Lip–larynx coordination in speech: Effects of mechanical perturbations to the lower lip

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(Received 6 August 1991; revised 10 June 1993; accepted 26 November 1993)

This experiment investigates the coordination between the larynx and the lips and jaw in voiceless consonant production using an experimental paradigm where a mechanical perturbation is applied to an articulator. Three subjects received unexpected perturbations to the lower lip during the transition between the first vowel and the first stop in /i'pip/. Movements of the upper articulators (lips and jaw) were recorded using an optoelectronic technique. Laryngeal responses were monitored using transillumination; intraoral pressure and the acoustic signal were also recorded. Results showed that laryngeal abduction was delayed following lip perturbation and that the duration of the laryngeal adduction gesture was lengthened. The oral movements toward closure of two of the subjects were modified and all subjects showed modification of the oral release movements in the perturbed conditions. All subjects showed an increased movement velocity and displacement of the upper lip, lower lip, and jaw in the oral opening phase. First trial compensation, however, was not observed in two of the three subjects. The results are discussed with respect to the speech perturbation literature and the notion of coordinative structures.

PACS numbers: 43.70.Aj, 43.70.Bk, 43.70.Fq

INTRODUCTION

Two generalizations are frequently made about the patterns of spoken language. The first is that speech is the product of a large number of components (i.e., articulators, features, gestures, etc., depending on the theoretical framework). The second is that these components are grouped in different ways over time to produce different lexical items. It is this grouping process that motivates the present work. Specifically, laryngeal–oral coordination is examined.

In general, models of speech production assume that the articulators are coupled for a brief period of time so as to produce sound units. In some speech sounds, articulators "cooperate" to valve the airway at a particular point in the vocal tract (e.g., the tongue and jaw in alveolar stop consonants). In other sounds, the articulators produce acoustic effects at different parts of the vocal tract (e.g., velar and labial contributions to /m/). Understanding this coordination has proven to be a difficult task, since we have no direct window into the planning or control processes. One valuable experimental paradigm for examining such coordination is the introduction of unexpected perturbations to ongoing motor acts. In a standard experiment, the subject is attached to a small torque motor and during some trials the motor is used to unexpectedly generate a brief load. The rationale for this research is that the nature and time course of responses to the load are thought to reveal the motor organization and reflex structure of the ongoing act. This paradigm has been applied to different types of movements in humans such as posture control (e.g., Nashner and McCollum, 1985), hand and finger movements (e.g., Traub *et al.*, 1980; Rothwell *et al.*, 1982; Cole *et al.*, 1984), and respiratory control (Newsom Davis and Sears, 1970).

While dynamic disturbances may seldom occur during normal speech production (however, see footnote 1), a number of studies have used a perturbation methodology to study speech coordination (e.g., Folkins and Abbs, 1975; Folkins and Zimmermann, 1982; Abbs and Gracco, 1984; Kelso *et al.*, 1984; Gracco and Abbs, 1985, 1988, 1989; Shaiman, 1989; Shaiman and Abbs, 1987). Most commonly, a sudden and unexpected load has been applied during ongoing speech to one of the articulators such as the jaw and the compensatory responses in the lower lip, the upper lip, and the tongue have been examined using kinematic records and/or electromyography (EMG).

The present experiment was designed to extend the perturbation paradigm to studies of remote articulators that are tightly coordinated in time during normal speech production. By remote articulators we mean ones that are not involved in producing the same constriction in the vocal tract. More specifically, we were interested to see if perturbations applied to the lower lip during the formation of the bilabial closure for a voiceless stop are compensated for by the larynx in order to maintain articulatory phasing. The lips and larynx can be considered remote in this con-

 0001-4966/94/95(6)/3605/12/$6.00

text because the biomechanical coupling between them is limited.

In the production of voiceless stops, several articulatory activities occur simultaneously and thus have to be temporally coordinated. Among these are the formation of an oral occlusion and an abduction–adduction gesture of the glottis. It is well known that the timing of these two gestures is critical for producing distinctions of voicing and aspiration (cf. Abramson, 1977; Löfqvist, 1980; Löfqvist and Yoshioka, 1984). How this coordination is achieved is not well understood.

Some studies have explored the potential role of oral air pressure in this interarticulator coordination by experimentally venting the oral cavity during the closure interval for voiceless stops. The results of these studies are, however, equivocal. Perkell (1976) found that the duration of the oral closure tended to increase and voice onset time (VOT) to decrease in the vented trials compared to controls. Putnam and Shipp (1975) examined the activity of the intrinsic laryngeal muscles in a similar experiment but did not find any difference in the activity pattern of the posterior crico-arytenoid muscle in the vented trials. Putnam *et al.* (1976), however, reported some laryngeal EMG adjustment to a venting manipulation. None of the experiments applying the venting paradigm, however, has monitored laryngeal articulatory movements.

In one of the early speech perturbation experiments Folkins and Abbs (1975) noted that cessation of voicing in a sequence of vowel and bilabial voiceless stop was often delayed 15–25 ms when the jaw was loaded, even though the kinematic analysis showed that the upper and lower lips made spatial compensations. A study by Shaiman and Abbs (1987) specifically investigated laryngeal responses to lower lip perturbations in voiceless stop production and observed a similar delay in voicing offset. Shaiman and Abbs suggest that an active delay in glottal abduction follows a perturbation of the lower lip. However, their technique for monitoring laryngeal activity, electroglottography, did not provide a reliable record of laryngeal abduction and adduction.

There are two possible explanations for the acoustic pattern reported by Folkins and Abbs (1975) and Shaiman and Abbs (1987). The first is that no laryngeal adjustment is made and that the extended voicing is simply the result of delayed closure and an extended period of airflow allowing the laryngeal vibrations to continue for a longer period. A second possibility is that the laryngeal opening gesture is delayed when closure is delayed. This can be tested by examining the onset of the laryngeal opening gesture relative to the onset of a preceding vowel. By the first account, there should be no difference in this interval between the perturbed and control trials while by the second account the perturbed intervals should be longer.

In the present study, kinematic recordings were made of the jaw, the lips, and the glottis. In addition, oral pressure was also recorded. An increase in oral pressure is an integral part of normal stop production, and pressure variations associated with lip perturbations can thus be used to assess the effectiveness of articulatory compensations to maintain the stop closure. The kinematic and aerodynamic records were further supplemented with acoustic duration measurements.

I. METHOD

A. Subjects

Three subjects with no known communication disorders were tested. Two of the subjects had no prior experience with the perturbation experimental paradigm while the third (subject SN) had participated in a related experiment. Subject AL is the second author. All three subjects are researchers in the area of speech production and thus are not naive as to the purpose of the experiment.

B. Apparatus and data recording

The subject sat in a dental chair with the head fixed. Perturbations were introduced through a paddle (1 cm wide) that rested on the subject's lower lip. The paddle was connected to a dc brushless torque motor which applied a small constant tracking load of approximately 3 g throughout the experiment. At a predetermined point in time, the motor generated a 50-g load that was used to perturb the lip. The control signal to the motor was a step pulse that resulted in a torque rise time in the range of 2–3 ms. Perturbations were introduced on 12% of the trials, and the duration of the perturbation pulse was 1.0 s. Thus, once the load was introduced during a trial, it remained on for the duration of that trial.

