In: Bell-Berti, F., & Raphael, L. J. (1995). Producing Speech: Contemporary Issues. For Katherine Safford Harris. AIP Press: New York.

31 On the Dynamics of Temporal Patterning in Speech

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With deep respect and affection for Katherine S. Harris, and her fascination with processes of durational change in the production of speech.

I. INTRODUCTION

What are the dynamics that underlie the temporal cohesion among the gestural or segmental components of a given speech utterance? The present chapter describes recent results from an ongoing series of studies at Haskins Laboratories that has begun to provide answers to this question (e.g., Kollia, 1994; Löfqvist & Gracco, 1991; Munhall, Löfqvist, & Kelso, 1994; Saltzman, 1992; Saltzman, Kay, Rubin, & Kinsella-Shaw, 1991; Saltzman, Löfqvist, Kinsella-Shaw, & Rubin, 1992; see also Gracco & Abbs, 1989, performed prior to the first author's move to Haskins). These studies employ a paradigm in which mechanical perturbations are applied to the articulatory periphery, typically the lower lip or jaw, during spoken utterances in order to probe the functioning of the speech production apparatus. The resultant changes in the temporal structure of the utterances are used to provide information about the dynamics of intergestural timing. For example, transient mechanical perturbations delivered to the speech articulators during repetitive speech sequences (Saltzman, 1992; Saltzman et al., 1991), or to the limbs during unimanual rhythmic tasks (Kay, 1986; Kay, Saltzman, & Kelso, 1991), can alter the underlying timing structure of the ongoing sequence and induce systematic shifts in the timing of subsequent movement elements. As will be elaborated shortly in greater detail, these data imply that the relative phasing of speech gestures is not rigidly specified over a given sequence. Rather, such results suggest that gestural patterning evolves fluidly and flexibly over the course of an ongoing sequence, governed by an intrinsic intergestural dynamics. Furthermore, these data suggest that the intergestural dynamical system functions as a sequence-specific central timing network that does not simply drive the articulatory periphery in a unidirectionally coupled manner. Rather, central and peripheral dynamics are coupled bidirectionally, so that feedback information from the articulatory periphery can influence the state of the central "clock."

There are two important methodological points to be made regarding this type of study. First, it is important to analyze extended, repetitive sequences, e.g., /...pæpæpæ.../, using steady-state, *phase-resetting* techniques. These methods were pioneered in studies of the effects of perturbations on the temporal structure of general biological rhythms (e.g., Glass & Mackey, 1988; Kawato, 1981; Winfree, 1980). In particular, such analyses are used to determine whether perturbations delivered during an ongoing rhythm have a permanent effect (i.e., phase shift) on the underlying temporal organization of the rhythm. Phase-resetting techniques have been used in many kinematic and neurophysiological studies of the control and coordination of rhythmic movements (e.g., Lennard & Hermanson, 1985; Lee & Stein, 1981). In such studies, what is measured is the amount of temporal shift introduced by the perturbation, relative to the timing pattern that existed prior to the perturbation. This phase shift is measured after the transient, perturbation-induced distortions to the rhythm have subsided, and the system has returned to its pre-perturbation, steady-state rhythm. The finding

of a post-perturbation, steady-state phase shift using this method would support the hypothesis that there exists a central timing network that both drives the articulatory periphery and whose state is altered (phase-shifted) by feedback specific to events at the periphery. A further, crucial aspect of the phase-resetting methodology is that, across trials, perturbations are delivered so as to sample all phases of the utterance's repeated syllable "cycle," e.g., from maximum lip opening for one instance of /æ/ to the next in /...pæpæpæ.../. The complete analysis thus consists of testing for post-perturbation, steady-state phase shifts for all phases of perturbation delivery, in order to examine the variation, over the course of the syllable, in the sensitivity or receptivity of the central clock to peripheral, perturbation-induced events.

It is important to note that relatively lengthy, repetitive utterances are required for the steady-state, phase-resetting technique, in order to be able to distinguish temporal articulatory distortions that are attributable to central resetting processes from those that are attributable to the systematic yet transient behavior of the articulatory periphery. The utterances must be repetitive, since the units of analysis are cycles and, by definition, successive cycles must be approximately identical. The utterance must be relatively long since, even in the minimum cycle-period case, where each cycle is only one syllable long, one needs: a) a steady-state measure of pre-perturbation behavior that includes approximately 5–10 cycles; b) 1–2 (occasionally 3) more cycles during which the perturbation is applied, at least in our previous experiments (Saltzman, 1992; Saltzman et al., 1991) and in the experiment reported below; c) several more cycles (occasionally none) in order to settle back to within a criterion degree of closeness to the pre-perturbation behavior; and d) approximately 2 – 10 cycles to provide a steady-state measure of post-perturbation behavior.

The second methodological point hinges on the fact that normal speech does not consist of extended, rhythmic repetitions of a single syllable. Therefore, in order to be sure that central phase shifts identified using phase-resetting techniques actually reflect processes governing normal utterances, it is necessary to bridge the theoretical gap between phase-resetting results and those obtained from perturbing discrete, word-like sequences, e.g., /pəsæpæpl/. Because of the relatively short duration of such discrete sequences, the system cannot be relied upon to settle down and to "shake off" the effects of the perturbation in the time between the offset of the perturbation and the end of the utterance. In effect, one can reliably study only the transient responses to perturbations in such sequences. Thus, in order to relate steady-state, phase-resetting data meaningfully to transient data obtained by perturbing discrete utterances, it is necessary to study the transient responses of *repetitive* as well as discrete utterances, preferably using a within-subject experimental design. Once the relation between the steady-state and transient patterns is understood for the repetitive data, a conceptual link can be forged between the transient patterns of

the repetitive and discrete data, and shared dynamical principles governing articulatory behavior can be identified.

