CHAPTER 2

NEURAL SYSTEMS IN SPEECH PHYSIOLOGY

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<tr>
<td>APA</td>
<td>arcuate premotor area (primate brain)</td>
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<tr>
<td>CA</td>
<td>anterior commissure</td>
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<tr>
<td>CAT</td>
<td>computerized axial tomography</td>
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<tr>
<td>CCZ</td>
<td>caudal cingulate zone</td>
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<tr>
<td>CM</td>
<td>corticomotoneurons</td>
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<tr>
<td>CNS</td>
<td>central nervous system</td>
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<tr>
<td>CP</td>
<td>posterior commissure</td>
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<td>CPG</td>
<td>central pattern generator</td>
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<td>CSA</td>
<td>cortical sucking area</td>
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<td>CT</td>
<td>cricothyroid</td>
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<td>EMG</td>
<td>electromyography</td>
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<tr>
<td>$F_n$</td>
<td>vocal fundamental frequency</td>
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<tr>
<td>GPe</td>
<td>globus pallidus (external section)</td>
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<td>GPI</td>
<td>globus pallidus (internal section)</td>
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<td>GTO</td>
<td>Golgi tendon organ</td>
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<td>Hz</td>
<td>Hertz</td>
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<td>HRP</td>
<td>horseradish peroxidase</td>
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<td>HSV-1</td>
<td>herpes simplex virus type 1</td>
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<td>IA</td>
<td>interarytenoid</td>
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ICMS  intracortical microstimulation
IEMG  integrated electromyography
JOR  jaw opening reflex
LCA  lateral cricoarytenoid
LL  lower lip
MD  medial dorsal thalamic nuclei
MENT  mentalis
MI  primary motor cortex
N  Newton
NA  nucleus ambiguus
NPA  nucleus para ambiguous
NPB  nucleus para brachialis
NPBm  nucleus para brachialis medialis
NRA  nucleus retroambigualis
NTS  nucleus tractus solitarius
OO  orbicularis oris
OOI  orbicularis oris inferior
OOS  orbicularis oris superior
$P_{co2}$  partial pressure of carbon dioxide
$P_{o2}$  partial pressure of oxygen
$P_a$  intraoral air pressure
PAG  periaqueductal grey
PC  Pacinian corpuscles
PCA  posterior cricoarytenoid
PD  Parkinson's disease
PET  positron emission tomography
PFC  prefrontal cortex
PMC  premotor cortex
$P_g$  subglottal air pressure
PTN  pyramidal tract neuron
R  right
RA  rapidly adapting
cCBF  regional cerebral blood flow
RCZa  rostral cingulate zone (anterior)
RCZp  posterior cingulate zone (posterior)
RFN  retrofacial nucleus
SA  slowly adapting
SAI  type I slowly adapting
SAII  type II slowly adapting
SI  primary sensory cortex
SMA  supplementary motor area
SNpr  substantia nigra (pars reticula section)
TA  thyroarytenoid
TBI  traumatic brain injury
UL  upper lip
VA  ventral anterior thalamic nucleus
VAmc  magnocellular part of VA
VAp  parvocellular part of VA
Vca  perpendicular line crossing the anterior commissure
VL  ventral lateral thalamic nucleus
VLM  medial part of VL
VLO  oral part of VL
VPL  ventral posterolateral division of ventral posterior nucleus
VPLc  caudal part of VPL
VPLo  oral part of VPL
VPm  ventral posteromedial division of ventral posterior nucleus

SPEECH AS A PHYLOGENETICALLY ADVANCED MOTOR SKILL

More than 100 years ago John Hughlings Jackson made several remarkable clinical observations on the function of the human cerebral cortex. Perhaps most controversial was his contention that "convolutions [of the cerebral cortex] contain nervous arrangements representing movements" (1874, p. 37). At about the same time, pioneering experiments on the electrically excitable motor cortex of the dog (Fritsch & Hitzig, 1870) and monkey (Perrier, 1873) made it clear that a disproportionately large part of the precentral motor cortex was allocated to control a very small proportion of muscles involved in the fine, precise movements of the body (cf. Evarts, 1981a, 1981b). Neurosurgical exploration of motor responses evoked from human precentral and postcentral gyri confirmed the disproportionately large representation for distal limb and vocal tract structures (Penfield & Rasmussen, 1950; Rasmussen & Penfield, 1947). The somatotopic localization of body parts in the sensorimotor cortex is shown in Figure 2–1. The sensory sequence is located in the postcentral gyrus, whereas the motor sequence is located in the precentral gyrus. This disproportionality, combined with the fact that many more neurons originating from the motor cortex form direct monosynaptic projections to brain stem and spinal lower motor neurons (Kuypers, 1964, 1973; Phillips & Porter, 1977), suggests that phylogenetic elaboration of the motor cortex has evolved to provide for finer motor control of existing muscles in new movement patterns.
Figure 2–1. Somatotopic localization of body parts in the human motor cortex. Parts of the body are scaled in proportion to the extent of their cortical representation. A similar pattern for somesthetic sense is found in the postcentral gyrus. (Reprinted with permission from “Further Studies of Sensory and Motor Cerebral Cortex of Man,” by T. Rasmussen and W. Penfield, 1947, Federation Proceedings, 6, 452; Carpenter, 1978).

One set of new movement patterns special to humans, involving phylogenetically “newer” structures of the brain, is speech. As a motor skill, speech is performed with speed and accuracy, improved with practice, flexible in achieving spatiotemporal goals, and virtually relegated to automaticity in the mature speaker (Netsell, 1982). Speech is often regarded as a highly adaptive, afferent guided motor control system (Abbs & Cole, 1982; Barlow & Netsell, 1986b, 1986c; Evarts, 1982; Gracco & Abbs, 1985; Grillner, 1982; Lubker & Gay, 1982; Lund, Appenteng, & Seguin, 1982; McClean & Smith, 1982; Smith, Moore, McFarland, & Weber, 1985). However, compared to our present understanding of limb motor control, actual data concerning the neurophysiology of speech production are unavailable. This is due in large part to (1) the biomechanical complexities of the vocal tract, (2) the complex organization of nuclei and pathways that transmit signals to and from structures of the vocal tract, (3) the wide range of forces and movements involved in speech production, and (4) the lack of an animal model for neurobiologic research into speech production.