Lip and jaw movements were recorded using a modified Selspot system; infrared light-emitting diodes were placed at the vermilion border of the subject's upper and lower lip and on the chin. The air pressure in the pharynx was recorded using a catheter pressure transducer (Gaeltec, model 12CT/4F) introduced through the nose. Before insertion, the transducer was calibrated in water at body temperature to minimize base line drift due to temperature changes.

Laryngeal articulatory movements (abduction and adduction) were recorded using transillumination. A fiberscope inserted through the nose provided illumination of the glottis, and the light passing through the glottis was sensed by a phototransistor placed on the neck just below the cricoid cartilage. Comparisons between transillumination and fiberoptic films (Löfqvist and Yoshioka, 1980) and also between transillumination and high-speed films (Baer *et al.*, 1983) have shown good agreement. During the experiment, the fiberoptic image was recorded on video tape. The recording was used later to assess whether the view of the glottis remained unobstructed and that no fogging of the fiberscope lens occurred during a trial.

The physiological and the aerodynamic signals were recorded on FM tape for subsequent processing. Conventional acoustic recordings were made simultaneously on the tape recorder. A signal showing the onset and the offset of the load in the loaded trials was also recorded. The signals were digitized with 12-bit resolution for data processing. The speech signal was sampled at 10 kHz, while the movement and pressure signals were sampled at 200

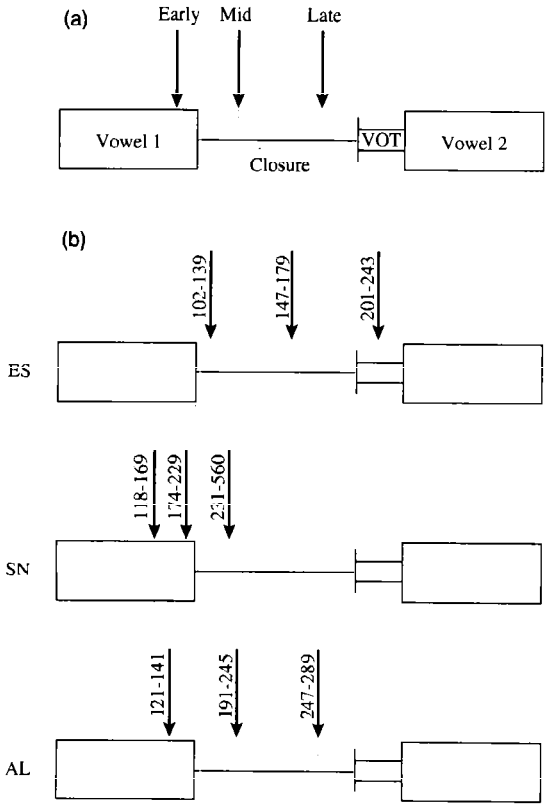

FIG. 1. (a) Schematic representation of the stimuli showing the intended location of the load onsets. (b) Schematic representation of the stimuli showing the average location of the load onsets, indicated by arrows, relative to the average control trial. The numbers above each arrow show the range of load onset times in ms relative to vowel onset.

Hz. After sampling, the movement signals were smoothed with a seven-point triangular window. Movement velocity was obtained from the position signal using a central difference algorithm; see Kay *et al.* (1985) for a description of the digitizing and filtering procedures used.

C. Procedure

The subjects produced the nonsense utterance "/i'pip/ again" 400 times within the experiment with a short pause between each repetition. Perturbations were introduced at three temporal lags from voice onset in the first /i/; the onset of the load was controlled on-line by a microcomputer. The times of load onset were determined individually for each subject by pre-experiment measurements of typical utterance durations. The aim was to perturb the lower lip at three different times during the transition from the first vowel to the voiceless bilabial stop. As shown in Fig. 1(a), these times were as follows: (1) just before vowel offset; (2) early during oral closure; and (3) late in the oral closure interval (just before release). For each subject the actual perturbation locations were divided into three nonoverlapping conditions. The mean values and the range for the three conditions can be seen in Fig. 1(b). The control conditions were composed of the trials immediately preceding each perturbed trial.

TABLE I. Acoustic measurements. Mean durations of the first vowel, oral closure, and voice onset time for the first stop in the nonsense utterance /i'pip/ in ms. The standard errors of the means are in parentheses.

Condition	V1	Closure	VOT
ES			
Early perturbation	90 (4.0)	88 (2.4)	54 (1.9)
Control—early	87 (4.4)	108 (3.4)	50 (2.0)
Midperturbation	82 (3.6)	97 (4.4)	68 (2.5)
Control—mid	87 (4.3)	103 (2.2)	53 (2.3)
Late perturbation	86 (5.2)	104 (2.6)	55 (2.7)
Control—late	88 (5.6)	102 (3.2)	51 (2.5)
SN			
Early perturbation	252 (4.4)	107 (4.3)	51 (1.6)
Control—early	224 (6.6)	125 (1.9)	44 (1.7)
Midperturbation	259 (7.7)	105 (6.4)	53 (2.9)
Control—mid	237 (9.3)	125 (2.6)	43 (2.6)
Late perturbation	242 (8.3)	108 (5.1)	64 (2.2)
Control—late	236 (6.3)	124 (1.9)	51 (2.6)
AL			
Early perturbation	176 (6.4)	93 (7.0)	79 (4.7)
Control—early	159 (4.3)	121 (1.3)	48 (1.8)
Midperturbation	161 (4.6)	96 (1.6)	73 (1.6)
Control—mid	156 (6.5)	123 (1.4)	42 (1.6)
Late perturbation	161 (4.7)	104 (1.8)	66 (2.8)
Control—late	159 (4.3)	121 (1.0)	47 (1.4)

D. Data analysis

The smoothed transillumination signal was measured to determine the duration of the opening and closing gestures associated with the production of the first /p/. Onsets and offsets of laryngeal movements were indicated by zero crossings in the first derivative of the transillumination record. For movements of the upper articulators, the measurements were made on the vertical displacement signal with this axis defined relative to the camera's x and y axes. The lower lip displacement represents the net vertical lip movement with the jaw movement subtracted. Onsets and offsets of the oral movements were identified in the smoothed velocity signals and taken as the point in time where the signal was 5% of peak velocity for each trial.

All data were analyzed using analysis of variance. Because of lost data due to equipment malfunction the number of analyzed trials differed between the subjects. For subjects SN, Al, and ES, respectively, 96, 98, and 82 trials were analyzed. In addition, for ES, the air pressure and transillumination signals could not be analyzed on one and two trials, respectively. As a result, the degrees of freedom in the analyses of variance differ between variables for ES.

