A DESCRIPTIVE STUDY COMPARING PHONOLOGICAL AND ARTICULATORY PATTERNS OF CLEFT PALATE AND CEREBRAL PALSYED SPEAKERS

A thesis submitted in partial satisfaction of the requirements for the degree of Master of Arts in Communicative Disorders

by

Joan Gullerud

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The Thesis of Joan Gullerud is approved:

Philip Reid

Edward P. Hall

Rolland Reeve, Chairman

California State University, Northridge
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ABSTRACT

A DESCRIPTIVE STUDY COMPARING PHONOLOGICAL AND ARTICULATORY PATTERNS OF CLEFT PALATE AND CEREBRAL PALSIED SPEAKERS

by

Joan Gullerud

Master of Arts in Communicative Disorders

Articulatory and phonological aspects of cleft palate and cerebral palsied speech were examined and compared to determine similarities and differences in order to increase our understanding of structural and neurological contributions to speech production.

The sample consisted of five subjects, two cleft palate children and three cerebral palsied children. Articulation and phonological patterns of the subjects were assessed and results were compared with findings reported for each group in the literature.

Both groups evidenced inadequate velopharyngeal functioning and were found to have a greater amount of hypernasality present than in a normal population. The cleft palate group exhibited a more constant condition of hypernasality while hypernasality was found to be inconsistently present in cerebral palsy. The two etiologies provide
an explanation of this phenomenon, that is, a structural disorder is a
static condition, ever-present, while a neurological disturbance is a
dynamic process, ever-changing. Both disorders, however, showed
inconsistencies due to phonemic context, length of utterance, etc.

The greatest number of articulatory errors for both groups were
evidenced in high-pressure consonants such as fricatives and affricates.
However, these errors were replaced by different sounds in the two
groups. Cleft palate speakers demonstrated laryngeal and pharyngeal
substitutions and distortion of sounds by nasal emission, indicating an
adequate but misdirected air stream. Cerebral palsied speakers, on the
other hand, showed omissions and substitutions of simpler sounds
suggesting reduced breath support as well as weakness and incoordination
of other articulators. This appeared to differentiate a focal
disturbance, such as cleft palate, where respiration and other
articulators are not involved, and a generalized disability where
other processes are often affected.

Finally, because of the generalized effect of the cerebral
palsied condition, speech was more often found to be reduced in
intelligibility than for cleft palate speakers.
Chapter 1

INTRODUCTION

Cleft palate and cerebral palsy were selected for this investigation because they represent structural and neurological disorders respectively. Defective articulation in cleft palate due to abnormality of the external speech organs and not to lesions of the central nervous system (CNS) is known as dyslalia (Wood, 1971). Dysarthria represents a disorder of articulation due to impairment of the part of the CNS which directly controls the muscles of articulation and is evidenced in cerebral palsy (Wood, 1971). Dyslalia, as defined by Wood (1971), also entails defective articulation due to faulty learning. Since a functional component can also exist in cerebral palsied speech, the clinical separation of dyslalia and dysarthria is not always clear. Terms used to describe defective articulation are fraught with much inconsistency among writers and also between countries. Great Britain, for example, affords dysarthria a broader definition to include structural as well as neurological disorders (Committee Report on Speech Therapy Services, 1969). But for purposes here, the delineation employed associates dyslalia with structural defects and dysarthria with neurological defects.

Comparisons of articulation and phonological patterns between cleft palate and cerebral palsied speakers are rare, if not absent, in the literature. Possibly because the need has not arisen. Certainly, differential diagnosis is adequate and their medical and therapeutic
management are quite diverse. It is believed, that a detailed and systematic examination of the similarities and differences in the two conditions could refine or perhaps extend the clinical data in each area. As is often the case, such scrutiny may lead to useful information about normal speech production. Moreover, this analysis may demonstrate that disorders as dyslalia may not be exclusively structural, nor dysarthria exclusively functional. Such a datum can be valuable to diagnosis and to planning appropriate therapy as revealed in the report by Hoopes et al. (1970) that "neurogenic" hypernasal speech all too often serves as a "wastebasket" diagnosis of hypernasal speech in the absence of an overt or submucous cleft palate. They examined eight patients diagnosed with "neurogenic" hypernasality and found subtle anatomical abnormalities sufficient to account for velopharyngeal incompetence in four patients. This has important implications for therapy since intelligent management of speech problems demands accurate diagnosis.

Definitions

Cleft Palate

In the limited sense, the term "cleft palate" denotes a congenital fissure resulting from incomplete merging or fusion of embryonic processes that normally unite in the formation of the soft palate, the roof of the mouth, the premaxilla, or the upper lip (Perkins, 1977).

Very often, the "cleft palate" is surgically closed and although residual effects such as velopharyngeal incompetence and deviant hypernasal speech may be evident, the original structural deformity is
repaired. Nevertheless, the condition is still referred to by the general term "cleft palate" as is cleft of the lip and submucous cleft.
In this paper, the term "cleft palate" will be used in this general sense unless a more specific term is needed for clarity.

Cerebral Palsy

Often cerebral palsy is defined in terms of motor dysfunction alone but other times it is seen as an aggregate of handicaps. Cruickshank (1976) viewed cerebral palsy "as one component of a broader brain-damage syndrome comprised of neuromotor dysfunction, psychological dysfunction, convulsions, or behavior disorders of organic origin" (p. 2). The disorder cerebral palsy comprises, has in common motor dysfunction but differs widely in etiology, pathology and associated disorders.

A clinical classification based upon the character of the disordered movement consists of spasticity (50 to 60 percent), dyskinesia--athetosis and others (20 to 25 percent), ataxia (one to 10 percent), mixed types (15 to 40 percent), and atonia (rare). The focus of the cerebral palsy discussion here will be on spasticity, athetosis and ataxia. Topographic classification, degree of severity, muscle tonus and associated functions are a part of the clinical classification (Denhoff, 1976), but will not be referred to.

Etiology

Both cleft palate and cerebral palsy have a multiplicity of causes, some of them similar, none of them fully understood. And both have possible genetic influences that are not satisfactorily defined.
In general, the time cleft palate appears as a developmental failure is almost always during the second month of gestation, while cerebral palsy can be acquired any time from conception through two or three years after birth.

Associated Conditions

Associated conditions may interact with one another and affect speech in diverse ways for both cleft palate and cerebral palsied conditions. Darley, Aronson and Brown (1975) have stated that sensory, perceptual and intellectual problems frequently associated with cerebral palsy are likely to blur the effect of the motor impairment on speech production. Awareness of these problems is therefore necessary. Although associated conditions may be reduced in number and severity in cleft palate, they nevertheless may affect communication skills and thus, also need to be considered.

Associated conditions in cleft palate and cerebral palsy are listed in Table 1 below.
## Table 1

Associated Conditions in Cleft Palate and Cerebral Palsy

<table>
<thead>
<tr>
<th>ASSOCIATED CONDITIONS</th>
<th>CLEFT PALATE</th>
<th>CEREBRAL PALSY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Associated Anomalies</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Feeding Problems</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Motor Development</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Dental Conditions</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Personality &amp; Emotional Adjustment</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sensory &amp; Perceptual Development</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Auditory</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Visual</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>Tactile</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>Speech Development</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Language Development</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Intelligence</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**Key**

- **0** = present
- **0** = questionable whether present
- **-** = absent
Chapter 2

BACKGROUND INFORMATION

The production of speech is an extremely complex form of human behavior that depends upon the organic integrity of the central nervous system and the structure and function of the many organs comprising the speaking mechanism of the body, and also upon numerous environmental, psychosocial and developmental factors (Bzoch, 1971b). The following review of the literature examines the effects of deviant structure on the speech mechanism by looking at the cleft palate condition and the effects of disrupted organic integrity of the CNS are examined by considering the condition of cerebral palsy. Particular attention is given to the structure and function of the velopharyngeal port and tongue, and how they affect "typical" phonological patterns, nasality and intelligibility in cleft palate and cerebral palsy.

Cleft Palate Speech

Apparently, typical "cleft palate speech" does not exist. Morris (1971) wrote that the cleft palate population is so heterogeneous in variables relating to communication skills that it is difficult to meaningfully describe "cleft palate speech." Not only does real variation of articulation skills and speech patterns in this population exist, but research is so confounded with varying terminology, classifications and measurement procedures that valid comparison of different studies is rarely feasible. Aware of these shortcomings, the nature
of "cleft palate speech" is briefly described in the following para-
graphs in relation to etiological factors.

Nature and Etiology

The nature of cleft palate speech is discussed in the paragraphs
below by first examining the possible structural etiologies such as
velopharyngeal incompetence, dental abnormalities and clefts of the
lip, and then by reviewing various etiologies not directly related to
structural anomalies.

Velopharyngeal incompetence. Velopharyngeal incompetence occurs
commonly in the cleft palate population and is considered the most
important etiological factor in its speech. Incompetent velopharyngeal
closure can result from clefting, submucous cleft, disproportionate
soft palate and pharynx, immobile soft palate due to scarring, neuro-
motor dysfunction, trauma or surgical intervention, faulty learning
(functional), or a combination of these factors.

The two most frequent consequences of velopharyngeal incompetence
affecting speech production in cleft palate speech are hypernasality
and problems with ineffective oral pressure build-up.

Hypernasality, an excessively undesirable amount of perceived
nasal cavity resonance during the phonation of vowels, is generally
considered the major deviation in cleft palate speech disorders.
However, Bzoch (1971a) found the incidence less prevalent than other
factors such as laryngeal and pharyngeal fricative substitutions and
delayed speech and language development.