Earlier work using this experimental paradigm has focused on intergestural timing changes that occur across syllables between successive bilabial closing gestures for /p/ during both the discrete utterance /pəsæpæpl/ and the repetitive utterance /...pæpæpæ.../ (Saltzman, 1992; Saltzman et al., 1991). The data described below represent preliminary results from a study (Saltzman, Löfqvist, Kinsella-Shaw, & Rubin, in preparation) that generalizes this approach to laryngeal devoicing gestures as well as to bilabial gestures. In addition, the study examines these same utterances for changes in intergestural timing that occur intrasegmentally between the bilabial closing and laryngeal devoicing gestures for /p/, and across syllables between successive laryngeal devoicing gestures for /p/. This study consists of data from two subjects, each of whom participated in two sessions. In each session, subjects produced alternating blocks of the repetitive sequence /...pæpæpæ.../ and the discrete sequence /pəsæpæpl/.

The preliminary data described below are of the first session's repetitive data for one subject (ES), and include 70 perturbed and 16 control trials. The data for the discrete sequence spoken by this subject (both sessions) have been described elsewhere (Saltzman et al., 1992), and are used here for comparisons with the repetitive data.

II. GENERAL EXPERIMENTAL METHODS: REPETITIVE DATA

A. Equipment and Data Processing

The subject sat in an adjustable dental chair, with his head restrained in an external frame. A small paddle connected to a torque motor was placed on the lower lip with a tracking force of 3 gm, in order to deliver step pulses of downward force (50 gm) at random times during the experimental trials. Timing of perturbation onset was controlled by a VAXstation II/GPX. Oral articulatory movements were measured optoelectronically using infrared light-emitting diodes mounted on the upper lip, lower lip, lip paddle, nose (the nose LED acted as a spatial reference), and a custom-made jaw splint. Laryngeal abduction and adduction movements were recorded using a transillumination technique in which a fiberoptic endoscope was introduced through the nose and placed in the pharynx in order to illuminate the larynx. The amount of light passing through the glottis, which depends of the degree of laryngeal opening, was detected by an optical sensor placed on a neck collar just below the cricoid cartilage. During the experimental session, the transilluminated larynx was displayed on a video monitor to ensure that the view of the larynx was unobstructed and that the endoscope's lens was not fogged. The acoustic speech signal and control voltage

applied to the torque motor were recorded. All data were fed into a 16-track FM tape recorder for later digitization and signal processing.

B. Protocol

Twelve blocks of 25 trials were performed during each of two sessions. Each session lasted approximately 3 hours each. Blocks alternated between *repetitive* and *discrete* experimental conditions. In the discrete condition, each trial consisted of the sequence /pəsæpæpl/; in the repetitive condition, each trial consisted of a sequence of approximately 20-30 repetitions of the syllable /pæ/, spoken at a syllable rate comparable to that used in the discrete trials. Details of the protocol for the discrete-blocks condition are described in Saltzman et al. (1992); details of the protocol for the repetitive-blocks condition are described below.

For the repetitive blocks, perturbations were delivered during a random sampling of 80% of the trials; perturbation duration was preset in an external timing circuit to equal the subject's average syllable duration measured during pretest repetitive trials. On each perturbation trial, the perturbation was delivered during the *nth* syllable (*n* varied randomly from 8-11), and after *m%* of the predetermined syllable duration (*m* varied randomly from 1-100). Task instructions were to not actively resist the perturbation, and to return to a steady rhythm similar to that produced before the perturbation as quickly and easily as possible.

C. Definition of Cycle Types

Bilabial and laryngeal movements were analyzed using a lip-aperture (LA) trajectory that was defined by subtracting the upper lip signal (UL) from the lower lip signal (LL), i.e., LA = LL – UL (Figure 1, top panel), and a glottal opening trajectory that was defined using the transillumination signal (Figure 1, middle panel). Individual cycles were then defined between successive peak openings, and four cycle types were identified:

a) *pre-perturbation* cycles included the trial's first cycle through the last cycle before perturbation onset;
b) *perturbation* cycles included all cycles that overlapped the perturbation interval;
c) *transient* cycles were defined as those cycles following the perturbation during which cycle periods deviated from the average pre-perturbation cycle period by more than approximately 2.5 standard deviations; and
d) *post-return* cycles were defined from the last transient cycle to the end of the trial.

Analyses were limited to a maximum of 20 cycles for any given trial.

III. BILABIAL DATA

A. Phase-resetting Analyses

Cycle phase, ϕ, was defined to be zero at all peak bilabial openings. For all other points between peak openings, phase was defined as (t / T_i), where t is the time (in secs) from the most recent peak preceeding a given event of interest, and T_i is the period (in secs) of the cycle containing the event. Thus, phase values of events occurring in a given cycle were defined in a normalized range from zero to one. In the perturbation trials, the phase of perturbation delivery was defined with respect to the time of perturbation offset. This offset time served as a temporal anchoring point for "strobing" both backward and forward in time into the pre-perturbation and post-return cycle sequences, respectively, using the average pre-perturbation cycle period to define the strobe period. The within-cycle strobe phases from the pre-perturbation and post-return cycles were then averaged to define an *average old phase*, ϕ_{old}, and *average new phase*, ϕ_{new}, respectively. *Phase shift*, $\Delta\phi$, was then defined as $(\phi_{new} - \phi_{old})$ (modulo 1). Thus, $\Delta\phi$ is the amount that a given trial's post-return rhythm has been shifted relative to its pre-perturbation rhythm ($\Delta\phi > 0$ denotes phase advance; $\Delta\phi < 0$ denotes phase delay). The same measures were obtained for the control (no perturbation) trials, where calculations were anchored to the end of a randomly timed, but not delivered, "phantom perturbation."