II. RESULTS

A. Acoustic patterns

Durations of acoustic events were measured to give an overall summary of the perturbation effects. Table I shows the average durations and standard errors of the first vowel, oral closure, and voice onset time for the first stop in the utterance "/i'pip/ again" for each of the three per

TABLE II. Oral closing kinematics. Displacement in cm, peak velocity in cm/s, and movement duration in ms are shown for the movements of the jaw, upper lip, and lower lip. The standard errors of the means are in parentheses.

	Jaw			Upper lip			Lower lip		
Condition	Disp.	Vel.	Dur.	Disp.	Vel.	Dur.	Disp.	Vel.	Dur.
Subject ES									
Early	0.60	8.06	131	0.44	7.40	133	0.19	5.33	61
	(0.04)	(0.49)	(4.24)	(0.01)	(0.35)	(6.08)	(0.01)	(0.32)	(4.44)
Control	0.57	6.93	141	0.43	7.34	134	0.18	4.74	63
	(0.04)	(0.35)	(4.95)	(0.03)	(0.56)	(7.32)	(0.01)	(0.38)	(4.15)
Mid	0.60	7.62	133	0.44	7.49	130	0.19	4.66	56
	(0.04)	(0.55)	(4.67)	(0.01)	(0.53)	(7.66)	(0.01)	(0.38)	(2.94)
Control	0.61	7.52	136	0.41	7.26	124	0.16	4.76	59
	(0.04)	(0.54)	(3.75)	(0.01)	(0.38)	(6.19)	(0.01)	(0.36)	(3.67)
Late	0.56	7.19	136	0.40	6.98	127	0.17	4.72	62
	(0.03)	(0.52)	(4.13)	(0.02)	(0.44)	(3.72)	(0.01)	(0.25)	(6.39)
Control	0.63	8.01	136	0.40	7.15	117	0.18	5.01	61
	(0.05)	(0.61)	(6.52)	(0.02)	(0.30)	(4.28)	(0.02)	(0.48)	(5.25)
Subject SN									
Early	0.65	9.50	131	0.48	7.73	133	0.30	5.24	111
	(0.03)	(0.33)	(4.87)	(0.01)	(0.15)	(8.07)	(0.03)	(0.37)	(9.55)
Control	0.51	6.69	161	0.40	5.29	194	0.138	3.00	76
	(0.02)	(0.37)	(8.7)	(0.02)	(0.35)	(12.5)	(0.01)	(0.25)	(6.76)
Mid	0.54	7.47	138	0.49	6.56	191	0.19	4.25	75
	(0.04)	(0.55)	(6.8)	(0.01)	(0.24)	(9.82)	(0.02)	(0.40)	(8.43)
Control	0.57	7.62	147	0.36	5.05	169	0.12	2.93	62
	(0.03)	(0.31)	(7.04)	(0.01)	(0.29)	(8.61)	(0.03)	(0.40)	(7.71)
Late	0.53	7.28	144	0.45	5.80	192	0.15	3.53	76
	(0.03)	(0.37)	(7.91)	(0.01)	(0.35)	(12.1)	(0.02)	(0.41)	(8.47)
Control	0.51	6.58	151	0.37	4.70	187	0.18	3.85	85
	(0.05)	(0.45)	(8.51)	(0.01)	(0.24)	(10.8)	(0.02)	(0.30)	(8.68)
Subject AL									
Early	0.46	7.55	150	0.42	7.68	105	...	...	...
	(0.03)	(0.45)	(12.7)	(0.01)	(0.22)	(3.81)	...	...	...
Control	0.44	6.80	133	0.34	6.60	108	0.18	4.97	63
	(0.02)	(0.33)	(4.58)	(0.01)	(0.26)	(4.71)	(0.01)	(0.17)	(2.12)
Mid	0.35	5.90	121	0.39	6.60	146	...	...	...
	(0.02)	(0.35)	(5.33)	(0.01)	(0.23)	(5.08)	...	...	...
Control	0.40	6.38	127	0.35	6.75	111	0.19	4.97	64
	(0.02)	(0.32)	(4.84)	(0.01)	(0.19)	(5.48)	(0.01)	(0.14)	(1.98)
Late	0.43	6.65	129	0.34	6.66	112	...	...	...
	(0.02)	(0.26)	(5.85)	(0.01)	(0.33)	(4.80)	...	...	...
Control	0.40	6.40	133	0.34	6.84	106	0.19	5.08	62
	(0.02)	(0.27)	(5.44)	(0.01)	(0.20)	(4.32)	(0.01)	(0.20)	(1.60)

turbed conditions and for the accompanying control conditions. The most consistent pattern is the increase in VOT duration in the perturbed trials. All three subjects showed reliably longer VOTs in the trials in which a perturbation was delivered [ES: $F(1,76)=67.63$, $p<0.001$; SN: $F(1,90)=27.82$, $p<0.001$; AL: $F(1,92)=160.62$, $p<0.001$] and there was no systematic trend as a function of the timing of the perturbation onset. Closure duration, on the other hand, was shortened by the perturbation in all cases [ES: $F(1,76)=10.07$, $p<0.01$; SN: $F(1,90)=26.99$, $p<0.001$; AL: $F(1,92)=160.67$, $p<0.001$] except subject ES's late perturbation condition in which the perturbations were delivered very close to the point of oral release [condition X time interaction, ES: $F(2,76)=6.47$, $p<0.01$]. For two of the subjects (early and mid perturbation conditions for SN and early perturbations for AL), some of the perturbation

TABLE III. Oral opening kinematics. Displacement in cm, peak velocity in cm/s, and movement duration in ms are shown for the movements of the jaw, upper lip, and lower lip. The standard errors of the means are in parentheses.