**GENERAL PRINCIPLES OF MOTOR SYSTEMS FOR SPEECH**

There are several general signal-processing schemes applicable to the motor subsystems of the speech apparatus. It appears unlikely that any single scheme is adequate to describe the large number of central and peripheral neural networks active during speech. Rather, the motor act of speech undoubtedly involves
combinations of “hard-wired” neural networks similar to pattern generators, feedback and feedforward mechanisms, and a variety of adaptive neural processes. Some of the more general principles of motor control are considered in the following sections.

**Control Theory—Feedback and Feedforward Control Systems**

Control theory provides a general description of how systems of sensors and effectors can be designed to produce desirable behaviors. One major distinction in control theory is closed-loop versus open-loop control. According to Ito (1974, 1975) closed loop feedback control is the most basic form of engineering control wherein the output is returned to the input (Figure 2–2a). In some cases (negative feedback), the feedback information can be considered as an error signal that tends to correct system output deviations so that the desired output is achieved with greater accuracy. In other cases (positive feedback), the feedback signal tends to augment the system output, perhaps for the production of rhythmic behavior.

In contrast to closed-loop systems, open-loop systems produce output signals without the influence of feedback. The output signal may give rise to the input signal of another control system (feedforward) (Figure 2–2b). For example, an afferent signal generated from an open-loop pathway involved in jaw control may be directed to modify the activity of a different vocal tract structure such as the lips or tongue. In this example, the jaw is operating in an open-loop fashion since it is not affected by the output (afferent signal). However, some open-loop systems direct their outputs or feedforward the flow of afferent information to a different local network. In the previous example, the lips and tongue are recipients of feedforward sensory information originating from mechanoreceptors and muscle afferents of the jaw. In an open-loop, feedforward scheme such as the one given in the previous example, information from any relevant receptor system which is available may be utilized, at the time a movement is planned, to “construct” an optimized motor command (Ghez, 1979; Grillner, 1982; Weiss, 1941). Open-loop systems have the great advantage of speed: however, the precision of output in the affected structure is dependent upon the internal calibration (e.g., learned relations between muscle state and mechanosensory experience). Abbs and Gracco (1984) note “for control in a system where the boundaries are internal and hence extremely predictable (e.g., the coordinated movements for producing oral shapes for speech), open-loop prediction may be as precise as feedback with the additional advantages of speed and stability” (p. 720).

A substantially more complex form of neural control important for speech, involving some aspects of open-loop and closed-loop mechanisms, is known as efference copy. Efference copy provides the central nervous system with a comparator by which intended motor output can be compared with actual
output (Von Holst, 1954). Specifically, voluntary movements are thought to involve two sets of signals, both of which are feedforward in operation. The first neural signal is directed to the effector organs (muscles), and a second simultaneous “copy” signal is sent to sensory systems which function to preset the sensory system for the anticipated consequences of the motor act (Kelso & Stelmach, 1976).

Considerable progress has been made concerning the pathways which may mediate the efference copy mechanism. Anatomical studies carried out in the past ten years show that the input to the pontine nuclei (and other relays to cerebellum) is considerably stronger from the sensorimotor cortices (SI—primary somatosensory cortex, and MI—primary motor cortex) than from association areas. This pattern of organization favors a major role for the cerebrocerebellar projection in relation to providing the cerebellum with efference copy signals from MI together with sensory feedback from SI (see Evarts, 1982). As movement occurs, the efference copy signals from motor cortex to cerebellum would be compared to feedback signals reaching the cerebellum from SI, and error signals would be returned to MI via the dentate nucleus and ventrolateral thalamus (Dhanarajan, Ruegg, & Wiesendanger, 1977).

During efference copy, the nervous system utilizes information about issued motor commands (Grillner, 1982). During speech, rapid sequences of different motor acts (sounds and gestures) follow each other. Each motor act corresponds to a critical configuration of the oral cavity. The brain obviously “knows” not only which “speech gesture” it is planning but also which one is being carried out at that particular instant. Information about the expected “oral configuration” in any given instance is thus continuously available. It would be surprising if the CNS did not utilize this information to “design” the command signal to reach the new target in the most expeditious way. Efference copy information and afferent information used to “construct” movements is well suited for a system controlling rapid motor sequences (Grillner, 1982).

**Spatiotemporal Goals**

Speech is often described as a goal-oriented process. For example, the target of a vowel is thought to be encoded by the brain in terms of area-function (vocal tract shape) features that yield particular acoustic end products (Lubker & Gay, 1982). In kinematic terms, these area functions can be modelled by a series of constrictions along the length of the vocal tract that are executed using a large number of central mechanisms, including open loop- and feedback-dependent processes (Allen & Tsukahara, 1974; Eccles, 1977; Gurin & Levik, 1979).

The speech articulatory goal of lip rounding appears to be a learned, language specific act (Abelin, Landberg, & Persson, 1981). The motor goals represented by this act are chosen by the speaker as a part of learning his or her language. These speech motor goals are not “hard-wired” into the system at birth, but are encoded within the CNS as the human animal learns the communication system of the particular language into which it is born. A comparison of the lip rounding gesture in Swedish and American speakers indicates the Swedes move their lips further, with greater velocity and with greater precision of goal achievement, than do American speakers. These data also suggest that different motor control strategies may be required when these labial movements are used in meaningful speech as compared to when they are used in non-meaningful vocalizations. These observations concerning the goals of labial protrusion are thought to be true for a number of other speech articulatory movements involving respiratory, laryngeal, velopharyngeal, and perioral structures.

Evarts (1982) has suggested that afferent inputs mediated by forward-looking control systems (feedforward) can give rise to articulatory compensations that are complete in spite of dynamic mechanical perturbations impinging on one or more components of the articulatory apparatus. The implication is that the brain encodes a representation of the goal of articulation, and that afferent input is evaluated in terms of this goal. Therefore, goal
orientation is essential to voluntary movement because the articulatory movement goal depends upon the coordination of a great variety of open-loop and closed-loop reflex processes.

**Motor Equivalence**

Motor equivalence is defined as the capacity of a motor system to achieve the same end product with considerable variation in the individual components that contribute to that output (Hebb, 1949). In achieving a particular vocal tract goal, the specific contributions of individual articulators may vary from one production of a particular element to another, as long as the desired end product is achieved (Lashley, 1951; MacNeilage, 1970; Netsell, Kent, & Abbs, 1980).

