Analysis of articulatory dynamics in dysarthria following brain injury in childhood using electromagnetic articulography and electropalatography

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Two physiological assessments, electromagnetic articulography (EMA) and electropalatography (EPG), were used simultaneously to investigate the articulatory dynamics in an 18-year-old male with dysarthria 9 years following traumatic brain injury (TBI). Eight words consisting of [TEXT NOT REPRODUCIBLE IN ASCII] in word initial and word final positions were produced up to 10 times. A nonneurologically impaired male served as a control subject. Six parameters were analyzed using EMA: velocity, acceleration, deceleration, distance, duration, and motion path of tongue movements. Using EPG, the pattern and amount of tongue-to-palate contact and the duration of the closure/constriction phase of each consonant produced were assessed. Timing disturbances in the TBI speaker's speech were highlighted in perceptual assessments in the form of prolonged phonemes and a reduced speech rate. EMA analysis revealed that the approach and release phase durations of the consonant productions were within normal limits. Kinematic strategies such as decreased velocity and decreased distances traveled by the tongue, however, may have counterbalanced each other to produce these appropriate results. EPG examination revealed significantly longer closure/constriction phase periods, which may have contributed to the prolonged phonemes and reduced speech rate observed. The implications of these findings for the development of treatment programs for dysarthria subsequent to TBI will be highlighted.

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Traumatic brain injury (TBI) is a leading cause of death and disability in the Western world (Sosin, Sniezek, & Thurman, 1996). Studies have indicated the incidence of TBI in children to be approximately 200 per 100,000 per year (Annegers, 1983; Cahill, Murdoch, & Theodoros, 2001). A range of disabilities can be seen in this population, particularly in those who have suffered from severe brain injuries. In a study carried out by Emanuelson, von Wendt, Lundalv, and Larsson (1996), it was found that almost all of the 25 children with severe TBI under investigation were left with some major impairments. Dysarthria is a commonly reported consequence of severe TBI in children (Costeff, Groswasser, & Goldstein, 1990). The term dysarthria refers to a group of neuromotor speech disorders caused by disturbances in the muscular control of the speech production mechanism as a result of lesions sustained within the central and/or peripheral nervous systems (Darley, Aronson, & Brown, 1975). A dysarthric speech disturbance not only can have significant negative effects on a child's ability to reintegrate into society and educational settings, but its effects often carry into adulthood. Little research, however, has been carried out on the long-term outcome of this disorder. The available reports are typically brief and rely on perceptual assessments or by self-report (e.g., Costeff, Groswasser, Landman, & Brenner, 1985; Costeff et al., 1990; Emanuelson et al., 1996). As knowledge of the mechanisms underlying a specific disorder is crucial for its treatment, advanced understanding of dysarthria following childhood TBI is required if more effective diagnostic and therapeutic techniques are to be developed (Theodoros, Shrapnel, & Murdoch, 1998).

In the past, perceptual analysis has been the preferred means of assessing dysarthric speech. Perceptual analysis has continued to be an important tool for speech assessment. However, the limitations of this approach are recognized and an extensive range of instrumentation has been developed to examine the underlying pathophysiology of the speech production mechanism. As stated by Hirose (1986), physiological analysis is a valuable complement to perceptual analysis for a number of reasons. Dysarthric speech is the end product of the motor characteristics of the articulators. Therefore, an examination of the motor patterns of the articulators appears to be a more direct and objective approach to assessing the nature and severity of dysarthric speech. In addition, speech is the result of several subsystems working together (i.e., articulation, velopharyngeal, laryngeal, and respiration), thus a deviant speech characteristic can be the result of impairment in any of these subsystems or a combination of them. The causal relationship is not always clear (Theodoros, Murdoch, & Chenery, 1994). Physiological, assessments can be used to measure each of these subsystems in isolation and to identify specific contributing physiological deficits.

Brink, Garrett, Hale, Woo-Sam, and Nickel (1970) observed impaired articulation to be common in the speech of a group of children with TBI. Theodoros et al. (1998) also found articulatory disturbances to be some of the most impaired aspects of their TBI child's speech production. Despite this finding, little research has been carried out in the articulatory disturbances exhibited in this population. Considering the frequency of its occurrence and the significant impact it can have on an individual's life, improved understanding of the articulatory deficits following childhood TBI is necessary.

There is a range of instruments available designed to assess the different aspects of the articulatory components of speech. Theodoros, Murdoch, and Stokes (1995), Theodoros et al. (1998), and Cahill, Murdoch, and Theodoros (2000) have utilized a tongue pressure transduction system to examine the tongue function following TBI. They identified reduced tongue strength and endurance as likely contributing factors to the consonant imprecision and overall speech intelligibility perceived. Although pressure and force transduction systems examine the static and force-related aspects of articulatory functioning, imaging techniques allow researchers to directly investigate the dynamic features of speech production. Ultrasound examination and cineradiography are examples of imaging techniques. Both techniques have been useful in examining a number of articulatory parameters. However, they have their individual limitations. Ultrasound examination has been a valuable resource because it is noninvasive and inexpensive. It is limited, however, in that the tongue tip cannot be tracked. Cineradiography, in contrast, appears to be one of the most useful methods in the recording of articulatory movement. Unfortunately, the use of this technique is generally restricted as it exposes the patient to radiation (Theodoros, Murdoch, & Goozee, 2001).

A safe, nonradiological imaging technique called electromagnetic articulography (EMA) has been developed that overcomes some of the limitations of ultrasound and cineradiography. This technique uses alternating electromagnetic fields to track articulatory movements during speech. Murdoch and Goozee (2003) used this technique to explore the articulatory dynamics of four children with dysarthria following TBI. A number of deviant speech characteristics were exhibited by the children in this study, including decreased speech rate, prolonged word durations, and distorted consonants. EMA analysis was used to help explain the nature of these perceptual characteristics. In the analysis of the data, there was difficulty in perceptually deciding whether the word-initial consonants and/or the following vowel were prolonged, thus resulting in the perception of prolonged word durations. Through EMA analysis, it was revealed that for two of the children with TBI, the consonant approach phase durations were significantly longer than the control group's durations. For one child, this appeared to be the result of an impairment in generating speed with her tongue, resulting in slow movements up to the palate. For the other child, increased distances traveled by the tongue appeared to be the main factor underlying the longer consonant durations. These findings would not have been possible without the use of EMA.

Further investigation of Murdoch and Goozee's (2003) data led to the speculation that the distorted consonants observed in these TBI children's productions were the result of disturbances in tongue placement and/or the amount of tongue contact against the hard palate. However, EMA could not provide evidence to feasibly support this supposition, because its primary function is to track and record movements of the tongue in the mid-sagittal plane. The researchers acknowledged the limitations of EMA and suggested the use of another imaging technique, electropalatography (EPG), to provide complementary information to EMA regarding the location and amount of tongue contact against the palate. EPG utilizes an artificial acrylic palate to detect tongue-to-palate contact during continuous speech in real time. Murdoch and Goozee (2003) emphasized that only through the simultaneous use of these two techniques could a complete picture of the underlying mechanisms of the lingual consonant distortions exhibited by the children with TBI be made. The simultaneous use of EMA and EPG in the healthy adult population has been reported by a number of researchers (e.g., Ellis & Hardcastle, 2002; Fitzpatrick & Ni Chasaide, 2002; Hoole, Nguyen-Trong, and Hardcastle, 1993).

This present study investigates, through the simultaneous use of EMA and EPG, the articulatory dynamics of an adult with dysarthria following severe TBI sustained in childhood. It was hypothesized that the speaker in the study would exhibit aberrant articulatory dynamics underlying his disordered speech. The complemented use of EMA and EPG was expected to provide a greater understanding of the mechanisms that underlie dysarthria of childhood TBI.

METHOD

Speakers

One male speaker with mild dysarthria subsequent to a severe TBI (Glasgow Coma Score = 3) participated in the study. The speaker was 18.2 years of age and sustained a severe TBI 9 years, 3 months prior to the EMA and EPG assessment. A computed tomography (CT) scan taken immediately after the accident revealed diffuse axonal injury with no midline shift or cerebral edema and no basal skull fracture. Further investigation using electroencephalography (EEG) in the same year of the accident indicated cortical dysfunction over the right posterior temporal region. Areas of porencephaly (the occurrence of cavities in the brain substance) in both frontal lobes were further indicated in a CT scan conducted 3 years after the accident. The most recent EEG investigation, conducted 7 years after the accident, described abnormal EEG patterns arising predominantly from the frontal region. Areas of cortical dysfunction that were potentially epileptogenic were recorded. At the time of the EMA and EPG assessment, no physical disability or limb weakness was apparent. Medication (Tegretol), which was reported by the speaker's mother to cause fatigue in the speaker, was being taken to control epilepsy. The TBI speaker reported that with increased fatigue, his motivation for speech may be affected. The TBI speaker had a negative history of speech difficulty prior to the TBI, and no lip, tongue, or jaw surgery had been undertaken.

Four perceptual assessments were conducted at the time of the EMA and EPG assessments. The results were analyzed independently by two qualified speech-language pathologists and then combined. These results revealed a slow speech rate (97 words per minute) as a prominent deviant characteristic in the TBI speaker's speech (Assessment of Intelligible Dysarthric Speech [ASSIDS], Yorkston & Beukleman, 1981). This moderate impairment in speech rate was supported by ratings from a speech sample analysis (Darley et al., 1975; FitzGerald, Murdoch, & Chenery, 1987) and nonspeech lingual movements that were judged to be slow and laborious (Frenchay Dysarthria Assessment, Enderby, 1983). Mild impairments in speech intelligibility at the word (intelligibility level at 77.55%) and sentence (intelligibility level at 97.13%) were also evident in the ASSIDS. A word intelligibility level of 95.65% was reported by the Multiple Word Intelligibility Test (Kent, Weismer, Kent, & Rosenbek, 1989). Mild impairments in phoneme length (i.e., prolongation) were a prominent deviant speech feature (Darley et al., 1975; FitzGerald et al., 1987).

A male 19.1 years of age with normal speech and neurological development served as a control subject for the study. He presented with perceptually normal speech as judged by a qualified speech-language pathologist and had a negative history of lip, tongue, or jaw surgery. Both the TBI speaker and the control speaker were native Australian English speakers.

This present study was given ethical clearance by the School of Health and Rehabilitation Sciences Ethics Committee at The University of Queensland.

Procedure

The EMA and EPG assessments were conducted simultaneously in an electrically shielded, quiet room. The speakers were each seated in a straight-backed chair.

EPG Assessment

The Reading Electropalatograph (EPG3) system was used to record details of the timing and location of tongue-to-palate contacts during production of a list of target words (see Test Material section). Each speaker was fitted with his own artificial palate made of acrylic, approximately 1 mm thick. The palate was moulded to fit the upper palate and teeth and contained 62 silver touch-sensitive electrodes exposed to the lingual surface. These electrodes were arranged in eight rows and eight columns according to a predetermined scheme based on anatomical landmarks (Hardcastle, Gibbon, & Jones, 1991) to ensure that adequate coverage of the entire palate was achieved and that consistency between the two speakers was maintained. Changes in tongue-to-palate contacts were sampled at 10 ms time intervals (sampling rate 100 Hz). Acoustic data also were recorded by the EPG system, at a sampling rate of 10 kHz.