	Jaw			Upper lip			Lower lip		
Condition	Disp.	Vel.	Dur.	Disp.	Vel.	Dur.	Disp.	Vel.	Dur.
Subject ES									
Early	0.54	8.76	96	0.22	4.38	80	0.35	8.02	92
	(0.03)	(0.55)	(2.11)	(0.02)	(0.24)	(4.23)	(0.02)	(0.50)	(6.36)
Control	0.72	9.04	126	0.40	5.14	135	0.21	4.26	90
	(0.04)	(0.56)	(1.89)	(0.01)	(0.16)	(3.52)	(0.01)	(0.22)	(4.35)
Mid	0.71	9.0	121	0.34	5.83	97	0.34	6.18	108
	(0.05)	(0.65)	(1.99)	(0.02)	(0.25)	(6.07)	(0.04)	(0.47)	(11.0)
Control	0.72	9.02	127	0.42	4.98	144	0.21	4.54	76
	(0.03)	(0.41)	(2.39)	(0.01)	(0.15)	(5.25)	(0.01)	(0.18)	(4.61)
Late	0.79	10.8	124	0.45	6.25	136	0.20	4.39	99
	(0.03)	(0.54)	(2.75)	(0.02)	(0.27)	(5.05)	(0.01)	(0.30)	(8.60)
Control	0.76	9.44	131	0.43	5.10	144	0.21	4.61	77
	(0.03)	(0.57)	(2.80)	(0.03)	(0.31)	(5.68)	(0.02)	(0.25)	(3.28)
Subject SN									
Early	0.59	6.51	156	0.47	6.33	129	0.27	3.68	125
	(0.02)	(0.35)	(5.33)	(0.02)	(0.22)	(7.28)	(0.02)	(0.22)	(8.46)
Control	0.60	5.47	171	0.38	4.98	123	0.08	1.54	72
	(0.02)	(0.34)	(3.79)	(0.01)	(0.17)	(2.33)	(0.01)	(0.15)	(9.36)
Mid	0.61	6.44	161	0.43	5.88	122	0.35	4.07	152
	(0.03)	(0.32)	(4.74)	(0.02)	(0.23)	(3.31)	(0.03)	(0.29)	(9.55)
Control	0.63	5.80	167	0.37	4.84	122	0.09	2.16	85
	(0.03)	(0.38)	(4.17)	(0.01)	(0.16)	(1.97)	(0.02)	(0.23)	(13.8)
Late	0.65	6.40	160	0.37	5.20	115	0.44	3.66	177
	(0.02)	(0.33)	(3.07)	(0.02)	(0.17)	(2.86)	(0.03)	(0.31)	(11.4)
Control	0.61	5.52	173	0.36	4.69	125	0.12	1.90	98
	(0.04)	(0.46)	(2.81)	(0.02)	(0.17)	(2.98)	(0.01)	(0.19)	(13.7)
Subject AL									
Early	0.46	5.76	148	0.39	4.93	163	0.37	4.77	183
	(0.03)	(0.25)	(8.1)	(0.01)	(0.14)	(7.4)	(0.06)	(0.23)	(12.5)
Control	0.45	5.44	148	0.40	4.38	167	0.24	2.48	102
	(0.02)	(0.30)	(3.22)	(0.01)	(0.20)	(3.89)	(0.01)	(0.12)	(5.02)
Mid	0.47	6.07	132	0.36	4.83	136	0.67	5.94	221
	(0.01)	(0.20)	(4.18)	(0.01)	(0.20)	(4.65)	(0.01)	(0.24)	(4.73)
Control	0.40	4.72	148	0.43	4.70	161	0.24	2.33	122
	(0.02)	(0.24)	(2.97)	(0.01)	(0.14)	(2.42)	(0.01)	(0.10)	(12.9)
Late	0.54	7.38	136	0.37	5.50	123	0.67	9.07	219
	(0.01)	(0.35)	(1.49)	(0.02)	(0.24)	(3.37)	(0.01)	(0.53)	(12.9)
Control	0.41	4.96	147	0.42	4.68	160	0.25	2.46	112
	(0.01)	(0.24)	(2.70)	(0.01)	(0.18)	(3.49)	(0.01)	(0.09)	(11.2)

onsets occurred during the first vowel. In these three conditions the duration of the vowel was longer in the perturbed conditions than in the control.

B. Oral articulator response to perturbation

1. Oral closing movements

The three subjects showed different patterns of response to the perturbation in the movements of the upper lip, lower lip, and jaw closing movements. In general, an early onset of perturbation was more likely to produce oral compensations. The overall kinematics of the lower lip raising movements toward the /p/ closure were significantly altered for one of the subjects. For subject SN, the displacement and peak velocity of the lower lip gesture increased in the perturbed trials; the effect was significant [$F(1,90)=16.72$, $p<0.01$] for displacement, and for peak

velocity [$F(1,90)=13.42$, $p<0.01$]. There were also significant interactions between condition and load onset time for displacement [$F(2,90)=9.25$, $p<0.01$] and for peak velocity [$F(2,90)=6.19$, $p<0.01$] due to the lack of effect in the late perturbation condition. The duration of lower lip raising was only influenced in the early perturbation condition. The perturbed trials showed longer movement durations than the control condition [condition X time interaction, $F(2,90)=3.46$, $p<0.05$]. For subject AL, the onset of the load occurred very close to the onset of lower lip raising resulting in a movement pattern that was difficult to measure consistently. Hence, the results for the lower lip kinematics of this subject have been left out. Subject ES showed no significant differences to the overall lip raising kinematics. (See Table II for descriptive statistics of the oral closing kinematics.)

The upper lip lowering movements towards the labial closure were also different in the perturbed trials. Displacement [$F(1,90)=72.68$, $p<0.01$] and peak velocity [$F(1,90)=55.28$, $p<0.01$] increased for subject SN. Subject AL showed similar effects in the earlier perturbation conditions for displacement [condition X time interaction, $F(2,92)=10.77$, $p<0.01$] and peak velocity [condition X time interaction, $F(2,92)=4.30$, $p<0.05$]. Subject ES's upper lip lowering movements were not significantly altered by the perturbations. For subject SN, the jaw raising movements were shorter in duration in all perturbation conditions [$F(1,90)=6.5$, $p<0.05$] and peak velocity was higher for the early perturbation condition than the control condition [condition X time interaction, $F(2,90)=7.14$, $p<0.01$]. Subjects ES and AL showed no significant differences in jaw movement as a function of the perturbation.

2. Oral opening movements

In contrast to the closing movements, the oral opening movements showed consistent patterns in the perturbed trials. For all three subjects, the oral opening movements were larger, faster, and longer in duration in the perturbed condition than the control trials. The load on the lower lip remained on for 1 s after perturbation onset and thus continued to exert an effect on the oral kinematics. The lower lip lowering movements in the perturbed conditions differed from the control for all three subjects. The displacement during the perturbed conditions was significantly greater than in the control trials [ES: $F(1,76)=25.85$, $p<0.01$; SN: $F(1,90)=21.27$, $p<0.01$; AL: $F(1,92)=218.67$, $p<0.01$]. All three subjects showed reliable condition X time interactions [ES: $F(2,76)=7.38$, $p<0.01$; SN: $F(2,90)=4.03$, $p<0.05$; AL: $F(2,92)=21.41$, $p<0.01$]. For subjects AL and SN the interactions indicate greater differences between the perturbed and control movements in the late versus the early conditions. For subject ES, the opposite pattern is observed. (See Table III for descriptive statistics of the oral opening kinematics.)

