ACOUSTIC AND TEMPORAL CORRELATES OF DYSARTHRIC SPEECH

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A Dissertation Submitted as part fulfillment
for the final year M.Sc. (Speech & Hearing) to University of Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING, MYSORE 570006
MAY - 2000

Certificate

This is to certify that the dissertation entitled "Acoustic and temporal Correlates of Dysarthric Speech is the bonafide work done in part fulfillment for the degree of Master of Science (Speecb and Hearing) of the student with Register No. M9808.

Mysore
May 2000

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Certificate

This is to certify that the dissertation entitled Acoustic and temporal Correlates of Dysarthric Speech has been prepared under my supervision and guidance.

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May 2000

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Declaration

I *hereby declare that this dissertation entitled Acoustic and Temporal Correlates of Dysarthric Speech" is the result of my own study under the guidance of Dr. N.P.Nataraja, Professor and Head of department of Speech Sciences, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier at any other University for any other diploma or Degree.*

Mysore
May 2000

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A thought to prospective readers

"Ideas are nobody's property ;

They belong to who ever expresses them best"

Emilio Cecchi

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Gratitude is the hardest of all

Emotions to express

There is no word capable of covering all

That one feels until we reach a world

Where thought can be adequately expressed in words

Thank You will have to do :

First and foremost I thank god almighty for the countless blessing he has bestowed on me.

Fear thou not; For I am with Thee (Isaiah 41:10)

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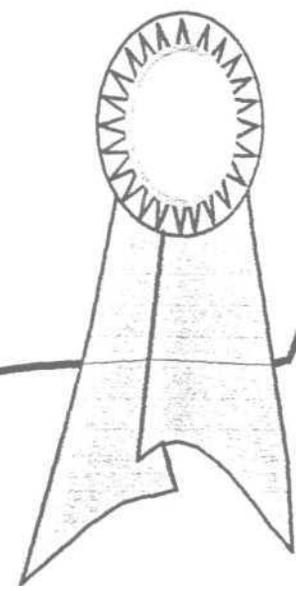

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INTRODUCTION

" Man's capability for oral communication probably evolved in parallel with the evolution of human brain. A precise record of the emergence of this singularly human faculty is lost in the fog of prehistory, but it has been assumed that as long as man has possessed oral speech, he has been susceptible to its disturbance or loss. Trauma and disease have disrupted man's ability to communicate and have repeatedly underscored the dependence of this skill on the integrity of the central and peripheral nervous system. (Rosenbek and Lapointe, 1978)

Since time immemorial man has shown interest in exploring nature and understanding natural process. Growth of science in this century has accelerated such endeavors. Progress in technology has been immensely helpful in understanding many natural processes in human anatomy and physiology, as well as in diagnosis and treatment of many disorders.

A major consideration of research workers in speech science and speech language pathology has been to attempt to describe and explain the manner in which speech system operates. " System " is used here to refer to the interacting, interdependent components of a functional unit that is only partially accessible to direct observation (Attanasia, 1987). "Speech, a motor act consists of complex ballistic movements. Unlike many motor activities, speech requires a complex blend of actions in synchrony to produce even a simpler response" (Kelso, Tuller, and Harris, 1983). Sensory motor integration is a necessary condition for normal speech production.

Speech is a highly integrated physiological motor act. For each speech sound there is a separate neuro muscular configuration that involves as a functional unit, all

musculature of the speech organs. Any disturbances of this neuromuscular configuration as a result of weakness, paralysis or incoordination of the speech musculature or as a result of lesions in the nerves supplying the musculature results in speech dysfunction's.

Dysarthria is such a condition, a Greek word, meaning literally disturbance of articulation. As the word is usually employed in the field of speech pathology it implies any impairment of articulation caused by agenesis of or damage to the nerve centers or tracts (other than those of the language areas of the cerebral cortex) immediately involved in direct control of the musculature used in the enunciation and pronunciation of vowels and consonants (West and Ansberry (1968)). Under dysarthria there is a diverse group with a degenerative natural course, often referred to as degenerative dysarthrias, with an onset after childhood and in most cases with signs and symptoms appearing gradually as in Parkinson's disease, Amyotropic lateral sclerosis, Huntingtons chorea, Wilson's disease etc. (Yorkston & Beukelman, 1988)

The traditional methods of speech evaluation of dysarthric patients by neurologists were by using speech material such as "tongue twisters". Since then speech evaluation strategies have become more scientific, organized and informative. The various techniques used can be grouped under two headings: 1. Perceptual assessment and 2. Instrumental analysis

Although perceptual analysis is the major tool used by speech-language pathologist to gather information concerning speech production, characteristics of persons with various speech disorders, there are good reasons to explore the potential of instrumental analysis for enhancing and refining this information. Especially in the case of motor speech disorders where speech characteristics may pose a particular

challenge to the fragile stability of perceptual judgements such as phonetic transcriptions (Shriberg & Kwiatkowski, 1982) or psychophysical scaling (Shiavetti, Metz & Sitler, 1981), instrumental analysis may be particularly attractive. Among the different types of instrumental analysis (e.g. aerodynamic, electromyographic, Cinegraphic) that could be used for analysis of characteristics of speech disorders, acoustic analysis is highly recommended (Hirano, 1981; Natraja 1972; Rashmi, 1988; Anitha 1994). According to Hirano (1981) "this may be one of the most attractive methods of assessing phonatory function or laryngeal pathology because it is non-invasive and provides objective and quantitative data". Acoustic analysis can be done using methods such as spectrography, peak analysis, inverse filtering and other computer-based methods apart from using other instruments, which is economical both in terms cost and time.

Acoustic analysis can be informative because it afforts quantitative analysis that carry potential for description sub system and for determining the correlates of perceptual judgements of intelligibility, quality and type of dysarthria (Kent et. al. 1999). Therefore acoustic analysis can be a valuable complement to the perceptual evaluation, in the belief that instrumental methods will over come some of the limitations of the subjective perceptual assessments (Collins, 1984). Kent & Netsell (1975), Rajkumar (1983), Nataraja & Indira (1982), Ramig et. al (1988) have used acoustic analysis to describe speech of dysarthrics.

The neurological disturbance seen in dysarthria can potentially affect every component of the speech production system. Predictably, these changes in the subsystems of speech productions lead to changes in the acoustic characteristics of speech. Therefore acoustic characteristics of speech in dysarthrics are studied to note

their deviations from comparable normal individuals of same **age** and sex groups. Since these acoustic characteristics reflect the changes in speech production system and its function, they have been used to study the nature and function of speech mechanism. Although the physiologic and phonetic interpretations of acoustic data are uncertain, they are useful in testing certain hypothesis about changes in anatomy, motor control and physiological functions. Thus in the event of abnormal structural and functional changes there will be a corresponding changes in the acoustic characteristics of speech. Therefore an insight into the varied characteristics of speech would facilitate in differentiating normal from abnormal. This will in turn contribute to the early detection of diseases of different neural sub systems, their diagnosis and management.

The abnormal vocal fold vibration, abnormal resonance, mal-positioning of articulators may all contribute to poor speech characteristics in dysarthrics. Some of the above parameters have been found to be affected in dysarthrics (Ramig et al. 1988; Yorkston et al., 1988; Hanson et al., 1984; Kent et al., 1975). Several attempts have been made to study the speech of dysarthrics based on perceptual and acoustic analysis . However,studies correlating the findings of these two methods are scanty. Further, not much information is available in Indian context on these parameters in dysarthrics. So, the present study aims at determining the changes in acoustic parameters in the speech of dysarthrics.

Statement of the problem

The problem was to know how the various acoustic parameters have changed in dysarthrics (with degenerative natural course) . The present study therefore aims at analyzing some of the acoustic aspects in the speech of dysarthrics.

Purpose of the study

The purpose of the study was to test the following hypothesis:

Main hypothesis

There is no significant difference in the acoustic parameters of speech of dysarthrics and normal subjects

Auxiliary hypothesis

1) There is no significant difference between the three types of dysarthnc subjects: Parkinson's disease, amyotropic lateral sclerosis and Wilson's disease in terms of comparable acoustic & temporal parameters.

a) Mean fundamental frequency for phonation for /a/, *!\l, IvJ*
b) Mean intensity in phonation for /a/, *l\l*, /u/
c) Extent of fluctuations in frequency and intensity
d) Speed of fluctuations in frequency and intensity
e) Frequency range in phonation
f) Intensity range in phonation
g) Formant frequencies (Fl, F2, F3)
h) Band width (B1,B2,B3)
i) Word duration
j) Vowel duration
k) Burst duration
1) Closure duration
m) Consonant duration
n) Voice onset time (VOT)

To test these hypothesis, totally 12 subjects both males and females were selected who belong to the same age range. This consisted of six normal subjects and six subjects with dysarthria. The normal subjects had normal speech, language,

hearing and had no other significant history of any other problem. The dysarthric group consisted of three subjects with Parkinson's disease, two with Amyotropic Lateral Sclerosis and one with Wilson's disease. Three trials of sustained phonation of /a/, /i/, & /u/ and a list of 50 familiar words in Malayalam produced by each subject were recorded. Thus, speech samples recorded for all the subjects were used for further analysis. The following frequency and intensity measures were obtained by analyzing the voice samples of all the three vowels using necessary computer programs.

a) Mean fundamental frequency in phonation
b) Mean intensity in phonation
c) Extent of fluctuations in frequency in phonation
d) Speed of fluctuations in frequency in phonation
e) Extent of fluctuations in intensity in phonation
f) Speed of fluctuations in intensity in phonation
g) Frequency range in phonation
h) Intensity range in phonation
i) Formant frequencies (Fl, F2, F3)
j) Band width (Bl, B2, B3) of formant frequencies

The speech samples of normal & dysarthric subjects were analyzed both perceptually and acoustically. The perceptual analysis was done by three judges. The computer programs of VSS (Voice and Speech Systems, Bangalore) were used for analysis speech to derive the following parameters

a) Word duration b)vowel duration c) burst duration d) closure duration
e) consonant duration f) Voice Onset Time

Implications of the study

The study provides information useful in

1. understanding the deviant acoustic parameters in dysarthrics
2. early detection of diseases with different neural sub system involvement
3. estimating the importance of neuromuscular control of speech production
4. Planning the management strategies for dysarthrics.

REVIEW OF LITERATURE

Language is undoubtedly the most important factor that differentiates human beings from other animals. Language constitutes both a set of symbols (codes) and set of procedures (rules), which combine to form words, phrases and sentences and used for the communication. Language is itself a system of abstract logic. It allows human beings to extend their rational ability. Indeed it has often been virtually equated with human beings abstract logical ability (Chomsky, 1966).

Although there are various ways of using language, the sending and receiving of spoken messages are the most frequently used and important ways of sharing the thoughts and feelings with each other. Speech may be viewed as the unique method of communication evolved by human beings to suit the uniqueness of their mind. By its great flexibility it permits human beings to produce a variety of signals commensurate with the richness of their imaginations. Speech may be defined as a form of oral communication in which the transformation of information takes place by means of acoustic energy. The speech wave forms are the result of interaction of one or more sources with the vocal tract filter system (Fant, 1960).

Speech is produced without observable efforts by human beings. The range of speech variation is immense and yet considered normal. Only a small part of information is conveyed by speech. Less than one percent of this is used for linguistic purpose, as such the rest gives other kinds of information about specific character of vocal tract of the speaker, which enables one to recognize the speaker's voice, physical well being and emotional state, attitude towards the entire context in which the speech event occurs. It can also carry other informations about the speaker with reference to the conventions of social class, occupation and style.

"Compared to the mechanism of human speech, the hardware of an **atom** bomb or a space missile is a simple engineering work" (DeuPree,1965). Speech is a complex process involving several intricate and diverse activities. Speech has several underlying bases such as social, physical, physiological, neurological, phonetic, linguistic, psychological, genetic, and semantic (Gray and Wise 1959). Speech can be considered as the skilled, willful and elaborate movement of muscles used for initiating vocal sounds and the moulding of these sounds into meaningful oral communication, or more simply, the voluntary modification of the outgoing breath stream into meaningful sounds of speech. The production of speech is exercised by the simultaneous, highly coordinated and specifically differentiated function of various systems: respiratory, phonatory and articulatory. Speech can be viewed from different aspects which are overlapping and inter-related i.e., acoustic, the motor and the perceptual aspects. Thus speech is a motor phenomenon. The motor activity involved in speech production is controlled by the nervous system. Speech involves the production of sequences of movements, which are controlled by several areas of the nervous system. Given its extreme complexity, the speech production is usefully conceptualized in terms of its different physical levels. These levels are illustrated in fig. 1.

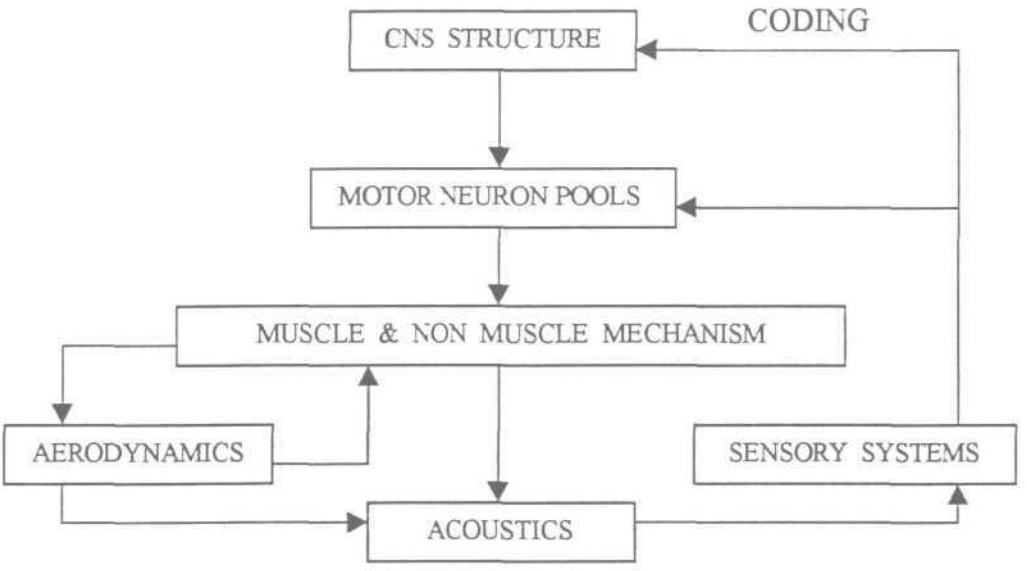


Fig.l: A schematic diagram of the major levels of speech production, showing primary lines of interaction and communication.(Clinical management of Dysarthric speakers : Yorkston & Beukelmann, 1988)

Certain brain centers and neural pathways are consistently involved in the neural process underlying the control of speech movements (Yorkston et.al. 1988). The net result of these processes is a highly organised discharge of the motor neurons innervating the speech muscles. Motor neuron discharge results in muscle fiber shortening and the generation of muscle forces. These muscle forces interact with the mechanical properties of muscular and non-muscular tissues (e.g. elasticity *and* Inertia.). In some cases, speech muscle forces result directly in structural displacements as in tongue movements for consonant and vowel productions. In other cases, muscle forces function primarily to change the mechanical properties of a structure as in regulation of the vibratory mass and tension of the vocal folds.

Movements of structures such as the chest wall, vocal folds, and tongue produce the precise changes in upper and lower respiratory airway pressure and flow required for speech production (Warren 1982,1986). Vocal tract pressure and flow changes result in the transient, turbulent, and periodic sound sources of speech. These sound sources are modified by the resonance or filter properties of the vocal tract to produce the rapid changes in acoustic spectral characteristics of human speech. Such changes are lawfully related to vocal tract positions and movements (Fant, 1960; Lindblom& Sundberg.1971; Minifi, 1973).

In studying the neurophysiological aspects of movements it is often required to distinguish between different stages of movement control process viz., planning, programming and execution, (Yorkston et al, 1988). They further opine that the consideration of these stages may also aid in the categorization of speech movement disorder. According to Paillard (1983), motor planning involves selection of an appropriate movement strategy in the light of intended goals and prevailing physical conditions. The intended goals of speech production may logically be thought of as

linguistic units such as phonemes, words or phrases (Abbs, 1990). In the planning of speech utterances, such units may be represented or coded in terms of spatial, aerodynamic, or acoustic targets. A general strategy for the achievement of such targets may also be a component of the speech motor plan.

Fig.-2: Schematic representation of different stages of movement control process

A second stage in the movement control is called motor programming. This entails provisional specification of precisely how the motor plan is to be achieved (Yorkston et al, 1988) for example, which muscles are to contract, how much and when. Programming is also likely to involve pre-tuning the excitability of various sensory and motor pathways to be involved in the ensuing movement process

The execution stage involves the direct activation of motor neurons, muscle contraction and movement. Through the execution process, the discharge of motor neurons may be influenced to varying degrees by numerous brain centers and sensory pathways.

The cerebral cortex is recognized as the major structure for speech and language processing. In right-handed and most left-handed individuals the left hemisphere has been considered to be specialized for the speech and language functions. Conceptualization of the physiological organization of cerebral cortex for speech movement control has been greatly influenced by observations on the effects of cortical surface electrical stimulation (Penfield & Roberts, 1966; Sessle & Wiesendanger, 1982; Woolsey, 1958). This work resulted in maps delineating the cortical regions most directly involved in movement control. These regions, illustrated

in fig3, include the primary motor cortex (area4), the pre-motor cortex (lateral area b), and the supplementary motor area (medial area). Broca's area (area 44) and somato-sensory cortex (areas 3,1 and 2).

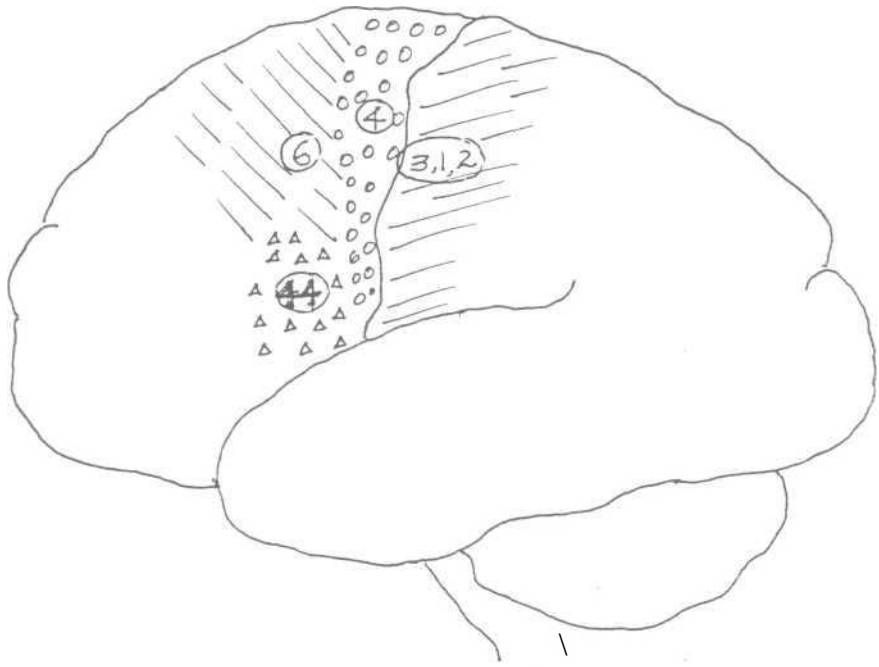


Fig.-3 : Showing the Brain speech and language areas in the cerabral cortex

Stimulation procedures involving the use of very low current strengths within the cortex are now providing a more detailed view of the spatial relationship of cortical areas to muscle activities (Weisendanger, 1981). Abbs and Welt (1985) and have suggested that multiple representations provide a partial basis for the control of diverse speech gestures by a single structure, for example lip movements for rounding and closure.

PRIMARY MOTOR CORTEX

Various characteristics of the primary motor cortex indicate that it is a major point of sensory-motor integration immediately prior to the lower motor-neurons. In primates, some (area 4) neurons have mono-synaptic projections to lower motor-neurons (Kuypers, 1958), that is, individual axons are linked directly to the motor-neurons with one synapse. Primary motor cortex activity is well correlated with

muscle force changes in learned movements (Evarts 1969; Hoffman & Luschei, 1980) and lesions in this area results in muscle weakness.

Area 4 neurons are also most responsive to sensory input from regions for which they provide motor innervation. This sensory input projects over the somato-sensory cortex (area 3, 1 and 2). Area 4 is very much involved in the execution stage of movement control. Area 6, on the other hand, is more involved in movement planning and programming (Porter, 1983). This is suggested by the fact that area 6 shows more complex activation patterns in relation to movement and an absence of short-latency responsiveness to peripheral stimulation. Area 6 has extensive projections to area 4, and it appears to be very important in shaping the pattern of motor output from area 4 (Yorkston et al, 1988).

CEREBELLUM

The cerebellum has long been recognized as a highly developed and specialized center for movement control, and it is likely to be involved in several stages of the speech movement process. The cerebellar cortex receives sensory inputs from the tongue, lips, jaw, larynx, and auditory system (Larson & Pfingst 1982), and it rapidly integrates this information in contributing to speech motor processing. Two distinct cortico-cerebellar pathways have been to be important in the regulation of motor cortex output for speech (Eccles 1977; Kent & Rosenbek, 1982; Kornhuber 1977; Neilson & ODwyer, 1984; Netsell, 1982). The important paths of communication are summarized in figure 4A.

One pathway involves neural projections from area 6 to the lateral cerebellar hemispheres (LH) via pontine nuclei. A return pathway to areas 4 and 6 occurs via deep cerebellar and ventral thalamic nuclei. Animal studies have suggested that this corticocerebellar loop is important in the planning and programming of learned

movements (Brooks 1979; Thach 1980). A second major corticocerebellar pathway involves collateral projection of descending corticospinal and corticobulbar fibers to the intermediate cerebellar hemispheres (I.H). This pathway provides the cerebellum with immediate information on descending cortical motor output. There is a return pathway to cortical area 4 from the intermediate hemispheres via deep cerebellar and ventral thalamic nuclei. The intermediate hemispheres also have descending projections to brain stem and spinal motor centers via the red nucleus. The characteristics of the various pathways support the general view that the intermediate cerebellum utilizes sensory input to effect rapid modifications of cortical motor output during movement execution. (Yorkston et. al, 1988).

BASAL GANGLIA

The basal ganglia are a collection of large subcortical nuclei which comprise a major portion of the "extrapyramidial" motor system. They make specialized contributions to speech movement control, and is suggested by the relatively distinct nature of speech seen in individuals with Parkinson's disease, a disorder of basal ganglia. The major projections to the basal ganglia arise in the frontal cortex and converge on the caudate nucleus and putamen (i.e., the striatum). These pathways form the initial segment of a loop that project back to the motor cortex via the globus pallidus and thalamic nuclei (see figure 4B). The cortical-putamen pathway appears to be especially important for motor control processes, as it is composed of fibers projecting from the premotor cortex, whereas the caudate nucleus receives primary inputs from the more anterior regions of the frontal lobe. Neural output from the putamen projects to the globus pallidus and substantia nigra, then to the primary motor cortex via ventral thalamic nuclei. Thus, a multisynaptic pathway is formed

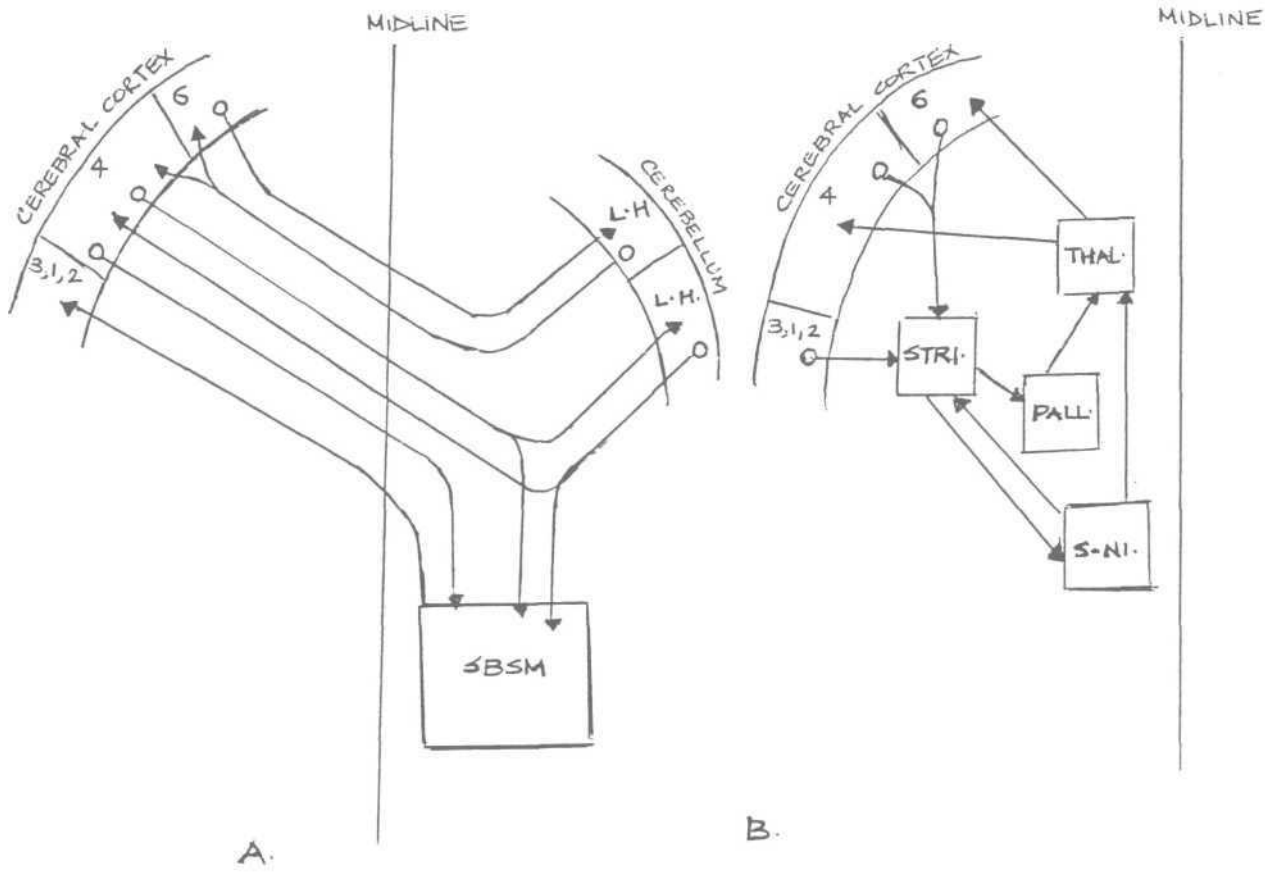


Fig.-4: Schematic illustration of the motor system pathways interconnecting the cerebral cortex cerebellum, and basal ganglia. Circles indicate output, and arrows indicate input. (A) Cortical- cerebellar pathways. L.H. and I.H. refer respectively to the lateral and intermediate cerebellar hemispheres. The projection from L.H. to the cerebral cortex occurs via ventral thalamic nuclei. SBSM refers to spinal and brain stem sensorimotor centers. (B) Pathways interconnecting regions of the cereberal cortex with various basal ganglia structures; striatum (Stri.), which includes the caudate nucleus and putamen, globus pallidus (Pall.), substantia nigra (S, Ni.). Basal ganglia communication to the cortex occurs via pathways projecting over the ventral nucler of the thalamus (Thai.). Eccles, 1977.

between the premotor cortex and primary motor cortex over the basal ganglia and thalamus. Apparently, the segregation of body parts presented within the primary motor cortex is maintained in this pathway (DeLong, Georgopouluos & Crutcher, 1983). With respect to the oro-facial system, there is a major pathway projecting from substantia nigra to supplementary motor cortex and then to area 4. In general, the basal ganglia are seen to be important in the planning and programming of learned movements. One important function may involve setting the kinematic parameters of movements, for example, recent electrophysiological studies of limb control in monkeys suggest that output from the globus pallidus is particularly important in regulating the direction and amplitude of movement (Anderson & Horak, 1985; DeLong & Georgopoulis, 1981).

MOTOR UNITS

Descending pathways from various brain centers converge on lower motoneurons within the brain stem and spinal cord. Motoneurons directly innervate muscles fibers, and thus, they represent an important interface between the rest of the nervous system and the mechanical systems involved in the movements. Each motoneuron innervates a unique set of muscle fibers, and taken together, they comprise a motor unit.

MOTONEURON POOLS

The motoneuron cell bodies associated with a muscle tend to be grouped together in cell aggregates or pools within the brain stem or spinal cord. The motoneuron within a given pool innervate the muscle fibres of a single muscle (Henneman, 1980). Various cranial nerve nuclei and the ventral regions of the spinal cord contain groups of motoneuron pools that innervate speech muscle systems. For

example, the nucleus ambigous is a collection of motoneuron pools within the medulla which innervate the intrinsic muscles of the larynx. Other cranial nerve motor nuclei of particular importance in speech motor control are the facial nucleus, the motor trigeminal nucleus, and the hypoglossal nucleus. Motoneuron axons project to their associated speech muscle fibres over the trigeminal, facial, hypoglossal, vagus, accessory, glossopharyngeal, and respiratory nerves.

Like any neuron, when a motoneuron's transmembrane potential is driven to its threshold by synaptic input, it produces an action potential which propagates the length of the axon. This results in the release of acetylocholine at the motor end plate. A muscle action potential then travelles through the associated muscle fibers, causing the release of calcium ions which then bind to muscle protein filaments. This results in the sliding action of the protein filaments, which is the basis for muscles shortening and the generation of brief forces or muscles twitches underlying whole muscle contraction (Stein & Lee, 1981).

The existence of different types of motor units is an important concept in motor physiology (Bruke &Edgerton, 1975). Examination of muscle tissue reveals that some muscle fibers are white, some are red and some are intermediate in colour. These obvious differences in colour have histochemical, mechanical and electrophysiological correlates. Red muscle fibers have a high mitochondrial and capillary supply, and an oxidative or aerobic metabolism. White muscle fibers have a reduced blood supply, few mitochondria, and anaerobic metabolism.

Electrical stimulation of motor unit axons and recording of their mechanical twitches have revealed some major differences in the physiology of motor unit types. On repeated electrical stimulation at low rates of red muscles motor units show low level twitch forces with slow rise or contraction times and they tend to maintain their

force levels over long periods of stimulation. In the same procedure, white muscle motor units show larger twitch force levels with faster contraction times and their peak force levels are much reduced after prolonged stimulation, that is, they fatigue easily. Based on these results, which have been replicated many times (Bruke & Edgerton, 1975; Lewis, 1981), red muscle motor units are referred to as slow-twitch fatigue-resistance (types), White muscle motor units as fast-twitch fatigueable (type FF), and intermediate muscle motor units as fast-twitch, fatigue-resistance (typeFR). In general, FF units tend to have larger motoneurons, axons, and muscle fibers, with larger muscle fibers partially accounting for their larger twitch forces. It is seen that type S motor units are the first and most frequently recruited under a number different conditions of activation (Desmedt, 1981; Henneman, 1980). Because most activities require low force levels to be sustained for a long time, for example, postural adjustment. This represents an effective adaptation of neuromuscular systems to the normal demands of animal movement. When rapid and /or large changes in muscle force are required, FF type units are recruited. The wide range of motor unit properties are apparently utilized in different muscle systems to achieve their unique demands (Clamman, 1981). For example, the small extra-occular muscles have very short contraction times to accommodate rapid eye movements, whereas leg muscles have much longer contraction times and generate large twitch tensions that are better matched to the more massive structures to be moved. Within muscle systems, however, there is further specialization; for example, the anterior tibial muscle is composed of a mixture of S, FF and FR type units, whereas the soleus muscle is composed exclusively of S type units. Netsell (1982) has suggested that the muscles used in speech production tend to have motor units with properties that are intermediate to those for the eyes and the limbs. Among the lip muscles, there is

considerable variability; for example, orbicularis oris has muscle fiber composition typical of S and FF type units whereas platysma is an equal mix of fibers consistent with S, FF and FR type units (Schwarting, et. al. 1982).

SPEECH MUSCLE BIOMECHANICS

Speech muscles may be thought of as mecham'cal systems that respond to neural input and produce movement. To appreciate the effect of neural signals on speech structure movements, one needs to briefly consider the mechanical characteristics of muscles involved in speech production.

MECHANICAL ELEMENTS OF MUSCLE

Fig 5, presents a simplified model depicting the mechanical characteristics of muscle. Individual motor units discharge, they produce single, twitch-like forces and these forces of different muscle units summate to produce whole muscle contractile force (F). This force acts to displace a tissue mass (M) (which is a complex process). Muscle tissue has an elastic and an internal fluid friction or viscosity components. The magnitudes of these forces change during the course of a movement. Thus, movement characteristics depend not only on the strength and timing of the muscle contraction, but also on the current mechanical state of the muscle and surrounding tissues (Lewis, 1981). Reduced displacements during rapid movements are probably due in part to this mechanical characteristic of muscles, generating pressures much less than that are required for speech. Thus, the timing of magnitude of inspiratory and expiratory muscle activation must be coordinated in relation to the continuous

changes in passive tissue mechanics of thoracic and abdominal structures (Draper, Ladefoged, & Whitteridge, 1959;Hixon, 1959).

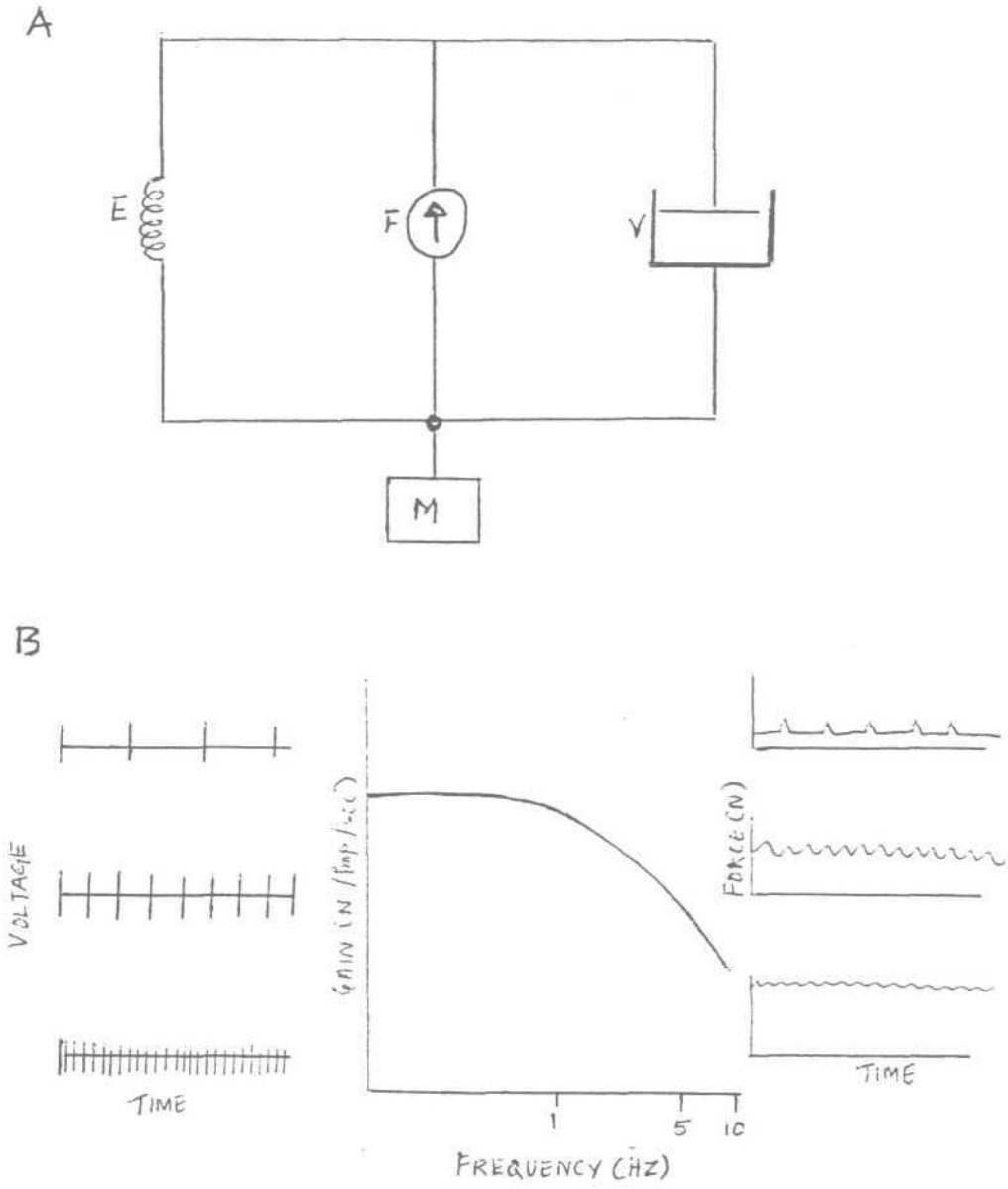


Fig.5(A) Biomechanical elements of muscle systems. Abbreviations: - E-elasticity, F- Force, V-Viscosity, M-Mass, (B) Summery description of low pass filter characteristics of muscle. The three waveforms displayed at the left refer to three rates of electrical stimulation applied to motor nerve innervating a muscle. The three forces on the right indicate the corresponding changes in force out put (in Newtons) recorded from the muscle. The middle plot summarizes the results of this type of experiment, showing the change in gain (Newtons divided by stimulation rate, impulses/second) as a function of the frequency of stimulation. (REF. YORKSTON AND BEUKELMAN 1988)

Muscle as a Low-Pass Filter

While considering the relationship between motor neuron activity and movement, physiologists have found it very useful to conceptualize muscle as a low-pass filter, that is, a system that transfers energy at low but not high frequencies. Motor units can discharge at sustained rates of about 20 to 35 Hz (Freund, 1983). But at high rates, mechanical properties of muscle fibers prevent motor units from generating distinct muscle-twitch forces (Fig 5B). From the same figure, it may be seen that at the lowest stimulation rate, relatively large muscle twitch curves result from each stimuli pulse. As stimulation rate increases the twitch curves tend to fuse until there is almost complete fusion of the individual twitch curves. The input-output relationship in this experiment may be depicted by a gain or frequency response curve like that shown in the middle of the Fig 5 B. The general form of the curve is like that of low-pass filter, and it is typical of all muscles. In the speech muscles the frequency response curve tends to fall off markedly above 3 to 4 Hz (Cooker, Larson & Luschei, 1980; Muller, MilenKovic & McLeod, 1984).

Conceptualizing muscle as a low-pass filter can be of use in evaluating aspects of both normal and abnormal speech. For example, when movement rates are increased beyond a certain point, there is invariably a reduction in the structural displacement which is most apparent at maximal diadokokinetic rates. This may be due to mechanical properties of muscle as well as a tendency for antagonistic muscles to be co-activated at rapid movement rates (Freund, 1983).

Sensory system contributions to speech motor control

The issue of contribution of sensory input to the co-ordination of speech muscle actions is intrinsic to the problem of how speech movement control in coded

in nervous system, and thus, it is significant for study of dysarthrias. Basic information on the anatomy and physiology of the human nervous system indicates that sensory afferents have strong diverse synaptic input to the numerous brain centers involved in the control of speech movement (Carpenter, 1976, Larson & Pfingst, 1982). Reflex experiments showing short-latencies in EMG recordings following stimulation of different sensory systems demonstrate short. Latency projections of Cutaneous, muscle, and auditory receptors to motoneurons innervating the speech muscles (Bratzlabsky, 1976; Spair, McClean, & Larson, 1983; Smith and Luschei, 1983).

Spatial Targets

An important feature of speech movement control is that relatively constant spatial targets are consistently achieved with structures that are continually changing their positions and biomechanical states (MacNeilage, 1970). The brain routinely integrates sensory information from peripheral mechanoreceptors in controlling speech movements. Experimental evidence for this is provided with the observation that with controlled changes in jaw opening as produced with bite blocks, the spectral pattern of the first periodic waveform of a vowel is relatively invariant (Lindblom. Lubker&Gay, 1979).

The muscle actions associated with the production of individual speech sounds vary widely as a function of phonetic environment. (Fromkin, 1966, MacNeilage & Declerk, 1969; Ohman, 1967). It is also known that the movement parameters of individual articulators vary widely across repetitions of constant phonetic sequences involving the same overall system output. Although other explanations are plausible, it seems likely that these features of speech movement control are partially dependent

on the use of sensory information in the organization of input to speech muscle motoneurons.

Studies of sensory function

The principal technique used to study sensory function in speech production has involved observations of speech motor output following controlled alteration of the normal pattern of sensory input during speech (Borden, 1979). In many cases, sensory alterations results in adaptive or compensatory motor responses. For example, presentation of masking noise is followed by increased vocal intensity (Lane & Tranel, 1971) or application of force perturbations to the jaw result in rapid compensations by the lips (Folkins&Abbs, 1975).

It has recently been observed that small force perturbations (10-55 grams) applied unpredictably to the lower lip during speech result in rapid compensatory responses by the upper and lower lips (Abbs & Gracco, 1984; Gracco & Abbs, 1985). The magnitude of the compensations are in proportion to lower lip displacements and are adequate to ensure a normal production of the speech utterance. These findings strongly suggest that perioral mechanoreceptor information is integrated on a continuous or rapid- intermittent basis within the neuromotor systems controlling speech movement.

Efferent control of sensory information

An important concept in motor physiology is that sensory processing at the spinal and brain stem levels may be under efferent or descending control by higher centers in the brain. This is most obviously manifested in the gamma motoneuron system which regulates muscle-spindle receptor sensitivity. It is now recognized that

alpha and gamma motoneurons are coactivated during movement. This provides for continuous response of muscle spindles during muscle shortening.

Another type of efferent control of sensory information involves direct descending input to the primary afferent neurons and interneurons within brain stem and spinal sensory pathways. Recent experiments on cat locomotion suggest that the sensitivity of peripherial reflex pathways are dynamically modulated during movement execution (Forssberg, 1979), that is for some behaviors, the brain actively controls how and when particular forms of sensory input will influence motoneuron discharge. Continued research is required to determine whether this is an important aspect of speech motor control.

Any disturbance of this neuromuscular configuration as a result of the weakness, paralysis or incoordination of the speech musculature or as a result of lesions in the nerves supplying the musculature results in speech dysfunction which are known as dysarthrias.

Description of disturbances in speech due to neurological involvement appears in early Egyptian hieroglyphics and can be traced periodically through the medical literature of several civilizations. A manuscript in a surgical papyrus composed at about 3500 B.C., contains the first use of the word "brain' and present 13 descriptions of skull fractures, bleeding from the nose and ears following fractures which are cited as disturbances of speech. McHenry (1969) quotes a case presentation which was perhaps the earliest description of neurogenic speech disturbance although the disorder could be either dysarthria or aphasia. The medical writings of the Greeks also contain such descriptions. Only scattered reference to the nervous system are found among the Homeric Greeks (Pre-Hippcratic); but with Hippocrates, an ancient clinical descriptive neurology, including references to speech loss was born.