Velopharyngeal closure and degree of perceived hypernasality are
generally agreed to be related, although a one-to-one direct relationship between them has not been found. For example, Subtelny et al. (1961) reported that 32 percent of the non-nasal adult cleft palate speakers did not have complete closure. Bzoch (1971b) stated that certain phonatory variations, certain articulation errors, and certain distortions of voice quality all generally unrelated to velopharyngeal insufficiency appear to influence perceptual judgements of nasality.

A second consequence of velopharyngeal incompetency has to do with adequate oral pressure build-up for consonant production. Oral pressure is necessary in the production of speech. Cleft palate speakers misarticulate most frequently on those sounds for which oral pressure is highest for normal speakers, namely fricatives, affricates and plosives, with fricatives being the manner of production most difficult (Subtelny and Subtelny, 1959; McWilliams, 1958; and McWilliams and Musgrave, 1971). This suggests that adequate air pressure is not built up in the oral cavity. The most reasonable explanation for this lack of pressure is an abnormal air leak through the velopharyngeal valve during articulation (Spriestersbach and Powers, 1959). As with hypernasality, no direct one-to-one relationship has been found between velopharyngeal adequacy and oral pressure build-up, but Spriestersbach and Powers found almost without exception that subjects unable to develop adequate breath pressure, showed marked deficiencies in articulation. A greater degree of closure appears necessary for intelligible production of fricatives than for other phonetic groups (Subtelny et al. 1961).

Compensatory movements for this inability to build up air pressure
in the oral cavity manifest themselves in various ways. One way is the use of pharyngeal and laryngeal fricative and plosive substitutions which can be explained in terms of a universally valid law concerning articulation: "In the presence of a cleft palate, it is physically impossible to produce distinct plosive and fricative sounds at the places of articulation that lie distally to the deficient palatal valve." (Luchsinger and Arnold, 1965, p. 675). Therefore, the articulation place tends to shift centrally from, or below the deficient palate. Only the pharyngeal zone between the tongue base and pharyngeal wall, and the laryngeal zone between the vocal cords, or in the laryngeal vestibule are available for this shift. Resultant substitutions are glottal stops, pharyngeal stops, and pharyngeal and laryngeal fricatives. These substitutions which attempt to valve the airstream at a point in the respiratory tract nearer the source of air pressure than the velopharyngeal port are learned behavior, compensatory in nature. Such behavior may persist even after physical management of the incompetent velopharyngeal port has been achieved (Bzoch, 1971a). Bzoch found that these laryngeal and pharyngeal substitutions were the commonest definable category of abnormal speech in cleft palate.

Another compensatory movement which can occur singly or with the substitutions already mentioned is nasal emission. When there is velopharyngeal closure and when the nasal passage is occluded, air pressure is directed through the oral cavity. Hence the air flow in the oral cavity can be temporarily occluded, then released, for the production of fricatives. Without velopharyngeal closure, air pressure that would
normally be directed through the mouth during the production of a plosive, fricative or affricate will escape from the oral cavity into the nasal cavity. This nasal emission of air pressure may be audible and may distort the acoustic signal of the speech sound (Morris, 1971).

Generally, the major determinant to the amount of nasal emission is the size of the velopharyngeal opening (Morris, 1971; Bzoch, 1971b; and McWilliam and Musgrave, 1971), however, certain writers have pointed out that caution in interpreting the relationship between the two should be practised. Peterson (1975) reported that persistence in oral-nasal misarticulation may be a residual of earlier velopharyngeal incompetency; and Bzoch (1971b) stated that an anterior fistula may also result in nasal air flow unrelated to the velopharyngeal mechanism. Also, nasal emission could be present but undetected due to a weak voice or a cold blocking the nasal passages.

Some speakers develop a nasal grimace, that is, the narrowing of the nares, perhaps in an attempt to prevent, at the nostrils, the emission of air pressure which they cannot prevent at the velopharyngeal port (Morris, 1971). Also possible, although rare (Morley, 1970), is a nasopharyngeal snort, most frequently associated with fricatives which is produced by the passage of air through a closed sphincter but not so tightly as to prevent the passage of air as on snorting.

**Dentition and occlusion.** Bzoch (1971a) concluded that the presence of cause-effect relationships between dental and occlusal hazards and speech production was not predictable. Dental deviations in isolation seldom impose severe problems on articulation, but when other deficits occur also, their disturbing effect on speech is
greatly enhanced (Starr, 1971).

Bzoch (1971a) found that unilateral or bilateral lisping, particularly evidenced on the /s/ sound was the single most common error related to dental abnormalities. However, other factors can cause lisping. For example, lateralization can also be a result of an anterior fistula.

Cleft lip. Cleft lip usually has little or no effect on speech. If the surgically repaired cleft lip is of sufficient size and mobility to allow opening and closing the mouth, articulation problems are probably not significant (Morris, 1968).

Tongue movement and sensation. If an individual with cleft palate uses pharyngeal and velar fricatives, the tongue is likely to be habitually retracted resulting in reduced tongue tip and blade movements. Or if there is a large fistula in the hard palate, the tongue may function to close the gap resulting in a high carriage, that is, a forward carrying of the tongue. Laurence and Phillips (1975) reported that some speakers who have a history of palatal problems use deviant lingual contacts to produce consonant sounds. They found the greater the inadequacy of the velopharyngeal mechanism, the more deviant lingual contacts used. Also, use of deviant lingual contacts was significantly related to the presence of abnormal intelligibility and abnormal nasal resonance. Therefore they concluded that these compensatory lingual contacts reduced intelligibility and increased hypernasality.

Matthews and Byrne (1953) found gross tongue movements similar
in normal and cleft palate speakers, however fine speech movements were quicker in the normal speakers. They concluded that their results do not support the hypothesis that the cleft palate group had an overall inferior tongue flexibility but only that they are slower in rate of sound production.

Ringel et al. (1970) found that subjects with articulatory defects made more errors on oro-sensory discrimination tasks than did subjects with normal speech patterns. Furthermore, sensory errors tended to increase as a function of severity of articulation defect. However, these tasks are not requisite to normal speech production and the validity of these tasks is uncertain (Ringel, 1970).

Acquired conductive hearing loss. Hearing loss of a conductive type is more prevalent in cleft palate than the general population but impairs the development of articulation proficiency for only a relatively small percentage of these children (Morris, 1968). However, if a loss does develop it is likely to occur at a young enough age to hinder the acquisition of articulation skills.

Etiology not directly related to structural anomalies. Various influences may be present in cleft palate speech patterns that are not directly related to the structural deviation. McWilliams and Musgrave (1971) reported that a slow maturation of consonants occurs in cleft palate children not to be confused with consonant deficiencies resulting from velopharyngeal valving problems. Other agents such as emotional factors (Wirts, 1971), intelligence (Lewis, 1971), as well as structural problems other than the velopharyngeal relationship might
contribute to the total speech picture. Also, the habituation of inappropriate speech postures often persist long after the anatomical deviation has been corrected. Bzoch (1971a) stated that this inappropriate learned behavior exists as a major reason for articulation problems. He felt the insidious nature of many of these early compensatory articulation patterns might be due to early habituation of extremely atypical neuromuscular conditioned motor patterns. This manner of early speech behavior involves efferent neuromuscular pathways completely different than those utilized in normal emerging speech patterns in early childhood. Bzoch believed this to be a good reason for the relative difficulty of modifying such patterns after they have been reinforced over any considerable period of time.

Aspects of "cleft palate speech" that have been discussed are summarized in Table 2 below.

Table 2
Categorical Aspects of Deviant Speech in Cleft Palate (Bzoch, 1971a)

<table>
<thead>
<tr>
<th>Speech Aspect</th>
<th>Number Affected Out of 1000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glottal stop, velar, pharyngeal &amp; laryngeal fricative substitutions</td>
<td>564</td>
</tr>
<tr>
<td>Delay in speech &amp; language</td>
<td>502</td>
</tr>
<tr>
<td>Hypernasal resonance</td>
<td>431</td>
</tr>
<tr>
<td>Nasal emission causing consonant distortion</td>
<td>423</td>
</tr>
<tr>
<td>Developmental dyslalia</td>
<td>340</td>
</tr>
<tr>
<td>Dysphonia characterized by aspirate voice</td>
<td>313</td>
</tr>
<tr>
<td>Dysphonia characterized by hoarseness</td>
<td>150</td>
</tr>
<tr>
<td>Lisping &amp; other articulation distortions related to dental &amp; occlusal abnormalities</td>
<td>128</td>
</tr>
<tr>
<td>Hyponasal resonance</td>
<td>120</td>
</tr>
<tr>
<td>Articulation deviations due to loss of hearing</td>
<td>60</td>
</tr>
<tr>
<td>Communication problems from visual distraction of the listener due to nasal &amp; facial grimacing</td>
<td>42</td>
</tr>
</tbody>
</table>
Vowel and consonant patterns are presented below and then the types of misarticulations and the consistency of errors will briefly be discussed.

Vowels. Although often nasalized, vowels are not greatly deviant for the cleft palate population when judged only on the basis of phonemic characteristics.

Consonants. (1) Manner of production. Fricatives, affricates and plosives are the most defective sounds in cleft palate speech while nasals and glides are the least affected (Moll, 1968). Fricative errors are the most prevalent (Subtelny and Subtelny, 1959; Byrne, Shelton and Diedrich, 1961). The fricative /s/ is the phoneme most frequently and consistently in error (McWilliams, 1958) and is considered the most sensitive indicator of velopharyngeal valving adequacy (McWilliams and Musgrave, 1971).

(2) Place of articulation. Sounds which involve lingual contacts are much more defective than are sounds which involve only the lips in cleft palate speakers. Among sounds which involve lingual contacts, articulation proficiency does not appear to change consistently with the anteroposterior placement of the contact (Moll, 1968). However, more anterior valves tend to be replaced with "back of the tongue" plosives or fricatives which sound more deviant than anterior valves.