Prior to the recording of the data, a 45-minute period was provided to allow the speaker to become accustomed to speaking with the EPG palate in his mouth. During this period, the speaker freely conversed with the researchers, and the list of words to be used in the assessment was read aloud three times to ensure that intelligibility and speech rate would not be compromised by reading difficulties. A qualified speech-language pathologist, who was unrelated to the study, listened to the speaker's speech before the palate was in situ and then after the desensitization period. Subject preparation for the EMA assessment began when it was judged by the speech-language pathologist that no significant change in the speaker's speech could be detected as a result of wearing the EPG palate.

EMA Assessment

The commercially available Electromagnetic Articulography AG-100 system (Carstens Medizinelektronik GmbH, Germany) was used to track movements of the tongue along the midsagittal plane during production of the target words. Three transmitter coils were attached to a lightweight plastic helmet, which was positioned around the speaker's head. These transmitter coils generated alternating magnetic fields at different frequencies (range 10-20 kHz), which in turn induced alternating electrical signals in a set of five miniature receiver coils (~~2 X 2 X 3 mm in size). By determining the magnitude (or relative strength) of these signals, the distance between a single receiver coil and a transmitter coil could be calculated by the system and, in turn, the position of each receiver coil recorded. Two receiver coils were fixed along the midline of the speaker's tongue at distances of 1 cm and 4 cm from the tongue tip to capture movement of the tongue tip and the posterior portion of the tongue, respectively. Two other receiver coils were attached to the bridge of the nose and the maxilla above the upper central incisors to serve as fixed reference points to detect any displacement of the helmet. Small amounts of biologically safe adhesive (Cyano-Vaneer Fast, a cyanoethyl liquid) were used for attachment inside the mouth, and micropore tape was used for attachment on the nose. Each receiver coil was positioned with the axis perpendicular to the midsagittal plane, parallel with the axes of the transmitter coils. A pulley system was used to suspend the helmet from the ceiling to reduce the weight of the helmet and to minimize any possible negative effects on the helmet position due to extraneous movements of the head and shoulders.

The position of each of the receiver coils was sampled at 500 Hz. The initial EMA recordings included a trace of the occlusional plane, which served as a means of data validation. This trace was achieved with an unaffixed receiver coil attached to the top of a small custom-made plastic tracing stick, which

was in the form of a T-bar (see Murdoch & Goozee, 2003, for further details). A lapel microphone connected to the EMA system was used to record acoustic data at a sampling rate of 16 kHz during the assessment. Once the EMA preparation was completed, the EPG palate was returned in situ, and approximately 5 minutes was provided prior to recording of the test material for each speaker to become accustomed to the two receiver coils on the tongue and the EPG palate.

Test Material

Eight single syllable words of CV or CVC structure (where V = [TEXT NOT REPRODUCIBLE IN ASCII]; see Appendix), were read aloud 10 times in random order by each of the speakers. The target consonants chosen for analysis included the alveolar stop [TEXT NOT REPRODUCIBLE IN ASCII], the alveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII], the postalveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII], and the velar stop [TEXT NOT REPRODUCIBLE IN ASCII]. This selection of lingual consonants was chosen to exemplify differences in the place and manner of production. Each of the consonants occurred in the initial and then the final position of a word, in consideration of articulatory differences often found in syllable-initial and syllable-final consonants (Krakow, 1999; Manuel, 2002; Manuel & Vatikiotis-Bateson, 1998). The words were preceded by a neutral schwa and contained the low [TEXT NOT REPRODUCIBLE IN ASCII] vowel to facilitate expansive movements of the tongue to and from the hard palate. Any words where a minimum of three repetitions could not be achieved due to an error in reading (as verified by the researchers) or technical limitations of the assessment programs (e.g., the principal receiver coil becoming detached from the tongue, sampling malfunction for acoustic and/or EPG) were eliminated from the study. As a result, the postalveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII] in the word initial position was omitted from the study as it failed to satisfy this criteria in the case of the TBI speaker. All productions recorded were phonetically transcribed by two qualified speech-language pathologists who were unrelated to the study.

EPG Analysis Procedures

The recorded EPG data were loaded into an analysis program called EPGLAB, a dedicated EPG analysis graphical user interface written in the Matlab[TM] mathematical computing environment (Scott & Goozee, 2002). To compare the spatial and timing aspects of the tongue-to-palate contacts produced by the TBI speaker and the control speaker, the EPG data were first analyzed in the form of totals displays, whereby the total number of tongue-to-palate contacts within a particular region of the palate is plotted as a function of time (i.e., over consecutive frames; Hardcastle et al., 1991). For the anterior consonants, [TEXT NOT REPRODUCIBLE IN ASCII], the totals displays were generated from the number of contacted electrodes in the anterior region (i.e., anterior four rows of electrodes). For the velar stop, [TEXT NOT REPRODUCIBLE IN ASCII], the totals displays were computed from the sum of the contacted electrodes in the posterior four rows of electrodes.

In the analysis of the tongue-to-palate contact spatial parameters, the frame of maximum contact, which is an important articulatory feature in consonant productions (Gibbon, 1990), was selected. To aid comparison between the numerous frames of maximum contact generated for each target consonant (i.e., up to 10) produced by the TBI speaker and the control speaker, "representative" frames of maximum contact were generated. This was achieved by combining the total number of frames of maximum contact generated for each of the target consonants. The frequency with which each electrode was contacted in the production of each target consonant over the total number of repetitions recorded could be viewed as a percentage (see Figure 11 for examples). The end product was a diagram which closely resembled each of the frames from which it was composed. These diagrams created an overall picture of the tongue-to-palate contact pattern formed for the individual target consonants. Two spatial parameters were analyzed in this study: the mean number of tongue-to-palate contacts at the frame of maximum contact for each of the consonants analyzed; and the pattern of tongue-to-palate contacts, determined by visual inspection of the representative frames of maximum contact.

In the analysis of the tongue-to-palate timing parameters, only the closure/stable constriction phase duration of each consonant was analyzed, as the EMA assessment was expected to provide a comprehensive profile of the speakers' approach and release phases of the target consonants. Two annotation points were automatically identified by EPGLAB on the totals displays and were used to measure the duration of the closure/stable constriction phase.

Annotation Point 1. Onset of Closure (stops)/Stable Constriction (fricatives) was identified as the first frame that showed tongue-to-palate contact at the four central electrodes along any of the rows in the anterior region of the palate for the alveolar stop. In the case of the velar stop, the onset of closure occurred when the four central electrodes in either of the two most posterior rows were initially contacted. For the analysis of the fricatives, the onset of stable constriction was identified as the first frame that showed the minimum number of noncontacted electrodes (when counted from the palate's midsagittal line in both lateral directions) in any one of the rows in the anterior region of the palate.

Annotation Point 2. Onset of Release was identified as the final frame that showed closure along the palate in the case of stops. For fricatives, the onset of release of stable constriction was identified as the final frame that showed maximum constriction.

Two types of comparisons were carried out using the closure/stable constriction phase durations calculated for the target consonants. The mean closure/stable constriction phase duration of each target consonant was compared between the TBI speaker and the control speaker, and a comparison of the mean closure phase duration of the consonants in the word initial position and the word final position was also conducted within each speaker, given that the articulatory features of a consonant may be affected by its position in a word (Manuel, 2002).

EMA Analysis Procedures

The kinematic data recorded using the AG-100 EMA system were analyzed using the EMA analysis programs, Tailor and Emalyse (Carstens Medizinelektronik GmbH, Germany). Before the kinematic data analysis commenced, data validation procedures were carried out to modify the movement data to make it suitable for analysis. Initially, this involved downsampling the data from 500 Hz to 250 Hz and filtering the reference coils (Filter 40, cutoff = 10 Hz). A dynamic position correction procedure was then performed to correct for any movements of the head in relation to the helmet. Finally, the movement data were rotated within the x-y plane to ensure that the occlusional plane was parallel with the x-axis so that consistency between the two speakers was maintained.

The kinematic parameters chosen to be analyzed for the approach phase (i.e., tongue movement up to the palate) and release phase (i.e., tongue movement away from the palate) of the target consonants [TEXT NOT REPRODUCIBLE IN ASCII] included:

1. maximum velocity (mm/s)

2. maximum acceleration and deceleration (m/[s.sup.2]) calculated for the principal receiver coil (i.e., the receiver coil positioned on the section of the tongue presumed to be responsible for closure/stable constriction along the palate),

3. distance (mm) traveled by the principal receiver coil, and

4. duration (ms) of the approach and release phases.

These values were averaged over the total number of repetitions recorded for each target consonant to produce a representative value for the various parameters. In addition, the movement trajectories or motion paths of the tongue receiver coils recorded in the midsagittal plane during each consonant production were visually inspected.

These parameters were investigated for the target consonants produced in the word initial and word final position with the exception of two circumstances. First, maximum deceleration for the principal receiver coil was not recorded for the release phase. This parameter was eliminated, as deceleration of the tongue in the release phase was considered to be related more to the positioning and stabilization of the tongue for the production of the vowel immediately following the consonant rather than the consonant itself (Kuehn & Moll, 1976). Second, the release phase of the consonants in the word final position was disregarded, as the precise tongue position at which the consonants ended was difficult to determine without a following vowel to constrain its movement.

RESULTS

EMA Results

In the next section, the means obtained by the TBI speaker and the control speaker for the kinematic parameters and the movement trajectories analyzed for the individual consonants, [TEXT NOT REPRODUCIBLE IN ASCII], are contrasted. These consonants were investigated in the word initial and word final positions, with the exception of the word initial [TEXT NOT REPRODUCIBLE IN ASCII] for which less than three productions were recorded for the TBI speaker.

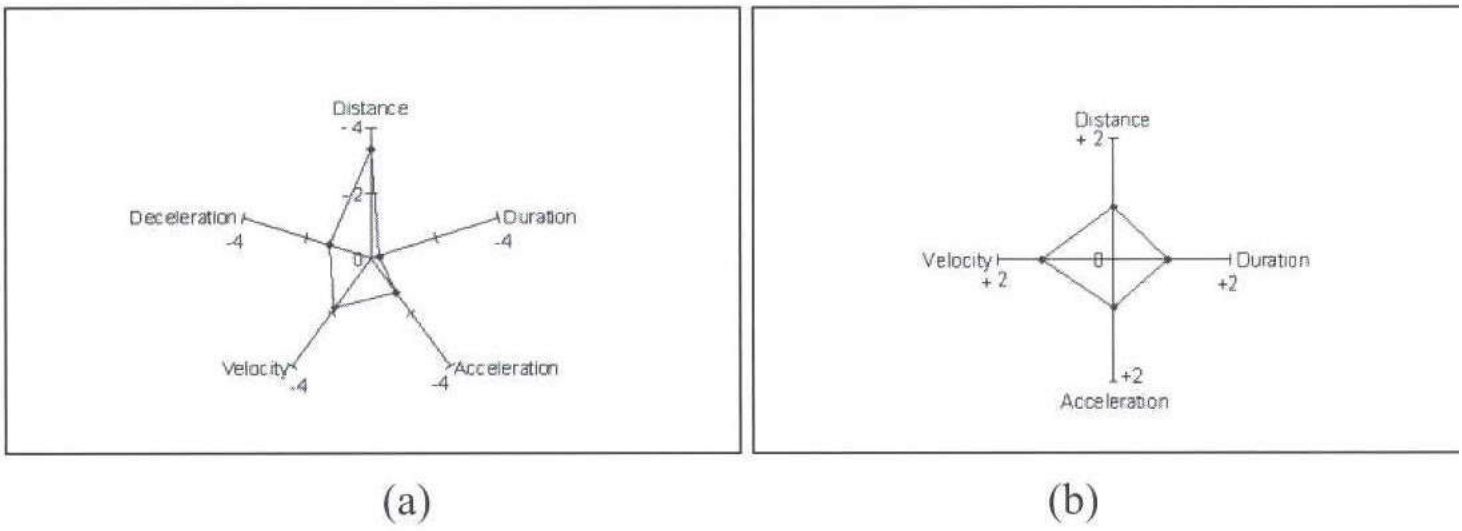


Figure 1. Difference (in standard deviations) in mean kinematic parameter values calculated f the TBI speaker and control speaker during the (a) approach and (b) release phase of /t/ in word i tial position. Negative values indicate smaller values being achieved by the TBI speaker compar to the control speaker.