In "Epidemics' Hippocrates describes hemiplegia, convulsions, paralysis of the right arm and loss of speech, which would have been aphasia, dysarthria or combination of both. "—the woman was seized with a fever in the 3rd month of pregnancy. There was pain in the joints, head, neck and around about the right articulate clavicle. Very shortly, the tongue became unable to articulate and the right arm was paralysed.... Her speech was delirious....speech later was indistinct but she was no longer paralysed...." (McHenry 1969).

During the middle ages and renaissance few advances were made from the original descriptions of the Greeks in understanding the neurogenic speech loss. The seventeenth and eighteenth centuries also were somewhat barren of detailed reports on the topic but by the nineteenth century the revival of interest and curiosity merged into a coalescence of scientific inquiry and clinical writing. Most of the observations and detailed case reports during this time revolved around the disturbance of speech, auditory comprehension, reading and writing which were eventually called aphasia. But it was also during this period that investigators such Jackson (1878) suggested that many disturbances of speech were not necessarily linked to symbolic impairment. Such non-symbolic impairments were termed "dysarthria ".

The term dysarthria is more comprehensive and precise than the traditional definition - imperfect articulation of speech caused by nervous system breakdown.

Further, the changes in the definition of term dysarthria are as under :

[11] Dysarthria is a Greek term dys + arthroun, which means inability to utter distinctly". As the word is usually employed in the field of speech disorders it implies any impairment of articulation caused by agenesis of, or damage to the nerve centers or tracts (other than those of the language areas of cerebral cortex) immediately

involved in direct control of the musculature used in the enunciation and pronunciation of vowels and consonants (West and Ansberry, 1968).

The concept of unitary dysarthria is being refined by using the term " dysarthophonia" by Rosenbeck and La Pointe (1978), Green (1964) and Peacher (1950). Here the respiratory, resonatory systems are also usually being found involved leading to i) articulatory ii) respiratory iii) phonatory iv) resonatory and v) prosodic disturbances. According to Peacher (1950), " problems in oral communication due to paralysis, weakness or in-coordination of the speech musculature resulting from the damage of the central or peripheral nervous system lead to dysarthria. " " The term covers all motor disturbances of speech exclusive of symbolic and integrative functions. "

Arnold (1969) has also considered dysarthrias as disorders of oral speech resulting from lesions within the cerebral centers, pathways and nuclei of the nerves involved in the speech event. Darley et al (1969) initially considered dysarthria as a collective name for a group of speech disorders resulting from disturbance in muscular control over the speech mechanism, due to damage to the central or periphral nervous system. (They designated it as a problem in oral communication due to paralysis, weakness or incoordination of the speech musculature). Later on, Darley, Aronson and Brown (1975) while discussing definitions of dysarthria have stated that..."the term will encompass coexisting motor disorders of respiration, phonation, articulation, resonance and prosody. It will also encompass isolated single process impairments such as an isolated articulation problem due to cranial nerve XII involvement, an isolated palato pharyngeal incompetence of neurogenic origin or an isolated dysphonia due to unilateral vocal fold paralysis. "

Considering all the above definitions, it can be concluded that "dysarthrias" are manifested as disrupted oral communication due to paralysis, weakness, abnormal tone or incoordination of the muscles used in speech and encompass co-existing motor disorders of respiration, phonation, resonation, articulation and prosody.

Dysarthria originates as a result of lesion either in the central nervous system or peripheral nervous system. For the lesions in the CNS or PNS the nature of the etiology may be vascular neoplastic, traumatic, infectious, toxic, metabolic . The nature of the cause does not result in the distinctiveness of certain patterns of dysarthria. Rather the different parts or levels of the motor system are impaired. The site of lesion and its extent will determine the abberations of movement that occur in specific sets of muscles implicated in speech. Since the symptoms in various dysarthrias overlap, it is difficult to pinpoint a fixed etiology for each of the disorders.

Pathologic Neuromuscular conditions Associated With Dysarthria

Dysarthrias are found to be associated with various pathologic neuromuscular conditions, for example spasticity, athetosis, rigidity, tremor, hypokinesis and flaccidity. Each of these conditions is relatively distinct within clinical neurophysiology and the nature of their associated neural mechanisms.. In some cases it would have been possible to find out the specific neuromotor pathology as related to specific symptoms of dysarthria. For example, the effect of flaccid paralysis of the spinal musculature on respiratory function during speech has been studied by Putnam and Hixon (1984). In other cases the relationship between neuromotor pathology and dysarthric speech patterns is not well understood. The association between dysfunction of basal ganglia and variability in speaking rate in Parkinson's disease is such a case (Netsell, Daniel & Celesia, 1975). In either instance, an understanding of

the mechanisms of pathologic neuromuscular conditions is important for research and clinical practice with the dysarthrias.

While considering the mechanisms of various pathologic motor signs, clinical neurologists often distinguish between primary or negative symptoms and secondary or positive symptoms. Negative symptoms are those resulting directly from functional loss of certain neurons and positive symptoms are seen as release phenomena resulting from disinhibition of healthy neurons. Thus, paralysis due to alpha motoneuron dysfunction is considered a negative symptom, whereas hyperactive reflexes associated with cortical damage are considered a positive symptom.

Adams (1973) suggests that different types of neuromuscular dysfunction are appropriately classified as paralysis, disorders of muscle tone, or forms of involuntary movements. This tripartite distinction is useful; however, motor disorders are generally discussed in relation to those parts of the nervous system that are primarily involved in disease process or that constitute the site of lesion. Modern text books in neurology typically consider disorders of movement in relation to the dysfunctions of lower motoneurons, upper motoneurons, cerebellar and basal ganglia (Adams & Victor, 1985). This general organization is used here in reviewing pathologic neuromuscular conditions associated with dvsarthria.

Lower Motoneuron and Motor unit Dysfunction

The alpha motoneurons within the brainstem motor nuclei and within the anterior horns of the spinal cord are known as lower motoneurons. These cells and the muscle fibers they innervate comprise different motor unit types discussed earlier. Various types of trauma or disease state may affect selected portions of the motor unit

and thereby produce characteristic signs of neuro-motor dysfunction (Woodbury, Gordon & Conrad, 1965). Destruction of the motor neuron cell body or axon results in the abolition of muscle contraction in the affected motor units. Because the motor unit is the final common pathway for muscle contraction, both reflex and voluntary movements are impaired. Destruction of lower motor neurons results in a condition of flaccidity or muscle softness, reduced reflex magnitudes and muscle atrophy. Reduced muscle tone or hypotonia, lack of resistance to passive movement is also often noted, and is believed to result in part from the absence of reflex contributions from muscle spindles and other mechanoreceptors. In some muscle systems, gamma motor neurons are likely to be affected by the lesion, and these are essential in maintaining appropriate levels of muscle spindle sensitivity, which in turn contribute to normal muscle tone.

Particular diseases may affect specific portions of the motor unit and result in characteristic motor signs. For example, the muscle end plate is selectively affected by myasthania gravis, which is characterized by weakness & heightened fatiguability. Myotonia is a condition of the muscle fiber membrane which prevents muscle from relaxing normally and thus impairs voluntary control. The contractile mechanism of the muscle fiber membrane which prevents muscle from relaxing normally and thus impairs voluntary control. The contractile mechanism of the muscle fiber may be affected by progressive muscular dystrophy which is manifested primarily as weakness.

In lower motoneuron disorders, small spontaneous visible contractions known as fasciculations sometimes may be observed. Fasciculations are triggered by events intrinsic to the motoneuron rather than synaptic input. Their presence in Amyotropic

Lateral Sclerosis and progressive muscular atrophy is believed to result from disease processes affecting the motoneuron.

UPPER MOTONEURON DYSFUNCTION

The term upper motoneuron encompases the pyramidal tract, corticospinal neurons, and cortico bulbar tract (Adams & Victor, 1985). The pyramidal tract includes only those neurons of cortical origin that descend over the internal capsule and decussate at the medullary pyramids. The corticospinal neurons include the pyramidal tract and more indirect pathways such as the corticorubrospinal and corticoreticulospinal tracts. The corticobulbar tract includes fibers that descend along with the corticospinal tracts and project to the motor nuclei of the brain stem, the reticular formation, and sensory relay nuclei. The cell bodies of upper motoneurons are found in several areas of the cerebral cortex, the primary ones being area 4, area6, and the parietal lobe.

Upper motoneuron lesions can occur at several levels of the nervous system and they seldom involve strictly corticospinal or corticobulbar pathways. For example, the close anatomical association of the ventral thalamic nuclei and the internal capsule makes it likely that lesions at that level will affect both upper motoneuron pathways and ascending thalamo-cortical projections from the basal ganglia and cerebellum. This point is clear in relation to the suggestion of Neilson and O'Dwyer (1984) that the characteristics of dysarthria in athetoid cerebral palsy, may be due in large part to inappropriate sensory processing over ascending thalamic pathways.

Upper motoneuron lesions tend to be characterized by conditions of excessive muscular tone known as spasticity. In the limbs, this is most notable in leg extensor

muscles and arm flexors, which also shows increased excitability to stretch stimuli. This increased reflex excitability is most likely due to both the removal of inhibitory influences and the increase of faciliatory drive to lower motoneurons. The neural mechanisms underlying spasticity are undergoing continued study by clinical neurophysiologists (Feldman, Young, & koella, 1980).

Recently Barlow and Abbs (1984) studied cranial- muscle fine motor control in a group of adult spastic dysarthrics They were particularly interested in the contribution of muscle-spindle dysfunction to disorders of fine motor control, and they analyzed the performance of three muscle systems known to have differing numbers of muscle spindles; the lips, tongue, and jaw. They did not observe greater deficits in motor control with the jaw and tongue, which are known to contain muscle spindles. This led Barlow and Abbs (1984) to suggest that deviant muscle-spindle activity is not a major cause of motor performance deficit in spastic dysarthria.

BASAL GANGLIA DYSFUNCTION

The principal structures of the basal ganglia are the caudate nucleus, putamen, globus pallidus, substantia nigra, and subthalamic nucleus. These structures and their associated pathways have a distinct distribution of neurotransmitters that is an essential aspect of their function in motor control. Understanding in this area was greatly advanced by studies of Parkinson's disease, a basal ganglia disorder involving loss of biogenicamine neurotransmitter, particularly dopamine. Dopamine is normally found in high concentration in the substantia nigra, but postmortem examination of the brains of Parkinsonian patients has shown low concentrations of dopamine and other biogenic amines.

Dopaminergic neurons project from the substantia nigra to the striatum and thalmus and their action is inhibitory. Thus, the loss of such neurons results in disinhibitory effects (i.e., reduced inhibition) on striatal and thalamic neurons. This has led to the view that pathologic motor symptoms in Parkinson's disease represent a type of release phenomenon, in which excitatory neuronal activity, normally held in check by basal ganglia input, are allowed to exert an abnormal level of influence.

Diseases of basal ganglia typically involve involuntary movements, disorders of muscle tone, and both reductions and exaggerations in the extent of movement. Hypokinesia or reduced activity is a prevalent symptom in some basal ganglia disorders, particularly Parkinson's disease. It is distinguished from paralysis in that it can occur without significant weakness. Another motor symptom that may have an origin similar to hypokinesia is bradykinesia. Bradykinesia refers to reduced velocity of movement and slowed speed of reaction.

Rigidity is a disorder of muscle tone, which unlike spasticity, shows a uniform amount of stiffness in response to passive movements. It is often present in the oral-facial musculature of Parkin-sonian individuals, and it may contribute to bradykinesic speech movements.

Another prominent sign of basal ganglia dysfunction is tremor. Generally, tremor refers to an involuntary oscillatory movement. A distinction is made between normal and pathologic tremor; the former typically occurs at frequencies of 8 to 12 Hz, and the later of frequencies of 3 to 6 Hz, although there can be an overlap in these ranges. For example, on prolonged sustained activation of a muscle, nonimpaired subjects will show a downward shift in tremor frequency to approximately 4Hz and an increase in tremor amplitude (Gottlieb & Lippold, 1983; Stiles, 1976). The mechanisms of tremor have been associated with reflex, central

oscillatory, and mechanical factors (Stein & Lee, 1981). Three types of pathologic tremor are usually distinguished; resting, postural, and action. Resting tremor is present when a structure is not maintaining a fixed posture of executing a movement. Postural tremor occurs during maintenance of a fixed posture, and action tremor during volitional movement.

Disorders of basal ganglia are sometimes characterized by exaggerated or hyperkinetic movement. Three major forms of hyperkinesia are chorea, athetosis, and dystonia. Chorea refers to rapid, unpredictable movements which may be simple or complex in form. Athetosis refers to an inability to maintain a fixed posture due to slow involuntary movements. Chorea and athetosis are sometimes seen in combination in individuals with Huntingdon's chorea, a condition involving the striatal neurons. The concept of dystonia is closely related to athetosis, and generally refers to a postural exaggeration of an athetoid-like movement.

CEREBELLAR DYSFUNCTION

Lesions of the cerebellum in humans result primarily in conditions of ataxia and hypotonia. Ataxia refers to a disorder of volitional movement that involves errors in rate, range, force and direction of movement (Holmes, 1979, Thatch, 1980). The ataxic individual often overshoots spatial targets with the limbs and then produces excessively large corrective movements. When these corrective movements become rhythmic, they are termed *intention tremor*, which is most marked at the end of movements. Other terms used to describe ataxic behavior are *asynergia*, a lack of muscle coordination, and dysmetria, errors in the range of movement.

The cerebellum is believed to be responsible for much of the automatic nature of motor behavior. This is strongly suggested by the observations of Holmes (1979)

on individuals having ipsilateral cerebellar lesions. One of his patients who had a lesion in the right side of the cerebellum commented that " the movements of my left hand are done subconsciously, but I have to think out each movement of my right arm." The loss of automaticity is reflected in a "decomposition of movement, or the performance of actions in successive parts rather than as a whole" (Thatch, 1980). The decomposition of movement is borne out in the speech patterns of individuals having dysarthria in association with cerebellar lesion.

Hypotonia, or a reduced resistance to passive movement, is another symptom common in human cerebellar disorders. It is believed to result from reduced activity of both alpha and gamma motoneurons (Gilman, 1970). It has been suggested by some that hypotonia is the primary deficit or negative symptom in cerebellar dysfunction and that ataxic symptoms are secondary or positive symptoms. Others suggest that the principal function of the cerebellum is coordination and that ataxic symptoms are primary in cerebellar disorders. Thatch (1980) indicates that this dispute is largely semantic, as none of the observed deficits provides an unmistakable clue to the nature of cerebellar function." Attempts have been made to classify dysarthrias into different types based on age of onset, general course, disease processes, neuroanatomic areas involved etc.

These attempts play an important and essential role in the understanding of dysarthrias as there are many other communication disorders caused by lesions of the CNS and /or PNS. These disorders can be brought under two heads : that is ,

1) Those attributable to the impairment of speech musculature - dysarthrias.
2) Those attributable to the impairment of a higher level mechanism, the motor speech programming - apraxias

Both of these types involve distinctive gestalts of impairment of various basic processes of speech but do not involve impairment of language as an integral features which is the case in aphasia.

Also, there can be communication disorders without the involvement of the CNS or PNS. These are grouped under dyslalias. In order to rule out the participation of each one of them, these should be differentially diagnosed. *Hence there is a need for classification of dysarthrias.*

Classification of dysarthria

Darley, Aronson, and Brown (1975) discuss several approaches for classifying the dysarthrias. These approaches are listed in Table A. Each system has advantages and disadvantages.

Classification bv age of onset suggests that dysarthria in a state of evolution (child) may require different remedial approaches from those used with dysarthria in a state of dissolution (adult). Typically, the symptoms, course, and causes differ between congenital dysarthria and acquired dysarthria. But this system promotes an artificial dichotomy. For example, injuries to the nervous system that cause dysarthria in children are frequently pooled under the term *cerebral palsy.* Similar injuries resulting in dysarthria in adults direct one in search of a specific cause.

The most popular historic approaches for classifying the dysarthrias are those that focus on the general cause, disease process, neuroanatomic area, or the cranial nerves involved. These efforts attempt to use the dysarthric patient's speech and voice characteristics either to localize the damage in the nervous system or to identify the cause. Froeschels (1943), Luchsinger and Arnold (1965), and Brain (1965) formulated systems that classified the dysarthrias according to the neuroanatomic site

of lesion and Peacher (1950) and Grewel (1957) developed classifications that combine both the nervous system involved and the specific cause. While these approaches are useful in confirming the localization of the lesion or the system involved and possible identification of cause of dysarthria, they are limited in providing useful information for the speech pathologist who is required to evaluate and treat the dysarthric patients.

Systems that are used to classify the dysarthrias according to the speech processes have been found to have some overlap. These approaches can be arranged into a hierarchy based on the increasing precision of yielding information about dysarthria. First, the dysarthrias can be classified in terms of the speaking process (or processes) that is involved. Is the patient's respiratory activity adequate for speech? Are the abdominal and thoracic muscles functioning to produce and regulate adequate air pressure for speaking? Or, as is the case in the spinal form of multiple sclerosis, does the patient display weak respiratory muscles inadequate for obtaining and controlling air supply? If so, the dysarthria can be classified as having a significant respiratory component. Similarly, appropriate questions can be asked about phonation: Do laryngeal muscles permit adequate voice production and pitch change? In terms of resonance, do pharyngeal and palatal muscles contract to manipulate the size, shape and number of cavities needed for normal selective amplification of sound? And in articulation, are tongue, lips and facial muscles adequate to permit the speed, range, strength, and coordination of movement necessary to produce adequate intelligibility? In prosody, do respiratory, laryngeal, and articulatory muscles act in sequence to provide pitch, intensity, and time variations needed for normal stress? Typically, a dysarthria will involve more than one speech process. When it does, the

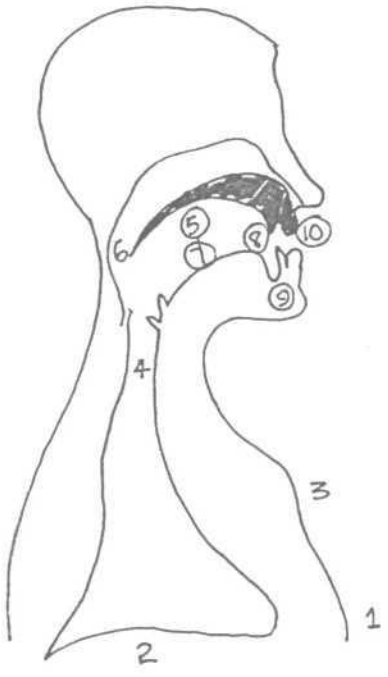
speech pathologist attempts to determine the relative contribution of each to the overall severity of dysarthria.

Table A. Showing possible classification systems of dysarthrias -Darley et. al. (1975).

Types	Examples
Age at onset	Congenital or acquired
General cause	Vascular, neoplastic, traumatic, infectious, etc.
Disease processes	Multiple sclerosis, myasthenia gravis, Parkinsonism, etc.
Neuroanatomic area involved	Cerebral, cerebellar, brainstem, etc.
Cranial nerves involvedV,	VII,IX,X,XI,XII
Speech processes involved	Respiration, Phonation, resonance, articulation, prosody
Speech valves involved	Respiratory, laryngeal, pharyngeal, velar, lingual, dental, labial
Speech events involved	Neural, muscular, structural, aerodynamic, acoustic, j perceptual
Perceptual characteristics	Pitch, loudness, voice quality, respiration, prosody, articulation, general impression

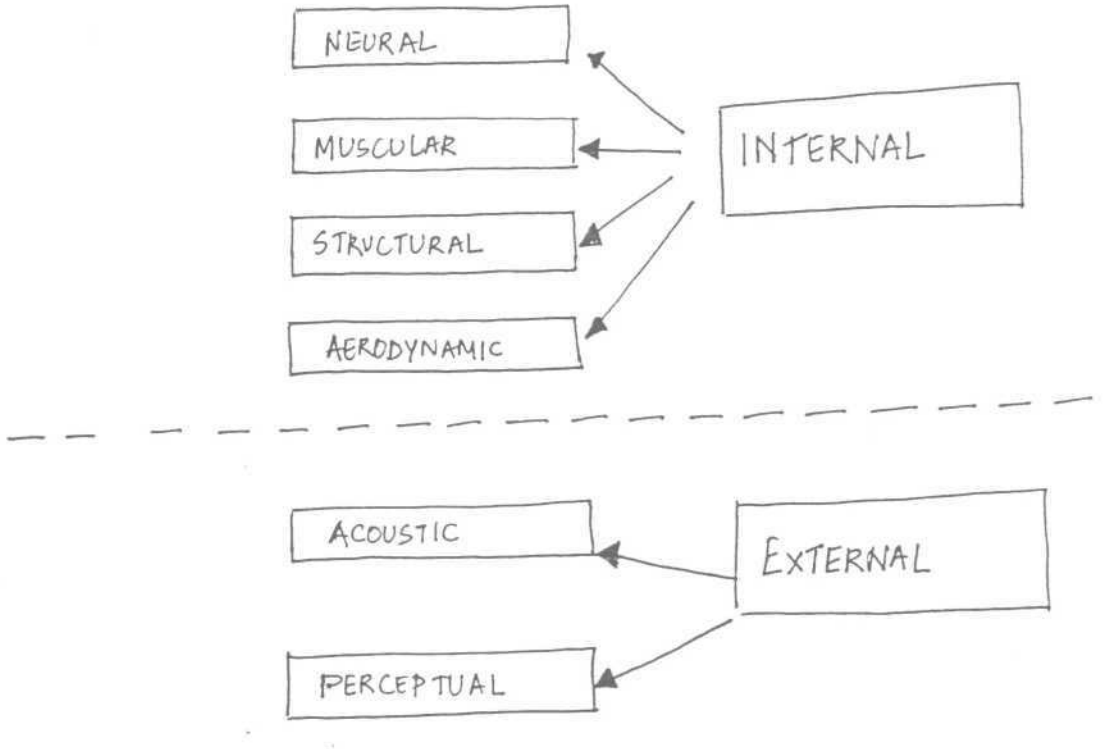
Second, classification based on the integrity of the muscular valves used in speech providems more precise information than the classification based on speech process systems involved. Netsell (1972, 1971) has described the speaking mechanism as being comprised of a series of functional components. This is illustrated in figure 6. Each component represents an area in which muscle activity-interrupts (valves) or releases the air used in speech. Dysarthria results when a neurological disorder disrupts the speed, range, strength, and coordination of movement necessary for normal valving of the air stream. Each numbered component represented in figure 6 can be assimilated into the speech process classification just discussed. For example, abdominal muscles (one), diaphragm (two), and rib cage and associated muscles (three) comprise respiration. Laryngeal muscles (four) produce phonation. Velopharyngeal muscles (five), tongue-pharynx (six), and tongue muscles (seven and eight) create resonance. And, velopharynx (five), tongue-pharynx (six), tongue muscles (seven and eight), jaw (nine), and lips (ten) produce articulation. Prosody is the result of coordinated valving of one or more of the ten functional

FIG 6: FUNCTIONAL COMPONENTS OF THE SPEAKING MECHANISM [NETSELL, 1971, 1972]



1 → ABDOMINAL MUSCLES
2 → DIAPHRAGHM
3 → RIBCAGE AND ASSOCIATED MUSCLES
4 → LARYNX
5 → VELOPHARYNX
6 → TONGUE - PHARYNX
7 → TONGUE (MIDDLE)
8 → TONGUE (ANTERIOR)
9 → JAW
10 → LIPS.

FIG 7: TWO WAYS OF OBSERVING DIFFERENT LEVELS OF SPEECH EVENTS.



components. This system permits greater precision in determining the location of disordered speech movements (e.g., weak diaphragmatic contraction) than is possible with a speech process classification (e.g. respiratory involvement). More precise classification leads to highly focused remedial efforts that are more effective.

Kent and Hixon's model (1976). (fig.6) Organizes motor speech activity into events occurring at several levels. These events are reproduced in figure 7 . Internal events include neural and muscle activities, structural movements, and the dynamics of air pressure and airflow. As one descends through these four levels, the knowledge about dysanhria increases and the need for interdisciplinary interaction and elaborate instrumentation decreases. Hardy (1967) has discussed most of these events and has offered several suggestions for physiologic research in dysarthria.

Neural and muscular events can be observed using EMG. This technique looks at the integrity of the nervous system by recording the electrical activity in muscles used for speech. The cooperation of a neurologist and elaborate instrumentation are necessary for this purpose. Leanderson, Meyerson and Persson (1972), Leanderson, Persson, and Ohman (1970), Netsell (1972), Netsell and Cleeland (1973), and Netsell, Daniel, and Celesia (1975) have studied EMG activity in dysarthric patients.

Structural events, particularly movements of the lip, jaw, tongue, velum, pharynx, and larynx, can be observed using cinefluorography (x-ray). This technique permits a look at the range and speed of structural movements during speech. It requires collaboration with a radiologist and x-ray technician and availability of elaborate, expensive equipment. Kent and Netsell (1975); Kent, Netsell, and Bauer (1975); Netsell (1973); Netsell and Kent (1976); and logemann et al. (1974, 1973) have provided evidence of structural movement abnormalities in dysarthric patients.

The aerodynamics of speech may be measured using the elaborate instrumentation described by Hixon (1972) or by more basic instruments, such as the U-tube manometer. Measurements range from lung volume and breath pressure to oral and nasal airflow. Marquardt (1973), Netsell (1973), Netsell, Daniel, and celesia (1975), and Putnam & Hixon (1984) have reported air volume, pressure, and flow characteristics in dysarthric patients.

Two speech events, acoustic and perceptual, are observed externally. Acoustic analysis requires instrumentation to obtain a visual representation of the speech signal. Primary measurements include observation of the physical properties of sound: frequency, intensity, and temporal relations. Kent and Netsell (1975), Lebrun, Buyssens, and Henneaux (1973);Lehiste (1965); and Ludlow and Bassich (1984) have used acoustic analysis to describe dysarthric speech.

Perceptual analysis requires the trained ears of experienced clinicians, the speech samples under variety of talking tasks and a system for classifying samples that are heard. Because most speech pathologists do not have access to the instrumentation or interdisciplinary personnel necessary for evaluating and classifying dysarthrias according to neural, muscular, structural, aerodynamic, and acoustic events, perceptual classification is convenient and popular.

Darley, Aronson, and Brown (1969; 1975) conducted studies known as the Mayo Clinic studies. By listening to speech samples collected from over 200 dysarthric patients with unequivocally diagnosed neurologic lesions or diseases, they concluded that dysarthrias resulting from damage in different parts of the nervous system sound different and can be differentiated according to specific perceptual dimensions. Utilizing 38 dimensions of speech and voice. Darley, Aronson, and

Brown (1969) developed a perceptual classification system composed of six different types of dysarthrias. Each has specific perceptual characteristics, each indicates the probable origin of nervous system disruption, each is associated with specific causes, and each results from specific abnormal neuromuscular condition.

Table B lists six different types of dysarthrias. The first five are relatively "pure" types, distinguished by specific perceptual characteristics that specify the probable location of nervous system involved. Each results from discernible causes and each is characterized by specific neuromuscular conditions that result in abnormal speech movements. Experienced speech pathologists can use their trained ears to listen to neurologically impaired patients and classify. If, for example, a patient displays breathy voice quality, hypernasality, and consonant imprecision, he or she is classified as having a flaccid dysarthria. This classification implies that the lesion involves the lower motor neuron system and results from infection, tumor, CVA, congenital condition, a specific lower motor neuron disease, or trauma. The information is conveyed to the patient's physician, who compares it with the results of his or her neurologic evaluation and special tests. Typically, the speech classification will agree with the medical information, and the physician will diagnose the patient as having bulbar palsy, which is a medical diagnosis consistent with involvement of the brainstem. If the patient demonstrates spastic dysarthria, and if the physician agrees, the diagnosis would be pseudobulbar palsy, implying a lesion of the upper motor neuron system. Other pure types are ataxic dysarthria, resulting from cerebellar involvement; hypokinetic dysarthria (Parkinson's dysarthria), resulting from extrapyramidal involvement; and hyperkinetic dysarthria, also resulting from extrapyramidal involvement.

The mixed dysarthrias listed in Table B occur as frequently as the pure types. The first three mixed dysarthrias listed are based on empirical evidence collected in the Mayo Clinic. The final type, unlike others, permits classification of dysarthrias involving multiple systems; for example, a spastic- ataxicflaccid dysarthria resulting from a tumor that has metastasized to involve upper motor neurons, the cerebellum, and lower motor neurons.

The three mixed types, based on perceptual data, demonstrate the validity of the system. Darley, Aronson, and Brown (1969) verified the perceptual characteristics in amyotrophic lateral selerosis (ALS). The perceived characteristics combine those heard in pure flaccid dysarthria and pure spastic dysarthria. The neuromuscular condition represents movement problems seen in both. Therefore ALS involves both upper and lower motor neurons and is classified as a mixed, spastic-flaccid dysarthria. Similarly, Berry et al. (1974) demonstrated that the perceived characteristics in patients suffering from Wilson's disease imply upper motor neuron, cerebellar, and extrapyramidal involvement. Finally, Darley, Brown, and Goldstein (1972) found that the movement disorders and the perceptual characteristics in multiple selerosis (MS) varied according to the system or systems (upper motor neuron cerebellar, or lower motor neuron) involved and could be identified.

Thus dysarthrias can be classified in different ways. Until the availability of sophisticated instrument it is sufficient to classify according to Kent's (1976) and Hixon's (1976) levels of speech events. The perceptual classification system developed in the Mayo Clinic is useful. Different types of dysarthrias can be identified by their perceptual characteristics. These predict the cause, the localization of neurogenic involvement, and the neuromuscular condition. It must be remembered

Table B : Mayo clinic perceptual classification of dysarthrias (Darley et. al. 1969, 1975)

Type	Perceptual characteristics!	Localization	Causes	Neuromuscular Condition
Flaccid dysarlhria	Brcalhy voice quality, hypcrnasalily, consonant imprecision, audible inspiration.	Lower motor neuron	Viral infection (c.g.,Poliomyclitis),tumor, CVA, congenital conditions, disease (c.g.,myasthenia gravis), palsies(e.g.,bulbar,facial)trauma	Flaccid paralysis, weakness, hypotonia, muscle atrphy, fnsciculations.
Spastic dysarthria	Strained-strangled-harsh voice quality, hypemasality, slowrate, consonant imprecision	Upper motor neuron	CVA, tumor, infections(e.g.,encephalitis),trau ma, congenital conditions (e.g.,spastic cerebral palsy)	Spastic paralysis, weakness, limited range of movement, slowness of movement.
Ataxic dysarlhria	Imprecise consonants, excess and equal stress, irregular articulalory breakdown	Cerebellar system	CVA,tumor,trauma,congenitalc ondition(e.g.,ataxicccrcbral plasy,Fricndrcich's alaxia) infection, toxic effccts(e.g.,alcohol)	Inaccurate movement, slow movement, hypotonia.
Hypokinetic dysarthria	Monopitch,monoloudncss, reduced stress, imprecise consonants, inappropriate silences, short mshes	Extrapyramidal system	Parkinson's discase.drug induced (e.g.,reserpine or phenothiazine)	Slow movements, limited range of movement, immobility, paucity of movement, rigidity, loss of automatic aspects of movement, resting tremor
Hyperkinetic dysarthrias Predominantly quick	Imprecise consonants, prolonged intervals, variable rate, monopitch, harsh voice quality,inappropriate silences, distorted vowels, excess loudncss variation	Extrapyramidal system	Chorea, infection, Gilles de la Tourett's syndrome, ball ism	Quickinvoluntary movements (e.g.myoclonic jerks, ties etc.), variable muscle tone
Predominantly slow	Imprecise consonants, straincd-strangled-harsh voice quality, irregular articulatory break down, monopitch, monoloudncss	Extrapyramidal system	Alhctosis(c.g.,acquircd orcongenital),infection, CVA, tumor, dystonia, drug induced(c. g., t ranquui 1 izcrs),dy s-kincsia(c.g.,torticallis,or lardive dyskincsia)	Twisting and writhing movements, slow movements, involuntary movements, hypertonia
Mixed dysarthria Spastic-flaccid	Imprecise consonants, hypcrnasality, harsh voice quality, slow rate , mono-pilch, short phrases, distorted vowels, low-pitch, mono-loudncss, excess and equal stress, prolonged intervals	Upper and lower motor neurons	Amyotrophic lateral sclcrosis,trauma,CVA	Weakness, slow movement, limited range of movement

Spastic-ataxic-hypokinetic	Reduced stress, monopitch,monoloudness,imprecise consonants, slow rate, excess and equal stress, low pilch.irrcgular articulatory breakdown	Upper motorneuron.cerebellar.extrapyramidal	Wilson's disease	Intention tremor, rigidity spasticily, slow movement
Variable (spastic-ataxic-flaccid)	Variable(e.g.,slow rate, harsh voice quality.irregular articulatory breakdowns)	Variable (e.g.,upper motor neuron,cerebellar, lower motor neuron)	**Multiple** selerosis	Variable (e.g., spasticity, weakness, slow movement,limited range of movement., inaccurate movement.
Others	Variable	Variable	Multiple CVAs Tumor, Trauma, disease etc.	Variable

Table C : Summary of Mayo Clinic research on motor speech disorders

Dysarthria type	Neurologic conditions	Location of ncuropathology	Neuromuscular movement tone deficit	Clusters of deviant speech dimensions	Most distinctive speech deviations
Flaccid	Bulbar palsy	Lower motor neuron	Muscular weakness; hypotonia	Phonatory incompetence; resonatory incompetence;Phonatory-prosodic insufficiency	Marked hypcrnasality, often with nasal air emission; continuous breathiness; audible inspiration.
Spastic	Pseudobulbar palsy	**Upper motor neuron**	Reduced range, force, speed, hypertonia.	Prosodic insufficiency; Articulatory-resonatory incompetence; phonatory stenosis	Very imprecise articulation; slowrate;low pitch;harsh strained-strangled voice
Ataxic	Cerebellar ataxia	Cerebellum	Hypotonia; reduced speed; inaccurate range,timing, direction.	Articulatory inaccuracy;prosodic excess; phonatory-prosodic insufficiency.	Excess and equal stress;phoneme and interval prolongation; dysrhythmia of speech and syllabic repetition; slow rate; some excess loudness variation.
Hypokinetic	Parkinsonism	Extrapyramidal system	Markedlyreduced range; variable speed of repetitive movements; movement arrest; rigidity.	Prosodic insufficiency plus four uncorrelated dimensions	Monopitch, monoloudness, reduced overall loudness; variable rate; short rushes of speech; some inappropriate silences.
Hypcrkinetic : 1 .quick	(a) Chorea (b) Myoclonus (c) Gilles de la Tourette's syndrome	Exlrapyramidal system	Quick.unsustaincd, random, involuntary movements	Nearly all clusters of speech dimensions	(a) Highly variable pattern of imprecise articulation;episodes of hypcrnasality;suddcn variations in loudness; (b) Rhythmic hypcrnasality; rhythmicphonatory

					intcruption; (c) Sudden ticklike grunts, barks, coprolalia.
2. Slow	(a) Athetosis (b) Dyskinesias (c) Dystonia	Extrapyramidal system	Sustained, distorted movements and posture; slowness; variable hypertonus	Cluster unreported Articulatory inaccuracy; prosodic excess; prosodic insufficiency; phonatory stenosis	(a)Distinctive deviations unreported Distinctive deviations unreported Prolongations of phonemes, intervels; unsteady rate, loudness.
3. Tremors	Organic voice tremor	Extrapyramidal system	Involuntary,rhythmic, purposeless oscillatory movements		Rhythmic alterations in pitch, loudness; voice stoppages.
Mixed	(a) Amyotrophic lateral sclerosis (b) Multiple sclerosis (c) Wilson's disease	Multiple motor system	Muscular weakness; limited range, speed	Prosodic excess, prosodic insufficiency; articulatory-resonatory incompetence; plionalory stenosis; phonuiory incompetence rcsonatory in competence	(a) Grossly defective articulation; extremely slow, laborious rate; marked hypcrnasalily; severe liarshness, strained-strangled voice; nearly complete disruption of prosody (b) Impaired control of loudness; harshness (c) Reduced stress; monopilch; monoloudncss; similar to hypokinetic dysarthria except no short rushes of speech

that the diagnosis of dysarthrias listed in Table B are based on speech characteristics . Medical diagnoses (e.g., bulbar palsy) differ in vocabulary, but they can be compared with the diagnoses based on speech characteristics. There are seven categories

1. Bulbar palsy
2. Pseudo bulbar palsy
3. Amyotrophic Lateral Sclerosis
4. cerebellar disorders
5. Parkinsonism
6. Dystonia
7. Chorea

Types of Dysarthrias - Description of Speech deviations and Neurological findings

Flaccid Dysarthria

In the execution of speech four of the cranial nerves are directly involved. They are trigeminal (V), Facial (VII), Vagus (X) and hypoglossal (XII)

A lesion in (i) the motor nucleus of these nerves and (ii) the peripheral nerves running from the nucleus to muscles, will result in the weakness of innervated muscles. This condition is referred to as flaccidity. Flaccid dysarthria results from the speech disorders due to the lesions of the lower motor neurons of each of nerves. The speech aberrations will be different in each case depending upon the nerves involved and the extent of involvement.

If the lesion is in V nerve, then the muscles of mastication will be weak. The patient will find it difficult to elevate his mandible to close bis mouth and keep it closed. The patient cannot move his mandible voluntarily to either side. If pressure is exerted on his lower teeth with a tongue depressor he may not be able to close his teeth together.

If the lesion is in VII nerve, then there is difficulty in pursing and retracting the lips; firming his cheek and facial muscles to permit impounding of air for phonemes requiring intraoral pressure. On examination of the speech mechanism one finds a droop of the affected side of the face in case of unilateral lesion, the unaffected side pulling upward and outward during a smile. The nasolabial fold will appear flattened on the weak side. With a bilateral lesion the lips may not be closed at rest; the smile elicited will be transverse; lip rounding and protrusion will be inadequate, when the patient puffs out his cheeks one can readily break the labial seal by pressing the cheeks.

If the lesion is in Xth nerve, then the patient may show palato-pharyngeal weakness or laryngeal weakness or both, with unilateral paralysis of the levator muscle of the soft palate, it can be seen that the weak side will hang lower than the intact side at rest, on phonation, the intact side will rise, the weak side will not. With bilateral paralysis the entire palate will elevate little or not at all on phonation. The gag reflex will be absent or diminished. When laryngeal muscles are affected, unilaterally or bilaterally laryngoscopic viewing will reveal failure of adduction to the midline or the vocal folds may appear bowed.

When the lesion is in XII nerve, the tongue will be weak, may appear smaller than average, may display atrophy of the borders or look shrunken and furrowed and may demonstrate fasciculations (small visible transient contractions of parts of a muscle). Protrusion and elevation of tongue tip may be difficult. In the case of a unilateral lesion the tongue on protrusion deviates to the affected side, in bilateral lesion weakness protrusion is symmetrical but limited in extent. Lateral weakness

may also be evident, leading to failure of the lateralized tongue to resist pressure exerted on the cheek.

In myasthenia gravis, which is a particular form of lower motor neuron disease, in which the impairment is an electrochemical one at the myoneural junction, any or all of these functions may be impaired. The most deviant aspect of speech and voice observed in a series of cases unequivocally diagnosed as presenting lower motor neuron impairment are:

1. Cluster of deviations indicative of resonatory incompetence are hypernasality, often of severe degree, audible nasal emission of air in the production of consonants which require large intraoral breath pressure, and abnormally short phrases during contextual speech attribute at least in part to air wastage at the palatopharyngeal port.

2. A cluster of speech and voice changes indicative of phonatory incompetence are breathy voice quality resulting from poor vocal fold adduction and air escape, audible inhalation of air (inspiratory stridor) due to inadequate abduction of vocal folds during inhalation and abnormally short phrases during contextual speech attributable at least in part to inefficient laryngeal valving and the patients inhaling more often than normal as they run out of air.

3. A cluster of deviations indicative of phonatory prosodic insufficiency, probably due to hypotonia (reduced tonus) of the laryngeal muscle are monotony of pitch, monotony of loudness and harsh voice.

4. Imprecise articulation of consonants can be, probably, attributed to impaired tongue movements and insufficient intraoral breath pressure.

The examination of Table C shows that breathy voice, hypernasality, audible nasal emission of air are more noticeable in flaccid dysarthria than in any of the other types. On neurologic examination patients with flaccid paralysis are found to have impairment of all types of movements, whether voluntary, automatic, or reflexive. The most prominent signs are weakness and hypotonia that is, reduced muscle tone, in myasthenia gravis. The most prominent feature is progression of weakness with continued use of muscles. Further Hyporeflexia, muscle atrophy.Further fasciculations are visible and fibrillations are evident on EMG. (Table D)

TABLE - D : Showing speech characteristics of Flaccid Dysarthria

XII nerve damage

Laryngeal	Velopharyngeal	Oral
Normal	Normal	Tongue : Weakness (Unilateral or bilateral and reduced range of motion)
		Imprecise vowels and lingual consonants

X nerve damage

Laryngeal	Velopharvngeal	Oral
Hoarseness, breathiness excessively low volume, diplophonia	Hypernasality and nasal emission.	Normal

VII nerve damage

Laryngeal	Velopharyngeal	Oral
Normal	Normal	Lip : Weakness (Unilateral or bilateral and reduced range of motion)
		Inprecise vowels and labial ' consonants

V nerve damage

Laryngeal	Velopharyngeal	Oral
Normal	Normal	Mandibular muscles : weakness
		Inprecision of vowels and labial consonants

Multiple nerve damage (XII, X, VII, V) effects

Laryngeal	Velopharyngeal	Oral
Breathiness reduced volume, inhalatory stridor	Hypernasality and nasal emission	Inprecision of vowels and labial consonants

Spastic Dysarthria

The musculature involved in speech production receive upper motor neuron supply from both the cerebral hemispheres. The term *pseudobulbar palsy* is used, to designate paresis or paralysis of the musculature supplied by these upper motor neurons, then the cause is bilateral, nerve supply to tongue and lips are inadequate, even a one-sided lesion can produce a speech problem. The final common pathways to the speech muscles are not impaired so the muscles are not flaccid. The distorted signals from upper motor neuron lead to changes in the muscle stretch reflexes and muscle tone that are identified as spastic, and the resulting speech changes are called spastic dysarthrias.

The characteristic clusters of disorders of speech and voice dimensions that commonly appear in spastic dysarthria are: -

1. A cluster of phonatory signs indicating phonatory stenosis, that is, a narrowing of glottis, leading to harsh voice quality, excessively low pitch, a strained strangled sound indicative of effortful voice production, the production of an effortful grunt at the end of an exhalation, and pitch breaks.