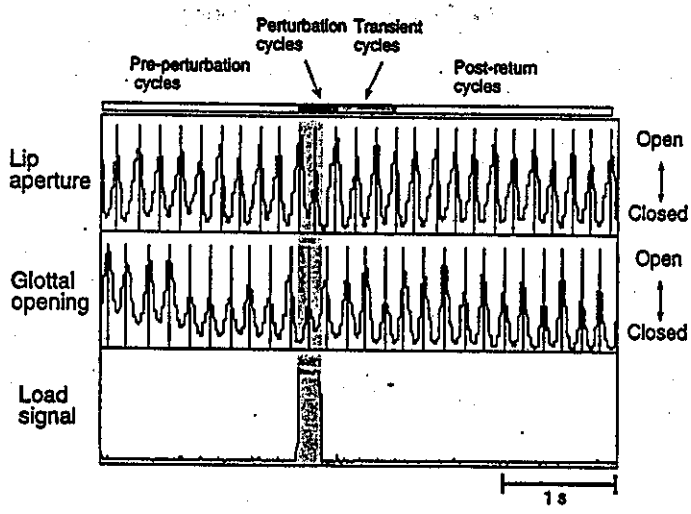

FIGURE 1. Data trajectories for a single repetitive trial: lip aperture (top panel), glottal opening (middle panel), and torque load signal (bottom panel). The sign of the loading signal is inverted in the figure to emphasize that the downward force on the lower lip acted to increase lip aperture. The boxes above the top panel mark the different cycle types for the trial.

Figure 2 illustrates the results of our analyses (one subject, one session), using $\Delta\phi$ data from the perturbation trials that were binned and averaged according to intervals of ϕ_{old}. For the control trials, $\Delta\phi$ data were simply pooled across all old phases of the "phantom perturbations." T-tests were computed for each bin to test whether the perturbation-induced phase shifts differed from control values. To adjust for an elevated Type I error rate due to multiple comparisons, α - levels were selected by dividing 0.01 and 0.05 by the number of comparisons made. As can be seen in the figure, the rhythm showed a phase advance in the 0.2–0.4 interval that was significantly different from the no-perturbation control trials ($p < 0.05$). This pattern replicates the bilabial phase-resetting results found in earlier studies (Saltzman, 1992; Saltzman et al., 1991), and lends further support to the hypothesis that central intergestural dynamics are sensitive to appropriately timed mechanical perturbations of the articulatory periphery, and that such events can permanently reset the rhythms of such central "clocks."

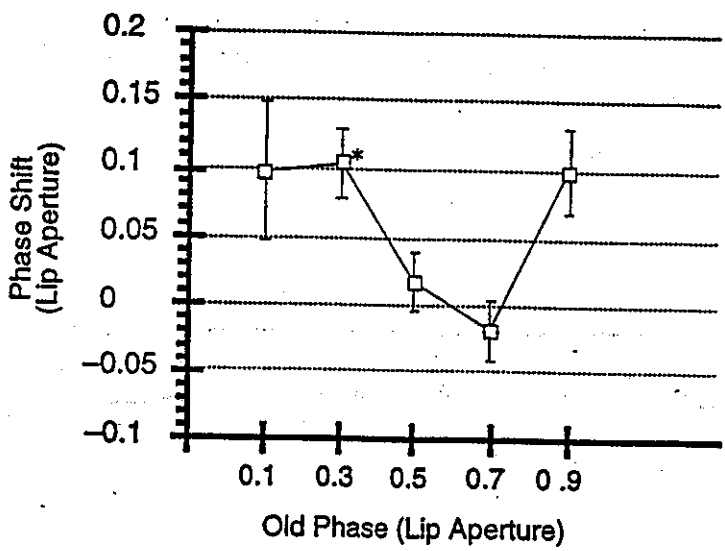

FIGURE 2. Phase shifts ($\Delta\phi$) of lip aperture trajectories (y-axis), binned and averaged according to average old phase (ϕ_{old}) values (x-axis). Bin labels represent the centers of these bins. Each point represents the average phase shift associated with a given bin. Error bars denote standard errors.

B. Transient Analyses

What are the origins of these steady-state phase shifts? One simple hypothesis is that most of these shifts are induced during the application of the perturbation. Thus, we focused next on the timing/phasing changes that occur during the first and second *perturbation cycles* (i.e., the first two cycles that overlapped the perturbation interval; see Figure 3), and their relation to steady-state phase-resetting. As with the phase-resetting analyses described earlier, control trial values were calculated for first and second "perturbation cycles" that were defined by randomly timed, but not delivered, "phantom" perturbations.

First perturbation cycle

For each trial, the duration of the bilabial first-perturbation cycles (dur_1) was normalized with respect to the session's average control "dur_1" values, using the formula (experimental – $\overline{control}$ / $\overline{control}$). Figure 4 (open squares) displays these duration change data in percentage form after binning and averaging according to the time of perturbation onset, which was normalized using the formula: (perton – t_{on_1}) / $\overline{prepert}$, where perton = onset time of perturbation, t_{on_1} = onset time of first-perturbation bilabial cycle, and $\overline{prepert}$ = the average pre-perturbation bilabial cycle duration for the trial. Protected t-tests indicated that cycle durations were significantly shortened relative to controls for the first ($p < 0.01$) and second ($p < 0.05$) bins, and significantly lengthened for the final bin ($p < 0.01$).