Quantitative Kinematic Data

During the approach phase of the alveolar stop [TEXT NOT REPRODUCIBLE IN ASCII] production in the word initial position, the TBI speaker's tongue tip (i.e., principal receiver coil) reached maximum velocity, mean maximum acceleration, and deceleration values that were consistent with the mean values reached by the control speaker (i.e., within 2 standard deviations [SD] of the control speaker's mean; Figure 1a). Although the duration of the approach phase for the TBI speaker remained consistent with the control speaker, the distance traveled by the TBI speaker's tongue tip was 3.32 SD below that of the control speaker. In the release phase of the alveolar stop [TEXT NOT REPRODUCIBLE IN ASCII] production in the word initial position, the TBI speaker achieved mean maximum velocity and acceleration values that were within 2 SD of the control speaker's mean values (Figure 1b). The mean distance traveled by the TBI speaker's tongue and the mean length of time taken to travel this distance were also within 2 SD of the control speaker's mean values.

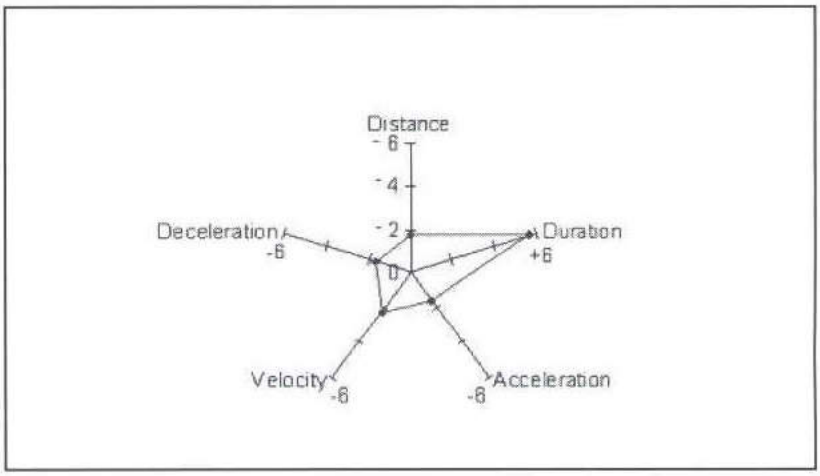


Figure 2. Difference (in standard deviations) in mean kinematic parameter values calculated for the TBI speaker and control speaker during the approach phase of /t/ in word final position. Negative values indicate smaller values being achieved by the TBI speaker compared to the control speaker.

During the approach phase of the alveolar stop [TEXT NOT REPRODUCIBLE IN ASCII] production in the word final position, the TBI speaker's tongue tip did not reach as high a mean maximum velocity as the control speaker (Figure 2). Rather, his mean maximum velocity was calculated to be 2.2 SD below the control speaker's mean value. The mean maximum acceleration and deceleration values, however, were calculated to be consistent with the control speaker's mean acceleration and deceleration values. Although the mean distance traveled by the TBI speaker's tongue tip was within 2 SD of the control speaker, the duration of the approach phase was 5.7 SD greater than that recorded for the control speaker.

During the approach phase of the alveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII] produced in the word initial position, the TBI speaker's tongue tip traveled at a velocity that was 2.03 SD slower than the control speaker (Figure 3a). However, the mean maximum acceleration and deceleration values were calculated to be consistent with the control speaker's mean values. Although the mean duration of the TBI speaker's approach phase was also within 2 SD of the control speaker's mean duration, the distance traveled by the TBI speaker's tongue tip in this length of time was calculated to be 2.24 SD less than the control speaker. The TBI speaker's alveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII] release phase was characterized by mean maximum velocity and acceleration values that were 2.05 and 2.03 SD below the control speaker's mean values, respectively (Figure 3b). The mean distance traveled by the TBI speaker's tongue tip and the time taken to travel this distance, however, was shown to be within 2 SD of the control speaker.

In the approach phase of the alveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII] produced in the word final position, the TBI speaker reached a mean maximum velocity that was 3.69 SD below the control speaker's mean maximum velocity value (Figure 4). While the mean acceleration value recorded for the TBI speaker was consistent with the control speaker, a slower deceleration (-2.39 SD) rate was demonstrated. The mean distance traveled by the TBI speaker's tongue was 4.95 SD less than the mean distance recorded for the control speaker. His mean approach phase duration was consistent, however, with the control speaker.

For the postalveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII], due to the small sample size that was recorded for this consonant in the word initial position, the consonant in the word final position only was analyzed. The TBI speaker's postalveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII] approach phase was characterized by a mean maximum velocity that was 2.38 SD below the control speaker's mean value, and mean maximum acceleration and deceleration values that were within 2 SD of the control speaker's mean values (Figure 5). The mean distance traveled by the TBI speaker's tongue tip was 3.53 SD less than the control speaker's. The duration of the approach phase, however, was shown to be consistent with the control speaker's mean duration.

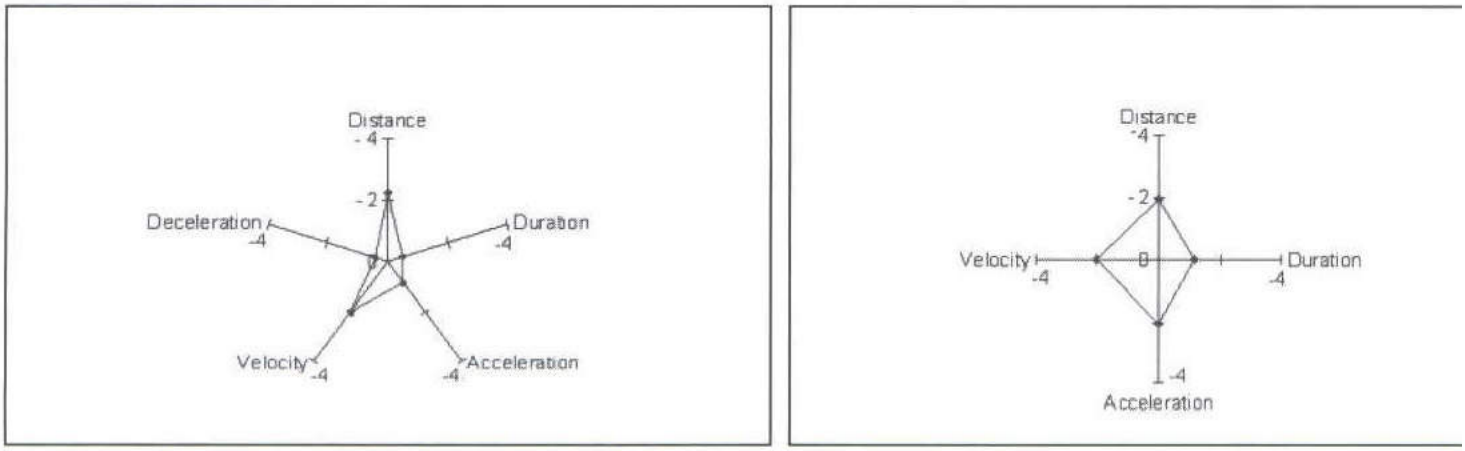


Figure 3. Difference (in standard deviations) in mean kinematic parameter values calculated the TBI speaker and control speaker during the (a) approach and (b) release phase of /s/ in word tial position. Negative values indicate smaller values being achieved by the TBI speaker compa to the control speaker.

During the approach phase of the velar stop [TEXT NOT REPRODUCIBLE IN ASCII] production in the word initial position, the back portion of the TBI speaker's tongue (i.e., principal receiver coil at 4 cm from the tongue tip) reached a slower mean maximum velocity (-2.97 SD) than the back portion of the control speaker's tongue (Figure 6a). His mean maximum acceleration and deceleration values were, however, consistent with the control speaker. The distance traveled by the TBI speaker's tongue back was, on average, 3.58 SD shorter than the control speaker's mean distance, while his mean approach phase duration was 2.41 SD greater than the control speaker's mean duration. In the release phase of the velar stop [TEXT NOT REPRODUCIBLE IN ASCII] production, the TBI speaker exhibited a mean maximum velocity value that was 2.75 SD below the control speaker's mean, while his mean maximum acceleration value was within 2 SD of the control speaker's mean (Figure 6b). The mean distance traveled by the back portion of the TBI speaker's tongue was calculated to be 2.44 SD shorter than that achieved by the control speaker's tongue. The release phase duration obtained by the TBI speaker, however, was consistent with the control speaker's mean duration.

The TBI speaker's velar stop [TEXT NOT REPRODUCIBLE IN ASCII] approach phase in the word final position was characterized by mean maximum velocity, acceleration, and deceleration values that were within 2 SD of the control speaker's means (Figure 7). The mean distance traveled by the TBI speaker's tongue was 2.23 SD below the mean distance value of the control speaker. The mean duration value, however, was consistent with the mean duration value calculated for the control speaker.

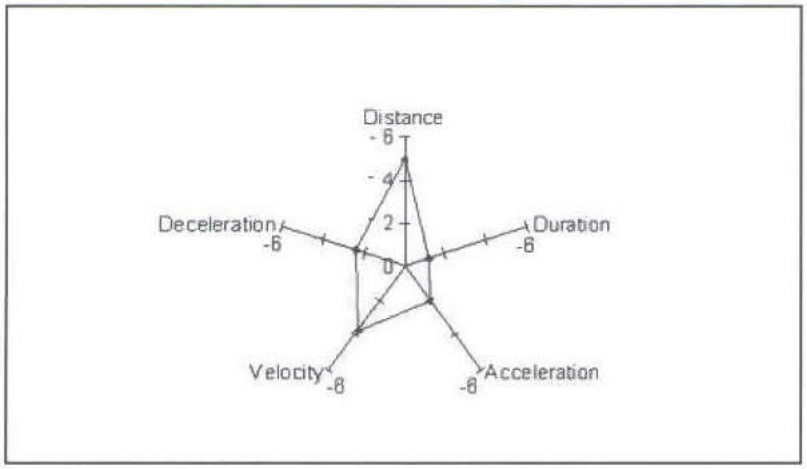


Figure 4. Difference (in standard deviations) in mean kinematic parameter values calculated for the TBI speaker and control speaker during the approach phase of /s/ in word final position. Negative values indicate smaller values being achieved by the TBI speaker compared to the control speaker.