(3) Voicing. In cleft palate speech, voiceless consonants are more defective than voiced ones (Moll, 1968). This is probably because voiceless consonants require a greater intraoral breath
pressure than do their voiced cognates and this, of course, is a problem in cleft palate.

Types of misarticulations. Distortions of consonant sounds are considered the most outstanding characteristic of cleft palate speech (McWilliams, 1958; Morris, 1971). Fricatives, because of their long duration, are subject to the most distortion. Distinctions between distortions, substitutions, and omissions are not always easy to discern. But, in general, a distorted sound tends to have the same place of articulation as the test sound, and a substituted sound tends to have the same manner of production as the test sound. For example, nasal emission produces consonantal distortions and glottal stops are frequent substitutions for plosives. The prevalence of omissions may be less than reported since when any attempt to produce a sound is made, it is not omitted.

Consistency. In general, articulation of consonants is highly inconsistent within and between cleft palate speakers. Individuals are relatively inconsistent in articulation of specific speech sounds and in most cases the correct sound is produced some of the time. However, speakers who substitute the pharyngeal fricative seem relatively consistent in their use of it. Similarly, distortion by nasal emission is less variable within speakers.

Intelligibility

Percentages of individuals with normal speech range from 0 to 90 percent in the literature which renders this data virtually meaningless (Moll, 1968). Moll selectively considered the data and concluded that
55 to 80 percent of individuals with surgically managed cleft palates achieve normal speech.

**Velopharyngeal Adequacy**

Incidence of nasality is similarly disparate. Nasal voice quality is more prevalent in the cleft palate population than in the noncleft population, exact quantification cannot be reliably given. Although conflicting results are reported in the literature, Moll (1968) concluded for a priori reasons that a real relationship exists between nasality and articulation in cleft palate speakers. However, many findings show that no one-to-one correlation exists between (hyper)nasality and articulation proficiency or intelligibility (Subtelny, Koepp-Baker and Subtelny, 1961; Subtelny and Subtelny, 1959).

The review presented suggests that for the cleft palate population velopharyngeal incompetence is the most prevalent etiological factor for deviant speech, but that no one-to-one relationship exists between various attributes of production such as hypernasality, nasal emission or intelligibility, and the amount of velar insufficiency.

**Cerebral Palsied Speech**

If some question as to the reality of a typical "cleft palate speech" was posed, then, the existence of a general "cerebral palsied" speech is even more doubtful. Lencione (1968) stated that essentially there is no speech problem that is uniquely characteristic of the cerebral palsied child per se. Rather, speech problems may range from very mild disorders to severe impairment dependent on the extent and
range of the neuromuscular, neurosensory, and psychosensory damage. Morley (1972) felt that the extent of brain damage could not be considered a useful index in predicting severity of dysarthria since a severe dysarthria can occur in children who have no obvious signs of cerebral injury and alternately little or no speech involvement may be present in the face of massive brain damage. If the extent of the lesion is not particularly diagnostic of dysarthria in cerebral palsy, then perhaps the site of lesion is more telling. And indeed, in theory, discrete lesions in various functional systems within the CNS are thought to produce specific speech characteristics. That cerebral palsy entails a number of these systems, makes it difficult to discuss the speech of this population as a unitary disorder. Furthermore, lesions are often mixed, obscuring cause and effect in speech patterns even more. Mecham et al. (1966) reported that even within any one type of cerebral palsy, problems are so diverse as to almost preclude the possibility of a composite picture.

Not only are there varying speech characteristics as a result of different parts of the brain being damaged to different extents and in various mixed lesions, but also associated conditions are present in varying combinations and intensities, and differentially affect speech acquisition and production. Unlike the cleft palate condition, neurological dysfunction affects the entire process of speech production. Respiration, phonation, resonance and articulation can all be affected. Whether a deficit is due to an articulatory problem, faulty respiration function or phonatory deficit is often not evident. Similarly, opinions differ on whether aberrations are due to the
dysarthria or compensatory movements.

With these complications in mind, cerebral palsy is discussed below in terms of a syndrome consisting of disrupted neuromuscular function with abnormal reflex behavior. Disturbances in posture and movement for the three main types of cerebral palsy are summarized in table form. Finally the nature and etiology of dysarthria in cerebral palsy and the resultant phonological system are described.

Neuromuscular Function and Dysfunction

Human voluntary movements including speech acts entail complex functional systems. Many neuromuscular influences on the executive function of speech serve to direct, adjust, coordinate and monitor speech production. While neurologists differ in their beliefs on various issues in brain functioning in normals and those with brain injury, most writers agree that impairment of neuromuscular function occurs in cerebral palsy as a result of brain damage. Neurphysiology of motor speech control is beyond the scope of this review, but in general terms, Melyn and Grossman (1976) stated:

Movement is a series of postures produced by various stimuli... It is the equilibrium of input and output within the central nervous system which produces the desired movement or lack of motion. Any disharmony between forces produces an imbalance resulting in an exaggeration of one force, or release of an opposing force, previously silent. (p. 74).

This imbalance may occur in cerebral palsy.

One imbalance that is particularly prevalent in cerebral palsy has to do with reflex behavior. Normally, as myelination of the fiber tracts proceeds in the human nervous system, primitive early reflex patterns become increasingly subservient to higher cortical centers.
When this occurs, the "pure" reflex can no longer be elicited, and the resulting movement or posture may be the same movement but now mediated by higher cortical functions, that is, volitional acts. Pathologically, some reflexes are never normal and others do not appear but most of the dysfunction in cerebral palsy relates to the persistence or exaggeration of these subcortical reflexes beyond the expected age of disappearance, due to lesions within the cerebral cortex and pyramidal and extrapyramidal systems. The presence of these primitive reflexes constitute dysfunctions of postural mechanisms in cerebral palsy that affect movement including motor speech control. When one primitive reflex persists, this may prevent the next stage of development and may create abnormal movement patterns and postural sets in the infant (Gesell and Amatruda, 1947). For example, babbling, the precursor of speech, often is delayed in cerebral palsied children because of the persistence of reflex behavior which prevents the free selection of movements. The child is therefore unable to experiment with sounds. Motor abilities are reduced due to restriction of movement and weakness, and accordingly sound-movement associations are reduced.

Prominent features of the three main types of cerebral palsy summarized from Melyn and Grossman (1976) and Darley, Aronson and Brown (1975) are presented below in Table 3.
Table 3
Prominent Features in Three Types of Cerebral Palsy

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>SPASTICITY</th>
<th>ATHETOSIS</th>
<th>ATAXIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>lesion</td>
<td>pyramidal tract</td>
<td>extrapyramidal tract</td>
<td>cerebellum</td>
</tr>
<tr>
<td>main problem</td>
<td>tonicity and hyperactivity of</td>
<td>inconsistency, involuntary movements and</td>
<td>inaccuracy of movement and lack of</td>
</tr>
<tr>
<td></td>
<td>stretch reflex</td>
<td>distorted postures (overflow of activity)</td>
<td>direction control (incoordination)</td>
</tr>
<tr>
<td>muscle tone</td>
<td>increased (hypertonic)</td>
<td>fluctuates (waxes and wanes)</td>
<td>decreased (hypotonic)</td>
</tr>
<tr>
<td></td>
<td>possible hypotonus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>reflexes</td>
<td>hyperactivity of stretch</td>
<td>retained primitive reflexes</td>
<td>dysfunction of postural reflexes</td>
</tr>
<tr>
<td></td>
<td>reflexes and retained primitive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>movement</td>
<td>weak and slow with face, tongue</td>
<td>weak, slow, writhing, and inconsistent with</td>
<td>weak, slow and dysrhythmic with difficulty</td>
</tr>
<tr>
<td></td>
<td>and distal areas more affected</td>
<td>face, tongue and distal areas more affected</td>
<td>in maintaining posture and</td>
</tr>
<tr>
<td></td>
<td>than proximal areas</td>
<td>than proximal areas</td>
<td>equilibrium</td>
</tr>
<tr>
<td>range of</td>
<td>limited</td>
<td>inconsistent</td>
<td>restricted in some</td>
</tr>
<tr>
<td>movement</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Nature and Etiology

Within cerebral palsy, there are fairly distinctive dysarthric features for the different types. However, deviant speech characteristics are similar in many respects too, and in the final analysis, it may be difficult to distinguish by listening alone, one type of cerebral palsy from another. In addition, many individuals with cerebral palsy have a mixed type. For these reasons, dysarthric patterns for spasticity, athetosis and ataxia are not discussed separately.
Respiration. Several aberrations of breathing patterns are seen in cerebral palsy which interfere with feeding, and speech acquisition and production (McDonald and Chance, 1964; Mecham et al., 1966; Lencione, 1976). The breathing rates of all young babies are very rapid and irregular. The cerebral palsied child frequently retains his or her infantile rapid rate for many years which might make vocal play, the antecedent of speech, difficult to indulge in. Feeding problems often arise from incoordination of nasal breathing and swallowing.

