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Compensatory articulation in aphasia

Jean H. Kim

School of Communication Sciences and Disorders
McGill University, Montreal
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ABSTRACT

Compensatory articulation was investigated in normal and brain-damaged individuals by comparing vowel production under normal and perturbed speaking conditions. The effects of fixed mandibular positioning on first and second formant frequencies of the vowels [u i æ a] were investigated in ten normal subjects, six nonfluent aphasics and six fluent aphasics. Adaptation to perturbation was examined under compensatory and noncompensatory conditions, in which the degree of mandibular opening posed maximal and minimal interference respectively, with reference to normal articulatory positioning. F1 and F2 values, determined by linear predictive coding, were measured at the onset and midpoint of glottal pulsing to identify changes in compensation over time. Results of statistical analyses indicated variable effects of perturbation in both the normal and aphasic subject groups. Analyses of the data with respect to perceptual difference limens suggested that formant deviations in compensatory and noncompensatory productions of all subjects groups would have resulted in changes in vowel quality. The results were interpreted as indicating that compensatory performance, although evident, was neither complete nor immediate in any of the subject groups tested. Moreover, articulatory reorganization and compensation for

fixed mandibular positioning appeared to be preserved in nonfluent and fluent aphasics. The findings are discussed with respect to models of speech motor programming and neurogenic models of speech production.

RÉSUMÉ

L'articulation compensatoire a été étudiée chez des individus cérébrolésés et chez des individus non cérébrolésés en comparant la production de voyelles dans des conditions normale et perturbées. Les effets du positionnement mandibulaire fixe sur les premier et deuxième formants des voyelles [u i æ a] ont été étudiés auprès de dix sujets normaux, de six aphasiques non fluents et de six aphasiques fluents. L'adaptation à la perturbation a été étudiée sous des conditions compensatoire et non compensatoire dans lesquelles le degré d'ouverture mandibulaire créait, respectivement, une interférence maximale et minimale, en référence à la position articulatoire normale. Les valeurs de F1 et de F2, déterminées par encodage linéaire prédictif (linear predictive coding), ont été mesurées au début et au milieu de la vibration glottale afin d'identifier les changements de compensation dans le temps. Les résultats des analyses statistiques indiquent des effets variables de la perturbation chez les sujets aphasiques et chez les non cérébrolésés. L'analyse des données, relativement au changement minimum perceptible, suggère que les déviations de formants dans les productions compensatoire et non compensatoire de tous les groupes de sujets auraient entraîné des changements dans la qualité des voyelles. Les résultats ont été interprétés comme indiquant que la performance

compensatoire, bien qu'évidente, n'est ni complète ni immédiate dans aucun des groupes de sujets. De plus, la réorganisation et la compensation articulatoires pour le positionnement mandibulaire fixe semblent préservées chez les aphasiques fluents, ainsi que chez les non fluents. Ces résultats sont discutés relativement aux modèles de programmation motrice de la parole et aux modèles neurologiques de production de la parole.

CHAPTER 1

Theories of speech motor control

Research in the area of speech motor control, guided by diverse theoretical viewpoints, has proven to be an exciting yet challenging field of active discussion and debate. Theories and models of speech motor production have evolved through gradual modifications of earlier theories as well as by radical shifts in the approach to investigating speech production. More specifically, in exploring the processes and mechanisms involved in translating linguistic intent to articulatory movements, the perspectives have changed dramatically from a view of movement as a direct output of linguistic units to a purely mechanical system without any assumptions of, or attachment to linguistically-based inputs (Smith, 1992). Three major theoretical perspectives will be reviewed in order to illustrate the progression of speech motor control research and to introduce some fundamental issues which have engaged the attention of researchers for several decades. This review includes a summary of a phoneme-based theory of invariant motor commands (Liberman, Cooper, Shankweiler & Studdert-Kennedy, 1967; MacNeilage, 1970), goal-oriented theories (Ladefoged, DeClerk, Lindau & Papcun, 1972; MacNeilage, 1970, 1980), and action theory (Folkins & Linville, 1983; Kelso & Tuller, 1983).

The phoneme-based theory of invariant motor commands describes speech production as the sequential selection and production of individual phonemes, each associated with a unique and invariant set of muscle commands and vocal tract configurations (Liberman et al., 1967). The formulation of this theory was motivated by the widespread assumption that speech could be segmented into phonemic units (MacNeilage, 1970). According to MacNeilage (1970), studies demonstrating categorical perception of phonemes and the occurrence of spoonerisms which involve the transposition of phoneme segments (e.g. "tasted the whole worm" instead of "wasted the whole term"), were among some of the findings used to attribute a psychological and behavioral reality to the concept of phonemes. Given the apparent psychological reality of phonemes, associating them with a unique set of motor commands for their production appears to have been logical. However, the invariant articulatory configurations posited in this perspective were challenged by the overwhelming inter- and intra-subject variability that was evident in repetitions of a given utterance, as determined by acoustic, EMG, and articulatory movement analyses (e.g. Abbs, 1986; MacNeilage, 1970; Smith, 1992). In addition, factors such as the rate of speech, stress, and contextual environment were observed to contribute to the variable nature of speech production (Smith, 1992).

The characterization of speech production as a dynamic

and exceptionally variable process generated different responses among researchers. For some, the lack of observable and measurable invariance triggered a spirited search for invariant physiological correlates of phonemes (Abbs, 1986; MacNeilage, 1970; Perkell, 1980). Others, who questioned the validity of phonemes as the underlying units of planning in speech production, explored the role of alternate linguistic structures, such as the syllable, in speech motor control (Kent, 1976). MacNeilage (1970) approached the "problem" of variability in yet a different manner.

In 1970, MacNeilage introduced a goal-oriented theory based on spatial targets which not only preserved the phoneme as the unit of production but also incorporated the evidence which suggested that a direct correspondence between linguistic units and motor commands did not exist. He approached the problem of variance by highlighting the remarkable capacity of the speech mechanism to produce acceptable speech despite the observed variability. In an effort to explain this ability, he proposed that the speech motor system operated on the basis of achieving invariant goals, referring to the phenomenon of "motor equivalence" whereby an end-result is achieved by variable movement patterns (Hebb, 1949). More specifically, phonemes were viewed as being encoded in terms of spatial goals (i.e. articulatory positioning within the oral space), which could be reached from variable starting positions and by variable

movement patterns. MacNeilage's theory will be evaluated on two points: 1) the operation of motor equivalence in speech production and 2) the nature of the goals (i.e. spatial) in achieving motor equivalence.

Motor equivalence, which illustrates the goal-oriented nature of motor programming, appears to be characteristic of motor systems in general (Abbs, 1986; Hughes & Abbs, 1976; Perkell, 1980). MacNeilage's proposal that speech motor control also operates on the basis of motor equivalence has been supported by considerable research. In speech production, at least two processes have been examined as evidence of motor equivalence: reciprocal compensation (Hughes & Abbs, 1976) and compensatory articulation (Fowler & Turvey, 1980; Lindblom, Lubker & Gay, 1979; Perkell, 1980).

Reciprocal compensation refers to the interactions observed between articulators in natural speech production. For example, Hughes and Abbs (1976) described a labial-mandibular relationship in vowel production. Inferior-superior displacement measures were obtained for the upper lip, lower lip and jaw in normal and fast speaking rate conditions, the latter being more demanding for articulatory reorganization. Analysis of the displacement data indicated a coordination of articulatory movements between the jaw and lower lip, which was maintained for both speaking rates. For example, in order to achieve the relatively large vertical opening required for [a], small jaw displacements were

consistently paired with significantly more lower lip movement. In contrast, a larger jaw displacement was associated with reduced lower lip movement. Hughes & Abbs (1976) described their observation as reflecting a "co-cancelling" effect of individual articulator variability.

The second process is compensatory articulation, which refers to the exceptional ability to reorganize the activity of the articulators in the presence of a perturbation in a manner which retains the acoustic and perceptual properties of normal productions (Fowler & Turvey, 1980; Lindblom, Lubker & Gay, 1979; Lindblom & Sundberg, 1971; Lubker, 1979). One common method of investigating compensatory articulation has been to examine speech production with a bite-block placed between the speaker's teeth in order to fix the position of the jaw.

For example, Lindblom et al. (1979) investigated vowel production under normal and bite-block conditions in Swedish subjects. The first and second formant frequencies of the perturbed productions were compared to the formant patterns obtained in the normal productions and to the patterns predicted by Lindblom and Sundberg's (1971) articulatory model which described the acoustic consequences of variations in jaw positioning. In this way, Lindblom and his colleagues were able to assess the degree of compensation that occurred on a scale ranging from "perfect" to "zero compensation". Compensation was considered to be perfect if the formant

patterns observed in the perturbed condition were identical to those obtained in the normal conditions. In contrast, zero compensation would have been reflected in large discrepancies between the formant patterns, with formants in the bite block condition conforming to the model of Lindblom and Sundberg (1971).

An analysis of their data indicated that most subjects showed near perfect compensation, as the formant patterns were within the range of normal productions. Moreover, compensation occurred immediately, coincident with the onset of phonation, leading them to speculate on the minimal use of feedback systems in speech motor control (Lindblom et al., 1979). (A subsequent section will consider the issue of feedback in greater detail.)

The compensatory phenomenon has also been reported in electromyographic (Abbs & Gracco, 1984; Folkins & Abbs, 1976; Lamarre & Lund, 1975) and displacement studies (Folkins & Abbs, 1975; Folkins & Linville, 1983; Tye, Zimmerman & Kelso, 1983). Its robustness is further exemplified by a study cited by Abbs (1986) which demonstrated that subjects showed compensatory reorganization in the presence of a perturbation despite instructions not to do so.

However, there has been increasing evidence to indicate that articulatory compensation is neither complete nor equivalent across the various categories of speech sounds (Flege, Fletcher & Homiedan, 1988; Fowler & Turvey, 1980;

McFarland & Baum, in press). For example, in a study designed to investigate the immediacy of compensatory articulation, Fowler & Turvey (1980) observed a degradation in intelligibility ratings for vowels produced under conditions of perturbation. Although acoustic differences were not statistically significant for either the first or second formants, closer inspection of the patterns revealed an overall increase in F1 for the perturbed productions, a trend consistent with models demonstrating formant shifts in the absence of compensatory reorganization. Perceptual measures indicated that the increases observed in F1 were sufficient to affect vowel quality. Naive listeners, presented with a sample of normal and perturbed productions, demonstrated significantly more errors in identifying bite block vowels than those produced normally. Therefore, Fowler and Turvey (1980) concluded that although their subjects went "a long way towards compensation", some limitations were evident as they did not demonstrate perfect compensation for the perturbation (p.316).

In a more recent study, McFarland and Baum (in press) investigated the effects of increased jaw opening on vowel and consonant production in French speaking adults. The sounds selected for investigation were three vowels [u i a], three stop consonants [p t k] and the fricatives [s š]. Subjects were required to produce the sounds under normal and bite-block conditions, for which temporal and spectral measures

were obtained. Temporal analyses revealed only a minimal effect of increased jaw opening on vowel and consonant duration. Significant differences were found only between normal and perturbed productions of the fricatives. In contrast, spectral measures indicated an effect of the bite block in all sound classes investigated. Differences in vowel formants were reported to be both statistically significant and perceptually salient, exceeding formant frequency difference limens (Flanagan, 1955). Similarly, centroid frequencies of the stops and fricatives in the bite block conditions were significantly different from those calculated for normal productions.

In order to investigate possible learning effects in compensatory articulation, vowel and consonant productions were examined following a 15-minute conversation with a 10mm bite block in place. Post-conversation measures of duration again showed a minimal effect of articulatory perturbation. However, in stark contrast to the earlier task, subjects showed more complete compensation for the bite block in vowel productions. A similar adjustment was not observed for the consonants, leading the authors to conclude that the development of compensatory strategies varies across the phoneme classes investigated (McFarland & Baum, in press). They speculated that the discrepancy in adaptation time between vowels and consonants was due to the greater articulatory precision required for consonant production.

The findings of these investigations (Fowler & Turvey, 1980; McFarland & Baum, in press) suggest that compensation for articulatory perturbations may not be complete, immediate or similar across all phoneme categories. Several explanations have been posited for partial compensation including individual learning differences (Lindblom et al., 1979) and mechano-anatomical limitations (Fowler & Turvey, 1980; Hughes & Abbs, 1976; Lindblom et al., 1979). In addition, Fowler and Turvey (1980) suggested that deviations from full compensation in bite block studies could reflect a distortion in the airflow caused by the bite block itself.

Despite the evidence for incomplete compensation to articulatory perturbation, the operation of some type of motor equivalence in speech production is unequivocal (Lindblom et al., 1979, Lindblom & Sundberg, 1971). In addition, as mentioned by Fowler & Turvey (1980), reports of incomplete compensation must not be interpreted as zero compensation. McFarland and Baum (in press) also made the distinction between incomplete and zero compensation, stating that although significant differences were found in the spectral measures of normal and perturbed conditions, a comparison of the perturbed formant patterns with those predicted by Lindblom and Sundberg's (1971) model revealed that the subjects had made some articulatory adjustments.

A number of recent studies have focused on how compensation, whether partial or complete, is achieved (Gay,

Lindblom & Lubker, 1981). For example, Gay et al. (1981) conducted an X-ray study designed to compare the articulatory gestures associated with the production of bite-block and normal vowels. Comparisons were based on vocal tract shape and area along the length of the vocal tract. Differences in vocal tract shape, determined by superimposing the outlines obtained for each condition, were minimal for all vowels tested. In particular, Gay and his colleagues (1981) highlighted the fact that subjects maintained the point of maximum constriction in order to yield accurate vowel production.

Deviations in area functions between bite block and normal productions were usually less than 5mm, with the minimum deviation consistently occurring at the point of maximum constriction (Gay et al., 1981). The researchers concluded from their findings that compensatory gestures were selective in nature, such that the resulting area function would give rise to acoustically equivalent productions. They proposed that vowel targets are "coded neurophysiologically with respect to the acoustically most significant area function features, the points of constriction along the length of the tract" (p.809). The goal is therefore not simply a spatial target, but an acoustic one.

In fact, a decade after his original proposal of spatial targets, MacNeilage himself considered acoustic goals to be more likely than spatial goals in speech motor control as

auditory information was the "main source of goals" in the acquisition of speech (MacNeilage, 1980, p.617). In proposing acoustic targets, MacNeilage also outlined several weaknesses inherent in viewing speech production as the achievement of spatial goals. For example, it was noted that the goal of some speech sounds, such as diphthongs, involved continuous movement rather than a fixed articulatory position in the oral space. In addition, MacNeilage (1980) made reference to a study conducted by Folkins and Abbs (1975) which demonstrated that spatial coordinates in the production of sounds which were assumed to involve fixed positions (e.g. bilabial closure for [p]) were found to vary; (see also Folkins & Linville, 1983; Gracco & Abbs, 1986).

In Gracco and Abbs' (1986) investigation of normal bilabial closure, a kinematic analysis of individual articulator movements (i.e. upper lip, lower lip and jaw) and the overall combined movement for multiple repetitions of the utterance "sapapple" was conducted. Results indicated greater variability in measures of peak displacement and velocity for individual articulator movement than the overall combined movement. The authors argued that articulatory movement could not be specified according to spatial targets as the spatial coordinates of labial contact, as determined by variable articulatory movement, were unique for each trial. However, the relative invariance of the combined movement patterns was interpreted as supporting the view of speech

motor planning operating with reference to a target. They hypothesized the target to be acoustic in nature as subjects' utterances were perceptually adequate despite kinematic variability of individual articulators. Moreover, Gracco and Abbs (1986) concluded that speech was encoded with respect to acoustic targets for the simple reason that speech production is listener-oriented; the ultimate goal of speech production is to be perceived by the listener.

Despite proposals for acoustic targets (Gracco & Abbs, 1986; MacNeilage, 1980; Perkell, 1980), some researchers have questioned their validity as well (Tye et al., 1983). Tye and her colleagues (1983) challenged the necessity of auditory targets in achieving motor equivalence by comparing compensatory skills in vowel production in hearing and hearing-impaired speakers. The hearing-impaired group was comprised of one congenitally deaf subject and two adventitiously deaf subjects. Cinefluorographic and perceptual analyses provided converging evidence to suggest that although the hearing-impaired subjects had more variable vocal tract configurations, they showed compensatory skill in the presence of a bite-block. As with the hearing subjects, hearing-impaired speakers produced vocal tract shapes which generally maintained the areas of maximum constriction. Based on these findings, Tye et al. (1983) concluded that auditory targets were not necessary for achieving compensatory articulation, particularly because such a representation had

never been available to the congenitally deaf speaker. Although they did not deny the possibility that normally hearing individuals use auditory targets to guide their production, they were not regarded as being a necessary feature.

Another major issue raised by much of this literature is the role of feedback in the control of speech production. Two levels of control have been posited in speech production: central and peripheral (Abbs & Eilenberg, 1976; Folkins & Zimmerman, 1981; Lindblom et al., 1979). Central mechanisms, which operate at the cortical level, involve the formulation of linguistic intent, processing of abstract linguistic structures and the initiation of motor commands (Abbs & Eilenberg, 1976). Peripheral mechanisms involve lower level neural systems including brainstem and spinal processes including lower motor neurons (Abbs & Eilenberg, 1976; Folkins & Zimmerman, 1981).