The peak velocity of the lower lip lowering movements also showed reliable differences for all three subjects. The movements in the perturbed conditions had higher peak velocities [ES: $F(1,76)=35.59$, $p<0.01$; SN: $F(1,90)=97.27$, $p<0.01$; AL: $F(1,92)=353.61$, $p<0.01$]. Two of the subjects showed reliable condition X time interactions [ES: $F(2,76)=15.71$, $p<0.01$; AL: $F(2,92)=33.74$, $p<0.01$]. Subject AL's peak velocities were higher in the later perturbation conditions whereas ES showed the opposite pattern. The duration of the lower lip lowering movements increased in the perturbed trials for all subjects [ES: $F(1,76)=10.64$, $p<0.01$; SN: $F(1,90)=51.87$, $p<0.01$; AL: $F(1,92)=149.74$, $p<0.01$]. Upper lip raising movements from closure differ in peak velocity for all three subjects. In general, the peak velocity of upper lip raising was higher in the perturbed conditions than in the control trials [ES: $F(1,76)=4.83$, $p<0.05$; SN: $F(1,90)=38.62$, $p<0.01$; AL: $F(1,92)=8.8$, $p<0.01$]. Subject ES showed a condition X time interaction for peak velocity [$F(2,76)=10.38$, $p<0.01$) because the average peak velocity in the early perturbation condition was lower than the control value. In contrast, displacement and duration of the upper lip raising movement did not show any systematic patterns across subjects. The jaw peak velocity generally increased in the perturbed conditions. Subjects SN and AL produced jaw lowering movements at a significantly higher velocity [SN: $F(1,90)=7.98$, $p<0.01$; AL: $F(1,92)=24.4$, $p<0.01$]. For subject AL this effect increased in size for the later perturbation conditions [condition X time interaction, $F(2,92)=4.86$, $p<0.01$].

The duration of the jaw movement was significantly shorter for the perturbed conditions for all three subjects [ES: $F(1,76)=56.15$, $p<0.01$; SN: $F(1,90)=11.62$, $p<0.01$; AL: $F(1,92)=6.88$, $p<0.05$]. Subject ES showed a condition X time interaction [$F(2,76)=18.78$, $p<0.01$]. No systematic patterns were observed across subjects for jaw lowering displacement.

C. Laryngeal response to perturbation

Two aspects of the laryngeal response to the lip perturbation were examined. Folkins and Abbs (1975) and Shaiman and Abbs (1987) reported that lip perturbation prior to oral closure caused voicing to be extended. The increase in vowel duration reported above for subjects SN and AL is consistent with this finding. Figure 2 shows the interval from the onset of the vowel to the onset of laryngeal abduction. As can be seen in this figure, in the three cases in which the perturbation onsets occurred during the vowel (early for AL, early and mid for SN), the laryngeal opening onset was delayed. (Note that the interval shown in Fig. 2 is not necessarily the same as the acoustic vowel duration. Subject ES, for example, consistently begins abduction after oral closure.)[2]

The second set of analyses examined the time course of the laryngeal gestures. The effect of the perturbation, in general, was to increase the duration of the glottal cycle. If we look at the abduction versus adduction movements, we see that this lengthening of the cycle is almost totally due to increases in the laryngeal adduction duration. As can be seen in Fig. 3, the three subjects showed no reliable differences in the duration of the abduction gestures between the perturbed and control trials. For some of the conditions this may be a relatively trivial finding since torque onset occurred after or late in the abduction gesture in some

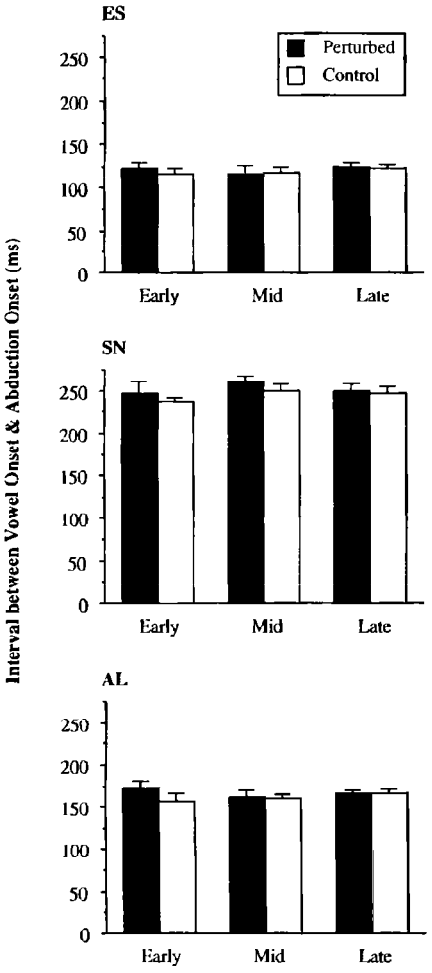

FIG. 2. The interval from vowel onset to onset of glottal abduction for loaded and control trials. Standard errors are shown for each mean.

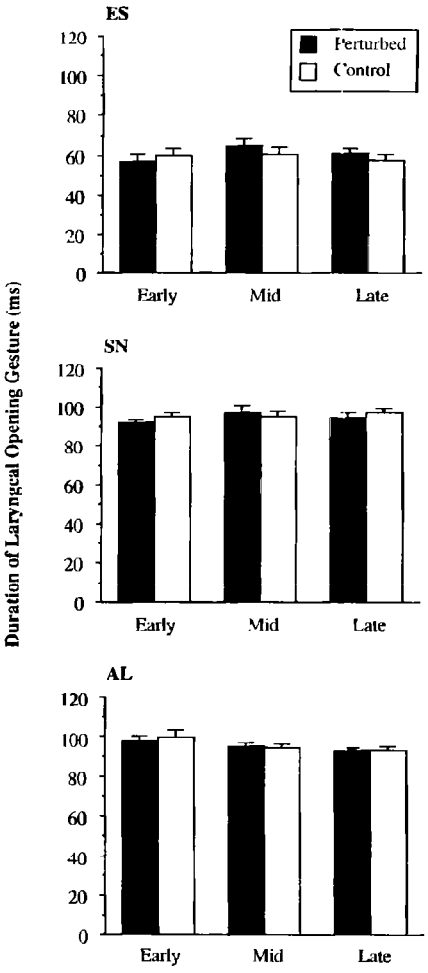

FIG. 3. Duration of the laryngeal abduction (open ng) gesture for loaded and control trials for all three subjects. Standard errors are shown for each mean.

trials. However, the failure to observe differences in this gesture even in subject SN's and AL's early perturbations suggests that the timing of the perturbation onset is not the sole explanation of this pattern. On the other hand, laryngeal adduction was longer in duration in the perturbed trials than in the control trials [ES: $F(1,74)=8.75$, $p<0.01$; SN: $F(1,90)=6.57$, $p<0.05$; AL: $F(1,92)=33.75$, $p<0.01$]. This can be seen in Fig. 4. Note that the load onset timing has no strong influence on the magnitude of the laryngeal closing change. Although transillumination is an uncalibrated signal, we examined the peak glottal opening magnitude in the various conditions. No reliable differences in glottal aperture size were observed as a function of perturbation condition or perturbation onset timing.

D. Interarticulator timing

The relative timing of the laryngeal and oral gestures was assessed in a number of ways. The onset of laryngeal adduction (peak glottal opening) was measured relative to the onset of the acoustic release burst for the first /p/. It was found that adduction in the perturbed conditions began later than in the control trials [ES: $F(1,76)=48.63$, $p<0.001$; SN: $F(1,90)=26.98$, $p<0.001$; AL: $F(1,92)=280.6$, $p<0.001$]. In the control conditions, the laryngeal adduction onset preceded the oral release while in the perturbed conditions the adduction onset occurred closer to or after release (see Fig. 5).

