ABSTRACT

SPECTROGRAPHIC, PAUSIMETRIC, AND INTELLIGIBILITY MEASURES IN PARKINSON'S DISEASE

By

Dennis C. Tanner

The purpose of this study was to analyze acoustically and perceptually the speech of Parkinson subjects who were receiving anti-parkinson medication. The subjects used in this study consisted of 12 patients, 6 male and 6 female, diagnosed by physicians as having Parkinson's disease or parkinsonism. Each sex category was further subdivided into 3 subjects with severe symptoms and 3 subjects with mild symptoms. A group of 12 age and sex matched individuals was used as a control.

The following questions were formulated to define this research:

1. Is there a difference between Parkinson and normal subjects' duration of articulatory pauses, using the operational definition of a pause and the method of measurement introduced by Tosi (1965)?

2. Is there a significant difference in the Euclidian distances between the mean frequency of the first and second

formants of vowels uttered by Parkinson subjects and the same vowel uttered by normal subjects?

3. Do the Parkinson subjects differ significantly in intelligibility from normals as measured by the Multiple Choice Intelligibility Test by Black (1963)?

4. How do untrained judges perceive the speech of Parkinson subjects, in particular:

A. How do untrained judges perceive isolated vowel-bracketed plosives produced by Parkinson subjects?

B. Is there a difference between Parkinson subjects' and control subjects' production of isolated vowel-bracketed plosives as determined by untrained judges?

The distribution of articulatory pauses from the Rainbow Passage for both the Parkinson group and the control group were compared statistically for differences in mean duration of the pauses. The results of the statistical analysis indicated the control subjects' mean duration of articulatory pauses to be longer than the mean duration of the articulatory pauses for the Parkinson subjects.

Spectrograms of the cluster and CNC samples were obtained to determine the mean frequencies of the first and second formants of the following vowels for both the Parkinson group and the age-matched controls: /I/, / ϵ /, /ae/, /a/, / Λ /, /3/, /o/. Euclidian distances from the control subjects' mean frequencies and the Parkinson's subjects' mean frequencies were tested statistically for significance. The statistical analysis indicated no significant differences among subjects of each subgroup but significant differences among Parkinson and normals of corresponding subgroups were observed. Although no interaction was observed across the various subgroups, there was some homogeneity between vowelsex-formant number-type of diagnosis with the amount and direction of the Parkinson groups' frequency deviations.

Data on intelligibility of the Parkinson subjects was obtained by using the Black Test of Intelligibility and was compared with the normalized data provided with the test. The results of the intelligibility analysis suggest that Parkinson subjects diagnosed by their physicians as possessing either mild or severe involvement, will have reduced intelligibility corresponding to the severity of the disease. The results also suggest that women with a severe diagnosis have better intelligibility than men with the same condition, and vice versa for subjects with a mild diagnosis.

The perceptual features regarding plosive production were judged by a panel of untrained judges. The results indicated that judges perceive the isolated vowel-bracketed plosives as, in decending order of occurrence, a) other nontested plosives, b) fricatives, c) nasals, and d) glides. However, the plosive analysis also indicated that the Parkinson groups' production of isolated plosives were as intelligible as an age-matched control group. The major conclusions regarding the plosive analysis are that 1) the Parkinson subjects'difficulty in the production of plosives reported in the literature is only true relative to ongoing speech, and/or 2) the widespread use of anti-parkinson medication has improved the overall populations' production of plosives to the extent that they no longer differ from age-matched normals.

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Ву

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CHAPTER I

INTRODUCTION

Parkinson's disease, a disorder of the extrapyramidal system, is one of the major chronic disabilities of adulthood which creates a need for residential nursing care. The progression of the syndrome may range over twenty or more years. The disorder is characterized by muscular rigidity, tremor at rest, and impairment of voluntary movements. The speech of individuals with Parkinson's disease is often greatly disturbed and may include deviation in the normal processes of respiration, phonation, resonance, prosody, and articulation.

Cramer (1940), Nielson (1941), Merritt (1955), and Hoberman (1958) were some of the early authors to examine the verbal symptoms associated with the disease. Most of these early studies described perceptual features and were often limited by the lack of precise methodology and instrumentation.

Within the last fifteen years, the research into the verbal manifestations of Parkinson's disease has become more precise and abundant. Canter's (1963, 1965a, 1965b) three part analysis of Parkinson speech was the first comprehensive analysis of the respiratory, articulatory, and phonatory

characteristics of the disease. Canter observed imprecise production of plosive consonants and discoordination of phonatory and articulatory activity to be the main contributors to defective articulation. Meldolesi (1954), Brombart (1961), Leanderson et al. (1972), Logemann et al. (1973), and Darley et al. (1975) have also observed lingual and labial mobility deficits to be associated with the disease.

Schilling (1925), Nugent et al. (1958), De La Torre et al. (1960), Ewanowski (1964), and Kim (1968) are generally in agreement that decreased maximum breathing capacity and faster respiratory rates are symptomatic of Parkinson's disease. The phonatory characteristics in Parkinson's disease have been studied by Neilson (1941), Merritt (1955), Grewel (1957), Darley et al. (1969b), and Darley et al. (1975). Many of these studies report contradictory results and are thoroughly discussed in Chapter II.

The research is limited regarding certain acoustic aspects of Parkinson's disease. Lehiste (1965) included a Parkinson speaker in her spectrographic analysis of dysarthria. Canter (1963) defined a pause as "stoppage of speech" and referred to them in determination of the durational qualities of the syllable.

In addition to the fact that there is little research available specifically addressing the acoustic aspects of Parkinson's disease, most of the previous research was

completed before the widespread use of anti-parkinson medication. The use of levodopa (L-Dopa) and other anti-parkinson medication has, in effect, altered the "typical" clinical symptoms reported in early research. Nakano et al. (1973) report significant increase in overall speech intelligibility and improved labial movements with levodopa therapy. Yahr and associates (1968), Lee et al. (1971), Stern et al. (1972), Hunter et al. (1973), and others report mild to dramatic benefits from levodopa therapy. Today, most patients with Parkinson's disease receive varying dosages of anti-parkinson medication.

Purpose of the Study

The purpose of the present study was to analyze acoustically and perceptually the speech of Parkinson subjects who are receiving anti-parkinson medication. The following questions were formulated to define this research:

1. Is there a difference between Parkinson and normal subjects' duration of articulatory pauses, using the operational definition of a pause and the method of measurement introduced by Tosi (1965)?

2. Is there a significant difference in the Euclidian distances between the mean frequency of the first and second formants of vowels uttered by Parkinson subjects and the same vowel uttered by normal subjects?

3. Do the Parkinson subjects differ significantly in intelligibility from normals as measured by the Multiple

Choice Intelligibility Test by Black (1963)?

4. How do untrained judges perceive the speech of Parkinson subjects, in particular:

A. How do untrained judges perceive isolated vowel-bracketed plosives produced by Parkinson subjects?

B. Is there a difference between Parkinson subjests' and control subjects' production of isolated vowel-bracketed plosives as determined by untrained judges?

Definition of Terms

Definitions of major terms employed in this study are as follows:

<u>Articulatory pause</u>. As defined by Tosi (1965), articulatory pauses are ". . . flow of acoustic energy of which the relative amplitudes remain below a predetermined value of a parameter called 'pause maximum amplitude, L_p ,' provided the duration of such levels of amplitude is more than a predetermined amount of time, indicated by another parameter called 'pause-minimum duration, T_p .' The parameter L_p is expressed as a percentage or dB ratio with respect to the average peak amplitudes (pressure or voltage) of the rectified waves of the sample of sound analyzed. The parameter T_p is expressed in milliseconds" (p. 134).

<u>Formant</u>. Wood's (1971) definition of a formant is used in this study to designate a natural mode of resonance in the vocal tract characterized on a spectrogram by a dark area indicating a relatively high intensity of a group of frequency components in a vowel.

Intelligibility. In this study, intelligibility is defined as the capacity to be understood or comprehended; that which is measured by the Multiple Choice Test of Intelligibility by Black (1963).

<u>Plosive</u>. "Any speech sound made by creating air pressure in the vocal tract and suddenly releasing it" (Wood, 1971, p. 18). In this study, the plosives were "vowel-bracketed" or produced between the articulation of two identical vowels. The samples are isolated in the sense that the vowel-bracketed plosives were not taken from ongoing speech samples.

Parkinson's disease. In this study, Parkinson's disease is used synonymously with parkinsonism and paralysis agitans. The subjects in this study were labeled as having Parkinson's disease if their physician indicated Parkinson's disease, parkinsonism, or paralysis agitans upon diagnosis and had prescribed anti-parkinson medication.

Organization of the Report

Chapter I has introduced some of the deficits in the research dealing with the verbal symptomatology in Parkinson's disease. The studies reported in the introduction are more

thoroughly discussed in the literature review. Also, in the introduction, the terms pertinent to this study were listed and defined.

Chapter II consists of a review of the literature including studies that relate to epidemiology, physical symptomatology, classification, and psychological factors associated with the disease. The available studies that relate to respiration, phonation, resonance, prosody, and articulation are also reported.

Chapter III discusses the selection of the subjects, equipment used in the study, and the procedure used to obtain the data.

Chapter IV contains a presentation of the results of the statistical analyses. Appropriate charts and tables are presented to clarify the results.

Chapter V includes a summary of the study and conclusions which can be drawn from the results. Recommendations for future research are also presented.

The Appendices contain the raw data utilized in the analyses.

CHAPTER II

REVIEW OF THE LITERATURE

Epidemiology

In 1817, James Parkinson published the "Essay on the Shaking Palsy" (Parkinson, 1817). The disease is now referred to by his name, "Parkinson's disease" or "parkinsonism." Epidemiological studies are generally in agreement regarding the prevalence of the disorder. Pollock and Hornabrook (1966) report the prevalence of parkinsonism in Wellington, New Zealand, to be 106 per 100,000 population. Jenkins (1966) found a prevalence of 85/100,000 in the Australian state of Victoria. Brewis et al. (1966) found an overall rate of 112.5/100,000 in a survey of the city of Carlisle, England. A survey of patients seen at the Kenyatta National Hospital, Nairobi, during 1965, indicates that 75 patients were admitted for neurological diseases; two with parkinsonism (Ojiambo, 1966). Hamdi (1966) reviewed 24 cases of parkinsonism admitted to the Neurological Unit of the Republic Teaching Hospital in Baghdad, Iraq, in the years 1958, 1959, and 1960 and recorded a 1.1 percent occurrence of parkinsonism from the patients studied during those years. Hsueh and Jeang (1966) reported 1.5 percent occurrence of parkinsonism of patients 60 years and older in a large general hospital in Taiwan.

In the United States, Blumenthal (1965) reviewed the patient records of a Veterans Administration psychiatric hospital and reported that of 2,265 patients, those with "paralysis agitans" constituted 1.6 percent of the patients with neurological diseases and 0.2 percent of the total patient population. Hussar (1966) reviewed the autopsy protocols of 1,276 white male schizophrenic patients who died at age 40 or over during 1954-59 in Veterans Administration hospitals and reported that a clinical diagnosis of paralysis agitans had been made in 12 or about 1 percent. Autopsy confirmed eight of the original diagnoses. In a survey of the utilization of institutional facilities in Kansas, Lewis (1965) found that 268 (2.1 percent) of 12,869 admissions to adult care homes were for Parkinson's disease. Lewis also concluded that about 0.1 percent of patients hospitalized in short-term general hospitals were given the diagnosis of Parkinson's disease at discharge. Goodman (1953) proposes that parkinsonism is one of the major chronic disabilities of adulthood creating a need for residential nursing. DeJong (1958) estimated the prevalence of the syndrome in the United States to be between 1,000,000 and 1,500,000. The U.S. Department of Health, Education, and Welfare (1968) suggests that the prevalence of parkinsonism is generally uniform among widely scattered geographical regions and in different racial groups. H.E.W. concludes that, "Patients with parkinsonism comprise between 1 to 2 percent of patients with neurological disease" (p. 3).

Physical Symptomatology

In the incipient stage of the development of the disorder, the patient may have a gradual slowing down of activities. Early symptoms include diminishing of normal reciprocal movements, slight changes in facial expression, some loss of dexterity, and some difficulty in performing small coordinate movements such as shaving, dressing, manipulating buttons, and writing (Eliasberg, 1959). The first symptom may be a slight trembling of the thumb or monotone voice characteristics (Von Werssowetz, 1964).

Rigid muscles, often seen in Parkinson's disease, are usually equal in degree in all opposing muscle groups, although frequently unequal in intensity on opposite sides of the body and rotary movements of the trunk are markedly diminished (Von Werssowetz, 1964). The body and its segments often assume a general attitude of flexion (Wachs and Boshes, 1961). The rigidity often causes loss of automatic unconscious movements. Blinking of the eyelids and changes of facial expression may be affected early. Facial muscle rigidity produces an expressionless face. Von Werssowetz (1964) attributes the typical "mask-like" face of Parkinson's disease to contribute to speech difficulties. Rigidity often causes akathisia (restlessness) of the extremities (Hodge, 1959).

Tremor is another significant symptom in parkinsonism. The tremor is generally rhythmic, slow, and occurring at a rate of three to six movements per second, and alternating

between opposing muscle groups. Tremor of the tongue and jaw may be present (Von Werssowetz, 1964). Dyskinesia (loss of skill) in the upper limbs is common in the disorder and slowness of voluntary movements is often present although muscle power is usually adequate (Oliver, 1967).

Oculogyric crisis may be present in certain types of parkinsonism. During the crisis the eyes will often turn up forcefully and is commonly seen in the post-encephalitic type but seldom occurs in the idiopathic disease (Von Werssowetz, 1964). Occasionally, patients with Parkinson's disease experience a "Parkinson's crisis" as a result of psychological trauma or sudden withdrawal of anti-parkinson The Parkinson's crisis is a sudden severe exacerbadrugs. tion of tremor, rigidity, and dyskinesia accompanied by acute anxiety, sweating, tachycardia, and hyperphoea and may be accompanied by oculogyric crisis (Oliver, 1967). Many of the other symptoms of the disorder are probable combinations of tremor, rigidity, or dyskinesia. Von Werssowetz (1964) reports that many of the major signs of parkinsonism usually disappear during sleep.

