1987: Motor and Sensory Processes of Language (pp. 163-201)
edited by E. Keller & M. Gopnik
Hillsdale, NJ:
Lawrence Erlbaum Associates Inc

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7 Programming and Execution Processes of Speech Movement Control: Potential Neural Correlates

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ABSTRACT

Explicit consideration of nervous system anatomy and physiology underlying speech and language is critical to provide a more concrete basis for linguistic and behavioral theories of communication. The purpose of the present paper is to provide a data-based neuroanatomical model for the nervous system actions associated with the motor programming and execution of speech movements. Recent studies indicate that speech motor control requires the integration of multiple sensory signals with internally specified, general motor goals. Results from these recent investigations of speech sensorimotor control and considerations of neuroanatomical and neurophysiological findings from nonhuman primates offer some specific hypotheses regarding underlying nervous system operations. In particular, it is possible to evaluate the speech motor programming and execution contributions of the premotor, primary motor, and supplementary motor cortical areas, and the inputs to these important cortical sites from the cerebellum, basal ganglia, and other cortical regions. Although this model is based on data obtained primarily from the perioral region and its CNS representations, the hypotheses provided are sufficiently basic to reflect general operations of the nervous system in this critically human function.

INTRODUCTION

Scientists investigating the processes underlying human speech and language behavior face a difficult problem. The superficial manifestations of oral communication can be observed rather directly. However, the underlying neural processes, seemingly critical to optimal understanding, are almost wholly opaque. Although it is possible to draw certain inferences concerning these neural processes from speech and language deficits associated with focal brain damage, these inferences are limited inherently by the rapid and sub-

stantial reorganizational processes that occur with loss of nervous system tissue (cf. Asanuma & Arissian, 1984; Glassman, 1978; Laurence & Stein, 1978). To overcome these difficulties, multiple and often complementary approaches have evolved. Commonly, these approaches include:

1. Identification of various hypothetical constructs or abstractions from linguistic or information processing models,
2. Classification of oral communication patterns in relation to those constructs, and
3. Interpretation of those patterns (or deficits thereof) to hypothesized subcomponents of the unobservable processes.

The constructs or abstractions commonly employed include planning, programming, serial and parallel processing, parsing, syntax, phonology, modularity, etc. Implicit in the use of such hypothetical constructs or abstractions is the assumption that they reflect essential aspects of underlying nervous system organization and function. Indeed, one major difference among the various theoretical and methodological approaches to understanding speech and language behavior appears to be the degree to which hypotheses and dependent measures explicitly reflect extant knowledge of the nervous system. From our perspective, explicit consideration of nervous system anatomy and physiology is critical if we are to make long-term progress in this area. Without this constraint, it is all too easy to conjure up a large number of equally plausible, abstract hypotheses to explain a given set of communication behaviors or deficits thereof. Despite the intellectual exercise that is provided by such effects, one must recognize that little may be gained in understanding the critical nervous system functions for speech and language.

In the study of speech motor control and nervous system control of movement in general, some hypothetical constructs also guide the ongoing research. However, the primary goal is focused more explicitly on determining functional brain behavior relations. For example, most investigators recognize that the overall motor control process includes several stages or levels. These stages commonly are identified as planning, programming, and execution involving what are thought to be distinct operations occurring prior to or during generation of a motor output. While definitions of these constructs often are operationalized and the terms are not always used uniformly, there is basic agreement regarding their importance and their general sequence within the motor act. Moreover, active research is under way in many laboratories to determine the neurophysiological correlates of these processes. Planning, for example, is considered an early process preceding programming, primarily involving processes that include general identification of motor goals (Paillard, 1983). By contrast, the programming process appears to include the selection and adjustment of the nervous system

circuits required to achieve the precise muscular, kinematic, and temporal requirements for the intended motor act (Paillard, 1983; Schmidt, 1982). Finally, the execution process is thought to involve the actual generation of the final descending neural signals, including their moment-to-moment shaping by continuous afferent input.

In this context, one purpose of the present paper is to review some recent physiological data from speech motor control which appear to reflect two of these hypothesized processes: motor programming and execution. As suggested by the present authors (cf. Abbs, 1986; Abbs, Gracco, & Cole, 1984) in speech motor control, these two processes would seem to lie immediately downstream from linguistic planning stages and hence reflect the implementation of phonological goals. In contrast to much of the work on neural mechanisms underlying the upstream language processes, these data on speech motor programming and execution were obtained in normal subjects, utilizing techniques adapted from studies of limb motor control in waking animals and man. Moreover, based upon these experimental data and previous neuroanatomic and neurophysiologic studies in nonhuman primates, it is possible to consider the neural structures that might underlie the processes of speech motor programming and execution. As such, a second purpose of this paper is to refine the neuroanatomical and neurophysiological foundation for further investigations of speech motor control, speech neuropathologies, and voluntary motor behavior in general. In consideration of these neuroanatomical and neurophysiological data, we feel that the advantages of incorporating extant biological data into models of speech and language processes will be apparent.

FUNCTIONAL SENSORIMOTOR PROCESSES IN SPEECH MOVEMENT CONTROL

A fundamental premise of the present paper is that the generation of movements for speech involves the continuous utilization of sensory information from the muscle receptors and cutaneous mechanoreceptors that are distributed throughout the respiratory, laryngeal, and orofacial systems. The suggestion that sensory information is continuously utilized does not imply that this is the sole means by which speech movements are generated. Rather, recent data indicate that the rich supply of orofacial, respiratory, and laryngeal afferents continually interact with central operations (e.g., programs) to yield the speech movement patterns associated with oral communication. In the discussion immediately following, we will review briefly the various data that address this pivotal point and illustrate how it is possible to document the critical operation of sensorimotor control processes by selectively perturbing speech movements and observing the muscle and movement "corrections" that occur in response to those induced errors.

The control of multiarticulate movements such as speech demands the temporal spatial interaction of multiple structures. As such, controlling the movements of speech is not a unidimensional process, but must also include the coordination among multiple structures necessary for this skilled motor behavior. Recent results have implicated the contribution of afferent-dependent mechanisms in both the control of individual speech movements and in the coordination among them (Abbs & Gracco, 1984; Folkins & Abbs, 1975, 1976; Gracco & Abbs, 1985; Kelso, Tuller, Bateson, & Fowler, 1984). These studies have demonstrated the presence of task-dependent, functionally organized, short latency compensatory responses to unanticipated mechanical perturbations applied prior to or during a speech movement. Comparable sensorimotor actions have been demonstrated for other complex motor behaviors; e.g., rapid postural adjustments (Marsden, Merton, & Morton, 1981; Nashner & Cordo, 1981; Nashner, Woollacott, & Tuma, 1979), compensatory eye-head interactions (Bizzi, Kalil, & Tagliasco, 1971; Morasso, Bizzi, & Dichgans, 1973), wrist-thumb actions (Traub, Rothwell, & Marsden, 1980), and thumb-finger coordination (Cole, Gracco, & Abbs, 1984).

These recent results are in contrast to earlier investigations of sensory contributions to speech motor control that primarily involved experimental interference with various afferent inputs. For example, following anesthetic reduction in oral sensation, global measures indicate that overall speech production capability is disrupted only in subtle ways (Gammon, Smith, Daniloff, & Kim, 1971; Ringel & Steer, 1963; Scott & Ringel, 1971). Additionally, reduced or distorted auditory information results in only mildly distorted speech motor output (Kelso & Tuller, 1983; Lane & Tranel, 1971). Estimates of afferent-to-efferent neural transport delays via analyses of reaction times, coupled with the apparent ballistic nature and short duration of many speech movements, have also led some to the position that speech movements are primarily preprogrammed; i.e., sensory information is used only in long-term adaptation or speech skill acquisition (cf. Borden, 1979; Keller, this volume; Kent & Moll, 1975; see, however, Cole & Abbs, 1983). From this alternate perspective, speech movements would be generated from preset motor patterns or programs and executed independently of any afferent information. Similar theoretical positions have been postulated from limb studies demonstrating that functionally deafferented animals (Fentress, 1973; Polit & Bizzi, 1979; Taub & Berman, 1968) and man (Rothwell, Traub, Day, Obeso, Thomas, & Marsden, 1982) are capable of executing certain learned motor tasks (cf. Marsden, Rothwell, & Day, 1984 for review). One must be cautious in the interpretation of motor performance observed under conditions of sensory deficit. Such observations are not interpretable in relation to what the removed portion of the nervous system does, but rather what the rest of the nervous system does without the part in question (cf. Asanuma &

Arissian, 1984; Berenberg, 1984; Dostrovsky, Millar, & Wall, 1976; Glassman, 1978). Furthermore, recent studies have shown that movements executed in the absence of afferent information often are only grossly normal, essentially lacking their normal precision (Sanes & Evarts, 1983). Finally, more natural, multijoint behaviors appear more disrupted by reduced afferent input than stereotypic behaviors or movements around a single joint (Bossom, 1974; Polit & Bizzi, 1979; Rothwell et al., 1982). Although it is apparent that the observations in sensory deficit studies cannot be interpreted easily, the results do indicate that certain motor tasks can be carried out, in a somewhat crude manner, despite reduced or absent afferent input. Hence, these data suggest that the nervous system is capable of prespecifying some muscle contraction and movement parameters (i.e., as in a "generalized motor program," cf. Schmidt, 1982), albeit in a somewhat imprecise form.