In summary, while some of the TBI speaker's kinematic characteristics exhibited during production of the individual consonants, [TEXT NOT REPRODUCIBLE IN ASCII], appeared to be consistent with those of the control speaker, some discrepancies were noted. The parameters, mean maximum acceleration and deceleration, and mean approach and release phase duration, demonstrated few significant differences between the TBI speaker and the control speaker across the target consonants; the only notable feature was a trend of marginally reduced acceleration and deceleration values. In contrast, the parameters, mean maximum velocity and mean distance traveled, yielded numerous significantly different values between the two speakers; the TBI speaker typically achieved values that were significantly smaller than the control speaker's values.

Movement Trajectories

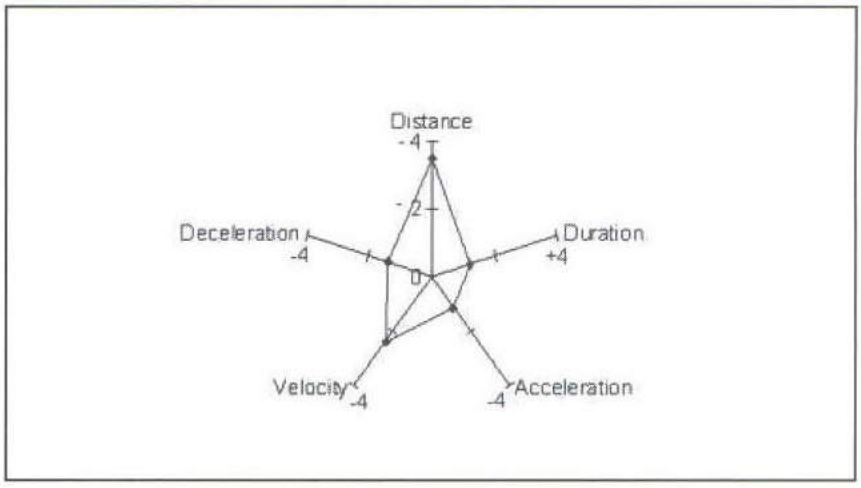


Figure 5. Difference (in standard deviations) in mean kinematic parameter values calculated for the TBI speaker and control speaker during the approach phase of /ʃ/ in word final position. Negative values indicate smaller values being achieved by the TBI speaker compared to the control speaker.

A number of differences between the movement trajectories exhibited by the TBI speaker and the control speaker were identified through visual inspection. In the production of the alveolar stop [TEXT NOT REPRODUCIBLE IN ASCII] in both word initial and word final positions, the general trend observed for the control speaker was movement of the tongue tip/blade receiver coil (i.e., principal receiver coil) up to the palate, followed by movement across or near the palate as indicated by abrupt changes in the direction of movement (see Figure 8a). For 10% of the control speaker's productions in the word initial position, however, excess lingual movement, as identified by suggested atypical movement of the tongue, at or near the palate was apparent (Figure 8b). For the TBI speaker's productions, the same general trend of the control speaker was evident. Excess movement at or near the palate was seen in 29% of his productions in the word initial position. One notable difference between the movement trajectories of the two speakers for both word initial and word final positions was the steeper rise of the control speaker's tongue tip/blade in the approach up to the palate, suggesting that the TBI speaker's tongue position prior to consonant production was considerably higher than that of the control speaker's.

The control speaker's production of the alveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII] in the word initial and word final position was characterized by a steep and direct rise of his tongue tip/blade up to the palate (Figure 9a). The slope of the TBI speaker's tongue tip/blade movement to the palate was, in comparison, notably more gradual than that of the control speaker's. Movement at or near the palate was observed for both speakers' movement trajectories, with that of the TBI speaker noticeably more extensive than that of the control speaker (Figure 9b). Consequently, the TBI speaker's tongue tip/blade position in the production of the alveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII], again, appeared to be significantly higher than that of the control speaker's.

For the postalveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII], the word initial productions were again omitted in the analysis due to the small sample size recorded. In the word final position, the control speaker's and the TBI speaker's tongue tip/blade movement trajectories demonstrated direct movement of the tongue tip/blade up to the palate, which stayed at or near the palate immediately following this approach phase. One prominent difference identified between the two speakers' movement trajectories, however, was the significant difference in their steepness up to the palate; the control speaker's movement trajectories were noticeably steeper than that of the TBI speaker.

In the production of the velar stop [TEXT NOT REPRODUCIBLE IN ASCII] in the word initial and word final positions, the control speaker's and TBI speaker's movement trajectories of the back portion of the tongue (i.e., principal receiver coil at 4 cm from tongue tip) demonstrated close similarities. In the word initial position, direct movements of the tongue back up to the palate were typically exhibited, followed by a forward moving arch in the movement trajectory (Figure 10a). In the TBI speaker's productions, however, 13% of the repetitions demonstrated excess lingual movement at or near the palate (Figure 10b), and an additional 38% demonstrated lingual sliding across the palate that was more extensive than that typically observed in the control speaker's movement trajectories. As evident for the other target consonants, the slope of the control speaker's movement trajectories as the tongue back traveled up to the palate appeared to be larger than that of the TBI speaker's, again suggesting the TBI speaker to display a higher tongue position prior to approach phase in comparison to the control speaker. In the production of the velar stop [TEXT NOT REPRODUCIBLE IN ASCII] in the word final position, both the control speaker's and TBI speaker's tongue back typically ascended up to the palate, at which it stayed momentarily, then descended away from the palate. For 22% of the control speaker's repetitions and 43% of the TBI speaker's productions, the tongue back was observed to stay at or close to the palate after the approach phase.

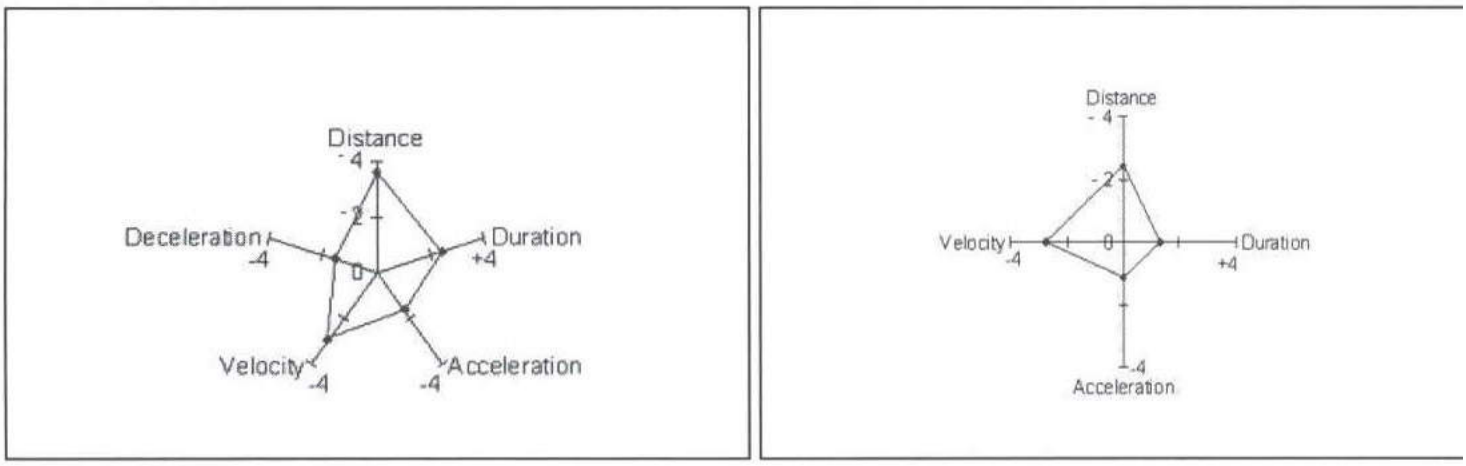


Figure 6. Difference (in standard deviations) in mean kinematic parameter values calculated the TBI speaker and control speaker during the (a) approach and (b) release phase of /k/ in word i tial position. Negative values indicate smaller values being achieved by the TBI speaker compa to the control speaker.

In summary, the control speaker's and the TBI speaker's movement trajectories in the production of the target consonants, [TEXT NOT REPRODUCIBLE IN ASCII], demonstrated similar trends of movement: ascending movement of the tongue to the palate followed by movement at or near the palate. Some discrepancies, however, were noted, including a higher tongue position displayed by the TBI speaker as reflected by a more gradual approach of the tongue to the palate, and more extensive lingual movements at or near the palate following the approach phase exhibited by the TBI speaker.

EPG Results

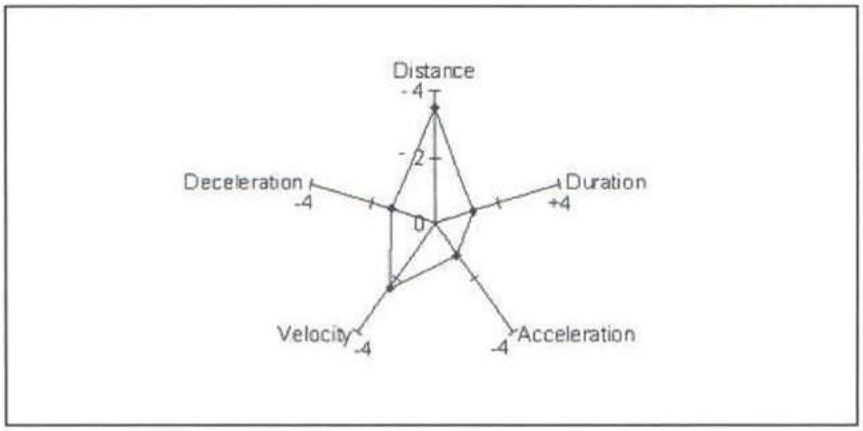


Figure 7. Difference (in standard deviations) in mean kinematic parameter values calculated for the TBI speaker and control speaker during the approach phase of /k/ in word final position. Negative values indicate smaller values being achieved by the TBI speaker compared to the control speaker.

In the next section, the spatial and timing parameters of the tongue-to-palate contacts produced by the TBI speaker and the control speaker for the individual consonants, [TEXT NOT REPRODUCIBLE IN ASCII], in the word initial and word final positions are described and contrasted.