Chusid (1973) reports that disturbed metabolism of brain amines may possibly be the basis for the development of parkinsonism. "Abnormally low concentrations of dopamine, norepinephrine, and serotonin in the basal ganglia and hypothalamus and their abnormal excretion in the urine have been reported, suggesting a defect in normal binding of amines" (p. 334). Von Werssowetz (1964) states that any dysfunction

of the extrapyramidal system can cause parkinsonism. It is, however, difficult to define exactly what the extrapyramidal system includes. Generally it is accepted that all motor mechanisms of the central nervous system, excluding those of the pyramidal tract are "non-pyramidal" or extrapyramidal motor systems (Jung and Hassler, 1960). Kreig (1953) defines the extrapyramidal system to include the extrapyramidal area of the cerebral cortex of each hemisphere, the thalamic nucleus, corpus striatum including caudate nucleus, putamen, pallidum, sub thalamus, and the rubrul and reticular systems. These structures are all loosely connected by numerous tracts which create a functional rather than an anatomic system. It is believed that the extrapyramidal system causes facilitation of impulses which pass from higher centers to the lower motor neuron cells of the agonist muscles, while at the same time suppressing the activity of the antagonist muscles (Asai and Schaltenbrand, 1960). Von Werssowetz (1964) lists the functions of the extrapyramidal systems: 1) integration and facilitation of primary movements of skeletal muscles, 2) integration of associated and ancillary movement patterns, and 3) control and inhibition of postural reflexes.

Chusid (1973) states that the "extrapyramidal system may be regarded as a functional system with 3 layers of integration: cortical, striate (basal ganglia), and tegmental (midbrain). The bulboreticular inhibitory and facilitatory area receives fibers from cerebral cortical

areas, the striatum, and the anterior cerebellum. The principal functions of the extrapyramidal system are concerned with associated movements, postural adjustments, and autonomic integration. Lesions at any level may obscure or abolish voluntary movements and replace them with involuntary movements" (p. 16).

Classification

Since James Parkinson's essay was published, many scientists and clinicians have classified the disorder. Von Werssowetz (1964) separates Parkinson's disease from the parkinsonian syndrome. Parkinson's disease is divided into idiopathic and arteriosclerotic categories and parkinsonian syndrome includes post infectious, toxic or metabolic, drug induced, and miscellaneous. According to Von Werssowetz (1964), Parkinson's disease differs from the parkinsonian syndrome by the absence of cortical involvement, lethargy, oculogyric abnormalities, and slowing and enfeeblement of emotional responses and behavioral disturbances. Generally, however, studies do not separate Parkinson's disease from parkinsonism. Oliver (1967) provides the following classi-

fication system:

Idiopathic Postencephalitic Atherosclerotic Drug induced Carbon monoxide poisoning Chronic manganese poisoning Chronic mercury poisoning Midbrain compression Traumatic

Controversy still exists regarding the pathologic differences among the various clinical forms of the disease. To compound the classification difficulties, no conclusive evidence exists that lesions of a specific structural component in the extrapyramidal tract are responsible for a specific symptom in the classification category. It is, however, generally accepted that there are characteristic differences between clinical forms of Parkinson's disease. Generally, it is believed that in the post-encephalitic form the substantia nigra is significantly affected (Spatz, 1925; Wolf, 1954). The globus pallidus, putamen, and the caudate nucleus are affected in the idiopathic type (Lhermitte, 1921; Keschner and Sloane, 1931; Vogt and Vogt, 1920). In the arteriosclerotic form of the disease, the most severely affected are the globus pallidus, striatum, and external capsule (Davidson, 1952; Denny - Brown, 1946). In many forms of the disease the cortex reveals significant changes (Benda, 1952; Davidson, 1942) and in certain instances the damage is particularly severe in the temporal lobes. Generally, in Parkinson's disease, the substantia nigra is considered as the site where pathologic alterations are most important, regardless of the suspected etiology (Greenfield, 1958; Baker and Nelson, 1959).

Psychological Factors

Studies describing the psychological, emotional, or mental status of patients with parkinsonism are contradictory. Some studies report widespread intellectual

impairment; others show focal brain damage. Emotional constriction and rigidity with impaired perceptual flexibility are often reported. Other studies report little or no emotional or intellectual deficits (H.E.W., 1968).

In Parkinson patients without severe motor deficits, Levita et al. (1964) found a decrease in verbal functions and impairment of arousal of attention and the maintenance of They were unable to relate cognitive deficits to the set. patient's age, voluntary movement impairment, or the laterality of sub-cortical involvement. As the disease increased in severity, they found consistent intellectual, cognitive, and perceptual losses. Nadvornik (1962) suggests that as the disease advances there is increased depression, organ fixation, and somatic preoccupation. Füenfgeld (1965) found the following psychological reactions in a study of 129 patients with the disease: 1) depression, 14.7 percent; 2) depression plus emotional lability or emotional inflexibility, 34.1 percent; 3) depression, emotional lability, and decreased spontaneity, 25.6 percent; 4) the above psychological abnormalities, plus disabling loss of interest and decreased vitality and will, 15.5 percent; 5) similar personality abnormalities with memory impairment and decreased judgment, decreased perception, concentration, insight and judgment, 10.1 percent.

Hartman-Von Monakow (1965) relates the mental status to the type of parkinsonism. The postencephalitic patients

demonstrate restlessness, irritability, and loss of inhibition especially when the disease develops early in life. When developed late in life, the postencephalitic parkinsonism patient is frequently characterized as having decreased initiative and spontaneity, slow psychic functioning, and decreased drive. Arteriosclerotic parkinsonism is often associated with disturbances of orientation, comprehension, memory, concentration, and affect as well as the same psychological changes reported in the postencephalitic parkinsonism.

Drugs and surgery also are reported to affect the psychological functioning in parkinsonism. H.E.W. (1968) reports, "Large doses of Dopa as amphetamine and related drugs can be expected to result in increased psychomotor activity which may be manifested by choreiform and athetotic activity, agitation, confusion, hallucination, and gross delirium" (p. 14). Hartman-Von Monakow (1972) found postoperative (stereotaxic surgery of the thalamus) psychological problems especially permanent psycho-organic symptoms and transient somnolence, hallucinations, and disorientation. Hoehn and Yahr (1969) studied a group of patients with parkinsonism and found about 25 percent had difficulty with speech, balance, and mental function after a unilateral thalamic lesion (172 cases), and further difficulty with these symptoms appeared after the contralateral lesion in 50 to 75 percent of the cases (43 cases). H.E.W. (1968) concludes that much of the psychological reactions seen in

parkinsonism are a mixture of disease, age, and drug effect.

Language Disorders

Studies differ regarding the extent of language deficits in unoperated parkinsonian patients. Although unoperated parkinsonism is generally associated with subcortical involvement (Levita, et al. 1964), specific language disturbances have been reported. Myers (1967) noted language disturbances with lesions of the thalamus. Ojemann and Ward (1971) have hypothesized that short-term verbal memory and verbal functions may be attributed to the dominant ventrolateral thalamus. Penfield and Roberts (1969) conclude, "The functions of all three cortical speech areas in man are coordinated by projections of each to parts of the thalamus and the elaboration of speech is somehow carried out by means of these circuits" (pp. 207-208). Levita and Riklan (1970) caution, however, that presence of neurological involvement is not necessarily associated with distinct verbal-cognitive changes in unoperated parkinsonism. Reitan and Boll (1971) report verbal, cognitive, and intellectual losses associated with the disease. Levita and Riklan (1973) conclude that the discrepancies in many studies may reflect differences in sampling.

Language deficits following surgery for relief of tremor and rigidity in Parkinson's disease have been widely reported in the literature (Cooper et al., 1968; Cooper, 1961; Mundinger and Riechert, 1963). Riklan and Levita

(1970) report the presence of language deficits, especially word-finding and alterations in articulation, in patients receiving subcortical surgery. Levita and Riklan (1973), using several scales of linguistic performance, conclude: 1) verbal-cognitive functions do not vary as a function of anterior-posterior, medial-lateral, or depth parameters of surgical lesion placement; 2) verbal-cognitive changes are not significantly or differentially related to lesion size or site within the confines of the ventrolateral nucleus of the thalamus.

Almgren et al. (1969) report that parkinsonian patients treated with a left-sided lesion as compared with a right-sided lesion, showed a more impaired postoperative performance on the Stroop Color-Word Test and a memory test for word pairs. In another study, Almgren et al. (1972) suggest that the differences in verbally expressed cognition between subjects with left-sided and right-sided ventrolateral thalamotomy extend well beyond the immediate postoperative period. Levita and Riklan (1973) report deficits in verbal functions decrease in degree or can no longer be observed within six months after surgery. Samra et al. (1969) examined 27 deceased parkinsonian patients who had undergone thalamic surgery and correlated premorbid language skills with the surgical lesions in each case. The following conclusions were drawn:

1. A lesion strictly confined to the ventrolateral nucleus of the thalamus may be followed by language and/or speech deficits.

2. No definite relationship existed between postoperative language or speech deficits and partial involvement of thalamic nuclei surrounding the ventrolateral nucleus; H fields of Forel; subthalamic nucleus or red nucleus. Also, mild encroachment on the internal capsule could be tolerated without language or speech deficits, as long as the pyramidal tract remained intact.

3. The size of the lesion was not related to postoperative language and/or speech deficits.

4. Postoperative language deficits were mild and improved in time; whereas speech disturbances could be either mild, moderate, or severe.

5. When language deficits did occur, they followed surgery on the left dominant hemisphere in most instances. In contradistinction, no definite relationship was found between the side of surgery and speech deficits.

6. Language and speech disturbances are most frequently associated with bilateral rather than unilateral surgery, regardless of the cerebral hemisphere involved in the second operation (p. 510).

Auditory Deficits

The existence of auditory disturbances in postencephalitic parkinsonian patients has been reported by Barontini and Cannizaro (1956). They found consistent audiometric results which suggest that patients suffering from this form of Parkinson's disease have hypoacusia of the "receptive type." Other studies suggest middle ear pathologies (Torrini, 1956) and inner ear disorders (Ferreri, 1931) as reasons for the apparent loss in hearing acuity. There is also evidence that perceptual disorders may be associated with lesions in the basal ganglia (Mettler, 1955). Jerger et al. (1960), using a battery of auditory tests, provided a comprehensive evaluation of the hearing deficits associated with parkinsonism. The study of 16 patients with the disease concluded that involvement of the central auditory pathways exists in some patients. No appreciable difference between experimental and control groups were observed on routine audiometric measures such as the pure-tone and conventional discrimination tests. However, on more difficult auditory tasks, a deficit closely related to the clinical types of the disease was observed. Idiopathic patients did not perform as well as the controls in this study.

The "Acceleration" and "On-Off" Phenomena

Canter (1963) reports the speech qualities of some patients with Parkinson's disease to be characterized by a slow rate of articulatory positioning. Conversely, Darley et al. (1969b) refers to occasional "short rushes of speech" in parkinsonian dysarthria. Occasional occurrences of rapid speech has also been reported by Grewel (1967), Laszewski (1956), and West et al. (1957). This speed-up of the articulatory movements is referred to as the acceleration phenomenon. The paradox that exists, in this aspect of the speech of parkinsonian patients, is how a patient can have such disparate symptoms as rigidity and reduced range of movements in one instance and uncontrolled acceleration behavior in another.

Netsell et al. (1975) offer the hypothesis that "undershooting" is the basis for the perception of accelerated speech in some cases of parkinsonism. According to the authors, "The speaker might excite the rigid musculature with normally timed neural control signals for voluntary movements (neuromuscular control signals) only to have the speech articulators fail to reach the necessary position for production of a particular speech sound before beginning the movement for the following sound" (p. 170). This failure to reach the intended position is the basis for the acceleration phenomenon. The authors also report that one subject could gain control of the acceleration behavior by speaking louder.

The "on-off" phenomenon (akinesia paradoxica), which is a rapid spontaneous fluctuation of symptoms and signs of parkinsonism, has long been recognized as a characteristic feature of the disorder (Claveria et al. 1973). Transient attacks of dysphonia have been noted and attributed to the phenomenon. Since the introduction of levodopa, the phenomenon has been reported with increasing frequency (Yahr, 1972; Demasio et al. 1973; Markham, 1972). When they occur, the attacks accompanying L-Dopa therapy are reported to be more profound and prolonged. Damasio et al. (1973) report the phenomenon to occur in approximately 10 percent of the cases. Barbeau (1972) suggests that the "on-off" attacks represent levodopa toxicity and suggests upper dose

limits. Claveria et al. (1973), in a study that related plasma levodopa levels to confusion, facial hypokinesia, and dysphonia also concluded that the phenomena is related to excessive plasma concentrations of levodopa.

Medication--Effects on Speech

Many clinical reports are in agreement regarding the immediate effects of levodopa therapy in Parkinson's disease. Hunter et al. (1973) report that two-thirds of the patients gain modest to spectacular benefits from L-Dopa therapy. Other reports indicate that sustained response and in many cases arrest of the disease can occur during prolonged administration of the drug (Lee et al. 1971; Stern et al. 1972).

Reports of general speech changes associated with L-Dopa therapy have been reported. Berkmayer and Hornykiewicz (1961) observed improvement in speech and other symptoms during treatment with L-Dopa. Yahr and associates (1968) report that the immediate speech changes associated with L-Dopa therapy are not as dramatic as the improvement in other physical symptoms. Hunter et al. (1973), in a study of 187 parkinsonian patients, report that after two years of L-Dopa therapy, parkinsonian disabilities continued to increase despite retention of partial responsiveness to levodopa. The same study also reported that while most benefited after 3 month's treatment, after 36 months only 40 percent of the patients remaining in the study

maintained their initial improvement. One of the interesting facts to emerge was that by 24 months into the study, individual scores for tremor, rigidity, and posture were largely maintained although initial improvement of speech and sialorrhea (excessive secretion of saliva) had been largely lost. The authors also note that the clinical patterns of deterioration varied considerably from one patient to another.

Nakano et al. (1973), in a double-blind investigation, studied speech intelligibility (Black test) and labial movements in relation to the effects of procyclidine hydrochloride (Kemadrin), levodopa, a placebo (lactose), and no drug therapy. A significant increase in overall speech intelligibility with L-Dopa was reported. Concentric needle electrodes were inserted at six points in the mouth for EMG readings. Oral exercises, (which included: entire smile, smile on the left side of the mouth and then the right side, labial eversion, counting, and phoneme and diphthong repetition) were also evaluated and graded for tonic activity, amplitude and frequency, and symmetry of labial movements. The results suggest that levodopa was the superior drug for benefiting speech. Significant improvement was noted in labial eversion, counting, and phoneme and diphthong repitition for both procyclidine and levodopa. Also with levodopa, benefits in symmetry, amplitude, and frequency of labial movements were noted.