Weak musculature, spasmodic muscle contractions, limiting the range of chest expansion, or a lack of coordination between different muscle groups can result in reduced vital capacity and shallow breathing which may manifest themselves in short phrasing and/or reduced intraoral breath pressure for the production of consonants. Involuntary movements in the respiratory musculature cause irregular breathing patterns which result in variations in vocal loudness and interruptions of vocalizations. A lack of coordination between agonist and antagonist musculature may cause "reversed breathing" where phonation is attempted after exhalation. Initiation of vocalization is consequently difficult. Also, an insufficient air supply reduces one's ability to sustain phonation. Sometimes in athetosis the brain centers for respiratory control are affected as well because of the proximity of the hypothalamus and the basal ganglia. The greatest incidence of breathing anomalies is considered to be in athetosis, then spasticity, and finally, ataxia.
Phonation. Any or all of the attributes of voice (pitch, intensity, quality, prosody) can be affected in cerebral palsy (McDonald and Chance, 1964; Mecham et al., 1966; Lencione, 1976). The laryngeal blocks which occur are usually a part of the more generalized neuromuscular dysfunction and they may be related to some of the respiration problems. When the child attempts to produce controlled exhalation for speech, the increased tension which occurs may spread to the laryngeal mechanism and produce various spasms. Initiating phonation may be difficult if the vocal folds first adduct in a spasm. During phonation, this adduction spasm may abruptly interrupt speech or result in explosive increases in loudness or abrupt changes in pitch.

When the vocal folds have increased tone, as in spasticity, this narrows the aperture and results in a harsh quality and a strained sound as though the breath stream were being squeezed through a narrowed glottis. Ataxic trembling of the whole larynx can also result in a harsh quality, that is, vocal pulse aperiodicity. A reduced range of laryngeal muscular movements causes alterations in prosody, namely monopitch and monoloudness. Slowness of movement may be demonstrated by breathiness where the vocal folds move slowly to the midline allowing air wastage. However, breathiness could also be due to an abductor spasm where the vocal folds come apart, or it could be compensatory in nature, such as economy of effort. Reduced loudness is often related to inadequate breath support.

As in respiration, there is a higher incidence of phonation aberrations in athetosis. Fluctuations in muscle tone result in
variations in pitch, that is rising intonations, or intensity or quality. Of course, the same manifestations could be a result of the inconsistent respiratory control.

Articulation and resonance. If the articulation process is viewed as valving the vocal tract in various places to impede the airstream in different manners, then generally we can say that spasticity will result in slower, less powerful movements of the articulators with a reduced range of movement and an inability to secure graded, synchronous movements. Hypotonia largely manifests itself as weakness and slowness of movement. Hypersensitivity may elicit various reflexes such as jaw jerk, bite reflex and sucking reflex that may have a detrimental effect on speech creating facial grimaces and distortions. In athetosis, the ability to make various articulatory movements may be present but this ability is inconsistent and comes and goes sporadically with fluctuating muscle tone and asynchronous muscle movement. The resultant movements are often explosive, jerky and arrhythmic with an overflow of activity. Dysrhythmia may also result from the unpredictable articulatory changes due chiefly to inaccurate direction control and inaccuracy of articulatory placement found in ataxia. Phonemes and the intervals between them are often prolonged in ataxia further contributing irregular rhythm with a somewhat more measured and more equally prosodic pattern than normal.

Articulatory movements, carried out at a reduced speed and range of movement, often consist of loose approximations with a lack of differentiation and a minimum of movement with many sounds omitted.
Mandible. If there is weakness in the mandible elevators, the mouth is often open causing drooling to be common since the mouth must be closed to swallow. Often chewing patterns are poor and articulation defective. The mandible interferes with speech production by pulling the tongue into a position which makes lingual-dental and lingual-palatal contacts difficult or impossible. In other words, the tongue's position is lower than normal and most cerebral palsied individuals have difficulties elevating the tongue so they would experience even more difficulties in this position. On the other hand, Kent and Netsell (1978) found increased range of jaw movements in athetoids could be a compensatory strategy used to achieve tongue elevation.

Muscles of the lower jaw are susceptible to stretch reflexes that cause spastic grimaces. Hyperextension of the mandible during eating and speaking, when extreme, causes mandibular facet slip (McDonald and Chance, 1964; Mecham et al., 1966).

Lips. Weakness in lip musculature will result in difficulty in protruding or retracting the lips or in difficulty maintaining bilabial approximation for feeding or articulation. If the child cannot maintain lip closure, drooling may also be a problem. When there are involuntary movements of the lips, patterns of lip movements are not predictable and the same difficulties mentioned above can arise. During a spastic grimace, the lips are retracted preventing lip closure. Similarly, alteration of muscle tone in athetoid facial distortions make sustained approximation of the lips difficult.

Tongue. The tongue, that most versatile articulator, is so well suited for the highly organized, finely incremented movements of
speech because of its finely-coordinated neuromuscular system providing muscular power, mobility and flexibility. The tongue with neuromuscular involvement will obviously be deficient on all or some of these dimensions.

Consonant productions in cerebral palsy are often omitted or distorted and this imprecision is not limited to the production of complex consonants only, even the production of isolated vowels is often distorted. Mecham et al. (1966) reported that 85 percent of sounds have some tongue elevation or retroflexion. It is therefore not surprising that aberrant tongue functioning in cerebral palsy has a marked detrimental effect on articulation and intelligibility. This detrimental effect can arise from inappropriate positioning of the tongue, reduced rates of tongue movement, and grossness of tongue shaping which may result from weakness, reduced range of movement, involuntary movements, reflexes, defective proprioception and/or incoordination.

In addition to all of these efferent processes, afferent impulses from the tongue may strongly participate in the control of tongue muscle activity. The theoretical relationship between this sensory input system and their output manifestations has led speech scientists to pose the question whether certain types of production disorders, which have hitherto been considered "motor" in origin, may in fact have an oral-sensory disturbance basis (Ringel et al., 1970). This relationship is a very complex issue and is not considered here. What is recognized is that in cerebral palsy both sensory and motor functions are defective, irrespective of their relationship to the final speech
product. Any tongue dysfunction that may have been present in cleft palate is intensified in degree and scope in the cerebral palsied population.

(4) Velum. Hypernasality is often present to a considerable degree in spastic cerebral palsy, an intermittent degree in athetosis, and is usually not a problem in ataxic cerebral palsy.

Hardy (1961) felt that velopharyngeal incompetence in cerebral palsy, in addition to hypernasality, leads to insufficient intraoral breath pressure which may contribute significantly to articulation problems. Although the tongue is almost always affected in cerebral palsy and surely contributes significantly to articulatory deficits, Hardy felt that the effects of insufficient velopharyngeal closure and resultant lack of oral pressure build-up may be obscured by the commonly held belief that the tongue is the primary causitive factor in indistinct speech in cerebral palsy.

Causes of an inability to impound oral pressure in cerebral palsy are different than the cleft palate population and may be the result of one or a combination of the following conditions:

(a) The palate may be partially or wholly paralyzed and air thereby escapes into nasal passages during speech attempts, precluding a pressure build up in the mouth.

(b) Oral pressure may not be possible due to paretic involvement or weakness of the respiratory musculature.

(c) Other articulators may be paretic or weak and therefore unable to impede the oral airstream to the extent needed for build-up of air pressure behind the tongue or lips, that is, inefficient valving. (Hardy, 1961).

In addition, any structural inadequacy may combine with marginal neurological dysfunction and create velopharyngeal incompetence.
Involuntary movements, spasms or incoordination could also cause inadequate closure. Finally, incoordination of velar closure with articulation may prevent oral pressure build-up at the appropriate time.

Netsell (1969) demonstrated the complexity of the problem of velopharyngeal valving by considering the time-varying aspect of the mechanism and the airstream. Briefly, four primary patterns of velopharyngeal dysfunction in neurological dysarthria described were gradual opening of the velopharyngeal port, gradual closing, anticipatory opening and retentive opening.

Netsell conjectured on the basis of the retentive opening patterns that the effect of velopharyngeal incompetence could contribute to the characteristic slow rate of speech associated with neurological dysarthria. He felt that slower movements of other articulators could be partly compensatory to allow time for the velum to close before the next oral consonant is produced.

Both Netsell's and Hardy's hypotheses have implications to treatment. Velopharyngeal incompetence, then, has not only been related to hypernasality in the cerebral palsied group but also to imprecise consonant articulation and reduced rate of production.

Differences in dysarthria among different types of cerebral palsy. Findings on spastic versus athetoid speech are not in complete agreement. Some studies have found differences between athetoids and spastics but others have not. Furthermore, in studies that have found differences, most find athetotic dysarthria more marked and less intelligible (Mecham et al., 1966; Lencione, 1976) but at least one study found spastic speech to be more involved (Clement and Twitchell, 1959).
Kent and Netsell (1978) said that studies very seldom make an effort to equate the athetoid and spastic groups for severity of the speech impairment or overall neuromuscular involvement. Mecham et al. (1966) concluded that cerebral palsy speech tends to be slow, jerky and irregular, labored or effortful and rather unintelligible. Because of the extreme variability within cerebral palsy types, generalizations about different types are not reliable.

Nevertheless, Table 4 is a consolidation of some of the aspects discussed concerning dysarthria in cerebral palsy as it relates to the three different types: spastic, athetoid and ataxic. There are many similarities among them and even though they differ on various features, the final speech product is often difficult to discern as spastic, athetoid or ataxic.