Spatial Parameters

Tongue-to-Palate Contact Patterns. Visual inspection of the representative frames of maximum tongue-to-palate contact generated for the TBI speaker's and the control speaker's productions of the individual consonants, [TEXT NOT REPRODUCIBLE IN ASCII], in the word initial and word final positions revealed a general agreement between the two speaker's contact configurations, which in turn conformed with typical patterns (Hardcastle et al., 1991; Figure 11). In the production of the velar stop [TEXT NOT REPRODUCIBLE IN ASCII], however, lateral contact and closure were expected to be formed in the posterior region of the palate. Instead, while posterior lateral contact was clearly evident in the TBI speaker's and control speaker's [TEXT NOT REPRODUCIBLE IN ASCII] productions, closure was apparent only in a small sample of the control speaker's productions in the word initial position and not at all in the TBI speaker's productions. Other EPG studies have reported similar findings for normal speakers (Dagenais, Lorendo, & McCutcheon, 1994; Hardcastle, Morgan Barry, & Clark, 1987). Both Hardcastle et al. (1987) and Dagenais et al. (1994) proposed that contact may have occurred posterior to the EPG palate in cases of no EPG closure. This is a likely hypothesis for this present study, as the majority of the velar stop [TEXT NOT REPRODUCIBLE IN ASCII] productions recorded for both the TBI speaker and the control speaker were phonetically transcribed as acceptable productions (see Phonetic Transcriptions section).

In addition to velar stop [TEXT NOT REPRODUCIBLE IN ASCII] productions, 10% of the control speaker's tongue-to-palate contact diagrams were identified that did not conform to the expected pattern as described by Hardcastle et al. (1991). Close examination of these frames revealed interruptions in tongue-to-palate closure, which appeared to be possibly the result of interference of the EMA coils. Similarly, 2% of the TBI speaker's frames displayed similar characteristics. These frames were, therefore, removed before the representative frames of maximum contact were generated and before further analysis was conducted.

Number of Contacts at the Frame of Maximum Contact. Figure 12 presents a comparison of the mean number of tongue-to-palate contacts at the frame of maximum contact produced by the TBI speaker and the control speaker in the word initial and word final positions for the target consonants, [TEXT NOT REPRODUCIBLE IN ASCII]. The velar consonant [TEXT NOT REPRODUCIBLE IN ASCII] was eliminated from this section of the analysis, as it was suspected that closure occurred posterior to the EPG palate for the majority of the productions; therefore, the frame of maximum contact recorded may not have been a true indication of the speakers' tongue-to-palate contact. The number of standard deviations the TBI speaker's values were away from the control speaker's values was investigated. Inconsistent findings were evident in the analysis. In the production of the alveolar stop [TEXT NOT REPRODUCIBLE IN ASCII] in the word initial position and word final position, the TBI speaker's mean number of tongue-to-palate contacts was consistent with that of the control speaker (i.e., within 2 SD). In contrast, for the TBI speaker's production of the alveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII] in the word initial and word final positions, the mean maximum number of contacts in the closure phase was 2.25 SD and 3.23 SD, respectively, less than the mean maximum number of contacts calculated for the control speaker. In the production of the postalveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII] in the word final position, the mean maximum number of contacts reached by the TBI speaker was consistent with that reached by the control speaker.

Timing Parameters. Comparison of closure / stable constriction phase duration across speakers. The mean durations of the closure phases exhibited by the TBI speaker and the control speaker for the individual consonants [TEXT NOT REPRODUCIBLE IN ASCII] are compared and presented in Figure 13. The velar consonant [TEXT NOT REPRODUCIBLE IN ASCII] was eliminated from this section of the analysis, as closure was not detected in any of the repetitions produced by the TBI speaker. In the analysis of the anterior consonants, a clear pattern emerged; the TBI speaker displayed significantly longer mean closure/stable constriction phases than the control speaker (range 2.25-4.03 SD greater than control speaker's means). The production of the alveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII] in the word initial position, however, was an exception, where the TBI speaker achieved a mean stable constriction phase duration that was consistent with the control speaker (i.e., within 2 SD).

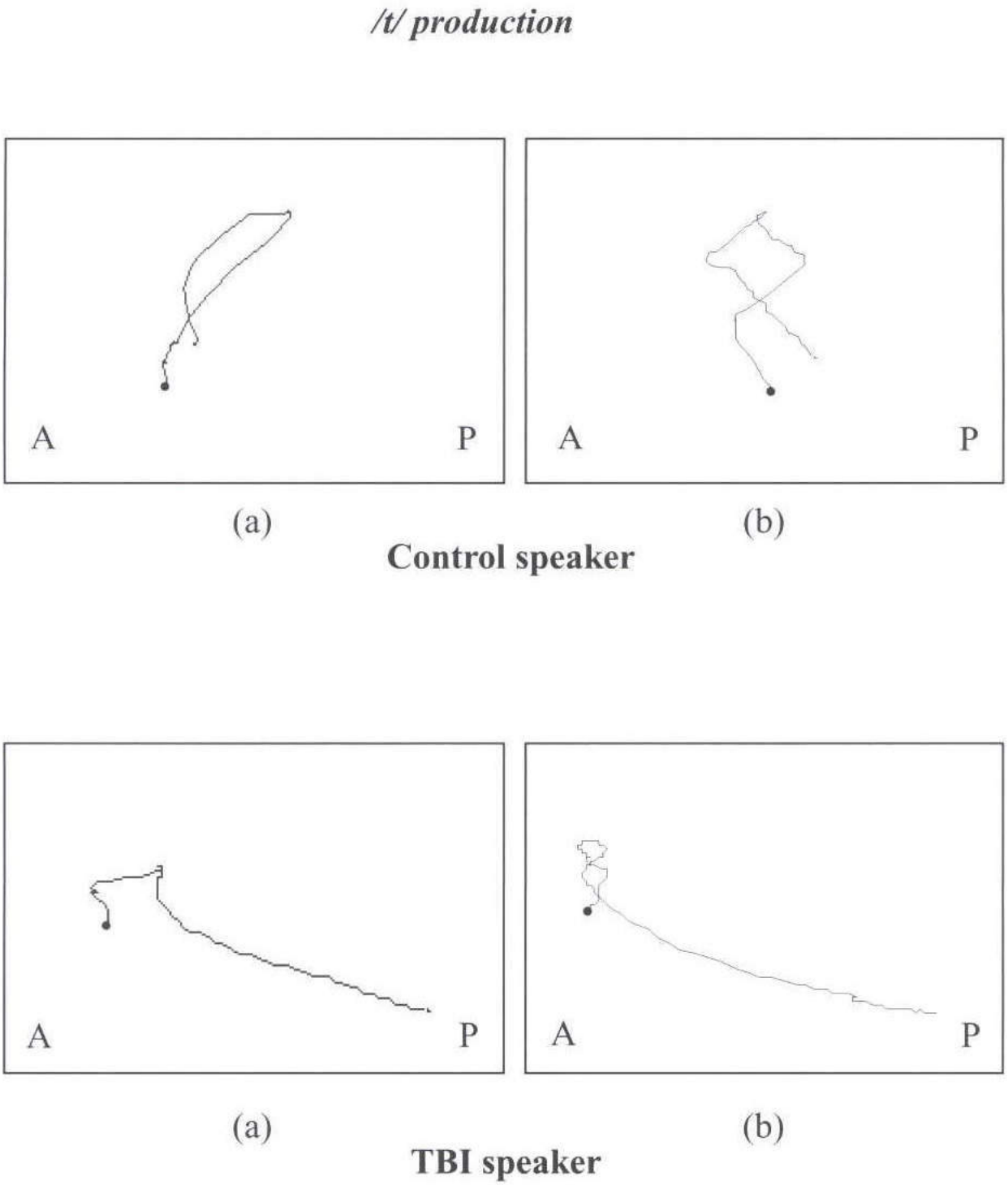


Figure 8. Examples of (a) typical and (b) seemingly atypical movement trajectories of the principal receiver coil (i.e., tongue tip coil) exhibited by the TBI speaker and control speaker during production of /t/ in word initial position (*Note:* trajectories not to scale). Onset of approach is indicated (●). Orientation of figures is indicated as anterior (A) and posterior (P).

Comparison of closure/stable constriction phase duration within speakers. The duration of the closure phase of [TEXT NOT REPRODUCIBLE IN ASCII] and [TEXT NOT REPRODUCIBLE IN ASCII] in the word initial position and in the word final position were contrasted for the TBI speaker and the control speaker. Neither of the target consonants [TEXT NOT REPRODUCIBLE IN ASCII] or [TEXT NOT REPRODUCIBLE IN ASCII] were included in this section of the analysis, as relevant data (i.e., word initial productions or [TEXT NOT REPRODUCIBLE IN ASCII] data due to failure to detect closures) were missing. In the control speaker's [TEXT NOT REPRODUCIBLE IN ASCII] production, the value recorded for the word initial position was 2.5 SD longer than that recorded for the word final position. Conversely, for the control speaker's [TEXT NOT REPRODUCIBLE IN ASCII] production, the stable constriction phase duration in the word initial position was consistent with the stable constriction phase duration in the word final position (i.e., within 2 SD).

For both [TEXT NOT REPRODUCIBLE IN ASCII] and [TEXT NOT REPRODUCIBLE IN ASCII] productions, the TBI speaker's closure/stable constriction phase duration in the word final position was within 2 SD of the closure/stable constriction phase duration in the word initial position.

Phonetic Transcriptions. The phonetic transcriptions of the words read aloud by the TBI speaker during the EMA/EPG assessment indicated that while the target consonants, [TEXT NOT REPRODUCIBLE IN ASCII], were perceived to be produced accurately in the word initial position on most occasions, when produced in the word final position, accuracy was significantly compromised. The TBI speaker's productions of [TEXT NOT REPRODUCIBLE IN ASCII] in the word initial position were judged to be identifiable, although unclear in 25% of the productions, by one of the two speech-language pathologists who acted as a judge. In contrast, the word final [TEXT NOT REPRODUCIBLE IN ASCII] was perceived by the two speech-language pathologists to be omitted or substituted with the velar stop [TEXT NOT REPRODUCIBLE IN ASCII] in 60 to 90% of the total number of productions.

The TBI speaker's productions of [TEXT NOT REPRODUCIBLE IN ASCII] in the word initial position were perceived to be produced accurately in all of the repetitions recorded. In the word final position, however, no correct productions were noted. Rather, the [TEXT NOT REPRODUCIBLE IN ASCII] productions were perceived to be omitted in 78% of the productions by one of the judges or substituted with the velar stop [TEXT NOT REPRODUCIBLE IN ASCII] in 88% of the productions by the other judge. The remaining productions were judged to be substituted with the alveolar stop [TEXT NOT REPRODUCIBLE IN ASCII]. In the word final [TEXT NOT REPRODUCIBLE IN ASCII] production, 10 to 30% of the TBI speaker's repetitions recorded were judged to be produced inaccurately, with the consonant being perceived as the alveolar affricate [TEXT NOT REPRODUCIBLE IN ASCII] or [TEXT NOT REPRODUCIBLE IN ASCII] or the alveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII]. The TBI speaker's velar stop [TEXT NOT REPRODUCIBLE IN ASCII] productions in the word initial position were perceived to be produced accurately on all occasions. In the word final position, although one judge rated the accuracy to be 100%, the other gave an accuracy rating of 63%, where substitutions of [TEXT NOT REPRODUCIBLE IN ASCII] and [TEXT NOT REPRODUCIBLE IN ASCII] were noted.