Integrating these latter findings into a theory of speech motor coordination and control thus requires the inclusion of an interactive process between ascending afferent signals and a generalized motor program. As suggested by Abbs et al. (1984), afferent information may be used to refine certain parameters of an abstract motor program in relation to the varying state of the periphery, yielding a more specific and detailed set of motor commands for actual motor execution (also cf. Abbs, 1979; MacNeilage, 1980). Support for this position has come from recent studies in which distinctly different compensatory response patterns are observed at different times prior to and during the motor execution of a speech movement gesture (Gracco & Abbs, 1982a, 1982b, 1985), seemingly reflecting the differential contribution of afferent mechanisms in the programming and execution of speech movements.

Prior to considering these most recent data, it is helpful to review the most salient findings of previous studies specifying the basic sensorimotor processes of speech. Our major approach to this problem has involved the adaptation and refinement of what has been shown to be a powerful technique available for investigation of sensorimotor mechanisms in human subjects—the unanticipated perturbation paradigm. Small, precisely controlled errors are introduced during the movement for a particular voluntary gesture, and the resulting muscle activity and movement changes are analyzed to determine the nature of the underlying sensorimotor actions. If applied carefully, this approach is not susceptible to confounding problems of adaptation and compensation associated with many other paradigms for evaluating sensory contributions; e.g., local anesthesia, masking of auditory feedback, fixing the jaw, etc. Additionally, unanticipated perturbation in conjunction with single neural unit recording has been used extensively in awake animals to determine the nervous system pathways and processes accompanying sensorimotor control in nonhuman primates (Conrad, 1978; DeLong, Alexander, Georgopoulos, Crutcher, Mitchell, & Richardson, 1984; Evarts &

Fromm, 1978; Evarts & Tanji, 1976; Georgopoulos, Kalaska, Caminiti, & Massey, 1983; Tatton & Bawa, 1979; Thach, 1978). These latter data support the contribution of ascending afferent signals to the motor control functions of the primary motor cortex, the somatic sensory cortex, the basal ganglia, the cerebellum, and as such permit more meaningful interpretation from parallel studies in man.

Application of the unanticipated perturbation technique in the investigation of speech motor control processes has revealed that when a small mechanical perturbation is applied to one speech structure, compensatory adjustments are observed in both the perturbed structure as well as the coactive unperturbed structures (Folkins & Abbs, 1975, 1976; Folkins & Zimmermann, 1982; Gracco & Abbs, 1985; Kelso et al., 1984). For example, if a perturbation is introduced to the lower lip during the elevation for a bilabial stop, compensatory adjustments are observed in both the upper lip and jaw. These data imply that sensory information is used not only to correct errors in individual movements, but also to make adjustments among the multiple movements involved in a given speech motor gesture; the latter observation thus suggests that sensorimotor actions contribute also to the coordination of speech movements. Compensatory adjustments in perturbed and unperturbed coactive structures are illustrated in Fig. 7.1; perturbation applied to the lower lip results in lower lip as well as upper lip adjustments. These upper and lower lip adjustments to lower lip perturbation have been defined previously as reflecting afferent-dependent open loop and closed loop control processes, respectively (Abbs & Gracco, 1984) or more recently as nonautogenic and autogenic sensorimotor actions (Abbs et al., 1984). That is, adjustments observed in the perturbed structure are designated as autogenic, while compensatory adjustments in a coactive but unperturbed structure are designated as nonautogenic. The terms autogenic and nonautogenic adjustments may be preferred because they do not limit the conceptualization of the underlying neural processes to extant engineering control schemes.

The responses presented in Fig. 7.1 and to be discussed subsequently were obtained during rapid lip closure for the production of a bilabial speech sound. Subjects were not aware of the purpose of the experiments and showed these compensations the first time they experienced a perturbation. The subjects' task consisted of sustaining the vowel *ae* and, upon hearing a tone, to close their lips as rapidly as possible and generate a *b*. Once the bilabial stop was produced, subjects resumed producing the vowel, and responded again when the tone was heard. Effectively, subjects were producing rapid lip closure from a static posture. Unanticipated perturbations were delivered to the lower lip on approximately 15% of the trials within a restricted time interval, as shown in Fig. 7.2; this 100 ms target interval was chosen for introduction of the perturbations to minimize contamination of natural control processes by voluntary adjustment.

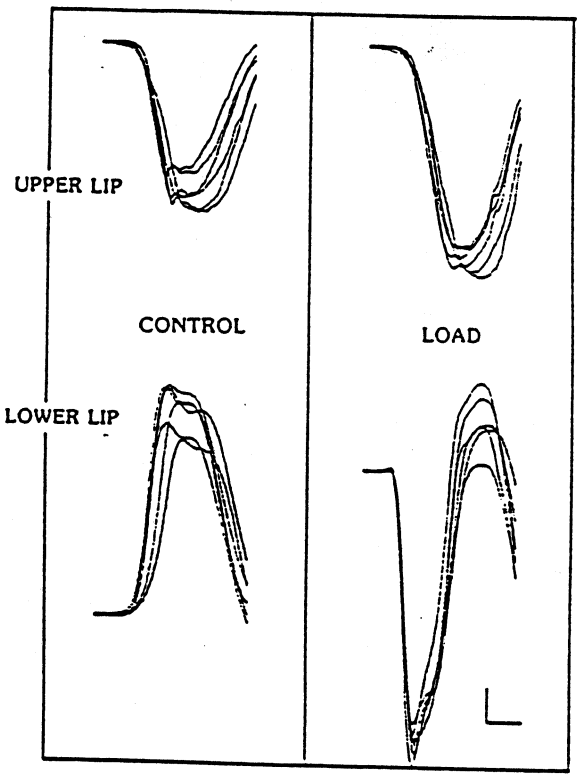

FIG. 7.1 Six control and load trials from a representative subject. Loads were introduced approximately 20ms before OOI onset. Load magnitude is 45gms. Calibration bars represent 1mm (vertical) and 50ms (horizontal).

Utilizing this paradigm, we have conducted a series of related studies involving the introduction of small unanticipated loads (10–40 grams) to the upper and lower lips prior to or during bilabial or labial-dental speech gestures. Load-induced movement changes (displacement, velocity, duration) and EMG changes (magnitude, latency) have been quantified to discern the operation of sensorimotor actions in the control of these speech gestures and their response characteristics. In the refinement of this paradigm, a number of issues have been explored formally and informally to determine its viability in revealing "normal" sensorimotor processes of speech movement control. The key observations, obtained from over 40 naive subjects, include:

1. *All subjects* compensated for unanticipated loads the first time they were introduced.
2. There were no trials for which speech was disturbed in any perceptible manner.

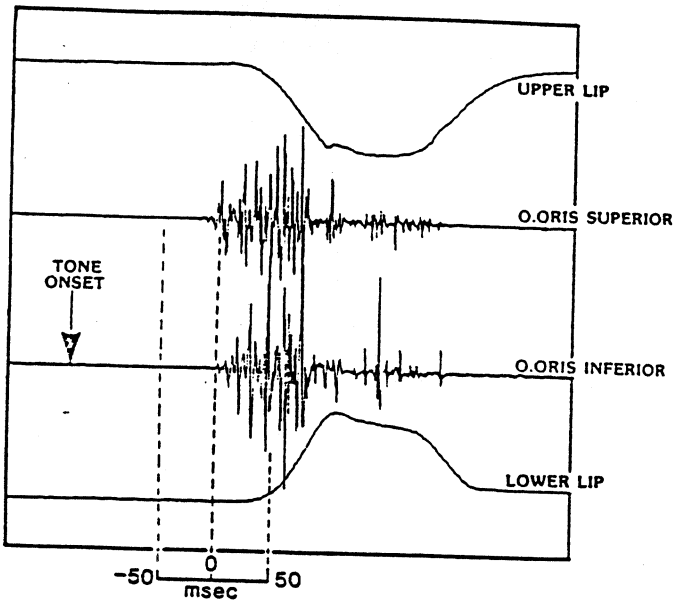

FIG. 7.2 Target interval for the placement of lower lip perturbations.

3. Subjects were *unable* to suppress a compensatory response if given instructions to "not respond" to the loads, indicating the ingrained nature of the underlying sensorimotor processes,

4. Compensatory patterns to perturbations introduced early in an experimental session did not differ, qualitatively or quantitatively, from those introduced near the end of a session,

5. Consistent and statistically significant changes in EMG and movement were observed *in all subjects studied*, even for loads as small as 10 gms (yielding perturbation displacements of 1.0 to 2.5 mm),

6. Magnitudes of compensatory displacement changes in the upper and lower lips were significantly and positively correlated to the magnitudes of the perturbation displacement.

7. *Inter*subject variability was remarkably small when factors such as range of normal lip movement, load magnitude and load onset time were normalized.