Compensatory vowel articulation, presumably involving the operation of motor equivalence, is defined as the ability of speakers to generate acceptable vowel qualities in spite of a fixed position of the mandible or other structures (Lubker & Gay, 1982). Producing vowels that require a relatively closed jaw position, such as /i/ or /u/ with a spacer held between the teeth fixing the jaw into a very open position confronts a speaker with a new, or at least unfamiliar, task as compared with producing the same vowel under normal, non-constrained conditions. Several studies have shown that under such conditions speakers are able to produce vowels that are acoustically correct with regard to formant frequency locations at the first glottal pulse (Gay, Lindblom, & Lubker, 1981; Lindblom, Lubker, & Gay, 1979; Lindblom & Sundberg, 1971; Netsell, Kent, & Abbs, 1978). These studies have demonstrated that the tongue is capable of reorganizing motor output. An example of this phenomenon, taken from Netsell et al. (1978), is shown in Figure 2–3. The reorganization in tongue height appears to be a goal-oriented process. In the example shown, compensatory adjustment in tongue height was maximum at points along the vocal tract where the normal vowel exhibited constrictions. At least for adult speakers, goal-oriented motor reorganization appears to require no learning since speakers are able to produce the correct vowels under bite-block conditions on the very first attempt, only a few seconds after inserting the bite-block and being cued for what vowel to produce.

![Figure 2–3. Vowel reorganization. Mid-sagittal line drawings of the upper airway for /i/ vowel productions by three adult speakers. Left side: superimpositions of lips, tongue, and pharynx when the jaw was free to move (solid lines) and held in a fixed position (dashed lines). Right side: superimpositions of tongue shapes referenced to the jaw which shows the extent of adjustment made with the tongue to achieve the similar tongue positions on the left. (Reprinted with permission from "The Organization and Reorganization of Speech Movement," by R. Netsell, R. D. Kent, and J. Abbs, 1980, Society for Neuroscience Abstracts, 6, 462.)](image-url)
**Motor Programs**

Broadly defined, the term “motor program” refers to the differentiation of knowledge into an appropriate movement configuration for action. Thus, CNS information concerning the current position of a limb, the location of a target, the forces acting on the limb, and other factors is used to define a sequence of muscle contractions to produce the desired movement.

Early notions portrayed the motor program as operating independently of peripheral feedback (Keele, 1968). However, recent neuroanatomic and neurophysiologic findings suggest that the motor program contains a set of specifications for the selection, sequencing, and timing of desired muscle groups to generate “target” forces and movements that may be modified by afferent feedback (Evarts, Bizzi, Burke, DeLong, & Thach, 1971; Newell, 1978; Newell & Barclay, 1982; Sterngerg, Monsell, Knoll, & Wright, 1978). Evarts (1982) has suggested that the motor program, processed in subunits of varying complexity and subject to modification by afferent input, is very important for speech motor control.

Another interesting concept regarding the operation of motor programs is the mechanism of “switching” (Pearson, 1976). Pearson has shown that the “switching” of the locomotion motor program from swing to stance is triggered by sensory input. Feedback during locomotion can be thought of as having two broad functions, the first being to switch the motor program from one phase to the next and the second being to modify the motor output within a single phase (Evarts, 1982). Tactile inputs are especially important in switching, while proprioceptive feedback provides continuous control of motoneuronal discharge during movement. From this example, one may hypothesize that the burst of afferent activity resulting from labial contact during the production of /p/ may provide the triggering sensory input to “switch” the motor program to the next phase.

Another creative implementation of the “program” concept in the context of speech motor control has been offered by Grillner (1982). In this scheme, speech is modeled as a sequence of individual sounds, each of which is produced by highly automated learned motor programs. Learned and innate movements, in some cases, are thought to share common neural mechanisms. In the simplest case the neural substrate for a learned motor act may thus only constitute a slight modification of a basic innate motor program. Grillner’s model implies that learned movements may result from a recombination of parts of different innate neural motor programs and other motor mechanisms.

**Central Pattern Generation**

A pattern generator may be thought of as a highly organized set of neurons which, when activated, yield a relatively stereotypic response such as locomotion or breathing. This concept has been applied to the control of more discrete, fractionated motor acts as well. For example, if all neurons in a given spinal network which normally act to flex and extend the upper limb are activated, locomotion will result. If only part of these neurons are activated another motor act will result such as finger movement or shoulder flexion. During evolution, more precise control of smaller fractions of pattern generators may account for the movement precision associated with manipulation and speech (Grillner, 1982).

There are well developed motor systems for mastication and swallowing with the pattern generating networks residing mainly in the brainstem (see Lund et al., 1982). Sound production results from a concomitant control of the respiratory airflow and the shape of the oral cavity. Grillner (1982) has suggested that the innate programs to control different sound sequences in animals may result from the coordinated actions of the respiratory brainstem pattern generator and fractions of the central pattern generators for mastication and swallowing. Given the diversity of sound production in humans, it logically follows that the CNS would require separate access to small fractions of the respective pattern generators and perhaps also directly...
to some motoneurons. Grillner goes on to state that learning of a sound motor program for speech would involve learning to combine in time the appropriate fractions of the motor apparatus.

Neural circuits in the spinal cord and brainstem play an important role in motor coordination. Segmental reflexes provide the nervous system with a set of elementary patterns of coordination that can be activated either by sensory stimuli or by descending signals from the brain stem and cerebral cortex. Most brainstem reflexes, including the perioral reflex, have relatively complex circuits (compared to stretch reflex). Although a few reflexes have primarily local actions on single muscles, most coordinate the actions of groups of muscles. With the exception of the jaw reflex, most reflex pathways are polysynaptic, having one or more interneurons interposed between the sensory and motor neurons (Gordon, 1991). This feature allows descending signals as well as other afferent inputs to modify the expression of the reflex, including the activation of networks of interneurons for the timing of reflex components. Divergent and convergent connections in reflex pathways play an important role in the spatial organization of reflex behaviors, determining which sensory inputs are enhanced or suppressed and which muscles contract or relax. Other types of connections determine the temporal organization of reflexes. For example, reverberating circuits—closed circuits of interneurons that re-excite themselves—are responsible for some reflexes that outlast the stimulus. And finally, more complex temporal patterns are produced by networks of interneurons called central pattern generators (CPG). Examples of CPG’s involving muscles of the vocal tract include respiration, suck, and mastication.