2. A cluster of features designated as prosodic insufficiency, probably resulting from restricted range of movements; monotony of pitch, monotony of loudness, reduction of the usual patterns of syllable and word stress, and shortness of phrases.

3. A cluster of three speech characteristics- imprecision of consonant articulation, distortion of vowels, and hypernasality- designated articulatory - resonatory incompetence, to indicate that muscle contractions are reduced in speed, force, and range of movement, leaving the palatopharyngeal port unable to close efficiently and the articulators unable to impede the breath stream.

4. Two features which constitute a portion of a larger cluster designated prosodic excess: equalization of stress with excessive stress on words and syllables usually unstressed, and slower than normal rate.

5. Breathiness of voice, which results either because of slowed movements of the vocal cords on adduction thus allowing wastage of air, or possibly a compensatory phenomenon adapted by the patient in trying to produce a less effortful phonation in the face of phonatory stenosis.

Some of these deviations are more prominent in spastic dysarthria than in any other type:i.e., low pitch level, with pitch breaks, harsh voice, the strained strangled sound of phonatory stenosis, slowed rate and shortness of phrases. On examination of speech mechanism it is seen that the lip, tongue and palatal movements are consistently executed more slowly than average and the extent of sluggish movements are restricted. Oral diadochokinetic rates will be slowed but still rhythmical.

The neurologic picture is one of paresis of the lower face and of the extremities of the opposite side in unilateral lesions of the brain, bilateral symptoms in case of bilateral lesions. Movement patterns rather than individual muscles are impaired. The most prominent features reported by the neurologist are that of increased muscle tone (spasticity), weakness (particularly of distal muscles) slowness of movement, and reduced range of movement. Other signs are hyperreflexia, absence

of superficial abdominal reflexes and the appearance of certain abnormal reflexes including the Babinski sign (extension of the great toe and fanning of the other toes when the sole of the foot is scratched) and the sucking reflex.

TABLE - E : Showing the speech characteristics of Spastic Dysarthria due to damage to bilateral upper motor neurons.

Laryngeal	Velopharyngeal	Oral
Hyperadduction of vocal folds	Inadequate palatopharyngeal closure	Slowness, Weakness
Strained, strangled voice, harshness excessively low pitch, monopitch	Hypernasality	Slow-rate (Alternate motion rate) Imprecision of consonants.

Ataiic Dysarthria:

Cerebellar dysfunction results in impairment of the coordination of skilled movements, including those of speech when the lesions are generalized or occur bilaterally. The timing of the component parts of movements is off, the force with which a movement is executed may be too strong or too weak, the amplitude or range of the movement may be poorly regulated and the direction of each movement may be poorly controlled. The result is a breakdown in the smooth rhythmic efficient production of speech; the resulting pattern of uncoordinated speech performance is labelled "ataxic dysarthria".

Following are the commonly observed disorders of speech and voice heard in groups of patients with cerebellar disease.

1. A cluster of three articulatory characteristics constituting of articulatory inaccuracy: imprecise production of consonants, distortion of vowels, and irregular articulatory breakdowns.

2. A grouping of four deviations constituting the cluster of prosodic excess, equalization of stress and excess stress on usually unstressed words and syllables, prolongations of phonemes, prolongation of intervals, and slow rate. The speech sounds too deliberately paced or "measured", the term "scanning speech" has been applied to this set of features heard frequently, but not exclusively in ataxic dysarthria.

3. Three signs of laryngeal involvement constituting the cluster Phonatory- prosodic insufficiency noticed are that of harsh voice quality, monotony of pitch, monotony of loudness. Four of these deviations mentioned above are more common in patients with ataxic dysarthria than any other dysarthric group, namely equalization of stress and excess stress on usually unstressed words and syllables, irregular breakdown of articulation, prolongation of phonemes, and prolongation of intervals between words.

On the oral speech mechanism examination it is observed that any oral movement will be performed jerkily, erratically, without fine control of direction or timing or extent. The performance may be quite variable from trial to trial, differing from the consistently slow and limited performance of the spastic dysarthria. Oral diadochokinetic repetitions may be normally rapid but are often remarkably dysrhythmic, accompanied by irregularities of pitch and loudness.

The usual neurologic findings in patients with cerebellar disease are flabbiness of muscles and reduction of muscle tone (hypotonia), Jerky muscular movements, wide-based-staggering gait, jerky, irregular arm movements, clumsy, slow finger movements, tremor in the use of a limb, and increase of that tremor towards the termination of the movement.

TABLE - F : Showing speech characteristics of Ataxic Dysarthria due to CerebellarDamage.

Laryngeal	Velopharyngeal	Oral
Approximate normal	Normal	Reduced control
Loudness variation, hoarse voice tremor	(Compensatory) Excessive and equal stress	(primary) Articulatory breakdown. Inprecision of consonants.

Hypokinetic Dysarthria:

In the disease of extrapyramidal system called Parkinsonism there is a general reduction of movement. This shows itself in speech also so the resulting dysarthria is labelled "hypokinetic dysarthria". Following are the most frequent characteristics of hypokinetic dysarthria identified in a series of Parkinsonian patients:

1. The most prominent characteristics are alterations of the prosody of speech i.e., monotony of pitch, reduced stress, and monotony of loudness.
2. Imprecise consonant articulation is often prominent with a marked reduction in the excursion of the articulators so that speech is often simply a slur.
3. Speech is sometimes arrested resulting in inappropriate silences and sometimes in repetitions of phonemes or syllables.
4. Speech is produced in short rushes, the rate at times seeming to accelerate within a phrase. Where as all other dysarthrias are characterised by a slower, than normal rate, many Parkinsonian patients speak at the rate judged to be faster than normals. The rate is often variable.
5. Voice quality is often breathy and loudness level is often reduced. Inadequate audibility is the presenting complaint of some Parkinsonian patients.

On the oral speech mechanism examination it is probably noted that repetitive tongue and lip movements are performed rapidly, sometimes inordinately rapid,but with reduced excursion so the performance is incomplete and the sounds produced are lacking in precision. Masked face with no expression, smile or blink.

On neurological examination salient features like rigidity (increased tone) that is often has a cogwheel character, restriction of range of movement, accomplishment of repetitive movements rapidly but with small amplitude, slowness of individual movements, and an alternating tremor at rest which subsides with movement are seen. Lesser signs of a confirmatory nature include loss of automatic associated movements (e.g. rotation of the body and swinging of arms while walking), paucity of movement, and hesitation and false starts in initiating movements.

TABLE - G : Showing the speech characteristics of Hypokinetic Dysarthria (Parkinsonism) caused due to damage of basal ganglia (substantia nigra).

Laryngeal	Velopharyngeal	Oral
Rigidity	Normal	Reduced range of motion
Monopitch excessively low volume & hoarseness		Accelerated rate imprecision of consonants

Hyperkinetic Dysarthria.

Patients with certain lesions of the extra pyramidal system will present involuntary movements, which interrupt ongoing purposeful movements. The term hyperkinesia is applied to all such occurences of involuntary movements. Some of the movement disorders are characterized by quick hyperkinesias, are myoclonic jerks, tics and chorea. Other involuntary movements rend to be slower, of more gradual onset, prolonged for variable periods, waxing and waning, these slow hyperkinesis include athetosis, tardative dyskinesia (drug-induced movement disorder), and dystonia.

The impairment in speech resulting from these movement disorders are known collectively as "hyperkinetic dysarthria". On examining the table H it can be seen that Hyperkinetic Dysarthria can be subdivided into quick, slow and tremors.

TABLE - H : Showing the subdivision of Hyper Kinetic Dysarthria.

Quick	Slow	Tremors
Chorea	Alhetosis	Organic voice tremor
Myoclonus	Dyskinesis	
Gillesde la Tourette's Syndrome	Dystonia	

1. Hyperkinetic dysarthria in chorea

In the ongoing speech performance of the patient with chorea one may observe sudden brief interruption of any of the basic motor processes of speech production.

a) Respiration may be interrupted by a sudden forced inspiration or expiration.
b) Phonation may be altered by sudden excessive loudness variations, voice stoppages, or voice breaks. Many subjects also present the signs of phonatory stenosis by presenting harsh voice and the strained strangled sound of effortful phonation against resistance.
c) Hypernasality frequently occurs, and because of the air wastage resulting from movements of palatopharyngeal incompetence.
d) Interference with the muscular adjustments of articulators are evident in impreciseness of consonant articulation and frequent vowel distortion.
e) Disturbances of prosody are prominent as momentary breakdowns of speech occur, as the patient tries to complete units of speech between these breakdowns and cautiously proceeds as though to avoid anticipated breakdowns. Among the alterations of prosody that have been observed are monopitch, monoloudness reduction of stress, prolongation of intervals, prolongation of phonemes and excessive stress on usually unstressed words and syllables and variable rate.

TABLE - I : Showing the speech characteristics of Hyperkinetic Dysarthrias (Chorea) due to damage of basal ganglia (globus pallidous)

Laryngeal	Velopharyngeal	Oral
Quick uncontrolled movements of intrinsic - extrinsic musculature	Normal	Quick uncontrolled movements
Sudden alterations of pitch and loudness phonatory arrest & strained, harsh voice		Sudden alterations in precision of vowels and consonants.

2. Palatopharyngeolaryngeal myoclonus :

Some patients display a repetitive rhythmic jerking of the parts of the speech musculature. Sometimes these myoclonic jerks involve only the palate, often the palate and pharynx, often the larynx as well. The myoclonic movements occur at a **rate** of one to two beats per minute and are often impossible to detect during contextual speech. During vowel prolongation they are usually audible as regular momentary interruptions in phonation. Myoclonic movements of the diaphragm can be detected in regular interruptions of respiration and outflow of air.

3. Organic voice tremor :

The tremors of laryngeal muscles may occur in association with tremor of other parts of the body known as essential or heredofamilial tremor, or it may occur in isolation. A mild voice tremor may not be noticeable in contextual speech but can be noted if the patient prolongs /a/. The tremulous tone results from rhythmic alterations in pitch and loudness.

TABLE - J : Showing the speech characteristics of Organic voice tremor due to brainstem lesions.

Laryngeal	Velopharyngeal	Oral
Rhythmic contractions of intrinsic-extrinsic musculature, 4-12/sec.	Normal	Approximate Normal
Rhythmic alteration of pitch and loudness (regular voice tremor) adductor phonatory arrests.		

4. Gills de la Tourette's Syndrome :

This Syndrome is characterized by multiple tics, and involuntary vocalization **that** includes caprolalia and echolalia (Shapiro, shapiro & Wayne, 1973)

TABLE - K: Showing the speech characteristics of Gilles de la Tourette's syndrome.

Laryngeal	Velopharyngeal	Oral
Grunt,bark squeal, shriek, scream, cough, throat cleaning gangling,moaning	Snort, sniff	"stuttering" unintelligible sounds echolalia, coprolalia
		Non-speech: whistling, clicking, lipsnacking, spitting

Hyperkinetic dysarthria in dystonia :

In the slowest of the movement disorders, dystonia, muscular contractions develops slowly, resulting in a distorted posture which is prolonged for a time and then subside. When these movements affect laryngeal musculature, phonation may be interrupted or the tone may become strained and strangled and the voice may resemble that of spastic dysphonia. Speech deviations occurring most frequently are distortion of vowels, excessive loudness variations, alterations in loudness from very soft to very loud and voice stoppages. Neurological examination shows suddenness of myoclonic jerks, the abrupt, muscle contraction in chorea which are slower than myoclonic jerks and may be momentarily sustained. There may be general slowing of voluntary movements and variable muscle tone in the quick hyperkinesias.

TABLE - L : Showing the speech characteristics of Dystonia due to damage to basal ganglia.

Laryngeal	Velopharyngeal	Oral
Slow uncontrolled movements of intrinsic-extrinsic musculature	Normal	Slow uncontrolled movements
Slow alterations of pitch and loudness. Phonatory arrest harshness		Slow alterations in consonant and vowel precision.

Mixed dysarthrias :

In those cases where there is impairment of more than one level of motor function the dysarthrias appear in a combined form. The resulting dysarthrias will display the features distinctive of each neurologic type, although the occurrence of certain deviations may observe the distinctive aspect of the "pure" dysarthrias. They are grouped under various categories such as:

a) spastic-flaccid	ex. Amyotreophic lateral sclerosis.
b) spastic-axtaxic-hypokinetic	ex. wilson's disease.
c) variable (spastic-ataxic-flaccid)	ex. Multiple sclerosis.

HI The Degenerative Dysarthrias :

On the basis of natural course,dysarthria may follow a number of patter-ns, including developmental (as in cerebral palsy in children), recovering (post onset traumatic head injury and stroke), stable (as in cerebral palsy in adults), (as in amyotrophic lateral sclerosis) or exacerbating- remitting (as in some cases of multiple sclerosis), (Yorkston et al, 1988)

The medical, speech characteristics of a diverse group of dysarthrias with a degenerative natural course deserves attention. Onset of these disorders occurs after childhood, and in most cases, the disorders are insidious, with signs and symptoms appearing gradually. Many progressive neuromotor disorders result in dysarthria, however only selected disorders that occur frequently in a clinical case load of speech/ language palhologists with those disorders that are uncommon but whose speech characteristics have been studied carefully and are reported in the literature have been reviewed. The review begins with of those disorders referred to by neurologists as moment disorders. Movement disorders can be divided into two

syndrome of Parkinsonism and those conditions characterized by abnormal involuntary movements known as the dyskinesis. The five dyskinesias are tremor, chorea, myoclonus, tics and dystonia.

Each of these categories will be reviewed briefly. Included among the dyskinesias reviewed here are dystonia, Huntington's disease, and Wilson's disease. Amyotrophic lateral sclerosis (a motoneuron disease), friedreich's ataxia (a spinocerebellar disorder), multiple sclerosis (a disease of the white matter), and myasthenia gravis (an autoimmune disorder characterized by abnormal fatiguability and weakness of skeletal muscles) are reviewed.

PARKINSON'S DISEASE

Parkinsonism is a general sydrome that encompasses the symptoms of "rest" tremor, rigidity, paucity of movement, and impaired postural reflexes, and is due to the loss of dopaminergic neurons in the basal ganglia (especially the substantia nigra) and brainstem. It can be divided into three subgroups depending upon its etiology and associated signs and symptoms:

1. Idiopathic or primary Parkinson's disease (also know as paralysis agitans).
2. Secondary Parkinsonism, which includes a number of disorders with extrapyramidal features and that have an identifiable causal agent, some of which would include toxin (l-methyl-4-phenyl-l, 23,3,6-tetrahydropyridine or MPTP), infections (von Economo's encephalitis), drugs (neuroleptics), repeated trauma or multiple strokes.
3. Heterogeneous system such as progressive supranuclear palsy, striatonigral, shy-Drager syndrome, or olivopontocerebellar (Marttila,1983)

In order to diagnose Parkinson's disease, at least two of the classic signs mentioned must be present. However, since most of the secondary and types of parkinsonisms also have these symptoms (particularly rigidity and bradykinesia), a

search must be made for signs and symptoms that are not typically seen. Some of these signs would be pyramidal tract signs (exaggerated reflexes, extensor plantar responses), intention tremor, ataxia or other evidences of cerebellar dysfunction, or profound early dementia (Marttila, 1983). The diagnosis of Parkinson's disease is made on clinical grounds. In the patient who does not show the classic signs of tremor, rigidity, and akinesia, computerized tomography (CT scans) may be helpful in the differential diagnosis.

Population Characteristics

The average annual incidence of parkinsonism (excluding drug-induced cases in U.S.) is 18.2 per 100,000. The prevalence in white populations is estimated to be between 66 and 187 per 100,000. There is no significant difference between males and females. The incidence increases sharply above the age of 64 and the peak of incidence is between 75 and 84 years of age. There has been a trend towards increased age at the time of diagnosis; in 1967, the mean age at onset was 55.3 years (Hoehn,& yahr, 1967; Rajput,offord, Beard, & Kurland, 1984).

Causes

There are three areas presently being investigated as possible etiologies: genetic, age-related, and environmental. There have been two familial subgroups identified with variants of Parkinsonism. The first, with autosomal dominant transmission, has tremor as the predominant sign with a strong family history of benign tremor. The second autosomal recessive form shows symptoms of akinesia and rigidity. However, twin studies have not shown any genetic transmission of typical idiopathic Parkinson's disease. It has been argued by some that Parkinson's disease is an accelerated form of normal aging with a loss of substantia nigra neurons. Again,

however, twin studies do not support this. Another argument against the aging theory is that the "parkinsonian" traits of normal elderly people do not respond to treatment with levodopa.

The third possibility, that of an environmental toxin, has received some support by the development of a severe form of Parkinsonism in a number of drug abusers. A derivative of meperidine,MPTP, has been shown to cause a severe loss of dopaminergic substantia nigra neurons. Although the pathology found after the use of this drug is not identical to that of Parkinson's disease (which includes other regions of the brain), it is the best model available. Other studies have suggested common exposures among patients from similar geographic areas that may trigger eventual cell death (Lang & Blair, 1984).

Course

Parkinson's disease typically has an insidious onset; in retrospect, patients recall increasing difficulties with "stiffness" and "muscle aches" that they had attributed to the normal course of aging. The problem that initiates the first visit to a physician is most commonly tremor. The tremor of Parkinson's disease is of the distal extremities and occur at rest (the pill-rolling phenomenon). Patients who initially show symptoms of tremor apparently have a slower progression of the disease, at least in the first 10 years (Hoehn & Yahr, 1967). In the early part of the course of the disease, the patient might notice increasing difficulty in repetitive or alternating movements such as walking. When a joint is passively moved through its range, a "catch" can be felt; this phenomenon is known as cogwheeling. This rigidity affects all striated muscles, causing difficulties in respiration, facial expression, swallowing,

mastication, and speech. Progression of rigidity can lead to flexion contractures of the fingers, elbows, cervical spine, hips, and knees with ensuing loss of mobility.

Bradykinesia (or akinesia, in its most extreme form) is slowness or decrease in spontaneous movements. Often the earliest manifestation of this is a decrease in the frequency of eye blinking (normal range 14 to 17 per minute). Paucity of facial movement leads to a mask-like appearance. With progression of the disease, the patient may not be able to perform simple volitional acts (called freezing) such as initiating ambulation or arising from a chair; these episodes can often be overcome by diverting the patients attention from the desired act or by an emotional response. Loss of postural reflexes, shuffling gait retropulsion (the tendency to Ml backwards), and festination (progressive rapidity of forward movement with a loss of control) all severely affect safe ambulation.

Controversy exists as to whether dementia is a feature of Parkinson's disease. In some patients, specific memory deficits are present on testing, and patients may complain of slowness in problem solving. However, objective testing of these patients is difficult because of the extreme slowness of motor responses, poor hand writing, and dysarthria. Agreement certainly exists that if dementia is prominent and occurs before major motoric disability, a diagnosis other than Parkinson's disease should be considered, for example, Alzheimer's disease or progressive supranuclear palsy (Morris, 1982).

Prior to treatment with levodopa, over one-quarter of patients were dead or severely disabled with in five years of their diagnosis; eights- percent were in this category after 10 to 14 years of observation, nearly three times that of the normal population (Hoehn & Yahr, 1967). It was initially though that treatment with

levodopa decreased the mortality to only 1.3 to 1.9 times higher than normal; recent studies question whether these apparent improvements were due to methodological inadequacies of these studies (Marttila, 1983; Rajput et al., 1984).

Speech characteristics

Extensive research has been focused on the description of the speech patterns of individuals with Parkinson's disease. The prevalence of speech disorder in the Parkinsonian population is high. Logemann, Fisher, Boshes, and Blonsky (1978) studied 200 Parkinsonian speakers and reported that 89 percent of their sample exhibited laryngeally related problems and 45 percent demonstrated articulatory problems. Of the 65 Parkinsonian patients studied by Buck and Cooper (1956), 37 percent had normal speech or were mildly involved, 22 percent had a moderate degree of speech involvement, and 29 percent had severely impaired speech.

Perhaps the most complete overview of Parkinsonian speech characteristics comes from the work of Darley, Aronson, and Brown (1969a, 1969b, 1975). They found the following as the speech characteristics of this group of patients: reduced variability in pitch and loudness, reduce loudness level overall, and decrease use of all vocal parameters for achieving stress and emphasis. Markedly imprecise articulation is generated at variable rates in short bursts of speech punctuated by illogical pauses and often by inappropriate silences. Voice quality is some times harsh, some times breathy.

Speech components

RESPIRATORY FUNCTION. With few exceptions, researchers have supported the conclusion that in Parkinsonian speakers, respiratory function is reduced as compared to normal speakers. De la Torre, Mier, and Boshes (1960)

observed reduced vital capacities in 17 Parkinsonian males that they studied. Two-thirds of the group demonstrated vital capacities that fell below 40 percent of predicted vital capacity for their age and sex. Irregular breathing patterns observed in the group were attributed to disruption in the normal agonist-antagonist synergy of the respiratory muscles. Ewanowski (1964) studied 12 Parkinsonian subjects and matched number of normal subjects and found no differences between quiet respiratory patterns of the two groups. Several investigators instructed their subjects to sustain phonation as a measure of respiratory support. Canter (1965a) and Boshes (1966) reported that their Parkinsonian subjects had reduced ability to sustain phonation. However, Ewanowski (1964) and Kreul (1972) reported similar ability to sustain phonation between the Parkinsonian and normal subjects. The differences in these results are probably related to the severity of the Parkinsonism in the various groups of subjects.

The pattern of respiratory support for speech has also been investigated. Kim (1968) employed an ink-recording respirometer with a face-mask. He reported that all of his patients but one showed a varied degree of ability to alter automatic respiratory rhythms to speak or voluntarily hold their breath. Both Kim and Hunker, Bless, and Weismer (1981) reported that dysarthric speakers with Parkinson's disease may have "inflexible" respiratory patterns for speech. In part, this inflexibility may be reflected in reduction of lung volume excursions or restricted use of chest wall part combinations to achieve lung volume displacements.

LARYNGEAL FUNCTION. Impaired performance of laryngeal subsystem for speech has been measured in numerous studies that consistently reported an important reduction in laryngeal function in patients with Parkinson's disease. Several

studies have shown that Parkinsonian subjects produce average fundamental frequency levels that are higher than normal speakers (Canter, 1965a,b, Kammermeier, 1969; Ludlow & Bassich, 1983). A reduction in pitch variability was reported by Grewel (1957). Ludlow and Bassich (1983) concluded that pitch variability was restricted, in that the downward pitch inflection at the end of sentences or parts of sentences is lacking. Although persons who routinely listen to Parkinsonian speakers often complain that they can not speak loud enough, the research reports are contradictory. Canter (1963) analyzed the speech of 17 speakers with Parkinson's disease and found that they did not differ from normal speakers in mean peak sound-pressure levels. Also, the two groups did not differ on the range of peak sound-pressure levels. Ludlow and Bassich (1983) reported that mean intensity in sentences was significantly reduced for Parkinsonian speakers. In addition, the literature would suggest that reduced loudness variability is common. Darley and colleagues (1975) reported that voice was frequently deviant in Parkinsonian speakers. Perhaps one contribution to the vocal pattern is the tendency of Parkinsonian speakers to be unable voluntarily to produce speech at very low intensity levels (Canter, 1965a). Ludlow and Bassich (1983) reported that the maximum range of intensity for Parkinsonian speakers on a loudness imitation task was reduced. As a group, Parkinsonian speakers also show extensive disorders of vocal quality. Darley and colleagues (1975) reported harsh voice quality and breathy voice (continuous) ranked sixth and seventh, respectively, as deviant speech dimensions for this population.

VELOPHARYNGEAL FUNCTION. A review of research reveals that although hypemasality is sometimes observed in Parkinsonian speakers, nasal emission is not. Mueller (1971) reported no measured nasal emission during speech in

any of the 10 Parkinsonian speakers he studied. Darley and colleagues (1975) reported that only 8 of their 32 subjects demonstrated hypernasality to a minor degree (mean severity value of 1.16 on a 7 point scale). No subject was judged to display nasal emission during speech.

ARTICULATION. Canter (1969b) reported that the primary articulatory characteristics of Parkinsonism result from inadequate articulatory valving during production of plosives and breakdowns in the coordination of laryngeal and oral activity. Longemann and Fisher (1981) reported that manner errors were most characteristic of Parkinsonism dysarthria. Spirantization of stops (the tendency of stops to be fricated) is characteristic of these patterns. Caligiuri (1985), Hirose, Kiritani, and Sawashima (1982), Hirose, Kiritani, Ushijima, Yoshioka, and Sawashima (1981), Hunker, Abbs, and Barlow (1982), and Leanderson, Persson, and Ohman (1970) suggest that persons with Parkinson's disease showed reduced articulatory displacements as compared to normal speakers and incoordination of agonist and antagonist muscles. Caligiuri (1985) noted that at normal speaking rates, rigid Parkinsonian speakers, exhibited significantly lower lip displacement amplitudes than nonrigid Parkinsonian speakers. Weismer (1984) reported that Parkinsonia subjects had longer vowel durations than both the geriatric and young adult subjects. The shortest closure durations were produced by Parkinsonian subjects for initial voiceless stops, whereas the longest durations were produced by geriatric subjects. For fricatives, Parkinsonian speakers had shorted durations than both the geriatric and young adult subjects. In summarizing his research, Weismer wrotethat " the data presented here suggests that Parkinsonian subjects have segmental and phrase-level durations which are slightly shorter than the corresponding durations of the appropriate control group, and that frequent spirantization is typical feature of

Parkinsonian dysarthria. Several characteristics of Parkinsonian dysarthria, such as continuation of vocal fold vibration into voiceless stop closures **and** somewhat inflated inter-and intrasubject variablities seem to be characteristic of geriatric speech and not unique to **the** neurogenic disorder. The only characteristic of parkinsonian dysarthria that might be considered an exaggerated aging effect is **the** shortened voiceless interval"

Overall speech

There is considerable variations in the speaking rate among Parkinsonian speakers. Canter (1969b) reported median speaking rate during oral reading for his subjects at 172.6 as compared to 177.6 words per minute (wpm) for normal subjects. However, the performance range for his subjects was 69.6 to 249.6 wpms. Kreul (1972) reported that his Parkinsoian subjects read aloud at a mean rate of 142.5 wpm, and Boshes'(1966) subjects read with a range from 50 to 70 wpm. Kammermeier (1969) reported a mean oral reading rate of 127 wpm (range 110 to 152). Netsell, Daniel, Celesia (1975) studied the "rushes of speech" that were demonstrated by one Parkinsonian speaker. Eleven of their 22 subjects demonstrated short rushes of speech. They observed that the reciprocal of these periods in the later syllables corresponds to rates in excess of 13 per second. Considering that the upper limit for voluntary control of such repetition rates is fewer than 10 per second, the 13 per second rate is interpreted as evidence that the subject is in some neuromuscular mode over which he (the speaker) has no immediate control. During the rushes of speech, the researchers report that lip contacts were not made during the production of /p/, thus supporting the conclusion that the speech articulator failed to reach the necessary position for production of a particular speech sound before beginning the movement to the following sound (articulatory undershoot).

Several clinical researchers not only suggest that there is variability among Parkinsonian speakers, but also that variability may be seen from task to task within a speaker. Weismer (1984) reports on intelligibility differences from situation to situation by stating that " although the data is derived from connected utterances, the clinical experience of large differences in intelligibility of Parkinsonian speech in the clinical setting versus "spontaneous" situations is confirmed in our experiment. Most of the Parkinsonian subjects in the current experiment were quite intelligible when producing the experimental sentences, but much less intelligible when engaged in spontaneous speech".

PROGRESSIVE SUPRANUCLEAR PALSY

Medical Aspects

Progressive supranuclear palsy is an extrapyramidal syndrome first described by Steele, Richardson, and Olszewki (1964). Symptoms include ophthalmoplegia (Mainly of vertical gaze), dystonic rigidity of the neck, pseudobulbar palsy, mild dementia, and spastic dysarthria. The following symptoms have been cited in subsequent reports: akinesia, lack of facial expression, poor postural reflexes, and hypokinetic dysarthria (Behrman, Carroll, Janota, & Matthews, 1969; Blumenthal & Miller, 1969; Hanson & Metter. 1980; Klawans & Ringel. 1971). Neuropathologic alterations are found in the following structures (Steele et al., 1964): subthalamic nucleus, red nucleus, substantia nigra, superior colliculus, periaqueductal grey matter, globus pallidus, and dentate nucleus of the cerebellum. The disease has an onset in middle and later part of life. Life expectancy after diagnosis averages five to seven years. This relatively uncommon disease is more frequent in males than females (Steele, 1972).

Diagnosis

Progressive supranuclear palsy can be distinguished from Parkinson's disease in several ways (Cummings & Benson, 1983). In progressive supranuclear palsy, the posture is extended rather than bowed as in Parkinson's disease. In progressive supranuclear palsy, rigidity primarily affects the axial structures, in Parkinson's disease, the limbs are primarily affected. Also, tremor is unusual in progressive supranuclear palsy.

Speech characteristics

Dysarthria is usually severe in individuals with progressive supranuclear palsy. Individuals may exhibit anarthria or mutism in the later stages (Steele, 1972). To date, few studies of speech characteristics of a group of individuals with progressive supranuclear palsy have been reported. However, clinical descriptions suggest the occurrence of both spastic and hypokinetic dysarthria and language deficits. Lebrun, Devreax, and Roussea. (1986) report that speech and language symptoms vary considerably from patient to patient.

DYSTONIA

Medical aspects

The term " *dystonia* " was coined by Oppenheim, who described patients with sustained posturing and also tonic and clonic spasms of muscles in different parts of the body. These spasms are typically activated with voluntary motor activity. Although dystonia is attributed to disturbances of the extrapyramidal system, the underlying neuropathology and mechanisms have not yet been described (Marsden & Harrison, 1974). Dystonias may be symptomatic of a neurological process such as cereberal anoxia, birth trauma, wilson's disease, encephalitis, and especially drugs such as phenothiazines and butyrophenones. However, they are often idiopathic or

inherited conditions. The severity of dystonia may range from catastrophic to a mild nuisance. There is neither weakness ncr wasting of muscle. Sensor.-, Sphincter, and reflex alterations do not occur. In EMG studies of dystonia, several research groups (Herz, 1944a,b; Yanagisawa & Goto. 1971) have observed a tonic, nonreciprocal pattern of activity in agonist and antagonist muscles during any voluntary or postural contraction.

The primary dystonias are slowly progressive disorders that can plateau anywhere in the course of the illness. They begin insidiously and almost always with action dystonia (Fahn, 1984). In cc~rast, most secondary dysxcnias begin with dystonia at rest and even with sustained postures. Some secondary dystonias have an obvious sudden beginning, such as on recovery from an acute encephalopathic event. Secondary dystonias may also be associated with metabolic disease (e.g., Wilson's disease, Hallervorden-Spatz disease). and tend to have a more racidly progressive course than do the primary dystonias. Some secondary dystcnias are due to environmental causes, such as head trauma, encephalitis, and exposure to toxins and tend to have a course that stabilizes and does not progress. Drugs that block the dopamine D2 receptor (antipsychcdcs and the substituted benzamides, e.g., metoclopramide) can induce two types of dystonia; acute dystonic reaction and delayed persistent dystonia (tardive dystonia). Acute dystonic reaction can be reversed readily with administration *of* anticholinergics or diazepam. Tardive dystonia is not only persistent, but is also frequently unresponsive to therapy .

Diagnosis

Marsden, Harrison, and Bundy (1976) report criteria for the diagnosis of idiopathic torsion dystonia as follows :

1. The presence of dystonic movements and postures (but arbitrarily excluding isolated spasmodic torticollis).
2. Normal prenatal history and early development
3. No history of any known precipitating illness or exposure to drugs known to provoke torsion dystonia prior to the onset of the disease
4. No evidence of intellectual, sensory deficit, pyramidal or cerebellar involvement on clinical examination
5. Failure of laboratory inveszgations, including copper studies, to demonstrate any cause for the disease

Population

In 72 individuals who were diagnosed as dystonic based on the preceding criteria (Marsden et al.,1976s the age of onset ranged from 1 to 59 years. Approximately 70 percent experienced onset in childhood and 30 percent experienced adult onset. The rado of females to males was 1.2:1. The duration of the disease was 16 years with a range of 1 to 47 years.

Signs, symptoms, and natural course

The distribution of signs and symptoms in dystonia is usually categorized as generalized (affecting many areas of the body), segmental (limited involvement,e.g., arm and neck, both arms, or neck and trunk but sparing the legs), and focal (signs limited to a single arm or a hand). Marsden and colleagues (1976), found generalized dystonia developed in 85 percent of those with onset at or before the age of 10 years, in 60 percent of those with onset between age 11 and 20 years, and only four percent of those with onset after age of 20 years. Cooper (1969) describes the initial patterns for three groups of patients:

1. Childhood form. Onset at four to six years of age; initial symptom nearly always is flexion inversion of the foot with progression to generalized dystonia within four to six years of onset.

2. Adolescent form. Onset at 8 to 13 years of age; initial symptom is usually in the foot, but sometimes in the arm; the rate of progression is slower than the childhood form.

3. Adult form. Initial symptoms usually start in the arm, this form usually develops into axial (trunk) dystonia with relative sparing of the extremities.

In the childhood form there appears to be two patterns of inheritance (Eldridge,1970). The autosomal recessive form begins in early childhood, is progressive over a few years, and is restricted to Jewish patients. The dominant form begins later, usually late in childhood or adolescence, progresses more slowly than the autosomal recessive form, and is not limited to an ethnic group. According to Marsden and colleagues (1976), the "typical" picture of segmental dystonia in adults was onset with dystonic posture and spasms affecting one arm, with subsequent spread to the other arm and neck or the neck alone. Focal dystonia usually involves symptoms in one area of the body, such as the arm (writter's camp) or the face (cranial dystonia). The syndrome of cranial dystonia, also known as blepharospasm-oromandibular dystonia, Breughel's syndrome, or Meige's syndrome, was described in 1910 by Henry Meige. The primary features of this syndrome are a blepharospasm, a prolonged tonic contraction of the orbicularis occuli muscles, and both fluctuating and sustained contractions of facial, lingual, and mandibular muscle groups (Golper, Nutt, Rau, and Coleman, 1983). Dystonic spasms disappear during sleep and are triggered by initiation of speech or presentation of food or drink to the mouth. The functional disabilities of the 72 patients in the study by Marsden and colleagues (1976) were assigned according to the criteria of Bundy, Harrison, and Marsden (1975):

Mild disability

Gradel: leading a normal life; no symptoms

Grade 2: mild disability; continuing full time work

Modarate Disability

Grade 3: works with difficulty

Severe Disability

Grade 4: not at work ; independent at home

Grade 5: Wholly dependent on others.

HUNTTNGTON'S DISEASE

Medical Aspects

Huntington's disease is a degenarative disorder of the nervous system characterized by a triad of clinical features including chorea, dementia, and a history of familial occurrence. Inheritence is via an autosomal dominant trait with complete penetrance. Thus, half the offspring of an afflicted individual will develop the disease. Males and females are equally likely to have the disease. Average age of onset of symptoms is 35 to 40 years and average course from onset to death is 14 years. Prevalence in the United States is 40 to 70 per one million population (Hogg,Massey, & Schoenberg, 1979).

Natural course

Personality changes usually occur before the onset of chorea. These alterations include: irritability, untidiness, and loss of interest (Cummings & Benson,1983). Transient facial grimacing, head nodding, and flexion extension movements of the fingers may be the first manifestation of the choreic movements. In advanced stages of disease, the speed of movement reduced and patients acquire **an** athetotic or dystonic character.

Diagnosis

The diagnosis of Huntington's disease is made on the basis of clinical findings of choreic form movement disorder and dementia occurring on a familial basis rather than on laboratory findings. Although there are no pathognomonic laboratory findings, diagnosis is supported by demonstrating diminished caudate volume on CT scans. Huntington's disease may be distinguished from other types of chorea, including sydenham's chorea, a self limited disease of children usually associated with episodes of inflammatory or infectious processes, and tardive dyskinesis, a movement disorder developed in individuals who are chronically exposed to neuroleptic drugs. The predominate movements in tardive dyskinesia usually involve the mouth and tongue, but hands, legs, trunk, and respiratory muscles may also develop choreoathetosis (Crane, 1968; Maxwell, Massengill, & Nashold,1970;Portnoy,1979). Because of the hereditary nature of Huntington's disease and the fact that age of onset occurs after child-bearing age, attention has focused on the study of "at-risk" individuals in order to identify the incipient signs of the disorder. At present, no test definitively discriminates non affected at-risk persons from presymptomatic carrier-victims of the disease(Cummings & Benson, 1983). Medical applications under investigation include use of levodopa, which increases chorea in symptomatic patients. This test, however, has not been completely validated with at-risk individuals (Klawans,Goetz,& Perlik,1980).

Speech characteristics

Speech symptoms may range from little or no dysarthria in case where choreic movements are restricted to the limbs and body, to speech that is so severely impaired

that it is unintelligible. Speech may be disrupted by sudden movements of the respiratory muscles, tongue, and face (Wilson & Bruce, 1955). Darley and colleagues !975) summarize the perceptual characteristics of 30 individuals with hyperkinetic dysarthria of chorea as follows: a highly variable pattern of interference with articulation, episodes of hypernasality, harshness, and breathiness, and unplanned variations in loudness. In the speaker's apparent attempt to avoid the inevitable interruptions and to compensate for them, the rate of speech is variably altered, phonemes and intervals between words are prolonged, stress is equalized and inappropriate silences appear.

Speech symptoms are so closely related to the underlying movement disorder that marked improvement in speech symptoms is dependent on modification of the severity of the movement disorders. Raming (1986) reported the results of a detailed acoustic analysis of phonation in individuals with Huntington's disease. She found abnormalities including low frequency segments (abrupt drops in fundamental frequency of approximately one octave), vocal arrests, and reduced maximal vowel duration. Changes due to behavioral speech intervention have not been reported, although speech may improve coincident with medication management of the choreic movements (Beukelman, 1983)

WILSON'S DISEASE

Medical Aspects

Wilson's disease is a rare, hereditary disorder caused by inadequate processing of the dietary intake of copper. Pathological changes occur in the liver, the brain, and the cornea of the eye as a result of excessive accumulation of copper in the tissue over a period of years. Neurological abnormalities include incoordination, tremor, dysarthria, drooling, dysphagia, and mask like face. Wilson's disease may present as a

neurologic syndrome, a psychiatric disturbance, or a hepatic disorder (Cartwright, 1978).

Natural course

Neurological begins to appear in adolescence or early adulthood. Darley and colleagues (1975) describe the natural progression of the disorder as follows: at later stages they usually exhibit severe ataxia with a bizarre intention tremor involving both upper extremities, marked rigidity of trunk and extremities, or a combination of the two. They also demonstrate marked dysarthria, dysphagia, drooling and masked expression. If undiagnosed and untreated, the disease is fatal.

Speech characteristics

Dysarthria was recognized as a prominent neurological feature when the disorder was first described by Wilson in 1912. Berry,Darely, Aronson, and Goldstein (1974) reported the results of a study in which they perceptually analyzed the speech of 20 individuals with Wilson's disease. The data suggested the presence of a mixed dysarthria with prominent ataxic, spastic, and hypokinetic features. Further speech samples were obtained at two points of medical treatment for 10 of the 20 individuals. Their findings indicated that a regimen of D-pennicillamine and a low copper diet produced a significant remission of dysarthria.

AMYOTROPHIC LATERAL SCLEROSIS (ALS)

Medical Aspects

Amyotrophic lateral sclerosis (ALS) is **a** progressive disease involving the motoneurons of both the brain and spinal cord in adults. Some motoneuron diseases, such as spinal muscular atrophy, involve primarily the lower motoneurons; others, such as primary lateral sclerosis, involves the upper motoneurons. Classical ALS involves both types of motoneurons. Upper motoneuron signs include muscle weakness, increased muscle tone (spasticity), hyper-reflexia, extensor plantar reflexes, and pseudobulbar palsy (manifested by hypertonic bulbar muscles, increased perioral reflexes, and exaggerated emotional responses) . Lower motoneuron signs include muscle weakness, muscular atrophy, and diminished or absence of deep tendon reflexes.

Population characteristics

The average world wide incidence of ALS ranges between 0.4 and 1.8 per 100,000 population, and the prevalence rates range between 4 and 6 per 100,000 population (Tandan & Bradley, 1985a). Nintyfive percent of all cases are sporadic. However, there are two familial inherited types of ALS. The familial adult type is based on an autosomal dominant inheritence, whereas with juvenile onset, the inheritence mechanism may be autosomal dominant or recessive (Tandan & Bradley, 1985b).For sporadic ALS in the United States and Europe, the mean age at onset is 56 years with a male to female ratio of 2:1 (Emery & Holloway, 1982). Between 14 and 39 percent of individuals with ALS survive for five years, about 10 percent live up to 10 years, and a few live for 20 years. There appears to be several factors that determine the course and duration of the disease for the individual patient. The prognosis becomes less positive progressively with each of the following symptoms; muscle atrophy, upper motoneuron involvement, respiratory insufficiency, and predominant bulbar (Brain stem) symptoms (Tandan & Bradley, 1985a).

Etiology

A long list of possible causative mechanisms have been investigated without any being overwhelmingly convincing (Tandan & Bradley,1985b; Amico &Antil, 1981). The possibility of genetic factors at least contributing to the development of the full-blown disease seems tempting in view of the fact that familial forms do exist. Other general factors that have been mentioned are the aging phenomenon and association of ALS with neoplasia. Some degree of loss of motoneuron cells does occur in normal aging; If ALS is a form of "premature aging" it is not clear as to what the mechanism would be. The incidence of malignancy coexisting with ALS is 0.7 to 10 percent above the normal population. However, no hormonal or other factor has been identified to explain a casual connection. A theory of viral causation is attractive because of the existence of such viruses as the poliomyelitis virus, which selectively affects anterior horn cells. A slow, virus-type infection is most likely although no viral particles have been identified and tissue transplantation in animals has not resulted in ALS. It is even more likely that the answer may be a combination of viral infection and immune factors; there has been evidence for increased cell-mediated immunity to poliovirus antigens and for the presence of circulating and renal immune complexes. Other suggested mechanisms have included exposure to metals and minerals, endogenous toxins, abnormal nucleic acids or membrane properties, or a defect in neurotransmitters. Patients have mentioned an increased history of trauma or surgery in ALS as opposed to controls: however, no explanation has been forwarded for this observation.