In contrast to the TBI speaker, the control speaker's productions of the target consonants, [TEXT NOT REPRODUCIBLE IN ASCII], were judged by both of the raters to be perceptually acceptable, with the exception of the velar stop [TEXT NOT REPRODUCIBLE IN ASCII], which was judged to be substituted with the alveolar stop [TEXT NOT REPRODUCIBLE IN ASCII] in 10% of the productions in the word initial position, and 10 to 20% of the time in the word final position. The percentage agreement, calculated as the percentage of time the two raters agreed that a production was correct or incorrect, was calculated to be 76%.

DISCUSSION

The TBI speaker in the present study presented with reductions in intelligibility and rate of speech, with rate of speech being the more compromised feature of the two. Indeed, impairments in the timing aspect of the TBI speaker's speech were highlighted in the form of phoneme prolongation as identified through perceptual analysis. The physiological techniques of EMA and EPG were used to examine the underlying basis of the reductions in intelligibility and rate of speech demonstrated by the TBI speaker. Examination of the articulatory parameters recorded using EMA and EPG revealed characteristic differences in the tongue movements exhibited by the TBI speaker and the control speaker that further highlighted the TBI speaker's timing disturbances. Objective analysis using EMA revealed consonant approach phase and release phase durations that were typically consistent with those recorded for the control speaker. Compensatory or deviant kinematic strategies may have been used, however, to achieve these durations. A prominent finding revealed by EPG analysis was the significantly longer closure and constriction periods exhibited by the TBI speaker in the production of the target consonants. This suggests that a prolonged closure/stable constriction phase may have been a contributing factor to the prolonged phonemes perceived.

Despite the findings of approach and release phase durations that were consistent with the control speaker, analysis using EMA revealed possible problems, including significantly decreased distances traveled by the TBI speaker's tongue and significantly reduced mean maximum velocities, which may have contributed to the deviant speech characteristics perceived. The movement trajectories revealed a typical pattern of more gradual rises of the TBI speaker's tongue to the palate in the approach phase when compared with the control speaker. This suggests that a higher tongue position was typically employed at the start of each approach phase, leading to shorter distances needing to be traveled by the tongue to reach the palate. A number of factors could have contributed to the significantly reduced distances recorded and the higher tongue postures employed. For example, a reduction in lingual proprioception may have been one such factor, so that the tongue could not follow an appropriate course to or away from the palate in the production of the target sounds. This suggestion is supported by reports of a speaker investigated by Goldstein, Ziegler, Vogel, and Hoole (1994) who complained of "being unaware of where his tongue was and which way it moved" (p. 204) following a severe closed-head injury. Alternatively, a reduction in jaw control and mobility may also have contributed to the reduced distances traveled to the palate, as reduced jaw control or mobility could result in a reduction in the amount of space available for tongue movement in the oral cavity. Indeed, reduced mobility may be indicative of excessive tone or hypertonicity in the muscles of the tongue and/or jaw. The TBI speaker's neuropathology, which consisted of diffuse axonal injury and cortical dysfunction, supports this hypothesis, as hypertonicity is regarded as a characteristic sign of upper motor neuron damage (Murdoch, 1990). Finally, a reduction in effort and/or motivation for speech, as reported by the TBI speaker to be the case at times, could also have contributed to the reduced distances recorded.

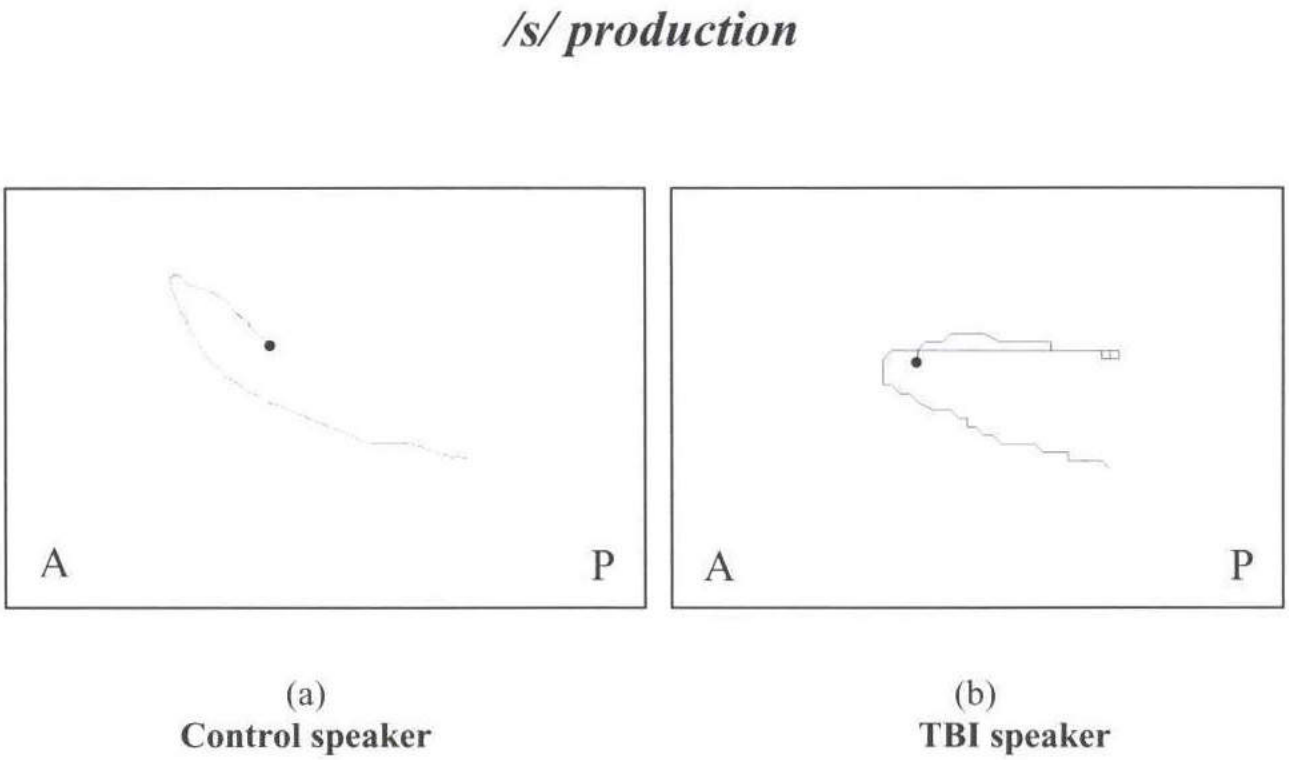


Figure 9. An example of a (a) typical movement trajectory of the principal receiver coil (i.e., tongue tip coil) exhibited by the control speaker and a (b) seemingly atypical movement trajectory by the TBI speaker during production of /s/ in word initial position (*Note:* trajectories not to scale). Onset of approach is indicated (●). Orientation of figures is indicated as anterior (A) and posterior (P).

Regardless of the cause, the reduction in distance may, in turn, have contributed to the reduced maximum velocity values recorded. With the significantly reduced distances traveled, there may not have been sufficient opportunity for the tongue to build up speed before reaching the palate. In fact, as there is a positive relationship between velocity and distance (Kuehn & Moll, 1976; Ostry & Munhall, 1985), if the value of one decreases, the value of the other would naturally be expected to follow. An alternate explanation to the reductions in velocity, however, may be an impaired ability to generate speed. Impaired speed generation capabilities may be related to motor-unit recruitment disturbances and/or reductions in the frequency of stimulation of muscle fibres within the tongue. Consequently, the related muscles are not able to function to their maximum capacity. The possibility of impaired speed generation capabilities being exhibited by the TBI speaker was supported by his mean acceleration values that were found to be typically marginally reduced, as well as nonspeech lingual movements that were observed to be slow and laborious. Whether the reduction in velocity was related to decreased distances being traveled by the TBI speaker's tongue or impaired speed generation capabilities, it appears that the reductions in velocity were compensated by the reduced distances, resulting in approach and release phases that were consistent with the control speaker.

In regard to the TBI speaker's prolonged closure and constriction phases as revealed by EPG, three possible explanations are proposed to explain this finding, including reduced oral tactile or kinesthetic sensation of the tongue and palate, reduced tongue strength, and a reduced ability of the tongue to decelerate sufficiently prior to contact with the palate. Disturbed sensation of the tongue, lips, jaw, and palate has been shown to significantly affect speech production, particularly articulatory precision (Duffy, 1995). Therefore, it is possible that the TBI speaker in the present study experienced decreased oral sensations, including reduced tactile and kinesthetic sensations, which were manifested in a decreased awareness of the length of time his tongue was in contact with the palate during the closure and constriction phases of his consonant productions. The excess lingual movement at the palate apparent in the TBI speaker's movement trajectories, rather than controlled and precise tongue-to-palate contacts as desired, may be further support of decreased tactile and kinesthetic sensations. Indeed, with the EPG palate in situ and thus with palate sensation removed, the effects of reduced tactile and kinesthetic sensation may have been magnified. Alternatively, reduced tongue strength may have contributed to the prolonged closure and constriction phases and excess lingual movement at the palate observed. With a weakened tongue, as supported by observations of the speaker's nonspeech lingual movements, the TBI speaker may have been unable to sustain the tongue position at the palate, producing unsteady movements at the palate.

Combined analysis of the EMA and EPG results revealed a possible connection between the typically marginally reduced lingual deceleration values found for the TBI speaker using EMA and his increased closure/stable constriction phases, as recorded by EPG. It can be speculated that the reduced lingual deceleration ability of the speaker may have resulted in a failure of the tongue to reach its target position on the palate in a well-controlled manner. Rather, the tongue may have hit the palate with excessive force or at an inappropriate speed, causing it to slide across the palate, which in turn may have prolonged the closure or constriction duration in the consonant productions. Indeed, comparatively more extensive lingual movement at the palate was one of the deviant features noted in the EMA movement trajectories of the TBI speaker.

In addition to affecting the rate of speech, disturbances in articulatory timing may contribute to a reduction in speech intelligibility. In the case of the TBI speaker, his articulatory timing disturbances may have manifested in the perception of consonant and vowel imprecision, consonant omission, and substitution noted in the phonetic transcription analysis. Prolonged closure phase durations, in particular, may affect the closure-release "burst" action required in the production of stops, resulting in a reduction in the crispness and precision of the consonants produced (Goozee, Murdoch, & Theodoros, 1999). Although examination of the closure phase duration of the target consonants using EPG revealed no discrepancy between the consonants in the word initial and word final positions, differences in the release "burst" action may have contributed to the significant perceptual differences between the consonants in these two positions, as highlighted in the phonetic transcriptions of the TBI speaker's productions in the EMA/EPG assessment. It has been

proposed that identification of consonants produced in word-initial positions is crucial to speech perception (Marslen-Wilson, 1980) and that speech motor control may be structured, at least partially, to produce precise word onsets (Adams, Weismer & Kent, 1993; Weismer, & Liss, 1991). Therefore, it is possible that, despite closure/stable constriction phases being lengthened, the enhanced articulatory accuracy perceived for the word initial consonants was partially caused by a greater amount of effort being exerted, which in turn may lead to a greater amount of air pressure being accumulated in the oral cavity, creating a more precise and more prominent release burst action. The perceived omission of the target consonants, [TEXT NOT REPRODUCIBLE IN ASCII], in the word final position in the phonetic transcriptions of the TBI speaker's speech may be attributed to a reduction in this air pressure buildup in the oral cavity.