Generally, if information from the peripheral mechanisms is used in the reprogramming of motor commands at the central level, an adaptive closed-loop system is assumed. Speech motor control in a closed-loop system is bidirectional as the controlling mechanism not only provides the output to the peripheral musculature but also receives input in the form of feedback, used for the revision of motor commands. Studies showing compensatory articulation have been cited as evidence for the operation of some form of a closed-loop system as

motor commands must be revised in the presence of a perturbation (Lindblom, et al., 1979).

However, studies designed to investigate the role of feedback in speech motor control have been contradictory, leading some to question the significance of postulating a closed-loop system. On one hand, for example, Perkell (1979, 1980) referred to a study conducted by Lindblom, McAllister and Lubker (1977) in which sensory feedback was reduced by applying a topical anaesthetic to the labial and oral mucosa. Analyses of subjects' vowel productions demonstrated that a reduction in sensory feedback was associated with noticeable difficulty in compensating for a bite-block. Lindblom and his colleagues therefore concluded that orosensory feedback was crucial in speech motor programming. On the other hand, however, Kelso and Tuller (1983) were unable to replicate Lindblom et al's (1977) findings, as they reported minimal effects of sensory deprivation on subjects' compensatory ability. Vowel productions were obtained under normal conditions and under various combinations of bite-block and reduced somatosensory and auditory information. Reduction in afferent somatosensory information was accomplished through bilateral anaesthetization of the temporo-mandibular joint (TMJ) and application of topical anaesthesia to the oral mucosa; auditory information was reduced by the presentation of white noise over a set of headphones. Acoustic analyses of the formant patterns revealed nonsignificant differences

between the normal and the most extreme condition of deprivation (i.e. a combination of the TMJ block, topical anaesthesia, masking noise and bite-block).

Although Kelso and Tuller (1983) cautioned that their procedure did not eliminate all peripheral information, nor could they preclude the use of feedback under normal speaking conditions, their findings clearly cast some doubt on the role of sensory information in speech motor programming. In addition, Kelso and Tuller (1983) noted that the operation of a peripheral feedback loop would require a certain amount of time, and is therefore incompatible with findings of immediate compensation, as demonstrated by acoustic equivalence at the onset of glottal pulsing (Lindblom et al., 1979).

An alternative to a closed-loop system is the open-loop system in which speech production occurs in the absence of feedback. In an open-loop system, a standard set of motor instructions is prescribed to the periphery, irrespective of the state of the vocal tract after the commands are initiated. Such a system is open in that motor programming is unidirectional; programs are not modified by information returning to the control site once the motor commands have been initiated. However, this does not preclude the use of feedback to determine the state of the vocal tract prior to production. A weakness of such a system lies in its inability to readily explain compensatory articulation in response to perturbations that are introduced during speech production,

presumably after the motor commands have been initiated.

In addition to the basic open and closed-loop systems, which are problematic in terms of their ability to explain compensatory reorganization observed for perturbations during speech production or the immediacy of such reorganization, alternative models of speech motor control have been posited. These include the central simulation model (Lindblom et al., 1979) and the dynamic model, based on action theory (Fowler, Rubin, Remez & Turvey, 1980; Kelso & Tuller, 1983).

In order to account for the immediacy of compensation exhibited by their subjects, Lindblom et al. (1979) proposed a central mechanism which was predictive in nature. The investigators hypothesized that articulatory patterns of movement were simulated by a central mechanism until an adequate (i.e. compensatory) pattern was defined and finally generated. The resulting movement would therefore have compensated for any discrepancy between the target and the presenting configuration of the vocal tract prior to production.

Folkins and Zimmerman (1980) investigated the predictive nature of the central mechanisms as described by Lindblom et al. (1979) by examining EMG activity in the muscles of jaw closure under normal speaking conditions and bite block conditions. They reasoned that if articulatory adjustments were the end result of a central simulation process, the detected inability to manipulate jaw position in the bite

block conditions should lead to a deactivation of the jaw muscles. EMG signals were obtained from the anterior temporalis, masseter, medial pterygoid and lateral pterygoid for three speaking conditions: normal, 5mm bite block and 15mm bite block. Conditions were compared with respect to incidence, timing and magnitude of bursts in EMG activity. No consistent differences across conditions were reported for any of the measures investigated. As muscle activity was not eliminated in the bite block conditions, for which subjects produced adequate speech, Folkins and Zimmerman (1981) questioned the view that compensatory articulation resulted from a central simulation process alone. Instead, they hypothesized that lower-level neural systems may be involved in articulatory coordination, in addition to the central planning and processing mechanisms.

Kelso and Tuller (1983) further challenged the operation of Lindblom et al.'s (1979) central simulation model, referring to its inability to explain compensatory reorganization for unexpected perturbations in which a load is introduced after the motor commands for a particular utterance have been initiated (Folkins and Abbs, 1975). The observation that compensation occurs in conditions for which subjects do not have access to preplanning (i.e. predictive) strategies suggested that a predictive mechanism alone could not adequately capture the compensatory nature of speech production. Kelso and Tuller (1983) instead proposed an

action theory perspective to account for the phenomenon of motor equivalence in speech production.

Advocates of action theory propose a mass-spring analogy in understanding the dynamics of the articulatory system such that motor equivalence is simply a property of the system's dynamics (Fowler et al., 1980; Kelso & Tuller, 1983; Tye et al., 1983). The focus of action theory is on the functional organization and grouping of the speech system, also referred to as coordinative structures, rather than viewing individual muscles as independent components in the speech musculature (Folkins & Linville, 1983; Fowler & Turvey, 1980). According to Gay and Turvey (1979), vowel production involves the organization of the articulators "into a single, autonomous system according to a particular equation (or set of equations) of constraint; and to produce a given vowel is (perhaps) to parametrize that system in a particular way" (p.346). When a particular component of the system, such as the jaw, is fixed by a bite block, the other components in the coordinative structure will automatically counteract for the loss of movement in a manner which preserves the equation without feedback. Therefore, compensatory reorganization is mechanically achieved. The advantages of viewing speech production as a largely mechanical system lie in its ability to explain the immediacy of compensation for articulatory perturbations whether they are introduced prior to or during speech production.

In contrast to the phoneme-based theory of invariant motor commands and goal-oriented theories presented earlier, action theory provides a novel perspective in studying speech production. The problem of speech motor control is approached much in the same manner as researchers study limb movement (Smith, 1992). Speech production is viewed as a mechanical system, operating with respect to equations of constraint, divorced from linguistically-based inputs. The linguistic units underlying articulatory gestures become less relevant in early versions of this theory.

Clearly, the theoretical perspectives that have been posited to explain compensatory articulation and speech motor control in general, are diverse. Nonetheless it is evident that some type of motor equivalence operates in the speech production system. A very interesting and as yet unanswered question relates to the neural mechanisms which underlie compensatory articulation skills. One method of investigating this issue is to explore compensation in individuals who have incurred brain damage. In the next chapter, current research on speech production deficits subsequent to focal brain damage will be reviewed with the aim of exploring the neural substrate for speech production.

CHAPTER 2

Speech motor control in aphasia

Investigations of speech disorders observed in aphasic populations have made significant contributions to our understanding of normal speech production (Mlcoch & Noll, 1980). The existence of different aphasia types and their associated patterns of speech errors have provided researchers with some organizational framework with which to model and study the processes involved in speech production (Mlcoch & Noll, 1980).

Darley, Aronson and Brown (1975) developed a neurophysiologic model of speech production in which three distinct stages of processing were described and localized to a particular cortical area (Mlcoch and Noll, 1980). The auditory speech processor (ASP), the earliest stage of speech production described in this model, is involved in the specification of phonemes and permissible phoneme sequences to be produced, prior to the transformation of linguistic intent into words and sentences. This level of processing is comparable to the phonology of language which defines the repertoire of phonemes and rules for their usage. Hypothesized to be anatomically located in the left mid-temporal lobe, lesions involving the ASP are considered to result in phoneme selection errors (i.e. literal paraphasias)

(Mlcoch & Noll, 1980).

According to the model, information from the ASP is relayed to the central language processor (CLP), thought to be situated at the junction between the mid-temporal, inferior parietal and anterior occipital regions (Mlcoch & Noll, 1980). The functions of the CLP include the selection of words (semantics) and the regulation of word order (syntax) for the production of spoken and written language. At the level of the CLP, the phonemes selected by the ASP are organized into meaningful units before being coded into motor commands.

The motor speech programmer (MSP), the third and final component of Darley et al.'s (1975) model is presumed to be localized in Broca's area. According to Darley et al. (1975), Broca's area and the MSP contained therein are essential in the motor realization of phonologic, semantic and syntactic units specified by the ASP and CLP. In contrast to the ASP and CLP which are hypothesized to involve high-level cognitive and linguistic processes, the MSP is considered to be a lower-level, motor-based component. Cortical damage to the MSP is suspected to result in apraxia of speech, an impairment in the "capacity to program the positioning of speech musculature for the volitional production of phonemes and the sequencing of muscle movements" (p.255). Darley et al. (1984) and others (e.g. Itoh & Sasanuma, 1984) suspect apraxia of speech may exist in the absence of a language deficit and therefore

separate from aphasia[1].

Itoh and Sasanuma (1984) made a similar distinction between the linguistic and motoric components of speech production in their model, outlining the following stages of processing: linguistic encoding, articulatory programming and execution. These stages are considered independent to the extent that speech deficits following cortical damage generally appear to reflect impairments in a particular stage of processing. More specifically, an impairment in linguistic encoding was thought to result in a language deficit or aphasia affecting the semantic, syntactic and/or phonological components of language. In contrast, Itoh and Sasanuma (1984) suggested disturbances at the level of articulatory programming resulted in apraxia of speech which, as noted earlier (Darley et al., 1975), is considered to be independent and separate from language. Finally, an impairment in the execution of motor programming was proposed to result in dysarthria.

Speech production deficits observed in individuals following brain damage has supported a modular view of speech production to some extent, wherein distinct stages of

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[1]It is important to note, however, that the terms Broca's aphasia and apraxia have been used interchangeably by some researchers and separately by others (McNeil & Kent, 1990). In the present study, the terms are grouped together under the general term, nonfluent, in reference to speech output which is labored, telegraphic and distorted (Damasio, 1991). This is in contrast to fluent output characteristics of Wernicke's aphasics, conduction aphasics and other aphasic syndromes in which articulatory movements appear smooth and fluid (Damasio, 1991).

processing appear to have been compromised. There has been considerable research to suggest that speech deficits observed in nonfluent patients reflect an impairment in the motor implementation of linguistic units, giving rise to distortions at the segmental level (Blumstein, 1990). Such errors have been explained by some as reflecting a disturbance in the phonetic stage of processing (Blumstein, 1990; Ryalls, 1987), a stage which is analogous to the motor speech programmer (MSP) or articulatory programming stage of Darley et al.'s (1975) and Itoh and Sasanuma's (1984) models, respectively. In contrast, the production deficits observed in fluent aphasics are reported to be primarily phonemic in nature such that the impairment lies in the selection of the sounds to be produced, not in their articulation (Blumstein, 1990; Itoh & Sasanuma, 1984). The observed differences between nonfluent and fluent aphasic groups have supported a dichotomous view of aphasic speech production.

By far, the greatest amount of research has been devoted to characterizing the nature of the speech production deficits exhibited by nonfluent aphasic patients. For example, one of the most common aspects of speech that has been analyzed in characterizing aphasic speech errors has been voice-onset time (VOT). VOT, the interval of time between the release of a stop consonant and the onset of voicing, is the principal acoustic feature which distinguishes the voiced and voiceless stop cognates in the English phonetic inventory (Blumstein &

Baum, 1987). VOT analyses of normal speech indicate that the voiced-voiceless distinction is achieved by producing two distinct nonoverlapping VOT ranges (Blumstein & Baum, 1987; Lisker & Abramson, 1964). The distribution of VOT in normal speakers is, therefore, bimodal.

Investigations of VOT productions in the aphasic population have revealed different patterns for nonfluent and fluent aphasics (Blumstein, Cooper, Goodglass, Statlender & Gottlieb, 1980; Itoh & Sasanuma, 1984; Shewan, Leeper & Booth, 1984, Tuller, 1984). For example, Blumstein et al. (1980) reported that fluent aphasics performed similar to normal subjects, maintaining the constraint of the two non-overlapping categories. None of their productions fell in the intermediate VOT range between the categories. The errors produced by these patients have been categorized as paraphasias or substitutions wherein a voiced target such as [d] was produced with a VOT corresponding to the voiceless cognate, [t] (Blumstein et al., 1980). These findings were taken to suggest that the impairment lay in the patients' ability to select the proper phoneme. As noted earlier, the articulatory programming of the misselected phoneme, however, was carried out correctly resulting in VOT values falling in an acceptable, albeit incorrect range (Blumstein et al., 1980).

Unlike the fluent aphasic group, nonfluent aphasics were unable to maintain distinct VOT regions for voiced and

voiceless productions. VOT values for both types of productions were found to overlap, resulting in an essentially unimodal distribution with VOT values falling outside the normal ranges. These productions were defined as articulatory implementation errors rather than planning or selection errors, as a change in the physical properties of the target had occurred which did not reflect a category substitution error (Blumstein et al., 1980). Specifically, Blumstein et al., (1980) concluded that the impairment in VOT production in nonfluent aphasic patients reflected a deficit in the precise interarticulator timing and coordination of laryngeal and supralaryngeal gestures (Blumstein, 1990; Freeman, Sands & Harris, 1978).

Deficits in interarticulator timing and coordination have also been reported in kinematic studies of velar movement in the production of nasal phonemes (Itoh, Sasanuma, & Ushijima, 1979). In this study, articulatory errors in an apraxic subject were attributed to a breakdown in the temporal integration of velar and lingual movement, a pattern not evident in conduction aphasics. In addition, greater variability was noted in the apraxic productions, leading the researchers to conclude that the observed differences reflected an underlying deficit in motor control in the apraxic patient.

However, not all aspects of speech production are compromised in nonfluent aphasics, suggesting that the

observed phonetic impairment is not pervasive. For instance, researchers have demonstrated that motor control is not compromised in producing durational differences in frication according to place of articulation, nor in producing differential vowel durations as a function of phonetic context (Baum, Blumstein, Naeser & Palumbo, 1990; Gandour & Dardarananda, 1984). Static properties of speech production such as consonant spectral patterns and vowel formants have also been reported to be preserved in nonfluent aphasic patients (Ryalls, 1986; Shinn & Blumstein, 1983). Moreover, nonfluent aphasics also produce phonological errors which may not be explained by articulatory implementation deficits. Such errors include metathesis and assimilation which implicate errors in the phoneme selection and planning stages of speech production (Blumstein, 1990).

The extent of the motor programming impairment characteristic of nonfluent aphasics remains elusive. By examining a range of articulatory phenomena, we may begin to determine those aspects of speech that are compromised and those that are robust to damage in nonfluent aphasics. Compensatory articulation, which has been studied as a means of understanding speech motor control in normal individuals, may extend our understanding of aphasic speech production as well.

Nonfluent aphasics who exhibit deficits in the programming and implementation of motor commands would be

expected to have some difficulty meeting the reprogramming and reorganization demands of compensatory articulation. Fluent aphasics, on the other hand, may demonstrate better compensation skills as compared to nonfluent aphasics as movement-level disturbances are less prominent in this population.

Few studies investigating aphasic speech under perturbed conditions have been conducted to date (Robin, Bean & Folkins, 1989; Sussman, Marquardt, Hutchinson and MacNeilage, 1986). Sussman et al. (1986) reported an investigation of compensatory articulation in a group of brain damaged individuals classified as Broca's aphasics. Recording of subjects' productions of the vowels [i] and [a] were obtained under normal and bite block conditions. Analyses of formant values (F1 and F2) with respect to the magnitude and direction of formant changes between normal and perturbed productions generated variable results. For the vowel [i], the jaw opening created by a 20mm bite block was shown to affect both formants, as indicated by a marked pattern of higher F1 and lower F2 values across subjects. These formant differences, reflecting a lack of tongue elevation and fronting necessary to minimize the acoustic effects of increased jaw opening, were interpreted as demonstrating a lack of compensatory articulation (Sussman et al., 1986). In contrast, similar analyses for bite block productions of [a] did not yield any consistent patterns in the formant values and, according to

the authors, both F1 and F2 were within the range of normal variation. Although the degree of compensation varied between the vowels investigated, Sussman and his colleagues (1986) concluded that compensatory skill had been compromised, as shown by subjects' performance in their perturbed productions of [i].

In an effort to localize the cortical or subcortical regions underlying compensatory articulation, the investigators searched for a relationship between lesion site and compensatory performance. Subjects were divided into three categories with respect to compensatory ability, as measured by the magnitude of formant differences between normal and bite block conditions. A correlation between skill and lesion site, as obtained from CT scans was then explored. Particular attention was given to Broca's area (Brodmann's areas 44 and 45) due to its hypothesized role in speech motor processing. Of four subjects considered to have good compensatory skill, an intact frontal operculum, particularly in the region of Area 44, was a common characteristic for three individuals. A CT scan of the fourth individual indicated a lesion involving Area 44 which had not affected compensatory ability. However, the importance of Area 44 in speech adaptation was further supported by the other subject groups; two of the three patients with the greatest formant deviations had infarcts which extended to Area 44 and surrounding regions. But again, the performance of one

individual was unexpected given the lesion site which did not involve the frontal operculum. Despite the two anomalous cases described, Sussman et al. (1986) put forward the suggestion that Area 44 was critical in achieving successful compensatory articulation, as it was implicated in most subjects who did not achieve "normal" compensation.