Signs, Symptoms, and Natural course

The most common presenting symptom is a focal or segmental weakness(63 percent); the most common form is paraparesis (20 percent), but the weakness may be more focal at outset. About one-third of all patients complain of hand clumsiness, another one-third of leg weakness, which may be manifested as tripping over carpets or on steps. Twenty-two percent show bulbar symptoms (dysarthria in 45 percent, dysphasia in 42 percent, dysphonia in 12 percent, and dyspnea in 6 percent). It is not unusual for patients to complain of muscle pain and cramping or paresthetic-like pains even though ALS is a disease of the motoneuron cells (Adam, 1986; Gubbay. Kahana, Zilber, & cooper, 1985)

ALS is a progressive disease with a median survival of three years from the time of onset of symptoms (Tandan & Bradly, 1985a). Those showing bulbar symptoms tend to have a more rapid course (median of 2.2 years). There is a subgroup of patients (perhaps up to 25 percent) who have a prolonged course; some have been reported to survive more than 20 years. Increasing weakness of the extremities, inability to swallow without aspiration, and decreased ability to speak ensure. Death is usually on the basis of respiratory failure or infection. Extraocuiar muscle movements are usually spread as in sphincter control. There have been some reports of dementia (in up to 5 percent) but there is some doubt that this is directly related to the presence of ALS.

Neuropathology

The motoneurons of the brain stem and spinal cord show simple atrophy, shrinkage, and cell loss (Hirano & Iwata, 1979). In individuals with extensive upper motoneuron signs there is a depletion of Betz cells and large pyramidal neurons from the fifth layer of the motor cortex and widespread of the corticospinal tracts (Hughes.

1982). In sporadic ALS the posterior columns of the spinal cord are uninvolved (Lawyer & Netsky, 1953).In the familial ALS, there is evidence of involvement of **the** posterior columns, Clarke's nucleus, and spinocerebellar tracts in at least 50 percent of the cases (Emery & Holloway, 1982). Studies have shown a reduction in the number of large motoneurons in the cervical and lumbar spinal cord (Tohgi, Tsukagnoshi, & toyokura, 1977). In the peripheral nervous system, several studies have reported a marked reduction in the number of large myelinated fibers in the ventral roots (Hanyu,Oguchi, Yanagisawa, & Tsukagnoshi, 1981; Sobue, Matsouka, & Maukai, 1981). Further, data indicates that ALS is predominantly a neuropathy, as evidenced by considerable loss of large myelinated fibers from all levels of the nerve.

Speech Characteristics

The speech characteristics associated with ALS vary, depending on the course of the disease. For individuals with initial symptoms appearing in areas served by the bulbar (lower cranial) nerves, motor speech and swallowing disorders occur quite precipitously. However, for individuals with initial symptoms in areas served by the spinal nerves, speech symptoms may occur late in the course of the disease. In either case, most persons with ALS are anarthric during the later stages of the disease and require an alternative communication system. Saunders, Walsh, and Smith (1981) reported that 75 percent of their 100 ALS patients were unable to speak at the time of their death.

Type of Dysarthria

The characteristic speech of the individuals with ALS has been classified as a mixed dysarthria by Darley et. al., (1975). Symptoms associated with both spastic and flaccid dysarthria are often present; however, as the disease progresses the contributions of each type of dysarthria may change. As the individual becomes excessively weak, the symptoms associated with the flaccid dysarthria usually become more apparent. As the disease progresses, the spastic symptoms often cannot be expressed by the weakened neuromuscular system.

Perceptual characteristics

The most extensive investigation of mixed dysarthria associated with ALS was completed by Darley et. al., (1975). According to these researchers, the most deviant speech disorders in order of rated severity were imprecise consonants, hypernasality, harsh voice quality, slow rate, monopitch, short phrases, distorted vowels, low pitch, excess and equal stress,prolonged intervals of reduced stress, prolonged phonemes, strained- strangled quality, breathiness, audible inspiration, inapproprite silences, and nasal emission.

Speech components

A review of the previous list of speech characteristics reveals that, as in most dysarthrias, individuals with ALS demonstrated impairment in all components of the speech mechanism. On a case-by case basis, the distribution of impairment varies from individual to individual.

RESPIRATORY FUNCTION. Putnam, Hixon, Stern (1982) studied two individuals with ALS who demonstrated abnormality for speech breathing. These individuals used limited lung volume ranges for speech that may have resulted from

reduction in vital capacity. For some individuals with ALS, respiratory impairment becomes so severe that they choose to be ventilated with a respirator.

LARYNGEAL FUNCTION. The phonatory subsystem may reveal a mixed dysarthria. Aronson(1980) writes: "If spasticity is predominant, hyperadduction of the true and false vocal folds, technically a pseudobullbar dysphonia, will require that the exhaled air be forced through a constricted glottis. Such elevated laryngeal resistance to the exhaled air stream, coupled with a reduced exhalatory force, decreases the volume of voice in addition to producing a strained hoarseness or harshness.... A greater flaccid (lower motor neuron) component produced adductor vocal-fold weakness... extreme breathiness and reduced loudness".

VELOPHARYNGEAL FUNCTION. Velopharyngeal incompetance with resulting hypernasality and nasal emission is commonly associated with ALS. Often the nasal emission is not easily perceived, because of the lack of respiratory support in these individuals. Nevertheless, inadequate closure of the velopharyngeal port decreases the ALS speaker's ability to impound air pressure in the oral cavity for consonant sound production.

ORAL ARTICULATION. The tongue and the lips frequently exhibit excessive weakness. Carrow, Rivera, and Mauldin, et al., (1974) found tongue atrophy to be a prevalent neurological sign in person with severely reduced speech intelligibility. Dworkin, Aronson, and Mulder (1980) measured tongue protrusion strength on a non speech task. Their data showed that healthy males could generate 2,086 grams (range= 1300 to 3,356 grams), whereas men with ALS averaged 1,129 grams (range =211 to 1,754 grams).

FRIEDREICH'S ATAXIA

Medical Aspects

Friedreich's ataxia is one of a heterogeneous group of spinocerebellar disorders. Cummings and Benson (1983) classify the spinocerebellar into three groups. Those affecting predominantly the spinal cord include Friedreich's ataxia and its variants and heriditary spastic ataxia. Those affecting predominently the cerebellum include cerebellar cortical and cerebellar nuclear (dentatorubral atrophy). Those affecting predominantly the brain stem and cerebellum include olivopontocerebellar atrophy.

The most common type of Friedreich's ataxia is the result of an autosomal recessive trait. Males and females are affected in equal proportion with age of onset between 11 and 12 years. Most patients die within 20 years of the onset of symptoms. The disorder is usually first observed as it affects the lower extremities with gait disturbance. Dysmetria of the upper extremities and dysarthria occur later. A number of abnormalities frequently occur as part of this syndrome, including skeletal deformities (pes cavus, hammer toes, and kyphoscoliosis), loss of viberation and position sense, absence of muscle stretch reflexes in the lower extremities, nystagmus, limb weakness, optic atrophy, pigmentary retinal , vestibular involvement, and myocardial (Brain & Walton, 1969; Menkes, 1974). A constant feature of the neuropathology of Friedreich's ataxia is the large myelinated sensory fibers, posterior roots, and dorsal root ganglion cells.

Speech characteristics

Dysarthria has long been recognized as a symptom of Friedreich's ataxia. In 1877,Charcot described it as a disease in which the tongue become too "thick". In 1964, Heck reported that speech defect is a common finding in Friedreich's ataxia,

with an estimated incidence of 63 percent to 93 percent. Brain & Walton (1969) state that the speech of persons with Friedreich's ataxia is invariably dysarthric in the later stages of the disease.

Perceptual characteristics

Numerous attempts have been made to describe precisely the charecteristics of the dysarthria associated with Friedreich's ataxia. In 1937, Zentay classified the dysarthric speech resulting from cerebellar lesion as ataxic speech, adiadokokinesis, explosive-hesitant speech, and scanning speech. By 1958, Alajouanine, Scherer, Sabouraund, and Gremy had studied ataxic speech using oscillographic tracings. They reported two patterns: the first showed amplitude variations from one word to another, which they labeled as explosive or scanned speech quality; the second was described as variations disturbing the continuity of phonemes.

In 1980, Joanette and Dudley rated the speech of 22 patients with Friedreich's ataxia using 16 of the speech dimensions reported by Darley et. al., (1975). They concluded that two speech factors were present : a general dysarthria, including reduced intelligibility, monoloudness, prolonged phonemes, inappropriate silences, imprecision consonants, and distorted vowels; and a vocal stenosis type, including harshness, pitch breaks, and manopitch level.

Respiratory functions

As early as 1929, Hiller studied the dysarthria in subjects Friedreich's ataxia and concluded that the primary speech problem of patients with cereballar lesions is one of respiratory control. In 1982, Putnam et. al., studied the respiratory kinematics and they reported that for all three cases, in spite of chest wall disorganization, weakness or component part deficits, the patients were still able to exchange enough

air and move it under enough pressure to produce an acoustic speech signal. However, in all three cases, velopharyngeal incompetence made respiratory efforts and compensations somewhat futile.

MULTIPLE SCLEROSIS

Medical Aspects

Multiple sclerosis is a disease of the white matter of the central nervous system which is characterized by progressive neurological deficits and, most commonly, a remitting/relapsing course. Although it is a relatively common disease and has been the object of intensive research in recent years, no definite knowledge exists of its etiology or effective therapies. The macroscopic lesions of Multiple Sclerosis are multiple plaques that are scattered throughout the nervous system, predominantly in the white matter. These are commonly seen in the periventricular area and tend to be symmetrical (McFarlin & Me Farland, 1982). Microscopically, the lesion is known to cause destruction of the myelin sheath with preservation of the axon, except in very chronic cases. The lesions are generally associated with small veins and venules, surrounded by lymphocytes, plasma cells, and macrophages. In an "acute" plaque, edema is seen in the vicinity of the affected nerve fiber. Resolution of this edema may be an explanation for the early reversal of some neurologic deficits after an exacerbation. The persistant neurologic signs are thought to be due to impaired saltatory conduction along the nerve axon (Hallpike, Adams, & Tourtelotte, 1983).

Research into possible etiologies indicates that there may be an environmental agent (e.g., a virus), a deranged immune response, or a combination of the two. Geographic studies indicate that the highest prevalence of the disease is in the higher latitudes in both the northern and southern hemispheres. Migrants from a higher

prevalence area to a lower prevalence area and vice versa have a prevalence that is midway between both areas. The critical age of exposure appears to be about 15 years. This may also indicate some genetic predisposition to the disease. Because of the similarities between MS and other demyelinating diseases (e.g., postinfectious, encephalomyelitis,subacute, sclerosing panencethalitis, and progressive multifocal leukoencephalopathy) that are caused by viruses and because of its similar onset to slow viral diseases (e,g.,Creutzfeldt-Jakob disease) a search for a viral cause has been made. No viral components have yet been identified; however, new nucleic acid hybridization techniques may prove fruitful in future identification. The presence of lymphocytes and macrophages in Multiple Sclerosis lesions and of elevated IgG levels in the cerebrospinal fluid have led to the consideration of a malfunctioning immune system. Again, however, there has been no evidence of cross- reactivity between patients; it seems more likely that the immune response is a reaction to an environmental agent (Ellison,Visscher, Graves, & Fahey, 1984).

Population characteristics

In the northern part of the United States, the prevalence is about one in 1000 of the population; it is one-third to one-half that in the southern state. About 95 percent of all cases begin between the ages of 10 and 50 years with a median onset age of 27years. Although it is a disease of younger people, if not unusual to be first diagnosed at 50 to 60 years; in these paitents, there are usually signs of chronicity. The female to male ratio is 1.5:1 (Arnason, 1982).

Signs, Symptoms, **and Natural** Course

Charcot first comprehensively described Multiple Sclerosis in 1877 as having nystagmus,scanning speech, and intention tremor. The most common symptoms in

this population are balance abnormalities (70 percent), impaired sensation (71 percent), paraparesis (62 percent), difficulty with micturition (62 percent),opu'c neuritis (55percent), and impotence (5-80percent) (Hallpike,et al.,1983). Optic neuritis is the acute or subacute loss of central vision with peripheral sparing in one eye; it is the first symptom of Multiple Sclerosis in 16 to 30 percenfof all patients. A young adult with isolated optic neuritis has a 17 to 65 percent risk of developing Multiple Sclerosis in later life. Other reliable symptoms of Multiple Sclerosis are intranuclear opthalmoplegia or bilateralocular paresis, tic douloureux or trigeminal neuralgia in a young adult, Lhermitte's symptom, acute transverse myelitis, and a "sensory useless" hand. Fatigue for which no objective explanation can be found is both a common and quite disabling complaint. On physical examination, vibration and position sense are frequently decreased or absent. Intentions tremor ataxia and hyperreflexia are common (Poser, 1984).

Vision and hearing are commonly ignored in evaluation. But cranial nerve dysfunction, scotomas, and decreased visual, and auditory acuity are not uncommonly present. Definite evidence of cognitive impairment is presents in over half of the patients. Neuropsychlogical tests have shown that the impaired abstract conceptualization and recent memory are the areas most frequently involved. Signs of bladder dysfunction appear at sometime in 50 to 80 percent of all the patients. This may include frequent urination, incontinence, or urinary retention; urinary tract infection and stones can be a source of significant morbidity in this population.

The average life expectancy in a young male after onset is 35 years. A number of studies have shown that disability scores calculated five years after onset correlate well with disability at 10 and 15 years; careful evaluation of function, therefore, can

be of prognostic value (Hallpike, et al., 1983). Prognosis is worse in males, if the age of onset is greater than 35 years, if a chronic progressive pattern is present at the onset, or if cerebellar symptoms occur at the initial presentation (Poser, 1984).

The clinical course can be divided into the following five classes:

1. Relapsing and remitting- About 70 percent of young patients with Multiple Sclerosis begin in this category with full recovery from neurologic signs and symptoms after each episode
2. Chronic progressive- This is most commonly present in patients older at the onset
3. Combined relapsing/remitting with chronic progressive- This is the eventual outcome in the majority of patients
4. Benign- About 20 percent of all patients have a normal life span with relatively normal functioning and little or no progression
5. Malignant- Five to ten percent of patients (usually young) show rapid and extensive involvement of cognitive cerebellar, and pyramidal systems, leading to death (Poser, 1984).

Speech characteristics

In 1868, Charcot described a characteristic triad of signs- nysagmus, intention tremor, and scanning speech-which he termed disseminated sclerosis, today called multiple sclerosis. Scanning speech referred to the prolonged phonation of words with slow and slurred articulation. However, as large groups of individuals with Multiple Sclerosis were studied, it became apparent that dysarthria was not a universal characteristic of Multiple Sclerosis. Ivers and Goldstein (1963) completed a retrospective study of 144 individuals with Multiple Sclerosis and reported that dysarthria was present in 19 percent of them. In their sample, dysarthria was the presenting symptom only in 2 percent of the patients. Darley, Brown, and Goldstein (1972) evaluated 168 patients with Multiple Sclerosis. Speech samples were perceptually analyzed, and they reported that 41 percent of the samples demonstrated

overall speech performance that was not essentially normal in terms of the impact on the listener. Beukelman, Kraft, and Freal (1985) reported on a survey of 656 individuals with Multiple Sclerosis. When a self-report technique was utilized, 23 percent reported a "speech and /or communication disorder". Perhaps the difference in the prevalence data reported in these studies resulted from the different methods used. In the reported studies, the prevalence of dysarthria in Multiple Sclerosis ranges from 19 percent to 41 percent. These differences may be due to the differences in population sampled and the evaluation methods used.

Obviously, severity of dysarthria in this population also varies. In the study by Beukelman and colleagues (1985) four percent of the respondents claimed to have communication so severely impaired that strangers were unable to understand them. Twenty-eight percent of this severely communicatively impaired group reported that they used augmentative communication approaches for communication.

Other Communication Problems

Although dysarthria is the most common communication problem observed in individuals with Multiple Sclerosis, aphasia has been infrequently reported. Olmas-Lau, Ginsberg, and Geller (1977) summarized the literature and reported that: "in several large reported series of multiple sclerosis patients, aphasia have been absent ... Other authors have rated the incidence of aphasia from 1 to 3 percent.". Poser (1978) reported two cases of aphasia in 812 individuals with Multiple Sclerosis. Although aphasia appears to be present in some cases of Multiple Sclerosis, Kraft (1981) points out that in the Multiple Sclerosis population intellectual dysfunction might be mislabeled as aphasia, because it also can affect language performance.

Perceptual Characteristics

Although "scanning speech" was included as an early symptom of Multiple Sclerosis by Charcot, there have been few careful studies of the dysarthric characteristics associated with this disease. Farmakides and Boone (1960) reviewed the case histories of 82 Multiple Sclerosis patients referred for speech therapy. They reported six characteristics that generally contribute to the dysarthric speech pattern - nasal voice quality, weak phonation and poor respiratory cycle, changes in pitch, slow rate, intellectual deterioration, and emotional liability.

Darley and colleagues (1972) rated the speech dimensions of 69 individuals with MS. They found that the perceptual speech patterns were consistent with a mixed dysarthria with both ataxic and spastic components. They summarize the speech characteristics as follows: the most prominent speech deviations in Multiple Sclerosis are impaired control of loudness, harshness, and defective articulation. Impaired use of vocal variability for emphasis, impaired pitch control, hypernasality, inappropriate pitch level, and breathiness are observed less frequently.

MYASTHENIA GRAVIS

Medical Aspects

Myasthenia gravis is an autoimmune disorder that is characterized by abnormal fatiguability and weakness of skeletal muscles. The cause of this weakness is a defect in neuromuscular transmission. In the normal neuromuscular unit, the terminal axon of a motoneuron displays complex branching; the membrane of the muscle and plate shows a similarly complex arrays of clefts. This arrangement increases the amount of surface area that can be involved in transmission and thereby increases the strength of the chemical stimuli that crosses the unit. Acetylcholine (ACh) is the chemical produced by the neuron which fits in to the receptor on the end

plate and allows for depolarization and contraction. An enzyme, acetylcholinesterase (AChE), breaks the ACh molecule into component parts and causes the receptors to become available again for the next impulse.

Population characteristics and Natural Course

Myasthenia gravis has a prevalence of about one per 20,000 in the United States. Congential and juvenile myasthenia are rare; however, some infants born to myasthenic mothers may show a transient form of the disorder. Females are affected about twice as often as males in young adulthood. After age 40, a slight preponderence of affected males may be seen. No genetic link is known (Seybold, 1983), and the natural history of myasthenia is essentially unknown. The most common initial symptoms involve the extraocular muscles (optosis, diplopia, and blurring of vision). Other presenting symptoms include leg weakness, generalized fatigue, difficulty in swallowing, slurred and nasal speech, difficulty in chewing, weakness of the face, arms, or neck, and trunk weakness or shortness of breath (Grob et al., 1981). Muscle weakness may not be present in the well-rested patient but can usually be elicited after exercise. Muscle atrophy is rare. Weight loss is most often due to difficulty in chewing or swallowing. Crises, due to myasthenia process itself or to overmedication, usually manifest as acute respiratory insufficiency, aphonia, and immobality. Al onset, about 40 percent of patients will have only occular signs and symptoms; within seven months about 60 percent of these will progress to generalized myasthenia gravis (Grob et al., 1981). The disease has an unpredictable course; spontaneous remissions can occur in any patient. Most patients show a fluctuating course with a particular muscle group primarily affected (Seybold, 1983). The maximal level of weakness of patients with generalized disease is reached within one to three years (Grob et al.,1981). The disease symptoms can be exacerbated by

enviromental (bright light, heat), physical (Pregnancy, viral illness, surgery), and emotional factors. The majority of patients with "idiopathic" cries had dysarthria or dysphagia at the time of occurrence, thereby predisposing them to aspiration (Cohen & Younger, 1981). Other organ system disorders are frequently associated with myasthenia gravis. Thymomas are present in at least 10 percent of patients; thymic hyperplasia is extremely common in younger patients. Other autoimmune disease, including thyroid disease, rheumatoid arthritis, systemic lupus erythematosus, and pernicious anemia, can be seen more often than in the normal population (Seybold, 1983).

The clinical signs and symptoms are sometimes sufficient to make the diagnosis of myasthenia gravis; more often, laboratory and pharmacological testing are necessary. A blood test for the AChR antibody is the simplest; however, since a large percentage of patients will not have a detectable level, a negative test does not rule out myasthenia gravis. Pharmacological testing involves the intravenous injection of short-acting cholinesterase inhibitors which inhibit the breakdown of AchR, a positive test will result in improved muscle strength or phonation for several minutes. Electrical testing for myasthenia gravis involves the fact that repetitive stimulation of a normal neuromusclar junction will produce action potentials with unchanging amplitudes. In the patient with myasthenia gravis, however, the muscle response amplitude decreases at least 10 percent between the first and the fifth responses. Testing under conditions of ischemia, heat, exercise, or after exposure to curare will increase the sensibility of the test. The last method to be used in specialized testing centers involves single-fiber electromyography. In a normal muscle, the time interval between the firing of two terminal branches of a motor unit is variable, a character

called jitter. In a disease of the neuromusclar transmission this can often be seen (Seybold, 1983).

Speech Characteristics

The severity of the speech characteristics demonstrated by individuals with myasthenia gravis is dependent on the severity of the syndrome, the effectiveness with which the symptoms are controlled with medications, and the fatigue level at the movement. According to Grob (1958), 15 percent of persons with myasthenia gravis have bulbar involvement that causes the symptoms and signs of dysarthria. Generally, the speech symptoms result from weakness of the muscles of the soft palate, pharynx, tongue, and larynx (Walton, 1977). This weakness is usually reflected in increasing hypernasality, deterioration of articulation, increasing dysphonia, and reduction of loudness level. Speech may become unintelligible (Darley et al., 1975). Speech abnormalities may be the initial symptom of myasthema gravis. Wolski (1967) reported a case study of a 14 year old girl with myasthenia gravis whose presenting symptoms were hypernasality and nasal emission. Aronson (1971) reported a case study of a 20 year old woman with myasthenia gravis who demonstrated a mild, breathy dysphonia, which had been previously diagnosed as a symptom of a psychogenic condition. Aronson (1971) presented this case study to "alert the clinician to the fact that voice changes can be one of the first and only signs of early neurologic disease".

Thus various neurological disorders lead, directly or indirectly to, various deviations in speech i.e. it may be from very mild to very severe dysarthria, making the speech totally unintelligible. In some cases the speech may be disturbed to a very great extent and it becomes impossible to use it effectively for the purpose of

communication. In such cases services of speech pathologist are sought and several therapy techniques have been developed for treating the same. Therefore it becomes very important to identify the neurological impairment responsible for the deviant speech production. The therapist can alter the speech parameters by making the patient to gain control over the deviant neuromuscular activity. The appropriate modifications and the choice of therapeutic techniques especially in the early stages of treatment must be based on a clear knowledge of the type of neuromuscular deficit underlying each aspect of the disorder speech.

Darley et al. (1969) opined that the speech production process relies on achieving appropriate muscle tone in the speech mechanism, muscle contraction at the right time, leading to the correct speed and force of movement, the correct range and direction of movement and the correct coordination and temporal sequencing of thousands of rapid successive (and simultaneous) interrelated, interdependent neuromuscular events. In dysarthrias there is deviation of muscular tone and contraction leading to deviations in range , force, rate, direction, rhythm and coordination which altogether make the speech abnormal. As speech pathologists main interest is in the acoustic output in the form of speech it becomes necessary to know the deviant aspect of muscle activities or neuromuscular activity underlying aspects of the deviant acoustic output or speech.

As the production of speech requires fine muscle coordination for its production even a mild neurological disorder related to speech may reflect itself in the form of speech deviation. Thus the study of speech parameters may be useful in the early identification of the neurological disorders. The assessment of dysarthric patient mainly aims at isolating the acoustic parameters which are deviant and to identify the

underlying deviant neuromuscular patterns so that this knowledge helps the therapist in planning the treatment program. That is having identified the parameters affected in the speech of a particular dysarthric patient., specific speech exercises can be devised to improve performance along the clearly defined lines and also this description framework provides more rigorous criteria for measuring improvement in patient's performance. Further such an analysis will be useful in differential diagnosis of various neurological disorders. Even it will become possible for the speech pathologist to identify the underlying neurological disorder by analyzing the speech of the dysarthria.

Various investigators have worked on analysing tie speech of the dysarthrias and found that a reduction in intelligibility is significant characteristics of dysarthric speech. In earlier times objective methods of measurrs intelligibility were not available. The approach to evaluate the dysarthric speech was based on highly subjective methods of classification. These methods were of questionable validity and of limited utility. Several workers (Zentay 1937, fros- es 1943, Fescher 1950, Grewel 1957) have made attempts to classify dysarthric speech. These classifications were based on localization of the lesion and subjective description of the speech resulting from the lesion. Descriptive terms such as "Shrred articulation", "Vague articulation," "unsteady voice' and scanning speech are typical (Morley 1955). Thus the need for experimental verification of the theories and methods for evaluating the dysarthric speech in a systematic manner has been felt.

Tikofsky and Tikofsky (1964) explored the applicability of intelligibility testing to the evaluation of dysarthric speech. Recorded samples of dysarthric speech was employed to measure speech intelligibility. They used three word lists. They concluded that it is possible to develop objective means of some aspects of dysarthric

speech through intelligibility testing and also that such measures when combined with other techniques will permit a better estimate of the nature and extent of dysarthric impairment.

Sarno (1969) suggested the use of a "functional intelligibility rating which should reflect, how well the speaker can make himself understood inspite of whatever speech impairment he may exhibit". Darley, Aronson and Brown (1969) employed a standard stimulus passage and scaling procedure in order to obtain an overall intelligibility measures. Tikofsky (1970) proposed a standard set of 50 single words to quantify intelligibility. Dysarthric speakers read the words and intelligibility scores were derived by computing the percentage of words correctly transcribed by nine listeners.

Yorkston and Beukelman (1975) compared intelligibility scores derived from a variety of quantification methods including estimate scaling procedures and six objective measurement techniques. The relative sensitivity and reliability of these measures across a wide range of dysarthric speakers was reported . Beukelman et al (1980) tried to determine the relationship between information transfer and speech intelligibility on measurements by single word and paragraph transcription across a wide severity range of dysarthric speakers. The results of the study revealed a close relationship between transcription intelligibility scores and information transfer (an index of the successfulness of communication.)

Canter (1965) abandoned single-word articulation tests when his pilot work revealed that certain Parkinsonism individuals performed normally on such tests yet displayed obvious articulatory difficulty in connected speech. Sarno (1968) in her review of Parkinsonian speech indicated that "the type or degree of phonemic

dysfunction does not necessarily correlate with the limitations noted in movements of the oral musculature. Further more, phonetic transcriptions are inadequate because most dysarthric articulation errors are distortions rather than substitutions or omissions.

Measurement of diadochokinetic rates of various speech related structures have also been reported in the literature as technique for quantifying dysarthric performance (Darley et al 1972). The use of diadochokinetic rates as a clinical measure of communication changes, however, is questionable because the repetitive sound units are not meaningful and do not occur in natural speech. The sensitivity of various techniques for quantifying dysarthria was explored by Yorkston and Beukelman (1980). They proposed a technique to objectively monitor changes in performance of dysarthric speakers over time during therapy. This technique depends on the multiple choice format developed for audiological testing by Black and Heagen (1963). Most of the analysis are based on perceptual identification of various deviant parameters of speech. However, the advancement in technology has permitted analysis of speech using various equipment such as:

Electromyography (Netsell and Cleeland, 1973)
U-tube manometry (Netsell 1976, and Hixon 1972)
Cinefluoregraphy (Kent and Netsell, 1975)
Aerodynamic studies (Hardy, 1967)
and Spectrography (Kent et al 1979, Lehiste 1965, Nataraja et. al., 1982, Rajkumar pandita, 1983)

Lebrun et al (1973) opined that ..."there is no doubt that perceptual characteristics are valuable in themselves but the inferences of acoustic and

physiological abnormalities from perceptual dimensions can be difficult and uncertain."

Acoustic analysis of speech

Among different types of instrumental analysis (e.g.,aerodynamic, electromyographic) that could be used in speech disorders, acoustic analysis can be highly recommended for following reasons. First, there is a well developed body of literature on acoustic characteristics of normal speech production (Baken 1987, Kent and Read, 1992; Klatt 1987) and growing literature concerning acoustic characteristics in various speech disorders, including those resulting from neurologic disease (Kent, Weismer, Kent & Rosenbeck 1989).

Second, the acoustic output of the vocal tract can be thought of as a bridge between speech production and perception and so is uniquely able to shed light on both problems of the mechanism associated with disordered speech and the effect of those problems on speech intelligibility.

Third, the acoustic output of the vocal tract contains the product of the entire speech system effort, rather than an isolated component of that effort. (Karren and Weismer 1997). To the extent that a speech disorder is defined by its anomalous communication product, acoustic analysis is may therefore prove to be valuable.

Fourth, acoustic analysis is completely noninvasive, and last, computer based analysis of speech acoustic have become highly sophisticated, accessible and relatively cheap. Acoustic analysis of speech is therefore within the reach of many clinicians for diagnostic, data keeping and research purposes.

Information is available in the acoustic signal concerning such factors **as** speaking rate, articulatory configurations for vowels and consonants, rate of change in the over all configurations of the vocal tract, flexibility of articulatory behavior, and aspects of phonatory behavior. The measurements made to draw inferences about articulatory and phonatory behavior often reveal a pattern that explains the reason for unintelligibility of speech and therapy that may be focussed on a particular aspect of speech production to improve intelligibility.

Acoustic representation of speech

There are varieties of ways in which an acoustic signal can be displayed and when it comes to the speech signal, the format of the display will impact the types of measures that can be made. In an attempt to quantify aspects of the speech signal, measures of temporal and spectral characteristics often are undertaken. (ForresLK.and weismer. G. 1997). Temporal characteristics reflect the duration of selected events where as spectral characteristics show the distribution of sound energy across frequency (i.e. the patteren of resonances of a given sound). The precise nature of these temporal and spectral measures will vary with the utterence produced. Factors that influence the choice of acoustic measures include manner of production and voicing for consonants, as well as source characteristics and nasalization.

Segmentation and Measurement of the speech wave

The speech wave is a complex, time varying signal from which temporal "Pieces" must be selected for analysis (Weismer et al 1997). The selection of these pieces can be made from waveform displays, which show sound energy amplitude as function of time (fig. VIII, top) and from spectrograms, which are three dimensional displays of frequency, time and relative amplitude (fig. VIII, bottom). The speech

signal, such as that displayed in figure, can be segmented to identify measurement intervals that are relevant to the structure of the utterance.

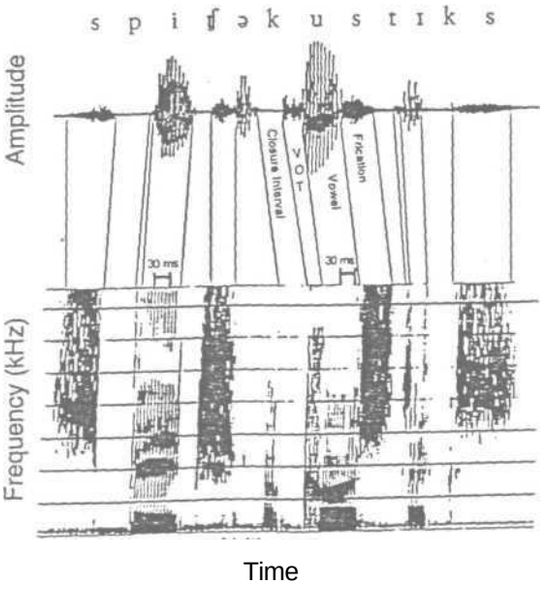


Figure VIE. Waveform (top) and spectrogram (bottom) of the utterance "speech acoustic." Note the segmentation of the utterance into consonantal and vocalic elements and the correspondence between these segments on the waveform and spectrogram. Note the 30 msec window in the center of the vowel *l\l* used for spectral analysis of formant frequencies.

Boundaries between sentences, phrases, syllables, phonemes and so forth must be determined before temporal measurements of specific intervals can be made. Because of the interaction of speech segments due to co-articulation, boundary identification is in some cases a difficult task. Operational definitions of the onset and offset of events must be provided and consistent application of these definitions must be maintained through out the analysis.

TABLE M : Showing the factors that influence segment durations and vowels formant frequencies.

Speaking rate
Phonetic context
Position in utterance ex: at end Vs beginning of utterance
Stress
Inherent chracteristics (e.g., vowel tongue height, ligrounding, voicing)
Type of speech material (e.g., isolated vowels Vs speech etc.)
Idiosyncratic speaker characteristics (e.g.,dialect, age, gender, vocal tract length)

Segment duration (word duration) has been studied extensively because they are thought to reflect principles of speech timing. The acoustic, evaluation of speech sound duration must take into account the relative nature of these measurements, because the durations depend on so many factors.(Weismer et al 1997). Thus, when comparing speech segment duration from a clinical speech acoustic evaluation of a given patient to values found in the literature (either for neurologically normal or impaired speakers), care must be taken to ensure the equivalence of factors such as segment identity, stress, dialect etc.

Spectral measures have also been studied extensively, because they can be related to vocal tract configuration and by interference, articulatory positions and movements. In general spectral measures that are used to describe a given sound class (e.g., vowels) are based on a rather small temporal window. Because the resonance's of the vocal tract are constantly changing, as a result of constantly varying articulatory movements, a large temporal window for spectral analysis might include too many varying acoustic features and thus "Smear" the analysis. In many cases, however, the time varying vocal tract resonances are the critical features of interest, and analysis of formant transitions (i.e., changes in formant frequencies over time) is required.

Fant (1960), in his classic work, showed that changes in vocal tract configuration have predictable influences on the acoustic output. Although strictly unique relations between vocal tract shape and acoustic output cannot be defined, general principles can be applied. For example, in the case of vowels (1) advancement of the tongue from a posterior to anterior location within the vocal tract results in an increase in the frequency of the second formant(f2) and a decrease of first formant(fl)

frequency (Fant, 1960; Stevens & House, 1955,1961); (2) lowering of the tongue from high(e.g.,/i/) to low (e.g.,/ae/) positions within the vocal tract increases the Fl frequency; and (3) elongation of the vocal tract by lip protrusion or, larynx lowering tends to result in a decrease of all formant frequencies. The relationships between articulatory configuration and spectral characteristics are some what more complicated for consonants, but in general it can be stated that the patter-n of resonances, or formants, associated with a stop, fricative, or affricate production is lawfully related to the size of the vocal tract cavity in front of the major constriction. For example, the spectrum of the stop burst for *hi* has a higher frequency representation than the spectrum for /k/, because for the smaller front cavity in *Ixl* articulation. Because of these types of relationships, information about the spectral characteristics of speech can be extremely useful in the investigation of normal and disordered production. That is, insight about the articulatory bases of perceived speech abnormalities can be obtained by analysis of the spectral characteristics of the speech signals. A few limitations to interpretations of speech spectra need to be emphasized. First, comparisons of spectra across subjects need to be made with care. Differences in vocal tract sizes of the cavities comprising the vocal tract will result in changes in the speech spectrum. Because information about the physical dimensions of the vocal tract is difficult if not impossible to obtain directly, comparison between individual speakers needs to be made cautiously. Second, as in the case of segment durations, variations in the speech material will impact the spectra, so comparisons between speakers must be made using the same sample. Last, within speaker variations can be quite large so frequent repetitions of the materials is required to obtain a reasonable estimate of speaker characteristics.

Techniques for spectral analysis include computer based Fourier analysis and Linear predictive analysis as well as spectography. Recently computer programs are used to make these measurements which generate the LPC and Fourier spectra in fractions of a second using a few key strokes. Thus, the measurement of formant frequencies is simple and straightforward. Advantages of this procedure include the relative ease in estimating formant frequencies from the spectrum as well as the utility of the procedure with a periodic signals. Fonnant frequencies for vowels have been studied extensively (Kent & Read, 1992) and as summarized in Table M} Many factors can affect these measures. The same caution about comparing vowel duration from clinical settings to previously published data applies. However in consonants as the energy is spread widely through out the frequency range, the peaks in consonant spectra are unlikely prominent as the stable peaks in vowel spectra. The lack of stability of peaks in consonant spectra makes it difficult to quantify the acoustic characteristics via a small group of formants (e.g., Fi^2,F3). The spectral shape of consonants depends on the over all distribution of energy across the frequency range of interest, rather than a few selected peaks. Description of acoustic characteristics of consonants therefore, must include information about the shape of the spectrum in addition to frequency. Quantification of spectral shape not only avoids the problem of finding stable peaks in the spectrum, but also seems to reflect the perceptual processing of consonant spectra (Tomiak, 1990).

Acoustic analysis in motor speech disorders

The classical departure point for understanding the speech production deficit in motor speech disorders is the Mayo classification system (Darley, Aronson & Brown, 1969ab, 1975). 38 perceptual dimensions were then combined in various ways to produce apparently unique clusters of dimensions for the different dysarthria types.

Weismer et al. (1992) delineated the likely acoustical correlates of some of the perceptual dimensions used in the Mayo system. Although the Mayo clinic studies made use of 38 perceptual dimensions a more limited set seemed to figure prominently in the descriptions of several different types of motor speech disorders. Some of these perceptual dimensions along with their corresponding acoustic characteristics or measures are listed in Table N

Table N : Showing Prominent perceptual dimensions and the likely acoustic correlates

Perceptual Dimension	Acoustic Correlate
Distorted vowels	Vowel durations Formant frequencies Formant transitions
Impresise Consonants	Consonant durations Consonant spectra Formant transitions
Hyper Nasality	LowFl frequency Low intensity formants, Spectral zero's
Mono Pitch	Flat Fo contour
Mono Loudness	Flat SPL contour
Harsh Voice	Jitter, Decrease S/N ratio
Stress Abnormality	Limited Fo range Vowel duration, Consonant duration

The deviancies seen in the acoustic correlates of the prominent perceptual dimensions in various conditions have been already reviewed. Further more Table 0 summarizes the various acoustic and perceptual studies carried out during the decade on dysarthrics, However the studies done using the perceptual and acoustic measures were equivocal and are relatively few.

The review of literature suggests that there are scanty attempts at studying the acoustic parameters of speech in dysarthrics with a course and their use in diagnosis and therapy. Therefore it was felt it is necessary to analyze the speech of dysarthrics acoustically, which will provide specific information relating to the possible

physiological contributions to the various perceptual characteristics. Such analysis may therefore aid in determining patterns , facilitate early detection of disease and provide a tool for monitoring disease progression.

This in turn, can lead the clinician to make better decisions regarding patient education and counseling and lead to more efficacious treatment.

Table O : Perceptual and Acoustic Studies done on Degenerative Dysarthrics

Author	Subjects (ss)	Tasks	Measures	Results
Darlcy et al, (1975)	32 Parkensons subjects	Connected speech (paragraph reading, conversation or sentence imitation)	Perceptual ratings of 38 speech dimension	All had consonant imprecision prosodic changes were more pronounced than articulatory ones
	30 ss with chorea	Connected speech (paragraph reading, conversation or sentence imitation)	Perceptual ratings of 38 speech dimension	27/30 imprecise consonants 27/30 distorted vowels
		Connected speech (paragraph reading, conversation or sentence imitation)	Perceptual ratings of 38 speech dimension	All had imprecise consonants 24/30 distorted vowels 24/30 had irregular articulatory breakdowns
		Connected speech (paragraph reading, conversation or sentence imitation)	Perceptual ratings of 38 speech dimension	Distortion of vowels, slow rate shortness of phrases & impression of consonants
Darley et al(1972)	168 ss with multiple sclerosis	Connected speech DDK rates	5 - point rating scale	59 % had normal speech 46 % had defective articulation
Logemann et al., (1981)	200 ss with parkinson's disease	Fisher - Logemann lest of articulation compctancc (sentence version)	IPA transcriptions of misarticulation	Classes most affected were stops-plosives, affricates and fricatives
Logemann etal, (1978)	200 ss with parkinson's disease	Sentences from Fishcr-logemann test of articulation competance	Judgements of presence or absence of misarticulations	45 % - articulatory disorders 89 % - laryngeal disorders sounds with greatest construction affected
Berry et al., (1974)	20 ss with Wilson's disease	Connected speech	Perceptual ratings of 32 speech dimensions	1. Reduced pitch variability 2. Imprecision in consonant production 3. Articulatory breakdown 4. Low pitch, harsh, strain voice quality 5. Slow rate
Canter (1965)	17 ss with Parkinson's disease	Connected speech	Spectrographic analysis	1. Maximum phonation duration reduced 2. Pitch range reduced

Weismer, et al., (1989)	9 ss with Parkinson's disease	Three sentences	Spectrographic analysis	1. Decreased duration of voiced segment 2. Reduced fundamental frequency 3. Formant transitions 4. Increased VOX
Metter etal, (1986)	10 SS with Parkinson's disease & supranuclear palsy	Connected speech samples	Spectrographic analysis	1. Rapid speaking form 2. Reduced syllable duration 3. Monotone fundamental frequency 4. Continuous voicing
Ramig etal.,(1988)	6 ss with Myotonic dystrophy 8 ss with Hunting tons disease 8 ss with Parkinson's disease 1 ss with ALS	Voice samples phonations of/a/ , /i/ ,/u/	Acoustic analysis	I. Fundamental frequency 2. Jitter, Shimmer 3. Harmonic to noise ratio contribute to differential diagnosis and document decreases progression
Zwirner et al., (1991)	18 ss with Parkinson's disease 13 ss with Huntingson's disease 8 ss with cerebellar ataxia	Voice samples phonation of /a/.	Acoustic and perceptual judgement	1. Higher variability in SDFO, Jitter and shimmer measures 2. No significant relationship between perceptual and acoustic measures 3. SDFO differentiated among neuropathological subgroups
Weismer (1984a)	8 ss with Paarkinson's S young adults 8 geriatric adults	Sentence rcpilation conventional and rapid rates	Spectrographic analysis	1. Spirantization most frequent in Parkiusosn's group 2. Voicing in to stop closure 1 Parkinson's group had segment and phase level durations slightly shorter than geriatric group.
Kent etal, (1979)	Individuals with cerebellar disease and alaxic dysarthria	Sample sentence rcpilation and convention	Physiological and acoustic analysis	Lengthening of segments equalization of syllabic duration. Abnormal transitional segments.

Nataraja etaL, (1982)	Ataxic dysarthria	Speech sample	Spcctrographic analysis	Prosodic excess, Prosodic phonatory insufficiency
Dcpaul & Brooks (1993)	5 ss with ALS	Speech sample	Spectrographic analysis	1. Compression of vowel space in /i/ & /u/ and expantion of vowel space in /a/ 2. High Fl and low F2
King ctal., (1093)	14 ss with Parkinson's disease	Sustained phoiiation and speech sample	Acoustic analysis	1. Maximum and mean fundamental frequency variability seen. 2. Sustained vowel phonation, also has variable suggesting phonatory variability in the subjects
Strandetal.,(1993)	4 ss with ALS	Sentence production task Sustained phonation of/a/	Acoustic analysis	Greater variability in phonatory performance and vocal quality were seen among the subjects

METHODOLOGY

Review of Literature, as presented in the previous chapter has shown that the acoustic analysis of speech of dysarthrics would be useful in the diagnosis and treatment of dysarthrics. Therefore the present study was designed to investigate the speech characteristics of dysarthrics using acoustic analysis. There have been attempts to extract various acoustic characteristics of speech in dysarthrics. However, the following parameters, which were considered as useful and feasible to measure, have been considered in the present study and are grouped as follows.