**NEURAL MOVEMENT CONTROL SYSTEM**

The neurophysiology of speech is the study of how the brain selects, sequences, and activates the muscles of the chest wall and vocal tract for speech movements. In the following sections, special consideration will be given to the functional organization and response properties of brain structures important in a general model of movement control, and where data permit, reference will be made to speech movement control. The significance of movement-related brain structures will be evaluated in terms of their anatomical and functional connectivity, response to electrical and/or natural stimulation, the effects of lesions, and their hypothesized role for speech.

**Connectivity of Primary Motor and Sensory Cortices**

Traditionally, a great deal of attention has been devoted to the role of the cerebral cortex in voluntary motor control. This is due to the fact that a substantial amount of information is processed in somatotopically represented areas of cortex located along the anterior and posterior margins of the central sulcus, including (1) output instruction sets for muscle activation, and (2) input data concerning the state of muscle contraction, position, and cutaneous sensation.

Much of what is known about the extent and subdivision of the primate sensorimotor cortex can be traced to the work of Woolsey and his colleagues (Woolsey, Settlage, Meyer, Sencer, Humay, & Travis, 1951). Often regarded as the "entry point" to the final common pathway, the primary motor cortex (MI) consists of Brodmann's area 4 and possibly the posterior strip of area 6. The approximate locations of areas 4 and 6 are shown in Figure 2–4. MI is dominated by somatosensory input. Pyramidal tract neurones (PTNs) located within the fifth layer of the motor cortex (inner pyramidal cell layer), serve as summing points of central command signals and peripheral feedback signals, and allow for rapid adaptations imposed by the environment via the corticospinal and corticobulbar tracts (Wiesendanger, Hummelsheim, & Bianchetti,
Figure 2–4. Schematic subdivision of the sensory and motor cortices based on Brodmann's areas. Primary motor and premotor regions of cerebral cortex indicated by numerals 4 and 6, respectively.

Note that the pyramidal tract is not exclusively the output of the motor area, rather its origin includes portions of the parietal and postcentral brain sites (Granit, 1977). Posteriorly, MI is bounded by the cytoarchitectonically distinct primary sensory cortex (SI). Other features that distinguish MI from SI include the origins of inputs. For example, areas 3a, 3b, 1, and 2 of SI receive thalamic inputs from ventralis posterior lateralis pars caudalis (VPLc), whereas MI receives inputs from nuclei ventralis posterior lateralis pars oralis and ventralis lateralis caudalis (Friedman & Jones, 1981).

The postcentral gyrus of the cerebrum constitutes the primary receiving areas for somatosensory information originating from mechanoreceptors located within skin, joints, and muscle (Mountcastle, 1957). The SI consists of several interconnected subareas (Jones & Powell, 1970; Pandya & Kuypers, 1969). For example, tract tracing methods used in the study of primate SI have led to the identification of subareas 3a, 3b, 1, 2, and 5 (see Evarts, 1981a; Manger, Woods, Jones, 1995; Merzenich, Kaas, Sur, & Lin, 1978). Each of these subareas receives different types of somatosensory information. Area 3a responds to stimulation of group I muscle afferents (derived from muscle spindles encoding position and velocity), area 3b is activated by slowly adapting mechanoreceptors in the skin, area 1 is activated by rapidly adapting cutaneous mechanoreceptors, and area 2 is primarily activated by joint movements.

Somatosensory cortex is generally considered to contain a number of complete somatotopic representations based upon mapping experiments in a number of species of primates (Kaas, Nelson, Sur, Lin, & Merzenich, 1979; Manger, Woods, & Jones, 1995; Merzenich et al., 1978; Paul, Merzenich,
Goodman, 1972). The region spanning cytoarchitectural areas 3a, 3b, 2 and 1, traditionally called S1, contains at least three complete representations and possibly a fourth, although the organization of 3a is still somewhat ambiguous (Kaas et al., 1979). Each complete map of the body corresponds to one cytoarchitectural area. Although there are discontinuities, the organization within each area is basically topographical.

The work of Vogt and Pandya (1978) and Jones, Coulter, and Hendry (1978) shows an inverse relation between the strength of thalamocortical inputs to the subsectors of the primary somatosensory cortex and the input of corticocortical projections to these subsectors. For example, subarea 3b, which has a thick granular layer, is dominated by primary afferent input from the thalamus with relatively little input from other cortical areas (Jones et al., 1978). By contrast, the lesser thalamic input from the ventrobasal complex to areas 1 and 2, compared with area 3b, is correlated with a relatively thin internal granular layer in these cytoarchitectonic areas (Jones, 1975). The output of area 3b via corticocortical projections is directed back into areas 1 and 2, but this projection is not reciprocated: instead of projecting back to area 3b, the major projection of areas 1 and 2 is to area 5 and MI. Thus, mechanoreceptor information reaching area 3b is not sent directly on to MI but is further processed in area 2, which also receives dense corticocortical input from areas 3b and 1.

**Somatotopy of Orofacial/Laryngeal Structures**

Maintenance of continuity in the neural representation of body parts, termed somatotopy, is a feature of numerous structures within the nervous system including the primary sensorimotor cortex (Woolsey et al., 1951). The amount of cortex devoted to a given muscle is presumed to reflect the frequency and intensity of the use of a muscle in normal behavior (Penfield & Rasmussen, 1950). Examples of body structures mapped to relatively large areas of the sensorimotor cortex include the fingers and hand for object manipulation and exploration and the lips, tongue, jaw, velopharynx, larynx, and chest wall for speech production. Discrete activation of individual muscles (individuation) in a finely graded manner (fractionation) is characteristic of skilled movements which have evolved with the elaboration of MI (Evarts, 1985; Kuypers, 1964; Phillips & Porter, 1977).

Early investigations using surface stimulation of the primary motor cortex suggested the existence of a rather simple somatotopic representation of movements (Lauer, 1952; Penfield & Boldrey, 1937; Walker & Green, 1938; Woolsey, 1958). This notion has been modified in recent years by a number of studies using intracortical microstimulation (Asanuma & Sakata, 1967; Clark & Luschei, 1973; Coul, Cusick, Pons, & Kaas, 1986; Huang, Sirisko, Hiraba, Murray, & Sessle, 1988; McGuinness, Siversten, & Allman, 1980; Sessle & Wiesendanger, 1982; Sirisko & Sessle, 1981, 1982, 1983; Zealor, Hast, & Kurago, 1983). The presentation of small electrical currents (<30 microamps) to motor neurons within the depths of the precentral gyrus has revealed the presence of multiple representations of individual muscle movements. For example, similar movements have been observed to occur at different depths in the same electrode penetration and within different rostrocaudal planes separated by as much as 6–8 mm (Huang et al., 1988). Threshold currents typically yield discrete responses limited to a small focus of movement in part of a muscle. Successive stimulation points separated by less than 250 microns (μm) can yield quite different muscle responses (Huang et al., 1988; McGuinness et al., 1980; Sessle & Wiesendanger, 1982). The discrete nature of these responses is explained, in part, by the columnar organization of the motor cortex (Asanuma, 1973; Asanuma & Arnold, 1975; Asanuma & Rosen, 1972; Asanuma & Sakata, 1967; Mountcastle, 1957).