The aforementioned observations collectively support the power of this technique in revealing sensorimotor mechanisms involved in goal-directed speech motor actions. Additionally, parallel observations indicate that these sensorimotor actions vary functionally with different speech tasks. For exam-

ple, if lower lip perturbations are introduced during a labial dental articulation for [f], upper lip responses (as shown in Figure 7.1) are absent (Abbs et al., 1984). Similarly, when jaw loads are introduced for [b] and [z] articulations, compensatory responses were task-specific, i.e., confined to the muscles and movements of the lips and tongue, respectively (Kelso et al., 1984). These latter data suggest that these patterns of sensorimotor action are a component of the phonetic intention or motor program of the speaker (cf. Abbs, 1986). Given these results, we felt justified to utilize this paradigm to address the hypothesis that sensory information is used differently in the motor programming for speech movements, as contrasted to its moment-to-moment utilization during movement execution (cf. Abbs et al., 1984; Gracco, 1984).

TEMPORAL VARIATIONS IN SENSORIMOTOR ACTIONS FOR SPEECH MOVEMENT CONTROL

In order to distinguish sensory contributions during the different stages of motor "preparation," motor programming and motor execution, unanticipated perturbations were introduced at different times prior to and during the initiation of a multimovement speech gesture. Perturbations introduced "early" (i.e., well before agonist EMG onset) presumably would yield compensations indicative of a pre-execution or programming process, while later perturbations would tap the stage of motor execution. In comparing the system responses under these two conditions, clear and consistent time-dependent variations in the form and loci of the compensatory responses were observed. Comparisons of nonautogenic (e.g., upper lip responses to a lower lip load) and autogenic (lower lip responses to a lower lip load) compensation revealed consistent differences in kinematic adjustments and latencies that varied with the time the load was introduced re: the onset of agonistic muscle EMG. An example of the kinematic changes accompanying load onset timing differences is presented in Fig. 7.3. These two single load/control comparisons reflect upper and lower lip movement changes to an early (38 ms prior to voluntary EMG onset) and a late (8 ms prior to voluntary EMG onset) lower lip perturbation. As can be seen, the autogenic lower lip response predominates for the early load, while the nonautogenic upper lip response predominates for the later occurring perturbation. Figure 7.4 shows the separate upper and lower lip perturbation adjustments (expressed as a ratio of perturbation displacement to compensatory change in displacement) for a group of five subjects. As shown, the lower lip provides greater relative compensation for loads introduced prior to muscle activation (10-55 ms pre-EMG onset) than the upper lip. These data also indicate that for early loads compensatory adjustments yield proportional and relatively consistent increases in both upper and lower lip displacements.

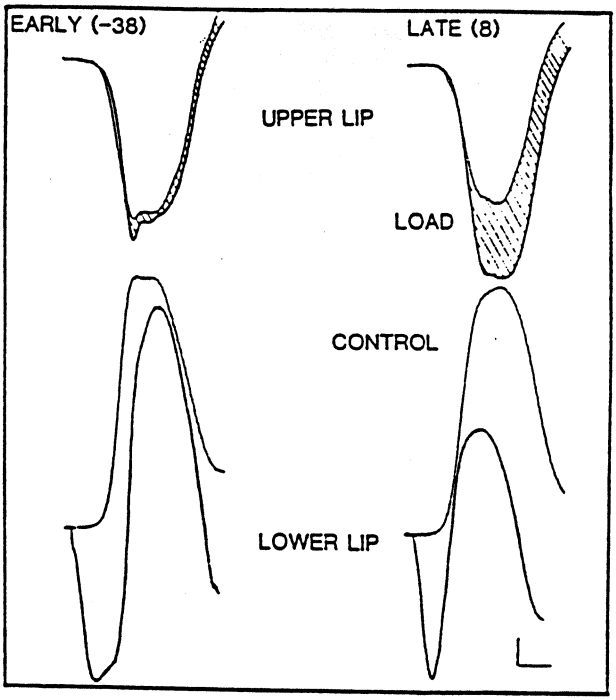

FIG. 7.3 A single load/control comparison demonstrating the load timing changes in the compensatory response. As can be seen, early occurring loads (before OOI onset) result in a larger increase in the lower lip response. Conversely, later occurring loads (after OOI onset) result in a larger increase in the upper lip response. Calibrations are the same as in Fig. 7.1.

An important aspect of the compensatory responses that occurred prior to muscle activation was their lack of time-locked response. That is, early loads do not result in fixed latency responses in the upper and lower lip muscles. Rather, the muscle changes are apparently incorporated into the previously programmed voluntary response at a wide range of latencies. Further, for these early loads, introduced 40 or 50 ms prior to EMG onset, compensatory responses from the upper and lower lips are adjusted as a unit apparently reflecting an overall modification of the parameters of a previously programmed voluntary action (Abbs et al., 1984; Gracco, 1984; Gracco & Abbs, 1985). Later occurring perturbations (after OOI onset) result in a lower lip response that is markedly reduced; by contrast, the upper lip contributes disproportionally as the load is introduced after muscle activation. As such, it appears that for perturbations introduced after motor execution has been initiated, the upper and lower lip contributions to the compensatory response reflect a different form of multimovement coupling than was

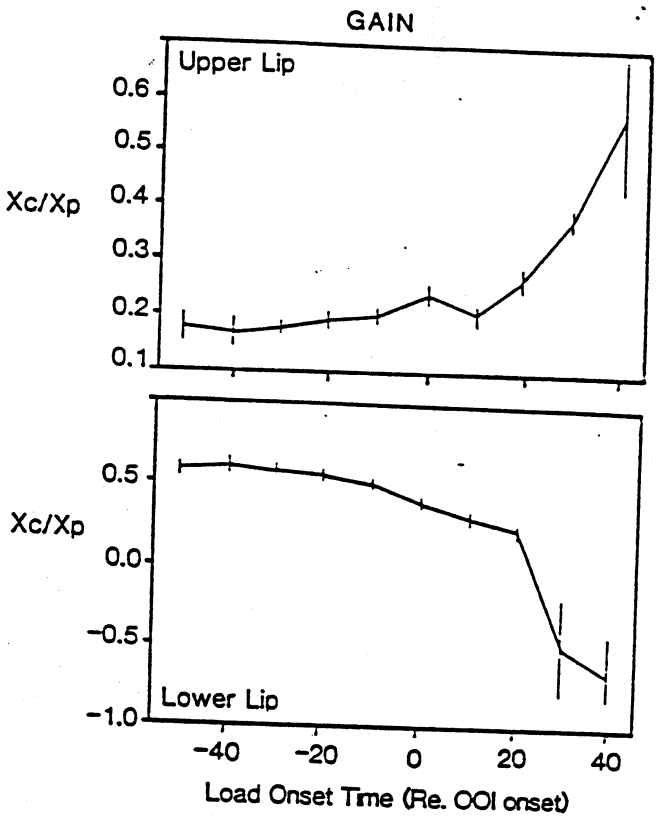

FIG. 7.4 Ratio of compensation displacement (Xc) to perturbation displacement (Xp) as a function of lower lip onset re: o. oris inferior onset. Motor task was /aba/. Compensation displacement (Xc) is the difference between the peak displacement of the upper or lower lip for the perturbed trial re: the preceding control trial. Data represent 509 trials from 5 subjects. Upper and lower lip gains were significantly different ($p < .01$) except at load onset time 20 ms post-OOI onset.

apparent for the early occurring perturbations. Instead of a proportional rescaling of both the upper and lower lip, it appears that responses to perturbations introduced immediately prior to or after the onset of muscle contraction actually reflect a functional decoupling of the two movements involved in this gesture.

SPEECH MOTOR PROGRAMMING AND EXECUTION

Given the above data, it might be hypothesized that perturbations introduced in the early phase of the motor act, seemingly prior to the activation of

pyramidal neurons for initiation of the movement, reflect the operation of a nervous system process characterized as "parameter estimation" (cf. Arbib, 1981); i.e., setting up the motor "program" to best fit the peripheral conditions. In contrast, compensatory responses to later loads (following muscle activation) reflect a predominantly predictive control mode characterized by nonautogenic adjustments. This later interval, presumably after the motor cortex has initiated its action, represents the time during which the motor task is being executed. A number of considerations, such as a high correlation between perturbation displacement and compensation displacement in the early interval and a reduction of that correlation in the later interval, reflect differences between these two processes.

These recent findings also augment the distinction we made in previous studies between autogenic (or corrective) and nonautogenic (or predictive) sensorimotor actions; these two processes appear to reflect two different nervous system control operations. In a more theoretical and evolutionary vein, a similar position has been recently offered by Goldberg (in press). Goldberg hypothesized that the nervous system is organized according to projectional (or predictive) and responsive (or corrective) control modes, representing extremes or poles of action. Based on our results of compensatory variations to lip perturbation, it appears that the compensatory responses to early loads (prior to muscle activation) reflect a predominant corrective (responsive) control mode characterized primarily by autogenic adjustments. In relation to the classical distinctions between corrective and predictive control processes (Houk & Rymer, 1981; Miles & Evarts, 1979; Rack, 1981), the predictive (projectional) actions have been suggested to be more robust under greater time constraints, as one might anticipate. Subsequently once motor action is initiated, the autogenic corrective actions, perhaps subject to the inherent instability of closed-loop delays, appear to be reduced in gain; their contribution is significantly less in the later interval. Consequently, the predominant control mode during execution appears to involve predictive nonautogenic adjustments. These observations suggest that the production of voluntary speech movements involves different sensorimotor actions during programming and execution. That is, for complex voluntary behaviors such as speech, the control mode or mode of action appears to involve both corrective and predictive sensorimotor actions (cf. Abbs & Gracco, 1984; Abbs et al., 1984, Gracco & Abbs, 1985).