1. Frequency parameters

a) Mean fundamental frequency in phonation for/a/./i/,/u/
b) Extent of fluctuation in frequency
c) Speed of fluctuation in frequency
d) Frequency range in phonation
e) Formant frequencies (F1,F2,F3)
f) Band widths (B1,B2,B3)

2. Intensity parameters

a) Mean intensity in phonation for /a/,/i/./u/
b) Extent of fluctuations in intensity
c) Speed of fluctuation in intensity.
d) Intensity range in phonation

1. Temporal parameters

a) Word duration
b) Vowel duration
c) Burst duration
d) Closure duration
e) Consonant duration
f) Voice onset time (VOT)

Subjects

The subjects comprised of two groups. The first group had six subjects with dysarthria and second had six normals. The subjects in these two groups were

matched in terms of age, sex and number. The mean age of both groups were around 55.9 years. All the subjects were literate, had Malayalam as their mother tongue and were well versed in reading and writing Malayalam. The first group comprised of three male subjects with Parkinson's disease, two female subjects with Amyotrophic Lateral Sclerosis and one female subject with Wilson's disease , who were randomly selected with severity ranging from mild to severe. Judgement regarding the severity was based on the reports provided by the speech pathologist and Neurologist of the Neuro-diagnostic center. These subjects were diagnosed as dysarthrics both by a neurologist and a qualified speech pathologist. The subjects were selected from the review cases at Srichitra Thirunal Institute for Cognitive Neuroscience's, Trivandrum. The descriptions of the cases with medical history have been given in Appendix-1.

Speech samples

Sustained phonation for about 5-6 secs of the vowels /a/, /i/**,** /u/ were recorded in order to extract both the frequency and Intensity measures. A list of 50 familiar words in malayalam native language were chosen from the test material used at the Department of speech science, All India Institute of Speech and Hearing, Mysore, was used as the speech material in order to measure the temporal parameters. Test material is given in Appendix-Et.

Instrumentation

_ AIWA PQ 1824 stereo tape recorder with H-legend external microphone.

_ Meltrack CR-X90 audio cassettes

_ VSS-12 bit A/D converter with speech interference unit and headphones/speaker.

A pentium (intel-200MHz) computer with 16 MB RAM, VGA and high resolution colour monitor.

Instrumental set up (Block Diagram)

Recording of Speech samples

Recording was carried out in a quiet room. The subjects were seated comfortably with the microphone at a distance of 6 cms from the mouth of the subject. Occupants of the test room were the investigator and the subject only. Following instructions were given to each subject prior to recording " Say /a/, three times each with comfortable loudness until I signal you to stop, later followed by /L' & /u/ ". Then read this word list (pointing to the word list to be read).

Thus the phonation and speech samples for each subject of both the groups were recorded using AIWA PQ 1824 stereo tape recorder with H-legend microphone on Meltrack CR-X 90 audiocassettes.

Analysis of Data

The analysis of the recorded data was carried out both perceptually and acoustically.

a) Perceptual Analysis

Only words were used. Three judges rated the recorded speech samples. These judges were trained speech pathologists. The recorded samples were played on a Philips stereo cassette deck F6112 tape recorder with attached headphones thus ensuring good listening conditions. Prior to the analysis, the judges were provided with a scoring sheet having the list of 28 deviant speech dimensions. The list is attached in Appendix-in.

The speech samples were rated on a five point scale by the judges. The scale was

1. Profound impairment
2. Severe impairment
3. Moderate impairment
4. Mild impairment

5. No impairment

The definitions of each dimension were read out to the judges before making the judgement. The judges were then requested to listen to the speech sample and describe (mark in the scoring sheet). The scoring sheet was based on the phonatory, respiratory, articulatory, resonatory and prosodic aspects of speech .

The dysarthric cases number 1, 4, 6 were reanalyzed by the judges in order to find the reliability of judgement.

b) Acoustic Analysis

Tape recorded [1] samples were used for analysis to obtained the required parameters from phonation and speech using the software Speech Science Lab (SSL) and VAGHMI " (Both from Voice and Speech Systems, Bangalore) loaded on a 200 MHz Pentium computer. For all analysis a block duration of 30 msec and a block shift of 10 msec was used. The speech samples were digitized using 12 bit ADC/DAC board at the sampling frequency of 16000Hz and were stored on the hard disk / floppies for further analysis.

Measurement of fundamental frequency in phonation (Fo)

The signal thus stored on the hard disk of the computer was submitted for analysis using the "INTON " ' programme of VAGHMI (VSS, Bangalore). The phonation signal was read in blocks or frames of 40 msec duration each. Autocorrelation technique was used to estimate the average fundamental frequency over this block of 40 msec. Intensity was measured as the RMS value in dB. Successive blocks were spaced by 10 msec. The minimum and maximum limits for Fo measurement were 50 Hz and 800 Hz. The analysis of the voice signal yielded the following parameters by digital display on the monitor of the computer.

1,2,3 Please see Appendix-iv for details

a) Frequency measures[4]
   1) Mean fundamental frequency
   2) Fundamental frequency range
   3) Extent of fluctuations in frequency
   4) Speed of fluctuations in frequency

b) Intensity measures [5]
   5) Mean intensity
   6) Range of intensity
   7) Extent of fluctuations in intensity
   8) Speed of fluctuations in intensity

The average of three readings was considered for obtaining parameters 1,3,4,5,7,8 where as the highest values of the three values was considered for parameters 3 and 6.

Extraction of vowel formant frequencies (F1, F2, F3)

The vowel formant frequencies (F1, F2, F3) were obtained only for the following three utterrances

/a/ - tʃettan, /i/ - Kitti, /u/ - tʃuttum

Using the 'SPGM' Program of the software ' Speech science lab ' a spectrogram of each utterance was obtained after identifying the target vowel, the cursor was placed in the middle of the vowel position so as to avoid formant transition and formant frequencies were determined by using the sectioning method through the use of linear predictive coding (LPC). This was done with 18 LPC coefficients. The frequencies at the peaks representing the formats were noted using the cursor.

4,5 Please see Appendix-v for details

Bandwidth

To Extract the vowel formant bandwidths (Bl, B2, B3) a spectrogram of the three utterances used for extracting formant frequencies was obtained using the "SPGM" program of the software "SSL" (speech science lab.)- After identifying the target vowel, the cursor was placed in the middle of the vowel position so as to avoid the formant transitions and the bandwidths were obtained by using the "PATPLAY" program of the software "SSL".

Measurement of Temporal Parameters

Temporal parameters were measured only for the following voiceless stop consonants /p/,/t/,/k/, in the following target words.

Consonant	**Target word**
/k/	Ka:ka,kattil.pakal
/p/	Paisa, kappal appol.
/t/	Pa:tta, tatta, mutta,

Word duration (in m sees)

The word duration was measured directly from the speech waveform, the waveform was displayed on the computer monitor using the "DISPLAY" program of SSL. The total word was identified based upon the regularity of the waveform. The total word was considered to extend from the beginning of the periodic signal to the end of the periodicity for the word. The duration was high lightened through the use of cursors. The highlighted position was played back through headphones, to confirm that the word under study has been high lighted and then the duration was marked correctly. Once this was confirmed, the duration of the highlighted position was read from the display on the monitor directly.

Closure Durations (in m secs)

The "DISPLAY" program of SSL was used to measure closure duration from the waveform, a gap between two periodic signals were highlighted using cursor. The highlighted portion was played to confirm that the closure has been marked correctly when the silence was perceived, then it was taken as closure. Once this was confirmed, the duration of the highlighted portion was read from the display.

Vowel duration (in msec)

The DISPLAY program of SSL was used to measure vowel duration also. The vowel duration was considered to extend from the beginning of the periodic marking to the end of the periodicity. This duration was highlighted through the use of cursors. The highlighted portion was played back through head phones to confirm that the vowel under study has been marked correctly and thus the duration has been identified correctly. Once this was confirmed, the duration of the highlighted portion was read from the display on the monitor directly.

Voice onset time (VOT) (in m sec)

VOT **(m** sec) of **/p/, /t/**, */k/* were measured with these sounds at initial position and medial position. The VOT were measured directly from the waveform. The "DISPLAY" program of SSL was used to display the waveform and the VOT was measured using the definition given by Lisker and Abramson (1967) i.e. the time interval between the burst (or brief interval of high intensity noise) that marks release of the stop closure and the onset of quasi - periodic pulsing that reflected laryngeal vibration was the voice onset time.

Burst Duration (in m sec)

The production of stops involves the complete closure of the vocal tract during which air pressure is built up in the mouth. On the release of the constriction the air pressure is abruptly released. The acoustic evidence of this release is abrupt or transient and burst duration in no longer than 5-40 m sec.

The "DISPLAY" program of SSL was used to measure the burst duration. The burst duration was considered to extend from the beginning of aperiodic marking to the end of aperiodicity which was highlighted and duration of the highlighted portion was read from the display on the monitor.

Statistical analysis

Descriptive statistics consisting of Mean and Standard deviation were obtained for all the parameters. To check whether there were any significant differences between the values of both the groups. Wilcoxons Matched Sign Rank Test was applied using SPSS program.

RESULTS AND DISCUSSION

The objective of this investigation was to study the acoustic parameters in different dysarthric groups and to explore the possibilities of using them for differential diagnosis of dysarthria. This was carried out by testing the following hypothesis.

1. There is no significant difference in the acoustic parameters between dysarthric and normal subjects.
2. There is no significant difference between the three types of dysarthric subjects: Parkinsons disease, Amyotropic Lateral Sclerosis and Wilson's disease in terms of comparable parameters as indicated (*)

1. Frequency parameters

a) Mean fundamental frequency in phonation for /a/, /i/, and /u/
b) * Extent of fluctuations in frequency
c) * Speed of the fluctuations in frequency
d) * Formant frequencies (F1.F2,F3)
e) * Frequency range in phonation
f) * Band widths (B 1,B233)

2. Intensity parameters

a) * Mean intensity in phonation for /a/, *l\l, IvJ*
b) * Extent of fluctuations in intensity.
c) * Speed of fluctuations in intensity
d) * Intensity range in phonation

3. Temporal parameters

a) * Word duration
b) * Vowel duration
c) * Burst duration
d) * Closure duration
e) * Consonant duration
f) * Voice onset time (VOT)

Note : (*) these parameters did not differ significantly between males and females in the normal group

Table 1A : Depicts Mean, SD values and Significance for all the frequency parameters in Normals and Dysarthric group.

\+ Significance at 0.05 level — No Significance

	NORMAL GROUP						DYSARTHRIC GROUP					
Frequency	MEAN (STANDARD DEVIATION)						MEAN (STANDARD DEVIATION)					
Parameters	/a/		/i/		/u/		/a/		/i/		/u/	
a) Fundamental Frequency	**(M) 165.24(0 (8.55)**	**(I') 2.12.2(1) (7.32)**	**170.17(0 (10.11)**	**254.23(1) (12.12)**	**175.7(0 (4.23)**	**237.15(1) (7.92)**	**156.24(0 (8.06)**	**222.2 (+) (10.11)**	**167.17 (+) (56.94)**	**196.76 (+) (55.93)**	**172.73 (+) (3.7)**	**203.11 (+) (11.8)**
b) Extent of Fluctuations	3.23 (+) (0.3253)		2.18 (-) (0.1528)		2.76 (+) (0.6807)		12.23 (+) (8.067)		5.67 (-) (7.082)		6.46 (+) (6.082)	
c) Speed of Fluctuations	1.24 (+) (0.4417)		1.75 (+) (0.7708)		1.26 (+) (0.9136)		18.52 (+) (2.364)		10.34 (+) (7.07)		11.33 (+) (2.133)	
d) Frequency Range	82.21 (-) (0.6324)		98.66 (-) (0.7071)		113.525 (-) (13.1521)		128.59 (-) (98.64)		107.19 (-) (96.72)		104.85 (-) (39.8172)	
c) Fonnant Frequencies												
Fl	783.80 (+) (58.59)		329 (+) (31.00)		354.2 (+) (34.50)		755.28 (+) (131.73)		336.72 (+) (67.06)		374.78 (+) (96.64)	
F2	1295.2 (+) (81.34)		2016.8 (+) (104.90)		1119.4 (+) (78.72)		1401.06 (+) (259.56)		2124.42 (+) (149.58)		1271.13 (+) (226.29)	
F3	2561.2 (-) (111.37)		2688.2 (-) (60.28)		2500.0 (-) (120.37)		2718.43 (-) (17.36)		2860.98 (-) (16.42)		2655.35 (-) (32.42)	
0 Band Widths												
Bl	165.4 (-) (61.8)		159.4 (-) (59.98)		442.6 (-) (245.52)		228.13 (-) (44.1)		121.5 (-) (59.06)		138.98 (-) (33.41)	
B2	322.6 (-) (101.8)		390.0 (-) (100.92)		639.36 (-) (104.62)		334.29 (-) (100.02)		212.35 (-) (71.88)		236.21 (-) (96.72)	
B3	531.2 (-) (137.32)		428.2 (-) (131.38)		787.2 (-) (112.03)		524.49 (-) (111.83)		343.59 (-) (129.61)		317.33 (-) (158.64)	

Tnblc IB : Depicts mean , ST) values and Significance for all the frequency parameters in Parkinsons disease ALS. Wilsons disease patients,

+ Significance at 0.05 level - Not Significant

	PARKINSONS DISEASE (N=.3)						AMYOTROPIC LATERAL SCLEROSIS (N=2)						WILSONS DISEASE (N=l)		
Frequency	MEAN STANDARD DEVIATION						MEAN STANDARD DEVIATION						MEAN		
Parameters	/a/	si	/i/	si	/u/	si	/a/	si	/i/	si	/u/	si	/a/	/i/	/u/
Fundamental Frequency	156.24 (14.5.1)		167.17 (15.01)		172.73 (17.58)		206.24 (8.06)		158.81 (56.49)		201.11 (1.7689)		254.11	273.68	202.73
Extent of Fluctuations	8.72 (2.93)		6.0167 (1.03)	-	6.94 (4.59)	-	22.3 (1.48)	+	4.49 (1.004)	-	4.95 (0.4313)	-	6.82	7.03	8.04
Speed of Fluctuations	28.1533 (28.22)	-	11.48 (10.14)	-	6.53 (2.13)	-	9.22 (2.35)	-	9.89 (7.07)	-	19.13 (9.3)	-	8.22	7.83	10.12
Frequency Range	152.27 (98.64)	-	74.48 (98.62)	-	13.74 (6.55)	-	107.02 (195)	-	173.45 (7.04)	-	215.03 (96.81)	-	100.72	72.83	157.83
Formanl Frequencies															
Fl	710.36 (88.89)	+	386.11 (12.16)	-	365.76 (8.37)	-	885.4 (72.82)	-	289.3 (71.2)	-	375.3 (12.87)	-	629.8	283.4	400.8
F2	1520.3 (67.13)	+	1781.18 (54.14)	-	1387.06 (50.7)	-	1273.6 (67.37)	-	2678.6 (80.8)	-	1232.8 (4.72)	-	1298.3	2045.8	1000.0
F3	2836.66 (81.8)	-	2739.43 (66.33)	-	2783.7 (108.89)	-	2576.6 (80.8)	-	3420.6 (70.7)	-	2367.0 (8.72)	-	2647.4	2106.4	2847.0
Band Widths															
Bl	324.4 (10.00)	-	83.8 (9.97)	-	133.03 (8.57)	-	130.0 (7.82)	-	170.2 (12.72)	-	150.4 (13.72)	-	135.6	137.2	134
B2	388.25 (8.4)	-	231.65 (10.3)	-	283.83 (8.77)	-	240.2 (2.72)		190.6 (11.72)	-	179.6 (12.92)	-	360.6	198.0	206.6
B3	688.63 (58.77)	-	416.85 (6.23)	-	385.4 (17.669)		340.2 (7.72)	-	220.2 (18.92)	-	248.6 (10.72)	-	400.7	370.6	214.6

The results of the acoustic analysis are discussed below in terms of each parameters studied. Statistical comparisons were done to find the difference between males and females in the normal group, which revealed that gender difference was significant only for Mean fundamental frequency parameter at 0.05 level as found using Wilcoxons Matched Pair Sign Rank test. Thus for the non-significant acoustic parameters males and females were considered as a single group.

FREQUENCY PARAMETERS

Inspection of Table 1A reveals that there is a significant difference between the dysarthric group and normals only in terms of the following frequency parameters.

a) Mean fundamental frequency for /a/,/i/, /u/
b) Extent of fluctuations in frequency for /a/, /u/ only
c) Speed of fluctuations in frequency for /a/, /i/, /u/
d) Formant frequencies.

Fl for/a/,/i/,/u/
F2 for /a/, *lul.* Only.

i) Mean Fundamental Frequency

Table 1A and Graph 1A &1B reveal the mean fundamental frequency in both male and female dysarthric subjects for vowels /a/,/i/,/u/ as

/a/-156.24 Hz ; /i/-167.17 Hz ; /u/-172.73 Hz

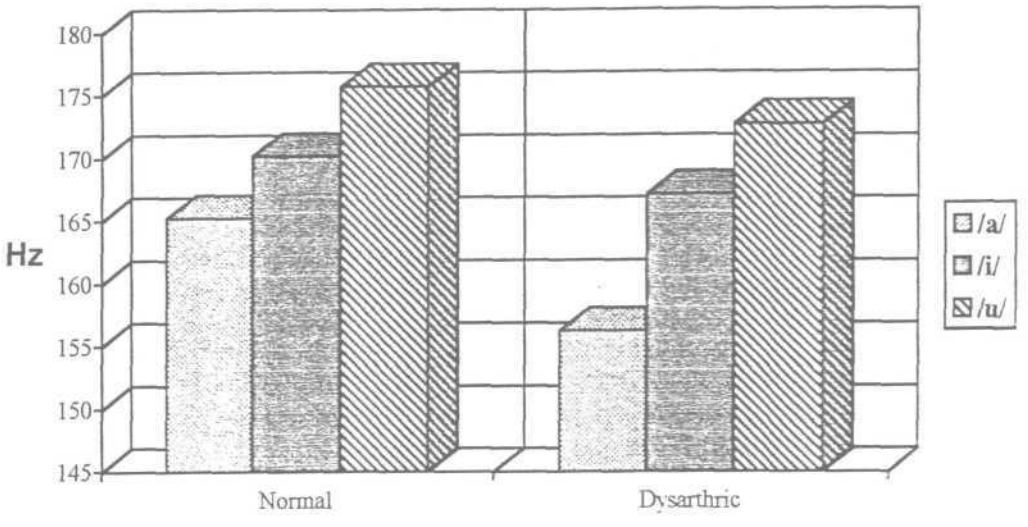
/a/-222.2 Hz ; /i/-196.76 Hz ; /u/-203.11 Hz respectively

Which is comparatively less than their normal counter pans whose mean fundamental frequency for *la/, IV, lul,* in both males and females were

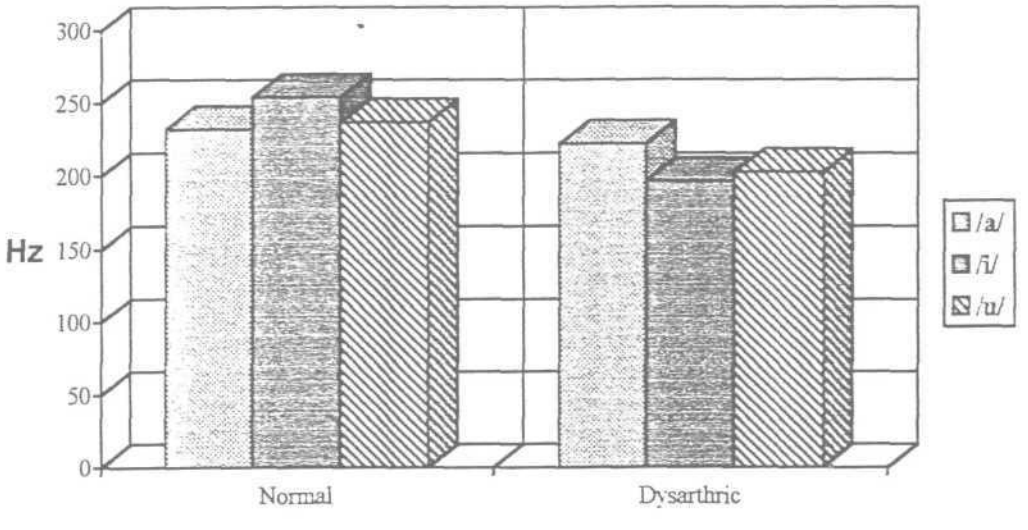
/a/-165.24 Hz ; /1/-170.17 Hz ; /u/-175.73 Hz

/a/-*2322* Hz ; /i/-254.23 Hz ; /u/-237.15Hz respectively

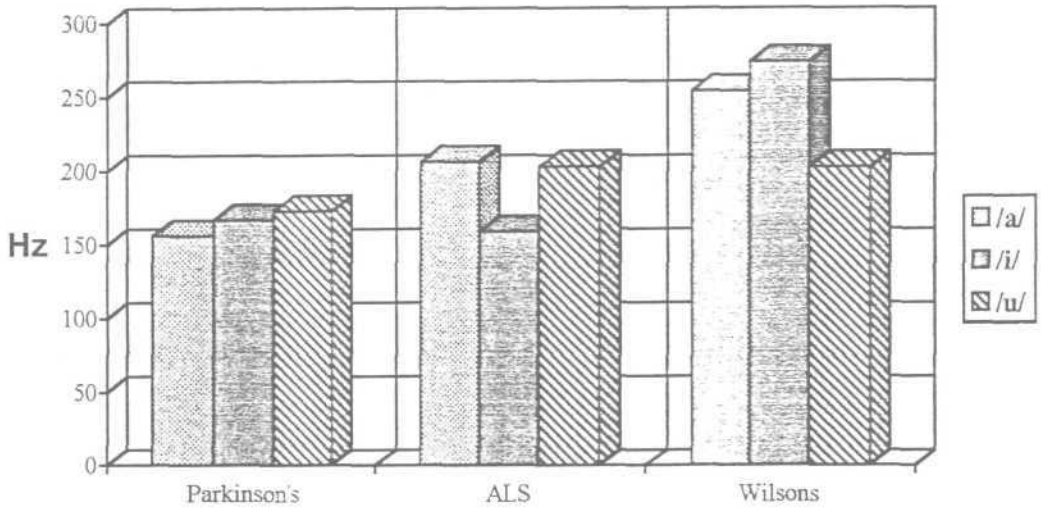
Graph 1A : Mean Fo (Hz) in males for normal and dysarthric group



Graph 1B : Mean Fo (Hz) in females for normal and dysarthric group



Graph 2 : Mean Fo (Hz) in Parkinson's, ALS, Wilsons disease



This difference seen between the two groups (including both males and females) were statistically significant for all the vowels /a/, /i/, /u/, as found using Wilcoxons Matched Pair Sign Rank Test.

Table IB and Graph 2 reveal that the mean fundamental frequency for /a/, /i/, *lul* in Parkinsons disease, Amyotropic Lateral Sclerosis and Wilson's disease as : /a/-156.24Hz, /i/-167.17 Hz, /u/-172.73Hz , /a/ - 206.24Hz, /i/158.3 Hz, /u/-203.3 lHz, /a/ - 254.3lHz, /i/ -273.68Hz, /*ul* -202.73Hz respectively.

From, the above data it can be understood that there is difference with in the dysarthic groups, however comparisons between groups cannot be established due to gender differences seen among the subjects.

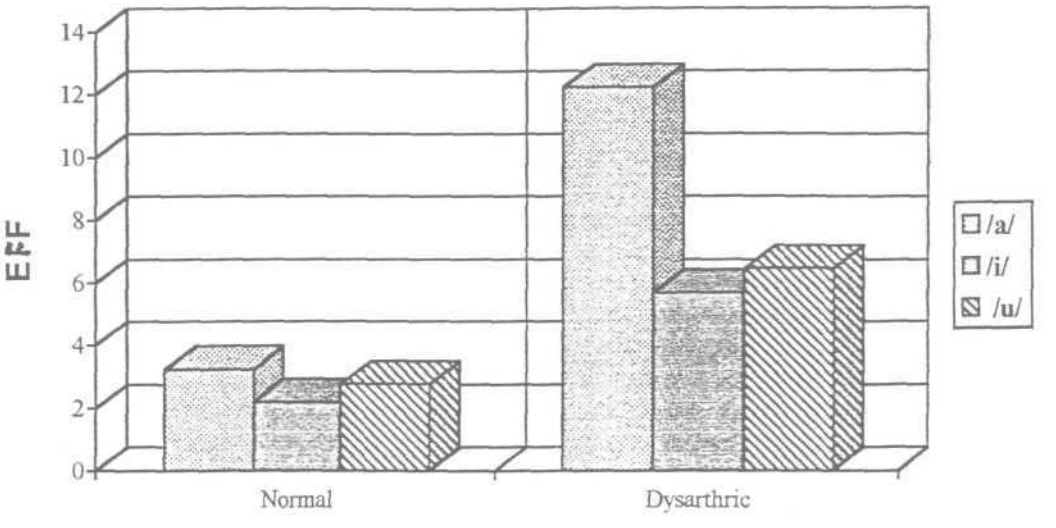
ii) Extent Of Fluctuations In Frequency

Table 1A Graph 3 reveal that the mean extent of fluctuations in frequency for /a/-12.93, /i/=5.67, *lul* - 6.46Hz in dysarthric group which is comparatively more than the normal group whose mean extent of fluctuations in frequency for /a/, *hi,* and *lul* are 3.23, 2.18, and 2.76Hz respectively. These differences between the two groups were statistically significant for vowels /a/, & *lul* but not for *hi* as found using Wilcoxons Matched Pair Sign Rank Test.

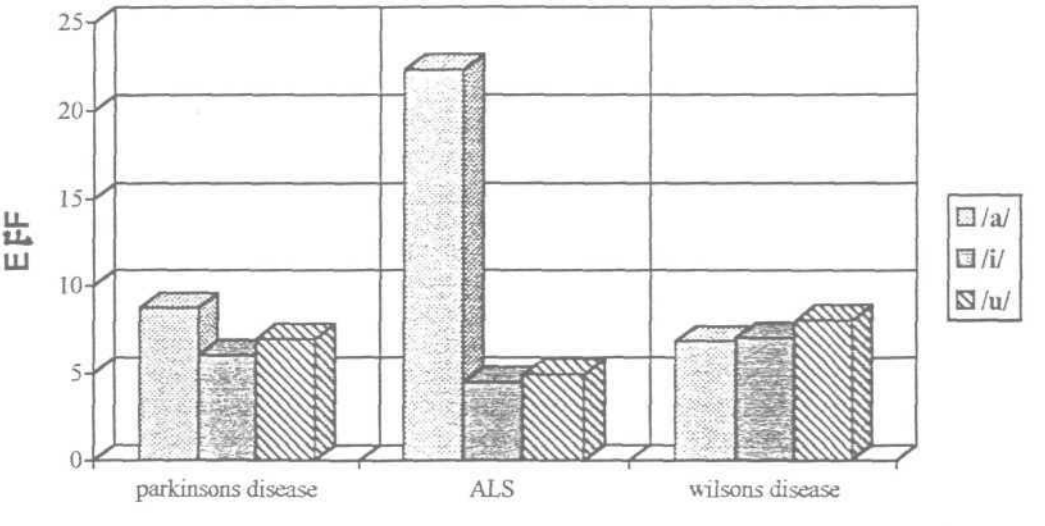
Table IB and Graph 4 reveal that the mean extent of fluctuations in frequency for /a/, /i/, */u/* in Parkinsons disease. Amyotropic Lateral sclerosis and Wilson's disease as: /a/ - 8.72; /i/ - 6.016; /u/ - 6.94, /a/ - 22.3; /i/ - 4.49, /u/ - 4.95, /a/ - 6.82; /i/ - 7.03; /u/ - 8.04 respectively.

From Table IB and Graph 4 it can be seen that there is difference within the dysarthric groups. However this difference was significant only for vowel /a/ at 0.05 level between Parkinsons and Amyotropic Lateral sclerosis, i.e.greater in Amyotropic

Graph 3 : Shows mean extent of fluctuations of frequency (EFF) in Normal and Dysarthric group



Graph 4 : Mean extent of fluctuations of frequency (EFF) in Parkinsons disease, ALS, Wilsons disease



lateral sclerosis than in Parkinsons disease cases and not with Wilsonsdisease when compared with other type of dysarthria.

Speed Of Fluctuations In Frequency

From Table 1A and Graph 5 it can be seen that the mean speed of fluctuations in frequency for /a/ =1.24; /i/ =1.75; /u/ = 1.26 in the normal group which is comparatively less than that of the dysarthric groups, whose mean speed of fluctuations in frequency for/a/, /i/and/u/are 18.52, 10.34 and 11.33 respectively.

These differences between the two groups were statistically significant for all the vowels at 0.05 level.

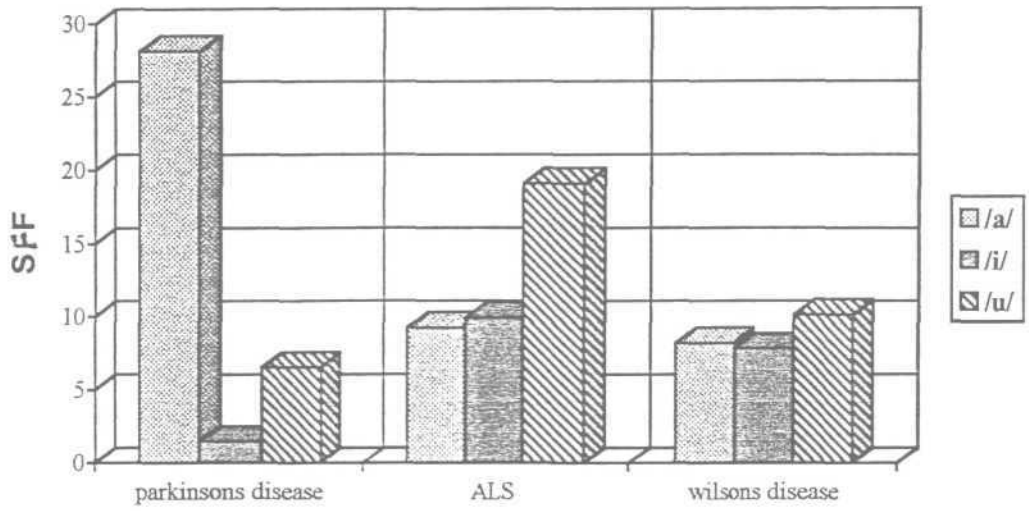
Further, inspection of Table IB and Graph 6 reveal that the mean speed of fluctuations in frequency for /a/, /i/ & /u/ in Parkinsons disease, Amyotropic Lateral Sclerosis and Wilsons disease as: /a/ - 28.15 ; /i/ - 11.48 ; /u/- 6.53, /a/ - 9.22 ;/i/ - 9.89 ; lul - 19.13,/a/ - 8.22 ; /i/ - 7.83 ;/u/ - 10.12 respectively.

From the above given data it can be seen that there is difference in the mean values of speed of fluctuations in frequency within the dysarthric groups. It is comparatively more in Parkinsons disease than in Amyotropic Lateral Sclerosis and, more in ALS than in wilsons disease. However, this difference was not statistically significant between the three dysarthric conditions.

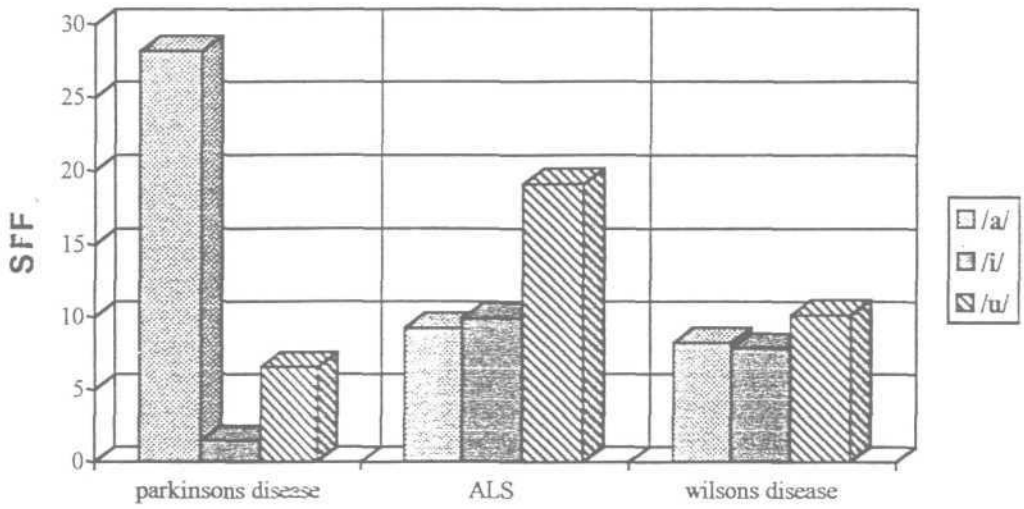
FORMANT FREQUENCY

Table 1A and Graph 7 reveal that the Fl, F2, F3 values for /a/ as 783.80Hz, 1295.2Hz, 2561.2HZ, for /i/ 329Hz, 2016.8Hz, 2688.2Hz, and for *IvJ* 354.2Hz, 1119.4Hz, 2500.0Hz in the normal group respectively, which are comparatively less

Graph 5 : Shows mean speed of fluctuations of frequency (SFF) in Normal and Dysarthric group



Graph 6 : Mean speed of fluctuations of frequency (SFF) in Parkinsons disease, ALS, Wilsons disease



F2& F3 in dysarthric group. The respective F1, F2, F3 values for /a/, /i/, /u/ in Hz are as follows:

/a/-755.28, 1401.06,2718.43.

/i/- 336.72 ,2124.42, 1271.13.

/u/-374.18- ,1271.3 ,2655.3.

This difference found between the two groups was statistically significant only for Fl and F2 of vowels /a/, /i/, /u/.

Further, inspection of Table IB and Graph 8 reveal that the formant frequency values for /a/,/i/, /u/ in Parkinsons disease, Amyotropic Lateral Sclerosis and Wilsons disease.

First formant frequency (Fl)

The first formant frequency for /a/ in Parkinsons, Amyotropic Lateral Sclerosis, Wilsons disease were 710.3 Hz, 885.4 Hz, 629.8 Hz, respectively.

The first formant frequency for /i/ in Parkinsons, Amyotropic lateral sclerosis, Wilsons disease were 386.11 Hz, 289.3 Hz, 283.4 Hz respectively.

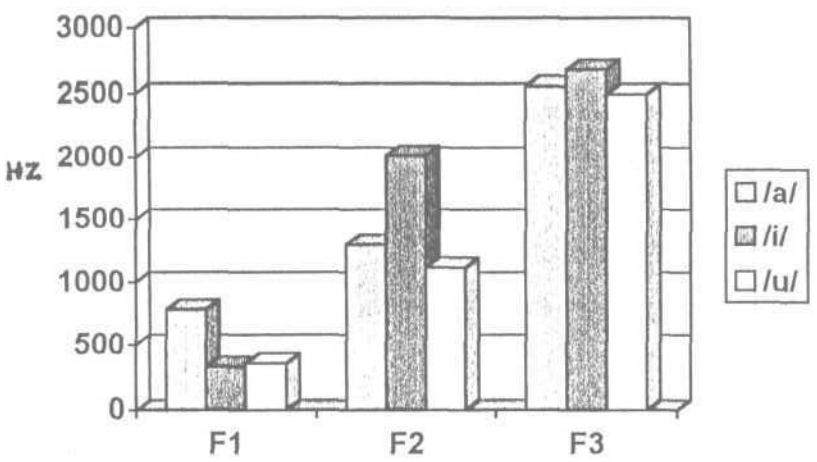
Further, the first formant frequency for /u/ in all the three dysarthric conditions were 365.76 Hz, 375.3 Hz, 400.8 Hz respectively.

This difference seen between the three dysarthric conditions was statistically significant at 0.05 level only between Parkinsons and Amyotropic lateral sclerosis for all the vowels.

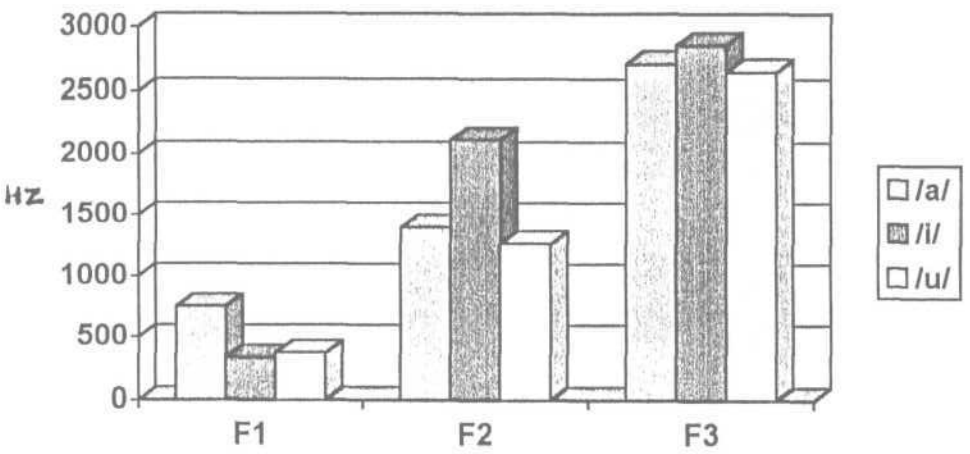
Second formant frequency (F2)

The second formant frequency for /a/ in Parkinsons, Amyotropic Lateral Sclerosis, Wilsons were 1520.3 Hz, 1273.6 Hz, 1298.3 Hz respecrively.For *I'll* the

Graph 7 : F1, F2, F3 (Hz) for /a/, /i/, /u/ in Normal and Dysarthric group

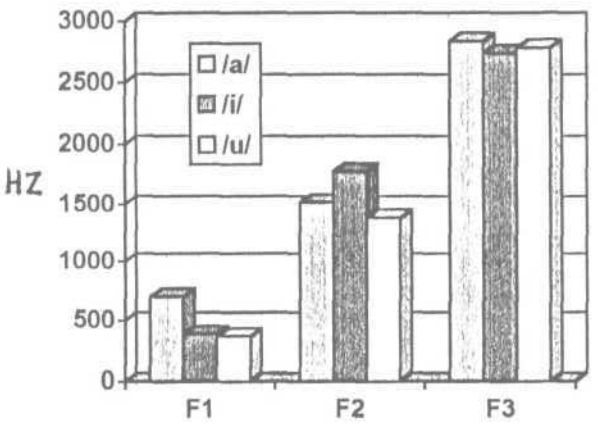


(Normal)

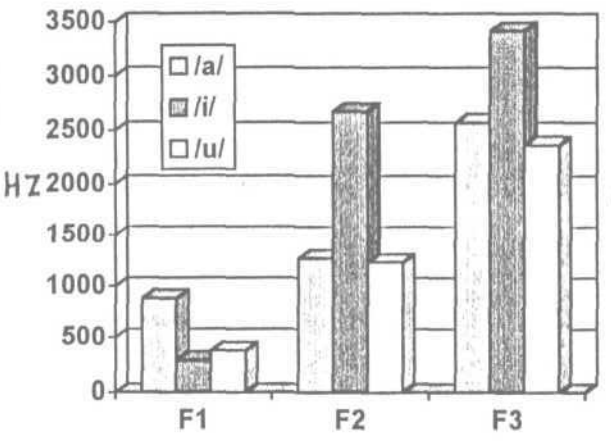


(Dysarthric)

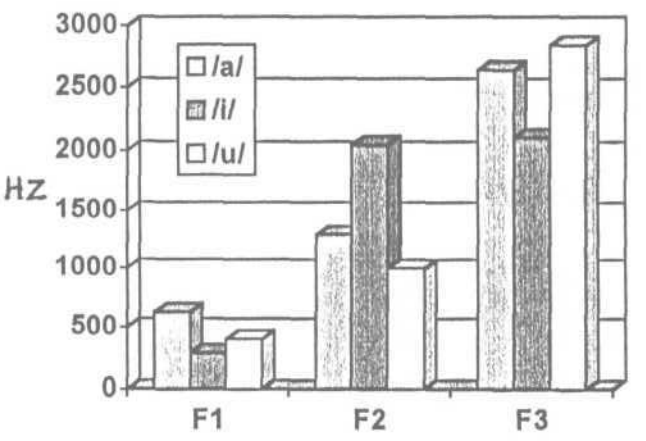
Graph 8 : F1, F2, F3 (Hz) for /a/, /i/, /u/ in Parkinsons, ALS, Wilsons disease



(Parkinsons)



(ALS)



(Wilsons Disease)

second formant frequency were as follows in Parkinsons disease- 1781.18 Hz , in Amyotropic Lateral Sclerosis - 2678.6 Hz and in Wilsons disease -2045.8Hz . For /u/ the second formant frequency values were as below:

In Parkinsons disease it was 1387.06 Hz ,in Amyotropic Lateral Sclerosis it was -1232.8 Hz and in Wilsons disease it was l000Hz..

This difference between the three dysarthric conditions were statistically significant at 0.05 level only ibr Parkinsons and Amyotropic lateral sclerosis for all the vowels.However no significant difference was found for any of the vowels when Wilsons disease was compared with the other dysarthric conditions.

Third formant frequency (F3)

On inspection of Table IB, it can be seen that there is a difference in the third formant frequency values between the dysarthric conditions for the three vowels 2/, /i/, /u/. However this difference was not statistically significant at 0.05 level.

Apart from these frequency parameters, other parameters were also studied i.e. the frequency range in phonation for /a/, /i/, /u/ and bandwidths (Bl, B2, B3) for vowels /a/, /i/, /u/ were also suciedd.

Table 1A reveals the there was a difference between the normal 2nd dysarthric group in terms of frequency range and bandwidths. However these differences were not statistically significant at 0.05 level. Further inspection of Table IB reveals a slight difference within the three dysarthric conditions in terms of frequency range and bandwidths. However, these differences were not statistically significant at 0.05 level.

Thus, the hypothesis stating that:

i) There is no significant difference between the dysarthncs and normal subjects in terms of frequency parameters is rejected

ii) There is no significant difference between the three types of dysarthrics i.e.: Parkinsons Disease, Amyotropic Lateral Sclerosis and Wilson's disease in terms of frequency parameters is also rejected

However the above two hypothesis were accepted only in terms of the following parameters where there was no significant difference seen at 0.05 level

- Frequency range in phonation.
- Band widths (B1, B2, and B3).