Parkinsonian Dysarthria

The characteristics of dysarthria are described in detail in the literature: Zentay (1937); Perello (1958); Aronson et al. (1968); and Darley et al. (1968). Mueller (1971) makes the following general conclusions regarding dysarthria: 1) the neuromuscular pathology underlying dysarthria appears to affect, to varying degrees, not only the articulatory but the phonatory and respiratory process of speech as well, 2) there are implications that the site and extent of the lesion determines, to a large extent, the speech pattern of the dysarthric, and 3) the need for further physiological research in dysarthria is obvious--especially the kind of research comparing various etiological groups of dysarthric individuals with one another and to the normal population (p. 333). Darley, et al. (1968) list seven subcategories of dysarthria: spastic, flaccid, combined spastic and flaccid, ataxic, hypokinetic, and two forms of hyperkinetic (quick and slow). Each of the above forms are reported to have unique combinations of deviation of the five basic motor speech processess: respiration, phonation, resonance, articulation, and prosody. According to the authors, parkinsonism produces "hypokinetic dysarthria" which:

. . . almost exclusively, is associated with repetition of initial sounds of words. This error suggests the occurrence of speech arrests analogous to the motor arrests observed in walking. Unusual prolongation of sounds is most frequently found in cerebellar disorders, where the timing of movements is faulty; however, one is likely

to hear prolongation of speech sounds in the hyperkinetic disorders and indeed in any patient whose rate of speech is substantially slowed. . . (p. 840).

Darley et al. (1969a), in an analysis of 32 patients with parkinsonism, found monopitch, monoloudness, and reduced stress to be the most striking phenomena in parkinsonism's hypokinetic dysarthria. Also, according to the authors, hypokinetic dysarthria is the only type of dysarthria in which the rate is not characteristically slow. As a group, hypokinetic dysarthrics are rated slightly faster than the other dysarthrias. Darley et al. (1969b) chart the neuromuscular effects seen in the dysarthrias. Table 1 shows the relationship between hypokinetic dysarthria and the other forms: CLR (cerebellar ataxia--ataxic dysarthria), PBP (pseudobulbar palsy--spastic dysarthria), BUL (bulbar palsy--flaccid dysarthria), ALS (amyotrophic lateral sclerosis--combined spastic and flaccid), PKN (parkinsonism--hypokinetic), DTN (dystonia--hyperkinetic) and CHO (chorea-hyperkinetic).

Tikofsky and Tikofsky (1964), in an intelligibility study of dysarthric speech, concluded that intelligibility testing can be employed to evaluate dysarthric speech. The authors were able to differentiate among dysarthrics in terms of listener based judgments and that intelligibility scores for normals differed markedly from the dysarthrics.

	CLR	NIQ	CHO	PBP	ALS	BUL	NXd
Direction of	Inaccurate	Inaccurate	Inaccurate due to	Normal	Normal	Normal	Normal
Movements	ataxia	slow in-	lesser degree,				
Rhythm (Timing	Irregular	voluntary movements Irregular	siow involun- tary movements Irregular	Regular	Regular	Regular	Regular
of Repetitive Movements Rate of Indivi-	Slow	Slow	Slow	Slow	Slow	Normal	Slow
dual Movements Rate of Repeti-	Slow	Slow	Slow	Slow	Slow	Normal	Fast
tive Move-							
ments Range of Indivi-	Excessive	Reduced to	Reduced to	Reduced	Reduced	Reduced	Reduced
dual Movements Range of Repeti-	to normal Excessive	normal Reduced to	excessive Reduced to	Reduced	Reduced	Reduced	Verv
tive Movements	to normal	normal	excessive				reduced
Force of Indivi-	Normal to	Normal	Reduced to	Reduced	Weak	Weak	Reduced
dual Movements	excessive Deduced	Fvccccim	excessive Avial often ev-	(paretic) Furessing	Funceita	Dedived	(paretic) Eveccine
concerning to prote		(biased)	cessive (biased)	(biased)	(biased)		(balanced)
			Extremities reduced				

Table 1. Neuromuscular defects in dysarthrias.

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Source: Darley, et al. (1969b).

Effects on Oral Mobility

Meldolesi (1954), in a study of postencephalitic cases, noted a delay in initiation of the swallow in some of the subjects. Calne et al. (1970) noted defective tongue movements in many of their subjects with Parkinson's disease. Brombart (1961) reported hesitant deglutition at the buccopharyngeal region in 5 of the 18 parkinsonian cases. Massengill (1967), in a cinefluorographic study, reported tongue thrusting patterns in all five parkinsonian patients studied. The author also reported that all five cases had experienced some difficulty swallowing. Logemann et al. (1973), in another cineradiographic study, reported that 12 of the 25 patients studied had lingual articulation disorders. According to the authors, "The lingual disability progressed from posterior to anterior, with labial involvement beginning sometime after posterior involvement of the tongue was evident" (p. 277). The authors conclude that lingual disability first affects swallowing and progresses to affect lingual mobility for speech and gradually moves from a posterior to anterior position.

Diadochokinetic rate is an effective measure of bradykinesia (abnormal slowness of movement) of the oral mechanism. It is known that the average diadochokinetic rate for normals is seven movements per second. Buck and Cooper (1956) measured the lingual diadochokinetic rates of 48 patients with Parkinson's disease and found that

three fourths of them had slow rates of 4 to 5 lingual alveolar movements per second. Although a "trend toward an association between a poor diadochokinetic rate and severe speech involvement" was noted, no significant relationship was found. Canter (1965) compared diadochokinetic performances of 17 parkinsonian patients with that of an age-matched group. He concluded that the parkinsonian group had impaired ability to perform rapid movements of the tongue tip, back of the tongue, lips, and vocal folds. The tonguetip involvement was observed to be typically greater than the lip involvement. It was observed that diadochokinesis was highly correlated with clarity of articulation, which was in turn highly correlated with over-all speech adequacy. Kreul (1972), in a study consisting of 23 patients with parkinsonism and control groups, concluded that syllable diadochokinetic rates fail to differentiate between normal subjects and subjects with parkinsonism. The author did however, determine that diadochokinetic rates for an interrupted vowel /i/ and a repeated vowel glide /u-i/ did differentiate between the two groups.

In general, labial muscles are normally activated in two functionally antagonistic groups for the production of rounding/closing and opening/spreading gesture movements (Leanderson et al. 1971). Parkinsonism affects the reciprocal innervation of the limb musculature (Schneider, 1968) and it has been suggested that the same type of disturbance

occurs in the facial musculature (Leanderson et al., 1972). In their study, it was revealed that a progressive articulatory deterioration of the stop consonants existed in patients with parkinsonism. Six muscles in one-half of the face were investigated and EMG examination indicated a well preserved reciprocal activation pattern initially. However, the second EMG examination revealed that the /b/-implosion involved no inhibition in the lip-opening muscles. The authors conclude, "Obviously, a deficient reciprocal activation seems to be the prime factor for the misarticulation of labial stop consonants, the production of which demands rapidly alternating closing and opening speech gesture components." In addition, L-Dopa was found to re-establish the reciprocal activation as well as reduce the hypertonicity in the subjects. Netsell and Cleeland (1973) report considerable success in removing undesirable lip retraction by using EMC feedback in one subject with complete bilateral retraction of the upper lip.

Darley et al. (1968) consider the articulatory errors in parkinsonism to be highly consistent. Darley et al. (1975) report that, "In the hypokinesia of parkinsonism, efficiency of articulation is diminished, since the range of movements is narrowed, the speed of single movements slowed, the speed of repetitive movements increased although their range is limited, and the force of movements reduced" (p. 187).

Doshay (1960) reports "slurred articulation" to be the most serious type of speech disturbance occurring in individuals with Parkinson's disease. Hoberman (1958) found labial and alveolar consonants to be the most defective phonemes found in the speech of parkinsonism. Hoberman suggests that parkinsonian patients perform normally for isolated voluntary movements and poorly for speech. Cramer (1940) reports that plosives lacked precision and were produced almost like fricatives in a group of postencephalitic parkinsonian patients. Canter (1965) also found imprecise production of plosive consonants and discoordination of phonatory and articulatory activity to be the main contributors to the defective articulation in his group of 17 patients with the disease. In a long term study to measure the effects of parkinsonism on control of the vocal tract, Logemann et al. (1973) observed fricatives to be produced with a reduction of constriction of the air channel. Radiographic analysis indicated that, "The tongue elevation was reduced in production of the affected speech sounds so that stop consonants . . . were converted to 'almost contacted' fricatives, produced at the same place of articulation" (p. 278).

Effects On Respiration

A number of studies deal specifically with the respiratory disorders in parkinsonism. Schilling (1925) reported respiratory deficits for both vegetative and speech

functions in a study of eight parkinsonian patients. Cramer (1940), in a study of six post-encephalitic parkinsonian patients, found 1) twice the normal frequency of inhalation, 2) negligible differences between vegetative breathing, profound breathing, and respiration during oral reading, 3) smaller than average vital capacities. 4) wastage of air before speaking, and 5) exhalation repeatedly interrupted by small inhalations. Laszewski (1956) attributes the speech deficits in most cases of parkinsonism to rigidity of the articulatory muscles than to restriction of vital capacity. Smith (1964), in a study of 23 parkinsonian patients, supports Laszewski's view by reporting a non-significant correlation (r = .05) between judged severity of speech defectiveness and vital capacity. Ewanowski (1964), in a study of 12 female parkinsonian patients, found no significant difference between guiet respiratory patterns between a parkinsonian group and a control group. He did, however, report that those with more severe neurologic impairment had faster respiratory rates.

Nugent et al. (1958) report decreased maximum breathing capacity and dyspnoea in 50 percent of parkinsonian patients. De La Torre et al. (1960) also reported reduced breathing capacity in 50 percent of the patients with the disease. In a study involving electromyography, Petit and Delhez (1961) found that the intercostal muscles interfered with respiratory function. The authors reported that the diaphragmatic

muscles appeared normal or near normal. Kim (1968), in a study of post-encephalitic parkinsonism, found respirometer tracings that indicated that during the resting state the patients had more rapid respiration with fewer variations in amplitude than the normals. The author considers the most significant finding to be that the post-encephalitic parkinsonians were limited in their ability to interrupt automatic aspects of respiration by volitional control as demonstrated by inability to hold breath.

Effects On Phonation

Most observations of the speech of parkinsonism refers to phonatory deficits. Merritt (1955) concluded that reduced loudness is a frequent characteristic of parkinsonism. Darley et al. (1968) observed reduced stress and monoloudness to be one of the major characteristics associated with the disease. Darley et al. (1969b) found two voice quality deviations, harshness and continuous breathiness, in their study of 32 adult subjects with parkinsonism. The authors attribute the occurrence of harshness, breathiness, and low pitch to the rigidity of laryngeal musculature.

A high vocal pitch has been reported by Nielson (1941) and Grewel (1957). Merritt (1955) however, observed a low voice to be associated with the disease. Canter (1963), in a well controlled study, measured the pitch levels (oscillograph) of 17 male patients with the disease and found that the parkinsonian group spoke at significantly

higher pitch levels than the age matched control group. The patients spoke at a median fundamental frequency of 129 cps on the average, while the control group was 106 cps.

Canter (1965) compared aged matched male parkinsonian patients with a control group in 1) their ability to produce different levels of vocal intensity, 2) the ability to phonate at extremely low and high pitch levels, and 3) to sustain phonation. To measure vocal intensity the author had each subject produce the syllable /no/ five times at four different levels of loudness: quiet, average, loud, and shouted. The readings were taken from a sound level meter. To determine vocal pitch measures, the subjects were instructed to produce the syllable /no/ at various pitch levels and ossillograms were made for measurement purposes. Sustained phonation was measured by having the subjects sustain the vowel /a/ three times in succession for as long as possible on a single breath. Canter concluded the following from the study: 1) The parkinsonian patients, as a group, were unable to produce "quiet" phonation at levels as low as those achieved by the control subjects. On "average" phonation, the two groups were essentially the same. The majority of the patients showed reduced ability to produce "loud" and "shouted" phonation. 2) Reductions in maximum pitch ranges were typical of the parkinsonian group. The parkinsonian patients averaged 1.25 octavies as compared to 1.84 for the controls. 3) The ability to sustain phonation

was impaired in the majority of the parkinsonian subjects (p. 49).

Darley et al. (1969b), in a study of 32 adult subjects with parkinsonism, reported monotony of pitch in 31 patients, monotony of loudness in all 32 of the subjects. Further, reduction of variability in pitch and loudness was reflected in the subjects' use of proper stress. All 32 subjects were judged to show some reduction of proper stress for emphasis.

Mueller (1971), in a study of ten patients with Parkinson's disease, matched them with a control group and found the parkinsonian patients to be reduced in the following areas:

 Phonation time during the sustained phonation of the vowel /a/,

2. Amount of air expended during the sustained phonations of /a/,

Phonation time for repeated productions of the syllable /s^/,

 Total number of syllables produced for repeated productions of /s^/,

5. Intraoral pressure during $/s^{/}$ productions. Mean oral airflow rates and volumes of air expended during repeated utterances of $/s^{/}$ did not differ statistically from the control group. However, the author suggests that the differences were indicative of respiratory and/or glottal

inefficiency in Parkinson's disease. The author concludes that the neuro-muscular involvement precludes the individual's ability to generate sufficient amounts of aerodynamic energy necessary for normal phonation and articulation.

Acoustic Aspects Of Dysarthric Speech

Lehiste (1965) analyzed the acoustic characteristics of dysarthric speech. Test material included three word lists for measurement purposes: 1) fifty monosyllabic CNC words, which are monosyllabic words with an initial and final consonant phoneme, 2) sixty monosyllabic words containing a number of initial and final clusters, and 3) fifty spondee words. The following measurements were made on each spectrogram:

 The center frequency of the first three formats at the target or steady state of the syllable nucleus.
 The duration of the syllable nucleus.