The relative contribution of one control mode over the other is most probably dependent on several factors. For example, the task or context may influence the relative predominance of one control mode over the other. A task requiring precise manipulation with no speed or time requirement may rely exclusively on corrective or autogenic adjustments. Conversely, a time-critical task, such as speech production, may rely more heavily on the use of predictive adjustments. Secondly, these two opposite but overlapping control modes further delineate the basic motor programming and execution stages in

speech motor control. That is, the programming of an action appears to involve reactive or corrective actions utilizing afferent-dependent refinement of a generalized motor program (cf. Abbs et al., 1984; Schmidt, 1982). The actual execution of the program involves predictive actions utilizing afferent information to shape or fine-tune the previously programmed response through preset transfer functions included as part of the program. In this respect, we do not view the motor program as a detailed specification of the motor "commands" for a particular pattern of muscle contractions and movements. Rather, in our view, the motor program is an algorithm which sets up the system for a process whereby on-line sensory input and general motor command prespecifications are "mixed" dynamically to yield appropriate intended goals (cf. Abbs et al., 1984 for a more detailed discussion of this important point). Finally, the ability to modify speech movements throughout the motor act indicates that movement control is a real-time continuous process and is sensitive to inputs during both pre-execution and movement times (cf. Georgopoulos, Kalaska, & Massey, 1981).

NEUROANATOMICAL CORRELATES

From the previous discussion, it appears that there are multiple sensorimotor actions underlying the generation of movements for speech production. In this section, drawing from research in both human and nonhuman primates, we will consider some of the potential neuroanatomic structures or pathways that might underlie these multiple sensorimotor processes. Due to the extent of this literature, the limited scope of this chapter, and the extensive interconnection among nervous system components, we will only consider pathways that appear to be most directly involved in speech motor control. As such, this analysis must be considered a simplification primarily intended to illustrate that (a) sensory input is utilized in multiple ways in motor control, and (b) certain sensorimotor pathways appear to be preferentially involved in motor programming while others appear to be involved preferentially in motor execution. It needs to be emphasized at this juncture that a strict division between motor execution and motor programming is not realistic nor ultimately productive, hence the use of the qualifying term, preferential. In addition, we will focus upon these pathways and their functions in a unidirectional manner, from sensory input to motor output. As such, the reciprocal nature of most CNS interconnections will be ignored.

SENSORIMOTOR CONTROL OF SPEECH MOVEMENT EXECUTION

In considering the sensorimotor mechanisms of speech motor execution, one is motivated to examine those pathways whereby sensory input gains most

direct (and short latency) access to motor neurons. Such direct access is important, for as noted in the observations reviewed above, motor execution is a time-critical process and perturbations introduced just prior to or after the onset of muscle contraction yield shorter latency responses than those introduced earlier.

Brainstem Pathways

For the lips, the shortest and most direct route by which afferents affect motor output is through the trigeminal afferent-to-facial MN connections in the lower brainstem, i.e., the perioral reflex pathway. As reflected in our previous analyses (Abbs & Gracco, 1984; Gracco & Abbs, 1982b), it appears that this seemingly most direct route (via brainstem connections) is not operative during orofacial speech motor behaviors. That is, using stimuli which are within the range of lip movement kinematics for speech (i.e., velocity, acceleration), there is a notable absence of short latency perioral reflex responses (Abbs & Gracco, 1984; Gracco & Abbs, 1982b). Lower lip perturbations occurring prior to agonist muscle onset (OOI) do not result in a reflex response at brainstem-mediated perioral reflex latencies (12-18 ms). Rather, it appears that the EMG changes in response to the perturbation occur over a wide range of latencies (22-85 ms). These latency values and the lack of perioral reflex contributions to these compensatory responses support a previous suggestion by Abbs and Cole (1982) that perioral system control for speech relies more heavily on supranuclear sensorimotor pathways. The reduction in the magnitude of lower lip EMG and movement responses for loads after the onset of OOI EMG is also notable (cf. Fig. 7.4), particularly since this variation is the opposite of what one might predict if the afferent influences were acting directly on the facial motor neurons. That is, if these responses were mediated via direct afferent input at the brainstem level, one would predict that the magnitude of the response would be proportional to the excitability of the motoneuron pool (cf. Houk, 1978). The observed reduction in response magnitude at a time when the motoneuron pool is most excited (immediately prior to and following EMG onset) is apparently due to influences that are out of phase with MN pool excitability, i.e., supranuclear centers. Given the acknowledged importance of multiple supranuclear sites in speech motor control, this is not surprising.

Cortical Sensorimotor Pathways

Recent neuroanatomical findings suggest several alternative supranuclear routes over which perioral afferents might influence coordination and control of speech movements during motor execution. These various pathways, dis-

cussed below, are illustrated schematically in Fig. 7.5. Our approach here is to begin at the orofacial motoneurons and proceed upstream to the sensory inputs.

For almost all voluntary movement, the major motor outputs of the central nervous system are directed through the primary motor cortex, MI. As reflected in Fig. 7.5, for orofacial cortical regions, the pyramidal cells of layers V and VI of the primary motor cortex make monosynaptic connections to cranial motor nuclei (lips, tongue and jaw) (Kuypers, 1958). Estimates of the minimal latencies of these descending pathways from the motor cortex to orofacial motoneurons have been obtained from observations with intracortical microstimulation (ICMS) in nonhuman primates. Using ICMS, Sirisko, Lucier, Wiesendanger, and Sessle (1980) reported EMG activation of facial muscles with latencies ranging from 7-22 ms. Similarly, Hoffman and Luschei (1980) reported response latencies of 6.5 to 7.0 ms in jaw muscles with intracortical microstimulation of a primary motor cortex jaw area. These direct connections suggest that the motor cortex output directly underlies execution of speech movements; i.e., the motor cortex is the final stage of

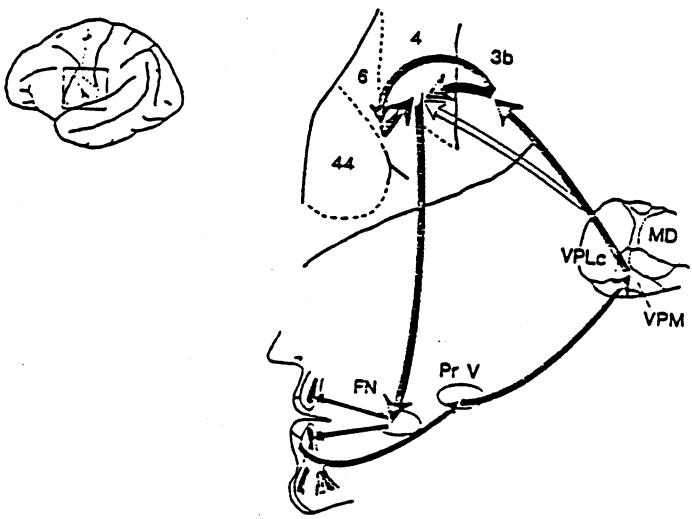

FIG. 7.5 Highly simplified orofacial "transcortical" pathways underlying the execution of speech movements. Abbreviations used include: PrV—Principal sensory nucleus of the trigeminal; Thalamic nuclei—VPM—ventral posteromedial VPLc—ventral posterolateral, caudal part; MD—dorsomedial; Cortical areas—3b—Somatic sensory, 6—lateral premotor, 4—precentral motor, FN—facial nucleus. The unoarkened arrow reflects a possible direct thalamocortical pathway based on data reported by Asanuma et al., 1979 (see text).

supranuclear control.[1] In this context, determining the supranuclear structures contributing to the sensorimotor processes of speech execution involves examining those sensory pathways that have the most direct influence on the pyramidal cells.

As illustrated in Fig. 7.5, there is a direct route from sensory receptors located in the periphery (cf. Evarts, 1981; Wiesendanger, 1978) to the primary motor cortex pyramidal cells (i.e., the so-called transcortical pathway). This input to motor cortex from muscle afferent and cutaneous stimuli is very powerful and appears largely to be somatotopically organized. For example, primary motor cortex pyramidal cells projecting to a given peripheral region (e.g., the index finger) preferentially receive sensory input from the muscles and skin associated with movement of that region (Asanuma & Rosen, 1972; Rosen & Asanuma, 1972). This pathway, originally suggested by Hammond (1956, 1960) from observations in human subjects, and more recently by Phillips (1969), may traverse two different routes, as shown in Fig. 7.6 (cf. Asanuma & Arissian, 1984; Evarts, 1981 for discussion). That is, the transcortical pathway is considered to involve sensory input directed to the motor cortex pyramidal cells both via SI and directly, the latter through either the nucleus ventralis posterolateralis pars oralis (VPLo) subdivision of the thalamus (Asanuma & Arissian, 1984; Asanuma, Larsen, & Yumiya, 1979; Lemon & van der Burg, 1979; Rosen & Asanuma, 1972) or the nucleus ventralis posterolateralis caudalis (VPLc) (Asanuma, Thach, & Jones, 1983).