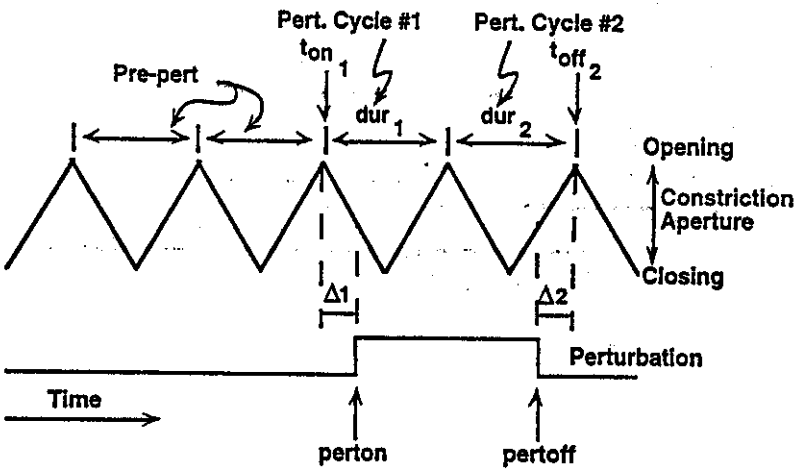

FIGURE 3. Schematic display of constriction (bilabial or glottal) aperture and perturbation trajectories for preperturbation cycles, and for perturbation cycles #1 and #2.

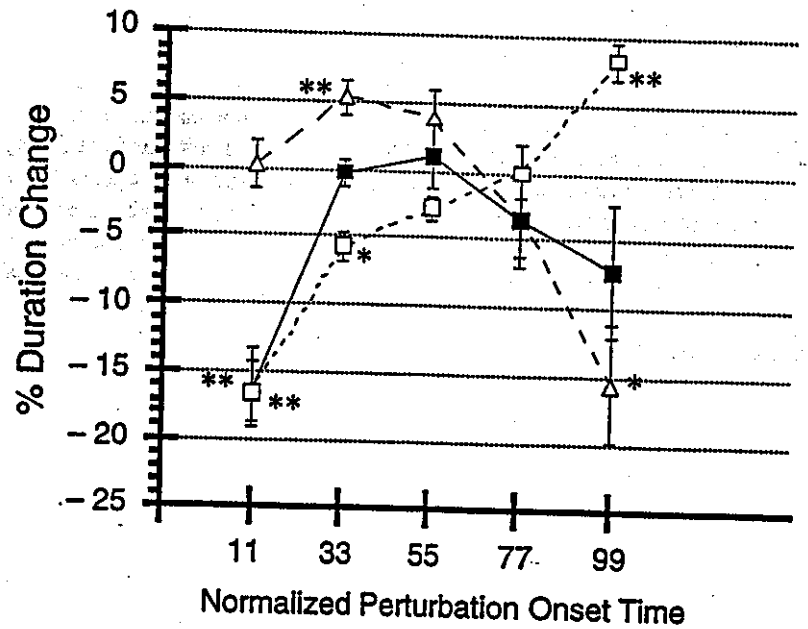

FIGURE 4. Percent durational change (100 × [experimental − $\overline{\text{control}}$] / $\overline{\text{control}}$) for the lip aperture trajectories' first perturbed cycles (open squares), second perturbed cycles (open triangles), and first-plus-second perturbed cycles (closed squares), binned and averaged according to normalized perturbation onset time (100 × [perton − t_{on_1}] / $\overline{\text{prepert}}$). Bin labels represent the centers of these bins. Error bars denote standard errors.

Second perturbation cycle

For each trial, the duration of the second-perturbation bilabial cycle (dur_2) was similarly normalized with respect to the session's average control "dur_2" values, using the formula (experimental − $\overline{\text{control}}$) / $\overline{\text{control}}$. Figure 4 (open triangles) displays these data after binning and averaging according to the same time base used for the first-perturbation bilabial cycle. Protected t-tests indicated that cycle durations were significantly lengthened relative to controls for the second ($p < 0.01$) and final ($p < 0.05$) bins, and significantly shortened for the final bin ($p < 0.01$).

C. Relationship between Steady-state and Transient Data

In order to test the hypothesis that the phase shifts observed in the steady-state were attributable to duration changes induced during the first two perturbation cycles, we first summed the normalized durations for each trial's first- and second-perturbed bilabial cycles, and binned these values using the same time base as in the previous separate analyses for these cycles (see Figure 4, filled squares). Protected *t*-tests indicated that the summed perturbation cycle

durations were significantly different (shorter) than the corresponding nonperturbation controls only in the first bin ($p < 0.01$).

We then rebinned, reanalyzed, and replotted the steady-state phase shifts using this same normalized time base (as opposed to the $\overline{\phi_{old}}$ time base originally used earlier in Section III.A; see Figure 2). In Figure 5, we have compared the steady-state data curve (open triangles) with the summed perturbation cycles' curve (open squares); these curves are virtually mirror images of each other, with significant effects only in the first bin. Given the opposite sign conventions used to define phase shifts and duration changes, these curves are in essence identical, indicating that the steady-state phase shifts are indeed induced in the first two perturbation cycles.