Etiology not directly related to the neurological dysfunction. Lencione (1968) reported that a substantial body of information had been accumulated indicating that the cerebral palsied child follows the same course of linguistic, including phonological, development as that of the nonhandicapped child. However, for the cerebral palsied child this development is much delayed. Mecham et al. (1966) reported a delay from two to four years is usual. Communication development depends upon development or maturation of many related areas. A certain degree of ability to control and integrate motor activities and a psychological readiness that will depend on emotional and adaptive maturation as well as intelligence are needed for the development of speech. Also perceptual and discrimination skills must develop into highly specialized sensory processes, especially within the modalities of vision, hearing and kinesthesia. Thus, many variables may obscure the dysarthria.
Table 4
Dysarthria in Three Types of Cerebral Palsy
<table>
<thead>
<tr>
<th><strong>MOVEMENT</strong></th>
<th><strong>SPASTICITY</strong></th>
<th><strong>ATHETOSIS</strong></th>
<th><strong>ATAXIA</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>slow, stiff, weak, limited range</td>
<td>slow, weak, inconsistent (fluctuates), range may be limited or increased</td>
<td>restricted tongue movements</td>
</tr>
<tr>
<td></td>
<td>indistinct articulation vowel distortion inability to secure graded synchronous movements</td>
<td>indistinct articulation vowel distortion jerky and arrhythmic</td>
<td>indistinct articulation vowel distortion jerky and arrhythmic, irregular speed and force somewhat measured: equalized prosodic pattern</td>
</tr>
<tr>
<td></td>
<td>retracted tongue spastic grimace</td>
<td>athetosis of tongue facial grimace: fluctuating muscle tone</td>
<td>drooling</td>
</tr>
<tr>
<td></td>
<td>drooling hypernasality</td>
<td>drooling intermittent hypernasality insufficient postural fixation: distorted postures for articulation</td>
<td>positional and directional orientation difficulty, lack of direction control</td>
</tr>
<tr>
<td><strong>PHONATION</strong></td>
<td>harsh labored, strained breathiness monopitch, monoloudness explosive increase in volume and pitch</td>
<td>harsh breathiness variations in quality, volume and pitch difficulty initiating vocalization interruptions in vocalization and volume</td>
<td>harsh labored monopitch, monoloudness excess loudness variation</td>
</tr>
<tr>
<td></td>
<td>reduced loudness</td>
<td>reduced loudness explosive, arrhythmic inconsistent</td>
<td>upward and downward sweeps in pitch measured, equalized prosodic pattern</td>
</tr>
<tr>
<td><strong>RESPIRATION</strong></td>
<td>shallow breathing short phrasing reduced intraoral pressure</td>
<td>shallow breathing short phrasing reduced intraoral pressure reversed breathing explosive, arrhythmic thoracic deformity inconsistent and uncontrolled</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Phonological Summary

The various etiologies that have been presented may manifest themselves in a cerebral palsied child's phonological system in various ways.

Vowels. Vowel distortions are present in all types of cerebral palsy.

Consonants. (1) Manner of production. In general, sounds requiring fine coordination are the most difficult for cerebral palsied individuals. Lencione (1976) and Mecham et al. (1966) found nasals least difficult (in initial and final positions) and fricatives, affricates and glides the most difficult (in all positions) to produce. Stops seemed least difficult when in the medial position of a word.

(2) Place of articulation. Mecham et al. (1966) reviewed the literature and reported that tongue tip sounds and especially those which require fine coordination were the most difficult for cerebral palsied speakers. Tongue tip simple sounds /n d t/, lip and back of the tongue sounds were easier than tongue-tip complex sounds /s z t d j/ (Lencione, 1976). Lingual-dental sounds /θ ø/ and alveolar sounds were difficult due to tongue protrusion and elevation difficulties found in cerebral palsy (Clement and Twitchell, 1959). Labials and velars were also found to be difficult.

(3) Voicing. Voiced consonants tend to be less difficult for the cerebral palsied population than voiceless cognates.

Types of misarticulations. Lencione (1976) and Mecham et al. (1966) found that omissions significantly exceeded distortions or
substitutions in cerebral palsy. The prevalence of omissions was greatest in final, medial and then initial, respectively. Types of misarticulation are influenced by phonemic context. Generally, a sound is produced in the easiest possible way and this behavior continues even though it may not be the best a person can do.

**Consistency.** Predominantly spastic involvement appears to evidence a relatively consistent error pattern in cerebral palsy. Inconsistency is one of the hallmarks of athetosis, phonological errors wax and wane as does the muscle tone and involuntary movements. Similarly in ataxia, lack of direction control and incoordination manifest themselves inconsistently in various substitutions and distortions for a single phoneme. There is such variation within the cerebral palsied population that it is difficult to describe any inter-group consistency.

**Intelligibility.** The incidence of cerebral palsied individuals with normal speech varies from study to study but it appears that about 70 to 80 percent of this group have some type of defect in articulation (mild to severe) which may be either functional or organic in origin (Mecham et al., 1966). Seventy-one percent of the athetoids and 31 percent of the spastics had unintelligible speech in Lencione's study (1954, as cited in Mecham et al. 1966). Wolfe (1950, as cited in Mecham et al., 1966) reported that 45 percent in his study had speech which was only partly understandable or was not understood at all.

**Velopharyngeal adequacy.** Velopharyngeal dysfunction in cerebral palsy is infrequently studied and therefore incidence figures are rare.
Hardy (1961) cited two studies that reported velopharyngeal closure problems. One found incidence in the cerebral palsied population to be 13 percent and the other 39 percent. Hardy felt that the latter incidence was more representative since a refined diagnosis of velopharyngeal adequacy had been used. Clearly, a need exists for well-defined research in this area.

Dysarthria in cerebral palsy is a difficult topic to delineate due to involvement in the multiple representation on many different levels of the motor speech process. Primary features are often obscured by many contributing influences. Cerebral palsy is a symposium of handicaps.

Some of the abnormalities in speech production which have been reviewed in cleft palate and cerebral palsy will next be compared with one another in order to obtain a better understanding of the nature and scope of both conditions.
Chapter 3

THE PROBLEM AND INVESTIGATIVE METHOD

Statement of the Problem

The purpose of the present descriptive study is to delineate differences and similarities between articulation and phonological systems of the two conditions, one structural, the other, neurological, in order to obviate contributing etiological factors from structure and function. Such comparisons drawn from research and clinical reports in the related literature can only be inferred and because of varying methods of investigation employed and the fact that the two populations are rarely, if ever, studied conjointly, one may not speak conclusively of their likenesses and differences.

The present investigation provides a model to avoid these methodological variables by evaluating samples from each population through the use of the same criteria, examiner and procedure. The findings are not expected to yield firm conclusions, because of the sample size. Rather, the findings are expected to determine if more extensive research is warranted utilizing the methodological model of this study.

The structural condition cleft palate is predominantly a focal disturbance with most articulatory deviations arising from a structurally inadequate velopharyngeal relationship. Conversely, cerebral palsy, a neurological disorder affects all of the processes of speech
production, not only the velum. Although both may have velopharyngeal incompetency, one due to a focal structurally insufficient closure and the other due to a generalized neurological dysfunction, it would seem that the respective effects on articulation and phonological systems, while having similarities, would essentially be different.

Speech errors arising from a structurally inadequate velum would be expected to produce consistent deviations since the structural condition is a given, unchanged phenomenon. Neurological dysfunction, on the other hand, is not always the same. For example, muscle tone varies in athetosis. Therefore inconsistency of error would be predicted for cerebral palsy.

Both groups could be hypothesized to have the most difficulty with high pressure consonants such as the fricatives and affricates since velopharyngeal inadequacy, whatever its origin, will attenuate adequate oral air pressure build-up. However, compensations available would be expected to be different since in cleft palate all other systems are adequately functioning but not so in cerebral palsy. Also, more severe involvement would be expected in the cerebral palsy population since many systems are involved.

**Method**

**Subjects**

Five children, two having repaired cleft palates and three diagnosed as cerebral palsied participated in this study. Ages ranged from 5 to 13 years. The cleft palate subjects were seen at the speech and language clinic at Manchester University and the cerebral palsied
subjects were evaluated at a school for the multiply handicapped in Manchester, England.

Subject A, a 5;6 year old female with a repaired unilateral complete cleft, evidenced a short soft palate and a wide post-pharyngeal space. A history of fluctuating conductive hearing loss was reported, and, at the time of the present testing, the subject had a cold.

Subject B was a 6;4 year old male with a repaired bilateral complete cleft, who evidenced a fistula in the hard palate. He also was reported to have had a history of conductive hearing loss.

Subject C was a 5;1 year old male who was regarded as a spastic quadriplegic with athetosis. He crawled, and was reported to be working with a walker and arm braces in physical therapy.

Subject D, a 13;1 year old female had a medical diagnosis of quadriplegia with athetosis. She was nonambulatory and wheelchair bound.

Subject E was a 13;3 year old male, and although there were very few direct signs of cerebellar involvement, was diagnosed as ataxic diplegia with right hemiplegia, right facial palsy and right eye affectation. He had had an encephalocele. The subject was seen in a wheelchair but he was reported to be ambulatory with a walking aid.

Procedure

Articulation testing. The Warner Articulation Test (WAT) was chosen to assess the speech systems of the two groups. This test elicits production of 116 words by means of picture stimuli. Initial
and final consonant sounds and clusters are assessed primarily in mono-
syllabic words but also in some polysyllabic productions. Sounds are
assessed in various phonemic contexts.

The WAT was considered appropriate for this comparative investi-
gation for several reasons. First of all, phonemes in initial and final
positions are tested in more than one context. Since the variability
of production is assessed, the consistency of an error can be examined.
Furthermore, whether a phoneme is in transition, or whether there are
any contexts where the subject produces the phoneme correctly can be
ascertained. A second reason the WAT was felt to be advantagious was
that it looks at stress and rhythm as well as articulation in longer
utterances in the section containing polysyllabic words. Some contrast
would be expected between cleft palate speech, where no difficulties in
stress and rhythm were reported in the literature, and cerebral palsy,
where inappropriate stress and rhythm behaviors have been reported for
all types of cerebral palsy. Finally, the simple and uniform phonetic
content of the WAT renders it adaptable for different ages of children
and for varying degrees of severity.

The WAT was administered to each subject in a quiet environment.
During administration, spontaneous responses were elicited but imitated
responses were accepted when a subject was unable to correctly name a
given picture. A phonetic transcription was made at the time of
administration but a tape recording was also obtained to enable the
examiner to validate questionable productions with others. The data
were then plotted on various graphs in the WAT and analyzed in terms
of a subject's phonetic inventory and phonological distribution of
sounds in initial and final positions.