The findings of the present study are in consonance with the earlier studies reported in the literature that the dysarthrics as a group differ significantly in terms of frequency parameters from normals of the same age group (King et. al., 1993; Strand .et. al; 1993; Watanbe et. al., 1994; Kent et. al 1992; Ramig et. al., 1988). These differences can be related to a variety of laryngeal and articulatory parameters including, decreased muscle mass, reduced crico-thyroid function, degree of spasticity or flaccidity to laryngeal muscle and restricted tongue movement patterns. Further, the difference seen within the three dysarthric conditions can be attributed to the differential neural subsystem involvement and factors related to subject.

However, the significant difference seen was only between Parkinsons and Amyotropic Lateral Sclerosis and not with Wilsons disease, the reason for which may that only a single subject was taken up in this category, in the present study.

2. INTENSITY PARAMETERS

Inspection of Table 2A reveals that there was a significant difference between the dysarthric group and normals in terms of the following intensity parameters.

a) Mean intensity for /a/, /i/, /u/.

b) Extent of fluctuations in intensity for /a/, /i/, /u/

c) Speed of fluctuations in intensity for /a/, /i/, /u/

Table 2B : Depicts mean , S.D and Significance values for **all** the intensity parameters in Parkinsons disease, ALS, Wilsons disease patients.

\+ Significance at 0.05 level - Not Significant

	PARKINSONS DISEASE						AMYOTROPIC LATERAL SCLEROSIS						WILSONS DISEASE		
INTENSITY	MEAN STANDARD DEVIATION						MEAN STANDARD DEVIATION						MEAN		
Parameters	/a/	si	/i/	si	/u/	si	/a/	si	/i/	si	/u/	si	/a/	/i/	/u/
Mean intensity (in dB)	34.13 (4.59)	+	34.36 (6.99)	-	39.61 (3.16)	+	45.37 (4.48)	+	42.93 (3.03)	-	46.76 (1.44)	+	33.47	40.37	43.72
Extent of Fluctuations	4.01 (0.44)	+	3.87 (0.46)	+	5.15 (0.677)	.	0.00 (0.00)	+	0.3267 (0.28)	+	4.28 (0.42)		2.87	0.00	0.00
Speed of Fluctuations	5.69 (1.20)	+	2.13 (112)	-	3.95 (101)	.	0.25 (0.26)	+	0.30 (0.28)	-	0.84 (0.68)	-	3.27	1.23	3.92
Intensity Range	22.75 (164)	+	15.37 (107)	-	16.61 (2.7)	.	2.37 (0.54)	+	2.72 (0.33)	-	3.76 (1.61)	-	20.75	16.75	14.27

Table 2A : Depicts mean , SD values and Significance for all the Intensity parameters in Normals and Dysarthric group.

\+ Significance at 0.05 level - Not Significant

	NORMAL GROUP						DYSARTHRIC GROUP					
INTENSITY	MEAN STANDARD DEVIATION						MEAN STANDARD DEVIATION					
PARAMETERS	/a/	Si	/i/	Si	/u/	Si	/a/	Si	/i/	Si	/u/	Si
Mean Intensity	49.9 (4.4917)	+	43.03 (2.37)	+	47.74 (7.85)	+	37.76 (3.73)	+	38.21 (3.03)	+	42.67 (1.44)	+
Extent of Fluctuations	0.00 (0.00)	+	0.00 (0.00)	+	0.148 (0.4619)	+	2.81 (0.28)	+	2.11 (4.28)	+	4.00 (3.16)	+
Speed of Fluctuations	0.00 (0.00)	+	0.00 (0.00)	+	0.00 (0.00)	+	3.47 (4.70)	+	1.37 (3.73)	+	2.90 (4.49)	+
Intensity Range	5.27 (4.49)	+	6.59 (2.32)	-	15.75 (7.85)	-	15.59 (1.075)	+	11.38 (1.64)	-	11.93 (2.76)	-

Table 2B : Depicts mean , S.D and Significance values for all the intensity parameters in Parkinsons disease, ALS, Wilsons disease patients.

\+ Significance at 0.05 level - Not Significant

	PARKINSONS DISEASE						AMYOTROPIC LATERAL SCLEROSIS						WILSONS DISEASE		
INTENSITY	MEAN STANDARD DEVIATION						MEAN STANDARD DEVIATION						MEAN		
Parameters	/a/	si	/i/	si	/u/	si	/a/	si	/i/	si	/u/	si	/a/	/i/	/u/
Mean intensity (in dB)	34.13 (4.59)	+	34.36 (6.99)	-	39.61 (3.16)	+	45.37 (4.48)	+	42.93 (3.03)	-	46.76 (1.44)	+	33.47	40.37	43.72
Extent of Fluctuations	4.01 (0.44)	+	3.87 (0.46)	+	5.15 (0.677)		0.00 (0.00)	+	0.3267 (0.28)	+	4.28 (0.42)		2.87	0.00	0.00
Speed of Fluctuations	5.69 (120)	+	2.13 (1.12)	-	3.95 (101)	-	0.25 (0.26)	+	0.30 (0.28)	-	0.84 (0.68)	-	3.27	1.23	3.92
Intensity Range	22.75 (164)	+	15.37 (1.07)		16.61 (2.7)		2.37 (0.54)	+	2.72 (0.33)		3.76 (1.61)		20.75	16.75	14.27

d) Intensity range for /a/ only.

i) Mean intensity in phonation

Table 2A and Graph 9 reveal that the mean intensity for /a/ = 37.76 dB; *Pd* = 38.21 dB; /u/ = 42.67 dB in dysarthric group which is comparatively less than the normal group, whose mean intensity values for /a/, /i/, /u/ are 49.9 dB, 43.03 dB, 47.74 dB respectively. This difference between the two groups were statistically significant for all the vowels /a/, /i/, /u/ as found using Wilcoxons Matched Pair Sign Rank Test.

Table 2B and Graph 10 reveal the mean intensity for /a/,/i/, /u/ in Parkinsons disease, Amyotropic Lateral Sclerosis and Wilsons disease as follows

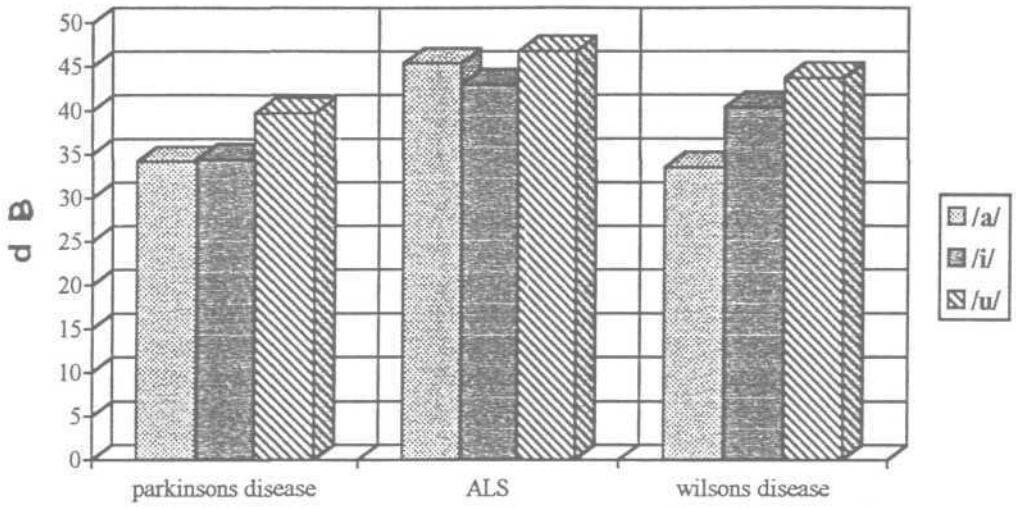
Parkinsons disease - /a/ - 34.13 dB ; /i/ - 34.36 dB ;/u/ - 39.61 dB ;
Amyotropic lateral sclerosis - /a/ - 45.37 dB ; /i/ - 42.93 dB ; /u/ - 46.76 dB ;
Wilsons disease - /a/ - 33.47 dB ; /i/ - 40.37 dB ; /u/ - 43.72 dB ;

From the above data it can be seen that there is difference within the dysarthric groups, However this difference was significant only for vowels /a/ and *lul* at 0.05 level in Parkinsons and Amyotropic Lateral Sclerosis and not when Wilsons disease was compared with the other two types of dysarthrias.

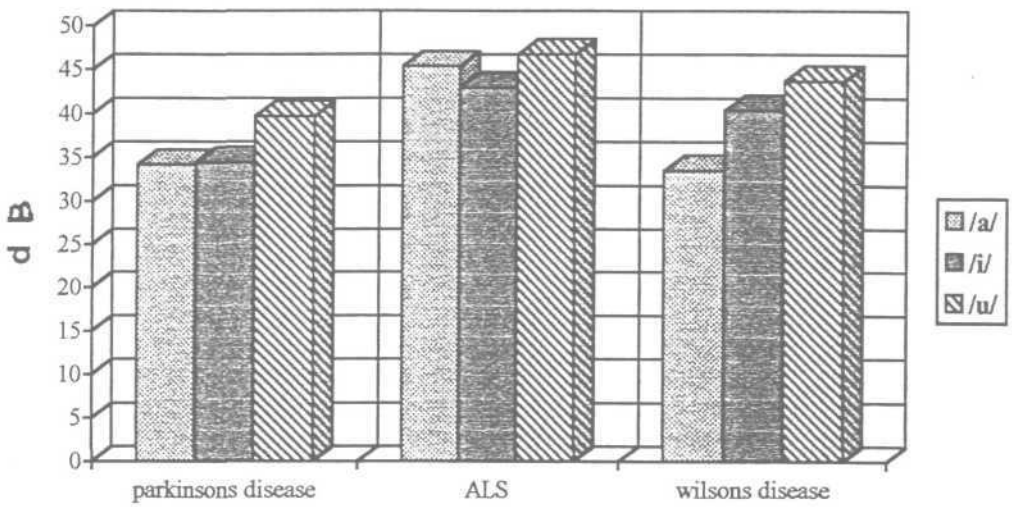
Extent of Fluctuation in Intensity

Inspection of Table 2A and Graph 11 reveals that the mean extent of fluctuation values in intensity for /a/ = 2.81; /i/ = 2.11; /u/ = 4.00 in dysarthric group was comparatively more than the normal group whose mean extent of fluctuations in intensity for /a/, /i/ were 0.00 and for *lul* = 0.148 respectively. This difference between the two groups was statistically significant for all the vowels /a/, /i/, /u/, as found using Wilcoxons Matched Pair Sign Rank Test.

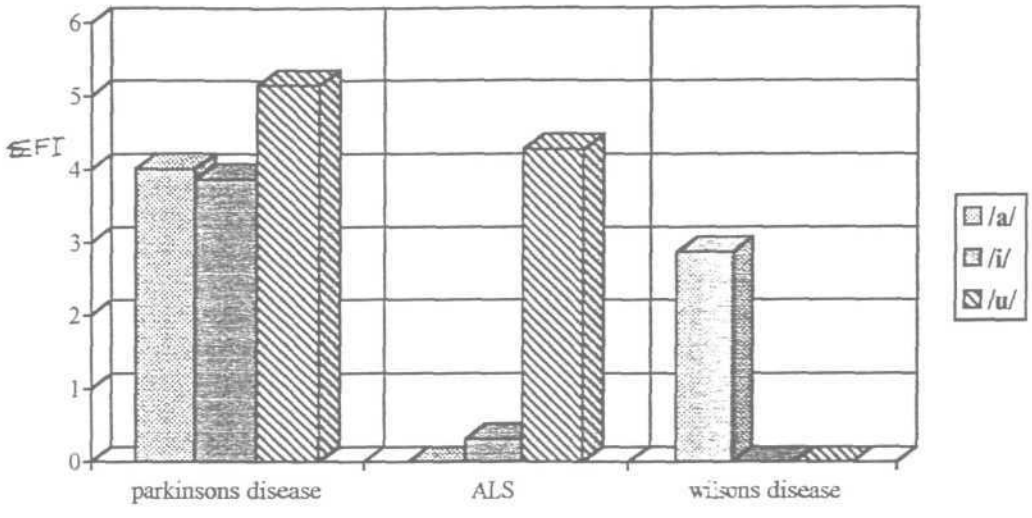
Graph 9 : Shows mean intensity (in dB) in Normal and Dysarthric group



Graph 10 : Mean intensity (In dB) in Parkinsons disease, ALS, Wilsons disease



Graph 11 : Shows mean extent of fluctuations of intensity (EFI) in Normal and Dysarthric group



Graph 12 : Mean extent of fluctuations of intensity (EFI) in Parkinsons disease, ALS, Wilsons disease

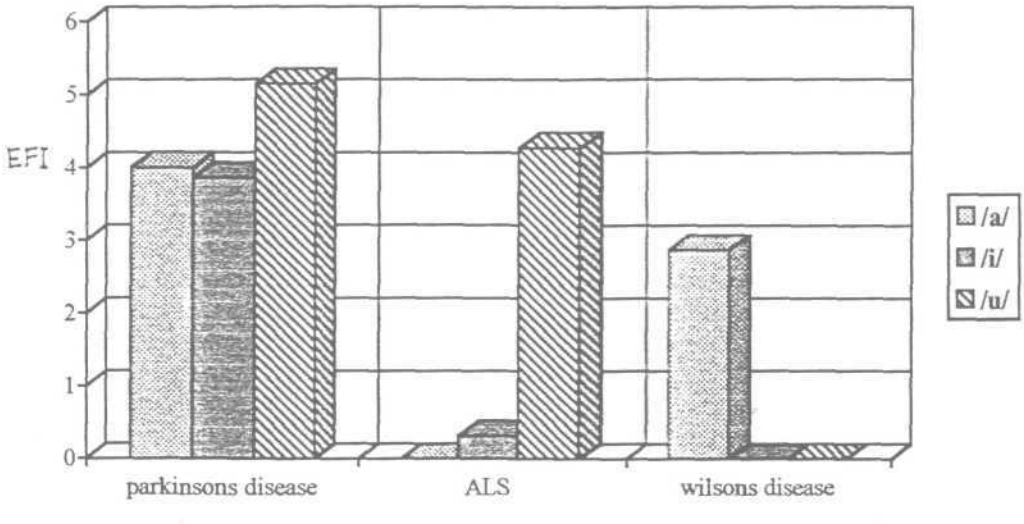


Table 2B and Graph 12 reveal that the mean extent of fluctuations in intensity for /a/, /i/, /u/ in Parkinsons, Amyotropic Lateral Sclerosis and Wilsons disease were as follows: /a/-4.01 ; /i/ - 3.87 ; /u/ - 5.15,/a/-0.00 ; /i/ - 0.3267; /u/ - 4.28, /a/-2.87 ; /i/- 0.00 ; /u/ - 0.00 respectively.

From Table 2B and Graph 12 it can be seen that there is difference within the dysarthric group. However the differences seen were significant for vowels /a/ and /i/, at 0.05 level between Parkinsons disease and Amyotropic Lateral Sclerosis i.e. greater in Parkinsons than in Amyotropic lateral sclerosis cases and no significant difference was seen when Wilsons disease was compared with the other two types of dysarthrias.

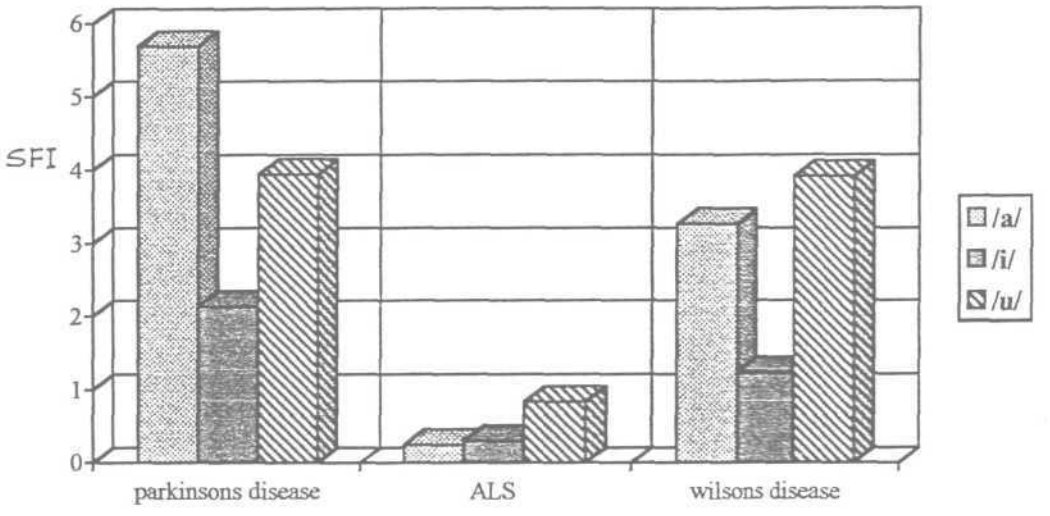
Speed Of Fluctuations In Intensity

Table 2A and Graph 13 reveal that the mean speed of fluctuations in intensity for /a/, =3.47, /i/ = 1.37; /u/ = 2.90 in dysarthric group which was comparatively more than that of the normal group whose mean speed of fluctuations in intensity for /a/, /i/ and /u/ were 0.00. The difference seen between the groups was statistically significant for all vowels at 0.05 level.

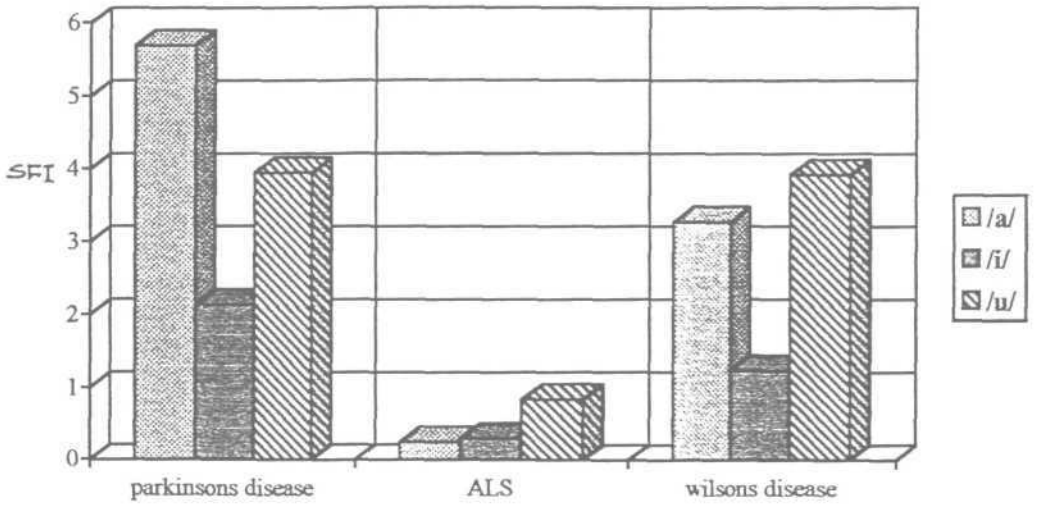
Further inspection of 2B and Graph 14 reveal that the mean speed of fluctuations in intensity for /a/, /i/, /u/ in Parkinsons, Amyotropic lateral sclerosis and Wilsons disease were as follows, /a/ -5.69; /i/ - 2.131 ; /u/ - 3.95, /a/ - 0.25 ; /i/ - 0.30 ; /u/ - 0.84, /a/ -3.27; /i/ -1.23 ; /u/ - 3.92 respectively.

From the above given data it can be inferred that there was difference within the dysarthric group, however this difference was significant for vowel /a/ only at 0.05 level for Parkinsons disease and Amyotropic lateral sclerosis following the order

Graph 13 : Shows mean speed of fluctuations of intensity (SFI) in Normal and Dysarthric group



Graph 14 : Mean speed of fluctuations of intensity (SFI) in Parkinsons disease, ALS, Wilsons disease



Parkinsons > Amyotropic Lateral Sclerosis. However ,no significance was seen when Wilsons disease was compared with the other two types of dysarthrias.

Intensity Range

Inspection of Table 2 A and Graph 15 reveal that the mean intensity range for /a/ - 5.27; /i/ - 6.59; *IvJ* -15.75 in normals which is comparatively less than that of the dysarthric group whose, mean intensity range values for /a/, /i/, and /u/ were 15.59; 11.38; 11.93 respectively. This difference seen between the groups was statistically significant for vowel /a/ only at 0.05 level.

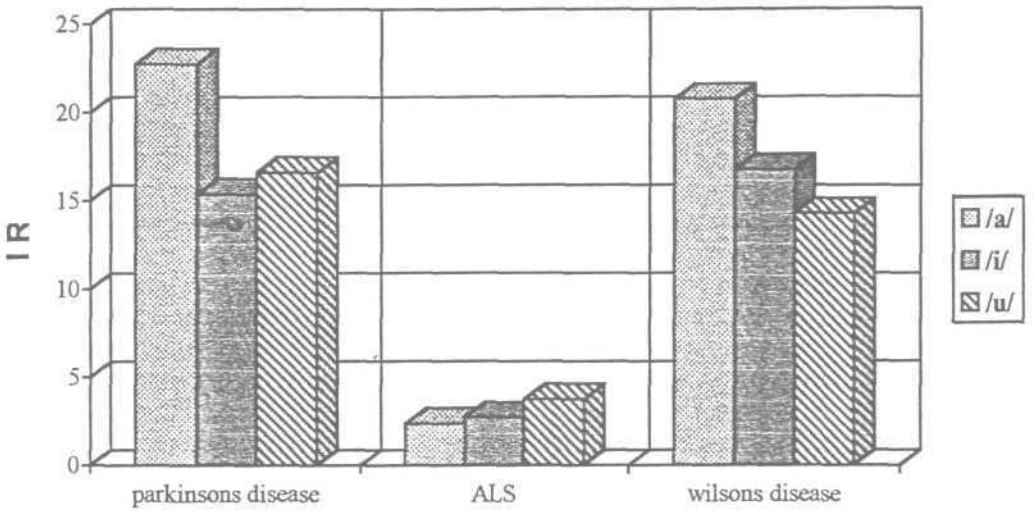
Furthermore inspection of Table 2B and Graph 16 reveal that the mean intensity range values for /a/,/i/ /u/ in Parkinsons disease: /a/ - 22.75 ; /i/ - 15.37 ; /u/- 16.61, Amyotropic lateral sclerosis: /a/ - 2.37 ;/i/ - 2.72 ; /u/ - *3.76,* and Wilsons disease : /a/ - 20.75 , /i/ - 16.75 ; /u/ - *14.27* respectively.

From these values it can be inferred that there was difference within the dysarthric group. However this difference was significant for vowels /a/ only at 0.05 level between Parkinsons disease and Amyotropic Lateral Sclerosis i.e. greater in Parkinsons disease than in Amyotropic Lateral Sclerosis cases. However it was not significant when Wilsons disease was compared with the other type s of dysarthrias.

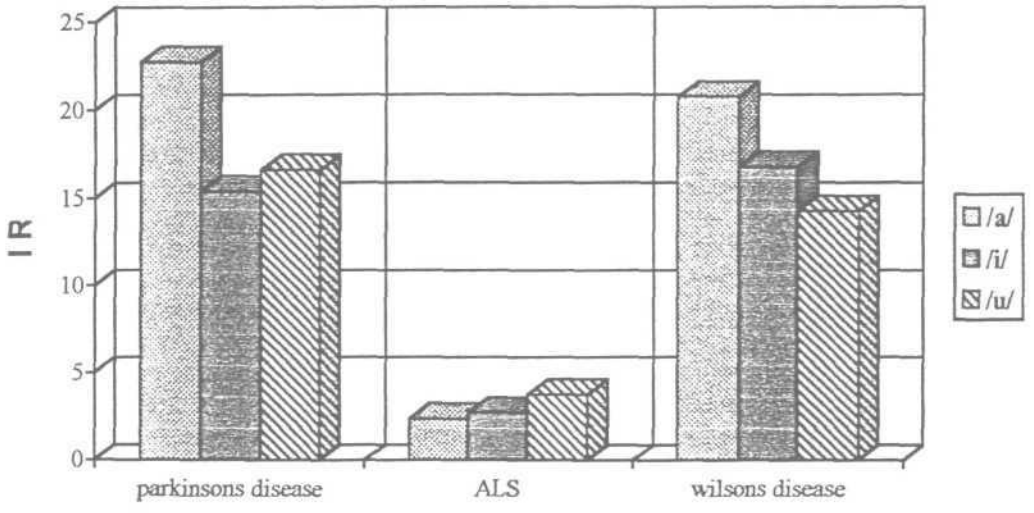
The findings of the present study are in agreement with earlier studies reported in literature that dysarthrics as a group differ significantly in terms of intensity parameters from normals of the same age group (Leuschel and Docherty 1996, King **et.** al. 1993, 1994). These differences may be attributed to the lack of control of the vocal system due to impaired neuro muscular control.

Further, the difference seen within the three dysarthric conditions may be attributed either to differential neural system involvement and to factors related to

Graph 15 : Shows mean intensity range (IR) in Normal and Dysarthric group



Graph 16 : Mean intensity range (IR) in Parkinsons disease, ALS, Wilsons disease



subjects age, sex, etc. However there was significant difference only between the subjects with Parkinsons and Amyotropic Lateral Sclerosis, but not with Wilsons disease, possibly owing to the fact that only a single subject was taken up in the category.

Thus the hypothesis stating

i) There is no significant difference between the dysarthrics and normals in terms of intensity parameters is rejected

ii) There is no significant difference between the three types of dysarthric conditions i.e.Parkinsons disease, Amyotropic Lateral Sclerosis and Wilsons disease in terms of intensity parameters is also rejected

TEMPORAL PARAMETERS

Word Duration

Table 3 a And Graph 17 Reveal The Mean word duration in the dysarthric group as 706.29 msec, and was comparatively more than in the normal group, whose mean word duration was 635.02 msec.

The difference seen between the two groups was statistically significant at 0.05 level as found using Wilcoxons Matched Pair Sign Rank Test. Table 3B and Graph 18 reveal the mean word duration in Parkinsons disease as 688.92 msec, Amyotropic Lateral Sclerosis as 757.77msec, and Wilsons disease (657 m sec). Further, it can be seen that there was difference within the dysarthric groups. However the differences were significant only between Parkinsons disease and Amyotropic Lateral Sclerosis at 0.05 level and not between Wilsons disease and the other two groups.

Table 3A : Depicts mean , SD values and Significance for all the Temporal parameters in Normals and Dysarthric group.

\+ Significant at 0.05 level - Not Significant

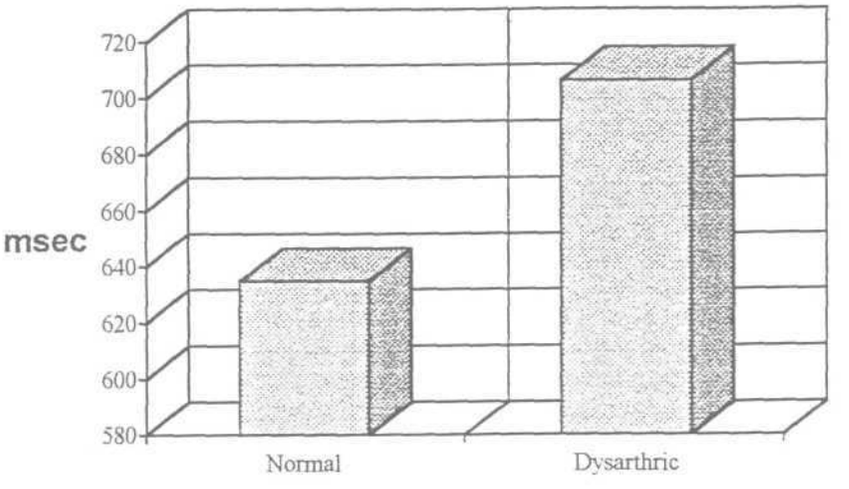
	NORMAL GROUP			DYSARTHRIC GROUP		
PARAMETERS	MEAN	SI	STANDARD DEVIATION	MEAN	SI	STANDARD DEVIATION
Word Duration	635.02	+	89.03	706.29	+	93.72
Vowel Duration						
/a/	96.30	+	11.30	**161.31**	+	40.40
/i/	120.0	+	15.19	184.46	+	42.54
/u/	117.76	+	28.37	195.73	+	73.72
Burst Duration						
/p/	16.58	+	1.03	4.66	+	1.232
/t/	15.62	-	2.04	14.98	-	3.022
/k/	15.94	+	2.86	8.06	+	5.03
Closure Duration						
/p/	88.47	-	16.24	58.69	-	10.23
/t/	8905	-	21.05	67.17	-	9.72
/k/	133.56	+	24.49	46.85	+	11.72
Consonant Duration						
/p/	53.2	-	11.72	35.42	-	40.72
/t/	63.2	-	8.72	63.8	-	90.82
/k/	64.2	-	12.42	32.8	-	40.84
Voice Onset Time						
/P/	20.20	-	4.60	14.02		3.27
/t/	**11.80**	+	0.83	21.26	+	8.86
/k/	24 40	+	17.37	14.85	+	6.72

Table 3B : Depicts mean , S.D and Significance values fui all the Temporal parameters **in** Parkinsons disease, ALS, **Wilsons disease patients.**

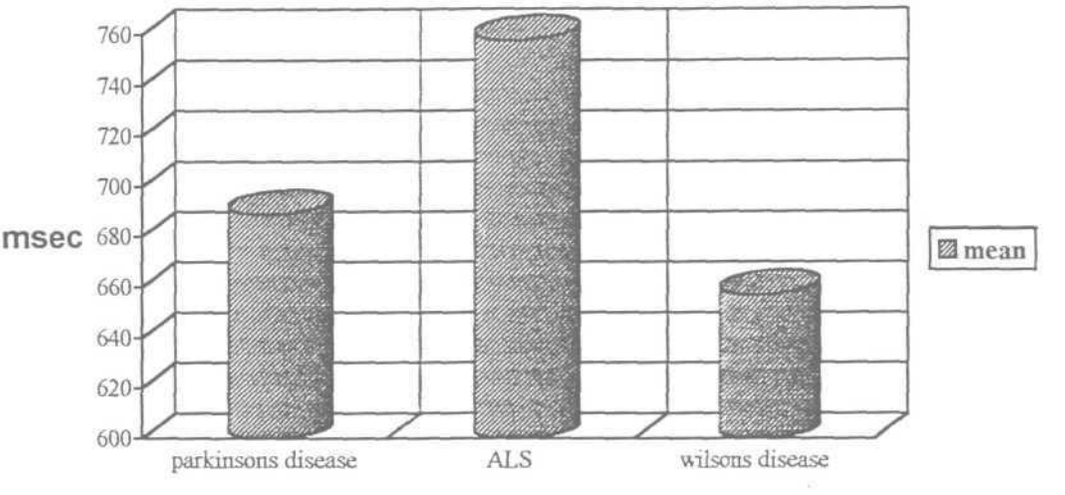
\+ Significance at 0.05 level - Not Significant

	PARKINSONS DISEASE			AMYOTROPIC LATERAL SCLEROSIS			WILSONS DISEASE
PARAMETERS	MEAN	SI	STANDARD DEVIATION	MEAN	SI	STANDARD DEVIATION	MEAN
Word Duration (msec)	688.92	+	90.72	757.77	+	117.528	657
Vowel Duration (msec)							
/a/	155.58	+	34.32	174.58	+	40.72	152.00
/i/	184.94	+	42.32	183.72	+	60.84	184.52
/u/	202.41	+	66.74	192.72	+	50.72	181.73
Burst Duration							
/p/	4.32	+	3.87	0.00	+	0.00	15
/t/	14.083	-	1.0104	13.72	-	4.87	20.25
/k/	10.33	+	8.98	0.00	+	0.00	17.42
Closure Duration							
/p/	50.85	-	808	55.31	-	8.22	89
/t/	59.73	-	9.72	63.44	-	8.72	97
/k/	59.33	-	10.72	35.17	-	7.35	32.8
Consonant Duration (msec)							
/p/	31.85	+	12.42	0.00	+	0.00	117
/t/	40.32	-	15.72	67.42	-	16.68	127
/k/	44.42	+	18.83	0.00	+	0.00	64
Voice Onset Time							
/p/	18.72	+	3.27	0.00	+	0.00	28
/t/	22.72	-	8.86	14.72	-	11.82	30
/k/	19.2	+	6.72	0.00	+	0.00	31.5

Graph 17 : Shows mean Word duration (m sec) in Normal and Dysarthric group



Graph 18 : Mean Word Duration (m sec) in Parkinson's, ALS, Wilsons disease



Vowel Duration

Inspection of Table 3 A and Graph 19 reveals that the mean vowel duration for /a/, /i/, /u/ in dysarthric group as 161.31 msec; 184.46 msec.; 195.73msec. respectively and was comparatively more than values of the normal group i.e. 96.30 msec for /a/; 120.0 msec, for /i/; and 117.76 msec, for /u/ respectively.

The difference seen between the groups was statistically significant for all the vowels /a/, /i/, /u/ as found using Wilcoxons Matched Pair Sign Rank Test.

Table 3B and Graph 20 reveal the mean vowel duration for /a/, *hJ,* /u/ in Parkinsons disease, Amyotropic lateral sclerosis and Wilsons disease as

/a/- 155.58 m sec.; /i/ -184.94 m sec.; /u/- 202.41 msec ,
/a/-174.58m sec.; /i/-183.72 m sec;/u/-192.72 msec . and
/a/- 152.0 msec.; /i/- 184.52 m sec.;/u/-181.73 msec respectively

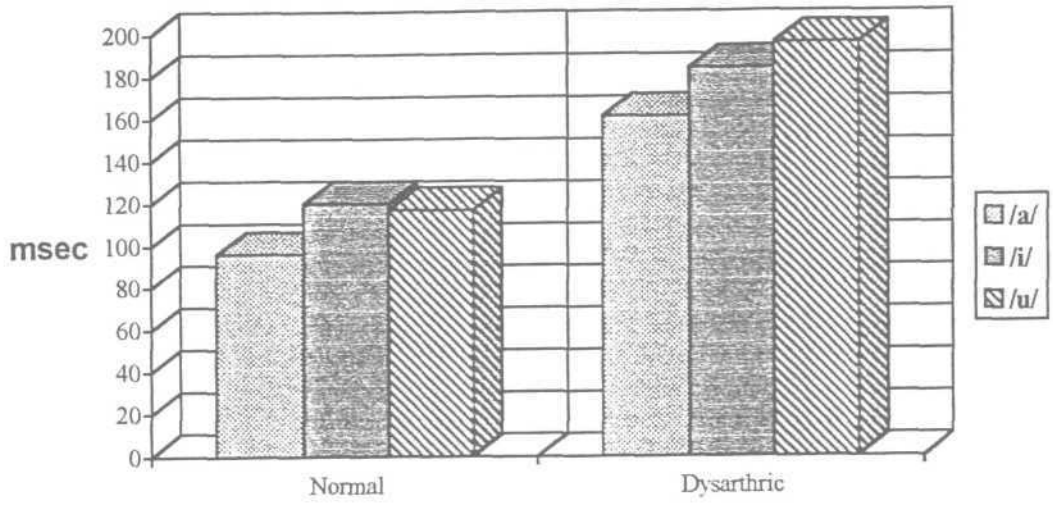
From the above data it may be seen that there is difference within the dysarthric groups. However this difference was significant for all vowels at 0.05 level only between Parkinsons and Amyotropic Lateral Sclerosis but not with Wilsons disease and Amyotropic lateral sclerosis or with Parkinsons disease.

Burst Duration

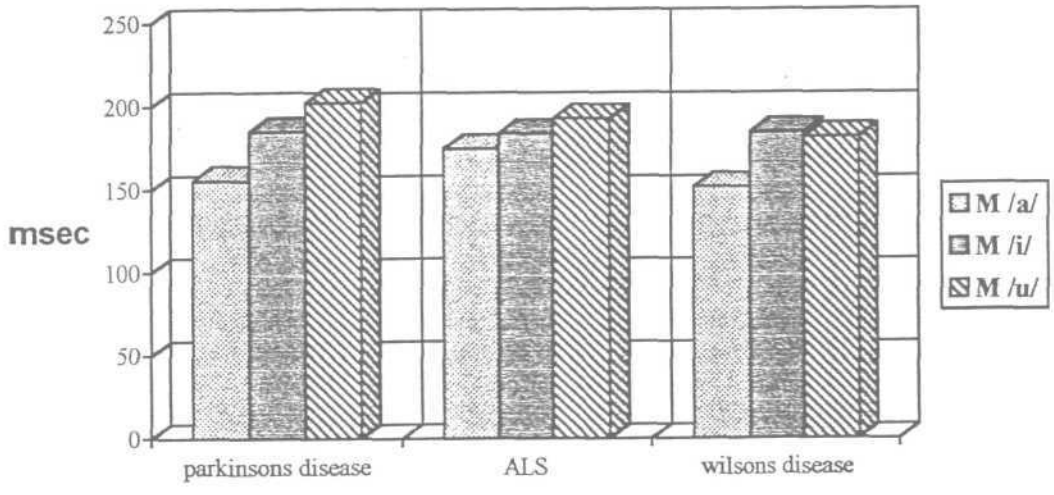
Table 3 A and Graph 21 reveal that the mean burst duration values for /p/, *IxJ, Dd* in dysarthric group as 4.66 msec.; 14.98 msec.; 8.06 msec, respectively which is comparatively less than the normal group which had mean burst duration values for /p/, /t/, /k/ are 16.58 msec; 15.62 msec; 15.94 msec respectively. The difference seen between the two groups was statistically significant for consonants /p/ & /k/ but not for /t/ as per Wilcoxons Matched Pair Sign Rank Test.

Table 3 B and Graph 22 reveals that the mean burst duration values within the dysarthrics were found to be different i.e. in Parkinsons disease /p/ - 4.32 m sec.;/t/ -

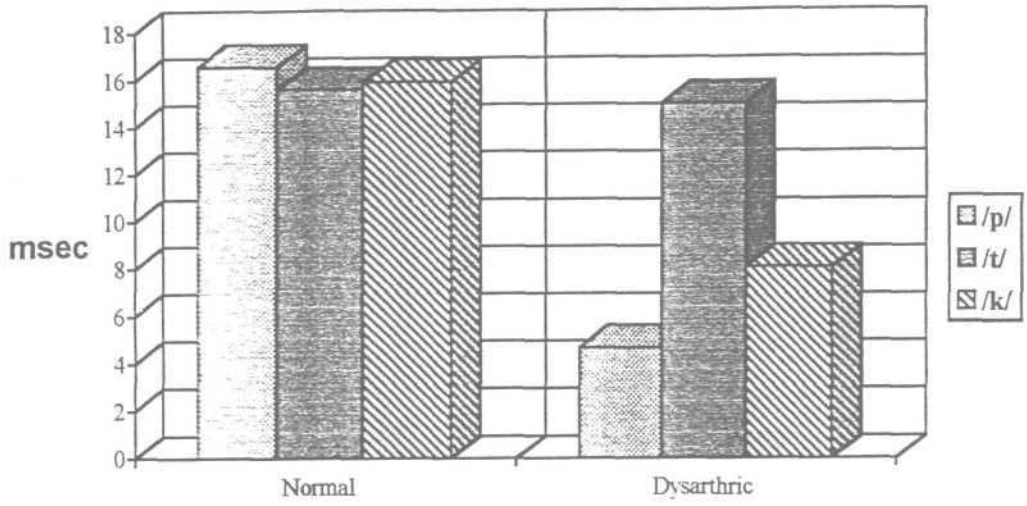
Graph 19 : Mean vowel duration (m sec) in normal and dysarthric group



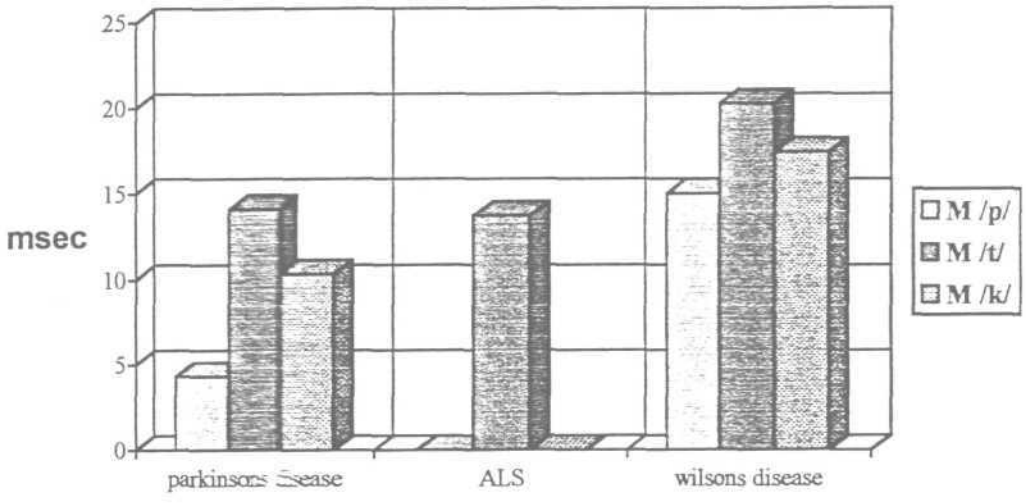
Graph 20 : Mean Vowel Duration (m sec) in Parkinson's, ALS, Wilsons disease



Graph 21 : Mean Burst duration (m sec) in normal and dysarthric group



Graph 22 : Mean Burst Duration (m sec) in Parkinson's, ALS, Wilsons disease



14.083 m sec; /k/-10.33 m sec, Amyotropic lateral sclerosis : /p/ - 0.00 m sec; /t/ - 13.72 m sec. ; */k/* - 0.00 m sec and **in** Wilsons disease :/p/-15.00 m sec; /t/ - 20.25 m sec.; /k/ -17.42 m sec respectively.

The difference seen within the group was statistically significant only for consonants /p/ & /k/ ar 0.05 level between Parkinsons and Amyotropic Lateral Sclerosis and not between Wilsons disease and Parkinsons disease, and between Amyotropic Lateral Sclerosis and Wilsons disease.

Closure Duration

Table 3 A and Graph 23 reveal that the mean closure duration values for /p/, /t/, */k/* in dysarthric groups as 58.69 msec; 67.17 msec; 46.85 msec, respectively and was less compared to the normal group whose mean values of closure duration for consonants /p/, /t/, /k, were 88.47 msec; 89.05 msec; 133.56 msec, respectively. This difference seen between the two groups was statistically significant only for consonant *Ikl* at 0.05 level.