The existence of multiple representations of muscles and movements in the facial motor cortex may be a reflection of the biomechanic
complexities and anatomic diversity of muscle subgroups in the orofacial region. Studies of orofacial kinematics and muscle activity in human speakers have shown that similar goals (movements) may be achieved by different patterns of muscle activation. Additional experiments are needed to consider the possibility that the "identical movements" reported in the ICMS studies are actually associated with different patterns of muscle activation. Therefore, a degree of caution is reserved in the suggestion that ICMS applied to different cortical loci yield exactly the same movement.

In primates, the neural representation of facial muscles is clustered in the rostral bank of the lateral central sulcus which is contiguous with the rostral, medial, and caudal borders of the more lateral regions representing the jaw and tongue muscles (Huang et al., 1988; Kuypers, 1981). An example of a somatotopic map of the face, tongue, jaw, and larynx for the monkey is shown in Figure 2-5. The neural representation of the tongue occupies the center of the precentral gyrus and extends from above inferiorly into the ovoid region of the laryngeal representation. The tongue and laryngeal regions are flanked by an anterior and posterior face field which are bridged in the middle, forming a horseshoe shaped face representation. The more exterior or lateral aspect of each face field represents the more lateralized muscles of the face such as the zygomaticus, and the central bridge area represents midline facial muscles, such as the orbicularis oris and mentalis (Zealard et al., 1983).

Contrary to the discrete nature of responses evoked in orofacial regions, the zones encountered in the laryngeal region are multifacelike zones involving the posterior criocarotid and one or more adductor or extrinsic muscles (Zealard et al., 1983). The widespread cortical representation of the posterior criocarotid is consistent with its postural stabilizing action on the arytenoid during the sliding phases of adduction and abduction (Fink & Demarest, 1978). Neural instructions arising from the ovoid region may be important for voluntary laryngeal activities requiring modification of the glottis.

Another issue concerns the number of complete somatotopic maps. Some investigators have suggested that at least two somatotopic representations of the facial musculature, each coursing in a mediolateral direction and separate, exist in primate mo-

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**Figure 2-5.** Somatotopy of orofacial structures. Map of face, tongue, jaw, and larynx zones for a monkey obtained using intracortical microstimulation. Triangles indicate laryngeal zones (large triangles thresholds less than 10 μamps, small triangles thresholds 6-10 μamps). F—face, T—tongue, and J—jaw zones. (Reprinted with permission from "Functional Organization of the Primary Motor Cortex Controlling the Face, Tongue, Jaw, and Larynx in the Monkey." In I. R. Titze and R. C. Scherer [Eds.], *Vocal Fold Physiology: Biomechanics, Acoustics and Phonatory Control*, pp. 57-73. Denver: The Denver Center for the Performing Arts.)
tor cortex (McGuinness et al., 1980; Zealear et al., 1983). This conclusion has been challenged by the observation that the two mediolateral clusters are in fact connected medially (Huang et al., 1988). Regarding this issue, Huang et al. (1988) stated “Indeed, the present study revealed no clear and consistent distinction of two separate and discontinuous rostral and caudal representations of the orofacial muscles in the precentral cortex” (p. 816).

Ipsilateral facial movements only occupy approximately 20% of the positive ICMS sites in the monkey face motor cortex of either hemisphere (Huang et al., 1988). Most of the ipsilateral representation was located in a narrow band in the anterior region (rostral) of the face motor cortex. Generally speaking, ipsilateral facial representations are located in motor cortex regions where contralateral movements are sparsely represented (see Figure 2–6, after Huang et al., 1988). Of the ipsilateral muscles represented, the orbicularis had the largest representation (45%) followed by zygomaticus major (24%) and platysma (20%). These incidence figures are commensurate with the kinematic role of these muscle subgroups for speech, swallowing, mastication.

Figure 2–6. Movement representation as defined by the technique of intracortical microstimulation for face, jaw, and tongue musculatures when right cortex of a monkey was unfolded. B, buccinator; C, levator anguli oris; H, digits; J, jaw open; j, jaw close; M, mentalis; N, nasalis; O, orbicularis oris; P, platysma; T, tongue protrusion; t, tongue retraction; Z, zygomaticus major; ipsilateral muscles are encircled. (Adapted from Huang, Sirisko, Hiraba, Murray, and Sessle, 1988.)
tion, and facial gesturing. Moreover, the most significant and residual movement deficits associated with traumatic injury to the facial motor cortex are often associated with these perioral muscles.

Consistent with these physiologic findings, neuroanatomic evidence indicates that projections from MI to the lower facial motor nuclear region are direct and predominantly contralateral in nature (Jenny & Saper, 1987). A large proportion of these corticobulbar projections are known to originate from the rostral bank of the central sulcus and the convexity of the precentral cortex (Huang et al., 1988; Kuypers, 1981). Electromyographic activity evoked by ICMS was characterized by latency values ranging from 10 to 45 ms for the face, jaw, and tongue muscles.

The findings from the microstimulation studies reviewed in the previous sections strongly suggest the face motor cortex is organized to coordinate the activity of numerous muscles within the face, jaw, and tongue. Moreover, a close correspondence exists between the outputs from these cortical regions and input channels originating from mechanoreceptors and muscle spindle afferents considered important for fine motor control.