Range and speed of tongue and lip movements. The range and speed of tongue and lip movements were evaluated in each subject on three types of tasks reported by Canning and Rose (1974). A tape recorder and a watch with a second hand were used. The children were given a practice session and at least two timed trials for each task, the faster one being recorded. It was explained to each child that the examiner wanted to see how quickly he or she could move his or her lips and tongue. The three tasks were:

1. Silent lateral tongue movements. Ten to and fro movements of the tongue from corner to corner of the mouth were counted.

2. Rapid repetition of six single consonant sounds. The consonants /t j k l w p/ were chosen as these require a wide range of the tongue and lip movements necessary for normal speech. The subject first imitated one of the consonants in isolation. The tester then demonstrated a series of rapid repetitions of this individual consonant and the subject practised. Ten repetitions were timed by the second hand on a watch twice.

3. Repetition of a sequence of consonants /pt k/. Each child was asked to sequence the consonants by imitation of /p At kə/. Only one of the children found this an easy task so the word "buttercup" was used for the others in place of /p At kə/. A child was asked to repeat the word many times as quickly as possible. Ten repetitions were counted for each child.

Tasks (2) and (3) are not only dependent on the range and speed of tongue and lip movements, but also the adequacy of breath support and
velopharyngeal closure since air pressure build-up is needed for the production of many of the consonants tested.

**Results**

Results of this investigation will be briefly summarized here in tabular form; and critical findings will be discussed and illustrated.

**Non-english Sounds**

Non-english sounds used by the subjects are presented in Table 5. Both groups used 8 of these 13 substitutions but in degree, the cleft palate group evidenced a higher frequency of these non-english sounds in his or her speech.

**Table 5**

Non-english Sounds Used by Subjects

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>w</td>
<td>nasal emission</td>
</tr>
<tr>
<td>l</td>
<td>lateral emission</td>
</tr>
<tr>
<td>β</td>
<td>bilabial voiced fricative</td>
</tr>
<tr>
<td>ð</td>
<td>bilabial voiceless fricative</td>
</tr>
<tr>
<td>p</td>
<td>labio-dental voiceless stop</td>
</tr>
<tr>
<td>b</td>
<td>labio-dental voiced stop</td>
</tr>
<tr>
<td>γ</td>
<td>labio-dental approximant</td>
</tr>
<tr>
<td>m</td>
<td>labio-dental nasal</td>
</tr>
<tr>
<td>ñ</td>
<td>palatal click</td>
</tr>
<tr>
<td>x</td>
<td>palatal fricative</td>
</tr>
<tr>
<td>q</td>
<td>uvular stop</td>
</tr>
<tr>
<td>y</td>
<td>uvular fricative</td>
</tr>
<tr>
<td>?</td>
<td>glottal stop</td>
</tr>
</tbody>
</table>
Manner and Place of Production

Table 6 represents the data in terms of manner and place of articulation. The format is taken from the WAT. On the top of the page is a representation of phonemes used by a normal speaker of English. Under that are the cleft palate subjects' data, after which follows the neurological group. This order will be consistent throughout. A "dash" (-) signifies an English phoneme not represented in the child's repertoire.

Table 7 shows which manner of production was the easiest and hardest for each group. Table 8 illustrates productions of the tongue-tip complex phonemes for each individual. The consistency and variability of each group is depicted in this representation.

Table 9 is a graphic representation of each subject's phonological system for initial and final consonant sounds. Again, this means of display was taken from the WAT.

Tongue and Lip Movements

The data for the range and speed of tongue and lip movements suggest the need for a more precise time measure. Therefore, results from these measures will not be presented. In general, both groups performed the tasks considerably slower than normal. In some cases the slowed rate may have reflected inadequate intraoral breath pressure and not necessarily slowed articulatory movements. For example, one cleft palate subject evidenced nasal emission on some consonants and inhaled for each production, no doubt slowing him down. Cerebral palsied subjects were often arrhythmic in their productions.
Table 6

Manner and Place of Production
<table>
<thead>
<tr>
<th>MANNER</th>
<th>PLACE</th>
<th>Bilabial</th>
<th>Labiodental</th>
<th>Dental</th>
<th>Alveolar</th>
<th>Palatal-</th>
<th>Palatal-</th>
<th>Palatal-</th>
<th>Velar</th>
<th>Other</th>
</tr>
</thead>
<tbody>
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<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>NORMAL</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>nasals</td>
<td>m</td>
<td>p b</td>
<td>f v</td>
<td>t d</td>
<td>n</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>stops</td>
<td>p b</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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Table 7

Manner of Production
Difficult and Easy

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Table 8
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Discussion

Cleft palate, a structural disorder and cerebral palsy, a neurological disturbance are similar in the respect that no typical speech patterns emerge from either group. Speech disorders in both populations vary on a continuum from mild to severe. However, on the severe end of the continuum it would be unlikely that cleft palate speech would ever reach the extreme of mutism or anarthria that is possible in cerebral palsy. Diversity in the literature within each group, making comparisons between the two populations difficult, has been discussed above. The present study undertaken was an effort to establish some uniformity in this milieu of diversity and suggests a format for future investigation.

Deviant speech in cleft palate arises fundamentally from one structure, namely the velum, and its inadequate relationship to the pharyngeal walls. This velopharyngeal inadequacy is considered to be the most important etiological factor in cleft palate dyslalia. Dysarthria in cerebral palsy, on the other hand, arises from a central neurological dysfunction which affects many or all of the organs and processes of speech. In addition to velopharyngeal inadequacy, abnormalities in respiration, phonation and other articulators often co-exist.

If phonation is deviant in cleft palate, usually the disorder is not a direct result of the condition itself, but rather secondary to it. For example, when hoarseness is present in a cleft palate speaker, it may be due to habitual glottal articulation which is a compensatory articulatory substitution used when there is velar inadequacy.
Hoarseness and harshness are thought to be the result of lingual, pharyngeal and laryngeal muscular imbalances associated with glottal articulation. Conversely, phonatory aberrations in cerebral palsy are usually due to the motor disorder itself. For example, spastic involvement in the laryngeal musculature may result in harshness, perhaps acoustically not unlike the harshness in cleft palate. However, it is in this case, a result of the increased muscle tone of the vocal folds which leads to a narrowed glottis and a resultant aperiodicity of the vocal tone. The increased muscle tone is not localized but rather permeates all the motor speech processes, and indeed motor processes throughout the body. An example of this overflow of activity was demonstrated when Subject D of this study, an athetoid, was attempting to lateralize tongue movement from corner of the mouth to corner (Task 1). The concomitant involuntary head movements with this exercise were considerable and probably contributed to the markedly reduced rate of lateralization of this task.

This difference, namely a focal versus a generalized disturbance is significant in the comparison of cleft palate and cerebral palsied speech and was hypothesized to effect consequential differences in the phonological systems of both conditions. This is discussed below by comparing the structure and function of the velum and tongue in both populations and then examining aspects of their phonological systems.

**Velopharyngeal Function**

Velopharyngeal inadequacy and hypernasality are more prevalent in cleft palate and cerebral palsied speech than in the normal population. And it appears that velar inadequacies are more common in cleft palate than
Where there is a structural deviation as in cleft palate such as a velum that is too short or a pharynx that is too wide, the velopharyngeal inadequacy is constant. Consequently, the resultant "nasal leak" is consistent. Certainly, functional contributions to the problem, phonetic context or speech rate alter the nasal resonance effect; but given these variables, the velopharyngeal measured relationship is the same.

This may not be the case in cerebral palsy. There are many factors which can have an effect on velopharyngeal closure or lack of closure, making it an "irregular leak". Reflexes, spasms, involuntary movements, and incoordination can all cause inconsistent velopharyngeal approximation. In addition, Netsell (1969) described four major different opening and closing patterns that would render nasal resonance inconsistent depending on phonetic context.

Hypernasality, then, is usually perceived as consistent in cleft palate speech and may be inconsistent in cerebral palsied speech.

The two categories, however, are not mutually exclusive. The absence of neurological influences in a structural disorder should not be assumed. Shelton (1971) felt that the diagnostician should consider possible deviations in neural function resulting from cleft surgery. Also, deviations in neural function which are related to congenital palatal insufficiency should be considered. Similarly, the possibility of structural deviations should not be precluded from contributing to hypernasality in cerebral palsy. Subject C, one of
the cerebral palsyed children provided a good example of this. According to the medical records, hypernasality was not a problem for this child until he had his adenoids removed. It appears that previously, Subject C was approximating closure with the benefit of the added mass of the adenoids. When these were removed, a probably weak soft palate was unable to compensate for the absence of the adenoid mass. So although neurological involvement is primary, the structural alteration was enough to reduce the efficiency of the velopharyngeal valve.

Oral pressure build-up which requires an intact velopharyngeal mechanism, has been shown to be a problem in both conditions. Furthermore, the consonants in English, the fricatives and affricates, that require varying degrees of oral pressure build-up, have been reported to be the most defective in both groups and were found to be the most defective in the present study. However, there are differences too; for in the cleft palate group, the main problem is directing the airstream, usually there is sufficient breath support and sufficient lingual coordination to produce the correct sound but the airstream escapes the oral cavity; but in cerebral palsy there may also be insufficient breath support due to respiratory involvement, or weakness or incoordination of other articulators so that they inefficiently impede the airstream, as well as nasal escape of the airstream. Obviously, the task of assessing such variables is not easy.