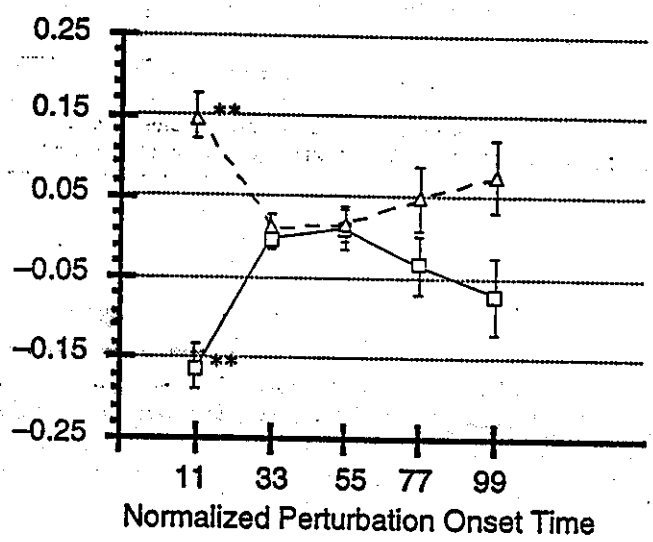

FIGURE 5. Lip aperture trajectory phase shifts ($\Delta\phi$: open triangles) and fractional duration changes ([experimental − $\overline{\text{control}}$] / $\overline{\text{control}}$: open squares) for the first-plus-second perturbed cycles (y-axis), binned and averaged according to normalized perturbation onset time ($100 \times$ [perton − t_{on_1}] / $\overline{\text{prepert}}$) (x-axis). Bin labels represent the centers of these bins. Error bars denote standard errors.

IV. LARYNGEAL DATA

A. Phase-resetting Analyses

For the laryngeal trajectories, cycle type definition, phase-shift computation, binning and averaging of the $\Delta\phi$ data according to intervals of $\overline{\phi_{old}}$, as well as statistical testing, proceeded as with the lip aperture analyses. Figure 6 shows that the laryngeal rhythm showed a steady-state phase advance in the 0.6 – 0.8 $\overline{\phi_{old}}$ interval that was significantly different from the no-perturbation control trials ($p < .01$).

Note that the sensitive old phase intervals differed for the laryngeal (Figure 6) and the bilabial (Figure 2) data. This was simply because the laryngeal and bilabial cycles are themselves out of phase with one another (see the data trajectories in Figure 1). As will be shown below in Section V.A, when both phase shifts are plotted on a common time base, it becomes clear that the phase shift behaviors of lips and larynx are virtually identical.

B. Transient Data; Relationship between Steady-state and Transient Data

As with the bilabial data, we computed and summed the normalized duration changes for each trial's first- and second-perturbed laryngeal cycles, and binned these values using the normalized time of perturbation onset for the trial. Again, as with the bilabial data, time normalization used the formula (perton – t_{on_1}) / $\overline{prepert}$, where perton = onset time of perturbation, t_{on_1} = onset time of the first-perturbation laryngeal cycle, and $\overline{prepert}$ = the average duration of the laryngeal pre-perturbation cycles for the trial. Protected t-tests indicated that the summed perturbation cycle durations were significantly different (shorter) than the corresponding nonperturbation controls only in the third bin ($p < 0.01$; see Figure 7, open squares).

In order to test the hypothesis that the phase shifts observed in the steady-state were attributable to duration changes induced during the first two perturbation cycles, we rebinned, reanalyzed, and replotted the *steady-state* phase shifts using the same normalized time base (as opposed to the $\overline{\phi_{old}}$ time base originally used above in Section IV.A; see Figure 6), and compared the steady-state data curve (Figure 7, open triangles) with the summed perturbation cycles' curve (Figure 7, open squares). As with the earlier bilabial analyses, these laryngeal curves are virtually mirror images of each other with significant effects only in the third bin. Again, given the opposite sign conventions used to define phase shifts and duration changes, these curves are in essence identical, indicating that the steady-state phase shifts are indeed induced in the first two perturbation cycles.

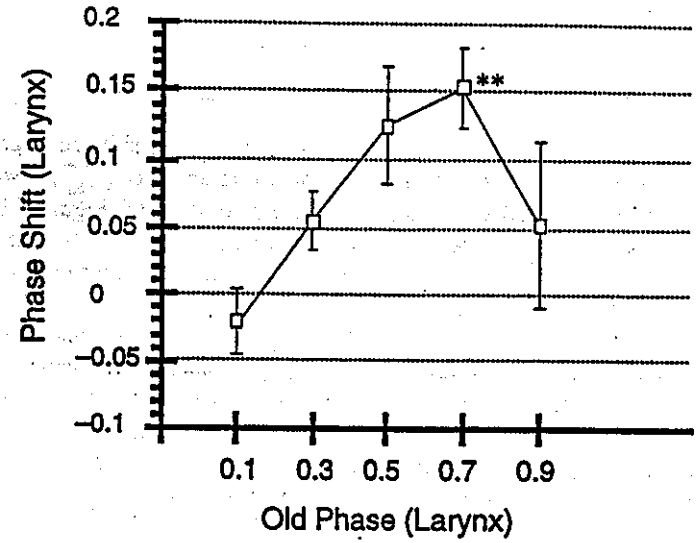

FIGURE 6. Phase shifts ($\Delta\phi$) for the laryngeal opening trajectories (y-axis) binned and averaged according to average old phase ($\overline{\phi_{old}}$) values (x-axis). Bin labels represent the centers of these bins. Error bars denote standard errors.