Sensory impulses originating from the perioral region also project to numerous thalamic and hence cortical sites. Perhaps the most secure and rapid transmission of sensory input from orofacial sites is via the trigeminothalamic projections, originating from the main sensory nucleus and subnucleus oralis of the trigeminal complex. Based on the types of trigeminothalamic synaptic junctions and terminal distribution of corticobulbar fibers, the main sensory nucleus represents the bulbar homologue of the dorsal horn (Kuypers, 1981). That is, similar to the dorsal column system, the trigeminothalamic system transmits information regarding touch and kinesthesia and this ascending information can be modulated through descending corticobulbar influences. In most species, including monkey and man, trigeminothalamic projections terminate in the ventral posteriomedial (VPM) nucleus of the thalamus (Smith, 1975). These large-diameter crossed and uncrossed projections are somatotopically organized (Kaas et al., 1984) and have been shown to reflect peripheral stimuli with a high degree of

[1] Although not dealt with specifically in this view, it is known that there are projections from the orofacial regions of MI and premotor cortex to the parvocellularis portion of the red nucleus, which in turn appears to project to the ipsilateral inferior olive (Humphrey, Gold, & Reed, 1984; Kuypers & Lawrence, 1967). The magnocellular division of the red nucleus also receives MI input (Humphrey et al., 1984; Kennedy, Gibson, & Houk, 1984; Kuypers & Lawrence, 1967) primarily from the leg and arm areas, and has been shown to project to the facial nucleus (Miller & Strominger, 1973) and to the interpositus nucleus of the cerebellum.

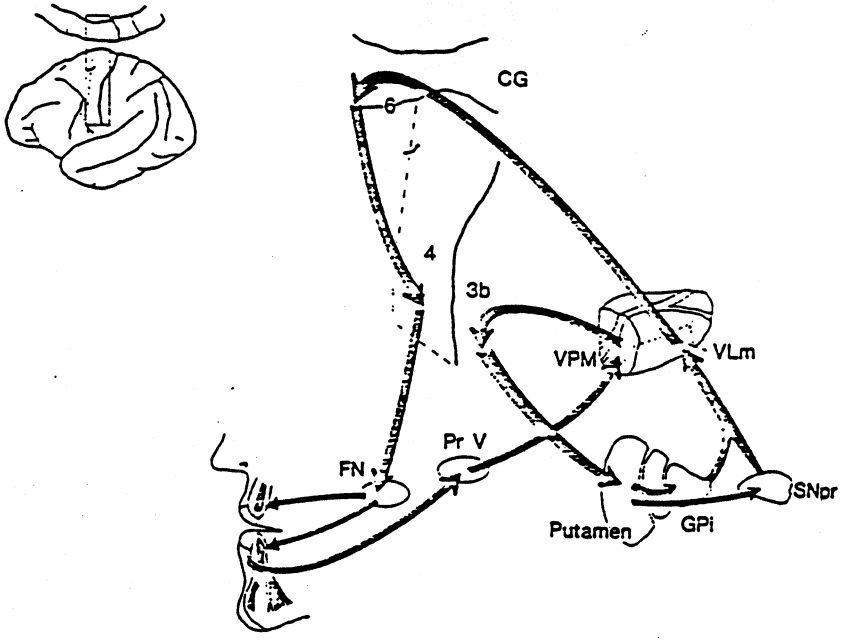

FIG. 7.6 Proposed pathway for speech motor programming incorporating the known basal ganglia—supplementary motor area (SMA) relations. Abbreviations used include: PrV—Principal sensory nucleus of the trigeminal; VPM—Ventral posteromedial nucleus of the thalamus, VLm—Ventral lateral nucleus, medial part; GPi—globus pallidus, inner segment, SNpr—substantia nigra pars reticulata; Cortical areas—3b—somatic sensory, 6—mesial premotor (SMA), 4—precentral motor; FN—facial nucleus; CG—cingulate gyrus.

reproductibility (Darian-Smith, 1966), indicating limited preprocessing and minimal convergence. Information transmitted through the trigeminal system via this pathway is in turn relayed to cortical somatic sensory areas 3b and 1 (Felleman, Nelson, Sur, & Kaas, 1983; Kaas, Nelson, Sur, Dykes, & Merzenich, 1984; Merzenich, Kaas, Sur, & Lin, 1978).[2]

The sensory projections from the perioral region to the somatic sensory cortex have been examined primarily by recording evoked potentials on the exposed cortex (Dreyer, Loe, Metz, & Whitsel, 1975); unfortunately, the latencies of these pathways have often not been reported. In one study (O'Brien, Pimpaneau, & Albe-Fessard, 1971), evoked potentials in the somatic sensory cortex of chloralose anesthetized monkeys to labial electrical stimulation were observed at latencies ranging from 5-12 ms. These authors

[2] It should be mentioned that Asanuma et al. (1979) reported the presence of a few facial VPM thalamic neurons activated antidromically from the motor cortex, suggesting the presence of a direct trigeminothalamic projection to MI independent of SI.

also reported responses in the face area of the motor cortex at slightly longer latencies (7-14 ms). Similarly, Hoffman and Luschei (1980) were able to activate motor cortex pyramidal cells by stretching the jaw closing muscles; the sinusoidal stretch used by these investigators yielded responses that were sensitive to the dynamic phases of the mechanical perturbation and were presumably of short latency. Given these data, it appears that direct ascending and descending pathways via the cortex are available with minimal loop times of as short as 14-20 ms (e.g., sensory ascending latencies of 7-10 ms and descending motor latencies of approximately this same minimum value). It should be noted that this sensorimotor pathway for the orofacial system is not well-elucidated; only a single study has reported both corticopetal and corticofugal responses in the same experiment and that experiment focused on the masticatory system (Hoffman & Luschei, 1980). Moreover, modern neuroanatomical techniques (e.g., axoplasmic tracing) have not focused specifically on supranuclear representations of orofacial mechanisms. However, the available data suggest that there are orofacial analogs to the so-called transcortical pathways which would involve minimal latencies and minimal processing; these pathways would be ideal for control during the time-critical processes of motor execution. In that respect, it would appear reasonable to suggest that the orofacial sensory projections to motor cortex (i.e., a transcortical pathway) are a major candidate for the autogenic sensorimotor adjustments observed in response to the later loads observed in our previous experiments. While the sensory pathways to primary motor cortex pyramidal cells are the most direct, there are sensory projections to other areas of the cortex that also may underlie the moment-to-moment adjustments necessary for motor execution. In this vein, the classical transcortical pathway traditionally has been considered to underlie autogenic or corrective adjustments; as such, other pathways may be involved in the prominent nonautogenic adjustments (i.e., upper lip responses to lower lip perturbations) just described.

One possible pathway for the nonautogenic adjustments is via sensory inputs to the cortical cytoarchitectonic area 6 (premotor cortex) in lateral regions of the precentral cortex (Fig. 7.7). This lateral premotor area has been shown to receive somatic sensory input from the orofacial region and in turn projects to the primary motor cortex. Neuroanatomically, the route for this sensory input to the lateral premotor cortex is ambiguous. However, electrophysiological data are quite compelling. For example, O'Brien et al. (1971) and Rizzolatti, Scandolara, Gentilucci, and Camarda (1981a) demonstrated that:

1. Lateral premotor responses to peripheral orofacial stimulation are almost as short in latency as those to the primary motor cortex (10-12 ms), and

2. This region of cortex is particularly sensitive to more complex, multimovement goal oriented stimuli (as contrasted to the primary motor cortex).

That is, this region of cortex appears to receive the kind of converging orofacial somatic sensory projections necessary for adjustments in the upper lip to lower lip perturbations or adjustments in the lips or tongue to perturbations applied to the jaw. Muakkassa and Strick (1979) and others have shown also that the face region of this premotor area has direct projections to primary motor cortex. As such, it might be suggested that one pathway underlying nonautogenic (intermovement) adjustments during the execution phases of the speech motor act is via orofacial somatic sensory projections to premotor cortex and, in turn, to the primary motor regions. Interestingly, select lesions of the orofacial premotor regions in monkeys result in dramatic deficits in the coordination of the lips, jaw, and tongue (cf. Larson, Byrd, Garthwaite, & Luschei, 1980; Luschei & Goodwin, 1975; Watson, 1975).

These considerations suggest that the nonautogenic sensorimotor compensations described previously in this paper may be mediated via a second cortical-cortical pathway, i.e., via the premotor cortical region. In this context, it is especially intriguing to consider how such adjustments are organized and reorganized with variations in phonetic goals (e.g., *p* vs. *f*) on a moment-to-moment basis. Seemingly, such variations would involve the processes referred to earlier as motor programming. In this case, the instructions for task-dependent sensorimotor actions (cf. Abbs et al., 1984; Kelso et al., 1984) would be "down-loaded" from other CNS structures for implementation in primary and nonprimary motor cortical regions. Responses to perturbations introduced well in advance of muscle activation would appear to reflect operations associated with these down-loaded processes. This consideration, of course, leads to the second central motor process of interest in the present paper, speech motor programming.