3. Formant positions at the point of transition form the initial consonant to the syllable nucleus and at the point of transition from the syllable nucleus to the final consonant.

4. The duration of the initial and final consonants.

5. The formant positions within the resonant consonants.

6. The frequency of energy concentrations in fricative consonants and in the releases of plosives.

Conclusions drawn from the study are as follows:

Deviations due to insufficient control of the voacl folds:

- 1. Laryngealization of syllable nuclei,
- 2. Laryngealization of consonants,
- 3. Breathy segments,
- 4. Voiceless transitions,
- 5. Devoicing of resonants.

Deviant pronounciations due to lack of control of the velum:

- 1. Denasalization of initial consonants,
- 2. Denasalization of final consonants,

- 3. Nasalization of syllable nuclei,
- 4. Nasalization of non-nasal consonants.

Deviations in articulation:

- 1. Palatalization,
- 2. Retroflexion,
- 3. Neutralization of the fortis-lenis opposition,
- 4. Distortions in the manner of articulation,
- 5. Distortions in the point of articulation,
- 6. Distortions in timing.

Lehiste reported the detailed articulatory deviations in the one Parkinson subject:

A considerable number of the distortions in the articulation of consonants involve changes in the manner of articulation. A sibilant /s/ was manifested as (Θ) (as in think) three times in initial position and seven times in final position. A bilabial voiceless fricative was produced in the words house and toss. The bilabial voiceless fricative was also once substituted for final /z/. Other substitutions for final /z/ included two occurrences of (Θ) and one occurrence of (\mathbf{f}) (as in ship). On the other hand, the sibilant /s/ was substituted for the final consonants in the words with and forth. A fricative was substituted for a plosive in nine cases. . . . The voiced velar fricative was also used once to replace final /r/ and once in place of initial /y/ (p. 57).

Lehiste also reported occasional substitution of glottal stops for final /t/ and /k/ phonemes. Legiste concludes that dysarthric subjects occasionally insert a pause in a sequence which does not correspond to a break between words. She observed the misplacement of the word boundary in spondee words in four instances.

Durational Qualities

Some authors have concluded that slow rates of speaking are common among parkinsonian patients (Brain, 1951; Grewel, 1957). Canter (1963) studied the durational

qualities of parkinsonism. He suggests that an individual's speaking rate depends on 1) the number of pauses, 2) the length of the pauses, 3) the length of the phrases, and 4) the duration of the spoken syllables. In his study, a pause was defined as the stoppage of speech. The duration was measured for each pause and the averaged values provided the "mean pause length." The length of a phrase was obtained by determining the time elapsed between successive pauses. Finally, syllable duration was measured using a phonation timer described by Steer and Hanley (1957). The study of 17 male parkinsonian patients and 17 male control subjects found the median speaking rate of the parkinsonian patients to be 172.6 words per minute as compared to 177.6 words per minute for the control group. Rate of speaking was determined by the time required to read the "Rainbow Passage" (98 words). However, statistical analysis revealed the differences in the two frequency distributions to not be reliable. Canter also did not find a statistically significant difference on the four measures of speaking rate. However, he did note individual cases of extreme deviation.

Summary

Individuals with Parkinson's disease comprise approximately 2 percent of the patients with neurological disease. The typical symptoms of muscular rigidity, tremor at rest, and impaired voluntary movements, influence to varying

degrees the speech production ability of these patients. It is believed that any dysfunction of the extrapyramidal system can result in Parkinson's disease. Abnormally low concentrations of dopamine, norepinephrine, and serotonin in the basas ganglia and hypothalamus suggest a deficit in the binding of amines.

Most research indicates some psychological reactions to the disease. The studies, however, do not agree on the specific psychological deficits that accompany or result from the disease. Depression and emotional lability are frequently reported. Many of the psychological reactions in Parkinson's disease are reported to be a combination of disease, age, and large doses of medication.

Language and auditory deficits have been reported. The language deficits are generally reported to accompany subcortical surgery for the relief of the parkinson symptoms. The results of the studies differ regarding the existence of language deficits in unoperated patients. The research regarding auditory deficits indicates that Parkinson subjects do not differ fron normals on pure tone and conventional tests. There is, however, some evidence that auditory deficits may be detected with more difficult auditory tests.

The dysarthria seen in Parkinson's disease has been labeled hypokinetic dysarthria. Deficits in respiration, phonation, resonance, prosody, and articulation have been reported. The verbal symptoms in Parkinson's disease are

often quite variable. Rapid fluctuation of symptoms have long been recognized. These "on-off" attacks have been reported more frequently since the introduction of L-Dopa and have been linked to excessive plasma concentrations of levodopa. The "acceleration phenomena," or short rushes of speech, have also been reported to accompany Parkinson's disease.

Studies indicating specific symptoms of monopitch, monoloudness, imprecise production of plosives, and reduced oral diadochokinesis in Parkinson's disease have been reported. The above deficits are reported to contribute to an overall reduced intelligibility.

There are no published studies involving spectrographic analysis of hypokinetic dysarthria. One spectrographic study of general dysarthria utilized a Parkinson subject in the sample. The author reported distortions of timing and confusion of word boundaries to be some of the characteristics associated with the speech of that subjects.

The recent widespread use of L-Dopa therapy and other anti-parkinson medication has resulted in reports of modest gain to complete arrest of the disease. Generally, reports indicate measurable improvement in most individuals. Specific improvement in intelligibility and labial movements has been reported to result from the anti-parkinson medication. Today, most patients receive anti-parkinson medication for relief of the Parkinson symptoms.

CHAPTER III

SUBJECTS, EQUIPMENT, MATERIALS, AND PROCEDURES

To reduce the confusion that may arise from a discussion of the apparatus and procedures employed in the four analyses, this chapter is divided into five sections. Section I, General Procedure, describes subjects, apparatus, and the procedure employed to collect speech samples from the Parkinson subjects and the control subjects. Section II, Pause Analysis, discusses the procedure and equipment employed in the detection and measurement of the articulatory pauses from these samples. Section III, Spectrographic Analysis, describes spectrograms from the speech samples and the procedures used to measure the mean frequencies of the first and second formants of seven vowels. Section IV. Intelligibility Analysis, discusses the apparatus and judging methods used in determining the overall intelligibility of the Parkinson subjects. Section V, Plosive Analysis, is divided into two parts. Part A concerns the procedure used to determine how the judges perceived the vowel-bracketed plosives. Part B explains the procedure employed to determine whether there was a significant difference between the Parkinson subjects' and control subjects' production of isolatedvowel bracketed plosives as determined by untrained judges.

Section I: General Procedure

Subjects

A total of 24 subjects were utilized in this study. All subjects spoke Standard American English of Midwestern dialect. Twelve of the subjects, six male and six female, were diagnosed by their physicians as having Parkinson's disease or parkinsonism. Each sex category was further subdivided into those diagnosed by their physicians as displaying severe Parkinson symptoms and those displaying mild symptoms. The subjects with Parkinson's disease were referred by their physicians or located from nursing home records. Seven of the twelve Parkinson subjects resided in nursing homes or medical care facilities. All subjects with Parkinson's disease were receiving anti-Parkinson medication and were history free of concomitant brain damage or other central nervous sytem dysfunction.

A control group, also history free of brain damage or central nervous system dysfunction, was sex and age matched $(\pm 3 \text{ years})$ with the Parkinson group. The Parkinson group mean age was 72.58 with a standard deviation of 10.67 years. The control group mean age was 72.50 with a standard deviation of 11.25 years. Table 2 lists the Parkinson and control subjects by age, sex, and severity of involvement.

	Parkinson Subjects			Contr	Control Subjects		
	Name	Sex	Age	Name	Sex	Age	
	A.F.	F	84	L.G.	F	84	
Severe	G.R.	F	78	B.C.	F	77	
	L.W.	F	81	G.C.	F	82	
	M.L.	F	72	A.D.	F	74	
Mild	B.F.	F	72	J.M.	F	72	
	B.R.	F	49	S.S.	F	46	
	C.R.	М	86	C.F.	M	83	
Seve re	C.D.	М	71	R.M.	М	74	
	J.K.	М	70	W.W.	М	72	
	B.S.	М	68	J.W.	М	68	
Mild	H.B.	М	81	L.S.	М	81	
	E.P.	М	59	B.B.	М	57	

Table 2. Parkinson and control subjects by age, sex, and severity of involvement.

Apparatus

The following apparatus was employed in the collection and analysis of the data:

Sound Spectrograph (Voice Identification, Inc.,
 VII, Series 700).

2. Pausimeter (Tosi, 1965).

3. Timer-Counter (Hewlett-Packard, 5326B).

4. High Speed Digital Printer (Mohawk Data Sciences Corp., Model 1200).

5. Open Reel Tape Recorder (Ampex Model AG600).

6. Amplifier and Speaker (Ampex Model 620).

 Portable Cassette Tape Recorder (Sony Solid State #110).

8. Audio Training Cassette Tapes (Memorex).

9. Magnetic Recording Tapes (Scotch AV 176, Low Noise).

10. CDC 6500 Computer.

Speech Samples

The following speech samples were used in this study:

1. <u>The Rainbow Passage</u>: A widely used, phonetically balanced passage presented in Appendix A.

<u>The Multiple Choice Test of Intelligibility by</u>
 <u>Black</u> (1963): List C was used and a sample of the form and scoring sheet is presented in Appendix B.

3. <u>Cluster and CNC monosyllables</u>: Monosyllables of a consonant-vowel-consonant variety. Complete lists are provided in Appendices C and D.

4. <u>Vowel-bracketed Plosives</u>: Four plosives /t/, /d/, /p/, and /b/ were bracketed by the vowels /I/, /a/, /u/ and are presented in Appendix E.

All samples were recorded on Memorex Audio Training Cassettes using a Sony Solid State Portable Tape Recorder #110. The samples were obtained at the subjects' place of residence. Room ambient noise levels were kept at a minimum. In the laboratory, the speech samples were transferred from the Audio Training Cassettes to the open Reel Magnetic Recording Tapes using the Ampex Model AG600 Reel Tape Recorder.

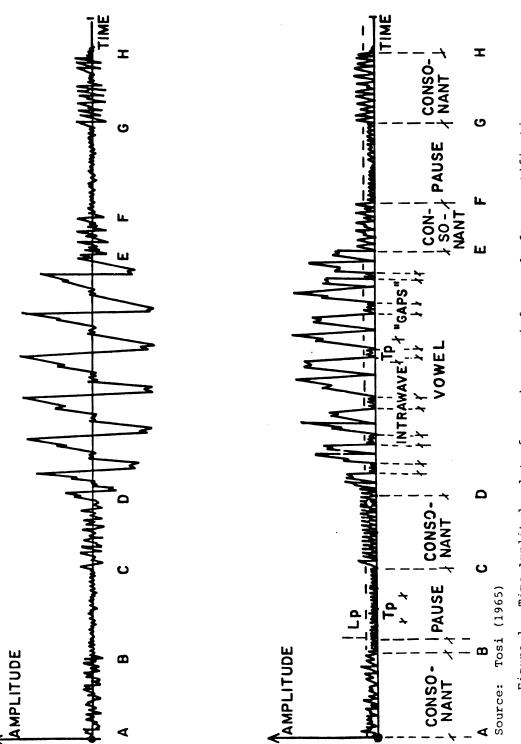
Section II: Pause Analysis

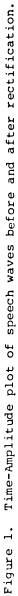
The purpose of the pause analysis was to determine whether there was a significant difference between the Parkinson subjects' and normal subjects' duration of articulatory pauses. Two minute samples of the subjects' reading of the Rainbow Passage (Appendix A) were used as the sample of ongoing speech. The tapes containing the Rainbow Passage were edited to remove any external noise. The operational definition of a pause, the method of pause detection, and the method of measuring the duration of a pause proposed by Tosi (1965) was followed in this analysis.

Tosi (1965) defined a pause as:

. . . flow of acoustic energy of which the relative amplitudes remain below a predetermined value of a parameter called 'pause maximum amplitude, L_p,' provided the duration of such levels of amplitude is more than a predetermined amount of time, indicated by another parameter called 'pause-minimum duration, T_p.' The parameter L_p is expressed as a percentage or dB ratio with respect to the average peak amplitudes (pressure or voltage) of the rectified waves of the sample of sound analyzed. The parameter T_p is expressed in milliseconds (p. 134).

Figure 1 illustrates the above definition. The speech wave is represented before and after rectification. According to Tosi's (1965) operational definition, only





the segments of the speech wave between BC and FG are "pauses."

Two parameters determine the boundaries of a pause. The first parameter, pause maximum amplitude (L_p) , puts an upper limit to the relative range of the acoustic amplitude. Pause maximum amplitude is expressed as a percentage with respect to the average peak amplitudes (V peak) of the ongoing sound (Rainbow Passage). According to Tosi (1965):

$$L_p = 100$$
 (V_L/V_{peak}) (% of V_{peak})

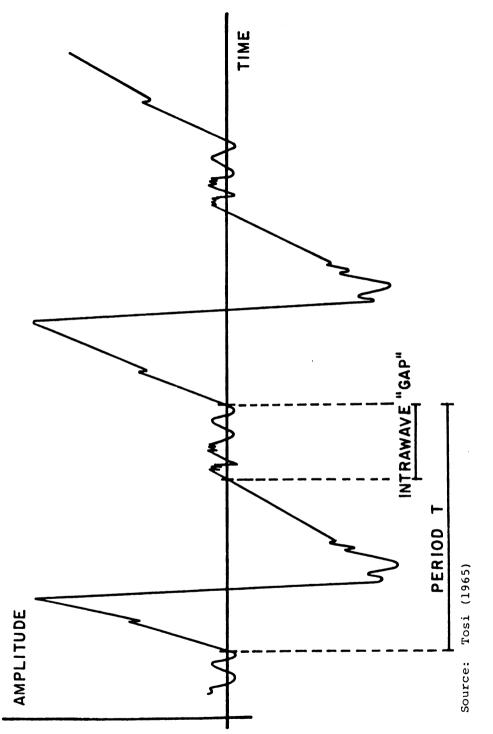
or alternatively:

$$L_p = 20 \log (V_L/V_{peak})$$
 (dB re V_{peak}).