These results should be interpreted with caution as the study has been criticized for numerous methodological flaws (Katz and Baum, 1987). For example, compensatory performance in Broca's aphasics was investigated with no reference to the performance of normal and other brain-damaged populations (i.e. fluent aphasics). Sussman et al.'s (1986) conclusions appear tenuous as subjects' performance could not be compared to normal variability in compensatory skill or to patients with a wider range of lesion sites (Katz & Baum, 1987). In addition, Katz & Baum (1987) also questioned Sussman et al.'s (1986) claims, as a statistical re-analysis of the data was found to contradict their conclusions. An analysis of variance on the formant frequencies revealed no significant differences between speaking conditions, suggesting that the subjects were in fact able to compensate for the perturbation (Katz and Baum, 1987). Clearly, compensatory articulation in Broca's aphasia requires further study before any definitive conclusions can be reached.

Another investigation, conducted by Robin et al. (1989) provides additional data relevant to the issue of compensatory

skill in brain damaged patients. Although the primary goal of the experiment was to examine articulatory velocity and temporal coordination in bilabial opening and their relation to speech errors, Robin et al. (1989) reported kinematic data for five verbal apraxic subjects in both normal and bite block conditions.

In the study, a 10mm bite block was inserted during two production tasks in order to examine its effect on peak articulatory velocity of the lower lip during attempted labial closure. In the first task, subjects were asked to repeat a list of words beginning with labial consonants. A comparison of peak velocity measures for the lower lip revealed no consistent differences between normal and bite block conditions, suggesting that the bite block had not interfered with the rate of lower lip movement. Interestingly, velocity measures obtained for the apraxic group were not noted to differ from those obtained from a single control subject. Similar results were reported for a second task in which subjects were asked to repeat a sentence at three speaking rates. As in the word production task, no systematic differences were observed between conditions nor between subject groups. The kinematic data provided by this study therefore suggest that subjects with apraxia of speech, typically considered to exhibit a disruption in the motor implementation of speech sounds, show intact compensation for fixation of the mandible in terms of lower lip velocity

measures in labial closure (Robin et al., 1989). Although these findings are based on kinematic measures of lip closure and are therefore not directly comparable to Sussman et al.'s (1986) acoustic investigation, they provide further evidence tc suggest that Sussman et al.'s conclusions may be open to question.

By investigating compensatory articulation in both nonfluent and fluent aphasics, the present investigation is expected to provide an extension to earlier studies aimed at understanding the nature of aphasic deficits in speech production. As compensatory articulation involves the reprogramming and implementation of articulatory motor commands in response to a perturbation, compensatory skill will provide a means of determining speech motor competence in both aphasic groups.

Speech production models, such as those proposed by Darley et al. (1975) and Itoh and Sasanuma (1984) would predict differences in compensatory performance in brain-damaged subjects depending on the extent to which the motor speech programmer (MSP) was affected. Nonfluent aphasic patients with impaired articulatory programming (i.e. MSP damage) would be expected to demonstrate impaired compensatory ability. In contrast, subjects with lesions sparing the MSP, and therefore likely to exhibit fluent speech are expected to be more successful in programming and implementing compensatory gestures.

In the present study, speech motor programming will be investigated for both compensatory and noncompensatory speaking conditions, as defined below (Katz, Olness, Baum & Kim, 1992; Lindblom et al., 1979). It has been reported that a large bite block presents maximum perturbation to articulatory positioning for high vowels (Lindblom et al., 1979). In contrast, maximum interference is achieved with a small bite block in the production of low vowels. In the current investigation, performance will be examined under conditions requiring considerable articulatory reorganization due to maximal perturbation as well as under conditions in which an articulatory change is introduced to the vocal tract which would interfere minimally with the achievement of an acoustic goal. The latter condition, referred to herein as "noncompensatory", will be achieved by utilizing a small bite block in the production of high vowels and a large bite block in the production of low vowels. A comparison of performance between the two compensatory conditions may shed some light on the extent of the motor speech programming deficit, if any, in the aphasic patients. A breakdown in performance in the noncompensatory conditions might reflect a particularly fragile speech motor control system.

Finally, the present study may contribute to theories of speech motor control with respect to the issue of feedback. The role of feedback in speech compensation will be investigated by comparing acoustic measures obtained at the

onset of glottal pulsing and at the midpoint of phonation in vowel production. Any changes in compensation would suggest the use of a feedback system and question the immediacy of compensation (Lindblom et al., 1979). Differential use of feedback between aphasic groups for the purpose of self-monitoring has been reported in the literature (Kennedy, 1983). For example, severe Wernicke's aphasics have been observed to be unaware of their own speech errors, suggesting a minor role of feedback in their speech production. In contrast, most individuals whose speech is characterized as nonfluent (i.e. Broca's aphasia) demonstrate greater awareness of their speech production errors. These differences in the use of feedback may be reflected in the present study in comparisons of compensatory performance over time. Details of the methodology and results of the present investigation are presented in the following chapters.

CHAPTER 3

Methods

Subjects

Aphasic Subjects: Six fluent and six nonfluent aphasic patients who were at least three months post onset were selected to participate in the present study. Inclusion in the study was limited to those who had suffered a single cerebral vascular accident resulting in a localized infarct. Patient selection was also restricted to those without significant dysarthria, oral cavity anomalies or prior speech, language, or neurological difficulties. In addition, subjects were required to pass a hearing screening at the speech frequencies of 0.5, 1 and 2 kHz with a threshold of 30 decibels or less in the better ear (ANSI, 1972b). All patients were native speakers of standard North American English. Nonfluent patients ranged in age from 43 to 79 years (mean age= 57). Fluent subjects had a similar range of 47 to 81 years (mean age= 72).

Patients were diagnosed on the basis of clinical assessment reports and results of the Boston Diagnostic Aphasia Examination (Goodglass and Kaplan, 1983). The Apraxia Battery for Adults (Dabul, 1979) was also administered to the nonfluent patients in order to determine the presence and severity of apraxia. Demographic and neurologic information

for the fluent and nonfluent patients is summarized in Table 1.

Control Subjects: The control group was comprised of ten individuals ranging from 64-80 years in age (mean age=72) who reported no history of neurological damage[2]. All were native speakers of standard North American English and were required to pass the hearing screening described above.

Stimuli

Productions of the phonemes [u i æ a] were recorded onto audio cassette by an adult male English speaker for presentation as model productions which subjects were asked to approximate. Fifteen repetitions of each vowel (five for each condition: normal, noncompensatory, compensatory) were recorded in randomized order, resulting in sixty tokens. A written cue for each token was prepared to accompany the auditory model. The written forms "oo", "ee", "aa", and "ah" were presented on 3" x 5" cards.

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[2] Although the control subjects were somewhat older than the aphasic patients, it was deemed unlikely that such age differences would influence compensatory abilities. However, if any age-related changes were to exist, they would tend to obscure any differences between the normal and impaired subjects, thereby "weighting" the investigation against our expected results.

Table 1. Demographic and Neurologic Summary of Aphasic Subjects

Subject	Age	Diagnosis	Site of Lesion
DI	43	Mild-moderate nonfluent aphasia; mild-moderate apraxia	Left parietal region
MS	43	Moderate-severe nonfluent aphasia; mild apraxia	Left Sylvian region with ventricular displacement
IG	79	Mild-nonfluent aphasia	Left parietal region
AK	59	Moderate-nonfluent aphasia; moderate-severe apraxia	Left MCA territory
GE	64	Nonfluent aphasia	Left caudate nucleus extending to anterior limb of internal capsule
MU	57	Moderate nonfluent aphasia moderate apraxia	Left frontoparietal region
IB	72	Fluent anomic aphasia	Left basal ganglia extending to paraventricular region
RM	47	Mild fluent aphasia	Left parieto-occipital region
JM	81	Moderate-severe fluent aphasia	Lesion information not available
RS	80	Mild fluent aphasia	Left thalamic region
AI	77	Moderate-severe fluent aphasia	Left temporoparietal region
BA	77	Moderate fluent aphasia	Left MCA territory

Materials

Bite blocks were constructed with a base and catalyst mixture of vinyl polysiloxane impression material putty (ANSI/ADA Spec.19: Type 1, very high viscosity). Two bite blocks with the following dimensions were prepared for each subject prior to testing: 25mm x 15mm x 7.5mm (large) and 5mm x 15mm x 7.5mm (small). Due to individual variability in dentition and patterns of occlusion, it was necessary to individualize the bite blocks in order to maintain a similar degree of jaw opening across subjects. At the time of testing, the interdental distance created by each bite block was measured. The bite blocks were adjusted to ensure a vertical distance of 22.5mm between the first premolars for the large bite block and 2.5mm for the small bite block. Bite blocks were placed between the first premolars on the left side (with the exception of one nonfluent patient) by the experimenter for each perturbed production.

Procedure

Subjects were instructed to approximate the auditory models of the target phonemes which were presented in free-field with a Sony CFS-W320 cassette recorder. Their rate of presentation was controlled by an examiner, timing them with bite block placement and presentation of the written cue by a second experimenter.

In trials requiring a perturbation, timing was

coordinated such that bite block placement was immediately followed by the auditory model and written cue of the target vowel. As the immediacy of compensatory abilities was being examined, an effort was made to reduce the holding period during which subjects could adjust to the perturbation prior to their productions. To further decrease the possibility of any adaptive effects between consecutive conditions requiring the same bite block, the bite block was removed after each production.

All sixty trials were presented to each subject. For each vowel, subjects were required to produce five randomly-ordered repetitions with each of the two bite blocks and an additional five under the normal unperturbed condition, for a total of fifteen repetitions. In conjunction with decreased holding time and the removal of bite blocks between successive productions, the randomization of bite block and normal conditions contributed to reduce the possibility of the subject developing articulatory adjustments to the perturbations. The randomized order of stimuli was held constant across subjects.

Subject productions were recorded onto an audio cassette using a Sony Professional Walkman WMD6C with a Sony ECM-909 directional microphone placed approximately eight inches from the speaker's mouth.

Acoustic Analyses

Productions from each task were digitized using the BLISS speech analysis system (Mertus, 1989) at a sampling rate of 10kHz with a 4.5kHz low-pass filter and 12-bit quantization. Once digitized, the isolated vowels were then analyzed by linear predictive coding (LPC) techniques to determine the first and second formant frequencies. LPC analyses were performed at the onset of the vowel and at its midpoint in order to identify any changes in compensation over time.

A range of acceptable formant values was established for each vowel so as to avoid potential errors generated in the LPC algorithm (e.g. mislabelling of higher formants when a lower formant cannot be detected). The F1-F2 vowel classification described by Peterson and Barney (1952) was used as a guide in setting the formant ranges, which are provided in Table 2.

If the LPC analysis at the onset did not yield a formant value within the specified range, the number of poles in the algorithm was adjusted from a default setting of 14 to 17 or 20. For analyses at the vowel midpoint, both the number of poles and the window position were manipulated. If no appropriate formant values were obtained despite the adjustment in poles, the window of analysis was shifted $\pm$20 ms from the original midpoint position. Formants remaining out of range were excluded from the data analysis.

Table 2. Formant frequency ranges employed in data collection

Vowel	F1	F2
[u]	200-500 Hz	750-1500 Hz
[i]	200-500 Hz	1800-2650 Hz
[æ]	600-950 Hz	1400-2000 Hz
[a]	600-950 Hz	900-1400 Hz

CHAPTER 4

Results

Mean F1 and F2 values, calculated separately for the onset and midpoint of vowel production, are presented in Figures 1-6. Individual data from which the figures were derived are provided in Appendices 1-12. For each subject group, separate analyses of variance (ANOVA) were conducted for F1 and F2, resulting in six 2x4x3 (measurement point X vowel x condition) ANOVAs[3].

Normal Controls

As demonstrated by Figures 1a and 1b, there appear to be small differences in F1 values across conditions at both the onset and midpoint of vowel production. A 2x4x3 (measurement point x vowel x condition) within-subjects ANOVA revealed significant main effects for vowel ($F(3,27)=315.83, p<0.001$) and condition ($F(2,18)=9.08$, $p=0.002$). The analysis also yielded a significant interaction between vowel and condition

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[3]The number of subjects processed for analyses was variable due to missing data. Data from 5 or more subjects were processed in 4 out of 6 ANOVA's. In the F2 analyses for both patient groups, missing data reduced the number of subjects to 4. In order to analyze the data, it was necessary to replace the missing data with those obtained at the alternate measurement point. These adjustments were few in number, accounting for only 3 and 2 of 144 cells in the nonfluent and fluent F2 analysis, respectively. Given their low incidence, these adjustments were not expected to affect the final results. In instances where neither the onset nor midpoint placement of the LPC window had yielded an F2 value, no further adjustments were made in the data.

Figure 1a. Mean F1 for normal and bite block productions of normal subjects measured at the onset of vowel production

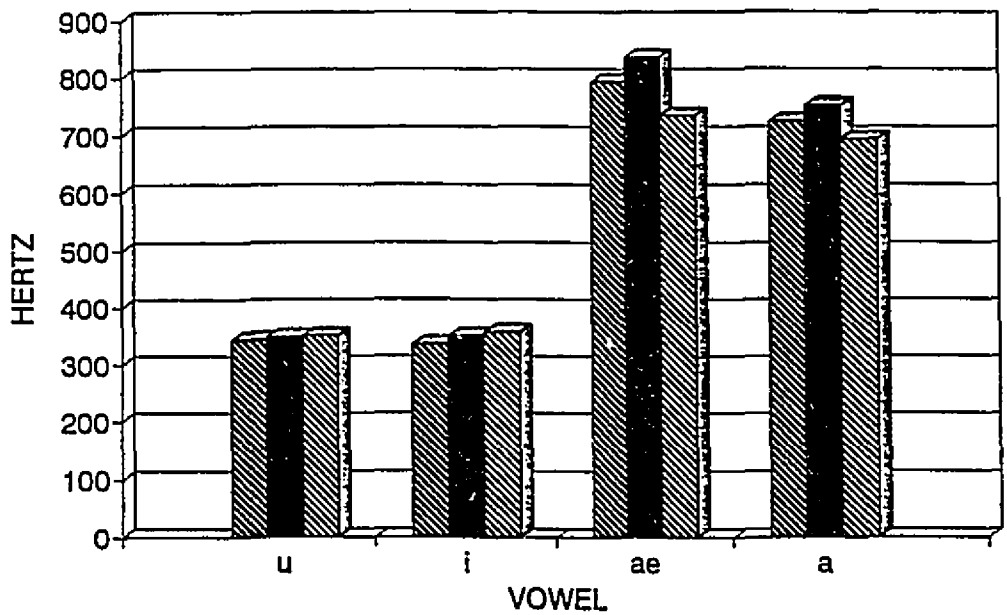
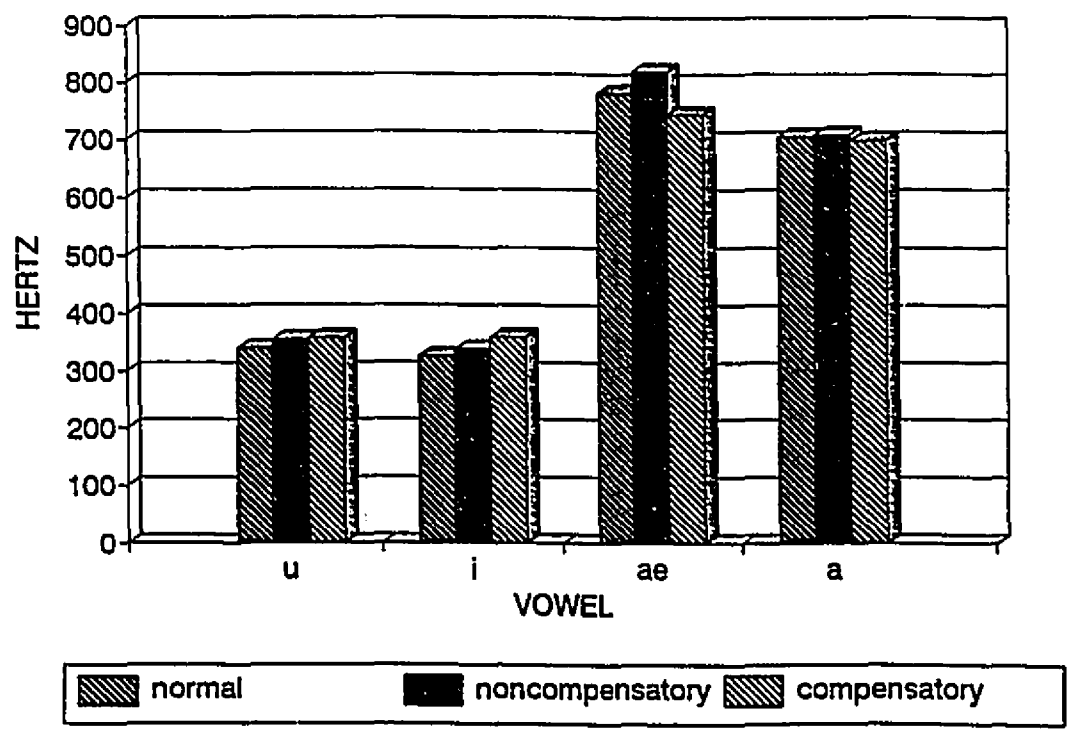


Figure 1b. Mean F1 for normal and bite block productions of normal subjects measured at the midpoint of vowel production



($F(6,54)=4.99$, $p<0.001$). A post hoc Newman-Keuls analysis ($\alpha=0.05$) of the vowel x condition interaction demonstrated that F1 was significantly lower for the compensatory productions of [æ] compared to normal and noncompensatory productions which did not differ significantly from one another. No significant differences in F1 emerged across conditions for the other vowels.