SENSORIMOTOR PROCESSES IN SPEECH MOTOR PROGRAMMING

There are several alternate and more indirect routes over which sensory input gains access to the primary motor cortex, possibly involving sensory processing and integration for purposes other than moment-to-moment adjustments of motor execution. Several of these indirect pathways involve brain sites thought to be important for motor control in a more executive capacity. Consideration of these pathways thus offers some preliminary hypotheses on some of the supranuclear structures potentially involved in speech motor programming. The two subcortical structures most often implicated in movement programming are the basal ganglia and the cerebellum (Allen &

Tsukahara, 1974; Brooks, 1979; Brooks & Thach, 1981; DeLong & Georgopoulos, 1981; Kemp & Powell, 1971; Paillard, 1983). Further, Schell and Strick (1984), based on their own neuroanatomic findings and reinterpretation of earlier data, suggest that the orofacial representations of the basal ganglia and cerebellum project in a topographic and segregated fashion to two different nonprimary motor cortical regions; the supplementary motor and premotor areas, respectively. Inasmuch as the primary motor cortex receives strong somatotopically segregated projections from premotor and supplementary motor cortices (Matelli, Camarda, Glickstein, & Rizzolatti, 1984; Muakkassa & Strick, 1979), these latter data suggest that afferents from the basal ganglia and the cerebellum influence the primary motor cortex via these nonprimary motor areas. Collectively, these data thus provide a basis for considering how orofacial sensory input might influence the process of speech motor programming. In the discussion that follows, we review these more recent neuroanatomical and neurophysiological findings which suggest that these subcortical and nonprimary motor areas comprise two major motor systems over which afferent input from the orofacial region gains access to motor output.

Basal Ganglia/Supplementary Motor Contributions

As noted, one sensorimotor pathway potentially underlying speech motor programming involves the basal ganglia. From this perspective, the basal ganglia are viewed as being upstream from the motor cortex and involved in task specifications that occur prior to a completed movement. Damage to the basal ganglia is known to result in numerous muscle tone and movement problems (cf. DeLong & Georgopoulos, 1981; Marsden, 1982). Although there has been controversy regarding a motor versus cognitive role of the basal ganglia, it now seems clear that the basal ganglia are composed of two functional subsystems: i.e., the caudate and putamen, serving "complex" and motor functions, respectively (cf. DeLong & Georgopoulos, 1981; DeLong et al., 1984). As illustrated in Fig. 7.6, the following discussion focuses primarily on the sensory input, orofacial representation, and motor output of the putamen in relation to its potential influences on motor cortex output. It should be noted that although many investigators view the basal ganglia as having a programming function, their potential role in moment-to-moment adjustments for motor execution is an issue of continuing interest (cf. Iansek & Porter, 1980; Liles, 1983).

The putamen receives sensory information from three major sources: (a) the substantia nigra pars compacta (SNpc), (b) the intralaminar nuclei of the thalamus, and (c) multiple cortical sites including the somatosensory, motor, and premotor areas (Dray, 1980; Jones, Coulter, Burton, & Porter, 1977; Künzle, 1975, 1976, 1977). Shown in Fig. 7.6 is only one of many possible projections to the basal ganglia. Neurons in the putamen have been shown to

respond to both passive and active limb movements at latencies between 25 and 50 ms (Crutcher & DeLong, 1984a, 1984b; DeLong & Georgopoulos, 1979). Similarly, studies of somatosensory input to the basal ganglia have shown that putamen neurons also respond to natural orofacial stimuli (DeLong & Georgopoulos, 1979). In the cat, Schneider, Morse, and Lidsky (1982) found that 42% of the units responsive to orofacial stimulation had a mean latency of 18 ms. The latency of basal ganglia responses to somatosensory stimuli, as compared to somatic sensory and motor cortex responses (cf. Evarts, 1973 [monkey]; Landgren & Olsson, 1980 [cat]), suggests an indirect, multisynaptic input pathway.

The major output from the putamen is directed through the inner segment of the globus pallidus and the pars reticulata portion of the substantia nigra (SNpr) (Nauta & Mehler, 1966). As reflected in Fig. 7.6, neurons related to orofacial movements have been found in the lateral portion of the SNpr and the ventrocaudal segment of the GPi (DeLong, Crutcher, & Georgopoulos, 1981; DeLong & Georgopoulos, 1979); cells in SNpr have been observed to discharge in relation to natural orofacial movements (DeLong et al., 1984; Mora, Mogenson, & Rolls, 1977). Further, it has been shown that portions of the basal ganglia associated with orofacial movements (SNpr) have a major efferent projection to a subdivision of the centrolateral thalamus, ventrolateralis medialis (VLm) (Carpenter & McMasters, 1964; Carpenter, Nakano, & Kim, 1976; Carpenter & Peter, 1972). As noted, recent work by Schell and Strick (1984) partially completes this picture demonstrating segregated inputs from VLm to the face area of the supplementary motor area (SMA) (cf. Fig. 7.6). The final set of data to this point are those of Muakkassa and Strick (1979) that demonstrate reciprocal connections between the face subregion of SMA and primary motor cortex. Inasmuch as anatomic and electrophysiologic studies have indicated an absence of direct projections from the SMA to facial, motor trigeminal or hypoglossal nuclei (Künzle, 1978; Macpherson, Marangoz, Miles, & Wiesendanger, 1982; Penfield & Welch, 1951), SMA influences on orofacial actions presumably are exerted via primary motor cortex. Hence, it appears that face area projections from the basal ganglia exert their motor control influence on MI via the SMA. As such, one way somatic sensory inputs to the basal ganglia can influence the control of orofacial movements for speech is via ascending projections to SMA, and, in turn, to primary motor cortex.[3]

[3] It should be noted that corticofugal projections via SNpr also are directed onto numerous brainstem sites including the superior colliculus, and reticular formation sites with the midbrain pons and medulla (Kuypers, 1981). Based on recent evidence from our laboratory (Gracco & Abbs, 1984) indicating the abnormal presence of perioral reflex responses to lower lip perturbation in Parkinson patients, it might be suggested that the inhibitory function often associated with the basal ganglia (cf. DeLong & Georgopoulos, 1981) may be mediated by these descending influences. The dysfunction of such hypothesized inhibitory influences is consistent with Parkinson patients' inability to suppress the mid-latency response to muscle stretch (Mortimer & Webster, 1979; Tatton & Lee, 1975).

Based on these data and consideration of the anatomical connections of the SMA with primary motor cortex, the suggestion that SMA is involved in executive level function relative to the primary motor cortex (Brinkman & Porter, 1979; Jürgens, 1984; Roland, Larsen, Lassen, & Skinhøj, 1980; Tanji, 1984) appears warranted. In addition to the basal ganglia inputs, the SMA also receives projections from numerous cortical sites including premotor, primary motor, somatic sensory, and parietal cortices (areas 1, 2, and 5) (Jones & Powell, 1970; Jürgens, 1984; Pandya & Vignolo, 1971). As with basal ganglia responses to peripheral stimuli, SMA latencies have been shown to be longer (greater than 20 ms from arm area; Brinkman & Porter, 1979) than for MI (7–15 ms from the fingers; Lemon, 1981). Additionally, SMA is less responsive to peripheral stimuli than MI (Brinkman & Porter, 1979; Lemon & Porter, 1976; Wise & Tanji, 1981), and apparently more selective. Tanji and Kurata (1982) reported SMA neurons to be selectively active dependent on the modality of the stimulus; i.e., visual, auditory, or somatosensory. This observation is in contrast to MI for which no such modality specificity has been reported. Timing of SMA activity has been shown to be related to movement with neurons responding both before and after the onset of muscular activity (Brinkman & Porter, 1979; Smith, 1979). The onset of neuronal activity in response to visual or auditory cues to move are earlier than those of precentral neurons, suggesting that SMA is involved in the earlier stages of premovement sensorimotor processing. Additionally, SMA has been shown to be involved primarily in movement sequences or complex movements; its apparent unresponsiveness to passive peripheral stimuli may merely reflect the multisynaptic and task-specific nature of the SMA contribution. Finally, as shown by Tanji and colleagues (Tanji & Kurata, 1982; Tanji, Taniguchi, & Saga, 1980), SMA activity is related to the actual motor preparation and not to the stimulus used to signal the initiation of a motor act.

Based upon observations in normal and impaired humans, the basal ganglia SMA pathway obviously is important for speech motor control and motor control in general. For example, it has been shown that basal ganglia dysfunction leads to speech and limb movement aberrations (Darley, Aronson, & Brown, 1975; Evarts, Teräväinen, & Calne, 1981; Flowers, 1976; Hunker, Abbs, & Barlow, 1982; Marsden, 1982; Wilson, 1925). These data, obtained exclusively in individuals with Parkinson's disease, range from perceptually observed deficits (e.g., imprecise consonants, monopitch, and monoloudness, cf. Darley et al., 1975) to reductions in amplitude and velocity of movements (Evarts et al., 1981; Hunker et al., 1982; Hunker & Abbs, 1984), elongated movement times (Evarts et al., 1981), and an apparent inability to utilize sensory information normally in scaling movement amplitude and force (Gracco & Abbs, 1984; Tatton, Eastman, Bedingham, Verrier, & Bruce, 1984). These observations are consistent with the suggestion that the basal ganglia ultimately influence the scaling of EMG and

subsequent kinematic parameters (DeLong et al., 1984; Horak & Anderson, 1984a, 1984b).

Interestingly, as noted previously, several investigators have suggested a similar executive function for the motor portions of the basal ganglia (cf. DeLong & Georgopoulos, 1981; Marsden, 1982). Moreover, there are similarities in the motor deficiencies of individuals with Parkinson's disease and SMA damage. Those basal ganglia-SMA dysfunction similarities include speech motor aberrations, ranging from speech arrest to imprecise articulation (Arseni & Botez, 1961; Caplan & Zervas, 1978; Damasio & Van Hoesen, 1980; Darley, Aronson, & Brown, 1975; Masdeu, Schoene, & Funkenstein, 1978).