Where:

V_{peak} = average peak amplitudes (pressure or voltage)
 of the sample of on-going sound analyzed;
V_L = absolute value of the pressure or voltage
 which puts an upper limit to the amplitudes

within pauses of on-going sound analyzed. In this study it was adopted $L_p = 5$ % V_{peak} because at that level no phonetic content was detected. The need for the second parameter arises from the concept of "interwave gap." This term refers to moments of relatively little amplitude as seen in Figure 2. Most complex waves of a fundamental frequency of 100 Hz and 150 Hz will have an interwave gap

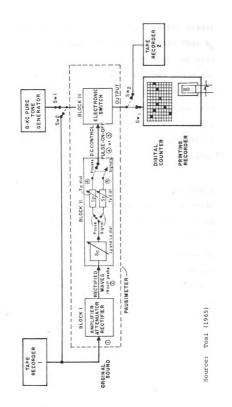




duration of less than 10 and 6.6 milliseconds respectively (Tosi, 1965). Most pauses are longer than these durations. Thus, the second parameter, pause-minimum duration (T_p) , is needed to define the other boundary of a pause. In this study it was adopted that $T_p = 5$ milliseconds because at this minimum duration interwave gaps were avoided for all the subjects' speech waves.

Tosi (1965) labeled and described the device and associated instruments for detecting and measuring pauses from ongoing speech as a "pausimeter." A block diagram of the instrument is provided in Figure 3.

The Ampex AG600 Reel Tape Recorder, playing back the Rainbow Passages, is connected to Block I of the pausimeter. Block I consists of an amplifier, an attenuator, and a rectifier. According to prescribed methods of operation, the amplifier or attenuator was adjusted so that the average amplitude peaks of the speech remained at approximately 0 level as indicated by a panel instrument. The output of Block I, consisting of the rectified speech waves with their peaks at a level of approximately 14 volts, is connected into the input of Block II. This unit consists essentially of three Schmitt trigger circuits. The first one, S_c , is activated only when the relative amplitudes of incoming rectified waves of the original sound are smaller than the value of L_p or greater than the value of L_s selected on a decade dial on the panel of the pausimeter. The output of





this Schmitt trigger S_c consists of trains of square waves lasting the same amount of time as the original sound wave trains of relative amplitudes smaller than L_p (pauses) or greater than L_s (signals). Square waves ceased when the relative amplitudes of the original sound waves became less than L_p (pauses) or smaller than L_s (signals). This output is fed into a second Schmitt trigger circuit, S_p , if the Pausimeter was processing pauses (switch in position "PAUSE"), or into a third Schmitt trigger circuit, S_s , if the Pausimeter was processing signals (switch to the position of "SIGNAL").

In the first case, Pausimeter processing pauses, the Schmitt circuit S_p is activated after T_p milliseconds when in its input there are no square waves coming from S_c .

Paritcular values of T_p are introduced by selecting a slope of a RC integrator through a panel dial T_p . This selected slope intercepts the S_p fixed triggering level at a point in time which is placed T_p milliseconds after no square waves are present in the S_p input. At the instant this interception happens, S_p is activated, emitting a 10 volt DC pulse. This pulse terminates T_p milliseconds after a train of square waves coming from S_c enters S_p . This 10 volt DC pulse, which is the output of Block II, turned ON the Electronic Switch, Block III. Therefore, when the Pausimeter is processing pauses, this Electronic Switch remains ON as long as a pause from the original sound is present,

and OFF when a signal is present.

In the second case, Pausimeter processing signals, the Schmitt circuit S_s is activated after T_s milliseconds when in its input there are square waves coming from S_c. Particular values of T_c are introduced by selecting a slope of a RC integrator through a panel dial T_s; this selected slope intercepts the S fixed triggering level at a point in time which is placed T_c milliseconds after the square waves from S_c are present in the S_s input. At the instant this interception happens, S_s is activated, emitting a 10 volt DC pulse. Such a pulse terminates T_s milliseconds after the square waves coming from S ceases. This 10 volt DC pulse, which is the output of Block II, turns ON the Electronic Switch, Block III. Therefore, when the Pausimeter is processing signals, this Electronic Switch remains ON as long as a signal from the original sound is present, and OFF when a pause is present.

In connection Sw₁, trains of 8 kz pure tones are allowed to pass through the Electronic Switch, Block III, during the time it is turned ON. These trains are fed into the Hewlett-Packard, 5326B Timer-Counter which counts their durations in milliseconds. These durations are then printed in a temporal order on a paper strip by the Mohawk Data Sciences Corp., Model 1200 High Speed Digital Printer, which is connected to the digital Timer-Counter.

Section III: Spectrographic Analysis

The purpose of the spectrographic analysis consisted of determining if there were significant differences in the Euclidian distances between the mean frequency of the first and second formants of vowels uttered by Parkinson subjects and the same vowels uttered by normal subjects.

Euclidian distances for each subject/vowel are defined as:

$$d = \sqrt{(F_1 - F_1)^2 + (F_2 - F_2)^2}$$

where:

- F₁ = mean frequency of the first formant of each
 subject/vowel,
- F₂ = mean frequency of the second formant of each
 subject/vowel,
- F'1⁼ average mean frequency of the first formant of each vowel as uttered by all subjects within the same subgroup (i.e., female, mild parkinson), F'2⁼ average mean frequency of the second formant of
- each vowel as uttered by all subjects within the same subgroup,

Seven vowels were utilized in this study, namely: /I/, ϵ /, /ae/, /a/, / Λ /, / γ /, /o/.

The CNC and Cluster lists provided in Appendices C and D were used in the spectrographic analysis. The Voice Identification Inc., Sound Spectrograph Series 700 was used to produce the wide band bar spectrograms for each word. A picture of the Spectrograph used in this study is provided below (Figure 4). Because most of the energy in speech can be found below 4000 Hz, the frequency control was set to display the energy below the upper limit of 4000 Hz. Following prescribed methods of operation, the voltage levels as read by the V.U. meter were held constant for each spectrogram.

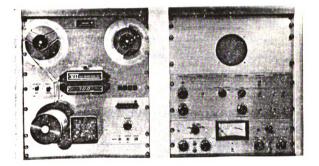


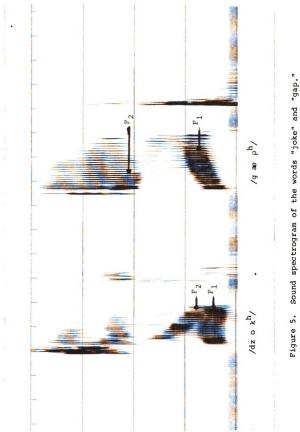
Figure 4. Voice Identification Inc. Sound Spectrograph Series 700

Essentially, the spectrograph performs a Fourier Analysis of 2.5 seconds of speech. The 2.5 seconds of speech is represented in terms of frequency along the vertical axis, time along the horizontal axis, and energy as darkened segments. A sample spectrogram is presented in Figure 5. The arrow indicates the dark resonance bars or formants.

Upon completion of each spectrogram, the appropriate vowel was labeled. The procedure for measuring the mean frequency of the first and second formants consisted of placing a clear plastic frequency overlay on each spectrogram and noting the mean frequency of the formant. The mean value of the formants for each vowel is presented in Appendices F-I. Calculations of Euclidian distances and the statistical analysis to test for significant differences was performed by using the CDC 6500 computer.

Section IV: Intelligibility Analysis

The purpose of the intelligibility analysis was to determine whether Parkinson subjects differed significantly in intelligibility from normals as measured by the Multiple Choice Test of Intelligibility by Black (1963). Form C was used and obtained from each Parkinson subject in the method described in the section "General Procedure." An example of the form and the scoring sheet is given in Appendix B.





Eighteen judges volunteered to participate in this analysis. It was arranged that three judges would respond to each subject. To reduce the effect of familiarity, no judge responded to more than two subjects. The following instructions were verbally given to each group of judges participating in this aspect of the study:

> You are going to hear subjects produce individual words. Please draw a line through the word on your answer sheet which most closely resembles the word you hear on the tape. There is no pattern to the answer. Base your choice only on what you hear.

For both the Intelligibility Analysis and the Plosive Analysis, the Ampex Model AG600 Reel Recorder and the Ampex Model 620 Amplifier and Speaker were combined to present the speech samples to the judges. Loudness levels were adjusted to a fixed volume. The judges were seated, facing the speaker at a distance of approximately twenty feet. The Voice Identification Project Office was used as the site for the judging and room ambient noise levels were kept at a minimum.

Section V (A & B): Plosive Analysis

The purpose of Part A of the Plosive Analysis was to determine how untrained judges perceived the vowel-bracketed plosives produced by the subjects with Parkinson's disease. Eight judges volunteered to participate in this aspect of the study. The eight judges were required to write what their perception of the test stimuli most closely resembled.

To reduce the effect of familiarity, the test stimuli was randomized. The following instructions were verbally given to each group of judges:

You are going to hear subjects produce consonants between two vowels. Please write on your response sheet your impression of what the consonant is. There is no pattern to the answer. Base your choice only on what you hear.

The results of the 96 response forms were transferred to the Plosive Analysis Open-Response Form A. An example of that form is provided in Appendix J.

The purpose of Part B of the Plosive Analysis was to determine if the Parkinson subjects' production of vowelbracketed plosives was perceptually different than similarly bracketed plosives of an age-matched control group. Judges were required to indicate, on a multiple choice form, which one of four vowel-bracketed plosives most resembled the stimulus. A copy of the Plosive Analysis Closed-Response Form B is provided in Appendix K.

Eighteen untrained judges, not participating in Part A, volunteered to participate in this aspect of the study. It was arranged that three judges would respond to each subject. To reduce the effect of familiarity, no judge listened to more than four subjects. Further, to reduce pattern responding by the judges, the taped stimuli was randomized. The following instructions were verbally given to each group of judges participating in Part B:

You are going to hear subjects produce consonants between two vowels. Please mark one of the four choices on your paper which most closely resembles the consonant you hear on the tape. You must respond to each item. There is no pattern to the answers. Base your choice only on what you hear.

CHAPTER IV

RESULTS AND DISCUSSION

The questions asked in this study were as follows:

1. Is there a difference between Parkinson subjects' and normal subjects' duration of articulatory pauses, using the operational definition of a pause and the method of measurement introduced by Tosi (1965)?

2. Is there a significant difference in the Euclidian distance between the mean frequency of the first and second formants of vowels uttered by Parkinson subjects and the same vowels uttered by normal subjects?

3. Do the Parkinson subjects differ significantly in intelligibility from normals as measured by the Multiple Choice Intelligibility Test by Black (1963)?

4. How do untrained judges perceive the speech of Parkinson subjects, in particular:

A. How do untrained judges perceive isolated vowel-bracketed plosives produced by Parkinson subjects?

B. Is there a difference between Parkinson subjects and normals in their production of isolated vowel-bracketed plosives as determined by untrained judges?

Pause Analysis

The purpose of the pause analysis was to determine if there was a significant difference between Parkinson subjects' and normal subjects' duration of articulatory pauses, using the operational definition of a pause and the method of measurement introduced by Tosi (1965).

Tables 3 and 4 indicate the mean articulatory pauses for normals and for each sub-group of Parkinson subjects. A two-way analysis of variance was completed to test for significant differences and sources of variation relative to the Parkinson subjects only. The results of that analysis are listed in Table 5. As the table suggests, sex, severity, and the interaction effect were not significantly different at the .05 (5.32) level of confidence. Significant difference was obtained beyond the 0.10 (3.46) level of confidence in the severity category.

To determine if there was a significant difference between Parkinson and control subjects' duration of mean articulatory pauses, a two-tailed Matched-Pair T-Test (T = 2.039) was computed. The results were non-significant at the .05 (2.201) level of confidence. Significant differences were obtained beyond the 0.10 (1.796) level of confidence. A one-tailed T-Test, hypothesizing longer mean pauses for the control subjects as compared to the Parkinson subjects, indicated a significant difference at the .05 (1.796) level of confidence. The results were non-significant at the .025 (2.201) level of confidence.

Sex	Severity	Name	Mean Duration Pause (msec.)	Standard Deviation (msec.)
F -		A.F.	435.78	1666.04
	Severe	G.R.	331.21	1680.43
		L.W.	234.90	843.36
		M.L.	184.99	664.60
	Mild	B.F.	238.71	396.66
		B.R.	125.08	553.93
М		C.R.	431.21	2011.92
	Severe	C.D.	442.23	30 39.33
		J.K.	179.07	805.71
	Mild	B.S.	158.97	793.42
		H.B.	238.47	1209.89
		E.P.	305.80	1037.02

Table 3. Mean and standard deviation of articulatory pauses extracted from the Parkinson group.

Average mean duration for Parkinson Females = 258.45 Average mean duration for Parkinson Males = 292.63

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Sex	Name	Mean Pause Duration (msec.)	Standard Deviation (msec.)
F	L.G.	257.26	1188.17
	B.C.	396.56	1150.72
	с.с.	320.10	1186.97
	A.D.	386.18	1107.15
	J.M.	877.82	3539.53
	S.S.	426.57	1599.83
м	C.F.	359.60	1398.37
	R.M.	499.90	2513.69
	W.W.	294.29	1070.28
	J.W.	295.09	1287.53
	L.S.	229.08	899.12
	в.в.	415.92	1662.62

Table 4. Mean and standard deviation of articulatory pauses extracted from the control group.

Average mean duration for Control Females = 444.08 Average mean duration for Control Males = 348.98

Source	d/F	Sums of Squares	Mean Squares	F-Ratio
Sex		3504.817	3504.817	. 34289
Severity	1	53651.139	53651.139	5.2489*
Interaction	1	898.562	898.562	.0879
Error	8	81770.644	10221.331	

Table 5. Analysis of variance of pause duration for Parkinson subjects.

* 5.32 required for significance at the 0.05 level of confidence.

Two additional two-tailed T-Tests were computed. The first T-Test (T = 0.0663) compared the six severe Parkinson subjects with their respective controls. The results of this test indicated non-significant differences beyond the 0.05 (2.571) level of confidence. The second test (T = 2.1897) compared the mild subjects with their respective control subjects and the differences were non-significant beyond the 0.05 (2.571) level but significant beyond the 0.10 (2.015) level of confidence.

The results of the Pause Analysis do not allow complete rejection of the hypothesis that there is no difference between Parkinson and normal subjects' duration of articulatory pauses. Significant differences were obtained at the 0.10 level of confidence and at the 0.05 level of confidence using the one-tailed T-Test.