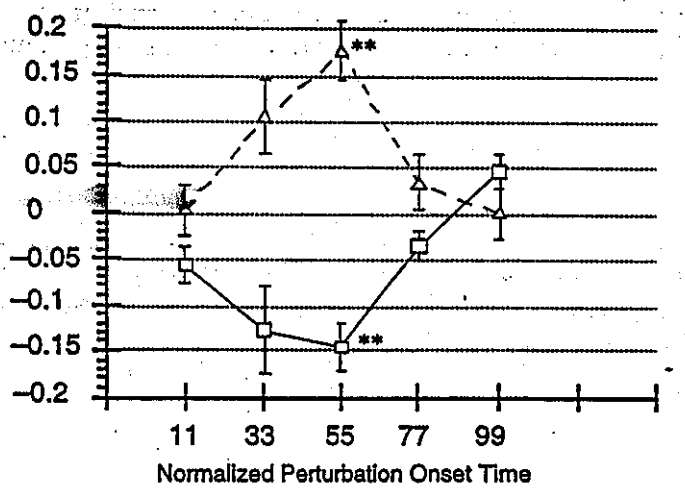

FIGURE 7. Laryngeal opening phase shifts ($\Delta\phi$: open triangles) and fractional duration changes ([experimental − $\overline{\text{control}}$] / $\overline{\text{control}}$: open squares) for the first-plus-second perturbed cycles (y-axis), binned and averaged according to normalized perturbation onset time ($100 \times$ [perton − t_{on1}] / $\overline{\text{prepert}}$) (x-axis). Bin labels represent the centers of these bins. Error bars denote standard errors.

V. LIP-LARYNX ANALYSES

A. Steady-State Shifts in Relative Phase between Bilabial and Laryngeal Trajectories

The evolution of the relative phase of the bilabial and laryngeal trajectories during each trial was measured in the following manner. Successive peak laryngeal openings were used to define "strobe" events in each corresponding bilabial cycle. Relative phase for each laryngeally-strobed bilabial cycle could then be defined by:

$$\frac{\left[\begin{array}{c}(\text{time of the } i^{\text{th}} \text{ laryngeal event}) \\ -\ (\text{time of the preceding bilabial peak})\end{array}\right]}{(\text{period of the strobed bilabial cycle})}$$

Average relative phase for each trial's strobed bilabial pre-perturbation cycles was defined computationally as [bilabial $\overline{\phi_{old}}$ – laryngeal $\overline{\phi_{old}}$]; average relative phase for the strobed bilabial postreturn cycles was defined computationally as [bilabial $\overline{\phi_{new}}$ – laryngeal $\overline{\phi_{new}}$]. Steady-state shifts in relative phase for each trial were then defined by the average postreturn relative phase minus the average pre-perturbation relative phase.

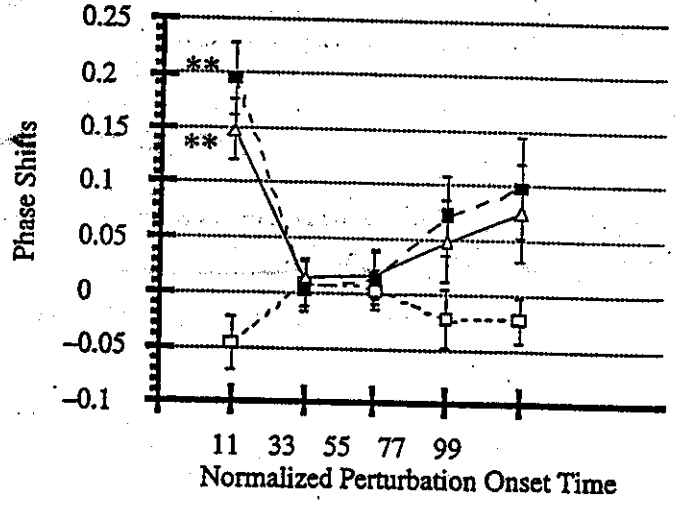

FIGURE 8. Phase shifts for laryngeal opening (closed squares), lip aperture (open triangles), and lip-larynx relative phase (open squares), binned and averaged according to normalized perturbation onset time ($100 \times [\text{perton} - t_{on_1}] / \overline{\text{prepert}}$). Bin labels represent bin centers. Error bars denote standard errors.

For ease of comparison with the results of the previous sections, we binned the data from each trial according to a normalized time base defined by the formula (perton − t_{on_1}) / $\overline{prepert}$, where perton = onset time of perturbation, t_{on_1} = onset time of the first-perturbation bilabial cycle, and $\overline{prepert}$ = the average duration of the bilabial pre-perturbation cycles for the trial. These binned data were averaged and tested against the corresponding mean value obtained in the control trials. As can be seen in Figure 8 (open squares), the perturbations did not induce any significant steady-state shifts in the relative phasing of lips and larynx. This makes sense since, as can also be seen in the figure, the individual phase-resetting behavior of the lips (open triangles) and larynx (filled squares) is virtually identical. When perturbations are delivered at the system's sensitive phase (i.e., the leftmost bin), the bilabial and laryngeal gestures appear to be phase-advanced as a relatively coherent unit, maintaining their relative phasing while they are advanced in absolute time.

B. Transient Data

The transient behavior of the relative phasing between bilabial and laryngeal trajectories was also examined. Transient shifts in relative phase for each trial were defined by the average relative phases computed for the first- and second-perturbed bilabial cycles, minus the average relative phases computed for the pre-perturbation bilabial cycles. We binned the data from each trial according to the same normalized time base used for the steady-state relative phase analyses. These binned data were averaged and tested against the corresponding mean value obtained in the control trials. There were no significant shifts in relative phase induced in the transient cycles (see Figure 9). These data indicate that even while a perturbation is being applied, the system acts so as to maintain the integrity of the bilabial-laryngeal intergestural unit.