Substitution, in particular backing or stopping (i.e., [TEXT NOT REPRODUCIBLE IN ASCII] to [TEXT NOT REPRODUCIBLE IN ASCII] or [TEXT NOT REPRODUCIBLE IN ASCII] to [TEXT NOT REPRODUCIBLE IN ASCII]), was another common speech process observed to compromise the TBI speaker's intelligibility of speech as highlighted by the phonetic transcriptions. A possible cause of these errors may be a tongue body that positions itself higher and closer to the palate than would an average tongue. Indeed, a higher tongue position was indicated by the EMA movement trajectories of the TBI speaker when compared with those of the control speaker. In speech production, a higher tongue body position could lessen the amount of space available in the oral cavity, creating some-what of a constriction in the back portion of the oral cavity and changing the acoustic features of the sound. Consequently, consonants that are normally made with less constriction at the back portion of the oral cavity (e.g., [TEXT NOT REPRODUCIBLE IN ASCII] and [TEXT NOT REPRODUCIBLE IN ASCII]) may be perceived as back consonants or as being stopped. Future research using EMA should thus consider the movement of the tongue in its entirety, as it is this movement that creates the individual consonants and not solely the primary tongue component presumed to be responsible for closure/stable constriction. Alternatively, other constrictions or obstructions located posterior to the EPG palate, not detected at the time of testing, could also have led to the perception of backing. Researchers need to be aware of this possibility in their studies.

In addition to being a valuable tool for examining the timing aspects of tongue contacts with the palate, EPG can also precisely determine the location and pattern of contact during speech. The spatial characteristics of the TBI speaker's tongue-to-palate contacts exhibited in the production of the target consonants appeared to be consistent with those of the control speaker. Some discrepancies were noted, however, in the number of tongue-to-palate contacts at the frame of maximum contact. For the production of the alveolar fricative [TEXT NOT REPRODUCIBLE IN ASCII], a smaller area of contact was recorded in both the word initial and word final positions. Discrepancies in contact area may be indicative of impairments in lingual motor control, which may, in turn, lead to a reduction in the intelligibility of a person's speech. Indeed, the excess movement noted at or near the palate evident in the TBI speaker's movement trajectories may be further support of impairments in lingual motor control. However, it must also be noted that while perceptual acceptability of the consonant [TEXT NOT REPRODUCIBLE IN ASCII] in the word final position was indeed compromised, as indicated in the phonetic transcriptions, in its production in the word initial position, the accuracy of production was reported to be 100%. The difference observed in the amount of contact between the TBI speaker and the control speaker suggested, therefore, not a problem in lingual control, but perhaps differences in palate morphology (e.g., palatal vault height and width). Again, the discrepancy perceived between the consonant in the word initial and word final positions may be attributed to the possibility of varying amounts of effort being exerted in the word production, affecting the accumulation of air pressure in the oral cavity, and ultimately, the clarity of the consonants perceived.

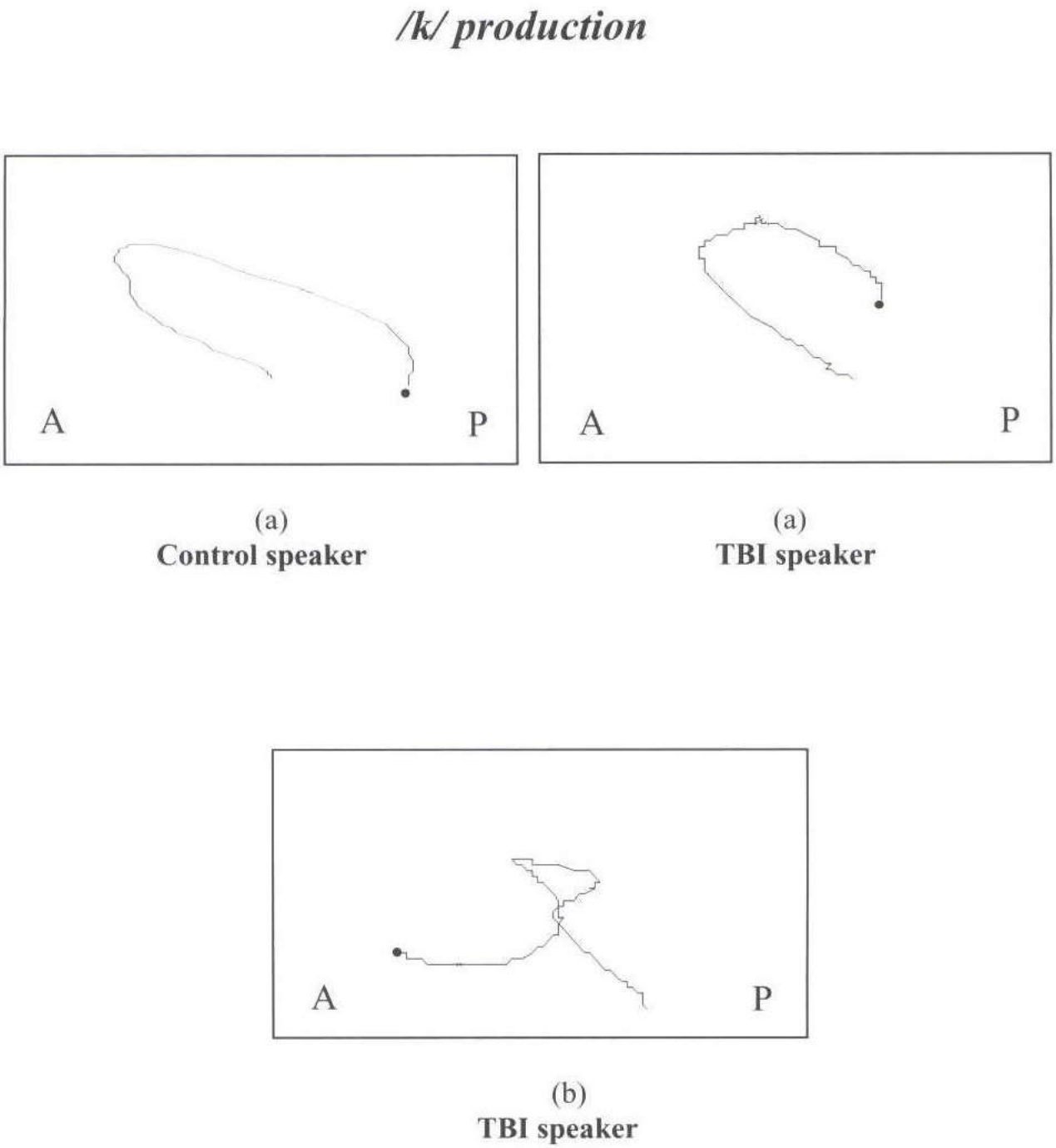


Figure 10. Examples of (a) typical and (b) seemingly atypical movement trajectories of the principal receiver coil (i.e., tongue back coil) exhibited by the control speaker and TBI speaker during production of /k/ in word initial position (*Note:* trajectories not to scale). Orientation of figures is indicated as anterior (A) and posterior (P).

CONCLUSION

Combined EMA and EPG analysis was able to pinpoint possible underlying mechanisms contributing to the reduced rate of speech and prolonged phonemes exhibited by an individual with dysarthria following childhood TBI. Objective analysis of the TBI speaker's speech using EMA revealed approach and release phase durations that were consistent with those recorded for the control speaker. However, further analysis revealed decreased distances traveled by the tongue and decreased velocity values that may have counterbalanced each other to result in these appropriate phase durations. In contrast, EPG analysis revealed significantly prolonged closure/stable constriction phase durations in the TBI speaker's consonant productions. The prolonged closure/stable constriction phase durations may have contributed to the perceived prolonged phonemes, and, ultimately, the slow rate of speech exhibited by the TBI speaker. Further EMA analysis revealed extensive lingual movements at or near the palate, which contributed to the prolonged closure/constriction phases recorded. Combined analysis of EMA and EPG, therefore, enabled a more complete interpretation of the speaker's tongue

dynamics than could perceptual assessments or the use of EMA or EPG alone. The ability to gain specific insights into the underlying nature of a speech problem is invaluable in treatment planning.

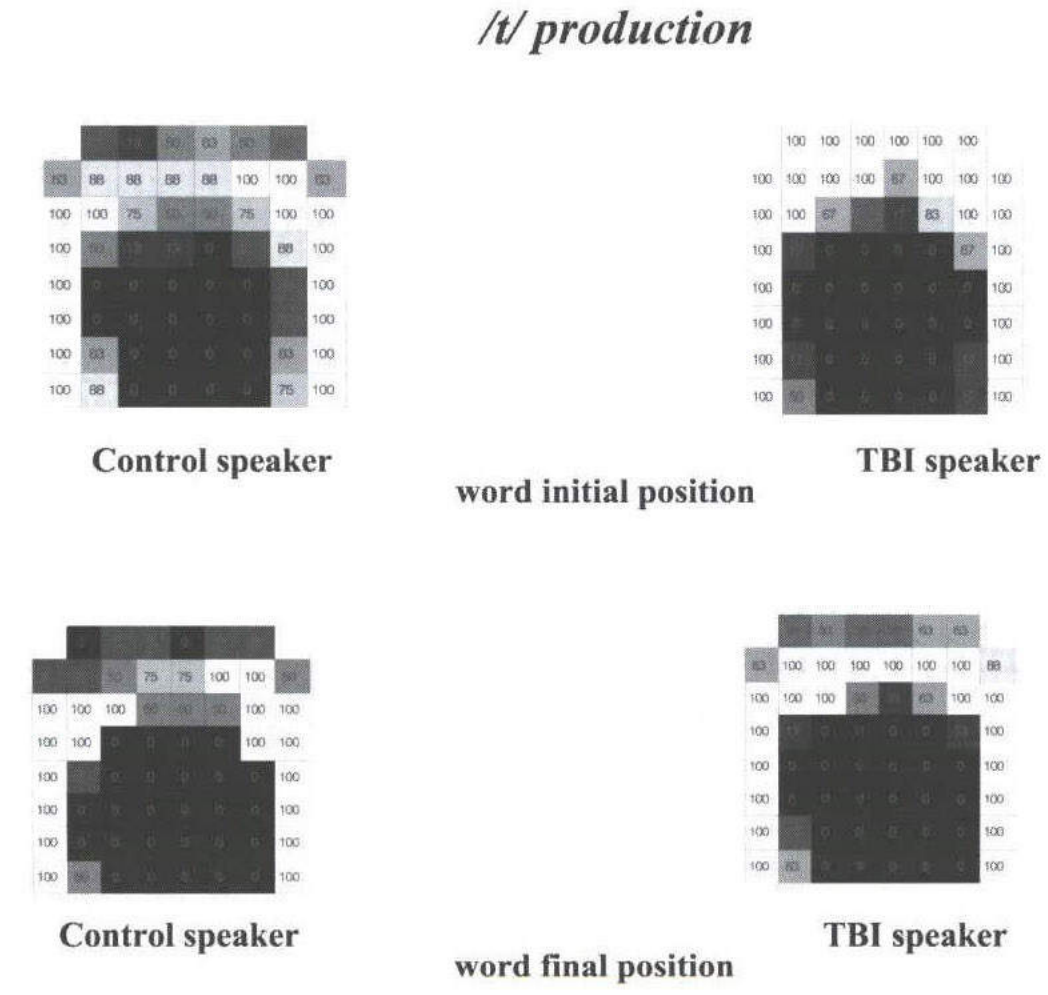
For the TBI speaker in this present study, treatment would be directed at decreasing the duration of his closure/stable constriction phases to develop more appropriate phoneme lengths and improve rate of speech. The potential underlying problem of excess lingual movement at the palate could be specifically targeted to achieve this, for example, by the use of a combined EMA and EPG biofeedback system where the individual could directly view his or her tongue movement in real time. As there appears to be high variability in the underlying nature of dysarthria following childhood TBI (Cahill et al., 2000; Murdoch & Goozee, 2003), a tailored treatment plan should be made for each patient if maximum effectiveness of the treatment were to be achieved. EMA and EPG can be used to help achieve this goal.