The test hypothesis used in the Pause Analysis was purposely stated in non-directional terms because there was no previous research to indicate that Parkinson subjects would have longer or shorter articulatory pauses. It was thought, however, that because Parkinson subjects have generally reduced energy levels for walking, talking, and other motor functions, the mean articulatory pause (low energy levels) would be longer. However, the fact that there was an observed tendency for the control subjects to have longer mean pauses is not surprising, considering the observation by Darley et al. (1969a) that hypokinetic dysarthria is the only type in which the rate of speech is not characteristically slow. Thus, if the general rate of speech is faster, it seems plausible that the duration of articulatory pauses would also be shorter.

The indication that the control subjects displayed longer mean articulatory pauses than the Parkinson subjects may be related to the Acceleration Phenomenon observed in the Parkinson syndrome. Generally, the Acceleration Phenomenon is considered as "occasional" occurrences of rapid speech. The Pause Analysis suggests that the tendency for rapid articulatory patterning or acceleration may be present during all or most ongoing speech of the Parkinson subjects. Netsell's et al.(1975) hypothesis, that the speech articulators fail to reach the intended position before beginning the movement for the following sound may explain the

observation of shorter articulatory pauses in the Parkinson subjects. If the Parkinson subjects undershoot the articulatory positions, then the distance between BC and EF, as seen in Figure 1, would be necessarily shorter. These distances would be shorter because the high energy levels for the following sound would begin sooner--thus, shorter mean articulatory pauses.

Because the results of the Pause Analysis were only marginally significant, cautious acceptance of the above discussion is recommended.

Spectrographic Analysis

The purpose of the spectrographic analysis was to determine whether there was a significant difference in the Euclidian distances between the mean frequency of the first and second formants of vowels uttered by Parkinson subjects and the same vowel uttered by normal subjects.

Table 6 indicates the average mean frequency of the first and second formants for each vowel. The Euclidian distances between points determined by the mean frequency of F_1 and F_2 of each vowel, uttered by each subject, and the centroids determined by the average means for each subgroup of subjects were computed. A statistical analysis performed with these Euclidian distances showed no significant differences among subjects of each subgroup. Euclidian distances for normal subjects were submitted to the same treatment with similar results.

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		Fl	$^{\rm F}2$	F1	F2	F1	F2	н Н	^F 2	н Н	$^{\mathrm{F}}2$	Fl	^Р 2	ъЪ	$^{\rm F}_{\rm 2}$
	Normal	508	1967	608	2100	608	1967	692	1425	650	1483	658	1192	508	875
Female	Parkinson's Mild	467	2317	617	2050	567	2133	717	1550	667	1317	583	1050	533	933
	Parkinson's Severe	433	1733	517	2217	467	2100	750	1433	633	1200	583	1083	767	700
	Normal	425	1742	575	1725	592	1875	633	1292	542	1325	517	925	442	792
Male	Parkinson's Mild	483	1533	550	1717	583	1867	667	1200	633	1367	500	967	483	817
	Parkinson's Severe	500	1517	633	1333	567	1833	650	1183	650	1433	550	1000	500	833

Another statistical analysis showed that the Euclidian distances corresponding to the Parkinson subjects differed significantly in almost all cases from the distances of normal subjects for all vowels and subgroups studied. The Parkinson subjects' deviations, however, were not consistent. For instance, Table 6 indicates that the vowel /ae/ uttered by both female and male Parkinson subjects, with either severe or mild diagnosis, present average mean frequencies of F_1 smaller than the normals. In other cases, like the vowel /a/, the average for F_1 is larger for both males and females with either severe or mild diagnosis than for normal averages. The vowel /æ/ showed a decrease of F_1 and F_2 proportional to the Parkinson severity.

Although no interaction was observed across the various subgroups, there was some homogeneity between vowelsex-formant number-type of diagnosis with the amount and direction of the Parkinson groups' frequency deviation re to normals. However, the reduced population of subjects used in this study does not allow a solid conclusion regarding this aspect.

Comparison of the mean frequencies of F_1 and F_2 of each vowel studied from the normal subjects used in this study and normal subjects used in other studies, indicated no significant differences in spite of the fact that the subjects used in this study were older than those utilized in other studies (Gray and Wise, 1958).

Intelligibility Analysis

The purpose of the intelligibility analysis was to determine whether Parkinson subjects differed significantly in intelligibility from normals as measured by the Multiple Choice Intelligibility Test by Black (1963). The normalized percentage of answers should reach 100 percent for normal speakers and normal listeners using a good audio system.

The percentages obtained for Parkinson subjects, as judged by untrained listeners through the linear response system, are listed on Table 7. All of the percentages were submitted to a statistical test (analysis of variance). Results indicated a significant difference (0.01) re to the normal percentages of intelligibility.

Sex	Severity	Subjects Percent	Average Percent
	Mild	96.13 96.00 90.91	$\mu = 94.35$ SD = 5.21
Male ·	Severe	88.94 90.30 56.11	$\mu = 78.45$ SD = 18.05
	Mild	91.91 87.86 92.96	$\mu = 90.30$ SD = 6.28
Female	Severe	95.30 67.18 89.13	$\mu = 83.90$ SD = 14.25

Table 7. Intelligibility of Parkinson subjects in percent.

The percentages indicated by the judges of this test (3 for each subject) determined that there was not a significant difference among the judges. This was interpreted as an indication of the reliability of the test.

The results of the intelligibility analysis suggest that Parkinson subjects, diagnosed by their physicians as possessing either mild or severe involvement, will have reduced intelligibility related to the severity of the disease.

The decreased intelligibility of the Parkinson subjects must be attributed to articulation since the loudness levels were adjusted to a fixed volume in each case. Subjects with a severe diagnosis possess an intelligibility significantly less than the normals (up to 22 percent reduction). Subjects with a mild diagnosis present a reduction of only up to 10 percent re to the normal. The results of this analysis also suggest that women with a severe diagnosis have better intelligibility than men with the same condition, and vice versa for subjects with a mild diagnosis.

Plosive Analysis

The purpose of the Plosive Analysis Part A was to determine how untrained judges perceived the vowel-bracketed plosives produced by the subjects with Parkinson's disease. The data for all subjects is presented in Table 8. Overall, the judges were correct in their responses to the vowelbracketed plosives on 80.3 percent of the items. Of the

Number and percent of errors perceived as voiced plosives, unvoiced plosives, fricatives, glides, laterals, and nasals by eight judges for all Parkinson subjects. Table 8.

	Total Error	Voiced Plosives	Unvoiced Plosives	Fricatives	Glides	Laterals	Nasals
Unvoiced	126	06	16	11	3	0	9
Plosives	100%	71%	13%	9 8	28	80	5 %
Voiced	101	58	16	14	7	I	ß
Plosives	100%	57%	16%	148	78	18	5%
Total	227	148	32	25	10	Г	11
	100\$	668	148	118	48	8	58
	100\$	8(80%	118	48	8	58

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errors listed by the judges, 80 percent were perceived as other plosives, 11 percent as fricatives, 5 percent as nasals, and 4 percent as glides. Table 9 indicates that, of the errors in the severe category, 74 percent were perceived as other plosives, 11 percent as fricatives, 7 percent as glides, 7 percent as nasals, and 1 percent as laterals. Table 10 indicates that, of the errors in the mild category, 86 percent were perceived as other plosives, 12 percent as fricatives, 1 percent as glides, and 1 percent as nasals.

The results of the Plosive Analysis Part A supports Cramer's (1940) and Canter's (1965) observation that plosives lacked precision and were produced almost like fricatives. The untrained judges perceived plosives as fricatives on 11 percent of the stimuli. However, judges were significantly more consistent in confusing some plosives as other plosives. The trend was especially evident in the judges' perception of unvoiced plosives as voiced plosives. In addition, subjects diagnosed as severe by their physicians tended to have more errors listed as nasals (7 percent) than those diagnosed as mild (1 percent). However, judges perceived plosives as fricatives equally between the mild (12 percent) and the severe (11 percent) categories.

The purpose of Part B of the Plosive Analysis was to determine whether there was any difference between the Parkinson subjects' and the age-matched control subjects' production

Table 9.	Number and percent of errors perceived as voiced plosives, unvoiced
	plosives, iricatives, glides, laterals, and nasals by eight judges
	for severe Parkinson subjects.

	Total Error	Voiced Plosives	Unvoiced Plosives	Fricatives	Glides	Laterals	Nasals
Unvoiced	59	37	6	ى س	5	:	9
Plosives	100%	638	15%	98	38 38	1	10%
Voiced	72	41	10	6	٢	1	4
Plosives	100%	57%	148	138	10%	1	5
Total	131	78	19	14	6	Т	10
	100%	59%	15%	118	78	18	78
	100%	<i>۲۰</i>	748	11%	78	18	78

Number and percent of errors perceived as voiced plosives, unvoiced plosives, fricatives, glides, laterals, and nasals by eight judges for mild Parkinson subjects. Table 10.

	Total Error	Voiced Plosives	Unvoiced Plosives	Fricatives	Glides	Laterals	Nasals
Ilnuciced	67	53	L	9		0	0
Plosives	100%	79%	10%	98	28	ł	ł
Voiced	29	17	و	ъ	0	0	H
Plosives	100%	598	218	178	ł	;	ж
Total	96	70	13	11	1	0	Г
	100%	738	13%	12%	18	;	18
	100\$	86	86%	128	18	ł	18

of vowel-bracketed plosives as determined by untrained judges. Analysis of the data suggests that untrained judges perceive isolated vowel-bracketed plosives with equal efficiency between the Parkinson subjects and the agematched control group. Table 11 indicates the total correct and the total errors for each subject in each category. Overall, the judges were correct in their responses to the vowel-bracketed plosives on 81 percent of the items. This compares to the 80.3 percent correct response rate as reported in Part A. Thus, both methods of plosive analysis, open and closed response, were considered as equal indicators of the judges' perception of the vowel-bracketed plosives.

The mean, variance, and standard deviation for the Parkinson group was 29.17, 33.06, and 5.75 respectively. For the control subjects, the judges were correct in their responses on 78 percent of the items. The mean, variance, and standard deviation was 28.08, 22.81, and 4.78 respectively. A difference between means T-Test showed the control score not to be significantly higher at the .05 level of significance.

In addition to the anlaysis for all Parkinson subjects, overall comparisons were made between severe and mild Parkinson groups and their respective control groups. Table 12 indicates that the mild and severe Parkinson groups, when compared to their control groups, were equally

Total correct for Parkinson subjects and control subjects. Table 11.