VI. COMPARISONS OF REPETITIVE AND DISCRETE DATA

What are the relationships between the patterns described above for productions of the rhythmic, repetitive sequence /...pæpæpæ.../, and those found for productions of the discrete sequence /pəsæpæpl/ (Saltzman et al., 1992)? Because of their very nature, the discrete sequences cannot be counted upon to settle back to a steady-state after perturbation delivery. Consequently, comparisons must be made using transient methods of analysis.

The two measures studied in the discrete sequences that are the most comparable to those studied in the repetitive sequences are: a) the duration of the so-called *vowel cycle* defined between successive maximum bilabial openings for /æ/ in /pəsæpæpl/. Its analog in the repetitive analyses is the second-

perturbation cycle; and b) the relative phase of the laryngeal peak inside the bilabial vowel cycle for devoicing the medial /p/. Its repetitive analog is the lip-larynx relative phase for the second-perturbation cycle. For historical reasons (e.g., Gracco & Abbs, 1989), we analyzed perturbation-induced changes in the duration of the discrete vowel cycle by binning the normalized duration change values according to a normalized time base defined by the formula (pertoff – t_{offd}) / $\overline{control_d}$, where pertoff = offset time of perturbation, t_{offd} = offset time of discrete bilabial cycle, and $\overline{control_d}$ = average duration of discrete bilabial cycles from the unperturbed control trials. The comparable time interval in the repetitive data is (pertoff – t_{offr}) / $\overline{control_r}$, where t_{offr} is the offset time of the second-perturbation cycle, and $\overline{control_r}$ is the average duration of unperturbed control second-perturbation cycles.

Comparisons between the discrete (open triangles) and repetitive (open squares) patterns are presented in the following two figures. The changes in bilabial cycle duration are comparable (Figure 10), showing significant lengthening and shortening, respectively, in the earlier and later time bins. The discrete relative phasing data shows significant increases in all bins, whereas the repetitive data show only a nonsignificant trend toward such increases (Figure 11).

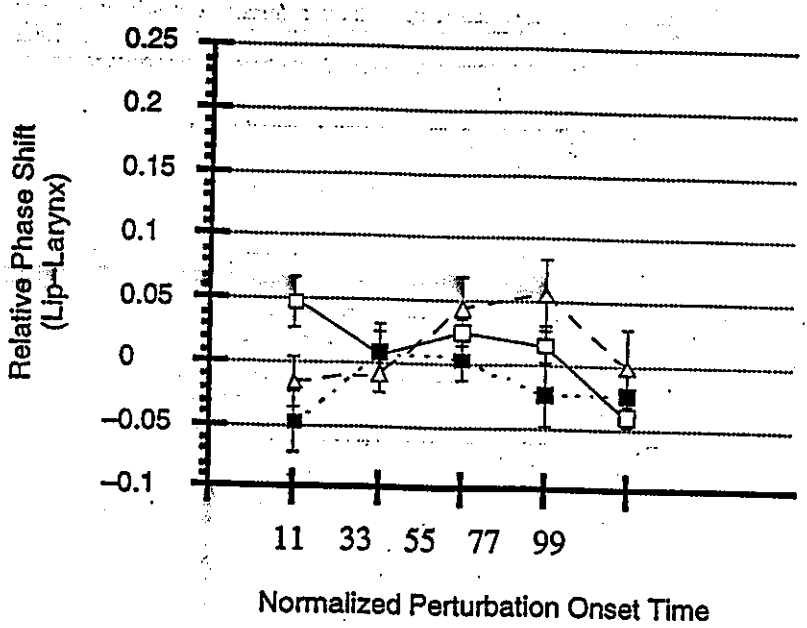

FIGURE 9. Shifts in lip-larynx relative phase for first-perturbed cycles (open squares), second-perturbed cycles (open triangles), and post-return cycles (closed squares), binned and averaged according to normalized perturbation onset time (100 × [perton – t_{on1}] / $\overline{prepert}$). Bin labels represent bin centers. Error bars denote standard errors.

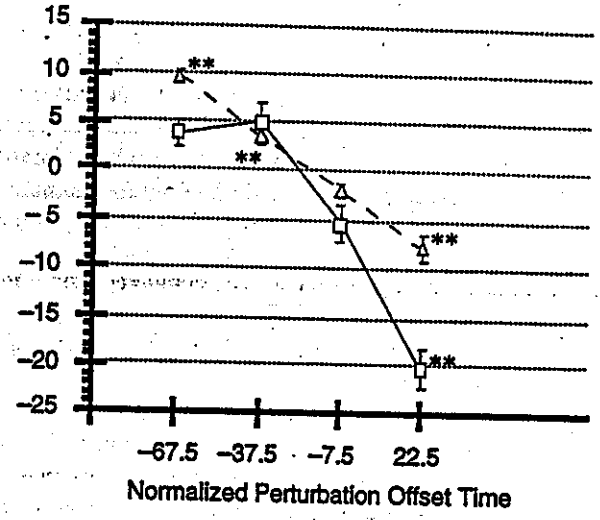

FIGURE 10. Normalized duration changes of lip aperture syllable (discrete data: open triangles; defined by [100 x {experimental − $\overline{\text{control}_d}$} / $\overline{\text{control}_d}$]) and second-perturbed cycle (repetitive data: open squares; defined by [100 x {experimental − $\overline{\text{control}_r}$} / $\overline{\text{control}_r}$]), binned and averaged according to their respective normalized time bases ([100 × {pertoff − t_{off_d}} / $\overline{\text{control}_d}$] for discrete data; [100 × {pertoff − t_{off_r}} / $\overline{\text{control}_r}$] for repetitive data). Bin labels represent bin centers. Error bars denote standard errors.