Despite advantages of the simultaneous use of EMA and EPG, one important limitation was noted. Before analysis of the EPG data began, a number of frames were removed due to tongue-to-palate contact patterns recorded that did not conform to the expected pattern. Close examination of these contact patterns revealed atypical patterns likely to be the result of impeding EMA receiver coils. Therefore, although the simultaneous use of EMA and EPG can provide important insights into the tongue dynamics of an individual, care must be taken in the interpretation of the results to ensure that accuracy of the information is not compromised. Other perceptual measures of intelligibility and more detailed phonetic analyses before and after the EMA coils and EPG palate are in situ may help quantify any effects of these instruments on the speaker's speech. To maximize the level of accuracy of data interpretation, a larger sample of control speakers against which deviant speech characteristics and articulatory kinematics may be compared would also be beneficial.

Future studies following this line of research should consider incorporating alternative assessment measures. One such measure could involve the analysis of airflow and intraoral air pressure throughout the production of a word. In the present study, one factor proposed to reduce the articulatory accuracy of the TBI speaker's speech was a reduction in air pressure being accumulated in the oral cavity, leading to consonant production that was compromised in clarity and precision. Finally, other factors that should be taken into consideration in future EMA and EPG based research and analyses include palate morphology and positioning of the whole tongue (i.e., analysis of nonprimary receiver coils in addition to primary coils). The analysis of such features would enhance the interpretation of EMA and EPG data and would aid the identification of deviant articulatory mechanisms underlying dysarthric speech disturbances.

APPENDIX

Word List:



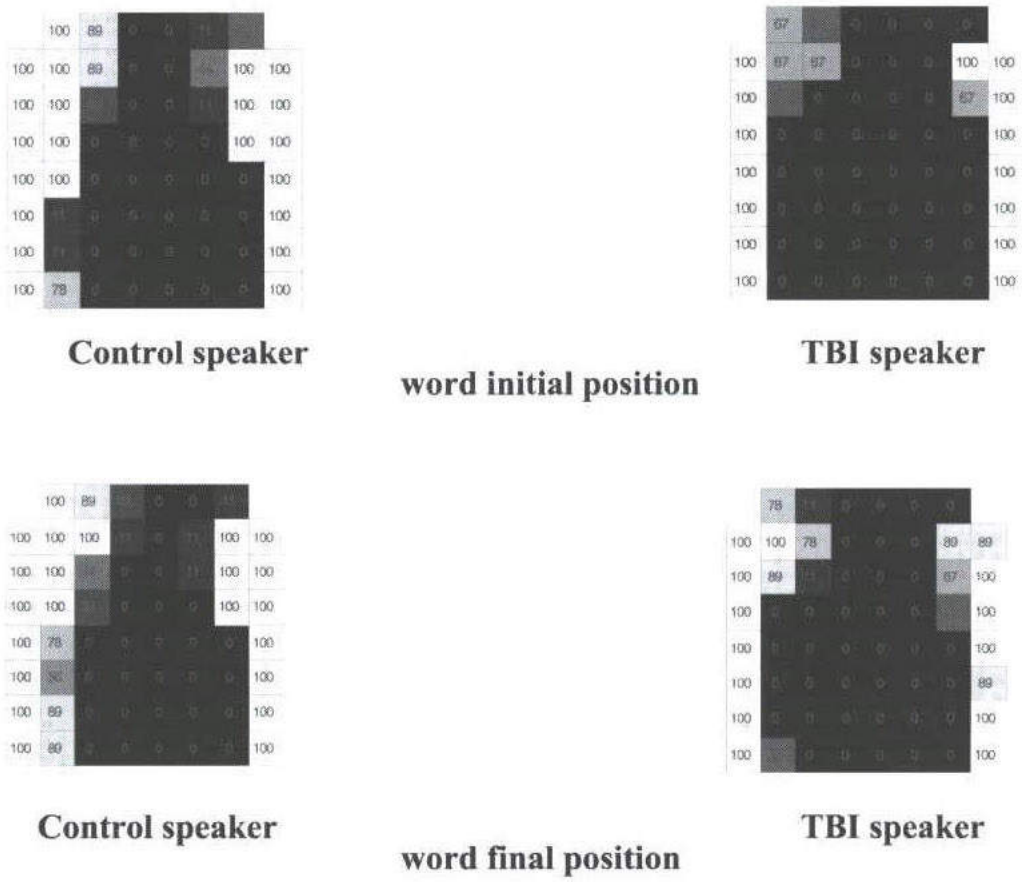


Figure 11. Representative frames of maximum contact generated for /t, s, ʃ, and k/ as produced by the TBI speaker and control speaker in word initial and word final positions. Top section of each palatogram represents the anterior region of the palate. The numbers in small boxes represent the percentage of time the sensor was touched over the total number of repetitions recorded for the target consonant. Different shadings of grey represent the varying percentages. *(continues)*

A tarp

A part

A sarge

A pass

A shark

A marsh

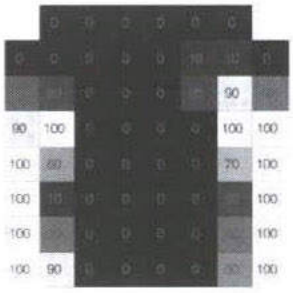
A car

/ʃ/ production

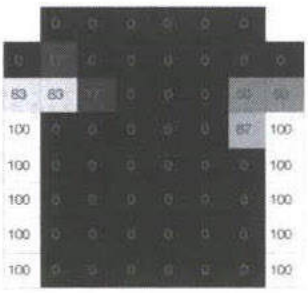
Control speaker

TBI speaker

word initial position



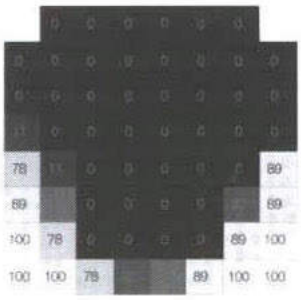
Control speaker



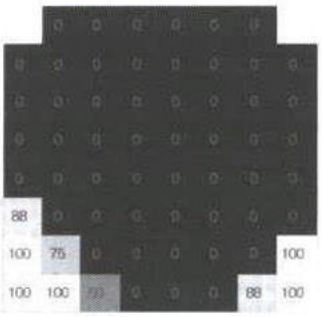
TBI speaker

word final position

/k/ production



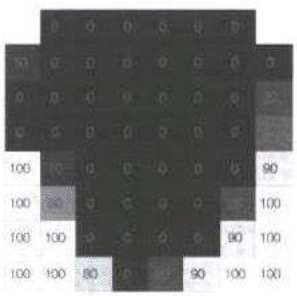
Control speaker



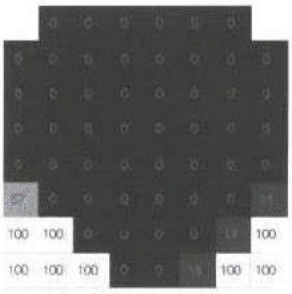
TBI speaker

word initial position

speaker



Control speaker



TBI speaker

word final position

Figure 11. *(continued)*

A park

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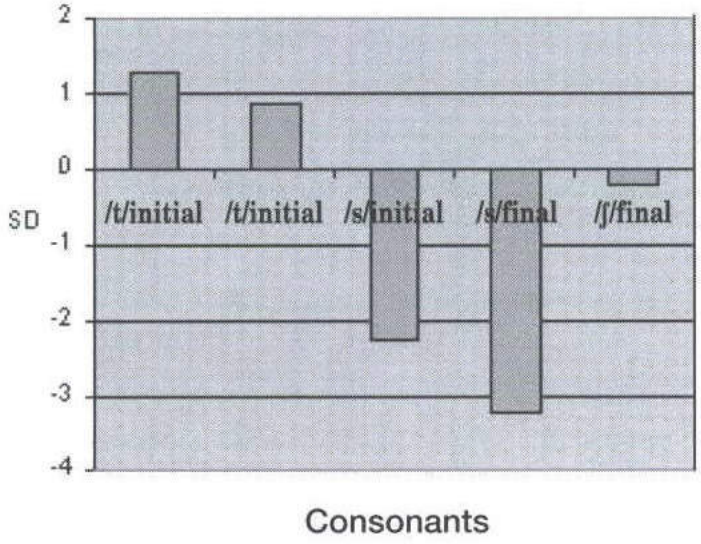

Figure 12. Difference (in standard deviations) between the TBI speaker and control speaker for mean number of contacts at the frame of maximum contact for /t, s, and ʃ/ in word initial and word final positions. Positive values indicate a greater number of contacts achieved by the TBI speaker compared to the control speaker.

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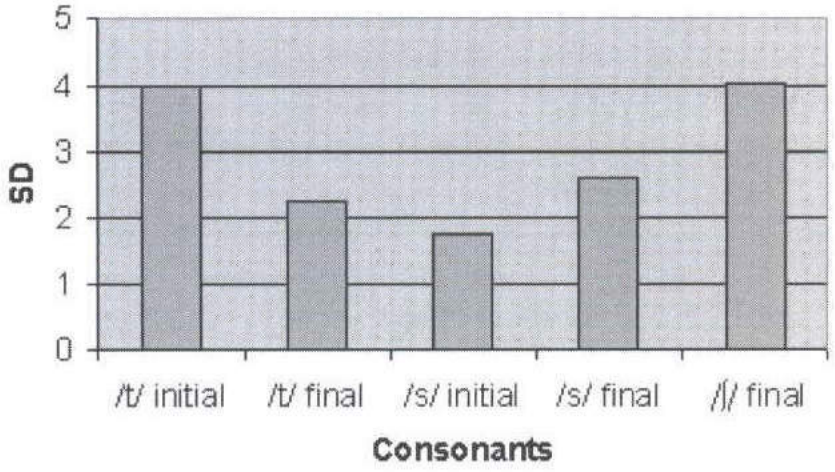

Figure 13. Difference (in standard deviations) in the mean closure/stable constriction phase duration exhibited by the TBI speaker and control speaker for /t, s and ʃ/ in word initial and word final positions. Positive values indicate a longer phase duration achieved by the TBI speaker compared to the control speaker.

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