ϵ Classi- Parkinson ϵ												
Severe A.F. 36 31 5 86.1 L.G. 36 29 7 80 Severe G.R. 36 22 14 61.1 B.C. 36 25 11 69 Severe L.W. 36 35 1 97.2 G.C. 36 20 16 55 Mild M.L. 36 29 7 80.6 A.D. 36 32 4 88 Mild B.F. 36 22 4 88.9 J.M. 36 32 4 88 Mild B.R. 36 32 4 88.9 J.M. 36 37 1 97 Severe C.R. 36 31 3 91.7 C.F. 36 20 16 55 Severe J.K. 36 15 21 41.7 R.M. 36 27 9 75 Severe J.K. 36 31 29.4 88.9 36 37 9 75 Severe </th <th>Sex</th> <th>Classi- fication</th> <th>Parkinson Subject</th> <th>*91dizzo¶</th> <th>ζοττεςτ</th> <th>Incorrect</th> <th>8 Correct</th> <th>Control Subject</th> <th>*91disso¶</th> <th>Correct</th> <th>Incorrect</th> <th>& Correct</th>	Sex	Classi- fication	Parkinson Subject	*91dizzo¶	ζοττεςτ	Incorrect	8 Correct	Control Subject	*91disso¶	Correct	Incorrect	& Correct
Severe G.R. 36 22 14 61.1 B.C. 36 25 11 69 Severe L.W. 36 35 1 97.2 G.C. 36 20 16 55 Mild M.L. 36 35 1 97.2 G.C. 36 20 16 55 Mild B.F. 36 32 4 88.9 J.M. 36 30 6 83 Mild B.R. 36 32 4 88.9 J.M. 36 30 6 83 Severe C.R. 36 31 3 91.7 C.F. 36 37 9 75 Severe J.K. 36 31 5 86.1 W.W. 36 27 9 75 Severe J.K. 36 31 29.4 88.9 36 37 91 75 Severe J.K. 36 J.W. 36 J.W. 36 27 9 75 Mild E.P.<	Ē4	Severe	A.F.	36	31	ъ	86.1	L.G.	36	29	2	•
Severe L.W. 36 35 1 97.2 G.C. 36 20 16 55 Mild M.L. 36 29 7 80.6 A.D. 36 32 4 88 Mild B.F. 36 32 4 88.9 J.M. 36 32 4 88 Mild B.R. 36 32 4 88.9 J.M. 36 37 1 97 Severe C.R. 36 32 4 88.9 S.S. 36 37 1 97 Severe C.R. 36 31 3 91.7 C.F. 36 27 9 75 Severe J.K. 36 31 5 86.1 W.W. 36 37 91 Mild B.S. 36 31 5 86.1 W.W. 36 37 9 75 Severe J.K. 36 31 29.4 88.9 36 7 9 75 Mild B.S.	٤ų	Severe	G.R.	36	22	14	61.1	B.C.	36	25	11	69.4
MildM.L.3629780.6A.D.3632488MildB.F.3632488.9J.M.3630683MildB.R.3632488.9S.S.3630683MildB.R.3632488.9S.S.3630683SevereC.R.3633391.7C.F.3627975SevereJ.K.36152141.7R.M.3627975SevereJ.K.3631586.1W.W.3633391SevereJ.K.3631586.1W.W.3633391MildB.S.3633391.7J.W.3633391MildG.B.3633391.7J.W.3632488MildG.B.3633391.7J.W.3627975MildE.P.3632488.9B.B.3627975MildE.P.3632488.9B.B.3627975MildE.P.3632488.9B.B.3627975MildE.P.3632488.9B.B.75975	ц	Severe	L.W.	36	35	Ч	97.2	G.C.	36	20	16	•
WildB.F.3632488.9J.M.3630683MildB.R.3632488.9S.S.3635197SevereC.R.3633391.7C.F.36201655SevereC.D.36152141.7R.M.3627975SevereJ.K.3631586.1W.W.3627975SevereJ.K.3631586.1W.W.3633391MildB.S.3631586.1W.W.3633391MildC.B.363129488.95637975MildG.B.3627129.4L.S.3627975MildE.P.3623129.4L.S.3627975MildE.P.3632488.9B.B.3627975MildE.P.3632488.9B.B.3627975Total4323508281.0Total4323379578	٤ų	Mild	M.L.	36	29	7	80.6	A.D.	36	32	4	•
WildB.R.3632488.9S.S.3635197SevereC.R.3633391.7C.F.36201655SevereC.D.36152141.7R.M.3627975SevereJ.K.36152141.7R.M.3627975SevereJ.K.3631586.1W.W.3633391MildB.S.3633391.7J.W.3632488MildG.B.36251129.4L.S.3627975MildE.P.3632488.9B.B.3627975MildE.P.3632488.9B.B.3627975Total4323508281.0Total4323379578	۲ų	Mild	В.F.	36	32	4	88.9	J.M.	36	30	9	•
Severe C.R. 36 33 3 91.7 C.F. 36 20 16 55. Severe C.D. 36 15 21 41.7 R.M. 36 27 9 75. Severe J.K. 36 31 5 86.1 W.W. 36 33 3 91. Mild B.S. 36 31 5 86.1 W.W. 36 33 3 91. Mild B.S. 36 33 3 91.7 J.W. 36 32 4 88. Mild G.B. 36 25 11 29.4 L.S. 36 27 9 75. Mild E.P. 36 32 4 88.9 B.B. 36 75. Mild E.P. 36 32 4 88.9 B.B. 36 27 9 75. Mild E.P. 36 32 8 81.0 Total 432 337 95 78. Total <td>ſщ</td> <td>Mild</td> <td>B.R.</td> <td>36</td> <td>32</td> <td>4</td> <td>88.9</td> <td>s.s.</td> <td>36</td> <td>35</td> <td>Ч</td> <td></td>	ſщ	Mild	B.R.	36	32	4	88.9	s.s.	36	35	Ч	
Severe C.D. 36 15 21 41.7 R.M. 36 27 9 75. Severe J.K. 36 31 5 86.1 W.W. 36 33 3 91. Mild B.S. 36 31 3 91.7 J.W. 36 32 4 88. Mild G.B. 36 25 11 29.4 L.S. 36 27 9 75. Mild E.P. 36 32 4 88.9 B.B. 36 27 9 75. Mild E.P. 36 32 4 88.9 B.B. 36 75. Mild E.P. 36 32 4 88.9 B.B. 36 75. Total 432 337 95 78. 78.	M	Severe	C.R.	36	33	m	91.7	C.F.	36	20	16	•
Severe J.K. 36 31 5 86.1 W.W. 36 33 3 91. Mild B.S. 36 33 3 91.7 J.W. 36 32 4 88. Mild G.B. 36 25 11 29.4 L.S. 36 27 9 75. Mild E.P. 36 32 4 88.9 B.B. 36 27 9 75. Mild E.P. 36 32 4 88.9 B.B. 36 27 9 75. Total 432 350 82 81.0 Total 432 337 95 78.	W	Severe	C.D.	36	15	21	41.7	R.M.	36	27	6	<u>с</u>
Mild B.S. 36 33 3 91.7 J.W. 36 32 4 88. Mild G.B. 36 25 11 29.4 L.S. 36 27 9 75. Mild E.P. 36 32 4 88.9 B.B. 36 27 9 75. Mild E.P. 36 32 4 88.9 B.B. 36 27 9 75. Total 432 350 82 81.0 Total 432 337 95 78.	W	Severe	J.K.	36	31	S	86.1	W.W.	36	33	m	91.7
Mild G.B. 36 25 11 29.4 L.S. 36 27 9 75. Mild E.P. 36 32 4 88.9 B.B. 36 27 9 75. Total 432 350 82 81.0 Total 432 337 95 78.	W	Mild	в.S.	36	33	m	91.7	J.W.	36	32	4	•
Mild E.P. 36 32 4 88.9 B.B. 36 27 9 75. Total 432 350 82 81.0 Total 432 337 95 78.	W	Mild	G.B.	36	25	11	29.4	L.S.	36	27	6	5.
. 432 350 82 81.0 Total 432 337 95 78.	W	Mild	Е.Р.	36	32	4	88.9	В.В.	36	27	6	75.0
			Total	432	350		ι.	Total	432	337		•

* Total number of responses available from three judges.

classification.
across
summary
Data
12.
Table

	Possible	Correct	Error	Total	Percent Correct
All Parkinson Subjects	432	350	82	432	81.0
All Control Subjects	432	337	95	432	78.0
Severe Parkinson Subjects	216	164	49	216	37.9
Age-matched Controls	216	154	62	216	35.6
Mild Parkinson Subjects Age-matched Controls	216 216	183 183	33 33	216 216	42.3 42.3

as intelligible to the judges in their production of isolated vowel-bracketed plosives.

The results of the Plosive Analysis Part B are opposed to studies which suggest that Parkinson subjects' production of isolated plosives are less intelligible than normals. Indeed, the results indicated that the Parkinson groups' production of isolated vowel-bracketed plosives were as intelligible as the age-matched controls to untrained judges. Therefore, one can conclude that the articulatory patterning necessary to produce the equally intelligible plosives must have been normal or "as normal" as the control groups production of vowel-bracketed plosives.

Three conclusions may be drawn regarding the results of Part B. First, one can conclude that the reported difficulty in the production of plosives by Parkinson speakers is only true relative to ongoing speech and thus a result of impaired oral diadochokinesis. This conclusion would tend to support Hoberman's (1940) assumption that Parkinson subjects perform "normally" for isolated articulatory movements. Second, one can conclude that the widespread use of anti-parkinson medication has improved the overall populations' production of plosives to the extent that they no longer differ from age-matched normals. This conclusion would be consistent with the observation that the anti-parkinson medication resulted in improved labial and other oral-muscular movements (Nakano et al. 1973).

The third conclusion one can draw is that the age-matched control group, average age 72.5, is not representive of all normals in that they might also be deviant in their articulation of plosives. There is, however, no research indicating that an older population produces plosives differently from a younger population.

Regardless of the reasons for the differences in conclusions, these results indicate that the Parkinson subjects used in this study do not experience singular deficits in activation of isolated plosive gesture movements as measured by plosive intelligibility.

CHAPTER V

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

Parkinson's disease (parkinsonism) is a progressive, extrapyramidal system disorder generally thought to be associated with slow degeneration of certain basal ganglia and resulting from a decrease in concentration of dopamine, which is essential to the transmission of neural impulses. Symptoms of the disorder may develop at any age but most commonly occur in the fourth or fifth decade of life. Progression of the symptoms may range over ten to twenty or more years. The disorder is characterized by muscular rigidity, tremor at rest, and impairment of voluntary movements. Approximately 2.5 million people in the United States suffer from this disorder. Among the symptoms of parkinsonism are disturbance of speech which may include deviations of any of the five basic motor speech processes: respiration, phonation, resonance, articulation, and prosody.

The subjects used in this study consisted of 12 patients, 6 male and 6 female, diagnosed by physicians as having Parkinson's disease. Each sex category was further subdivided into 3 subjects with severe symptoms and 3 subjects with mild symptoms. A group of 12 age and sex-matched

individuals was used as a control.

The following phonetic materials were used in this study: 1) The Rainbow Passage, 2) the words from list C from the Multiple Choice Test of Intelligibility by Black (1963), 3) Cluster and CNC monosyllables, and 4) Isolated vowel-bracketed plosives.

The distribution of articulatory pauses from the Rainbow Passage for both the Parkinson group and the control group were compared statistically for differences in mean duration of the pauses. The pauses were detected and measured by the instrument "pausimeter" (Tosi, 1965). The results of the statistical analysis indicated that the control subjects' mean duration of articulatory pauses were longer than the mean duration of the articulatory pauses for the Parkinson subjects.

Spectrograms of the cluster and CNC samples were obtained to determine the mean frequencies of the first and second formants of the following vowels for both the Parkinson group and the age-matched controls: /I/, / ϵ /, /ae/, /a/, / Λ /, /2/, /o/. Euclidian distances from the control subjects' mean frequencies and the Parkinson subjects' mean frequencies were tested statistically for significance. The statistical analysis showed no significant differences among subjects of each subgroup, but significant differences among Parkinson and normals of corresponding subgroups were observed. Although no interaction was observed across

the various subgroups, there was some homogeneity between vowel-sex-formant number-type of diagnosis with the amount and direction of the Parkinson groups' frequency deviations.

Data on intelligibility of the Parkinson subjects was obtained by using the Black Test of Intelligibility (1963) and was compared with the normalized data provided with the test. The results of the intelligibility analysis suggest that Parkinson subjects diagnosed by their physicians as possessing either mild or severe involvement will have reduced intelligibility corresponding to the severity. The results also suggest that women with a severe diagnosis have better intelligibility than men with the same condition, and vice versa for subjects with a mild diagnosis.

The perceptual features regarding plosive production were judged by a panel of untrained judges. The results indicated that judges perceive the isolated vowel-bracketed plosives as, in decending order of occurrence, a) other plosives, b) fricatives, c) nasals, and d) glides. However, the plosive analysis also indicated that the Parkinson groups' production of isolated plosives were as intelligible as an age-matched control group. The major conclusions regarding the plosive analysis are that 1) the Parkinson subjects' difficulty in the production of plosives reported in the literature is only true relative to ongoing speech, and/or 2) the widespread use of anti-parkinson medication has improved the overall populations' production of plosives

to the extent that they no longer differ from age-matched normals.

Conclusions

Within the design of this study and the instrumentation utilized, the following conclusions are appropriate:

1. Control subjects display longer articulatory pauses than do Parkinson subjects using the operational definition of a pause and the method of measurement introduced by Tosi (1965). Thus we can conclude that Parkinson patients speak at a more rapid rate when compared to normal individuals of the same age.

2. There is not a significant Euclidian difference between the mean frequency of the first and second formants of vowels uttered by Parkinson subjects within the same subgroup. There is a significant difference for the same vowels, as uttered by normal subjects and Parkinson subjects. The results suggest that Parkinson patients distort the first and second formants in monosyllables.

3. Parkinson subjects diagnosed by their physicians as possessing either mild or severe involvement will have reduced intelligibility corresponding to that diagnosis when measured by the Multiple Choice Intelligibility test by Black (1963). The results suggest that a medical diagnosis of severity will include intelligibility factors in that diagnosis.

4. Untrained judges perceive isolated vowel-bracketed

plosives uttered by Parkinson subjects as, in decending order of occurrence, other plosives, fricatives, masals, and glides.

5. There is no significant difference between Parkinson subjects and normals in their production of isolated vowel-bracketed plosives as determined by untrained judges. Thus, Parkinson individuals receiving anti-Parkinson medication do not appear to experience difficulty producing isolated plosives.

Recommendations

Considering the findings of the present study, the following recommendations for future research are:

 A study should be conducted to determine how judges perceive the plosives produced by Parkinson subjects in ongoing speech.

2. A study should be conducted to determine whether there is a difference between the plosive intelligibility in ongoing speech by Parkinson subjects as compared to normals.

3. A study should be conducted to determine how hypokinetic dysarthria differs acoustically from Lehiste's (1965) results concerning general dysarthria.

4. A study should be conducted to compare the mean frequencies of the first and second formants in other types of dysarthria, especially flaccid dysarthria.

5. A study should be conducted to measure the mean pause duration of subjects displaying the acceleration

phenomenon.

6. A study should be conducted to determine whether articulation and voice therapy is effective in improving the overall intelligibility of Parkinson subjects. REFERENCES

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APPENDICES

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APPENDIX A

THE RAINBOW PASSAGE

When the sunlight strikes raindrops in the air, they act like a prism and form a rainbow. The rainbow is a division of white light into many beautiful colors. These take the shape of a long round arch, with its path high above, and its two ends apparently beyond the horizon. There is, according to legend, a boiling pot of gold at one end. People look, but no one ever finds it. When a man looks for something beyond his reach, his friends say he is looking for the pot of gold at the end of the rainbow.

Throughout the centuries men have explained the rainbow in various ways. Some have accepted it as a miracle without physical explanation. To the Hebrews it was a token that there would be no more universal floods. The Greeks used to imagine that it was a sign from the gods to foretell war or heavy rain. The Norsemen considered the rainbow as a bridge over which the gods passed from earth to their home in the sky. Other men have tried to explain the phenomenon physically. Aristotle thought that the rainbow was caused by reflection of the sun's rays by the rain. Since then physicists have found that it is not reflection, but refraction by the raindrops which causes the rainbow. Many complicated ideas about the rainbow have been formed. The difference in the rainbow depends considerably upon the size of the water

drops, and the width of the colored band increases as the size of the drops increases. The actual primary rainbow observed is said to be the effect of superposition of a number of bows. If the red of the second bow falls upon the green of the first, the result is to give a bow with an abnormally wide yellow band, since red and green lights when mixed form yellow. This is a very common type of bow, one showing mainly red and yellow, with little or no green or blue. THE MULTIPLE CHOICE TEST OF INTELLIGIBILITY BY BLACK

Speaker 1 is drew crew moderate fight modesty mice modest grew forbade chink **53** Y pervade kink surveyed check 2 stayed survey chin spude intent drunk stung grunt intend stun 2 content stunned brunt (intense) runt busy_ (wade) quench physics physic waves went whence wave way when visit clearly fine PASE weary find past cast quarty sign kind task query nurse get popular 6 hopper gap first guess DILT guest opera burst only immense named 7 commence name emit main woman pullman emit main cement knave omen swain latter (last) slain Hame 8 ladder lash laugh plain rabbit glass gold pail crash poor bowl crab 0 polo cold craft palace crack. % Correct Errors

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	peaker 1 i		
1	groove	modern	vice
	drew	moderate	fight
	crew	modesty	mice
	grew	modest	bite
2	say	forbade	chink
	stay	pervade	kink
	stayed	surveyed	check
	spade	survey	chin
3	stung	drunk	intent
	stun	grunt	intend
	sun	brunt	content
	stunned	runt	intense
4	quench	busy	wade
	went	physics	waves
	whence	physic	wave
	when	visit	way
5	pass	clearly	fine
	past	weary	find
	cast	quarry	sign
	task	query	kind
6	popular	nurse	get
	poplar	first	gap
	hopper	birth	guess
	opera	burst	guest
7	immense	named	only
	commence	name	woman
	emit	main	pullman
	cement	knave	omen
8	latter	last	swain
	ladder	lash	slain
	lattice	laugh	flame
	rabbit	glass	plain
9	crash	gold	pail
	crab	bowl	poor
	craft	cold	polo
	crack	bold	palace

APPENDIX B

APPENDIX C

THE CLUSTER LIST

- 13. CHARGE 1. ARCH 2. BANGS 14. CLEARS 3. BARB 15. CROWN 4. BIND 16. CUTE 5. BLUSH 17. DARK 6. BOLT 18. DROP 7. BOUNCE 19. DWARF 8. BRONZE 20. ELSE 9. BUILT
- 10. CATS
- 11. CHAMP

12. CHANT

.