Sufficient but misdirected air pressure in cleft palate speech is evidenced in their laryngeal and pharyngeal fricatives and plosives, and in audible nasal emission. The air pressure is appropriate, only
is not directed through the oral cavity. The substitutions appear to be compensatory in nature. Often they are produced in the same manner as the sound they are replacing only the location of constriction of the breath stream has been shifted distally to the deficient velar valve. These types of substitutions, that is, laryngeal and pharyngeal fricatives, are not at all typical of cerebral palsy speech patterns and suggest that there may not be an adequate breath stream to produce these pressure sounds elsewhere. Not only is vital capacity often reduced in cerebral palsy, but the entire process from inhalation to exhalation to phonation to articulation is often asynchronous and uncoordinated resulting in inefficient valving of the available air stream throughout the entire process.

This is illustrated in the present investigation. Both cleft palate speakers evidenced nasal emission and one of them used a number of uvular and glottal substitutions. The cerebral palsied children, on the other hand, demonstrated no instances of nasal emission or pharyngeal substitutions. Glottal stop was used occasionally by one cerebral palsied speaker. Subject B (cleft palate) evidenced nasal emission on all sibilants while Subject C (cerebral palsy) substituted /t/ and /d/, simpler tongue tip sounds, for sibilant productions.

In both cleft palate and cerebral palsy the hypernasality or nasal emission could also be due to functional reasons. This would, however, be more likely in cleft palate since very often an inadequate mechanism may at some point in time be corrected by surgery and the previous behavior may still persist because of abnormal efferent neuro-motor pathways. Whatever the reason, Bzoch (1971a) felt that
inappropriate learned behavior exists as a major reason for articulation deviations among cleft palate speakers. Although functional problems can also be found in cerebral palsy, they probably would not be as prevalent since the neurological dysfunction cannot be corrected by surgery or any other method.

In short, velopharyngeal inadequacy in cleft palate manifests itself as an abnormal but regular phenomenon whereby the airstream is misdirected to other places of articulation. In cerebral palsy, an abnormal and irregular leak is often present and here it is not only a matter of misdirected airstream but could also be a reduced airstream and inefficient valving elsewhere.

Tongue Function

The function of the tongue and its possible efferent and afferent roles is not clearly understood. Investigators would like to show that various stereognostic and two-point discrimination tests are related to articulation ability and therefore invaluable in diagnostics and implications for therapy. But at this point, work is still experimental and results are inconclusive. That these afferent capabilities relate to articulatory proficiency seems a reasonable hypothesis. If this were the case, both cleft palate and cerebral palsied individuals would show deficits in afferent abilities, especially the cerebral palsied population.

Both conditions demonstrate deviant tongue carriage. In cleft palate a posture may be developed to compensate for inadequate velopharyngeal closure or an anterior fistula. But in cerebral palsy, abnormal tongue posture occurs as a result of the central motor...
disorder. For example, a spastic tongue may be retracted arising from hypertonicity; or if the mandible is weak, the tongue carriage may be low. Unlike cleft palate, tongue position does not seem to be compensatory in cerebral palsy. In both conditions, an abnormal tongue carriage may result in a reduced range of movement but in cerebral palsy, further reduction and restriction in movement arises from weak, stiff, uncoordinated, and athetotic muscular control. In cleft palate, an abnormal carriage may result in restricted tongue tip movement thus reducing the possibility of laying down normal neuromuscular pathways for the fine coordination with which this highly innervated articulator is endowed. Matthews and Byre (1953) found that their cleft palate subjects demonstrated adequate speed for gross movements and reduced speed for fine speech movements. They felt that the "cleft palate tongue" was misrepresented in the literature and that it may be slightly slower than normal but not reduced in overall flexibility and mobility.

In cerebral palsy, the tongue is often considered its most involved articulator. Reduction in strength, mobility, and flexibility are present in various degrees and combinations. These comprise an essential part of the condition and are not compensatory in nature. Tongue movement is so restricted that even vowels are affected. Usually vowels are considered to be less susceptible to abnormality being relatively open, unrestricted sounds. Infants vocalize using vowels long before consonants. All types of cerebral palsy have involvement with tongue functioning and vowels can be affected in all of them. Two of the three cerebral palsied children tested (Subjects
C and E) dentalized lingual-alveolar sounds and did not elevate their tongue tip to the alveolar ridge at all. In addition, Subject E evidenced vowel distortions and marked incoordination with complex tongue tip sounds. Subject C was unable to effect tongue protrusion and Subject D evidenced involuntary tongue protrusion on inappropriate sounds, for example the /l/ was interdental. The two cleft palate subjects demonstrated none of these problems. But one of them, Subject B did evidence lateralization of the airstream on certain sounds. Since Subject B has an anterior fistula in the hard palate, this lateralization of the airstream could be compensatory in origin since the tongue tip may be used to block the fistula.

Summarily, the tongue is almost always involved in cerebral palsy and may be for some but to a lesser degree in cleft palate. In a neurological dysarthria, tongue involvement is part of the whole syndrome while in a structural dyslalia, tongue malfunctioning is secondary to the structural condition. Both the scope and degree of involvement in cerebral palsy far exceeds that in cleft palate.

Other Articulators

Seldom are any other articulators affected in cleft palate. Repaired lip clefts rarely affect production of labial consonants. Conversely, other articulators such as the mandible and lips often are a part of the neurological pathology in cerebral palsy. Subject C, in the cerebral palsied group, evidenced a number of non-english labiodental sounds because of weakened oral musculature which made labial approximation difficult. In addition, abnormal reflex activity, involuntary movements, and spastic retraction of the lips can
interfere with articulation.

A spastic grimace, such as retraction of the lips, and an athetoid grimace, such as fluctuating muscle tone, are not under the control of the speaker, and can interfere with speech production. In cleft palate, a nasal grimace, constriction of the nares for example, occurs sometimes but here, it is used as a learned compensatory strategy to try to prevent nasal escape of air. Thus facial grimaces are a part of the condition in cerebral palsy and secondary to the condition in cleft palate.

In cerebral palsy the lower jaw may be weak, and therefore depressed, resulting in decreased efficiency for speech production. For example, elevation of the tongue and lip closure would be harder to effect. Poor mandibular, labial and tongue control also very often result in drooling. This was evidenced in two of the cerebral palsied children, Subject C and E, the same two who were unable to elevate their tongues. Also Subject C had difficulty achieving labial closure. However, the mandible is often reported in the literature to be used to compensate for inadequate tongue movement in a superior-infereior plane. That is, the jaw is raised and lowered to effect various tongue heights for articulation.

**Phonological Patterns**

Vowels. In the review of the literature vowels, apart from being nasalized, were not found to be perceived as otherwise deviant in cleft palate speech but were in cerebral palsy. The present investigation supported this assertion in that neither cleft palate speaker
evidenced vowel distortions, and two of the three cerebral palsied children did. The presence of distorted vowel production appears to show greater involvement in the oral musculature and especially the tongue.

Consonants. (1) Manner of production. The most common articulatory errors of manner of production in cleft palate and cerebral palsy are presented in Table 10 and agree completely with the cleft palate findings. (See Table 7, page 42.) For cerebral palsy, fricative and affricates were also found the most defective but only the approximant /r/ was difficult, all three subjects producing /w/ and /j/ correctly. Also, the plosives, although perhaps not as aspirated as normal, appeared to be relatively easy sounds to produce for these cerebral palsied speakers.

Table 10

<table>
<thead>
<tr>
<th>Manner of Production</th>
<th>Cleft Palate</th>
<th>Cerebral Palsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most Defective Sounds</td>
<td>fricatives—most sensitive affricates plosives high pressure sounds</td>
<td>fricatives affricates glides fine coordination sounds</td>
</tr>
<tr>
<td>Least Affected Sounds</td>
<td>nasals glides</td>
<td>nasals</td>
</tr>
</tbody>
</table>

While manner of production errors appear to be similar in both groups, as stated before, etiology is mainly due to the incompetent velar sphincter with resultant inadequate intra-oral pressure build-up
in cleft palate while in cerebral palsy other factors too, such as difficulty with finely coordinated sounds due to tongue involvement are causitive.

(2) Place of articulation. For cleft palate, sounds with lingual contacts are reported to be more difficult than labials. A tendency to replace more anterior valves with "back of the tongue" ones is also stated in the literature. In the limited data collected from the two cleft palate children here, deviant productions did not seem to be related to place; except that in places of articulation anterior to the velum, intra-oral breath pressure could not be adequately attained. Most of the non-english substitutions were more posterior than anterior, due to the inadequate velum. But any antero-posterior difficulties appeared to be related to inadequate oral air pressure and not to lingual placement inability. One example of a more posterior placement was Subject B's substitution of /q/, an uvular plosive for /k/, a velar one. This could be due to posterior carriage of the tongue. In short, placement errors may arise from antero-posterior difficulties due to velar insufficiency, but are likely to be unrelated to any tongue elevation, protrusion or agility difficulties.

Tongue tip complex sounds /s, z, t, d, 3/ are usually the hardest to produce for a cerebral palsied individual. Other than that generalizations about placement errors are difficult to make because of the many factors involved. Tongue tip simple, lip and back of the tongue sounds are thought by some to be the easiest but are reported by others to be difficult. Dentals and velar sounds are also reported to be difficult for a cerebral palsied person to make. It seems most
reasonable to conclude that in cerebral palsy there may be difficulties in the antero-posterior dimension of the tongue, for example, protrusion /θ,ð/; or on the vertical dimension, for example, elevation /t,s,tʃ/, due to both velar incompetence, and reduced agility and speed of the tongue. The articulatory problems in the three cerebral palsied subjects were diverse, and supported the above conclusions:

Subject C: no interdental sounds /θ,ð/: weakness, inability to protrude tongue;
Subject D: /l/ was sometimes correct and sometimes interdental: inconsistent, involuntary movement; and
Subject E: /k/ in final position produced as: /kʰ,ʌk,t,ʃ,ʃk,ʃʃ/: lack of direction control, incoordination.