Statistical analyses of F2 revealed a main effect for vowel ($F(3,18)=283.66$, $p<0.001$) and a significant vowel x condition interaction ($F(6,36)=6.62$, $p<0.001$). As shown in Figures 2a and 2b, lower F2 values are observed for bite block as compared to normal productions of [i] and [æ] at both measurement points. Newman-Keuls analyses ($\alpha=0.05$) of the vowel x condition interaction indicated a significantly lower mean F2 in compensatory productions as compared to both normal and noncompensatory productions of the vowel [i]. The difference between normal and noncompensatory productions was not significant. Compensatory productions of [æ] also resulted in a significant lowering of F2 as compared to normal productions; F2 values obtained in the compensatory condition were not significantly different from noncompensatory productions, nor were noncompensatory productions different from normal productions.

It would appear that the various noncompensatory speaking conditions created minimal articulatory interference as formant values were not significantly different from those

Figure 2a. Mean F2 for normal and bite block productions of normal subjects measured at the onset of vowel production

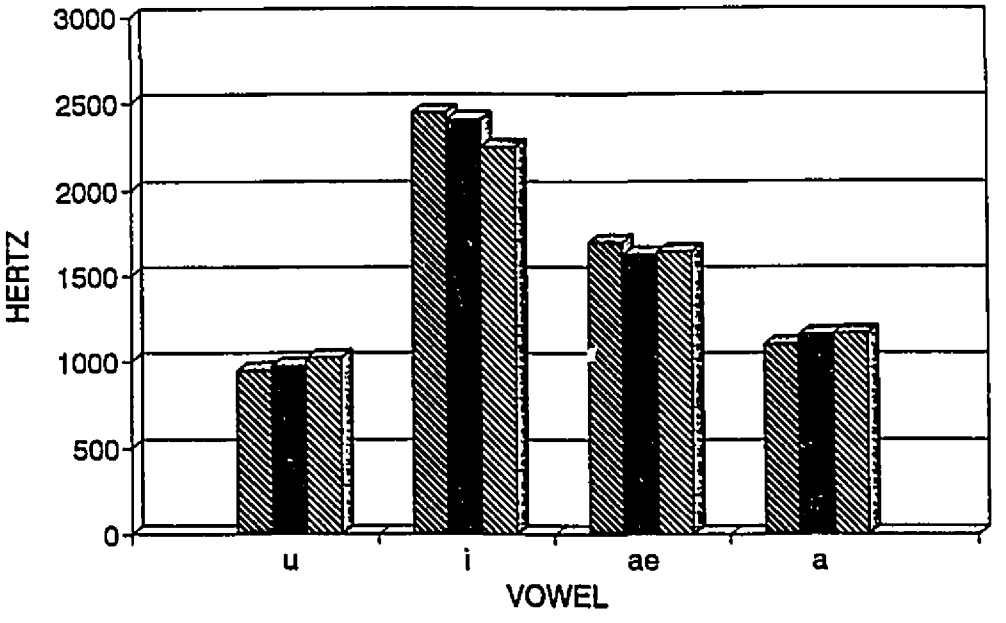
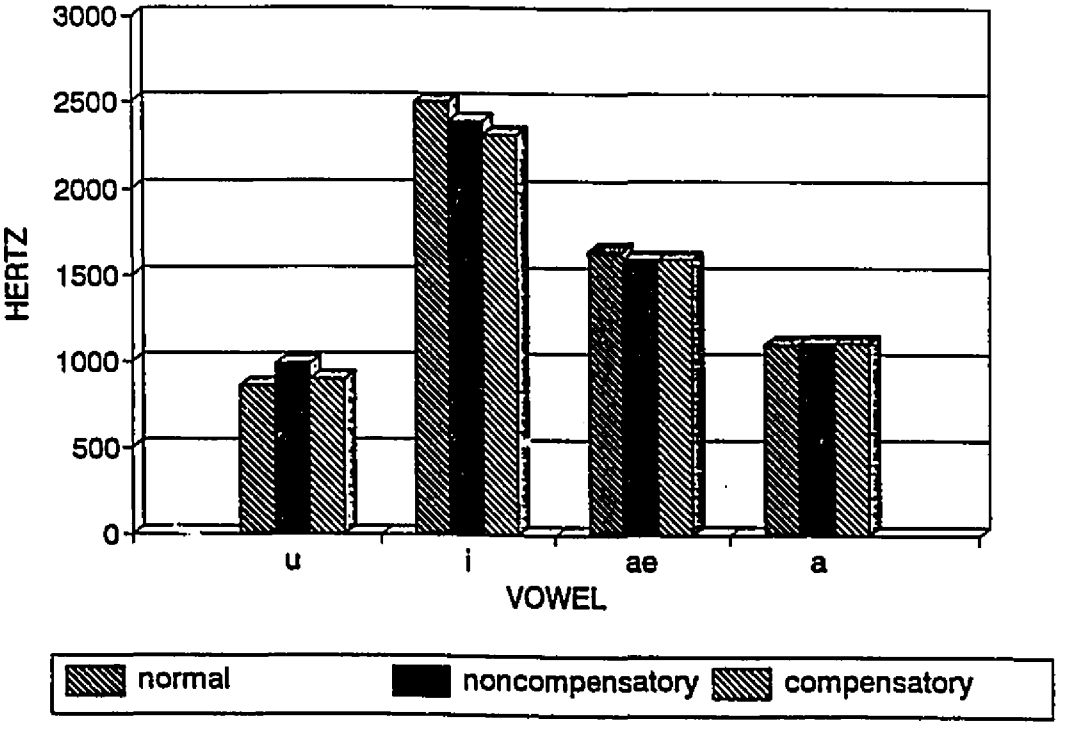


Figure 2b. Mean F2 for normal and bite block productions of normal subjects measured at the midpoint of vowel production



produced under normal speaking conditions. However, the statistical analyses would suggest that compensation for maximum perturbations was not always achieved by normal subjects.

Nonfluent aphasics

Mean F1 values for the nonfluent aphasic group are provided in Figures 3a and 3b. The figures reveal an overall increase in F1 for all vowels except [æ] in which a lowering of F1 was the trend observed in both bite block conditions relative to the normal condition at both measurement points. A measurement point x vowel x condition ANOVA yielded significant main effects of vowel ($F(3,12)=353.03, p<0.001$) and condition ($F(2,8)=8.16, p=0.02$). However, no significant differences between conditions emerged with a moderately conservative post-hoc test such as the Newman-Keuls test ($\alpha=0.05$). No additional main effects or interactions were found.

As shown in Figures 4a and 4b, the presence of a bite block had variable effects on F2 across the four vowels. Statistical analyses revealed a significant main effect for vowel ($F(3,15)=139.72, p<0.001$) and a vowel x condition interaction ($F(6,30)=2.70, p<0.05$). A post hoc Newman-Keuls test ($\alpha=0.05$) of the interaction demonstrated a significant difference between F2 values in the normal condition and both bite block conditions for [a] only. The two bite block

Figure 3a. Mean F1 for normal and bite block productions of nonfluent aphasics measured at the onset of vowel production

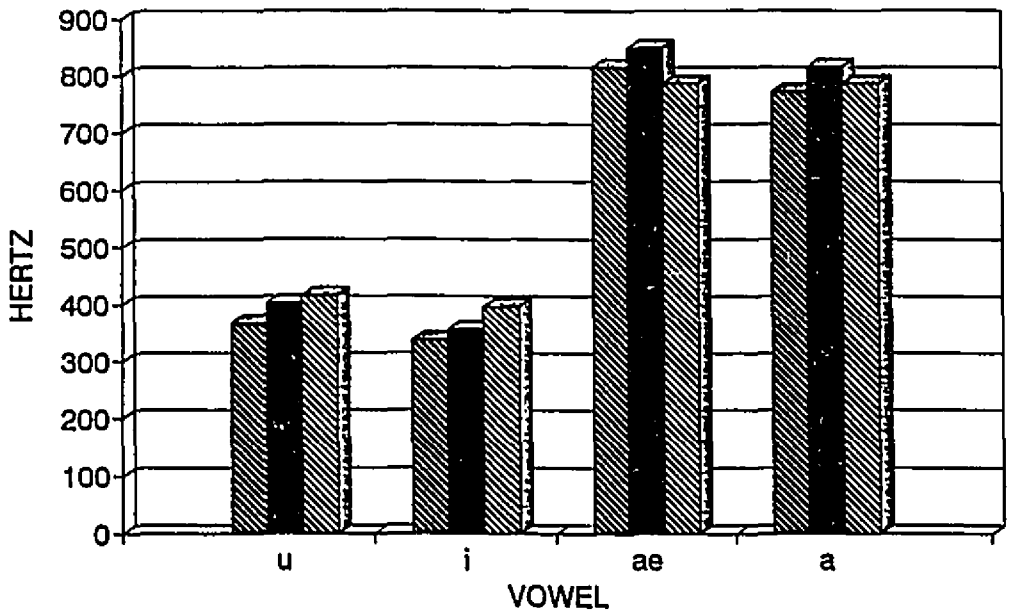


Figure 3b. Mean F1 for normal and bite block productions of nonfluent aphasics measured at the midpoint of vowel production

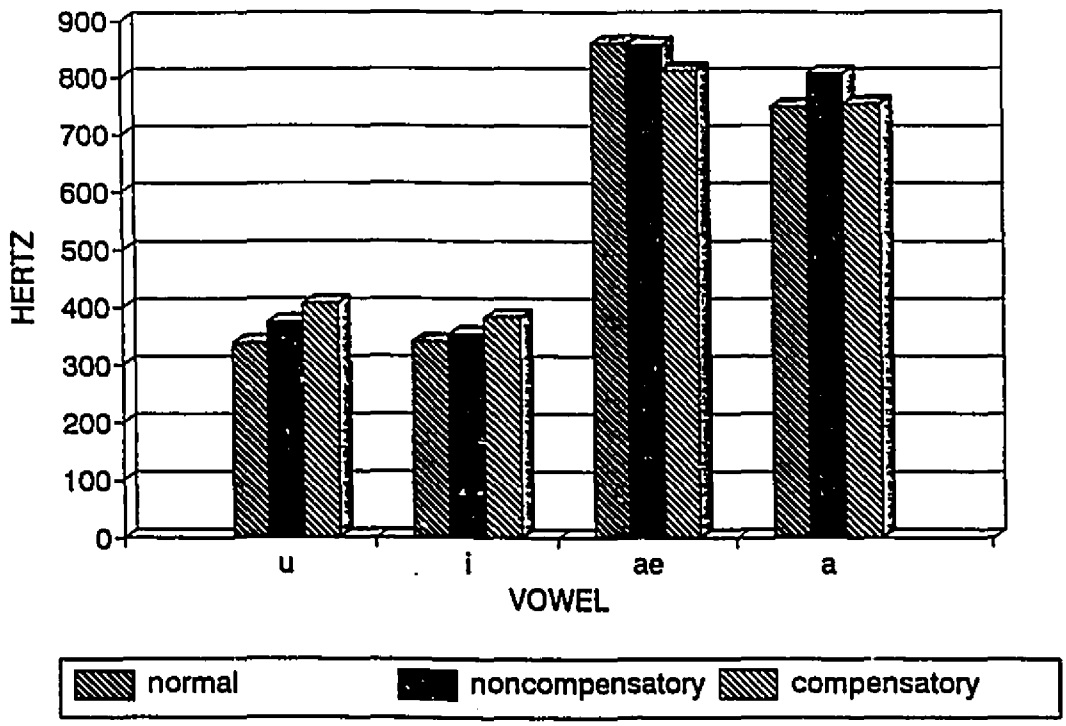


Figure 4a. Mean F2 for normal and bite block productions of nonfluent aphasics measured at the onset of vowel production

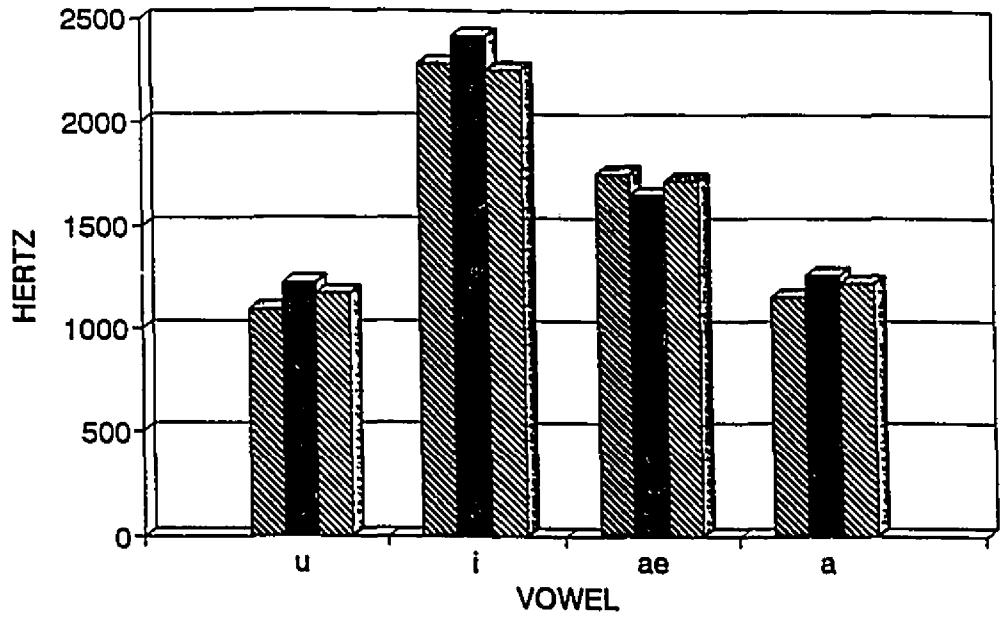
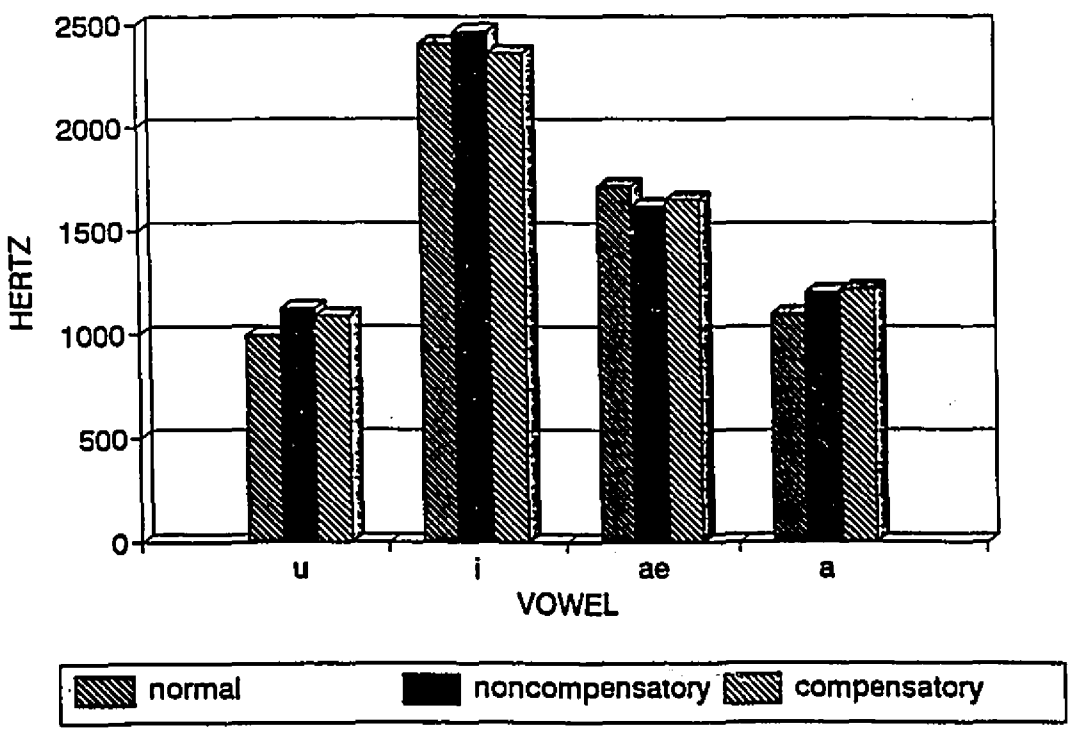


Figure 4b. Mean F2 for normal and bite block productions of nonfluent aphasics measured at the midpoint of vowel production



conditions did not differ from one another.