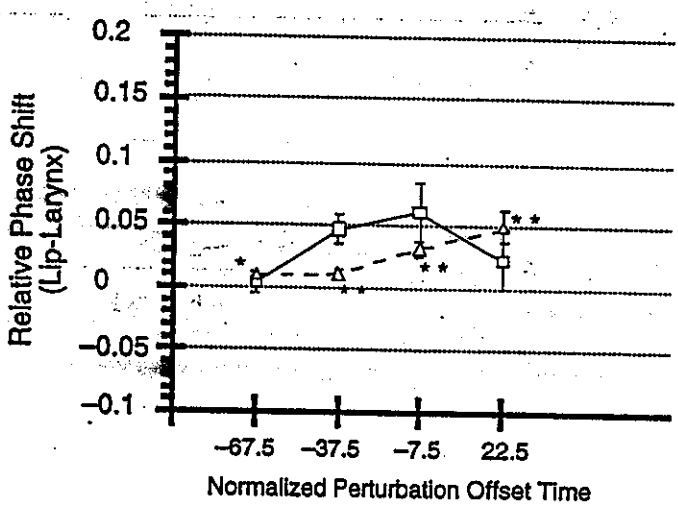

FIGURE 11. Shifts in lip-larynx relative phase for lip aperture syllable (discrete data: open triangles) and second perturbed cycle (repetitive data: open squares), binned and averaged according to their respective normalized time bases ([100 × {pertoff − t_{off_d}} / $\overline{\text{control}_d}$] for discrete data; [100 x {pertoff − t_{off_r}} / $\overline{\text{control}_r}$] for repetitive data). Bin labels represent bin centers. Error bars denote standard errors.

VII. DISCUSSION/CONCLUSIONS

The data described above provide several hints regarding the nature of the dynamics that underlie intergestural temporal cohesion, both within segments and between successive syllables. First, the steady-state phase shifts induced by the perturbations during the repetitive utterances (Sections III.A & IV.A) provide further support for the hypothesis that central intergestural dynamics can be "permanently" reset by peripheral articulatory events. These effects occur, however, only when the perturbation is delivered within a particularly "sensitive phase" of the cycle. During this period, the downwardly directed lower lip perturbation is opposing the just-initiated, actively controlled bilabial closing gesture for /p/. If one assumes that efferent commands to the periphery are strongest at gestural initiation, then these results imply that the timecourse of afferent sensitivity mirrors that of efferent strength.

The second hint is provided by the transient analyses of the repetitive utterances (Sections III.B and IV.B). These analyses indicate that, although systematic durational changes are induced in the first two perturbed cycles when perturbations are delivered during the utterances' nonsensitive phases, these changes are simply transient peripheral responses to the perturbation and do not indicate central phase-resetting. For example, Figure 4 shows how a perturbation-induced shortening of the first-perturbed cycle in bin 33 is cancelled by a lengthening of the following second-perturbed cycle; similarly, a lengthening of the first-perturbed cycle in bin 99 is cancelled by a shortening of the following second-perturbed cycle. These systematic but transient durational changes can be understood as cases in which the offset time of the second-perturbed cycle (t_{off_2} in Figure 3) is not altered by the perturbation, although the time of the preceding peak opening is either advanced (bin 33) or delayed (bin 99).

Relatedly, the perturbation-induced steady-state phase shifts are almost totally attributable to changes occurring during the first two perturbed cycles. For example, Figure 4 shows how a perturbation-induced shortening of the first-perturbed cycle in the utterance's sensitive phase bin (bin 11) is followed by no change in the second-perturbed cycle. Thus, the steady-state phase shift can be understood as a case in which the offset time of the second-perturbed cycle (t_{off_2} in Figure 3) and the times of the immediately preceding and all subsequent peak openings are advanced by the same amount. Methodologically, this result suggests that in order to distinguish centrally from peripherally induced durational changes in perturbed speech sequences, it is probably necessary to examine durational changes in at least two successive articulatory intervals (syllables?).

Third, steady-state and transient analyses of the repetitive data both support the hypothesis of greater intergestural temporal stability within segments/phonemes than between syllables. This conclusion is based on the existence of comparable steady-state phase resetting curves for the bilabial and

laryngeal trajectories (Figure 8), and the absence of shifts in the relative phasing of lips and larynx in the both the steady-state and in the first- and second perturbed cycles (Figure 9). Thus, even when the bilabial and laryngeal gestures of the /p/s in successive syllables are shifted in time relative to one another by the perturbation, they are shifted as a coherent unit.

Finally, comparisons of the discrete and repetitive data suggest that intergestural temporal cohesion between successive syllables is comparable in the two types of utterance (Figure 10). However, intergestural cohesion within segments/phonemes may be greater for the repetitive sequences (Figure 11). This difference may be attributable to the sustained, rhythmic nature of the repetitive sequences and/or to differences in the within-syllable position of the bilabial stop /p/ in the repetitive (syllable initial position) and discrete (syllable final position) sequences. Differentiating among these possibilities is a task for future research.

ACKNOWLEDGMENTS

This work was supported by grant support from the following sources: NIH Grant #DC-00121 (Dynamics of Speech Articulation) and NSF Grant #DBS-9112198 (Phonetic Structure Using Articulatory Dynamics) to Haskins Laboratories.

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