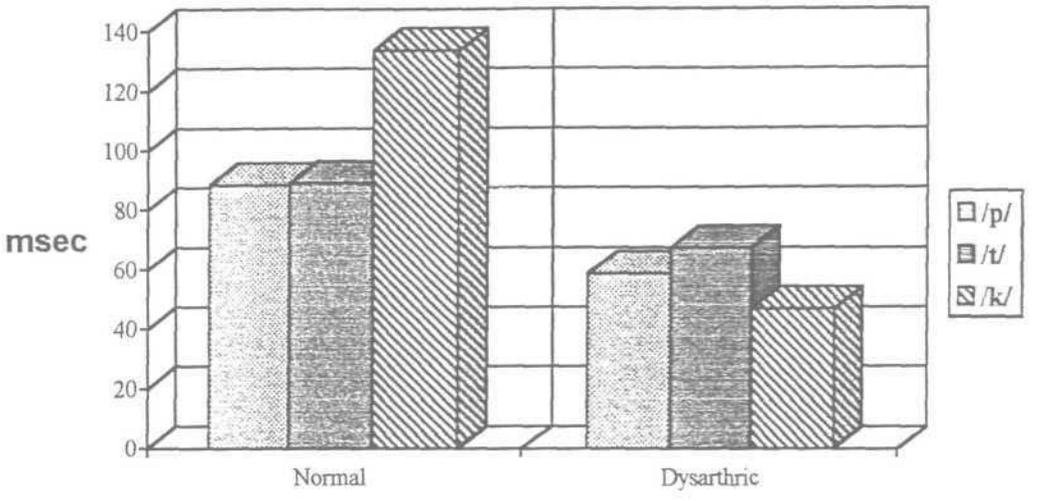
Inspection of Table 3B reveals that the mean closure duration values for the three dysarthrics Parkinsons ,Amyotropic Lateral Sclerosis, Wilson's disease as : /p/ - 50.85 msec; /t/ - 59 "3 msec ; *Ikl* -59.33 msec; and /p/ - 55.31 msec; /t/ - 63.44 msec; /k/ -35.17 msec, /p/ - 89.00 msec; /t/ - 97.00 msec; /k/ -32.80 msec, respectively.

However this difference within the dysarthric group was not statistically significant at 0.05 level.

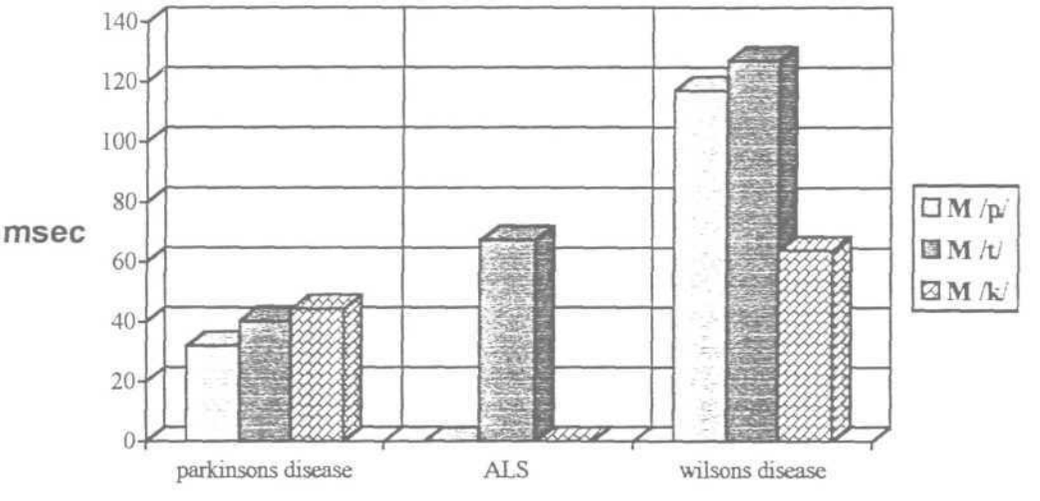
Consonant Duration

Inspection of Table 3 A reveal that the mean consonant duration for the normal and dysarthric groups. In normals it was found to be 53.2 msec for /p/; 63.2

Graph 23 : Mean Closure duration (m sec) in normal and dysarthric group



Graph 24 : Consonant duration (m sec) in Parkinson's, ALS, Wilsons disease



msec, for /t/; 64.2 msec, for /k/ whereas the dysarthric group had , 35.42 msec, for /p/; 63.8 msec, for /t/ ; 32.8 msec for /k/ respectively. However the difference seen between the two groups was not statistically significant at 0.05 level.

Table 3B and Graph 24 reveal that there was difference within the dysarthric groups in terms of consonant duration. The values of the consonant duration in Parkinsons disease for : /p/ was 31.85 msec ; /t/ was 40.32 msec ; /k/ was 44.42 msec, where as Amyotropic lateral sclerosis for : /p/ was 0.00 msec; /t/ was 67.42 msec ; /k/ was 0.00msec,similar Wilsons disease for : /p/ was 117.00 msec; *Ixl* was 127.00 msec; /k/ was 64.0 msec.

There was a difference within the dysarthric groups and the difference was found to be statistically significant for consonants /p/ and *fkl* at 0.05 level between Parkinsons and Amyotropic lateral sclerosis but not between Wilsons disease and Parkinsons and between Amyotropic Lateral Sclerosis and Wilsons disease.

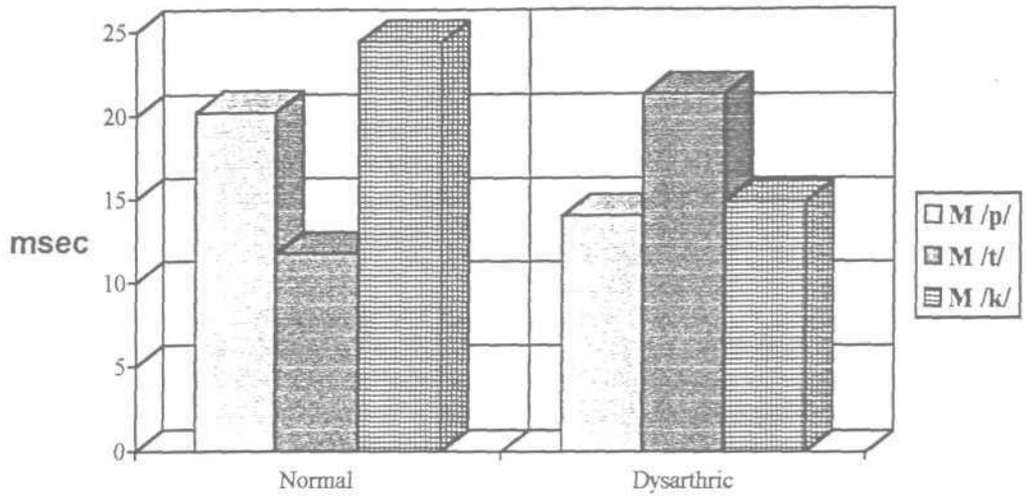
Voice Onset Time

Table 3 A and Graph 25 reveal the mean VOT for /P/ as 14.02 msec, for *Ixl* as 21.26 msec; and for /k/ as 14.85 msec, In dysarthric groups and is comparatively less than in the normal group which had showed the mean VOT for /p/, /t/, and /k/ as 20.20 msec; 11.80 msec; 24.40 msec respectively. The difference between the groups was statistically significant at 0.05 level consonants /t/& /k/ but not for /p/ as found using Wilcoxons Matched Pair Sign Rank Test.

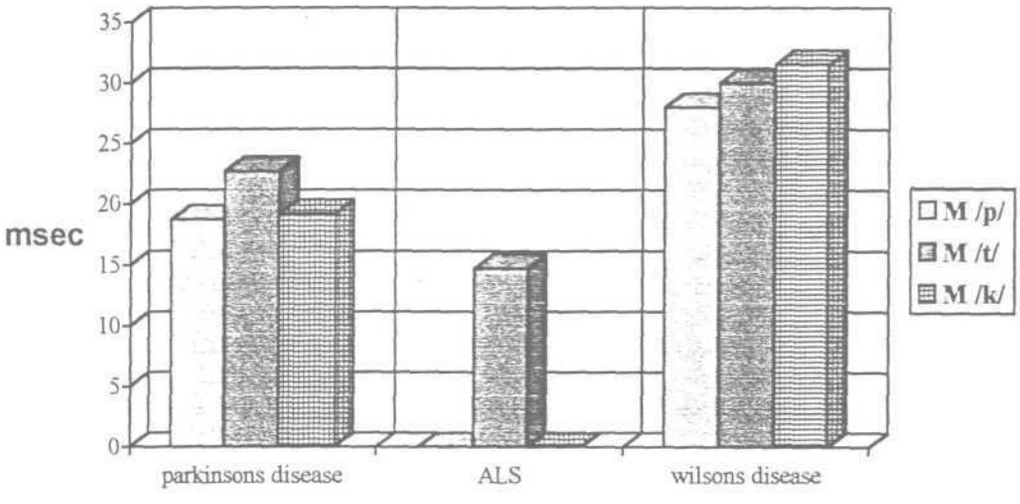
Table 3B and Graph 26 reveal the mean VOT values for /p/, /t/, */k/* in Parkinsons disease, Amyotropic lateral sclerosis and Wilsons disease as follows:

: /p/ - 18.72 msec; /t/ -22.72 msec; /k/ - 19.20 msec,
: /p/ - 0.00 msec; /t/ - 14.72 msec, /k/ - 0.00 msec and
: /p/ - 28.00 m sec; /t/ - 30.00 m sec, /k/ - 31.50 m sec. respectively

Graph 25 : Vot (m sec) for /p/, /t/, /k/, in Normal and Dysarthric group



Graph 26 : Vot (m sec) for /p/, /t/, /k/, in Parkinson's, ALS and Wilsons disease



From the above given data it may be seen that there was difference within the dysarthric groups, however the differences were statistically significant at 0.05 level for consonants /t/ *& /k/* between parkinsons disease and Amyotropic lateral sclerosis only but not between Parkinsons disease & Wilsons disease and between Amyotropic lateral sclerosis and Wilsons disease.

Thus the hypothesis stating that

1) there is no significant difference between dysarthrics and normal subjects in terms of temporal parameters is rejected.
2) there is no significant difference between the three types of dysarthric subjects: Parkinsons disease, Amyotropic lateral sclerosis and Wilsons disease in terms of temporal parameters is also rejected.

However the first hypothesis was accepted in terms of consonant duration only and the second in terms of closure duration.

The findings of the present study are in consonance with the earlier studies, which had reported that dysarthrics as a group differ significantly in terms of various temporal parameters from normals of the same age group (Caruso et. al. 1987; forrest et. al. 1997; Hertrich et. al. 1995). These differences are related to the impaired neuro muscular control of the speech production system in dysarthria.

Further, the differences seen with in the three dysarthric conditions can be attributed to the differential neural subsystem involvement and disease progression etc. However, the significant difference was seen only between Parkinsons and Amyotropic Lateral Sclerosis and not between Wilson disease and other two groups. The reason for which may be only a single subject was compared in this category, in present study.

In summary, the present study adds to the literature by supporting the findings of earlier studies conducted to delineate the acoustic and temporal correlates of dysarthric speech. From the present study it can be understood that there is a significant difference between the dysarthric and normal groups in terms of the following acoustic and temporal parameters.

a) Mean fundamental frequency in phonation for /a/, /i/, and /u/
b) Extent of fluctuations in frequency for /a/, /u/ only
c) Speed of the fluctuations in frequency for /a/, /i/, and /u/
d) First formant frequencies (Fl) for vowels/a/,/i/, and/u/
e) Second formant frequency (F2) for vowels /a/ & /u/
f) Mean intensity for /a/, /i/, /u/
g) Extent of fluctuations in intensity for /a/, /i/, and /u/.
h) Speed of fluctuations in intensity for /a/, /i/, and /u/
i) Intensity range for /a/ only.
j) Word duration
k) Vowel duration
1) Burst duration for consonants /p/ & /k/.
m) Closure duration for consonants /k/ only
n) Voice onset time for consonants /t/ & /k/.

It was also seen that this difference exists even within the dysarthric group. Thus rejecting the hypothesis that

1. There is no significant difference in the acoustical and temporal parameters between dysarthrics and normal subjects
2. There is no significant difference between the three types of dysarthric subjects : Parkinson's disease, Amyotropic Lateral Sclerosis and Wilsons disease in terms of comparable acoustic and temporal parameters.

DESCRIPTION OF DYSARTHRIC SPEECH

1. PARKINSONS DISEASE

Three male's ages ranging between 55-72 years were taken up who were diagnosed as having Parkinson's disease by a neurologist and a speech pathologist diagnosed them as having dysarthria.

PERCEPTUAL ANALYSIS

The most deviant speech dimensions as judged perceptually were :

- Low pitch level
- Mono pitch
- Mono Loudness
- Imprecise Consonants
- Distorted Vowels
- Hoarseness
- In-appropriate Silences
- Reduced stress
- Mild abnormality in (Overall Intelligibility)

ACOUSTIC ANALYSIS

Acoustic analysis revealed that the following parameters in Parkinsons disease were different from their normal counterparts.

a) FREQUENCY PARAMETERS

Inspection of Table 4 A reveals the following

1) Significant reduction in mean fundamental frequency for vowels / a / & / i / only
2) Significant increase in mean extent of fluctuations in frequency for vowels / a / & / i / only

Table 4A : Depicts Mean ,SD values, Significance for all the frequency parameters in Parkinsons disease and Normals .

\+ Significance at 0.05 level Not Significant

	PARKINSONS DISEASE (N=3)						**NORMAL GROUP (N=3)**					
Frequency	MEAN (STANDARD DEVIATION)						MEAN (STANDARD DEVIATION)					
Parameters	**/a/**	**Si**	/i/	**Si**	/u/	**Si**	**/a/**	**Si**	***/i/***	**Si**	***/u/***	**Si**
Fundamental Frequency	156.24 (14.55)	+	167.17 (35.03)	+	*172.73* (37.58)	-	160.24 (15.72)	+	190.24 (33.02)	+	174.12 (38.58)	-
Extent of Fluctuations	8.72 (2.93)		6.0167 (1.03)	+	6.94 (4.59)	-	2.76 (0.68)	+	0.4533 (0.1523)	+	2.22 (1.02)	-
Speed of Fluctuations	28.153 (28.22)	-	11.48 (10.14)	+	6.53 (2.13)	-	0.74 (0.44)	-	1.44 (0.77)	+	1.57 (0.91)	-
Frequency Range	152.27 (98.64)	-	74.48 (98.62)	+	13.74 (6.55)	+	62.88 (60.2)	-	127.95 (68.47)	+	44.6067 (35.6)	+
Formanl Frequencies												
Fl	710.36 (88.89)	+	386.11 (12.16)	+	365.76 (8.37)	+	720.266 (55.94)	+	323.6 (60.22)	+	326.98 (24.94)	+
F2	1520.3 (67.13)	+	1781.18 (54.14)	-	1387.06 (50.7)	+	1354.9 (58.9)	+	2132.6 (12.36)	-	1088.59 (54.3)	+
F3	2836.66 (81.8)	-	2739.43 (66.33)	-	2783.7 (108.89)	-	2463.76 (64.39)	-	2868.47 (78.67)	-	2374.76 (117.32)	-
Band Widths												
Bl	324.4 (10.00)	-	83.8 (19.97)		133.03 (18.57)	-	167.92 (9.8)	-	146.85 (16.8)	-	262.5 (116.72)	-
B2	388.25 (38.24)	-	23 1.65 (40.3)	-	283.83 (48.77)	-	276.05 (64.05)	-	283.65 (57.34)	-	394.46 (59.74)	-
B3	688.63 (58.77)		416.85 (6.23)		385.4 (17.669)		460.03 (69.72)	-	407.13 (107.83)	-	513.72 (139.49)	-

Table 4B : Depicts mean , SD values and Significance for all the Intensity parameters in Parkinsons and Normal subjects.

\+ Significance at 0.05 level - Not Significant

	PARKINSONS DISEASE GROUP (N=3)						NORMAL GROUP (N=3)					
INTENSITY	MEAN STANDARD DEVIATION						MEAN STANDARD DEVIATION					
PARAMETERS	/a/	Si	/i/		/u/	Si	/a/	Si	/i/	Si	/u/	Si
Mean Intensity	34.13 (4.59)		34.36 (6.99)		39.61 (3.16)	+	45.90 (3.73)	+	42.96 (2.08)	+	42.66 (2.08)	+
Extent of Fluctuations	4.01 (0.44)	+	3.87 (0.46)	+	5.15 (0.67)	+	0.00 (0.00)	+	0.00 (0.00)	+	0.00 (0.00)	+
Speed of Fluctuations	5.69 (1.20)	+	2.13 (1.12)	+	3.95 (1.01)	+	0.00 (0.00)	+	0.00 (0.00)	+	0.00 (0.00)	+
Intensity Range	22.75 (1.64)		15.37 (1.67)	-	16.61 (2.7)	-	2.59 (0.38)	-	5.4 (1.06)	-	3.51 (1.12)	-

Table 4C : Depicts mean , SD values and Significance for all the Temporal parameters in Parkinsons and Normal subjects.

\+ Significant at 0.05 level - Not Significant

	PARKINSONS DISEASE (N=3)			NORMAL GROUP (N=3)		
PARAMETERS	MEAN	SI	STANDARD DEVIATION	MEAN	SI	STANDARD DEVIATION
Word Duration (msec)	688.92	+	90.72	604.97	+	98.39
Vowel Duralion (msec)						
/a/	**155.58**	+	34.32	**106.25**	+	27.76
/i/	184.94	+	42.32	90.25	+	12.76
/u/	**202.41**	-	**66.74**	**110.41**	-	72.76
Hurst Duration (msec)						
/p/	4.32	+	3.87	8.73	+	1.006
/t/	14.083	+	1.01	15.33	+	0.577
/k/	10.33	+	8.78	15.71	+	11.15
Closure Duration (msec)						
/p/	50.85	+	8.08	80.92	+	8.07
/t/	59.73	-	9.72	90.83	-	11.72
/k/	59.33	-	10.72	80.73	-	12.72
Consonant Duration (msec)						
/p/	31.85	+	12.42	35.85	+	14.42
/t/	40.32	+	15.72	56.72	+	12.42
/k/	44.42	-	18.83	45.82	-	11.22
Voice Onset Time (msec)						
/p/	18.72	+	3.27	12.72	+	4.27
/t/	22.72	+	8.86	14.82	+	5.27
/k/	19.2	-	6.72	18.82	-	6.72

3) Significant increase in mean speed of fluctuations in frequency for vowel / i / only

4) Significant reduction in mean frequency range for vowels / i / & / u / only

5) Significant decrease in first formant frequency (Fl) for vowel / a / only

6) Significant increase in second formant frequency (F2) for vowel / a / & / u /

7) No significant difference in third formant frequency (F3) for vowels / a /, / i / & */u/.*

8) No significant difference in Bandwidths (Bl, B2, B2) for vowels / a /, / i / & *In I.*

b) INTENSITY PARAMETERS

Inspection of Table 4B reveals the following

1) Significant reduction in mean intensity for vowels / a / , / i / & / u / only

2) Significant increase in mean extent of fluctuations in intensity for vowels / a /, / i / / u / only

3) Significant increase in mean speed of fluctuations in intensity for vowel / a / , / i / , / u / only

4) No significant difference in the mean intensity range for all the vowels / a / , / i / , / u /

c) TEMPORAL PARAMETERS

Inspection of Table 4c reveals the following

1) Significant increase of mean word duration in Parkinsons disease compared to normals

2) Significant increase in the mean vowel duration for vowels / a / , / i / only

3) Significant decrease in the mean burst duration for all voiceless consonants / p / , / t / , / k / .

4) Significant decrease in the mean closure duration for consonant / p / only

5) Significant decrease in the mean consonant duration for consonants / p / & /1 / only

6) Significant increase in the voice onset time for all voiceless consonants / p /, /1 / ,/ k /.

The frequency of occurrence of laryngeal and articulatory dysfunction among patients with Parkinsons disease have been well documented in literature (Logeman et. al. 1978; Canter, 1963, 1965; Weismer 1984; Ramig et. al. 1988). The perceptual findings of the present study are in accordance with the earlier study reported in literature. (Darley et. al. 1969; 1969b; 1975).

Darley et. al. Reported that the most characteristic distinctive features of hypokinetic dysarthria comprise mainly of significantly reduced variability in pitch and loudness, reduced loudness level over all and decreased use of all vocal parameters for achieving stress and emphasis. Markedly imprecise articulation is generated at variable rates in short burst of speech punctuated by illogical pauses and often by inappropriate silences . Voice quality is some times harsh, sometimes breathy.

These findings reported can be correlated to the perceptual features such as monopitch, lowpitch level, inappropriate silences, reduced stress, hoarse voice quality distorted vowels, imprecise consonant articulation as found in the present study.

The various acoustic and temporal parameters that contributed heavily to the impression of the following perceptual features are summarized below.

Perceptual Findings	**Acoustic Correlates**
Low pitch level	Decreased mean fundamental frequency seen in Parkinsons disease
Mono pitch	Flat fo contour Decrease in frequency range
Mono loudness	Flat intensity (SPL) contour
Hoarse voice quality	Increase mean speed and extent of fluctuations of frequency & inensity seen in Parkinsons disease
Imprecise consonants & distorted vowels	Increased VOT Increased vowel duration Increased formant frequencies Decreased consonant duration Decreased closure duration Decreased burst duration
Reduced stress	Decreased intensity and frequency variations Decreased consonant duration

The acoustic measures relating to the deviant perceptual dimensions presented over here are in agreement with that reported by Forrest et. al. (1997). To conclude, it can be hypothesized the dysfunction seen in the speech production mechanisms in Parkinsons disease can be attributed to the progressive nature of the disease. Logeman et. al. (1973, 1978) hypothesized that the dysfunction begins with the laryngeal system and subsequently involves the posterior tongue, more anterior portion of the tongue and finally the labial articulators. They suggested progression takes places from most posterior vocal tract (voice) deficits, to deficits involving more anterior portions of the vocal tract may be related to a predictable pattern of neural degeneration in the Somatotopic representations of the speech articulators in

Parkinsons disease. Further, effects on the temporal parameters can be attributed to the classical movement deficit seen in Parkinson's disease in turn effecting the motoric aspects of speech production (Speech motor control)

Thus, from the present study we can conclude that there is significant difference between Parkinson's disease and normal subjects of age group and sex in terms of frequency, intensity and temporal parameters.

EL **Amyotropic lateral sclerosis (AIS)**

Two female's ages ranging between 42-70 years were taken up who were diagnosed as having Amyotropic Lateral Sclerosis by **a** neurologist and a speech pathologist diagnosed them as having dysarthria.

Perceptual Analysis

The most deviant speech dimensions as judged perceptually were :

- Low pitch level
- Imprecise Consonants
- Slow rate
- Mono pitch
- Distorted vowels
- Harsh voice quality
- Inappropriate silences
- Short rushes of speech
- Moderate abnormality in Overall Intelligibility.

Table 5A : Depicts Mean ,SD values, Significance for all the frequency parameters in Amyotropic Lateral Sclerosis and Normal subjects.

\+ Significance at 0.05 level — Not Significant

	AMYOTROPIC LATERAL SCLEROSIS (N=2)						**NORMAL GROUP (N=2)**					
Frequency	MEAN (STANDARD DEVIATION)						MEAN (STANDARD DEVIATION)					
Parameters	/a/	Si	/i/	Si	/u/	Si	/a/	Si	/i/	Si	/u/	Si
Fundamental Frequency	206.24 (8.06)	+	158.83 (56.49)	-	203.31 (3.76)	+	219.56 (8.55)	+	227.77 (11.12)	-	233.51 (12.96)	+
Extent of Fluctuations	22.3 (1.48)	+	4.49 (1.004)		4.95 (0.4313)		3.59 (7.77)	+	3.48 (0.32)	-	3.19 (2.04)	-
Speed of Fluctuations	9.22 (2.35)	-	9.89 (7.07)	-	19.13 (9.3).	+	1.62 (0.88)	-	1.99 (1.414)	-	1.04 (1.36)	+
Frequency Range	107.02 (1.95)	-	173.45 (7.04)		215.03 (39.81)		49.2 (0.63)	-	76.71 (0.70)	-	165.76 (13.15)	-
Formant Frequencies												
Fl	885.4 (12.8)	+	289.3 (11.2)	-	375.3 (12.87)	+	820.00 (16.78)	+	345.4 (12.11)	-	346.6 (8.72)	+
F2	1273.6 (7.37)	+	2678.6 (84.8)	-	1232.8 (4.72)	-	1382 (19.28)	+	2094 (8.72)	-	975.4 (9.82)	-
F3	2576.6 (80.8)	-	3420.6 (70.7)	-	2367.0 (8.72)	-	2573.6 (24.78)	-	2588 (12.13)	-	2137.2 (10.11)	-
Band Widths												
Bl	130 (7.82)	-	170.2 (12.72)	-	150.4 (13.72)	-	191 (10.72)	-	119.8 (22.84)	-	228.8 (75.84)	-
B2	240.2 (2.72)	-	190.6 (11.72)		179.6 (12.92)	-	273.8 (14.72)	-	203.6 (44.85)	-	335.8 (80.24)	-
B3	340.2 (7.72)	-	220.2 (18.92)		248.6 (10.72)	-	383.6 (19.28)	-	300.2 (112.83)	-	348.4 (90.72)	-

Table 5B : Depicts mean , SD values and Significance for **all the** Intensity parameters in Amyotropic Lateral Sclerosis and Normal subjects.

\+ Significance at 0.05 level - Not Significant

	AMYOTROPIC LATERAL SCLEROSIS (N=2)						NORMAL GROUP (N=2)					
INTENSITY	MEAN STANDARD DEVIATION						MEAN STANDARD DEVIATION					
PARAMETERS	/a/	Si	/i/	Si	/u/	Si	/a/	Si	/i/	Si	/u/	Si
Mean Intensity	45.37 (4.48)	+	42.93 (3.03)	-	46.76 (144)	-	52.9033 (4.49)	-	43.09 (2.32)	-	34.05 (7.85)	-
Extent of Fluctuations	0.00 (0.00)	-	0.326 (0.28)	-	4.28 (0.42)	+	0.00 (0.00)	-	0.00 (0.00)	-	0.26 (0.46)	+
Speed of Fluctuations	0.25 (0.26)	-	0.30 (0.28)	-	0.80 (0.68)	+	0.00 (0.00)	-	0.00 (0.00)	-	0.00 (0.00)	+
Intensity Range	2.37 (0.54)	-	2.72 (0.33)		3.76 (161)	+	6.60 (18)	-	2.62 (1.22)	-	23.54 (4.77)	+

Table 5C : Depicts mean , SD values and Significance **for all the Temporal parameters in** Amyotropic Lateral Sclerosis **and Normal subjects.**

\+ Significant at 0.05 level - Not Significant

	AMYOTROPIC LATERAL SCLEROSIS (N=2)			NORMAL GROUP (N=2)		
PARAMETERS	MEAN	SI	STANDARD DEVIATION	MEAN	SI	STANDARD DEVIATION
Word Duration (msec)	757,77	+	117.528	648.77	+	118.528
Vowel Duration (msec)						
/a/	174.58	+	40.72	100.375	+	10.11
/i/	183,72	+	60.84	138.725	+	40.72
/u/	192.72	-	50.72	107.52	-	12.11
Burst Duration (msec)						
/p/	0.00	+	0.00	16.375	+	9.82
/t/	13.72	-	4.87	16.785	-	10.41
/k/	0.00	+	0.00	10.5	+	11.12
Closure Duration (msec)						
/p/	55.31	+	8.22	50.31	+	7,22
/t/	61.44	+	8.72	60.45	+	7,42
/k/	35.17		7.35	52.72	-	8.42
Consonant Duration (msec)						
/p/	0.00	+	0.00	152.99	+	7.07
/t/	67.42	+	11.84	143.47	+	7.08
/k/	0.00	+	0.00	119.24	+	4.41
Voice Onset Time (msec)						
/p/	0,00	+	0,00	15.99	+	8.21
/t/	14.72	-	11.82	16.42	-	7.82
/k/	0.00	+	0.00	12.42	+	9.82

Acoustic Analysis

Acoustic analysis revealed that the following parameters in ALS were different from their normal counterparts.

a) FREQUENCY PARAMETERS

Inspection of Table 5 A reveals the following

1) Significant reduction in mean fundamental frequency for vowels / a / & / u / only
2) Significant increase in mean extent of fluctuations in frequency for vowel . a / only
3) Significant increase in mean speed of fluctuations in frequency for vowel u / only
4) No significant difference in frequency range for vowels / a / , / i / & /u/.
5) Significant increase in first formant frequency (F1) for vowel / a / & / u / only
6) Significant decrease in second formant frequency (F2) for vowel / a / only
7) No significant difference in third formant frequency (F3) for vowels / a /, / i / & **/u/.**
8) No significant difference in Bandwidths (B1, B2, B3) for vowels / a /, / i / & **/u/.**

b) INTENSITY PARAMETERS

Inspection of Table 5B reveals the following

1) Significant reduction in mean intensity for vowel / a /
2) Significant increase in mean extent of fluctuations in intensity **for** vowel / u /
3) Significant increase in mean speed of fluctuations in intensity **for** vowel / u /
4) Significant decrease in the mean intensity range for **vowel** / **u** /

c) TEMPORAL PARAMETERS

Inspection of Table 5C reveals the following

1) Significant increase of mean word duration seen in ALS compared to normals
2) Significant increase in the mean vowel duration for vowels / a / & / i / only
3) Significant decrease in the mean burst duration for consonants / p / & / k / only.
4) Significant increase in the mean closure duration for consonant / p / & /1 / only
5) Significant decrease in the mean consonant duration for all consonants / p /, /1 /, / k / .
6) Significant decrease in the voice onset time for all consonants / p / & / k / only.

The perceptual observations of changes in both phonatory and aiticulatory systems in ALS are reported in clinical research literature (Darley et. al. 1969a; 1969b; 1975, Caruso et. al. 1987). In contrast, the body of literature providing acoustic quantification of the aiticulatory and phonatory performance of ALS speakers is still quite small (Kent et. al. 1992; stand et. al. 1993). The perceptual descriptions and acoustic data presented over here indicate great variability in phonatory performance and vocal quality of ALS subjects. These differences may be due to several factors: the relative degree of spasticity Vs flaccidity in the laryngeal musculature; differential involvement of particular laryngeal and /or respiratory muscles; and differing respiratory/laryngeal strategies for compensatory performance. Furthermore the aberrant temporal characteristics reported here for ALS speakers could result from either a) the tongue slowing into or away from aiticulatory postures for particular sounds or b) slow and /or weak laryngeal gestures or c) some combination of both (Caruso et. al. 1987).

The various acoustic and temporal parameters that contribute heavily to the impression of the following perceptual features are summarized below.

Perceptual Findings	Acoustic Correlates
Low pitch level	Decreased mean fundamental frequency seen in ALS
Imprecise consonants	Decreased VOT Increased vowel duration Changes in formant frequencies Decreased consonant duration Increased closure duration Decreased burst duration
Slow rate	Increased word duration Increased closure duration Increased vowel duration
Mono pitch	Flat fo contour
Distorted Vowels	Increase inFl Decrease in F2 Increased vowel duration Decreased VOT.
Hoarse voice quality	Increased mean speed and extent of fluctuations of frequency & intensity .
Inappropriate silences	Increased closure duration Decreased VOT.
Short rushes of speech	Decreased Burst Decreased VOT.

Thus, from the present study we can conclude that there is significant difference between Amvotropic lateral sclerosis and normal subjects of the same age group and sex in terms of frequency, intensity and temporal parameters.

III Wilsons disease

One female aged 15 years was taken up who was diagnosed as having Wilsons disease by a neurologist and a speech pathologist diagnosed her as having dysarthria.

Perceptual Analysis

The most deviant speech dimensions as judged perceptually were :

- Monopitch
- Monoloudness
- Imprecise consonants
- Slow rate
- Harsh voice quality
- Distorted vowels
- Reduced stress
- Mild abnormality (Over all intelligibility)

Acoustic analysis

Acoustic analysis revealed that the following parameters in Wilson's disease were different from their normal counter parts. However, this difference was not statistically significant and can be attributed to the fact that only a single subject was taken up in this category, in the present study.

a) Frequency Parameters

Inspection of Table 6A reveals the following

1) Decreased mean fundamental frequency for all vowels /a/, /i/, /u/
2) Increased Extent and Speed of fluctuations in frequency **for** all vowels /a/, /i/, /u/

Table 6A : Depicts Mean , for all the frequency parameters in Wilsons disease and Normal subjects.

	WILSONS DISEASE (N=l)			**NORMAL (N=l)**		
Frequency	MEAN			MEAN		
Parameters	**/a/**	/i/	***/u/***	**/a/**	/i/	/u/
Fundamental Frequency	254.31	273.68	202.73	272.66	246.94	249.02
Extent of Fluctuations	6.82	7.03	8.04	3.92	4.77	3.52
Speed of Fluctuations	0.22	7.83	10.12	1.98	**2.2**	0.98
Frequency Range	100.72	72.83	157.83	206.22	57.54	215.83
Formant Frequencies						
Fl	629.8	283.4	400.8	902.0	312	451.06
F2	1298.3	2045.8	1000.0	942.5	1515	1499.8
F3	2647.4	2106.4	2847	2828.72	2347.79	3601.32
Band Widths						
Bl	135.6	137.2	134	106.64	276.25	141.05
B2	360.6	198.0	206.6	559.85	349.28	272.82
B3	400.7	370.6	204.6	289.75	425.83	384.4

Table 6B : Depicts mean , for all the Intensity parameters in Wilsons Disease and Normal subjects.

INTENSITY	WILSONS niSI-ASP. (N=l) MEAN			NORMAL (N=l) MEAN		
PARAMETERS	/a/	/i/	/u/	/a/	/i/	/u/
Mean Intensity	33.47	40.37	43.72	55.89	43.12	90.36
Extent of Fluctuations	2.87	0.00	0.00	0.00	0.00	0.36
Speed of Fluctuations	3.27	1.23	3.92	0.00	0.00	0.00
Intensity Range	20.75	16.75	14.27	10.65	18.1	36.89

Table 6C : Depicts mean , for all the Temporal parameters in Wilsons Disease and Normal subjects.

PARAMETERS	WILSONS DISEASE (N=l)	NORMAL GROUP (N=l)
	MEAN	MEAN
Word Duration (msec)	657	697.67
Vowel Diiralion (msec)		
/a/	152.00	118.3
/i/	184.52	171.8
/u/	181.73	160.29
Burst Duration (msec)		
/p/	15.00	40.54
/t/	20.25	14.16
/k/	17.45	27.51
Closure Duration (msec)		
/P/	89.00	187.44
/t/	97.00	140.91
/k/	32.8	70.82
ConsonaiU Duration (msec)		
/p/	117.00	128.24
/t/	127.00	118.34
/k/	64.00	67.24
Voice Onset Time (msec)		
/p/	32.20	30.20
/t/	34.40	33.40
/k/	34.50	32.20

3) Decreased frequency range for vowels /a/ & /u/ only

4) Increased frequency range for vowel /i/

5) Decreased first formant frequency (Fl) for all vowels /a/, /i/, /u/

6) Increased second formant frequency (F2) for all vowels /a/,/i/, /u/

7) Decreased Third formant frequency (F3) for all vowels /a/, /i/, /u/

However, these differences were not statistically significant

b) Intensity Parameters

1) Decreased mean intensity for all vowels /a/, /i/, /u/

2) Increased speed and extent of fluctuations in intensity for all vowels /a/, /i/, /u/

3) Increased intensity range for vowel /a/ only

4) Decreased intensity range for vowels /i/ & /u/

However, these differences were not statistically significant.

c) Temporal Parameters

1) Decreased mean word duration in Wilsons disease

2) Increased vowel duration for all vowels /a/, /i/, /u/

3) Decreased burst duration for consonants /p/ & /k/

4) Increased burst duration for consonants /t/ only

5) Decreased closure duration for consonants /p/, /t/, /k/

6) Decreased consonant duration for consonants /p/ & /k/

7) Increased consonant duration for consonant /t/ only

8) Increased voice onset time for consonant /p/, /t/, /k/

However these differences were not statistically significant.

The perceptual observations of changes in both phonatory and articulatory systems in Wilsons disease are reported in clinical research literature (Darley et al 1969a; 1969b; 1975; Berry et. al. 1974). In contrast, the body of literature providing acoustic quantification of the articulatory and phonatory performance of Wilsons

disease speakers is still quite small. Berry et. al. (1974) reported that the most characteristic distinctive feature of dysarthria in Wilsons disease comprise mainly of reduced pitch variability or stress patterns, a lock of precision in producing consonants, irregular articulatory breakdown, prosodic compensations. Phonatory patterns involving low pitch, harsh voice and vocal strain. These findings reported can be correlated to the perceptual features such as monopitch, Monoloudness, imprecise consonants, slow rate, Harsh voice quality, distorted vowels, reduced stress as found in the present study.

The various acoustic and temporal parameters that contributed heavily to the impression of the following perceptual features are summarized below.

Perceptual Findings	Acoustic Correlates
Monopitch	Flat FO contour Decreased frequency range
Mono loudness	Flat Intensity (SPL) contour
Imprecise Consonants & Distorted vowels	Increased VOT Increased Vowel duration Changes in Formant Frequencies Decreased Consonant duration Decreased burst duration
Slow rate	Changes in burst and consonant duration Increased Vowel duration
Harsh Voice Quality	Increased speed and Extent of fluctuations in both frequency and intensity-
Reduced stress	Decreased intensity and Frequency variations Decreased consonant and Vowel duration

Berry et. al. (1974) opined that the dysarthria in Wilsons disease contains components the resemble, in fact, the dysarthria in patients with cerebellar disorders

(ataxic), Parkinsonism (hypokinetic) and pseudobulbarpalsy (spastic). Analysis of deviant patterns seen in Parkinsons disease and Wilson disease patients in the present study indicate that dysarthria in Wilsons disease contains components that resemble, in fact the dysarthria of patients with Parkinsons disease, supporting Berry et. al. (1974) study. This similarity seen can be attributed to multiple system involvement within the nervous system with a mixture of neurologic symptoms as well as speech symptoms, it would be logical to call the dysarthria in Wilsons disease a " Mixed dysarthria". However, studies suggest that most individuals with early wilsons disease have a pure hypokinetic type of dysarthria, in distinguishable from that of Parkinsons disease. In more advanced cases they have the ataxic speech characteristics as well as the prominent prosodic deviations found in both hypokinetic and spastic dysarthria.

Thus from the present study we can conclude that there is difference between Wilsons disease and normal subjects of same age group and sex in terms of frequency, in intensity and temporal parameters, however this difference was not statistically significant.

Fig i) Frequency and Intensity Analysis waveform of a representative normal individual. For phonation /a/.

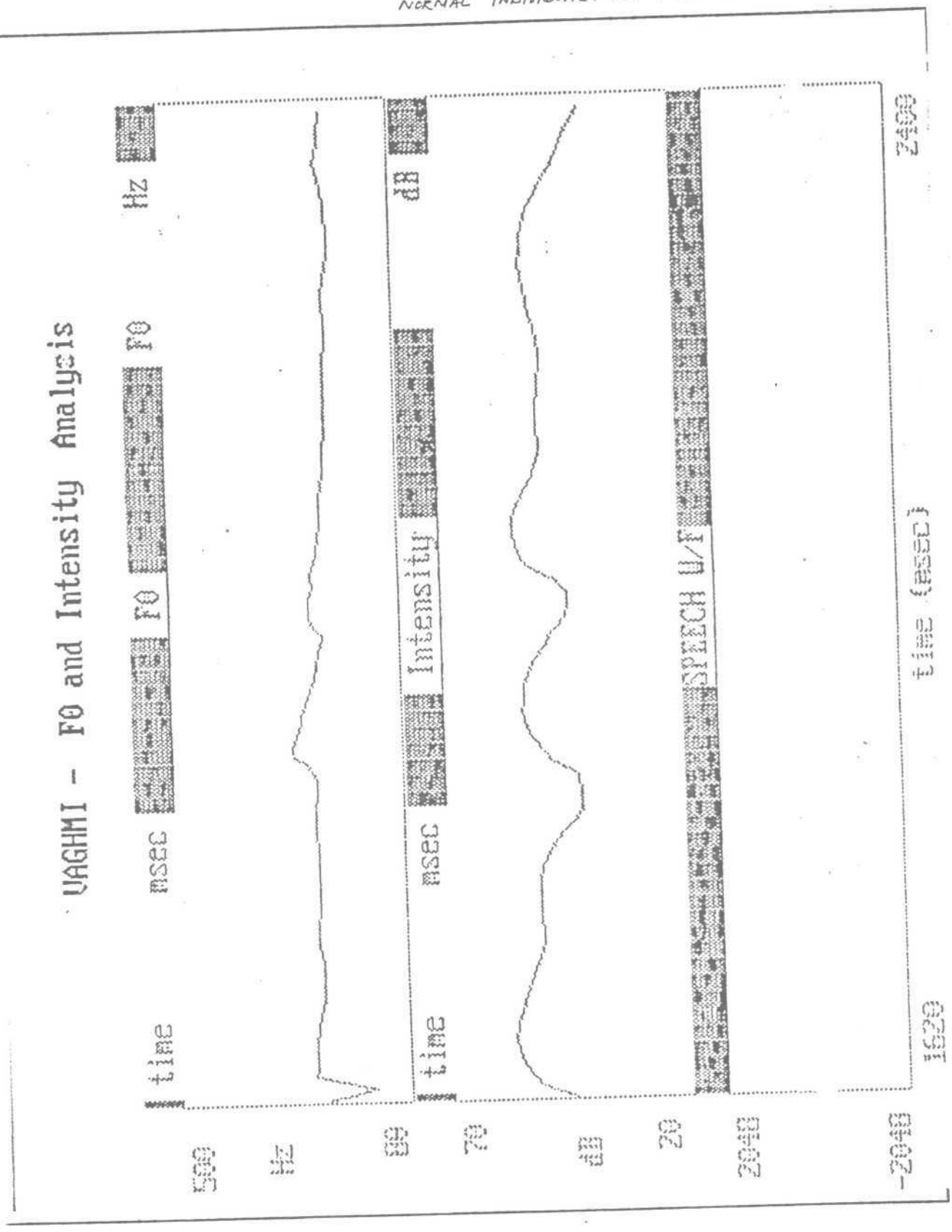


Fig ii) TEMPORAL ANALYSIS WAVEFORM OF A REPRESENTATIVE NORMAL INDIVIDUAL FOR THE WORD /kakal

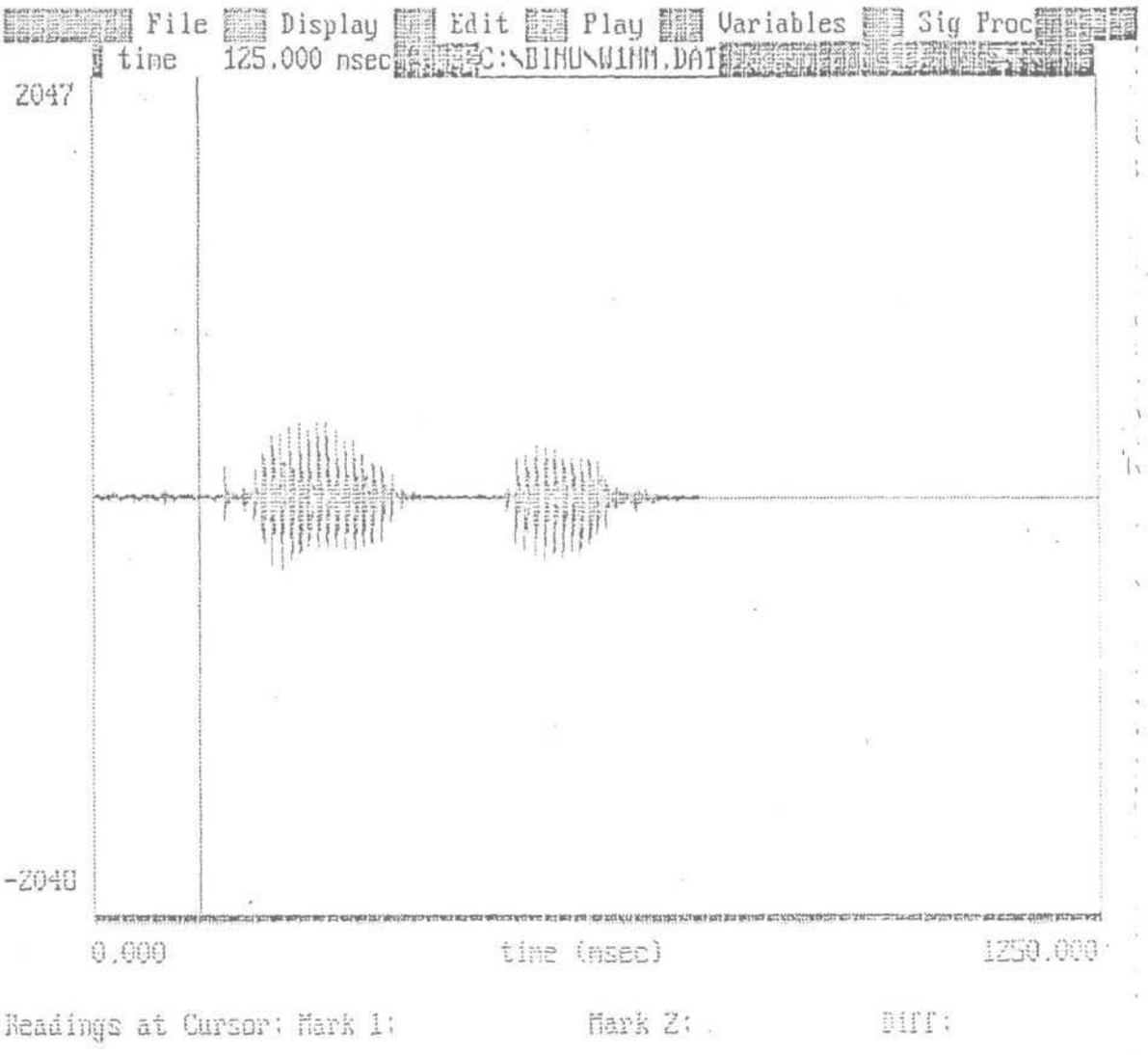


Fig (iii) Frequency and Intensity analysis of a representative subject with Parkinsons Disease for phonation /a/.