Fluent aphasics

Mean F1 values obtained for the fluent aphasic group are displayed in Figures 5a and 5b. As shown in the figures, the influence of speaking condition on F1 differed for the high and low vowels, with generally higher F1 values in the bite block conditions for [i] and [u] and somewhat decreased F1 values in the bite block conditions for [æ] and [a]. A measurement point x vowel x condition ANOVA yielded significant main effects for measurement point ($F(1,4)=12.69, p<0.05$), vowel ($F(3,12)=362.88, p<0.001$) and condition ($F(2,8)=14.54, p=0.002$). A comparison of F1 between the onset and midpoint of phonation revealed an overall decrease in formant value over time. The same trend was observed in all speaking conditions. A vowel x condition interaction also emerged ($F(6,24)=3.78$, $p<0.01$) which was further analyzed by Newman-Keuls tests ($\alpha=0.05$) revealing significant differences between the normal and compensatory F1 values of all vowels. The compensatory conditions for [i] and [æ], were also significantly different from the noncompensatory conditions. No significant differences emerged between normal and noncompensatory conditions for any of the vowels tested.

Mean F2 values for the fluent group are presented in Figures 6a and 6b. A measurement point x vowel x condition

Figure 5a. Mean F1 for normal and bite block productions of fluent aphasics measured at the onset of vowel production

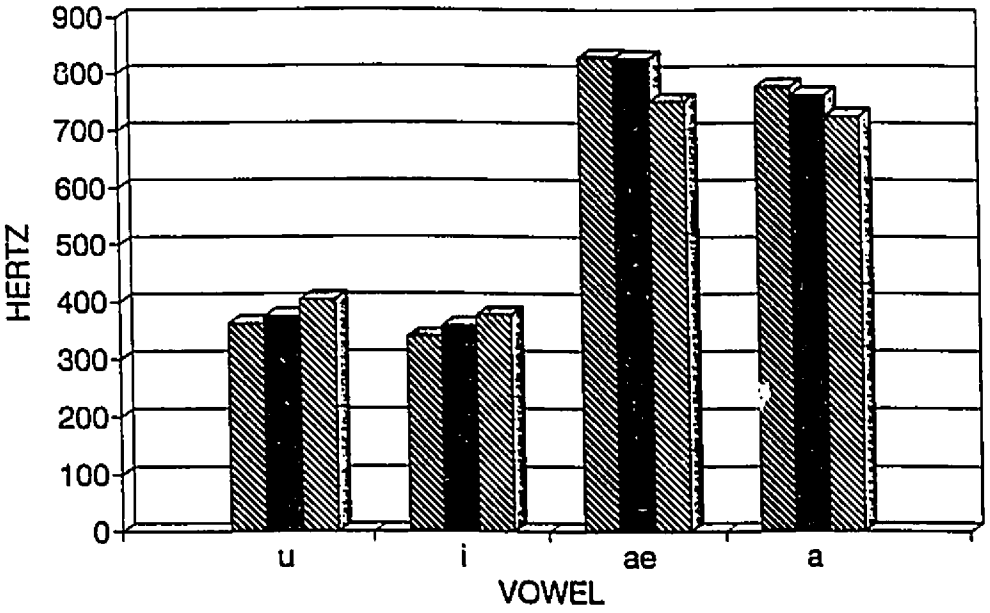


Figure 5b. Mean F1 for normal and bite block productions of fluent aphasics measured at the midpoint of vowel production

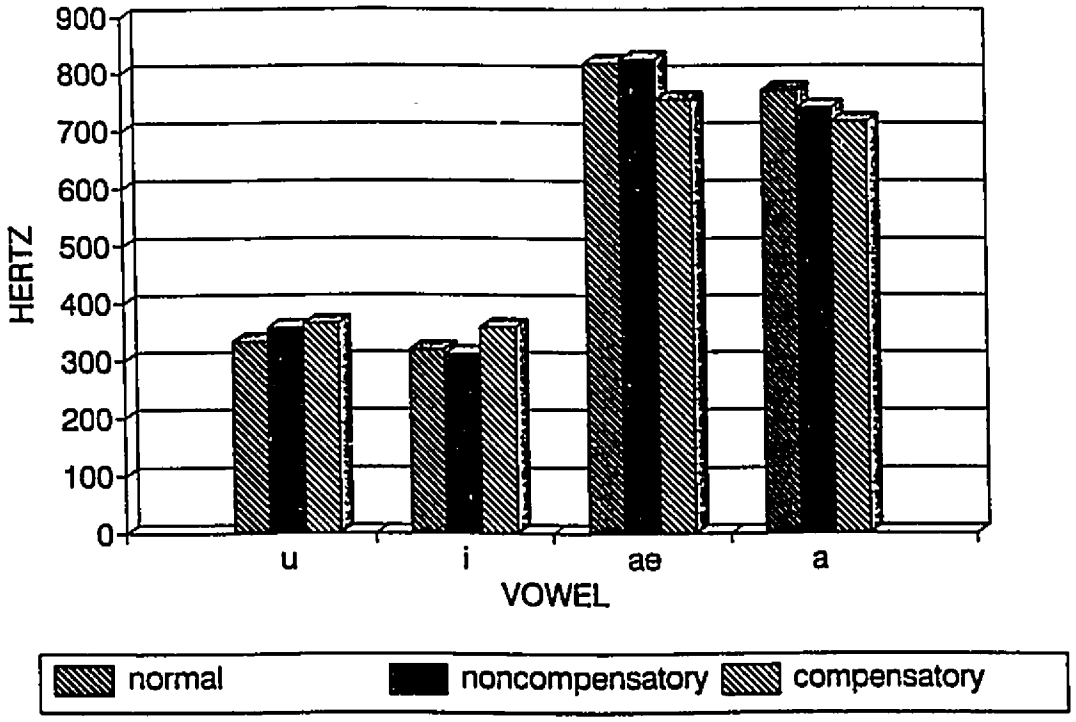


Figure 6a. Mean F2 for normal and bite block productions of fluent aphasics measured at the onset of vowel production

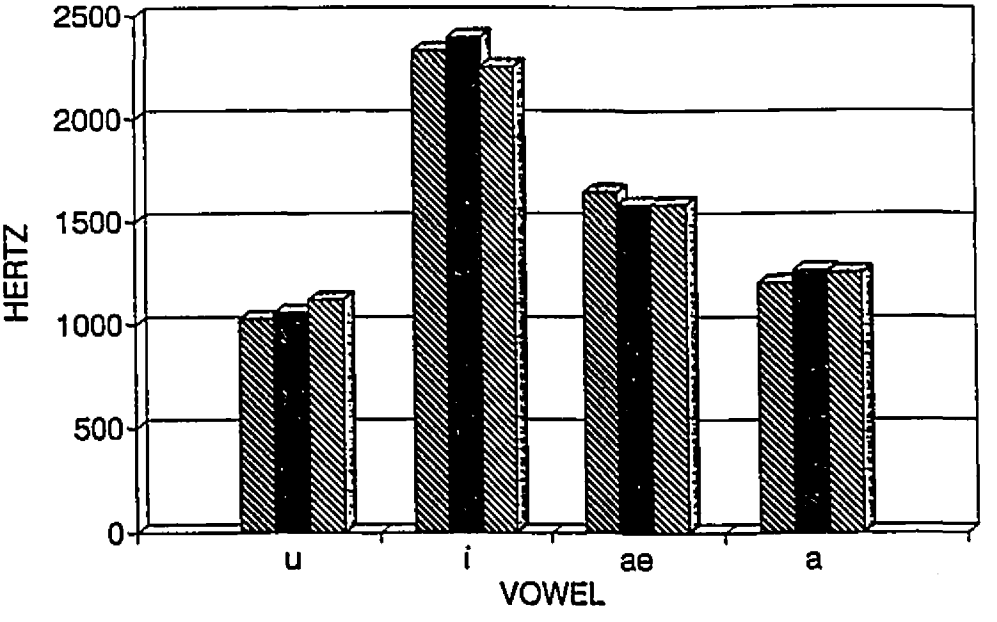
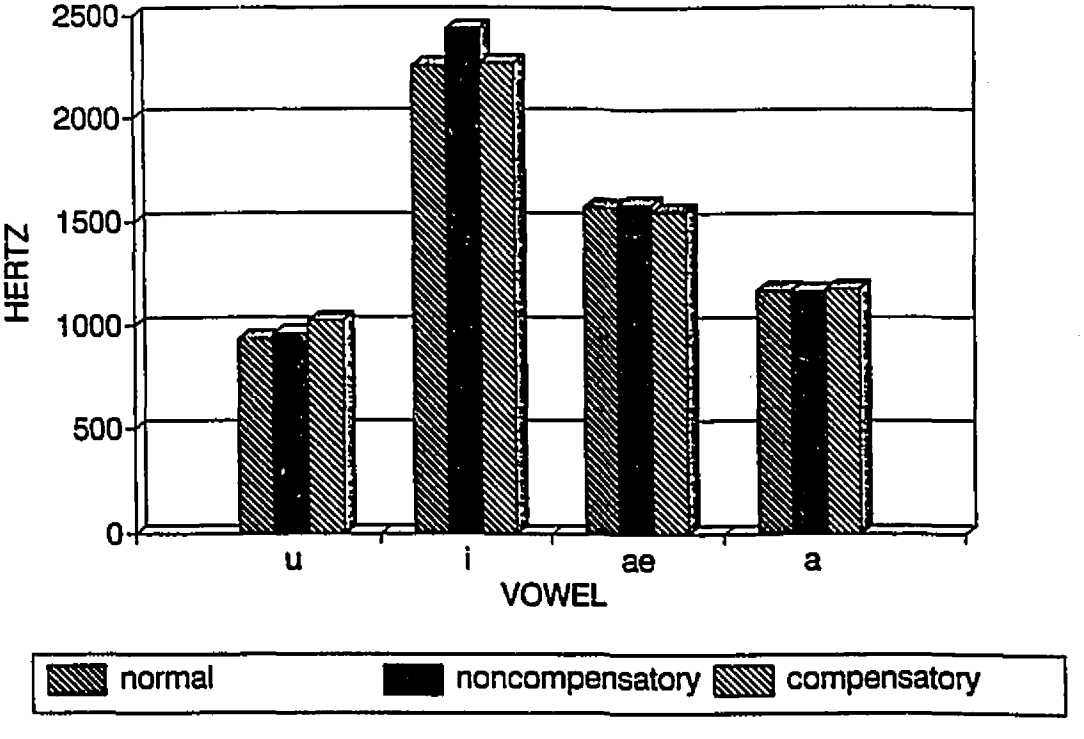


Figure 6b. Mean F2 for normal and bite block productions of fluent aphasics measured at the midpoint of vowel production



ANOVA yielded a significant effect of measurement point ($F(1,5)=27.47, p=0.003$) and vowel ($F(3,15)=159.38, p<0.001$) only. Similar to F1, a comparison of F2 between measurement points indicated that values at the midpoint were generally lower than at the onset of phonation in all speaking conditions. However, unlike F1 and any of the previous ANOVA's, no significant main effect, nor any interactions were found for speaking condition, suggesting that fluent patients were successful in maintaining normal F2 values. As similar findings were not obtained for F1, it would appear that the fluent subjects demonstrated partial compensation for maximum articulatory perturbation as did the normal and nonfluent subjects.

In addition, increased variability, which has often been a characteristic feature of aphasic speech production as compared to normal controls, was not evident in the present investigation. In general, as may be seen from Appendices 1-12, standard deviation values were quite comparable across groups and across conditions. The latter would suggest that articulatory perturbations did not contribute to greater formant variability as compared to normal vowel production. In some instances (e.g. F2 values of [æ] in normal subjects), formant variability under normal speaking conditions was shown to exceed that observed in corresponding compensatory and noncompensatory conditions.

Analyses with respect to perceptual difference limens

Following the statistical analyses, the data were further analyzed to determine whether formant differences that emerged across conditions would exceed perceptually significant boundaries.

The effects of formant frequency variation on the perception of vowels has been studied by several investigators (Flanagan, 1955; Hawks, 1994; Kewley-Port & Watson, 1994). Flanagan's findings suggested that a 3-5% variation of formant frequencies was the minimum perceptible change, or difference limen, resulting in a perceptible degradation in vowel quality. However, according to Flanagan (1955), difference limens of 3-5%, although perceptible, did not result in phonemic differences. In contrast, difference limens of a greater magnitude were more likely to extend into neighboring phonemic boundaries, giving rise to phonemic changes.

However, finer difference limens of 1-2% have been reported in more recent investigations (Hawks,1994; Kewley-Port & Watson, 1994). For example, Hawks (1994) conducted an extensive study of difference limens for synthetic vowels with multiformant and single formant variation. Difference limens for single-formant variation, although greater than those obtained for multiformant variation, were reported to be 1.82% and 1.97% for F1 and F2, respectively. Hawks (1994) therefore concluded auditory discrimination of formant variation to be significantly better than previously reported

when minimal stimulus uncertainty paradigms are invoked.

As the goal of the present study was to investigate a speaker's ability to maintain an acoustic and perceptual resemblance to a target (i.e. compensatory ability) rather than a listener's absolute discrimination skills, the larger difference limens (Flanagan, 1955) were selected to provide a better measure of compensatory ability. If the differences exceeded these maximal perceptual difference limens, it was expected that they would be salient to listeners. One caveat should be noted here regarding the interpretation of the difference limen data. That is, investigations of difference limens are conducted for synthetic stimuli under optimum listening conditions and typically for formants in isolation. Difference limens for the detection of minimal deviations in frequency may not be directly applicable to the perception of vowel quality in normal speech production. Therefore, the perceptibility of formant deviations in the bite block conditions may in fact be less salient than the difference limen data would suggest. Nonetheless, the difference limens provide a metric by which to evaluate deviations from normal values.

Three and five percent difference limens for each subject group were calculated separately for each vowel according to the mean F1 and F2 values obtained under normal speaking conditions. Mean F1 and F2 values used for this calculation were a group average of the formant values at the onset and

midpoint. Difference limens computed for each vowel and subject group are listed in Tables 3a-3c.

In analyzing the data according to the magnitude of formant deviations in terms of difference limens, compensatory and noncompensatory productions were classified according to the following categories: (i) less than 3%, (ii) between 3% and 5% and (iii) exceeding 5% difference limens as compared to normal productions. Productions exceeding either 3% or 5% difference limens (ii and iii) were considered "unsuccessful" approximations of the target. Therefore, successful compensation was considered to have taken place if difference limens were less than 3% (i).

Individual mean formant differences, calculated by subtracting the mean formant values obtained for the normal condition from that obtained in each bite block condition for each subject, vowel and measurement point are presented in Appendices 13-18. For each subject, 8 between-condition differences were calculated for each bite block condition, resulting in a total of 16 tokens for analysis. These differences were then classified according to the three difference limen categories outlined above. The numbers in parentheses accompanying the calculated differences indicate the difference limen that was exceeded. For example, a classification of (5) indicates that the difference exceeded the 5% difference limen for that formant. A classification of (3) indicates a difference which exceeds the 3% difference

Table 3a. F1 and F2 frequency difference limens for normal subjects (Hz)

Vowel	Mean F1	3%	5%	Mean F2	3%	5%
[u]	342	10	17	903	27	45
[i]	329	10	16	2475	74	124
[æ]	788	24	39	1674	50	84
[a]	715	21	36	1099	33	55

Table 3b. F1 and F2 frequency difference limens for nonfluent subjects (Hz)

Vowel	Mean F1	3%	5%	Mean F2	3%	5%
[u]	352	11	18	1042	31	52
[i]	339	10	17	2363	71	118
[æ]	837	25	42	1733	52	87
[a]	760	23	38	1130	34	56

Table 3c. F1 and F2 frequency difference limens for fluent subjects (Hz)

Vowel	Mean F1	3%	5%	Mean F2	3%	5%
[u]	348	10	17	984	30	49
[i]	329	10	16	2344	70	117
[æ]	825	25	41	1607	48	80
[a]	774	23	39	1185	36	59

limen only and is therefore, between 3 and 5%. Mean difference values not accompanied by a number in parentheses indicate that the differences were less than 3% difference limens and therefore, successful instances of compensation.

A summary of group performance according to the percentage of between-condition differences in each difference limen category, derived from Appendices 13-18, is provided in Tables 4-6 for each subgroup separately. The data in Table 4 indicate a surprisingly high percentage of unsuccessful productions in the normal subject group. For the noncompensatory condition, the percentage of mean differences, combined for F1 and F2, exceeding either the 3 or 5% difference limen ranged from 42-75%. An even larger range of 55-90% unsuccessful compensation was obtained for the compensatory condition.

Tables 5 and 6 display the findings for the aphasic groups. For the nonfluent patients, the percentage of productions exceeding either the 3 or 5% difference limen in the noncompensatory condition ranged from 30-93%; the percentage of unsuccessful compensation in the compensatory condition, ranged from 42-92%. Analysis of mean formant differences in the fluent patient group data indicated that 36-83% of noncompensatory F1 and F2 differences exceeded the 3 or 5% difference limen. Mean differences exceeding the 3 or 5% difference limen in the compensatory condition accounted for 33-100% of the fluent group's productions.

Table 7, which displays the percentage of successful bite block production for each subject, indicates that for most individuals, normal and aphasic, successful compensation was shown to occur less than 50% of the time, according to these relatively stringent criteria.