In contrast to observable motor deficits resulting from basal ganglia dysfunction, the motor contributions of the SMA are less clearly documented. Changes in cerebral blood flow (CBF) have been used to identify cortical areas active during certain complex motor gestures including speech (Larsen, Skinhøj, & Lassen, 1978; Orgogozo & Larsen, 1979; Roland, Larsen, Lassen, & Skinhøj, 1980). Subjects instructed to count or recite the days of the week displayed increased blood flow in the primary motor and supplemental motor cortex of both cerebral hemispheres. Blood flow changes were noted in SMA, basal ganglia (putamen and globus pallidus), and motor cortex during execution of independent finger movements, but not during simpler motor tasks such as repetitive flexion of a single finger or during sustained isometric contractions (Orgogozo & Larsen, 1979; Roland et al., 1980; Roland, Meyer, Shibasaki, Yamamoto, & Thompson, 1982). Similariy, electrical stimulation of the SMA in man produces a transient inability to speak (Penfield & Roberts, 1959). Damage to the anterior cerebral artery primarily results in volitional speech disturbances often described as a motor or transcortical aphasia (Gelmers, 1983; Rubens, 1975). In a summary of 12 patients with well-defined tumors involving the SMA, Arseni and Botez (1961) report two categories of speech disturbances: one affecting the functioning of the motor mechanism of speech and the other a more "dysphasic disorder." Similar observations had been made earlier by Critchley (1930); Chusid, de Gutiérrez-Mahoney, & Margules-Lavergne (1954); and Lapiane, Talairach, Meininger, Bancaud, & Orgogozo (1977). Although the studies reporting speech symptoms associated with SMA damage are not as numerous or, more importantly, as detailed, it appears that the motor aberrations in these patients may be similar to aberrations associated with basal ganglia damage.

Cerebellar/Premotor/Contributions

The second potential pathway by which sensory input may influence motor programming is via the cerebellum, a structure classically considered to be involved in coordination of complex movements (cf. Holmes, 1922). Because

of the parallel sensory projections to the cerebellum and somatic sensory cortex, one is led to believe that the former inputs are involved in functions other than moment-to-moment control of motor execution. Indeed a number of investigators have shown that cerebellar output via the dentate nucleus reflects more complex motor functions such as "motor set" (Hore & Vilis, 1984: Strick, 1978, 1983). Until very recently, however, the exact projections from the orofacial portions of the cerebellum to the motor cortex were ambiguous: earlier studies primarily utilized degenerative or destructive neuroanatomical techniques. Recent neuroanatomical and physiological observations, as summarized later, offer a more coherent picture of these projections.

As illustrated schematically in Fig. 7.7, the cerebellum receives orofacial sensory information from the periphery via direct trigeminal routes as well as more indirect cortical projections (Allen, Gilbert, & Yin, 1978: Allen & Tsukahara, 1974). Afferent input to the cerebellum terminates on the cerebellar cortex (Cody & Richardson, 1978; Miles & Wiesendanger, 1975; Snider & Stowell, 1944) and on the deep cerebellar nuclei (Chan-Palay, 1977; Eller & Chan-Palay, 1979). In the forelimb, somatic sensory stimuli have been

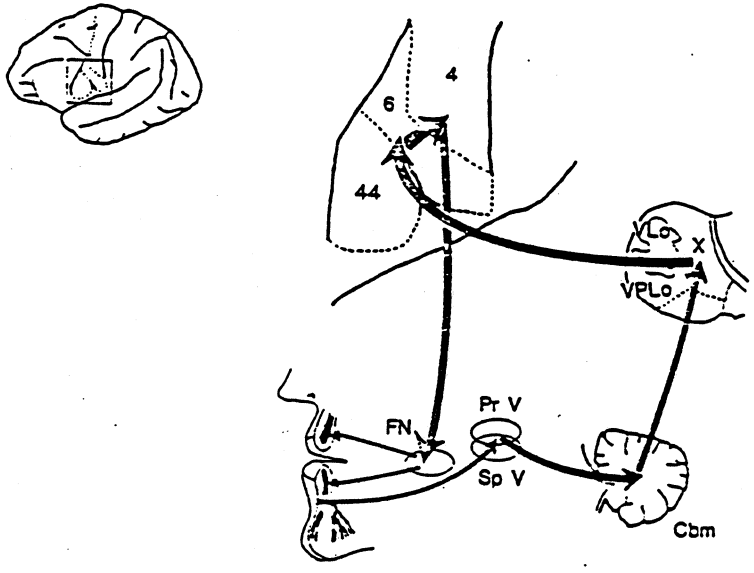

FIG. 7.7 Proposed pathway for speech motor programming incorporating the known cerebellar—premotor area (PMA) relations. Abbreviations used include: PrV—principal sensory nucleus of the trigeminal, spV—spinal portion of the trigeminal (interpolaris subdivision); Cbm—cerebellum (lateral dentate nucleus—although not indicated in the figure, the trigeminal projections terminate on the cerebellar cortical surface as well as directly to the deep nuclei; see text for detail); X—thalamic nucleus X, VPLo—ventral posterolateral nucleus, oral part, VLo—ventral lateral nucleus, oral part; Cortical areas—same as previous figures; FN—facial nucleus.

recorded in dentate and interpositus nuclei with latencies of 30-50 ms and 20-40 ms, respectively (Harvey, Porter, & Rawson, 1979; Strick, 1978). Cutaneous facial stimulation results in short latency input to Crus I and II and the paramedian lobule of the cerebellar cortex (Huerta, Frankfurter, & Harting, 1983), which contains the most expansive facial map (Shambes, Gibson, & Welker, 1978). In addition, retrograde HRP labelling in the monkey has shown direct and bilateral monosynaptic connections from the main sensory and spinal nucleus of V to cerebellar dentate nucleus (Chan-Palay, 1977). Similar HRP injections into the interpositus nucleus resulted in retrograde labelling in the mesencephalic nucleus, containing jaw muscle spindles, but not in the main sensory or spinal nucleus of V (Chan-Palay, 1977). However, facial representations in the interposed nuclei are reported by Thach, Perry, and Schieber (1982) in the monkey and Richardson, Cody, Paul, and Thomas (1978) in the cat. Thach et al. (1982) reported neurons in the posterior portion of dentate and interpositus that discharged in relation to facial movements. Recordings in the cat cerebellum have demonstrated projections onto interpositus nuclei from supraorbital, infraorbital, mental, and masseteric nerve branches (Richardson et al., 1978). These data indicate that orofacial afferents project directly to the dentate nucleus as well as indirectly to dentate and interpositus via the cerebellar cortex.

While precise somatotopy has not been described for the hemispheres of the cerebellar cortex, the lateral nuclei are known to contain a facial representation primarily located in caudal and posterior regions (Stanton, 1980; Thach et al., 1982).[4] As reflected in Fig. 7.7, the caudal portion of the dentate nucleus, which includes this facial representation, projects primarily to the ventrolateral thalamus area (Asanuma et al., 1983; Stanton, 1980) with a noticeable lack of cerebellar terminations in VLm (Schell & Strick, 1984) which receives basal ganglia projections as noted above. These thalamic projections from the caudal dentate are coextensive and overlap with corticothalamic projections from the face areas of the primary and nonprimary motor cortices (Kievit & Kuypers, 1977; Kuypers & Lawrence, 1967). Based on their retrograde HRP tracing study and considering the physiological studies of Sasaki et al. (1976, 1979), Schell and Strick (1984) indicate that the caudal dentate nuclei (the orofacial representation) project to the premotor cortex via thalamic area X. Thus, it appears that the caudal dentate does not project directly to motor cortex, but rather to the premotor area (cf. Fig. 7.7). Moreover, as noted previously, Muakkassa and Strick (1979) demonstrated direct inputs from the face area of premotor cortex to MI. As such, sensory inputs to the cerebellum appear to influence motor cortex output to orofacial motoneurons indirectly via the premotor cortex.

[4] Both interpositus and dentate nuclei project to thalamus; however, we will focus on the dentate projections as the literature suggests that dentate is more involved in motor programming (Allen & Tsukahara, 1974; Brooks & Thach, 1981; Thach, 1978).

Analyses of cerebellar outputs, as reflected in activity of the deep nuclei, reflect the executive role of this structure. Dentate activity appears to be governed by the "willed" action of the animal and related to the intended movement, seemingly independent of activity at the motor cortex or the lower motor neuron pool. More specifically, Thach (1978) demonstrated dentate activity which was correlated with position of the limb and the direction of the intended next movement. In contrast, interpositus activity was best related to the pattern of muscular activity of the limb, the reflexive behavior of neurons in motor cortex or in muscles when a stimulus (perturbation) is delivered (Thach, 1978). Strick (1978, 1983) has reported data on single unit recordings in dentate nucleus during several contrastive motor tasks, and suggested that this output of the cerebellum was involved preferentially in "motor set". In particular, it was demonstrated that single neural units in dentate did not vary directly with a sensory stimulus (as apparently do cortical cells), but rather responded to predetermined combinations of the intended movement and sensory stimuli. Similarly, Hore and Vilis (1984) eliminated components of EMG responses attributed to set by cooling the dentate nucleus. These latter data suggest that lateral cerebellar nuclear output to the motor cortex is not related to the particular parameters of movement (as would be predicted if this structure were involved in motor execution), but to the conditions under which a given movement was generated. Finally, prior to a voluntary movement, cells within the dentate have been shown to discharge earlier than cells within the motor cortex (Thach, 1975), and cooling of the dentate delayed both onset of voluntary movement as well as movement-related activity in motor cortex (Meyer-Lohmann, Hore, & Brooks, 1977). These data augment the suggestion that cerebellar output, as manifested in dentate, is upstream from and precedes activity in motor cortex. Further, data such as these strongly implicate the lateral cerebellar nuclei in the programming of voluntary movements.