To assess the oral kinematics we examined the movements of the upper and lower lips and in addition we calculated a measure of oral aperture. The measure of oral aperture which we used was the vertical distance between the upper and lower lip ireds. The onset of oral aperture opening relative to the acoustic release burst varied as a function of the perturbation [ES: $F(1,76)=23.17$, $p<0.01$; SN: $F(1,90)=46.49$, $p<0.01$; AL: $F(1,92)=9.9$, $p<0.01$). As can be seen in Fig. 6, this interval is shorter in the perturbed conditions. This indicates that the acoustic release occurred a shorter interval of time after the oral opening movements began in the perturbed trials than in the control trials.

The analysis of the timing of oral movement onsets relative to the laryngeal movement onsets shows a less consistent pattern than is observed for the relation of the oral and laryngeal kinematics to the release burst. The interval between the onset of glottal adduction and the onset of oral aperture opening showed no systematic patterns across

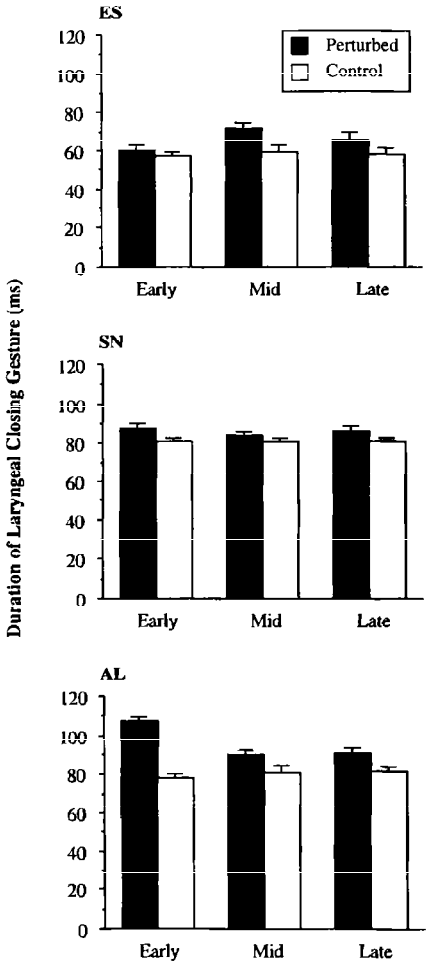

FIG. 4. Duration of the laryngeal adduction (closing) gesture for loaded and control trials for all three subjects. Standard errors are shown for each mean.

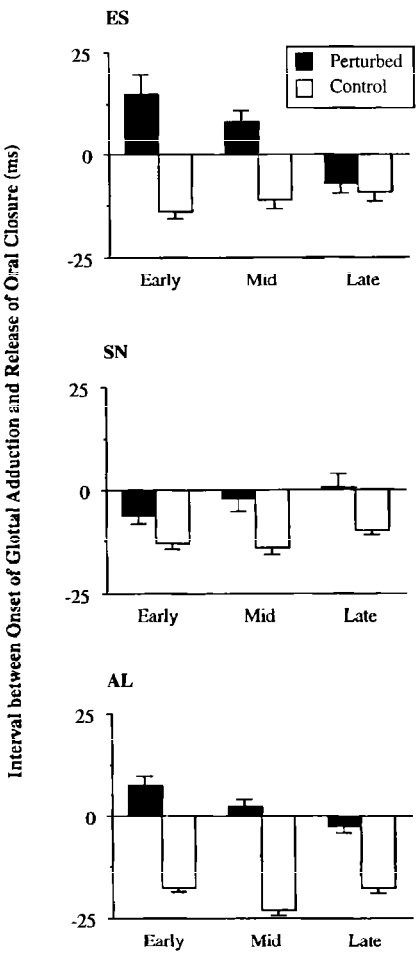

FIG. 5. Duration of the interval between the onset of glottal adduction and the oral release for all three subjects. Standard errors are shown for each mean. A positive value indicates that the glottal event occurs later than the acoustic release.

subjects as a function of the perturbation. Subjects ES and AL showed condition X time interactions [ES: $F(2,76)=5.36$, $p<0.01$; AL: $F(2,92)=3.28$, $p<0.05$] caused by the longer intervals in some perturbed conditions (ES: early and mid; AL: mid). However, the data for this interval are quite variable (Fig. 7). The intervals between the onset of glottal adduction and the onsets of upper and lower lip movements from closure also show a variable pattern. For two of the subjects (ES and AL), the onset of glottal adduction occurs closer to the onset of upper lip raising in the perturbed conditions than in the control conditions [ES: $F(1,76)=18.58$, $p<0.01$; AL: $F(1,92)=44.05$, $p<0.01$]. For subject SN there is no consistent pattern. The onset of lower lip lowering precedes glottal adduction for all subjects in all conditions; however, there are no systematic effects as a function of the perturbation condition.

E. Oral air pressure

Intraoral pressure values were obtained at three points in the utterance: (1) the offset of visible vibration in the pressure signal for the first vowel; (2) the onset of vibration in the pressure signal for the second vowel; and (3) peak pressure during the closure for the first /p/. Table IV shows these values for the three subjects.[3] None of the subjects showed reliable differences between the perturbed and control conditions. The pressures at vowel offset and vowel onset do not differ as a function of the perturbation conditions for any of the subjects. As has been reported previously (Hirose and Niimi, 1987), the pressure at voicing offset is higher than at voicing onset.

F. First perturbed trials

For two of the three subjects (AL, ES) some of the initial reactions to the perturbations were not complete compensations. In subject ES's first perturbed trial he produced what looked like a glottal stop at the onset of the second /p/ in /i'pip/ and maintained a closed glottis and open mouth until the torque went off.[4] At this point he finished the utterance without making a laryngeal gesture for the second /p/. In the second perturbed trial, a more normal closure duration was observed, however no laryngeal gesture was observed for the second /p/. Subject AL also showed some disorganization during the first perturbations. In particular, during the second loaded trial he released the oral closure with an appropriate phasing be-

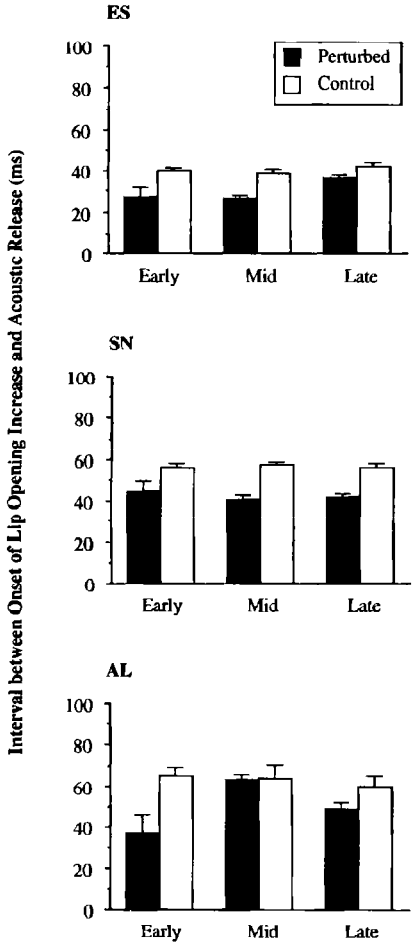

FIG. 6. Duration of the interval between the onset of oral aperture opening and the acoustic release in ms. Standard errors are shown for each mean. In all cases the onset of movement preceded the acoustic release.