Table 4. Summary of group mean differences according to difference limens for normal subjects

Vowel	Difference limen category	F1				F2			
		NC-N		C-N		NC-N		C-N	
		# Productions	% Productions	# Productions	% Productions	# Productions	% Productions	# Productions	% Productions
[u]	>5	8	40	5	25	11	58	9	50
	>3	5	25	6	30	1	5	1	6
	<3	7	35	9	45	7	37	8	44
	Total unsuccessful	13	65	11	55	12	63	10	56
	Total successful	7	35	9	45	7	37	8	44
[i]	>5	11	55	16	80	4	21	12	63
	>3	1	5	0	0	4	21	3	16
	<3	8	40	4	20	11	58	4	21
	Total unsuccessful	12	60	16	80	8	42	15	79
	Total successful	8	40	4	20	11	58	4	21
[æ]	>5	9	45	17	85	8	40	8	40
	>3	4	20	1	5	3	15	4	20
	<3	7	35	2	10	9	45	8	40
	Total unsuccessful	13	65	18	90	11	55	12	60
	Total successful	7	35	2	10	9	45	8	40
[a]	>5	11	55	8	40	10	50	11	55
	>3	4	20	4	20	3	15	3	15
	<3	5	25	8	40	7	35	6	30
	Total unsuccessful	15	75	12	60	13	65	14	70
	Total successful	5	25	8	40	7	35	6	30

Noncompensatory (NC), Compensatory (C), Normal (N)

Table 5. Summary of group mean differences according to difference limens for nonfluent aphasics

Vowel	Difference limen category	F1				F2			
		NC-N		C-N		NC-N		C-N	
		# Productions	% Productions	# Productions	% Productions	# Productions	% Productions	# Productions	% Productions
[u]	>5	8	67	7	58	7	64	8	67
	>3	0	0	0	0	1	9	3	25
	<3	4	33	5	42	3	27	1	8
	Total unsuccessul	8	67	7	58	8	73	11	92
	Total successful	4	33	5	42	3	27	1	8
[i]	>5	7	58	11	92	2	20	6	55
	>3	3	25	0	0	1	10	1	9
	<3	2	17	1	8	7	70	4	36
	Total unsuccessful	10	83	11	92	3	30	7	64
	Total successful	2	17	1	8	7	70	4	36
[æ]	>5	5	50	5	42	4	33	2	17
	>3	2	20	2	17	2	17	3	25
	<3	3	30	5	42	6	50	7	58
	Total unsuccessful	7	70	7	59	6	50	5	42
	Total successful	3	30	5	42	6	50	7	58
[a]	>5	6	50	6	50	9	75	7	58
	>3	1	8	4	33	2	17	3	25
	<3	5	42	2	17	1	8	2	17
	Total unsuccessful	7	58	10	83	11	93	10	83
	Total successful	5	42	2	17	1	8	2	17

Noncompensatory (NC), Compensatory (C), Normal (N)

Table 6. Summary of group mean differences according to difference limens for fluent aphasics

Vowel	Difference limen category	F1				F2			
		NC-N		C-N		NC-N		C-N	
		# Productions	% Productions	# Productions	% Productions	# Productions	% Productions	# Productions	% Productions
[u]	>5	7	58	11	92	10	83	8	73
	>3	1	8	1	8	0	0	1	9
	<3	4	33	0	0	2	17	2	18
	Total unsuccessul	8	66	12	100	10	83	9	82
	Total successful	4	33	0	0	2	17	2	18
[i]	>5	5	42	8	67	4	36	10	91
	>3	4	33	2	17	0	0	1	9
	<3	3	25	2	17	7	64	0	0
	Total unsuccessful	9	75	10	84	4	36	11	100
	Total successful	3	25	2	17	7	64	0	0
[æ]	>5	5	45	7	64	6	50	4	33
	>3	1	9	1	9	1	8	2	17
	<3	5	45	3	27	5	32	6	50
	Total unsuccessful	6	54	8	73	7	58	6	50
	Total successful	5	45	3	27	5	42	6	50
[a]	>5	6	50	8	67	3	25	4	33
	>3	2	17	0	0	4	33	0	0
	<3	4	33	4	33	5	42	8	67
	Total unsuccessful	8	67	8	67	7	58	4	33
	Total successful	4	33	4	33	5	42	8	67

Noncompensatory (NC), Compensatory (C), Normal (N)

Table 7. Percentage successful bite block productions for individual subjects

Subject	F1			F2		
	% of noncompensatory productions	% of compensatory productions	Total %	% of noncompensatory productions	% of compensatory productions	Total %
N1	13	25	38	21	7	29
N2	25	19	44	14	14	29
N3	13	13	25	38	31	69
N4	13	6	19	6	13	19
N5	13	19	31	19	19	38
N6	19	13	31	27	7	33
N7	19	13	31	31	13	44
N8	19	19	38	6	25	31
N9	13	13	25	25	25	50
N10	13	19	31	31	13	44
Group range	13-25	6-25	19-44	6-38	7-31	19-61
NF1	0	6	6	19	25	44
NF2	19	25	44	31	6	38
NF3	25	6	31	25	13	38
NF4	21	36	57	23	23	46
NF5	19	6	25	0	13	13
NF6	6	6	13	13	13	25
Group range	0-25	6-36	6-57	0-31	6-25	13-46
F1	0	14	14	0	14	14
F2	0	0	0	33	13	47
F3	31	6	38	31	19	50
F4	25	13	38	13	19	31
F5	25	0	25	19	13	31
F6	19	25	44	25	25	50
Group range	0-31	0-25	0-44	0-33	15-25	14-50

Normal (N), Nonfluent (NF), Fluent (F)

CHAPTER 5

Discussion

Results of the present investigation suggest that compensatory articulation is a preserved skill in brain-damaged individuals as differences in compensatory performance between normal and aphasic groups were not apparent. However, before between-group comparisons are drawn, implications of the findings for normal and aphasic speech production and in particular, with reference to the various models described in the literature, will be considered.

The present study corroborates the more recent investigations of compensatory articulation in which normal subjects have been shown to possess a remarkable but limited capacity to adapt to articulatory perturbation (Fowler & Turvey, 1980; Flege et al., 1988; McFarland & Baum, in press). In contrast to the earlier findings of Lindblom and Sundberg (1971) and Lindblom et al., (1979), statistical analyses of the F1 and F2 values extracted from normal and bite block vowel productions revealed significant effects of perturbation on vowel production. More specifically, for F1, a significant decrease was observed in compensatory productions of [æ] as compared to normal productions. Significant lowering in F2 was also shown for compensatory productions of both [æ] and [i].

Although systematic deviations in vowel formants between normal and bite block productions were reported by Lindblom et al. (1979), these differences were not judged to be perceptually significant in an informal listening task. However, analysis of the present data with respect to perceptual difference limens (Flanagan, 1955) leads to a different conclusion. In addition to being statistically significant, the formant differences observed in the compensatory productions of [i] and [æ] relative to normal productions were shown to exceed 3-5% difference limens, suggesting a change in vowel quality. Furthermore, a similar analysis of the vowels [u] and [a], for which normal and bite block productions did not differ significantly in terms of acoustic measures, revealed formant deviations that would were categorized as unsuccessful target approximations according to difference limen criteria. Again, it is important to point out that the difference limen metric utilized was quite strict; thus interpretation of these data in terms of perceptual salience must be done cautiously.

Interestingly, although formant patterns in noncompensatory productions were not statistically different from normal productions, a comparison of formant means indicated that these differences also exceeded the difference limen criteria for successful compensation. Thus, even relatively minor vocal tract perturbations may affect the speech quality of normal subjects. However, as mentioned

earlier, the limitations of employing difference limen data to evaluate bite block productions as being perceptually acceptable must be considered.

As reported by Fowler and Turvey (1980) and McFarland and Baum (in press), it is also important to recognize that the normal subjects in the present investigation did demonstrate partial compensation for the perturbations. Formant deviations between normal and bite block productions were shown to be smaller than predicted by Lindblom and Sundberg's (1970) model in many cases. According to Lindblom and Sundberg (1970), the magnitude of F1 deviation in the absence of any articulatory adaptation was several hundred Hertz for some of the vowels tested. As the magnitude of F1 deviation between normal and bite block productions was much smaller in many instances, some adaptation to perturbed jaw positioning appeared to have taken place. Moreover, it is important to recognize that the mere presence of the bite block within the oral cavity may have contributed to changes in its resonance characteristics. It is also important to point out individual differences in compensatory ability within the normal subjects which may, in part, be due to differences in vocal tract morphology and the degree to which the bite block perturbed normal articulatory configurations (McFarland & Baum, in press).

The use of feedback for purposes of making on-line adjustments by normal subjects was not evident as the data

revealed no significant formant changes between the onset and midpoint of vowel production. However, studies such as that conducted by McFarland and Baum (in press) suggest a long-term role of feedback in the learning and implementation of compensatory motor programs. Their findings of improvement in compensatory vowel production following only a 15-minute period of conversation with the perturbation in place could be indicative of feedback-mediated adjustments made over time as opposed to a moment-to-moment evaluation of and adjustment to the perturbation. Feedback may therefore be critical in the acquisition and maintenance of novel articulatory patterns necessary for compensatory articulation. Although, no direct evidence for the role of feedback was provided by the current results, the findings for normal speakers, coupled with results of other investigations (e.g. McFarland & Baum, in press) are most compatible with models of speech motor control that provide a role for sensory feedback in articulation (Mlcoch & Noll, 1980) as opposed to models which assume immediate or complete compensation due to dynamic muscle linkages (e.g. Kelso & Tuller, 1983).

Turning to the results for the nonfluent aphasics, the data suggested that, contrary to previous reports (Sussman et al., 1986), compensatory performance among nonfluent subjects was comparable to that observed in normal subjects. Similar to normal subjects, F1 values in the bite block conditions were shown to be significantly different from those produced

under normal speaking conditions. For F2, a significant increase in frequency was noted under compensatory and noncompensatory conditions for [a]. The bite blocks had no significant effect on the F2 values of the other vowels tested. As will be recalled, a considerable percentage of compensatory and noncompensatory F1 and F2 values exceeded 3-5% difference limens, indicating significant deviations from the target. But again, similar to normal speakers, nonfluent aphasics demonstrated partial compensation for fixation of the mandible as formant deviations between the normal and bite block productions were often smaller than Lindblom and Sundberg's (1971) model would predict had no articulatory adjustments occurred.

The similarity of findings for the normal and nonfluent subjects, (i.e. partial compensation for articulatory perturbations) would suggest that attempts to identify a cortical area or neural structure underlying compensatory articulation may be difficult. Claims of a discrete cortical region such as Area 44 (Sussman et al., 1986) or the operation of a single system such as a motor speech programmer localized to Broca's area (Darley et al., 1975) in achieving compensatory articulation are not supported by the present data. If such claims were accurate, one would expect to find a considerable deficit in compensatory ability in the nonfluent subjects. It may be argued, however, that these individuals may not have sustained damage to the critical area

(i.e. Broca's area) and were thus able to compensate in a manner similar to normals. As precise lesion information was not available for the brain-damaged subjects in the present study, this possibility merits consideration. In fact, for some patients (e.g. DI and AG), cerebral infarcts were confined to parietal regions, suggesting Broca's area had been spared. However, for most of the nonfluent speakers, some involvement of Broca's area or adjacent cortical and subcortical regions involved in the motor control of speech production can be assumed.

Alternatively, motor programming deficits in articulatory adaptation may only become apparent with increasing task complexity. It is possible that a breakdown in compensatory articulation may be observed in more demanding speech tasks, such as conversation as opposed to isolated vowel production. Or perhaps, group differences may emerge in the ability to learn compensatory motor commands over time.

The use of feedback in making short-term, on-line adjustments by the nonfluent group was not evident. As was the case for the normal subjects, formant values at the onset of vowel production were not significantly different from those at the midpoint. And, as always, an interpretation of null results must be made with great caution. Overall, as compared to the normal speakers, the performance of the nonfluent aphasic subjects was unremarkable. This suggests that the speech production deficits of these subjects do not

implicate the ability to reorganize motor commands in response to perturbation. Perhaps, as has been suggested in the literature, the speech production deficits of nonfluent aphasics are constrained largely to impairments in temporal control (e.g. Blumstein & Baum, 1987).

Consistent with findings for the normal and nonfluent subjects groups, compensation for articulatory perturbation in fluent aphasics was best described as variable or partial. Significant changes in F1 were noted for all the vowels tested when produced under compensatory speaking conditions. Somewhat surprisingly, analysis of the F2 data in the fluent group yielded a nonsignificant effect of bite block across all vowels tested. This finding would suggest that the fluent group exhibited some consistency in their ability to maintain the area function in a manner which preserved the F2 patterns of the target vowels. However, this interpretation should be tempered by several factors. As shown in Appendix 18, considerable individual variability was observed in achieving target F2 values, making it difficult to attribute complete compensation to the fluent group. Furthermore, as was the case with the normal and nonfluent subject groups, the difference limen data do not support such a claim. As may be recalled, F2 deviations in bite block productions as compared to normal productions were often shown to exceed perceptual difference limens (Table 6) and were therefore expected to be salient to listeners.

Although the statistical analyses for the fluent speakers suggested limited changes in production over time, formant changes between the onset and midpoint of vowel production were not limited to bite block productions, but were observed for normal productions as well. These findings could be indicative of subtle deficits in articulatory positioning in the fluent patients or an inability to maintain a steady-state vowel. Alternatively, the large variability within the group may account for the anomalous statistical findings. Regardless, as significant interactions with speaking conditions did not emerge, the use of sensory feedback in performing on-line compensatory adjustments in response to articulatory perturbation was not apparent.

Conclusion

In summary, all three subjects groups demonstrated partial compensation to articulatory perturbation. Complete articulatory reorganization in response to compensatory and noncompensatory speaking conditions was therefore not achieved immediately nor did it appear to be achieved on a moment-to-moment evaluation and correction basis (McFarland & Baum, in press). Furthermore, group differences were not apparent as variable compensatory performance between vowels and bite block conditions was the pattern observed for the normal controls and the nonfluent and fluent aphasic subjects. The findings of the present study have important implications for

both models of speech motor control and neurogenic models of speech production.

Several models of speech motor control such as the central simulation model (Lindblom et al., 1979) and the dynamic model (Fowler et al., 1980; Kelso & Tuller, 1983) have been proposed to account for the immediacy of articulatory compensation. However, results of the present investigation suggest that articulatory reorganization is not immediate but may be achieved over time. Although the use of feedback in achieving compensatory articulation was not evident in any of the subject groups tested, long-term use of feedback in the adaptation to articulatory perturbation requires further investigation.

Compensatory performance among all subject groups was comparable as each group exhibited variable and partial compensation, a finding that is inconsistent with current neurogenic models of speech production which postulate a discrete area or mechanism underlying speech motor programming. For example, the model of speech production proposed by Darley et al.(1975) would predict nonfluent aphasics, presumed to have damage to the motor speech programmer, to exhibit impaired compensatory articulation. However, as the nonfluent aphasics did not demonstrate impaired compensatory performance, one may speculate that there is more diffuse representation of motor speech programming (see e.g. Alexander, Naeser & Palumbo, 1987;

Barlow & Farley, 1989). However, as specific lesion information for the aphasic subjects was unavailable, this remains open to question.

Moreover, as compensatory abilities in the nonfluent group appeared to be unimpaired, results of the present investigation provide further evidence to suggest that the phonetic deficits typically associated with nonfluent speech production are not pervasive. Rather, movement-level disturbances may surface in dynamic processes of speech production (e.g. temporal coordination of articulators for the production of nasal phonemes) as opposed to more static features (e.g. vocal tract configuration for vowel production).

Although vowel production was examined under two bite block conditions to uncover possible differences in compensatory abilities for minimal and maximal perturbation, no consistent differences between compensatory and noncompensatory conditions were found for any of the subject groups. As mentioned earlier, deterioration in compensatory performance may emerge in tasks of greater complexity (e.g. in conversation) or in the production of different speech segments (e.g. consonants).

The need for further research on speech motor programming and in particular, compensatory articulation, in both normal and brain-damaged subjects is clear. One of the important issues in normal speech motor control which needs to be

explored is the capacity of normal individuals to achieve complete compensation over time. Similarly, the capacity of aphasic subjects to ultimately achieve complete compensation should also be investigated in greater detail. However, in order to determine the relationship, if any, between discrete cortical regions or structures (i.e. a motor speech programmer) and compensatory skill, more detailed lesion information will be essential. In addition, varying the speech task in complexity and analyzing both consonant and vowel segments may uncover changes in compensatory performance that were not revealed in the present investigation.

REFERENCES

Abbs, J.H. (1986). Invariance and variability in speech production: a distinction between linguistic intent and its neuromotor implementation. In J. Perkell & D. Klatt (Eds.), *Invariance and Variability in Speech Processes*. New Jersey: Lawrence Erlbaum Associates.

Abbs, J.H. & Eilenberg, G.R. (1976). Peripheral mechanisms of speech motor control. In N.J. Lass (Ed.), *Contemporary Issues in Experimental Phonetics*. New York: Academic Press.

Abbs, J.H. & Gracco, V.L. (1984). Control of complex motor gestures: orofacial muscle responses to load perturbations of lip during speech. *Journal of Neurophysiology*, 51, 705-723.

Alexander, M., Naeser, M. & Palumbo, C. (1987). Correlations of subcortical CT lesion sites and aphasia profiles. *Brain*, 110, 961-991.

American National Standard Institute (1972b). *Specification for Audiometers*. ANSI S3.6-1969, New York.