**Frontal Lobe Inputs to Primary Motor Cortex**

One of the classic concepts concerning the cortical control of movement is that “premotor” areas exist in the frontal lobe that have bilateral and direct access to the primary motor cortex (von Bonin & Bailey, 1947; Matsumura & Kubota, 1979; Muakkassa & Strick, 1979; Pandya & Vignolo, 1971). These “premotor” areas are thought to contribute to the organization of skilled movement and the programming of motor cortex output. Tract tracing techniques (horseradish peroxidase) in primates (Muakkassa & Strick, 1979) revealed the location of four spatially separate and somatotopically organized premotor areas with projections to the primary motor cortex (Figure 2–7). Following injections into the face area of the motor cortex, labeled neurons were found in separate areas of frontal lobe including: (1) the inferior limb of the arcuate sulcus (caudal bank), (2) rostrally in the supplemental motor area, (3) rostrally in the ventral bank of the cingulate sulcus, and (4) the lateral bank of the inferior precentral sulcus. The densest projections originate from the premotor cortex (PMC) and the supplementary motor area (SMA). These premotor areas represent important elements in parallel pathways that influence motor cortex output and motor behavior.

**Supplementary Motor Area (SMA)**

The SMA occupies the medial portions of area 6 and has been considered to function in programming motor sequences, including preparatory states for forthcoming movements (Brinkman, 1984; Kornhuber & Deecke, 1965; Orgogozo & Larsen, 1979; Roland, 1985; Roland, Larsen, Lassen, & Skinhøj, 1980; Wiesendanger et al., 1985). This region of the brain is also implicated in the control of posture, gating motor cortical reflexes, and initiating motor cortex output and movement (Brinkman & Porter, 1978; Pandya & Vignolo, 1971; Tanji & Taniguchi, 1978; Wiesendanger, Seguin, & Kunzle, 1973). Electrical stimulation of SMA results in complex, coordinated movements of the contralateral limbs and body, with occasional ipsilateral limb movements (Bancaud & Talairach, 1967; Penfield & Welch, 1951). This is in sharp contrast to the MI, where focal stimulation elicits highly localized and discrete movements of the contralateral musculature (Tanji, 1985). Studies of movement disturbance after ablation of SMA have shown that the effects are subtle, often transient, with no apparent paralysis. Again, this contrasts remarkably with effects of primary motor cortex lesions.

Some SMA neurons are activated by tactile inputs. However, the magnitude of such responses is smaller than that observed in the primary motor cortex. SMA units responsive to passive arm displacements have been iden-
Figure 2-7. Frontal lobe inputs to MI. A: location and approximate spatial extent of the four “premotor” areas which project to the primary motor cortex in primates are indicated by symbols (posterior arcuate, filled circles; precentral sulci, triangles; SMA, squares; cingulate sulcus, open circles). B: body representation in each of the four “premotor” areas depicted in A is indicated by the words “face,” “arm,” and “leg.” (Reprinted from Brain Research, 177, by K. F. Muakkassa and P. L. Strick, “Frontal Lobe Inputs to Primate Motor Cortex: Evidence for Four Somatotopically Organized ‘Premotor’ Areas,” pp. 176–182. Copyright 1979, with permission from Elsevier Science.)

tified in the posterior region where microstimulation effects in arm muscles can be elicited. The shortest latencies are around 15 msec (Wiesendanger et al., 1985). Because of the smaller magnitude of the movement-related activity, and the lack of strong correlations of neuronal activity with movement onset, the SMA is functionally more remote from the peripheral motor apparatus than the primary motor cortex (Wise & Tanji, 1981). SMA neurons are also responsive to visual and auditory signals at significantly shorter latencies than those of precentral cortex. The functional significance of these inputs remains to be studied. Tanji and co-workers (1980) have found that a significant number of SMA neurons manifest instruction-induced changes of activity during the period intervening between the instruction and a perturbation-triggered movement. These particular SMA neurons, however, are not active in association with the movement itself. It appears that SMA neurons may function in a conditional manner, dependent upon behavioral context or intentional set, or as triggered premotor inputs to summing PTNs in the primary motor cortex (Evarts, Shinoda, & Wise, 1984; Tanji, Taniguchi, & Saga, 1980).

The SMA, among other areas of the cerebral cortex, is considered an important region for speech motor control. Penfield and Roberts (1959), considered the SMA to be of special importance for the programming and execution of speech. The SMA, containing a somatotopic representation of vocal tract structures (Brinkman & Porter, 1978; Fried, Katz, McCarthy, Sass, Williamson, Spencer, & Spencer, 1991; Muakkassa & Strick, 1979), is
strategically situated as one of the primary premotor inputs to MI for speech motor programming.

Neuroimaging techniques have provided new and exciting data on the activity of motor areas of the brain, including the SMA, during speech motor processing in normal and neurologically impaired speakers. For example, data generated from computerized axial tomography (CAT) and regional cerebral blood flow studies (rCBF) support the hypothesis that the SMA functions as a premotor input to the MI, via Broca's area, for speech production in normal speakers (Gelmers, 1983; Jonas, 1981, 1987; Larsen, Skinhoj, Soh, Endo, & Lassen, 1977; Lassen, Ingvar, & Skinhoj, 1978; Roland, Skinhoj, Larsen, & Lassen, 1977; Roland et al., 1980). The SMA appears to be directly involved in the planning of propositional speech. In fact, small lesions limited to the SMA have been found to cause a pure disorder of speech initiation (Freedman, Alexander, & Naeser, 1984). The left SMA, more than the right SMA, is active during the formulation of novel speech. The output from the left SMA is processed by the ipsilateral Broca's area before being received by both the left and right MI for execution of the motor plan. Additional evidence that the SMA is involved in speech motor planning versus motor execution is realized from the observation that rCBF increases are found in the SMA while normal subjects think about counting without actually speaking (Lassen et al., 1978). Consistent with their hypothesized role as motor executors, both Broca's area and MI show no increases in rCBF during this task. These observations are somewhat at odds with an earlier notion in which the SMA, contrary to Broca's and Wernicke's areas, was hypothesized to be more involved in actual modulation of speech output than in ideational formulation (Botez & Barbeau, 1971).

If the left SMA is damaged, the normal sequence of events for propositional speech is impaired or impossible. However, in the presence of left SMA damage, nonpropositional speech remains intact since the adequate stimuli have direct access to MI from subcortical centers. In fact, if the left SMA is sufficiently damaged, it may be impossible for the subject to inhibit nonpropositional speech (swearing, etc.) (Jonas, 1987). Therefore, the SMA also plays an important inhibitory role in selectively gating inputs to MI that would otherwise disrupt an ongoing speech motor program. The following points summarize the role of the SMA in speech (Jonas, 1987).