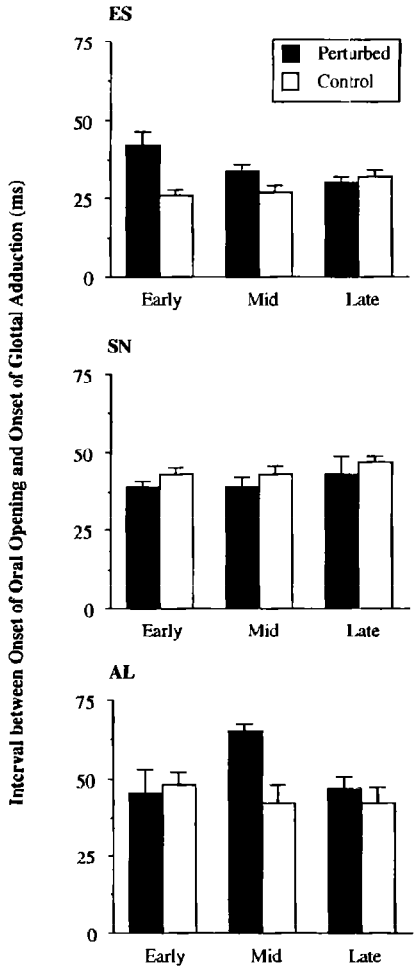

FIG. 7. Duration of the interval between the onset of oral aperture opening and the onset of glottal adduction in ms. Standard errors are shown for each mean. In all cases the onset of oral aperture opening preceded the onset of laryngeal adduction.

tween the oral and laryngeal gestures but then closed the lips again; these maneuvers resulted in an abnormally long VOT. On the other hand, subject SN who had participated in a related experiment and thus had some experience with the perturbations did not produce such behaviors on the first perturbed trials.

III. DISCUSSION

The results of the study indicate some laryngeal reaction following load onset to the lower lip. The laryngeal responses to the lip perturbations took two forms. First, there was a delay in onset of glottal abduction when the load was introduced before the offset of the preceding vowel. This effect accounts for the increase in the duration of the preceding vowel and is in agreement with the results presented by Folkins and Abbs (1975) and Shaiman and Abbs (1987). Second, the glottal abduction–adduction cycle increased due to an increase in the adduction phase; the abduction phase did not change in duration. These results indicate that in some very general way the articulators are coupled during speech production. While this finding might be expected from the tight timing between the two articulatory systems during normal speech, it extends the results of other perturbation studies to these remote articulators.

There appear to be oral responses to the perturbations as well. In most cases the lips and jaw system overcame the load allowing the production of an oral closure and thus the buildup of oral pressure. This is evident from the pressure measurements presented in Table IV. For two of the subjects (SN and AL) there was some evidence of adjustment in the upper lip closing movement in response to the lower lip perturbation. The displacement and peak velocity of upper lip lowering movements increased in the perturbed conditions. This pattern of behavior is in agreement with other studies using a similar experimental paradigm (cf., Folkins and Abbs, 1975; Abbs and Gracco, 1984; Kelso *et al.*, 1984).

The pattern of variability in the oral compensation produced by different subjects is consistent with the findings presented by Gracco and Abbs (1985). In the Gracco and Abbs study, the timing of the onset of the load to the lower lip was shown to influence the magnitude of the lower lip compensation. For early perturbations the lower lip made a larger contribution to the compensation than

TABLE IV. Pressure measurements. Intraoral pressure values at the moment of offset of voicing of the first vowel, at peak intraoral pressure during the intervocalic /p/, and at the moment of onset of voicing following the intervocalic /p/. The standard deviations are in brackets. Note that subject ES's data are presented in arbitrary units.

Condition	Pressure offset	Peak pressure	Pressure onset
ES			
Early perturbation	671.3 (228.6)	1002.6 (203.2)	299.9 (183.8)
Control—early	624.8 (180.4)	1164.8 (168.2)	358.1 (168.8)
Midperturbation	685.9 (247.4)	1192.4 (139.0)	389.0 (356.7)
Control—mid	596.9 (200.2)	1242.8 (287.1)	433.1 (224.8)
Late perturbation	680.8 (180.5)	1159.3 (224.0)	294.2 (147.0)
Control—late	609.1 (174.2)	1217.8 (265.1)	387.8 (145.4)
SN			
Early perturbation	11.5 (7.9)	12.7 (7.8)	6.8 (7.6)
Control—early	11.6 (7.4)	12.8 (7.4)	6.4 (7.4)
Midperturbation	11.4 (7.4)	12.5 (7.3)	6.6 (7.2)
Control—mid	11.4 (7.3)	12.6 (7.4)	6.2 (7.6)
Late perturbation	11.5 (7.2)	12.5 (7.1)	6.3 (7.4)
Control—late	11.6 (7.2)	12.7 (7.2)	6.7 (7.2)
AL			
Early perturbation	10.6 (6.7)	13.1 (7.0)	9.1 (7.2)
Control—early	12.2 (7.4)	14.2 (7.2)	10.0 (7.3)
Midperturbation	11.7 (8.0)	13.7 (7.9)	9.1 (7.9)
Control—mid	11.6 (7.9)	13.7 (7.9)	9.2 (7.7)
Late perturbation	11.4 (7.0)	13.3 (6.8)	8.9 (6.7)
Control—late	11.6 (6.6)	14.0 (6.7)	9.0 (6.5)

for later perturbations. In the present study, the three subjects received the loads at different times relative to oral closure. For subject SN the load onsets were delivered early relative to oral closure. In this subject's data, changes were observed in both the upper and lower lip motions compared to the control trials. For subject AL the load onsets occurred quite late relative to oral closure. This subject shows no lower lip response and upper lip adjustment only for the earlier load onsets. Subject ES received loads quite close to the onset of oral closure and thus showed no oral adjustments.

In spite of evidence of articulatory adjustments, the present results show significant acoustic differences between the loaded trials and control trials for the duration of the vowel preceding the stop, for stop closure duration, and for VOT. If the effectiveness of the compensations are judged by their acoustic consequences, it is obvious that these compensations are not always effective. Although we have not tested whether the perturbed and control productions could be distinguished by listeners, our impression is that this would indeed be the case. The increase in VOT in the perturbed condition is substantial in most cases.