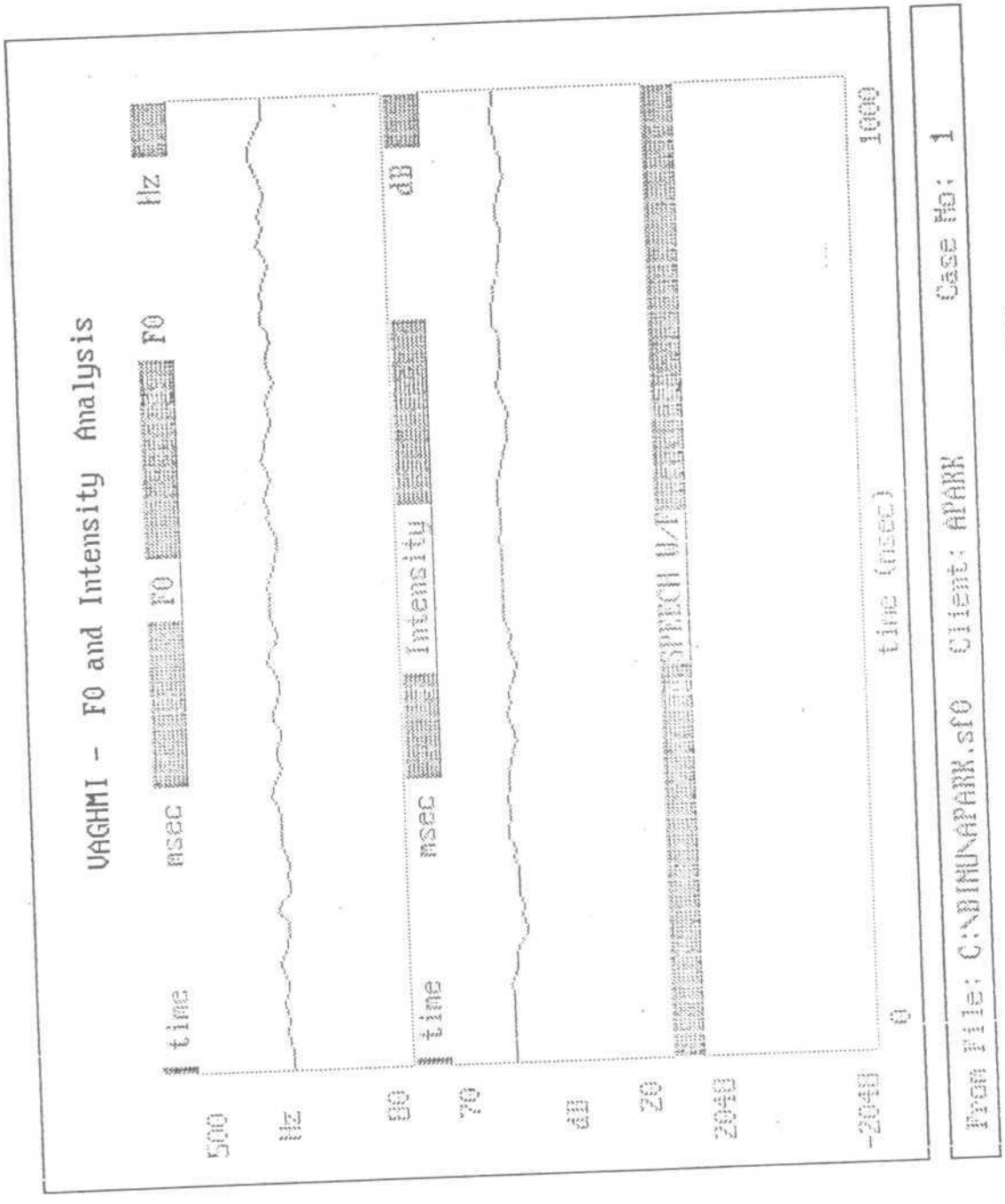


FIG (iv). FREQUENCY AND INTENSITY ANALYSIS OF A REPRESENTATIVE SUBJECT WITH AMYOTROPHIC LATERAL SCLEROSIS FOR PHONATION /a/.

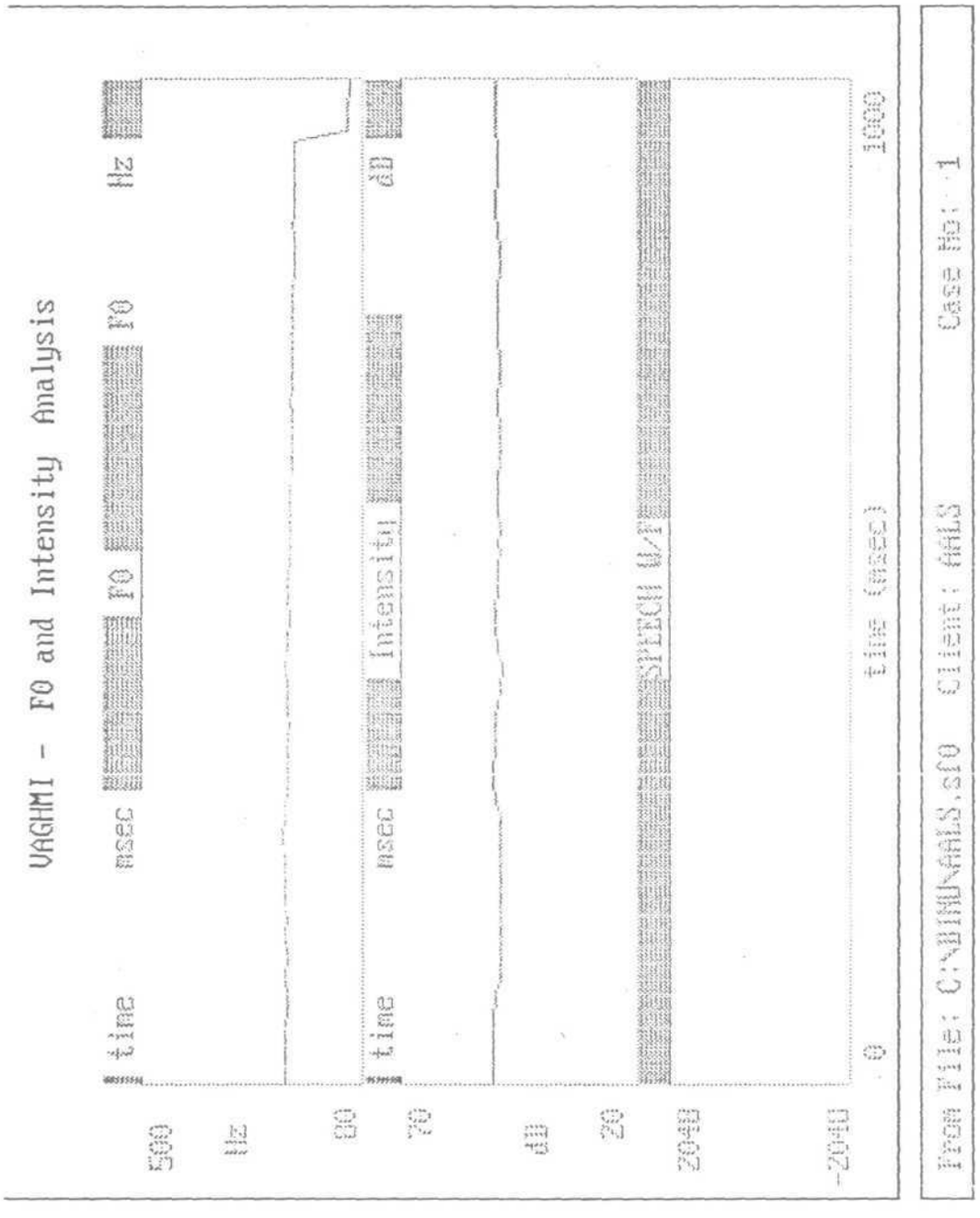


FIG V) FREQUENCY AND INTENSITY ANALYSIS OF A REPRESENTIVE SUBJECT WITH WILSONS DISEASE FOR PHONATION /a/.

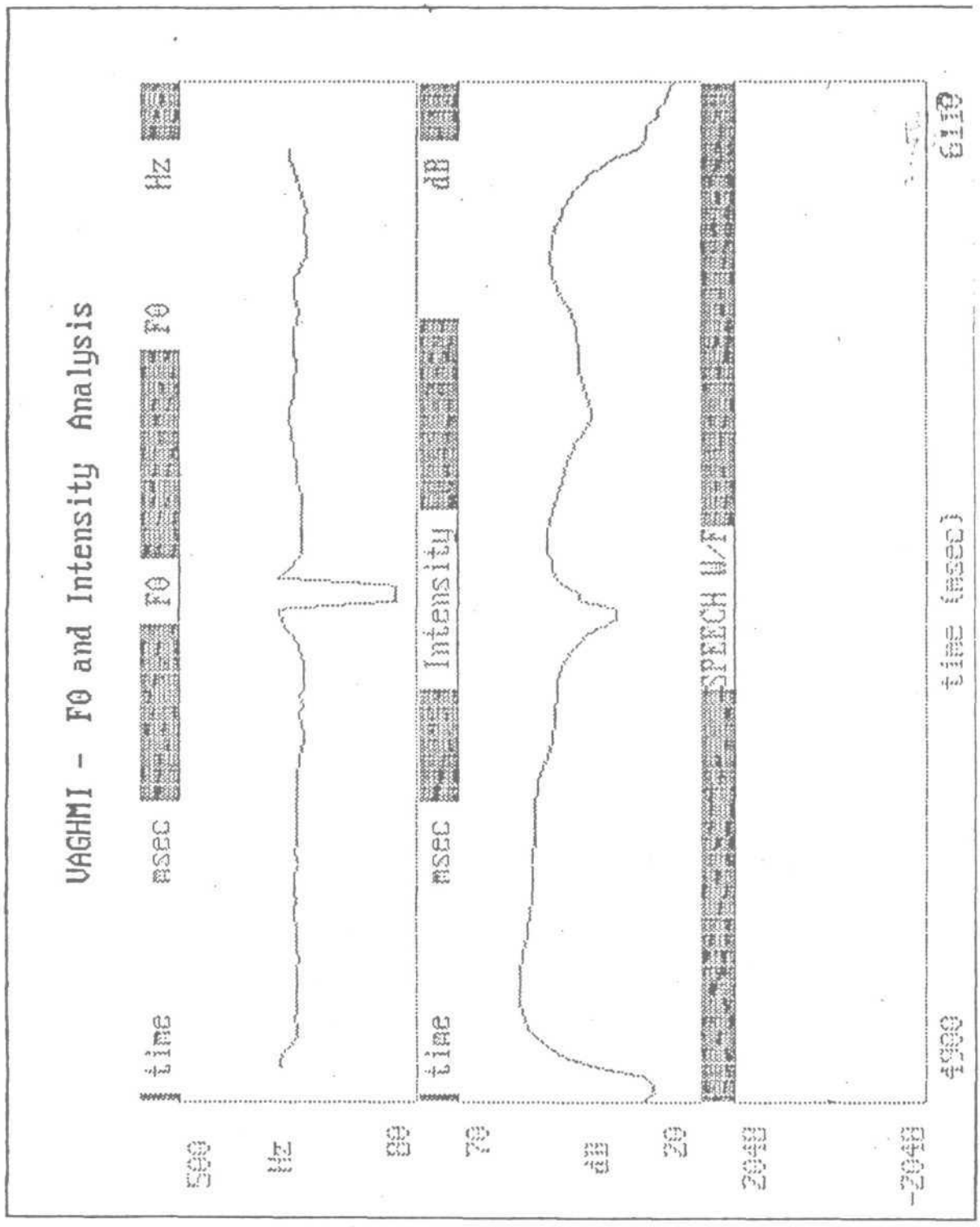


FIG vi) TEMPORAL ANALYSIS WAVEFORM OF A REPRESENTATIVE SUBJECT WITH PARKINSONS DISEASE FOR THE WORD /kaka/

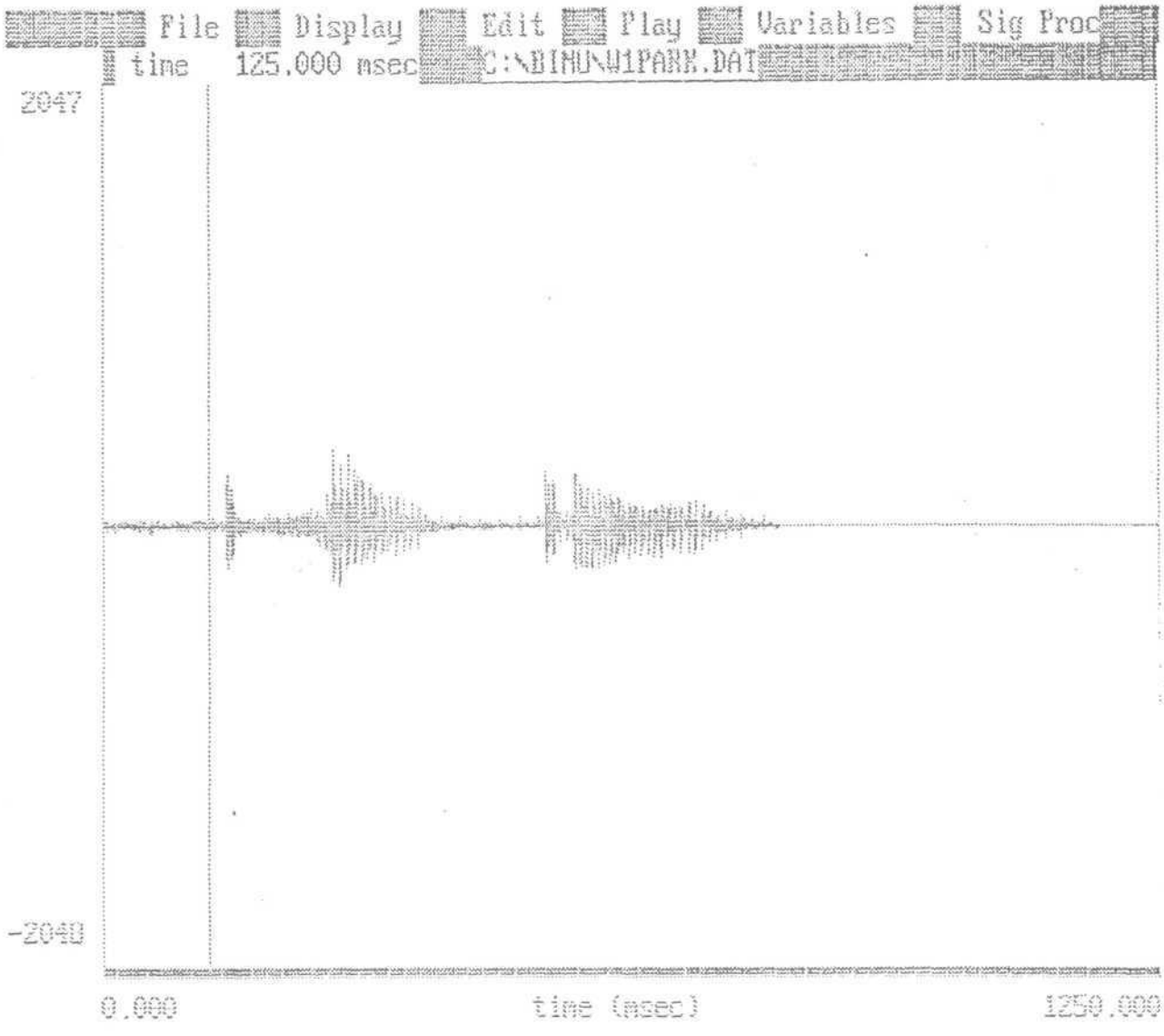


Fig vii) Temporal Analysis waveform of a representative subject with amyotropic lateral sclerosis for the word /kakal.

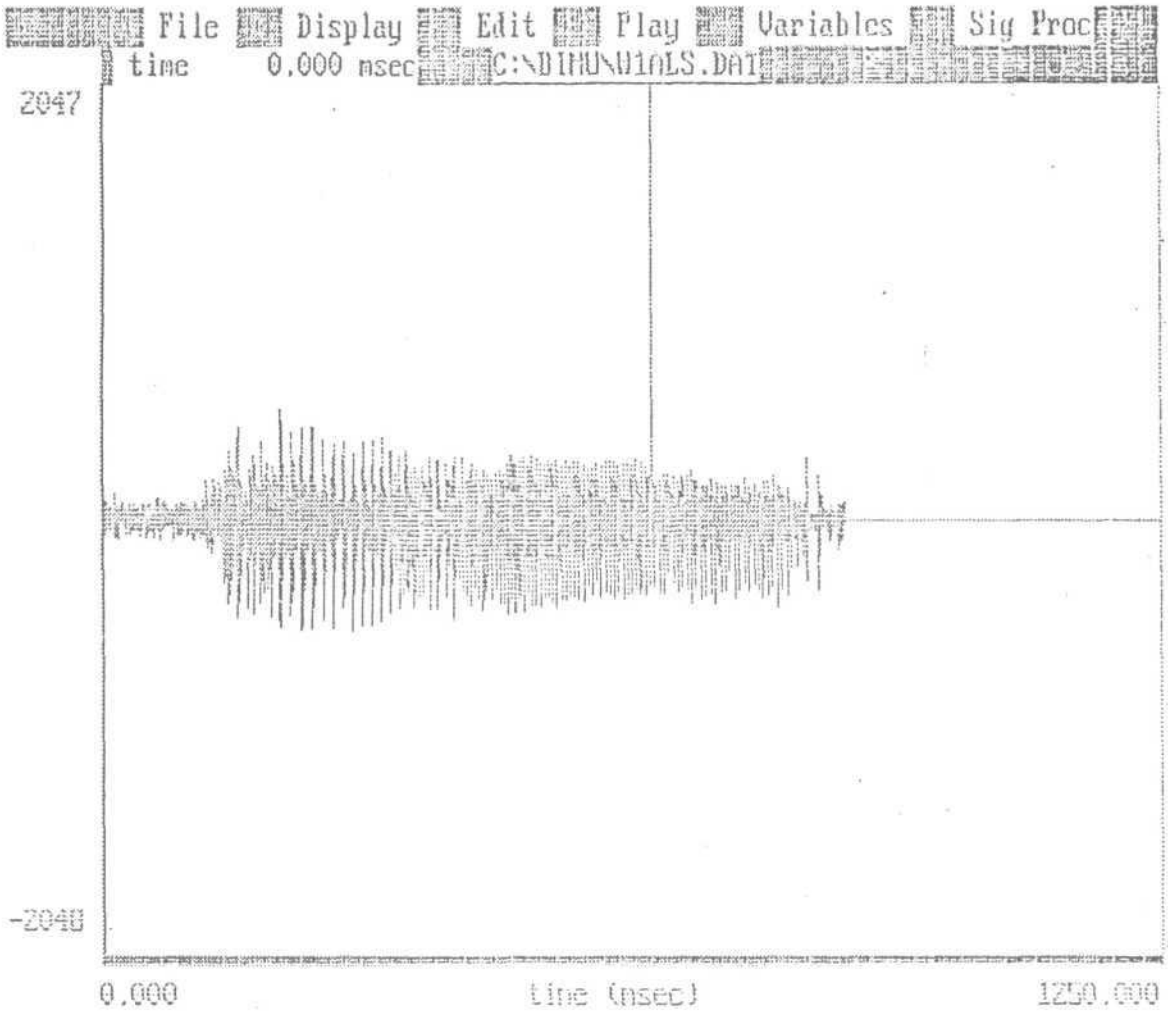
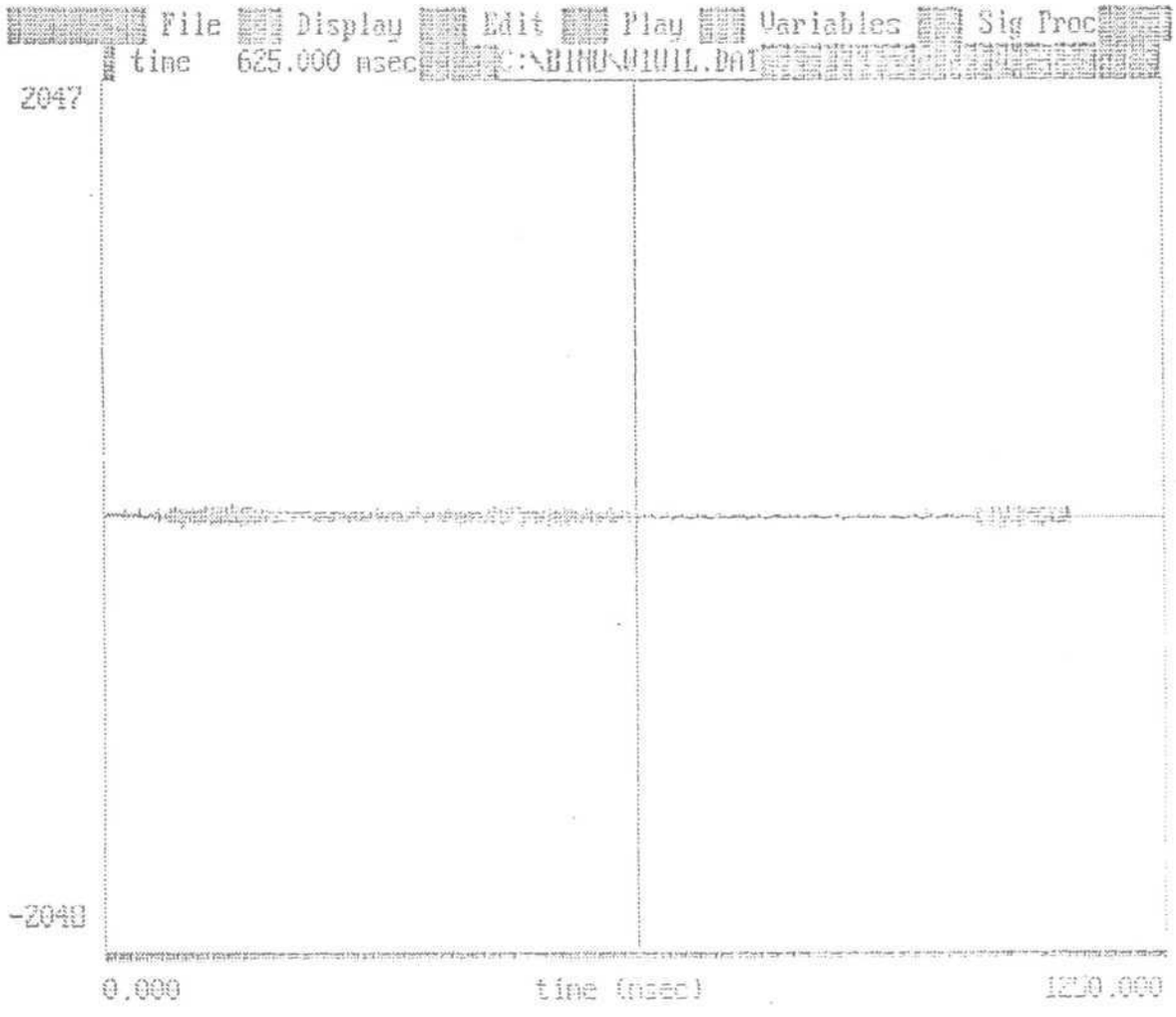


FIG (1x) TEMPORAL ANALYSIS WAVEFORM OF A REPRESENTATIVE SUBJECT WITH WILSONS DISEASE FOR THE WORD /kɛkɛ/.



Summary and Conclusion

Dysarthria is a Greek word, meaning literally disturbance of articulation. As the word is usually employed in the field of Speech Pathology it implies any impairment of articulation caused by agenesis of or damage to the nerve center or tracts (other than those of the language areas of the cerebral cortex) immediately involved in direct control of the musculature used in the enunciation and pronunciation of vowels and consonants (West and Ansberry, 1968). Several attempts have been made to analyze dysarthric speech using both physioacoustic and psychoacoustic methods. Darley, Aronson and Brown (1975) formed a classification system based on the perceptual judgement of dysarthric speech on 28 deviant speech dimensions. However studies correlating the findings of both the perceptual and acoustic methods are scanty. Further, not much information is available in Indian context on these parameters in dysarthrics. So, the present study was carried out to delineate the acoustic parameters in dysarthric speech. The purpose of the study was to test the. following hypothesis:

Main Null Hypothesis

There is no significant difference in the acoustic parameters of speech of dysarthrics and normal subjects.

Auxiliary Hypothesis

1) There is no significant difference between the three types of dysarthric subjects : Parkinsons disease, Amyotropic Lateral sclerosis and Wilson's disease in terms of comparable acoustic and temporal parameters.

a) Mean fundamental frequency for phonation for / a /, / i /, / u /

b) Mean intensity in phonation for / a /, / i /, / u /

c) Extent of fluctuations in frequency and intensity.

d) Speed of fluctuations in frequency and intensity.

e) Frequency range in phonation

f) Intensity range in phonation

g) Formant frequencies (F1,F2,F3)

h) Bandwidth (B 1.B2, B3)

i) Word duration

j) Vowel duration

k) Burst duration

1) Closure duration

m) Consonant duration

n) Voice onset time (VOT)

To test these hypotheses, totally 12 subjects both males and females were selected from the same age range. These consisted of 6 normal subjects and 6 subjects with dysarthria. All normal subjects were normal in terms of Speech, Language, and Hearing and had no history of neurological problems. Subjects with dysarthria consisted of three with Parkinsons disease, two with Amyotropic Lateral Sclerosis and one with Wilson's disease. Three trials of sustained phonation of / a /, / i / & / u / and a list of 50 familiar words in malayalam were recorded for all the subjects .Using the data on vowel prolongation the following frequency and intensity measures were obtained by using computer analysis.

a) Mean fundamental frequency in phonation for / a /, / i /, / u /

b) Extent of fluctuations in frequency in phonation.

c) Speed of fluctuations in frequency in phonation.

d) Frequency range in phonation

e) Mean intensity in phonation for / a /, / i /, / u /

f) Extent of fluctuations in intensity in phonation.

g) Speed of fluctuations in intensity in phonation.

h) Intensity range in phonation

i) Formant frequencies (F1 ,F2,F3)

j) Bandwidth (B1,B2,B3)

The speech samples of normal and dysarthric subjects were analyzed both perceptually and acoustically. The perceptual analyses was done by three judges. The computer programs of VSS (Voice and Speech Systems, Bangalore) were used for acoustic analysis and the following parameters were noted and compared.

a) Word duration

b) Vowel duration

c) Burst duration

d) Closure duration

e) Consonant duration

f) Voice onset time (VOT)

Analysis of results using Wilcoxons Matched Pair Sign Rank test showed that there was

1) Significant difference in the acoustic parameters of speech of dysarthrics and normal subjects

2) Significant difference between the three dysarthric subjects was seen only between Parkinsons disease and Amyotropic lateral sclerosis but not with Wilsons disease in terms of comparable acoustic and temporal parameters

3) Significant difference between Parkinsons disease, Amyotropic lateral sclerosis for few of the parameters when compared to the normal subjects of the same age group and sex.

4) No significant difference between Wilsons disease and normal subjects of the same age group and sex when each of the parameters were compared.

To conclude the present study adds to literature by supporting earlier studies conducted to delineate the acoustic and temporal correlates of dysarthric speech. Further a direct relationship between acoustic patterns and perceptual phenomena should not be always expected, as variability can exist across tasks as well as across subjects lending further support to the argument that the neuromuscular impairment in dysarthrics is complex and idiosyncratic. However, acoustic analysis of dysarthric speech can provide specific information correlating to possible physiological contributions to perceptual characteristics. Such analysis may therefore aid in determining patterns of differential neural subsystem , early detection of disease and providing a tool for monitoring disease progression. This in turn can lead the clinician to make better decisions regarding earlier and better patient education, counseling and more efficacious treatment.

Recommendations

1. Each type of dysarthric group can be taken and the deviant aspects can be analyzed, helping in differential diagnosis of dysarthrias based on speech characteristics.
2. Number of subjects can be increased in each group and analysis can be done.
3. The aerodynamic study of speech can be done to add to the information about the acoustic characteristics of dysarthric speech.
4. Artificial neural networks can be used for the classification of dysarthrics using acoustic parameters.
5. Multidimensional analysis of voice in dysarthrics can be done.

The study is limited to

1) Maximum of three subjects within each category of dysarthria

2) One subject with Wilsons disease

3) Analysis of words with voiceless consonants /p/, /t/, /k/

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APPENDIX - I

Description of the Dysarthric Subjects

Subject No.	Age /Sex	Duration of Illness	Diagnosis	Medication
1.	55 /Male	31/2 years	Stage II Parkinsons disease	Tab: Tidomet Plus Pacitane
2.	72 / Male	2 years	Stage II Parkinsons disease	Tab: Tidomet Plus Bromocriptine
3.	70 /Male	41/2 years	Stage II Parkinsons disease	Tab: Tidomet Plus Trihexy-phenidyl HCL Pacitane
4.	45 / Female	1 year	Amyotropic Lateral Sclerosis (bulbar / Spinal involvement)	Tab: Tizam
5.	70 / Female	2.5 years	Amyotropic Lateral Sclerosis (bulbar / Spinal involvement)	Information Not Available
6.	15/ Female	1 year	Wilsons disease	D-penicilla-mine D-isomer

APPENDIX - II

LIST OF WORDS (MALAYALAM) USED AS TEST MATERIAL.

1) കാക്ക	ka:ka:
2) നെറ്റി	neṯṯi
3) പൈസ	paisa
4) കട്ടിൽ	kaṭṭil
5) പാട്ട്	pa:tt
6) അപ്പോൾ	appoḷ
7) സന്ധ്യ	sandja:
8) പാട്ട	pa:ṭṭa
9) പണ്ട്	panda
10) പക്ഷി	pakʃi
11) സാമ്പാർ	sa:mba:r
12) ചേച്ചി	tʃe:tʃi
13) കിട്ടി	kiṭṭi
14) സിംഹം	simham
15) കപ്പൽ	kappal
16) പച്ച	patʃa
17) രാത്രി	ra:ṯri
18) തട്ട്	ṯaṭṭə
19) കഷ്ടം	kaʃṭam
20) പൂച്ച	pu:tʃa
21) മുന്ത്	mutt
22) പാറ്റ	pa:ṭṭa
23) ചിപ്പ്	tʃi:pə
24) പശ	paʃa
25) ചുറ്റും	tʃuṭṭum
26) ദൈവം	deivam
27) പല്ലി	palli
28) ചുണ്ട്	tʃuṇḍa
29) മുട്ട	muṭṭa

30) പുല്ല്	pullə
31) തങ്കം	tangam
32) ചന്ത	tʃəndə
33) മാങ്ങ	ma:ŋa
34) അഞ്ച്	andʒə
35) പേന	pe:na
36) തിന്നു	tinnu
37) മല്ലി	malli
38) ചേട്ടൻ	tʃe:ṭṭan
39) മുണ്ട്	muṇḍə
40) പകൽ	pakal
41) തെറ്റ്	ṭeṭṭə
42) മുറ്റം	muṯṯam
43) ചീത്ത	tʃi:ṯa
44) അപ്പൻ	appan
45) മൂങ്ങ	mu:ŋa
46) റോസ	ro:sa
47) മുഖം	mukʰam
48) രസം	rasam
49) മഞ്ഞ	manja
50) പണ്ട്	paṇḍə

APPENDIX -

Dimentions	Rating Scale				
Phonatory-Respiratory	1	2	3	4	5
1. Low pitch level					
2. Pitch breaks					
3. Hoarseness					
4. Breathiness					
5. Hoarseness					
6. Strained-stranghed sound					
7. Voice stoppages					
8. Audible Inhalation					
9. Forced Inhalation / Exhalation					
Articulatory					
1. Imprecise consonants					
2. Vowels distorted					
3. Irregular breakdowns					
4. Phonemes repeated					
5. Phonemes prolonged					
Resonatory					
1. Hyperaasality					
2. Nasal emission of air					
Prosodic					
1. Monopitch					
2. Monoloudness					
3. Excessive loudness variation					
4. Loudness decay					
5. Slowed rate					
6. Rapid rate					
7. Variable rate					
8. Short rushes of speech					
9. Reduced stress					
10. Excess & Equalized stress					
11. Intervals prolonged					
12. Inappropriate silences					

Rating Scale

1. Profound Impairment
2. Severe Impairment
3. Moderate Impairment
4. Mild Impairment
5. No Impairment

APPENDIX - IV

1 The tape recorder was calibrated by the following procedure. Tones of 125HZ, 250HZ, 500 HZ and 1000 HZ at 50 dB from Heterodyne Analyser Type 2010 (B& K) were recorded on the tape. The recorded signals from the tape were digitized using the procedure described in methodology. Then, using INTON programme on the computer referred in the methodology frequency and intensity were analyzed. The results, pointed below showed no variation in the frequency and intensity of the tones. Thus the fidelity of the tape recorder and the tape were tested and then used for the study.

Tones analyzed by INTON programme.

Measures	125 HZ	250 HZ	500HZ	1000HZ
Mean FO	125	250	500	1000
Extent of fluctuation in fundamental freq.	0	0	0	0
Extent of fluctuation in intensity	0	0	0	0
Speed of fluctuation in fundamental freq.	0	0	0	0
Speed of fluctuation in intensity	0	0	0	0
Maximum FO	126	250	500	1000
Minimum FO	125	250	500	1000
Maximum intensity	51	51	52	51
Minimum intensity	50	50	50	50
Range (FO)	1	0	0	0
Range intensity	1	1	2	1
Pause (%)	0	0	0	0

1. Developed by Dr. T.V. Ananthapadmanabha and associates. These soft ware have been tested and also used for analyzing voice and speech of normal and pathological subjects at the Department of Speech Sciences, All India Institute of Speech and Hearing, Mysore, India regularly. These programmes have been specially developed to derive parameters of voice and speech.

APPENDIX - V

a) Extent and speed of fluctuations in fundamental frequency in phonation (Ex.F.F / Sp.F.F)

The fluctuation in phonation in frequency was studied as the extent and speed of fluctuation. The fluctuation in frequency was defined as the variations +/- 3 Hz and beyond in fundamental frequency. The extent of fluctuation in frequency was defined as the means of fluctuation in fundamental frequency in a phonation of one second. The speed of fluctuation in frequency was defined as the number of fluctuations in fundamental frequency in a phonation of one second. The TNTON' programme provided the extent and speed of fluctuation for the phonation submitted for analysis by considering the whole sample i.e., by averaging the extent and speed of fluctuation obtained for the sample analyzed. The extent and speed of fluctuation for three trials of /a/ were averaged and the value considered as the extent and speed of fluctuation for /a/. Similarly, the extent and speed of fluctuation in fundamental frequency for the vowels /i/ and /u/ for subjects of all the three groups were obtained.

b) Extent and speed of fluctuation in intensity in phonation (Ex. F.L / Sp. F.I.):

Fluctuation in phonation in terms of intensity were studied as the extent and the speed of fluctuation. Fluctuation in intensity was defined as the variations +/- 3dB and beyond in intensity. The extent of fluctuation in intensity was defined as the means of fluctuations in intensity in a phonation of one second. The speed of fluctuation in intensity was defined as the number of fluctuations in intensity in a phonation of one second. The TNTON' programme, similar to extent and speed of fluctuation in fundamental frequency, provided the extent and speed of fluctuation in intensity for each trial of/a/. The average of three values was considered as the extent and speed of fluctuation in intensity for /a/. Similarly , the extent and speed of fluctuation in intensity for vowels /i/ and /u/ for all the subjects of all the three groups were determined.

c) Frequency range in phonation (FR) :

The difference between the maximum and minimum fundamental frequency in phonation was considered the frequency range in phonation. Three values of ranges were obtained for /a/ using all the three recordings of /a/ of each subject. The maximum of the three was considered as the frequency range for /a/ for each subject.

Similarly, the frequency range for /i/ and /u/ for each subject were obtained for all the three groups.

d) Intensity range in phonation (IR) :

The difference between the maximum and minimum intensities in phonation provided the intensity range in phonation. Three values of intensity ranges were obtained for /a/ using all the three recordings of/a/ of each subject. The maximum of the three trials was considered as the intensity range for /a/. Thus, the intensity range in phonation was obtained for all the subjects from the three groups. Similarly, the intensity range for /i/ and /u/ for each subject were obtained.

APPENDIX - VI

Definitions used in the study :

Dysarthria : disrupted oral communication due to paralysis, weakness, abnormal tone or the muscles used in speech and encompass co-existing motor disorders of respiration, phonation, resonation, articulation and prosody.

Deviant Speech Dimensions:

These were taken from the thirty-eight deviant speech dimensions given by Darley. Aronson and Brown (1975).

Phonatory-Respiratory :

1. **Low Pitch Level** :- Pitch of voice sounds consistently too low for individuals age and sex.
2. **Pitch Breaks** :- Pitch of voice shows sudden and uncontrolled variation (Falleselto breaks)
3. **Harshness** :- Voice is harsh, rough and raspy.
4. **Breathiness**:- (Transient): Breathiness is transient, periodic intermittent.
5. **Hoarseness** :- (wet voice):- Wet, "Liquid" sounds hoarseness.
6. **Strained-strained sound** :- Voice (phonation) sounds strained or strangled (an apparently efforatal squeezing voice through glottis).
7. **Voice stoppages** :- There are sudden stoppages of voiced air stream (as if some obstacles along vocal tract momentarily impedes flow of air).
8. **Audible inhalation** :- Audible breathy inspiration.
9. **Forced inhalation / exhalation** :- Speech is interrupted by sudden forced inspiration and expiration and expiration signals.

Articulatory :

1. **Imprecise consonants** :- Consonant sound lacks precision. They show shining, inadequate sharpness distortions and lack of crispness. There is clumsiness in going from one consonant bound to another.
2. **Vowels distorted** :- Vowel sounds are distorted through out their total duration.

3. **Irregular articulatory breakdowns** :- Intermittent non-systematic breakdown in accuracy of articulation.
4. **Phonemes repeated** :- Repetitions of phonemes.
5. **Phonemes prolonged** :- There is prolongation of phonemes.

Resonatory :

1. **Hypernasality** :- Voice sounds excessively nasal. Excessive amount of air is resonated by nasal cavities.
2. **Nasal emission of air** :- There is nasal emission of air stream.

Prosodic :

1. **Monopitch** :- Voice is characterized by a monopitch or monotone. Voice lacks normal pitch and inflectional changes. It tends to stay at one pitch level.
2. **Monoloudness** :- Voice shows monotony or loudness. It lacks normal variation in loudness.
3. **Excessive loudness variation** :- Voice shows sudden uncontrolled alterations in loudness, sometimes becoming too loud, sometimes too weak.
4. **Loudness decay** :- There is progressive diminution or decay of loudness.
5. **Slowed rate** :- of actual speech is abnormally slow.
6. **Rapid rate** :- of actual speech is abnormally rapid.
7. **Variable rate** :- Rate alternately changes from slow to fast.
8. **Short rushes of speech** :- By pauses.
9. **Reduced stress** :- Speech shows reduction of proper stress or emphasis patterns.
10. **Excess and equalized stress** :- Excess stress on unstressed parts of speech eg(I) monosyllabic words (II) Unstressed syllables of polysyllabic words.
11. **Intervals prolonged** :- Prolongation of interward or inter-syllable intervals.
12. **Inappropriate silences** :- when there are abnormal intervals or pauses in syllables and in between two words.