(3) Voicing. Voiceless sounds are reported more often defective in both groups than the voiced cognates. This was supported by the present inquiry. Greater intra-oral air pressure is needed for voiceless sounds and seems to be the main etiological factor for defective production of voiceless sounds. In cerebral palsy, an added laryngeal involvement could also contribute to the etiology.

Types of misarticulations. Distortion of consonants is considered the most outstanding characteristic of cleft palate speech. Nasal omission causes distortion, and the fricatives are the most susceptible to becoming distorted because of their relatively long duration. Distortions were present in the two cleft palate speakers in the form of nasal emission and lateral emission. Some lateral emission was evidenced in one cerebral palsied subject but overall fewer distorted consonants were in this group.

Omissions are reported in the literature to exceed distortions and substitutions in cerebral palsied speech, especially in the final
position. Cleft palate speakers have an average of 21 sounds in initial position of words and 18 in final positions. Cerebral palsied speakers have an average of 18 initially and 10 finally. This shows that cleft palate speakers have fewer omissions, and that cerebral palsied speakers have a greater number of omissions in the final position than in the initial position. This small sample therefore is in agreement with findings cited in the literature. Articulation proficiency for both conditions depends on the phonetic context. This has important implications for treatment, and can give indications as to the status of the articulator under question.

Since cleft palate is fraught with more distortions, and cerebral palsy exhibits a greater number of omissions, it would follow that cleft palate would have more non-english sounds than cerebral palsy. This seems to be the case for the subjects in this study when distribution of an occurrence is considered and not just the number of types of sounds. For although nasal emission is only one type of a non-english sound, it can affect a variety of sounds, for example plosives and fricatives.

This study showed that omissions may not be as prevalent in cleft palate speech as reported. If any attempt is made to produce the speech sound, it is not omitted. For example, Subject B used nasal emission and prolongation to signal the final consonant when there were two final consonants, [wɪlɪ] for ['wilz] and [krəs:] for [krəft].

Generally in the cleft palate population, substituted sounds tend to have the same manner of production. Usually in cerebral palsied speakers, sounds are produced in the easiest way possible and this
continues even though it might not be the best a person can do.

**Consistency.** Articulatory errors arising from a structural disorder were hypothesized to show greater consistency in occurrence than errors due to a neurological dysfunction. However, inconsistency can stem from various causes. Phonetic context and rate of speech production can influence nasality and consequently articulation because a significant relationship between degree of nasality and articulation ability exists. Other factors are fatigue and emotional stress. The phonological system of the cleft palate subject B appeared to be in a state of variation since so many different productions of a single phoneme were demonstrated. In cerebral palsy, inconsistency can result from fluctuating muscle tone, spasms, involuntary movement and lack of direction control. Subject E (ataxic) demonstrated this lack of directional control on many consonant productions. He could usually recognize his misarticulations and would attempt to self-correct: /məsʃ/ for /mæsk/ then /mətʃ/ for /mæsk/. While the cleft palate condition produces more consistency in hypernasality, it appears that due to other factors, articulation errors are often inconsistent as is the case with cerebral palsy.

**Intelligibility.** Considerable variation exists in reporting percentages of those cleft palate speakers and cerebral palsied individuals who achieve normal, intelligible speech. From reviewing the phonological systems of both groups, it would appear that although cleft palate speech may sound more deviant if there are non-english sounds, generally this population is more amenable to therapy. Since
much of the involvement is secondary to the velopharyngeal incompetence, aberrant behavior can be modified. Conversely, most of the associated involvement in cerebral palsy is part of the condition. Apparently, more cleft palate speakers achieve "normal" speech than do cerebral palsied individuals. The cleft palate speakers in this study were more intelligible than the cerebral palsied speakers. However, there were too few in either group to make generalizations.

Lack of intelligibility in cleft palate is mostly related to distortion of consonants and hypernasality. In cerebral palsy, it is related to indistinct articulation with consonants omitted, substituted and distorted, distortion of vowels, hypernasality, reduced intensity and possible prosodic abnormalities.

Speakers have a remarkable ability to compensate for physiological constraints imposed on particular articulators by adjusting articulatory maneuvers in order to achieve a desired acoustical end product. It is the interplay among various articulators which allows the speaker to approximate the desired sound even under difficult situations (Minifie et al., 1973). In cerebral palsy, there may be little with which to compensate.

Assessment and Treatment

Articulatory disorders among the cleft palate and cerebral palsied populations may stem from numerous sources. Careful evaluation is therefore necessary. In cleft palate it must be ascertained if the problem is structural or functional. If structural, the necessary surgical or prosthetic arrangements must be made with the appropriate specialists. If functional, further delineation must be made in
reference to etiology before speech therapy is commenced. Essentially
the task entails determining the errors and when they occur. Learning
when they occur is as important as hearing what they are since the
context in which the errors occur frequently gives clues about treat­
ment (Morris, 1971). Whether the speech problems are residual or
compensatory, whether they are a result of abnormal tongue placement
or faulty direction of the airstream will determine the direction
therapy should take. The possibility of neurological contribution,
hearing deficit or emotional factors must also be considered.

In cerebral palsy, the process of diagnosis and evaluation
multiplies in complexity. Mysak (1971) stated that discussion of
articulation disorders among the cerebral palseid is replete with
implications with respect to the differential diagnosis of possible
articulatory contributions in any one individual. The possible contribu­
tion of oroneuromotor maturation, adequacy of breath pressure, mental
retardation, hearing loss, and developmental articulatory disorder
should be considered. Furthermore, the particular phonetic lapse under
consideration must be determined whether to be due to consistent
limitations to direction and range of movements of the articulators; to
inconsistency in moving in appropriate directions with appropriate
range; to problems of intra-articulatory system coordination for example,
lack of coordination between labial and velopharyngeal closure activi­
ties for production of /b/; to difficulty in voluntarily reproducing
certain movements which can be effected spontaneously; to difficulty
moving various articulators separate from one another; to problems in
adequate and accurate sensory feedback from the articulatory system,
and to combinations of the above (Mysak, 1971). The possibility of a structural inadequacy should not be precluded. Mysak reported that after identifying the possible movement deficits, the diagnostician should determine whether the problems stem from retained primitive reflexes, or lack of differentiation among the articulators, or to involvement of the neuromotor systems, and so on. The complexities are many and the many decisions that are made will affect therapy effectiveness. There are many priorities that come before speech. Suitable posture, head control, breath support, phonation, valving movements all must be present before speech can effectively be attempted. Lencione (1968) has stressed that the total development of the cerebral palsied child must be a priority. She felt that the multiplicity of disorders in cerebral palsy makes it important that the speech pathologist have a basic understanding of the concept of growth and development in the interpretation of causes and consequences.

A careful examination of the phonological system can give invaluable information as to etiology of the disorder and therefore therapy direction in both cleft palate and cerebral palsied speakers. Knowledge of the differences and similarities of the two can also aid diagnosis. For example, if a cerebral palsied individual demonstrates hypernasality with pharyngeal and glottal substitutions, the possibility of reduced breath support can be ruled out since the air pressure is available at the location of the pharynx and larynx. A good understanding of both phonological systems can increase one's diagnostic skill in either area.
Chapter 4

CONCLUSION

The structural and neurological disorders that have been discussed both reflect complex entities. Some of the relations between them may never be fully understood. The discussion of this paper has revolved around differences and similarities between articulatory and phonological systems in the two conditions cleft palate and cerebral palsy.

Similarities arise because velopharyngeal inadequacy is present in both conditions. Both conditions evidence hypernasality, and both have the greatest difficulty with high pressure consonants. Many of the contrasts that have been presented stem from differences of structural and neurological etiological factors. The structural alteration in cleft palate produces a static phenomenon, one that will evidence certain consistencies in performance. The hallmark of a neurological dysfunction, on the other hand, will be inconsistency since the structure is adequate and able to make closure but does so only when the neurological events are times, coordinated and balanced appropriately.

A second basic difference arises from the scope of the disorders. Cleft palate is a focal disturbance, one in which other systems are more commonly normal. Therefore compensatory behaviors will reflect this. For example, pharyngeal and laryngeal substitutions reflect normal air pressure since respiration is not disordered in cleft palate. Cerebral palsy reflects a generalized dysfunction affecting movement and coordination of many or all of the processes of speech production.
Therefore available compensations become reduced. Pharyngeal and laryngeal compensations are not utilized because breath pressure is reduced at those levels as well. More likely the sound is omitted or a simpler sound, not at all related may be substituted.

The abnormality present in the speech of a cleft palate individual is usually due to the velar insufficiency, and the many compensatory mechanisms developed to cope with this structural deficiency. Speed and flexibility of the other articulators per se are usually not significantly reduced. However, in speech, inefficient directing of the airstream may result in an overall decreased speed of speech movements; and inappropriate tongue carriage may result in reduced tongue tip agility.

All the processes of speech which are differentially aberrant in cerebral palsied individuals contribute to the total picture of speech abnormality. These aberrations are part of the motor disorder and basically, few compensatory strategies are available to the cerebral palsied speaker because most systems are involved. Weakness and slowness of movement in speech and nonspeech activities are common to all types of cerebral palsy, and flexibility, motility and stability of the articulators are reduced to varying degrees in different types of cerebral palsy.

These differences and similarities in phonological and articulatory patterns in cleft palate and cerebral palsied speech did emerge from the limited data of the present study. This would seem to indicate that the procedure followed provides a viable model, and as such, suggests that investigation utilizing a larger sample would yield
significant findings of a more quantifiable nature in delineating structural and neurological contributions to the speech production in cleft palate and cerebral palsy.
REFERENCES