The nature of the observed adjustments. What are we to make of this pattern of response to the perturbation? There is clear evidence of laryngeal compensation but does the behavior at the larynx make sense functionally? It is conceivable that the delay in onset of glottal abduction following the perturbation was produced in order to preserve oral–laryngeal timing. A delay in the onset of oral closure is apparent in the three conditions in which the load onset occurred during the vowel (early and mid for SN and early for AL). A delay in onset of glottal abduction could preserve the articulatory timing at both closure onset and stop release. From an acoustic point of view, the timing of oral and laryngeal articulations at these two points in time during stop production is particularly critical.

While the delayed glottal abduction may thus be functional in preserving articulatory timing at the onset of stop closure, it is obvious that this adjustment did not preserve interarticulator timing throughout the production of the stop. In particular, the glottis began to close later with respect to oral release in the perturbed trials. This produces the observed increase in VOT for the perturbed trials (Löfqvist and Yoshioka, 1984). Moreover, the duration of the glottal adduction phase is also longer in the perturbed trials. This adds further to the increase in VOT.

The change in timing between peak glottal opening and oral release is produced by the change in closure duration. The shortened oral closure associated with the perturbed productions can be accounted for by the mechanical effects of the increased load on the lower lip;[5] that is, the load stayed on for the duration of the trial and caused the lower lip to descend earlier and more quickly than in the control trials. The shorter interval between the onset of oral aperture opening and the acoustic release burst is consistent with this idea. The peak velocity of the jaw, lower lip and upper lip release movements were also generally higher in the perturbed trials. The load could indirectly influence the upper lip kinematics by altering the contact force between the lips and the amount of lip compression during closure.

The relation between the onset of laryngeal adduction and the various oral kinematic measures was quite inconsistent. In part, this can be attributed to sources of variability in the measurements of lip movement. The contact forces at the lips can cause compression in both upper and lower lips and it may be impossible to distinguish independent motion of the two lips. This makes the upper and lower lip movement onsets confounded when the movements are from the closure position. Second, the placement of the ireds at the vermilion border of the lips may allow some motion at the point of contact between the two lips without corresponding motion at the ired. Finally, the load itself may be responsible for venting the closure at the point of contact of the paddle with the lips. The latter factor would produce changes in closure duration that were not strongly related to changes in the timing of oral movement onset.

The first trial reactions in the two subjects who were new to the paradigm warrant consideration.[6] There are many possible explanations for the difference between the present results and results reported in other experiments. The load rise time used in this experiment was very fast and presumably outside the physiological range. This could have influenced the results. There clearly must be natural limits on the extent to which compensations are achievable. If the loads are too great or if the load onset is temporally too close to a critical speech event, the subject will not be able to compensate. However, this explanation seems unlikely. The load magnitude used in the present experiment was similar to that used by other researchers.

Further, Gracco (1984) varied the rise time of the load, including values similar to that used here and found no effect for different rise times. Other technical differences exist between the present study and previous work though it is not clear which differences may be important. For example, the lip paddle used in the present experiment had a smaller surface area in contact with the lower lip than the device used by Gracco.

The difference between the first trial compensation in our study and the responses reported in previous papers certainly deserves further research. One of the priorities in this area should be to understand what is meant by compensation under these perturbation conditions. Little of the research on dynamic perturbations has assessed the acoustic or perceptual consequences in any way other than online judgments during the actual experiment. This casual assessment of the perceptual effects presumably produces a broad tolerance in the assessment of compensations. Even if more systematic analyses were performed, however, it is not clear what acoustic or perceptual evidence should be used to assess the adequacy of compensation. There is little agreement in the perceptual research community on the objects of speech perception (Diehl and Kluender, 1989; Fowler, 1989). Further, Crystal and House (1988) have shown that in more natural speech many stop consonants are produced in an incomplete fashion. In their corpus only 59% of stops included both an occlusion and plosive portion.

The coordination of the larynx and oral articulators observed in the present experiment is consistent with the idea that the independent articulators in speech are controlled as functional units or coordinative structures. In this view, groups of muscles and articulators act synergistically to achieve phonetic goals. The motor control units in speech are not just the patterns of activity required to move the articulators involved in the production of a specific phonetic segment. Rather, the units of speech are frameworks that specify the couplings between the articulators required to produce that phonetic segment (e.g., Saltzman and Munhall, 1989). It is hypothesized that the movements of individual articulators within a coordinative structure are adjusted in response to perturbations so that the goal of the coordinative structure is achieved. This cooperative behavior provides flexibility in articulation through a backdrop of task specific reflexes. The delay in the onset of glottal abduction that was observed for two of the subjects is consistent with this depiction of speech coordination. However, the failure of the larynx to initiate adduction movements earlier in response to the early oral release and the increased duration of the laryngeal adduction are less easily understood within this framework.

In summary, the results of the present study indicate that the laryngeal articulatory movements can be rapidly altered when a load is applied to the lip during voiceless stop production. While this finding is not completely unexpected given the tight coupling between oral and laryngeal articulators during normal speech, it extends the perturbation paradigm to spatially remote articulators. At the same time the results suggest some limitations on functional responses to maintain interarticulator timing in the face of mechanical perturbations. In particular, the proper phasing of the oral and laryngeal articulations at stop release was disrupted when a load was applied to the lower lip, resulting in an increased VOT in the perturbed productions.

ACKNOWLEDGMENTS

This work was supported by NIH Grants DC-00865 and RR-05596 to Haskins Laboratories, in part by ESPRIT-BR Project 6975—Speech Maps through Nuter Grant P55-1, and a grant from the Natural Sciences and Engineering Research Council of Canada to the first author. Helpful comments on earlier drafts were made by Vince Gracco and John Folkins.

[1]The New York Times (26 October 1986) reported a more "natural" perturbation incident that occurred during a performance of the opera "Tosca." Eva Marton, a soprano, was about to sing her aria, "Vissi d'arte" when a large baritone caught her in the jaw with his elbow. Ms. Marton's jaw was dislocated and she was unable to open or close her mouth. Undaunted, she sang the aria. According to Ms. Marton, "I shaped the vowels with my tongue."

[2]It would be important to know whether the onsets of the movements of the oral articulators were also delayed by the perturbations. If this was observed it would indicate that the oral and laryngeal articulators are being adjusted in a similar manner. However, too few load onsets occurred prior to the onsets of upper lip, lower lip, and jaw movements to examine this possibility in the present data set.

[3]Because of problems with the calibration signal for subject ES's pressure signal, the data for this subject are presented in arbitrary units.

[4]This response was so extreme that the trial was excluded from statistical analyses of the data.

[5]The mechanical interpretation of the shortened closure may not be as simple as it first seems. If the change in closure duration is caused by the presence of the load this would imply a time-varying pattern of resistance to the load. The oral system seems able to overcome the onset of the load and still make a bilabial closure while the oral system must be unable to resist the load following the onset of closure and thus the lower lip is pulled down too early. Why there should be this difference is unclear.

[6]It should be noted that subject SN and two other subjects in a previous study failed to show first trial compensation when the lower lip was perturbed during the production of /bæb/. All three subjects in this study failed to produce a good closure on the initial perturbed trial.

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