Barlow, S.M. & Farley, G.R. (1989). Neurophysiology of speech. In D.P. Kuehn, M.L. Lemme and J. Baumgartner (Eds.), *Neural Bases of Speech, Hearing, and Language*. Boston: College Hill Press.

Baum, S.R., Blumstein, S.E., Naeser, M.A., & Palumbo, C.L. (1990). Temporal dimensions of consonant and vowel production: an acoustic and CT scan analysis of aphasic speech. *Brain and Language*, 39, 33-56.

Blumstein, S.E. (1990). Phonological deficits in aphasia: theoretical perspectives. In A. Caramazza (Ed.), *Cognitive Neuropsychology and Neurolinguistics: Advances in Models of Cognitive Function and Impairment*. New Jersey: Lawrence Erlbaum Associates.

Blumstein, S.E. & Baum, S.R. (1987). Consonant production deficits in aphasia. In J. Ryalls (Ed.), *Phonetic Approaches to Speech Production in Aphasia and Related Disorders*. Boston: College-Hill.

Blumstein, S.E., Cooper, W.E., Goodglass, H., Statlender, S., & Gottlieb, J. (1980). Production deficits in aphasia: a voice-onset time analysis. *Brain and Language*, 9, 153-170.

Dabul, B. (1979). *Apraxia Battery for Adults*, Austin: Pro-Ed.

Damasio, H. (1991). Neuroanatomical correlates of the aphasias. In M.T. Sarno (Ed.), *Acquired Aphasia* (2nd ed). New York: Academic Press.

Darley, F., Aronson, A., & Brown, J. (1975). *Motor Speech Disorders*. Philadelphia: Saunders.

Flanagan, J. (1955). A difference limen for vowel formant frequency. *Journal of the Acoustical Society of America*, 27, 613-617.

Flege, J.E., Fletcher, S.G., & Homiedan, A. (1988). Compensating for a bite block in /s/ and /t/ production: palatographic, acoustic, and perceptual data. *Journal of the Acoustical Society of America*, 83, 212-228.

Folkins, J.W. & Abbs, J.H. (1975). Lip and jaw motor control during speech: responses to resistive loading of the jaw. *Journal of Speech and Hearing Research*, 18, 207-220.

Folkins, J.W. & Abbs, J.H. (1976). Additional observations on responses to resistive loading of the jaw. *Journal of Speech and Hearing Research*, 19, 820-821.

Folkins, J.W. & Linville, R.N. (1983). The effects of varying lower-lip displacement on upper-lip movements: implications for the coordination of speech movements. *Journal of Speech and Hearing Research*, 26, 209-217.

Folkins, J.W. & Zimmerman, G.N. (1981). Jaw-muscle activity during speech with the mandible fixed. *Journal of the Acoustical Society of America*, 69, 1441-1445.

Fowler, C.A., Rubin, P., Remez, R.E., & Turvey, M.T. (1980). Implications for speech production of a general theory of action. In B. Butterworth (Ed.), *Language Production*, Vol. 1. London: Academic Press.

Fowler, C.A. & Turvey, M.T. (1980). Immediate compensation in bite-block speech. *Phonetica*, 37, 306-326.

Freeman, F.J., Sands, E.S., & Harris, K.S. (1978). Temporal coordination of phonation and articulation in a case of verbal apraxia: a voice-onset time study. *Brain and Language*, 6, 106-111.

Gandour, J. & Dardarananda, R. (1984b). Prosodic disturbance in aphasia: vowel length in Thai. *Brain and Language*, 23, 206-224.

Gay, T., Lindblom, B., & Lubker, J. (1981). Production of bite-block vowels: acoustic equivalence by selective compensation. *Journal of the Acoustical Society of America*, 69, 802-810.

Gay, T. & Turvey, M. (1979). Effects of efferent and afferent interference on speech production: implications for a generative theory of speech motor control. *Proceedings of the Ninth International Congress of Phonetic Sciences*, Copenhagen.

Goodglass, H. & Kaplan, E. (1983). *The Assessment of Aphasia and Related Disorders*. Philadelphia: Lea & Febiger.

Gracco, V.L. & Abbs, J.H. (1986). Variant and invariant characteristics of speech movements. *Experimental Brain Research*, 65, 156-166.

Hawks, J.W. (1994). Difference limens for formant patterns of vowel sounds. *Journal of the Acoustical Society of America*, 95, 1074-1084.

Hebb, D. (1949). *The Organization of Behavior*. New York: Wiley.

Hughes, O.M. & Abbs, J.H. (1976). Labial-mandibular coordination in the production of speech: implications for the operation of motor equivalence. *Phonetica*, 33, 199-221.

Itoh, M. & Sasanuma, S. (1984). Articulatory movements in apraxia of speech. In J. Rosenbek, M. McNeil, & A. Aronson (Eds.), *Apraxia of Speech: physiology, acoustics, linguistics, management*. San Diego: College-Hill.

Itoh, M., Sasanuma, S., & Ushijima, T. (1979). Velar movements during speech in a patient with apraxia of speech. *Brain and Language*, 7, 227-239.

Katz, W.F. & Baum, S.R. (1987). Compensatory articulation in Broca's aphasia: the facts aren't in yet: a reply to Sussman et al. *Brain and Language*, 30, 367-373.

Katz, W., Olness, G., Baum, S.R., & Kim, J.H. (1992). Compensatory articulation in Broca's aphasia. Poster presented at Academy of Aphasia, October 1992, Toronto, ON.

Kelso, J.A.S. & Tuller, B. (1983). "Compensatory articulation" under conditions of reduced afferent information: a dynamic formulation. *Journal of Speech and Hearing Research*, 26, 217-224.

Kennedy, J.L. (1983). Treatment of Wernicke's aphasia. In W.H. Perkins (Ed.), *Language Handicaps in Adults*. New York: Thieme-Stratton.

Kent, R.D. (1976). Models of speech production. In N.J. Lass (Ed.), *Contemporary Issues in Experimental Phonetics*. New York: Academic Press.

Kewley-Port, D. & Watson, C.S. (1994). Formant-frequency discrimination for isolated English vowels. *Journal of the Acoustical Society of America*, 95, 485-496.

Ladefoged, P., DeClerk, J., Lindau, M., & Papcun, G.A. (1972). An auditory-motor theory of speech production. *UCLA Working Papers in Phonetics*, 22, 48-75.

Lamarre, Y. & Lund, J.P. (1975). Load compensation in human masseter muscles. *Journal of Physiology*, 253, 21-35.

Liberman, A., Cooper, F., Shankweiler, D., & Studdert-Kennedy, M. (1967). Perception of the speech code. *Psychological Review*, 74, 431-461.

Lindblom, B., Lubker, J., & Gay, T. (1979). Formant frequencies of some fixed-mandible vowels and a model of speech motor programming by predictive simulation. *Journal of Phonetics*, 7, 147-161.

Lindblom, B., McAllister, R., & Lubker, J. (1977). Compensatory articulation and the modeling of normal speech production behavior. Paper presented at the Symposium on Articulatory Modeling, July 1977, Grenoble, France.

Lindblom, B.E.F. & Sundberg, J.E.F. (1971). Acoustical consequences of lip, tongue, jaw, and larynx movement. *Journal of the Acoustical Society of America*, 50, 1166-1179.

Lisker, L. & Abramson, A. (1964). A cross-language study of voicing in initial stops: acoustical measurements. *Word*, 20, 384-422.

Lubker, J. (1979). Motor control of speech gestures. *Proceedings of the Ninth International Congress of Phonetic Sciences*, Copenhagen.

MacNeilage, P.F. (1970). Motor control of serial ordering of speech. *Psychological Review*, 77, 182-196.

MacNeilage, P.F. (1980). Distinctive properties of speech motor control. In G.E. Stelmach & J. Requin (Eds.), *Tutorials in Motor Behavior*. Amsterdam: North Holland.

McFarland, D.H. & Baum, S.R. (in press). Incomplete compensation to articulatory perturbation. *Journal of the Acoustical Society of America*.

McNeil, M.R. & Kent, R.D. (1990). Motoric characteristics of adult aphasic and apraxic speakers. In G.E. Hammond (Ed.), *Cerebral Control of Speech and Limb Movements*. North-Holland: Elsevier Science Publishers.

Mertus, J. (1989). *Bliss*. Brown University, Providence, R.I.

Mlcoch, A. & Noll, J. (1980). Speech production models as related to the concept of apraxia of speech. In N.J. Lass (Ed.), *Speech and Language: Advances in basic research and practice*, Vol 4. New York: Academic Press.

Perkell, J.S. (1979). On the use of orosensory feedback: an interpretation of compensatory articulation experiments. *Proceedings of the Ninth International Congress of Phonetic Sciences*, Copenhagen.

Perkell, J.S. (1980). Phonetic features and the physiology of speech production. In B. Butterworth (Ed.) *Language Production*, Vol 1. London: Academic Press.

Peterson, G.E. & Barney, H.L. (1952). Control methods used in a study of vowels. *Journal of the Acoustical Society of America*, 24, 175-184.

Robin, D.A., Bean, C., & Folkins, J.W. (1989). Lip movement in apraxia of speech. *Journal of Speech and Hearing Research*, 32, 512-523.

Ryalls, J.H. (1986). An acoustic study of vowel production in aphasia. *Brain and Language*, 29, 48-67.

Ryalls, J.H. (1987). Vowel production in aphasia: towards an account of the consonant vowel-dissociation. In J.H. Ryalls (Ed.), *Phonetic Approaches to Speech Production in Aphasia and Related Disorders*. Boston: College-Hill.

Shewan, C., Leeper, H., & Booth, J. (1984). An analysis of voice-onset time (VOT) in aphasic and normal subjects. In J. Rosenbek, M. McNeil, & A. Aronson (Eds.), *Apraxia of Speech: physiology, acoustics, linguistics, management*. San Diego: College-Hill.

Shinn, P. & Blumstein, S. (1983). Phonetic disintegration in aphasia: acoustic analyses of spectral characteristics for place of articulation. *Brain and Language*, 20, 90-114.

Smith, A. (1992). The control of orofacial movements in speech. *Critical Reviews of Oral Biology and Medicine*, 3, 233-267.

Sussman, H., Marquardt, T., Hutchinson, J., & MacNeilage, P. (1986). Compensatory articulation in Broca's aphasia. *Brain and Language*, 27, 56-74.

Tuller, B. (1984). On categorizing aphasic speech errors. *Neuropsychologia*, 22, 547-557.

Tye, N., Zimmerman, G.N., & Kelso, J.A.S. (1983). "Compensatory articulation" in hearing impaired speakers: a cinefluorographic study. *Haskins Laboratories Status Report on Speech Research*, SR-73.

Appendix 1. Mean F1 and F2 produced by normal subjects in normal and biteblock productions of [u]

Subject	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
N1	323	344	328	316	341	322	970	1034	1030	924	925	799
N2	377	372	373	372	377	377	886			850	1461	875
N3	356	367	370	376	384	349	933	925	948	846	868	866
N4	267	289	303	345	345	356	1156	1044	936	898	944	918
N5	400	329	394	391	407	428	961	930	1167	960	961	974
N6	333	318	328	328	321	315	846	762		797	803	754
N7	359	351	373	316	343	363	910	910	1051	792	907	913
N8	340	380	343	315	326	325	904	952	908	839	1057	824
N9	347	340	345	306	292	322	911	960	852	827	813	801
N10	342	394	334	336	391	413	958	1222	1264	892	1131	1213
$\bar{x}$	344.4	348.4	349.1	340.1	352.7	357.0	943.5	971.0	1019.5	862.5	987.0	893.7
σ	35.1	31.5	27.7	29.9	36.0	39.3	83.7	124.5	139.0	54.7	194.8	130.1

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 2. Mean F1 and F2 produced by normal subjects in normal and biteblock productions of [i]

Subject	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
N1	331	330	334	306	313	344		2647	2541	2606	2610	2512
N2	376	383	397	348	348	357	2595	2494	2427	2581	2539	2516
N3	367	363	317	334	324	360	2511	2442	2341	2606	2426	2533
N4	305	276	351	337	345	402	2560	2381	1980	2627	2579	2077
N5	404	426	323	380	407	372	2583	2581	2305	2555	1821	2232
N6	308	347	330	316	336	335	2305	2225	2021	2471	2444	2357
N7	297	323	401	299	326	356	2200	2242	2158	2355	2299	2173
N8	297	336	337	305	314	313	2510	2425	2434	2385	2291	2341
N9	347	390	384	329	358	382	2538	2425	2323	2582	2566	2395
N10	320	315	372	281	305	369	2213	2184	1915	2280	2300	1989
$\bar{X}$	335.2	348.9	354.6	323.5	337.6	359.0	2446.1	2404.6	2244.5	2504.8	2387.5	2312.5
σ	37.1	43.0	31.4	28.3	29.7	24.9	160.2	152.0	214.0	123.9	233.7	190.2

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 3. Mean F1 and F2 produced by normal subjects in normal and biteblock productions of [æ]

Subject	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
N1	881	916	816	929	875	861	1740	1763	1815	1747	1671	1700
N2	938	900	820	899	769	794	1781	1587	1498	1742	1569	1533
N3	685	833	703	691	867	724	1546	1477	1649	1516	1523	1584
N4	747	935	708	726	915	749	1859	1741	1575	1806	1628	1653
N5	781	853	837	737	855	800	1800	1734	1771	1797	1707	1724
N6	847	868	778	854	886	783	1687	1588	1560	1584	1554	1478
N7	752	708	632	688	707	630	1669	1560	1646	1597	1617	1664
N8	765	764	688	731	748	660	1794	1682	1748	1711	1695	1609
N9	797	829	691	782	803	715	1579	1551	1538	1501	1525	1492
N10	738	745	676	786	778	738	1527	1531	1558	1490	1499	1516
$\bar{X}$	793.1	835.1	734.9	782.3	820.3	745.4	1698.2	1621.4	1635.8	1649.1	1598.8	1595.3
σ	75.4	75.7	71.5	85.2	68.7	68.5	116.3	100.4	109.2	124.9	75.6	88.5

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 4. Mean F1 and F2 produced by normal subjects for normal and biteblock productions of [a]

Subject	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
N1	824	940	853	844	741	828	1015	1238	1306	1105	1029	1164
N2	828	780	745	750	728	703	1238	1175	1303	1134	1122	1167
N3	658	682	617	632	668	625	1136	1141	1141	1162	1158	1151
N4	784	875	672	734	799	700	1173	1106	1036	1128	1081	1121
N5	738	732	742	670	683	698	1267	1331	1326	1330	1317	1310
N6	717	704	648	712	671	729	1060	1052	1003	1089	1034	1077
N7	637	663	632	619	627	621	1005	1109	1106	1006	992	1072
N8	718	779	710	747	777	710	977	1218	1221	1024	1192	925
N9	635	694	650	640	706	616	1057	1095	1011	1023	1062	995
N10	701	657	665	711	694	759	1053	1066	1106	995	1078	1095
$\bar{X}$	724	750.6	693.4	705.9	709.4	698.9	1098.1	1153.1	1155.9	1099.6	1106.5	1107.7
σ	70.8	94.2	71.2	68.5	52.6	66.4	100.3	87.6	125.3	100.2	95.6	104.4

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 5. Mean F1 and F2 produced by nonfluent aphasics in normal and biteblock productions of [u]

Subjects	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
NF1	367	463	474	274	398	453	952	1222	1284	876	1117	1217
NF2	401	441	400	386	368	382	958	939	873	836	935	803
NF3	426	416	430	367	373	430	1412	1413	1374	1185	1314	1230
NF4	395	393	392	391	383	392	870	894	892	841	809	904
NF5	265	330	357	313	379	376	1384	1444*	1265	1284	1444	1135
NF6	346	372	432	295	337	401	979	1424	1337	929	1131	1237
$\bar{x}$	366.7	402.5	414.2	337.7	373.0	405.7	1092.5	1222.7	1170.8	991.8	1125.0	1087.7
σ	57.0	48.2	40.3	50.0	20.4	29.9	239.7	250.7	226.7	193.4	233.9	187.8

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 6. Mean F1 and F2 produced by nonfluent aphasics in normal and biteblock productions of [i]

Subjects	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
NF1	298	336	408	274	312	353	2227	2297	2183	2317	2364	2281
NF2	365	397	391	358	362	431	2478	2481	2051	2533	2536	2354
NF3	373	437	415	351	367	369	2561	2394	2420	2448	2491	2342
NF4	363	345	398	350	352	353	2213	2577*	2372	2213*	2577	2373
NF5	302	281	412	410	394	432	1885	2472	2216	2485	2404	2533
NF6	327	343	354	296	318	352	2337	2282	2294	2426	2393	2253
$\bar{X}$	338.0	356.5	396.3	339.8	350.8	381.7	2283.5	2417.2	2256.0	2403.7	2460.8	2356.0
σ	33.5	54.0	22.6	48.4	31.1	39.1	238.6	114.8	134.7	118.0	86.3	98.0

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 7. Mean F1 and F2 produced by nonfluent aphasics in normal and biteblock productions of [æ]