1. **Left SMA programs Broca's area which in turn activates MI for propositional speech.**

2. **Voluntarily activated nonpropositional speech (counting, alphabet) is initiated via a brief burst of SMA-Broca activity, after which MI operates independently.**

3. **MI may be activated for automatic nonpropositional speech in the absence of SMA-Broca activity (verbal response to an aversive stimulus).**

4. **The SMA also serves to regulate the amount and type of information flowing into MI. The SMA is strategically situated in the brain to activate MI for propositional speech and is also capable of selectively inhibiting conflicting inputs from subcortical centers responsible for initiating nonpropositional speech.**

Contemporary analysis of PET data in humans (Picard & Strick, 1996) suggests that the medial wall of the superior frontal gyrus (Brodmann area 6) contains two distinct motor areas, similar to those found in a monkey. These two areas appear to be distinguishable based on task complexity. A schematic of the medial surface of the brain, partitioned according to Talairach space is shown in Figure 2–8. The intersection of the bicomissural line (CA–CP) and a perpendicular line (Vca) that passes through the anterior commissure at midline defines the origin of Talairach space. Lod activated during the execution of simple tasks and somatosensory stimulation of the forearm are located caudal to the Vca line. More complex tasks produce increases in rCBF in areas rostral to the Vca line. Picard and Strick (1996) refer to these areas as pre-SMA (complex tasks—rostral to Vca line),
and SMA proper (simple tasks—caudal to Vca line). These functional areas within the medial aspect of the superior frontal gyrus are shown in Figure 2–9 for movements involving the arm, eye, and vocal tract. Complex versus simple speech tasks are organized along the rostral–caudal axis. Activation of SMA proper is enhanced during automatic performance of learned movements such as repetition of words, and the production of simple verbal associations. Even voluntary contraction of the chest wall during expiration activates SMA proper (Ramsay, Adams, Murphy, Corfield, Grootoord, Bailey, Frackowiak, & Guz, 1993). By contrast, the pre-SMA is heavily engaged during the initial stages of speech motor skill acquisition, including silent verb generation, complex ordered tasks, and novel associations.

**Premotor Cortex (PMC)**

The lateral portion of area 6 is known as the premotor cortex (PMC) in humans and as the arcuate premotor area (APA) in monkeys. This region of the cerebral cortex receives substantial inputs from the primary visual cortex (Pandya & Kuypers, 1969) and is considered to be involved in visually guided

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*Figure 2–8. A schematic of the medial surface of the brain, partitioned according to Talairach space. The intersection of the bicommissural line (CA–CP) and a perpendicular line (Vca) that passes through the anterior commissure at midline defines the origin of Talairach space. Numbers show approximate location of Brodmann's cytoarchitectonic areas. Boundaries are indicated by dashed lines in instances where they do not course along sulci. Boxes delimit the areas enlarged in Figures 2–9 and 2–11. (Adapted from Talairach & Tournoux, 1988; reprinted with permission from Oxford University Press from "Motor Areas of the Medial Wall: A Review of Their Location and Functional Activation," by N. Picard and P. L. Strick, 1996, Cerebral Cortex, 6, 342–353.)*
movements (Haaxma & Kuypers, 1975; Kubota & Hamada, 1978; Moll & Kuypers, 1977; Wiesendanger et al., 1985). Some neurons in the PMC also are responsive to somatosensory input resulting from passive and/or active limb displacement, with minimal latencies ranging from 13–15 msec (Bignall & Imbert, 1969; Rizzolatti, Scandolara, Matelli, & Gentilucci, 1981a, 1981b; Wiesendanger et al., 1985).

Similar to SMA, the overall responsiveness of PMC neurones to somatosensory stimuli is weaker than for neurones in MI. Wiesendanger and others suggest that the somatosensory input is directed primarily to neurones at the input stage of the cortical compartment and perhaps less to the output neurones. Since most cells within the PMC are responsive to visual inputs, this region of the brain may be less important than the SMA.
for speech production since visual information is not considered essential for execution of this motor skill.

**Cingulate Motor Areas**

Traditionally, the cingulate cortex has been considered to be part of the limbic system (Broca, 1878; Papez, 1937). The results from recent experiments in primates clearly indicate that the cingulate cortex is not homogeneous, and that portions of this region are more directly involved in somatic motor control than others (Dum & Strick, 1991, 1993). In fact, these authors have divided cingulate cortex into two subdivisions. The first is known as the cingulate gyrus and is found on the interhemispheric surface. The cingulate gyrus is thought to be involved in the expression of emotions, control of autonomic function, attention to sensory inputs, and processing of noxious stimuli. The cingulate gyrus lacks direct projection to primary motor cortex. The second subdivision of the cingulate cortex is the cingulate sulcus which can be found on the dorsal and ventral banks of that sulcus. Intracortical stimulation of these surfaces is capable of evoking a wide variety of movements in forelimb, hindlimb, trunk, and orofacial muscles. Stimulation of the rostral cingulate motor cortex was effective in producing facial movements and is consistent with the location of the medial wall areas that project to the face representation of primary motor cortex (Muakkassa & Strick, 1979). The central representation of facial and neck/upper trunk movements for two species of macaque monkeys is shown in Figure 2–10a and 2–10b. Dum and Strick (1991, 1993) found three separate cingulate motor areas, each of which has dense interconnections with primary motor cortex. Limb representations within the three cingulate motor areas project monosynaptically to the spinal cord and orofacial representations within the cingulate sulcus project to appropriate brain stem motor nuclei. Picard and Strick (1996) completed a compelling synthesis of their anatomical work on motor areas of the medial wall in relation to 29 published reports using positron emission tomography (PET). The co-registration between anatomical and functional imaging techniques is remarkable. As shown in Figure 2–11, the location of rCBF increases in the anterior cingulate region for speech production tasks is found among two primary sites in the rostral cingulate zone (anterior to the Vca line of Talairach). By contrast, simple arm movements were localized to the caudal cingulate zone (generally proximal and posterior to the Vca line of Talairach). Studies of regional cerebral blood flow are consistent with anatomic and microelectrode maps of the anterior cingulate region. The functional significance for multiple cingulate motor areas lies in the hypothesis that each premotor area “may differentially generate and/or control specific aspects of motor behavior” (Dum & Strick, 1991). These areas appear to be involved in the planning, preparation, and execution of motor acts (Shima, Aya, Mushiake, Inase, Aizawa, & Tanji, 1991). Motor areas of the cingulate sulcus are target of outputs from the basal ganglia and cerebellum. These features of organization are similar to other premotor areas in the frontal lobe and favor the cingulate motor areas as an integral neural substrate for the central control of movement (Dum & Strick, 1993).