The role of the cerebellum as a brain center underlying coordination of movement and the effects of premotor cortical lesions lend credence to the concept that the cerebellum and the orofacial region of premotor cortex work in conjunction, perhaps in a serial fashion. The influence of this cerebellar-premotor pathway in orofacial control and speech is apparent from several sets of data. For example, damage to the cerebellum results in an ataxic dysarthria (Darley et al., 1975; Kent & Netsell, 1975; Kent, Netsell, & Abbs, 1979; Lechtenberg & Gilman, 1978) seemingly characterized by a breakdown in normal speech movement generation. Further, speech aberrations following frontal lobe damage suggest that the premotor area is also involved in the control of orofacial movements (cf. Fromm, Abbs, McNeil, & Rosenbek, 1982; Itoh, Sasanuma, Hirose, Yoshioka, & Ushijima, 1980; Schiff, Alexander, Naeser, & Galaburda, 1983; Tonkonogy & Goodglass, 1981). As noted by Mohr (1976):

> a 'restricted' lesion to these areas (lateral premotor) leads one to view their function as mediating a more traditionally postulated role as a premotor association cortex region concerned with acquired skilled oral, pharyngeal, and respiratory movements, involving speaking as well as other behaviors but not essentially language or graphic behavior, per se. (p. 22)

Similarly, single neural unit and behavioral studies in nonhuman primates have shown the premotor area to be involved in the guidance of movements based on sensory signals (Godschalk, Lemon, Nijs, & Kuypers, 1981; Halsband & Passingham, 1982; Rizzolatti, Scandolara, Matelli, & Gentilucci, 1981b). Similar to cerebellar studies, the premotor cortex has been shown to reflect activity associated with motor set or programming (cf. Weinrich & Wise, 1982; Weinrich, Wise, & Mauritz, 1984). Parallel to the earlier suggestion that the basal ganglia-SMA damage may produce some comparable speech movement aberrations, it also has been suggested that cerebellar and premotor cortical damage may yield similar speech movement aberrations (cf. Kent & Rosenbek, 1982 for discussion based on acoustical analyses). The movement discoordination noted by Fromm et al. (1982) in a study of apraxia of speech and the dysprosodic similarities between ataxic dysarthrics and apraxic patients described by Kent and Rosenbek (1982) appear to support such a suggestion. Based on the behavioral data presented here and the parallel neuroanatomical considerations, it appears that the cerebellar-premotor system occupies an executive position relative to the primary motor cortex and may perform overlapping or related functions in the programming of speech. Obviously, however, more detailed investigations are needed.

SYNTHESIS AND IMPLICATIONS

As discussed previously and summarized in Fig. 7.8, there is substantial neuroanatomical and functional evidence for the operation of multiple somatic sensory contributions to the processes of speech motor control. These multiple sensory influences upon orofacial motor output dramatically highlight suggestions over the last 10 years by many neuroscientists that our current challenge is no longer to determine whether sensory input is utilized in control of motor output, but rather to determine the exact nature of this important contribution (cf. Abbs & Cole, 1982; Evarts, 1982; Grillner, 1975). In that spirit, the present paper provides an initial neuroanatomical and neurophysiological framework within which to consider the differential contribution of sensory inputs to the operations of motor programming and motor execution. However, these two processes in turn each appear to be served by more than one set of pathways. For motor execution, there may be as

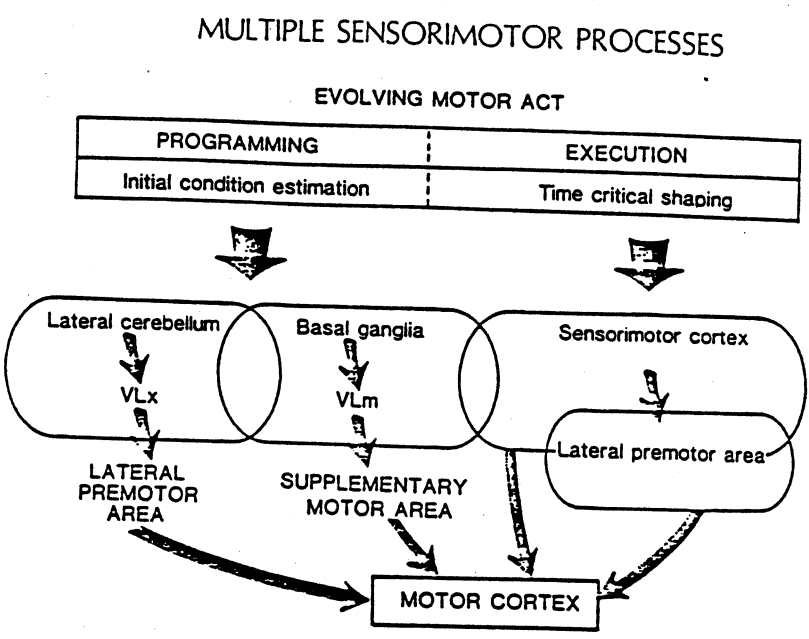

FIG. 7.8 Schematic summary illustrating the hypothesized multiple sensorimotor processes and neuroanatomical substrate involved in the programming and execution of speech.

many as three major sensorimotor pathways involved, including inputs to motor cortical pyramidal cells (the "upper motor neuron pool") via the somatic sensory cortex, the premotor cortex, and directly via the thalamus. All of these pathways appear to have the requisite short latencies and input/output characteristics to contribute to the time-critical adjustments for motor execution.

In parallel, it appears that while both the cerebellum and basal ganglia also receive sensory input, these centers, in conjunction with the nonprimary motor areas, may participate in more executive functions related to the programming of movement. Extrapolating from available data in the limbs, it appears that the cerebellum may be involved in establishing the necessary temporal-spatial relationship among the speech articulators (Soechting, Ranish, Palminteri, & Terzuolo, 1976) (i.e., the scaling of the upper lip, lower lip, and jaw for bilabial production) as well as initiating the timing between agonist and antagonist muscles (Brooks, Kozlovskaya, Atkin, Horvath & Uno, 1973; Meyer-Lohmann et al., 1977; Soechting et al., 1976; Trouche & Beaubaton, 1980). Several recent papers have provided very specific data to this point; e.g., the cerebellum appears to control the spatial gain between the head and eye movements in the vestibular-ocular reflex as

an off-line controller (cf. Lisberger, 1984). On the other hand, the basal ganglia sensorimotor pathway may influence the generated movement by scaling the EMG activity in the agonist and antagonist muscles (Horak & Anderson, 1984a, 1984b) to yield movements of appropriate magnitude and velocity.

The integration of sensory input with the motor programs via the basal ganglia and cerebellum may correspond, neuroanatomically, to the parameter estimation process suggested by Arbib (1981) (cf. Abbs et al., 1984). Interestingly, both of these subcortical systems influence the motor system via nonprimary motor areas that in turn impinge on the primary motor cortex. In this context, it needs to be reemphasized that, in our particular view, a motor program is not a representation of detailed movements and specific muscle contractions. Rather, we consider a motor program more as an "implementation algorithm" for carrying out motor execution, including appropriate modulation and gating of sensorimotor pathways. One could argue that these algorithms, including appropriate gating for on-line sensorimotor adjustments during motor execution, are down-loaded to the premotor and supplementary motor cortex and are ultimately manifest only indirectly in the observable motor output. These nonprimary motor areas have long been considered to have significant roles in motor control, particularly in the preliminary programming of complex movements.

Together, these results and discussion point to a dynamic hierarchical model of speech motor control utilizing distributed motor control processes and interacting neural subsystems. This hierarchical organization, as depicted graphically in Fig. 7.8, is dynamic from the standpoint that once the goal has been established, the locus of system control is time-dependent. Specifically, it is suggested that the cerebellar-premotor system is involved early in the programming process with little influence once execution has been initiated. In contrast, the basal ganglia-SMA system is predominantly involved late in the programming process and its influence appears to span both programming and execution. The remaining hypothesized pathways are preferentially involved in the execution of the motor program providing on-line sensorimotor adjustments. This flexible hierarchical organization allows for the operation of multiple, parallel subtask actions, each down-loaded and implemented via actions in the nonprimary and primary motor cortices. Together these distributed sensorimotor systems appear to interact dynamically to produce the coordinated movements of human speech.

ACKNOWLEDGMENTS

The authors express their gratitude to Eric Keller for his comments on an earlier version of this manuscript. Gratitude also is expressed to our bioengineer Richard Konopacki for his technical assistance.

This work was supported by National Institute of Neurological and Communicative Disorders and Stroke Grants NS20668, NS13274 and National Institute of Child Health and Human Development Grant HD-3352.

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