APPENDIX D

THE CNC LIST

- 1. BAR 13. HEAD
- 2. BELL 14. HOUSE
- 3. BUSH 15. HUT
- 4. CHIN 16. JAIL
- 5. DAB 17. JOKE
- 6. DIP 18. KERN
- 7. DIRGE 19. KEG
- 8. FADE 20. KID
- 9. FAITH
- 10. FOUR
- 11. GAP
- 12. GULL

APPENDIX E

THE VOWEL-BRACKETED PLOSIVES

1.	АТА	7.	IPI
2.	ADA	8.	IBI
3.	АРА	9.	UTU
4.	ABA	10.	UDU
5.	ITI	11.	UPU
6.	IDI	12.	UBU

APPENDIX F

PARKINSON SUBJECTS CNC LIST

				Me	ean Fr	equer	ncy of	Each	Form	nant
Severity	Sex	Subject		a	ε	٨	æ	>	0	I
Severe	F	A.F.	F F1 F2	750 1500	400 2050	550 1000	500 2150	500 1050	450 1150	400 2100
Severe	F	G.R.	F1 F2	750 1500	400 2000	450 750	400 650	600 1700	450 1150	100 1700
Severe	F	L.W.	F1 F2	650 1200	500 2500	400 750	400 650	600 1200	100 600	11(55(
Mild	F	M.L.	F F1 2	750 1450	500 2000	700 1300	650 2000	600 1000	500 1150	50(200(
Mild	F	B.F.	F F1 2	700 1400	500 2000	650 1400	600 2250	600 1100	500 1200	500 2200
Mild	F	B.R.	F1 F2	700 1150	550 2650	750 1300	650 2350	500 850	500 1050	500 2750
Severe	М	C.R.	F1 F2	700 1850	600 1300	750 1300	600 1850	600 1400	600 950	400 1250
Severe	М	C.D.	F1 F2	650 1400	650 2200	650 1400	600 1700	500 1000	500 1000	600 1800
Severe	М	J.K.	F1 F2	800 1250	500 2050	600 1200	500 2100	500 850	500 1050	40 200
Mild	М	B.S.	F1 F2	750 1200	650 1600	650 1050	700 1800	500 750	5 50 1000	600 1700
Mild	М	Н.В.	F1 F2	600 1000	500 2000	600 1400	700 1950	500 750	500 1250	450 2000
Mild	М	E.P.	F1 F2	550 1100	450 2150	550 1150	500 1800	450 700	450 1800	50(155(

APPENDIX G

CONTROL SUBJECTS CNC LIST

				Mean	Frequ	iency	of Ea	ch Fo	ormant
Sex	Subject		a	ε Ε	Λ	 æ	<u> </u>	 0	 I
F	L.G.	F_{F_2}	700 1400	550 1600	700 1400	600 1650	450 800	500 1450	400 1150
F	в.С.	F_{F_2}	750 1400	600 2100	750 1700	750 2000	750 1100	500 1400	700 2200
F	G.C.	F1 F2	500 2100	500 2350	700 1500	650 2100	600 1000	550 1050	500 2300
F	G.D.	F1 F2	500 1400	650 1500	550 1350	700 1400	500 900	500 1200	500 1300
F	J.M.	F F1 2	500 1200	500 1500	750 1600	500 1700	900 900	500 1200	400 2200
F	S.S.	F1 F2	550 1000	600 2500	700 1200	650 1600	500 900	500 1000	500 1100
М	C.F.	F F 2	650 1100	500 1900	700 2100	650 1800	400 650	400 700	500 1900
М	R.M.	F1 F2	700 1200	500 2200	700 1550	600 1750	500 750	500 1700	450 2200
М	W.W.	F F1 2	700 1150	450 1500	650 1300	500 1650	400 650	500 1400	400 1500
М	J.W.	F1 F2	700 1200	400 2000	600 1400	500 1700	450 900	500 1000	400 1800
М	L.S.	F1 F2	500 750	500 2300	700 1300	600 1050	500 800	450 1400	450 2100
М	в.в.	F_1 F_2	650 1250	500 1850	600 1300	600 1700	450 650	450 950	500 1600

APPENDIX H

PARKINSON SUBJECTS CLUSTER LIST

				Me	an Fr	equen	cy of	Each	Form	ant
Severity	Sex	Subject	-	a	ε	٨	æ	2	0	I
Severe	F	A.F.	F_{1}	750 1500	350 2450	700 1400	550 2300	500 1505	500 900	400 2150
Seve re	F	G.R.	F_{1}	700 1400	600 2100	600 1100	500 1900	650 1100	300 600	500 1900
Severe	F	L.W.	F_{F_2}	800 1400	600 2100	600 1100	350 2100	600 1100	300 600	400 1150
Mild	F	M.L.	F_{F_2}	700 1450	700 1650	700 1250	500 1900	650 1100	500 900	500 1950
Mild	F	B.F.	F_{1}	700 1500	600 2200	750 1500	700 2100	600 1150	600 1100	400 2500
Mild	F	B.R.	F1 F2	750 1700	550 2300	550 1200	500 2400	500 900	500 800	500 2500
Severe	Μ	C.R.	F F2	700 1000	500 1000	690 1700	700 1750	650 1800	400 700	550 1100
Severe	М	C.D.	F_{F_2}	600 1050	700 1600	700 1400	650 1700	500 100	600 900	600 1600
Severe	М	J.K.	F_{1}	650 1500	700 1400	600 1200	350 2050	500 1100	500 900	350 1850
Mild	М	B.S.	F_{F_2}	650 1250	600 1750	700 1250	700 1700	500 900	500 800	550 1500
Mild	М	H.B.	F F2	750 1350	550 1800	700 1500	600 1900	500 1100	500 850	500 1600
Mild	М	E.P.	F_{F_2}	600 1000	500 1600	500 1350	450 2000	500 900	450 800	400 1500

APPENDIX I

CONTROL SUBJECTS CLUSTER LIST

				Mean	Frequ	lency	of Ea	ch Fo	ormant
Sex	Subject		a	ε	٨	æ	2	0	I
F	L.G.	F_1 F_2	750 1300	600 2100	700 1500	600 2200	600 1000	500 800	500 2000
F	в.с.	F_1 F_2	750 1500	600 2100	750 1500	650 2200	750 1200	450 750	500 2200
F	G.C.	F F ¹ 2	800 1500	550 2500	700 1500	700 1500	650 1150	600 600	600 1700
F	A.D.	F F1 2	600 1150	600 2100	500 1500	600 1400	700 1400	500 900	500 1500
F	J.M.	F1 F2	750 1450	600 2100	500 1400	750 2000	700 1400	500 1050	500 1700
F	S.S.	F_{F_2}	500 1650	700 1700	750 1500	650 2500	550 1000	500 900	450 2500
М	C.F.	F F1 2	700 1100	600 1800	600 1150	650 1850	550 900	350 700	500 1800
М	R.M.	F F 2	600 1250	600 1500	550 1400	550 2100	600 1000	400 650	450 2000
М	W.W.	F_{F_2}	500 1250	550 1700	500 1300	500 1850	400 800	450 900	450 1600
М	J.W.	F1 F2	700 1300	500 1500	500 1500	600 1800	450 850	450 800	400 1800
М	L.S.	F_{F_2}	700 1500	600 2350	500 1400	700 1800	600 1000	500 850	400 1550
М	B.B.	F_{F_2}	600 1350	600 1500	600 1200	550 1850	500 1000	500 850	350 1700

APPENDIX J

PLOSIVE ANALYSIS OPEN RESPONSE SUBTEST FORM A

CLAS	ECT: SSIFIC	H.I ATIC		Male 3	e-Mil	1d 5	6	7	8	S Error	W Voiced Plosive	Unvoiced Plosive	Fricative	Glide	Lateral	Nasal	Total
1.	ATA	D	D	D	D	D	т	т	D	6	6				Γ		
2.	ADA	D	D	D	D	D	D	D	D	-							
3.	APA	В	В	В	В	в	в	В	В	8	8						
4.	ABA	в	в	в	в	В	в	В	В	-		1)
5.	ITI	D	G	G	G	D	D	D	т	7	7						
6.	IDI	D	D	D	D	D	в	В	D	2	2	1					
7.	IPI	в	в	в	Ρ	Р	В	Р	Р	4	4						
8.	IBI	в	В	В	В	в	В	в	В	-							
9.	UTU	G	т	т	т	т	D	D	т	3	3	L.,					
10.	UDU	D	D	D	D	D	D	D	D	-		11	1				
11.	UPU	Ρ	В	P	Р	Р	Р	В	P	2	2						
12.	UBU	В	в	В	В	в	В	В	В	-							
											sive	Plosive					-
CLAS	JECT: SSIFIC	E.I ATIO		Male 3	e-Mi 4	1d 5	6	7	8	IL Error	o Voiced Plosive	N Unvoiced F	Fricative	Glide	Lateral	Nasal	Total
Stim	SSIFIC nulus ATA	LATIO	ON: 2 T	3 T	4 T	5 T	т	т	т			Unvoiced	Fricative	Glide	Iateral	Nasal	Total
Stim	ATA ADA	LATIO	ON: 2 T D	3 T D	4 T D	5 T D	T D	T D	T A	11		N Unvoiced	Fricative	Glide	Lateral	Nasal	Total
Stim	ATA ADA APA	LATIO	2 T D P	3 T D T	4 T D P	5 T D P	T D P	T D P	T A P	11 - - 1	9	Unvoiced	Fricative	Glide	Iateral	Nasal	Total
Stim 1. 2. 3. 4.	ATA ADA APA ABA	LATIO	2 T D P B	3 T D T D	4 T D P B	5 T D P B	T D P B	T D P B	T A P D	11	9	N Unvoiced	Fricative	Glide	Iateral	Nasal	Total
Stim 1. 2. 3. 4. 5.	ATA ADA APA ABA ITI	1 T D P B T	2 T D P B T	3 T D T D T	4 T D P B T	5 T D P B T	T D P B T	T D P B T	T A P D T	11 - 1 2 -	9	N Unvoiced	Fricative	Glide	Iateral	Nasal	Total
Stim 1. 2. 3. 4. 5. 6.	ATA ADA APA ABA ITI IDI	1 T D P B T D	2 T D P B T D	3 T D T D T D	4 T D P B T D	5 T D P B T D	T D P B T D	T D P B T D	T A P D T D	11 - - 1 2 -	9	N Unvoiced	Fricative	Glide	Iateral	Nasal	Total
Stim 1. 2. 3. 4. 5.	ATA ADA APA ABA ITI IDI IPI	1 T D P B T D P	2 T D P B T D P	3 T D T D T D P	4 T D P B T D P	5 T D P B T D P	T D P B T D P	T D P B T D P	T A P D T D P	11 - 1 2 - -	9	r v Unvoiced	Fricative	Glide	Iateral	Nasal	Total
Stim 1. 2. 3. 4. 5. 6. 7. 8.	ATA ADA APA ABA ITI IDI	1 T D P B T D P P P P P	2 T D P B T D P B T D P B	3 T D T D T D P B	4 T D P B T D P B T D P B	5 T D P B T D P B T D P B	T D P B T D P B	T D P B T D P B	T A P D T D P B	11 - - 1 2 - - - 1	9	1 Nuviced	Fricative	Glide	Iateral	Nasal	Total
Stim 1. 2. 3. 4. 5. 6. 7. 8. 9.	ATA ADA APA ABA ITI IDI IPI	1 T D P B T D P	2 T D P B T D P	3 T D T D T D P	4 T D P B T D P	5 T D P B T D P	T D P B T D P	T D P B T D P B T D P B T	T A P D T D P B T	11 - - - - - - - 1 2	9	1 Nuviced	Fricative	Glide	Iateral	Nasal	Total
Stim 1. 2. 3. 4. 5. 6. 7. 8.	ATA ADA ADA ABA ITI IDI IPI IBI	1 T D P B T D P P P P P	2 T D P B T D P B T D P B	3 T D T D T D P B	4 T D P B T D P B T D P B	5 T D P B T D P B T B T B	T D P B T D P B T D P B T D	T D P B T D P B T D P B T D	T A P D T D P B T D	11 - - 1 2 - - - 1	9	1 Nuviced	Fricative	Glide	Iateral	Nasal	Tota
Stim 1. 2. 3. 4. 5. 6. 7. 8. 9.	ATA ADA APA ABA ITI IDI IPI IBI UTU	1 T D P B T D P P T	2 T D P B T D P B T D P B T	3 T D T D T D P B D	4 T D P B T D P B D P B D	5 T D P B T D P B T	T D P B T D P B T D P B T	T D P B T D P B T D P B T	T A P D T D P B T	11 - - - - - - - 1 2	9	1 T Nuviced	Fricative	Glide	Iateral	Nasal	Total

APPENDIX K

PLOSIVE ANALYSIS CLOSED-RESPONSE FORM B

1.	ABA	2.	ADA	3.	АТА	4.	APA
	АТА		APA		ADA		ADA
	ADA		ABA		APA		АТА
	АРА		АТА		ABA		ABA
5.	ITI	6.	IPI	7.	IDI	8.	ITI
	IDI		IBI		IBI		IBI
	IBI		IDI		IPI		IDI
	IPI		ITI		ITI		IPI
9.	UBU	10.	UDU	11.	UPU	12.	UBU
	UPU		UPU		UDU		UTU
	UDU		UBU		UTU		UDU
	UTU		UTU		UBU		UPU
Subj	ect Name	<u></u>					
	e Name						
Perc	ent Corre	ct					