Subject	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
NF1	756	833	778	817	863	757	1536	1505	1488	1539	1475	1500
NF2	884	913	865	893	909	899	1734	1424	1664	1715	1426	1651
NF3	850	943	682	855	915	707	1941	1898	1951	1856	1823	1876
NF4	858		902	921		913	1728	1773	1771	1694	1679	1716
NF5	808	784	804	854	767	819	1740	1597	1773	1750	1614	1664
NF6	738	770	682	813	822	775	1830	1722	1677	1738	1665	1511
$\bar{X}$	815.7	848.6	785.5	858.8	855.2	811.7	1751.5	1653.2	1720.7	1715.3	1613.7	1653
σ	58.8	76.9	91.3	42.2	62.1	81.5	133.8	177.0	153.3	103.0	145.0	139.6

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 8. Mean F1 and F2 produced by nonfluent aphasics in normal and biteblock productions of [a]

Subject	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
NF1	737	825	793	717	796	744	1065	1213	1276	960	1147	1263
NF2	825	845	867	865	904	895	1308	1321	1315	1261	1301	1315
NF3	803	810	746	761	752	721	1218	1306	1273	1286	1246	1361
NF4	826	851	830	704	865	743	1179	1246	1216	1117	1185	1207
NF5	745	734	715	710	732	677	1149	1350	1223	1028	1237	1122
NF6	696	822	766	733	790	730	1037	1139	1034	949	1115	1007
$\bar{x}$	772.0	814.5	786.2	748.3	806.5	751.7	1159.3	1262.5	1222.8	1100.2	1205.2	1212.5
σ	53.7	42.3	55.8	60.7	66.1	74.4	99.9	78.7	99.5	147.2	69.0	130.8

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 9. Mean F1 and F2 produced by fluent aphasics in normal and biteblock productions of [u]

Subject	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
F1	345	399	433	346	377	389	1145	1291	1269	1189	1240	1201
F2	289	336	346	295	322	325	1003	804	1118	857	804	1118*
F3	385	379	405	365	374	299	913	1113	991	860	951	827
F4	356	330	400	304	323	357	1060	910	1051	930	851	987
F5	324	324	352	295	285	342	915	1050	1215	846	871	993
F6	476	480	491	395	449	471	1153	1169	1090	942	1028	1008
$\bar{x}$	362.5	374.7	404.5	333.3	355.0	363.8	1031.5	1056.2	1122.3	937.3	957.5	1022.3
σ	64.2	59.6	53.8	41.9	57.8	60.6	106.7	176.7	103.4	129.8	159.5	127.7

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 10. Mean F1 and F2 produced by fluent aphasics in normal and biteblock productions of [i]

Subject	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
F1	347	388	407	364	350	396	1802*	2402	2356	1802	2293	2233
F2	289	337	361	292	304	345	2509	2498	2296	2316	2332	2115
F3	355	368	352	322	314	366	2262	2232	2006	2341	2308	2060
F4	326	333	334	264	264	278	2581	2334	2319	1844	2620	2545
F5	319	294	331	265	278	320	2285	2338	2205	2607	2466	2281
F6	403	433	477	401	358	451	2549	2545	2285	2597	2593	2368
$\bar{X}$	339.8	358.8	377.0	318.0	311.3	359.3	2331.3	2391.5	2244.5	2251.2	2435.3	2267.0
σ	38.7	48.5	56.1	45.4	37.6	60.3	292.7	115.4	127.1	353.8	146.3	175.9

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 11. Mean F1 and F2 produced by fluent aphasics in normal and biteblock productions of [æ]

Subject	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
F1		825	794	730	828	748	1931	1758	1798	1861	1713	1778
F2	871	787	649	807	762	705	1538	1556	1566	1559	1600	1500
F3	718	788	682	774	789	673	1553	1544	1453	1435	1582	1461
F4	881	870	795	876	890	790	1571	1475	1538	1449	1462	1456
F5	805	785	696	843	778	718	1777	1659	1642	1699	1624	1634
F6	882	905	893	881	907	893	1488	1479	1501	1422	1517	1439
$\bar{x}$	831.4	826.7	751.5	818.5	825.7	754.5	1643	1578.5	1583.0	1570.8	1583	1544.7
σ	71.0	50.6	91.8	59.6	60.7	78.6	172.8	110.5	123.0	176.8	87.0	134.5

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 12. Mean F1 and F2 produced by fluent aphasics in normal and biteblock productions of [a]

Subject	F1						F2					
	ONSET			MIDPOINT			ONSET			MIDPOINT		
	N	NC	C	N	NC	C	N	NC	C	N	NC	C
F1	781	721	773	837	734	722	1149	1389	1228	1190	1226	1185
F2	755	676	648	682	636	640	1088	1184	1226	1058	1050	1044
F3	724	826	678	735	750	682	1205	1197	1189	1094	1103	1108
F4	780	751	708	734	758	729	1087	1114	1165	1030	1075	1122
F5	748	742	676	798	783	690	1396	1340	1379	1336	1336	1332
F6	876	865	873	843	786	838	1296	1348	1322	1290	1184	1272
$\bar{X}$	777.3	763.5	726	771.5	741.2	716.8	1203.5	1262.0	1251.5	1166.3	1162.3	1177.2
σ	52.8	69.7	83.7	64.6	55.2	67.4	122.9	111.2	82.2	126.6	108.1	108.2

Normal (N), Noncompensatory (NC), Compensatory (C)

Appendix 13. Individual mean differences in F1 according to difference limens exceeded for normal subjects *

		[u]		[i]		[æ]		[a]	
Subject	Mm point	NC-N	C-N	NC-N	C-N	NC-N	C-N	NC-N	C-N
N1	onset	21 (5)	5	-1	3	35 (3)	-65 (5)	116 (5)	29 (3)
	midpoint	25 (5)	6	7	38 (5)	-54 (5)	-68 (5)	-103 (5)	-16
N2	onset	-5	-4	7	21 (5)	-38 (3)	-118 (5)	-48 (5)	-83 (5)
	midpoint	5	5	0	9	-130 (5)	-105 (5)	-22 (3)	-47 (5)
N3	onset	11 (3)	14 (3)	-4	-50 (5)	148 (5)	18	24 (3)	-41 (5)
	midpoint	8	-27 (5)	-10 (3)	26 (5)	176 (5)	33 (3)	36 (5)	-7
N4	onset	22 (5)	36 (5)	-29 (5)	46 (5)	188 (5)	-39 (5)	91 (5)	-112 (5)
	midpoint	0	11 (3)	8	65 (5)	189 (5)	23	65 (5)	-34 (3)
N5	onset	-71 (5)	-6	22 (5)	-81 (5)	72 (5)	56 (5)	-6	4
	midpoint	16 (3)	37 (5)	27 (5)	-8	118 (5)	63 (5)	13	28 (3)
N6	onset	-15 (3)	-5	39 (5)	22 (5)	21	-69 (5)	-13	-69 (5)
	midpoint	-7	-13 (3)	20 (5)	19 (5)	32 (3)	-71 (5)	-41 (5)	17
N7	onset	-8	14 (3)	26 (5)	104 (5)	-44 (5)	-120 (5)	26 (3)	-5
	midpoint	27 (5)	47 (5)	27 (5)	57 (5)	19	-58 (5)	8	2
N8	onset	40 (5)	3	39 (5)	40 (5)	-1	-77 (5)	61 (5)	-8
	midpoint	11 (3)	10 (3)	9	8	17	-71 (5)	30 (3)	-37 (5)
N9	onset	-7	-2	43 (5)	37 (5)	32 (3)	-106 (5)	59 (5)	15
	midpoint	-14 (3)	16 (3)	29 (5)	53 (5)	21	-67 (5)	66 (5)	-24 (3)
N10	onset	52 (5)	-8	-5	52 (5)	7	-62 (5)	-44 (5)	-36 (5)
	midpoint	55 (5)	77 (5)	24 (5)	88 (5)	-8	-48 (5)	-17	48 (5)

Measurement Point (Mm point), Compensatory (C), Noncompensatory (NC), Normal (N)

Appendix 14. Individual mean differences in F2 according to difference limens exceeded for normal subjects *

		[u]		[i]		[æ]		[a]	
Subject	Mm point	NC-N	C-N	NC-N	C-N	NC-N	C-N	NC-N	C-N
N1	onset	64 (5)	60 (5)	-	-	23	75 (3)	223 (5)	291 (5)
	midpoint	1	-125 (5)	4	-94 (3)	-76 (3)	-47	-76 (5)	59 (5)
N2	onset	-	-	-101 (3)	-168 (5)	-194 (5)	-283 (5)	-63 (5)	65 (5)
	midpoint	611 (5)	25	-42	-65	-173 (5)	-209 (5)	-12	33 (3)
N3	onset	-8	15	-69	-170 (5)	-69 (3)	103 (5)	5	5
	midpoint	22	20	-180 (5)	-73	7	68 (3)	-4	-11
N4	onset	-112 (5)	-220 (5)	-179 (5)	-580 (5)	-118 (5)	-284 (5)	-67 (5)	-137 (5)
	midpoint	46 (5)	20	-48	-550 (5)	-178 (5)	-153 (5)	-47 (3)	-7
N5	onset	-31 (3)	206 (5)	-2	-278 (5)	-66 (3)	-29	64 (5)	59 (5)
	midpoint	1	14	-734 (5)	-323 (5)	-90 (5)	-73 (3)	-13	-20
N6	onset	-84 (5)	-	-80 (3)	-284 (5)	-99 (5)	-127 (5)	-8	-57 (5)
	midpoint	6	-43 (3)	-27	-114 (3)	-30	-106 (5)	-55 (5)	-12
N7	onset	0	141 (5)	42	-42	-109 (5)	-23	104 (5)	101 (5)
	midpoint	115 (5)	121 (5)	-56	-182 (5)	20	67 (3)	-14	66 (5)
N8	onset	48 (5)	4	-85 (3)	-76 (3)	-112 (5)	-46	241 (5)	244 (5)
	midpoint	218 (5)	-15	-94 (3)	-44	-16	-102 (5)	168 (5)	-99 (5)
N9	onset	49 (5)	-59 (5)	-113 (5)	-215 (5)	-28	-41	38 (3)	-46 (3)
	midpoint	-14	-26	-16	-187 (5)	24	-9	39 (3)	-28
N10	onset	264 (5)	306 (5)	-29	-298 (5)	4	31	13	53 (3)
	midpoint	239 (5)	321 (5)	20	-291 (5)	9	26	83 (5)	100 (5)

Measurement Point (Mm point), Compensatory (C), Noncompensatory (NC), Normal (N)
* Difference limens exceeded are provided in parenthesis

Appendix 15. Individual mean differences in F1 according to difference limens exceeded for nonfluent aphasics *

		[u]		[i]		[æ]		[a]	
Subject	Mm point	NC-N	C-N	NC-N	C-N	NC-N	C-N	NC-N	C-N
NF1	onset	96 (5)	107 (5)	38 (5)	110 (5)	77 (5)	22	88 (5)	56 (5)
	midpoint	124 (5)	179 (5)	38 (5)	79 (5)	46 (5)	-60 (5)	79 (5)	27 (3)
NF2	onset	40 (5)	-1	32 (5)	26 (5)	29 (3)	-19	20	42 (5)
	midpoint	-18 (5)	-4	4	73 (5)	16	6	39 (5)	30 (3)
NF3	onset	-10	4	64 (5)	42 (5)	93 (5)	-168 (5)	7	-57 (5)
	midpoint	6	63 (5)	16 (3)	18 (5)	60 (5)	-148 (5)	-9	-40 (5)
NF4	onset	-2	-3	-18 (5)	35 (5)	-	44 (5)	25 (3)	4
	midpoint	-8	1	2	3	-	-8	161 (5)	39 (5)
NF5	onset	65 (5)	92 (5)	-21 (5)	110 (5)	-24	-4	-11	-30 (3)
	midpoint	66 (5)	63 (5)	-16 (3)	22 (5)	-87 (5)	-35 (3)	22	-33 (3)
NF6	onset	26 (5)	86 (5)	16 (3)	27 (5)	32 (3)	-56 (5)	126 (5)	70 (5)
	midpoint	42 (5)	106 (5)	22 (5)	56 (5)	9	-38 (3)	57 (5)	-3

Measurement Point (Mm point), Compensatory (C), Noncompensatory (NC), Normal (N)

* Difference limens exceeded are provided in parenthesis

Appendix 16. Individual mean differences in F2 according to difference limens exceeded for nonfluent aphasics *

		[u]		[i]		[æ]		[a]	
Subject	Mm point	NC-N	C-N	NC-N	C-N	NC-N	C-N	NC-N	C-N
NF1	onset	270 (5)	332 (5)	70	-44	-31	-48	148 (5)	211 (5)
	midpoint	241 (5)	341 (5)	47	-36	-64 (3)	-39	187 (5)	303 (5)
NF2	onset	-19	-85 (5)	3	-427 (5)	-31	-70 (3)	13	7
	midpoint	99 (5)	-33 (3)	3	-179 (5)	-289 (5)	-64 (3)	40 (3)	54 (3)
NF3	onset	1	-38 (3)	-167 (5)	-141 (5)	-43	10	88 (5)	55 (3)
	midpoint	129 (5)	45 (3)	43	-106 (3)	-33	20	-40 (3)	75 (5)
NF4	onset	24	22	-	159 (5)	45	43	67 (5)	37 (3)
	midpoint	-32 (3)	63 (5)	-	-	-15	22	68 (5)	90 (5)
NF5	onset	-	-119 (5)	587 (5)	331 (5)	-143 (5)	33	201 (5)	74 (5)
	midpoint	160 (5)	-149 (5)	-81 (3)	48	-136 (5)	-86 (3)	209 (5)	94 (5)
NF6	onset	445 (5)	358 (5)	-55	-43	-108 (5)	-153 (5)	102 (5)	-3
	midpoint	202 (5)	308 (5)	-33	-173 (5)	-73 (3)	-227 (5)	166 (5)	58 (5)

Measurement Point (Mm point), Compensatory (C), Noncompensatory (NC), Normal (N)

* Difference limens exceeded are provided in parenthesis

Appendix 17. Individual mean differences in F1 according to difference limens exceeded for fluent aphasics *

		[u]		[i]		[æ]		[a]	
Subject	Mm point	NC-N	C-N	NC-N	C-N	NC-N	C-N	NC-N	C-N
F1	onset	54 (5)	88 (5)	41 (5)	60 (5)	-	-	-60 (5)	-8
	midpoint	31 (5)	43 (5)	-14 (3)	32 (5)	98 (5)	18	-103 (5)	-115 (5)
F2	onset	47 (5)	57 (5)	48 (5)	72 (5)	-84 (5)	-222 (5)	-79 (5)	-107 (5)
	midpoint	27 (5)	30 (5)	12 (3)	53 (5)	-45 (5)	-102 (5)	-46 (5)	-42 (5)
F3	onset	-6	20 (5)	13 (3)	-3	70 (5)	-36 (3)	102 (5)	-46 (5)
	midpoint	9	-66 (5)	-8	44 (5)	15	-101 (5)	15	-53 (5)
F4	onset	-26 (5)	44 (5)	7	8	-11	-86 (5)	-29 (3)	-72 (5)
	midpoint	19 (5)	53 (5)	0	14 (3)	14	-86 (5)	24 (3)	-5
F5	onset	0	28 (5)	-25 (5)	12 (3)	-20	-109 (5)	-6	-72 (5)
	midpoint	-10 (3)	47 (5)	13 (3)	55 (5)	-65 (5)	-125 (5)	-15	-108 (5)
F6	onset	4	15 (3)	30 (5)	74 (5)	23	11	-11	-3
	midpoint	54 (5)	76 (5)	-43 (5)	50 (5)	26 (3)	12	-57 (5)	-5

Measurement Point (Mm point), Compensatory (C), Noncompensatory (NC), Normal (N)

* Difference limens exceeded are provided in parenthesis

Appendix 18. Individual mean differences in F2 according to difference limens exceeded for fluent aphasics *

		[u]		[i]		[æ]		[a]	
Subject	Mm point	NC-N	C-N	NC-N	C-N	NC-N	C-N	NC-N	C-N
F1	onset	146 (5)	124 (5)	-	-	-173 (5)	-133 (5)	240 (5)	79 (5)
	midpoint	51 (5)	12	491 (5)	431 (5)	-148 (5)	-83 (5)	36 (3)	-5
F2	onset	-199 (5)	115 (5)	-11	-213 (5)	18	28	96 (5)	138 (5)
	midpoint	-53 (5)	-	16	-201 (5)	41	-59 (3)	-8	-14
F3	onset	200 (5)	78 (5)	-30	-256 (5)	-9	-100 (5)	-8	-16
	midpoint	91 (5)	-33 (3)	-33	-281 (5)	147 (5)	26	9	14
F4	onset	-150 (5)	-9	-247 (5)	-262 (5)	-96 (5)	-33	27	78 (5)
	midpoint	-79 (5)	57 (5)	775 (5)	701 (5)	13	7	45 (3)	92 (5)
F5	onset	135 (5)	300 (5)	53	-80 (3)	-118 (5)	-135 (5)	-56 (3)	-17
	midpoint	25	147 (5)	-141 (5)	-326 (5)	-75 (3)	-65 (3)	0	-4
F6	onset	16	-63 (5)	-4	-264 (5)	-9	13	52 (3)	26
	midpoint	86 (5)	66 (5)	-4	-229 (5)	95 (5)	17	-106 (5)	-18

Measurement Point (Mm point), Compensatory (C), Noncompensatory (NC), Normal (N)
* Difference limens exceeded are provided in parenthesis