**Subcortical-Thalamocortical Relations**

**Basic Organization**

A topic of special significance in movement control concerns how two of the major subcortical motor systems, the cerebellum and the basal ganglia, differentially influence premotor and motor cortex. Early views on the anatomical connections between cortical and subcortical motor systems specified that efferents originating from cerebellar, pallidal, and nigral nuclei are highly segregated, manifesting little overlap in their projections to the ventrolateral thalamus (Asanuma, Thach, & Jones, 1983b; Carpenter, Nakano, & Kim, 1976; DeVito & Anderson, 1982; Kalil, 1981;
Figure 2–10. Maps of orofacial and neck movements evoked by intracortical stimulation of the motor areas on the medial wall. The medial wall of the hemisphere has been unfolded and reflected. A. Movements evoked by long-train intracortical stimulation in a macaque (Macaca mulatta). (Modified from Figure 4 of Mitz and Wise, 1987.) B. Movements of the face and neck evoked by normal and long-train intracortical stimulation in a different species of macaque (Macaca fascicularis). All movements were contralateral unless otherwise noted. The key applies to both A and B. (Adapted with permission of the Journal of Comparative Neurology from Luppino, Matelli, Camarda, Gallese, & Rizzolatti, 1991.) **Key:** arcuate sulcus, ArS; rostral limit of the superior limb of the arcuate sulcus, ArSS; cingulate sulcus, dorsal bank, CgSd; cingulate sulcus, ventral bank, CgSv; cingulate gyrus, CgG; central sulcus, CS; superior frontal gyrus, medial wall, SGm; frontal granular cortex, Fgc.
Figure 2-11. The location of rCBF increases in the anterior cingulate region is plotted in Talairach space. Symbols correspond to a synthesis of experimental observations reviewed by Picard and Strick (1996) from a pool of 29 studies in the PET literature. Circles designate activations observed during relatively simple tasks, whereas diamonds indicate activations related to more complex attributes of motor behavior. Activation loci related to arm movement tasks, eye movement tasks, and verbal tasks are plotted separately in A, B, and C. In D, activations during oculomotor and speech tasks are designated together as FACE. This alternation divides the rostral cingulate zone into anterior (RCZa) and posterior (RCZp) sectors activated in relation to complex tasks, most notably including speech. The caudal cingulate zone (CCZ) is defined by the activations associated with simple arm movement tasks located just in front and behind the Vca line. (Reprinted with permission from Oxford University Press from "Motor Areas of the Medial Wall: A Review of Their Location and Functional Activation," by N. Picard and P. L. Strick, 1996, Cerebral Cortex, 6, 342-353.)

Kim, Nakano, Jayarman, & Carpenter, 1976; Kuo & Carpenter, 1973; Mehler, 1971; Percheron, 1977; Stanton, 1980). The basic pattern of termination of subcortical efferents in the thalamus was thought to include: (1) rostral portions of the deep cerebellar nuclei project to the motor cortex via the nucleus ventralis posterior lateralis pars oralis (VPLo); (2) caudal portions of the deep cerebellar nuclei project to the PMC via area X; and (3) the globus pallidus projects to the SMA via the nucleus ventralis lateralis pars oralis (VLo) (Asanuma, Thach, & Jones, 1983a; Jones, Wise, & Coulter, 1979; Kalil, 1981; Kusama,
Mabuchi, & Sumino, 1971; Percheron, 1977). Schell and Strick (1984), using retrograde transport in primates, demonstrated that the SMA, APA (PMG), and MI receive thalamic input from separate, cytoarchitectonically well-defined subdivisions of the ventrolateral thalamus. Thus, each thalamocortical pathway was thought to be associated with a distinct subcortical input (Schell & Strick, 1984).

Details concerning the anatomical relations between cerebellum, basal ganglia, and motor cortex have been refined considerably over the past decade largely as a result of improved tract-tracing techniques. The recent work of Strick and colleagues (Hoover & Strick, 1993, 1998; Middleton & Strick, 1996; Strick, Dum, & Picard, 1995; Strick, Hoover, & Mushiake, 1993) has essentially redefined our understanding of basal ganglia–thalamic-cortical and cerebello–thalamocortical pathways in monkey using retrograde transneuronal transport of herpes simplex virus type I (McIntyre-B strain). Three days after injection of this virus into select regions of the motor cortex, densely labeled neurons were found in subdivisions of the VL (VPLo, VLo, and VPM) thalamus that are known to innervate MI. If survival time is increased to 5 days, the virus can pass from first-order neurons to second-order neurons in deep cerebellar nuclei (dentate and interpositus) and to second-order neurons in the output nuclei of the basal ganglia located in the internal segment of the globus pallidus (GPi). Additional information concerning these anatomical relations is given in the following sections.

**Basal Ganglia–Thalamocortical Relations**

The principal efferents from the basal ganglia include those originating from the globus pallidus and the substantia nigra. Efferents from the internal segment of the GPi terminate in three subdivisions of the ventrolateral thalamus, including nucleus ventralis lateralis pars oralis (VLo), parts of the nucleus ventralis anterior principalis (VAp), and nucleus ventralis lateralis pars medialis (VLM) (DeVito & Anderson, 1982; Kim et al., 1976; Kuo & Carpenter, 1973; Nauta & Mehler, 1966). Nigrothamic projections originate from the pars reticulata segment of the substantia nigra (SNpr) and terminate in two subdivisions of the ventrolateral thalamus: nucleus ventralis anterior magnocellularis (VAmc) and VLM. Carpenter, Carleton, Keller, & Conte, 1981; Carpenter et al., 1976; Carpenter & Peter, 1972; Carpenter & McMasters, 1964; Carpenter & Strominger, 1967). Additional projections from the substantia nigra also terminate in paralaminar medial dorsal thalamic nuclei (MD). According to Schell and Strick (1984), pallidal output is focused on subdivisions of the ventrolateral thalamus that project to the SMA (i.e., VLo and VLM) and part of the nigral output also is focused on thalamic regions that project to the SMA (i.e., VLM and MD). Anatomical evidence in monkeys indicates that the somatotopic organization of the pallidonnigral system is maintained in its thalamic projections. For example, the “face” representation in SNpr projects to a “face” representation in VLM which in turn projects to the “face” representations in the SMA. Interestingly, neither pallidal nor nigral efferents appear to terminate in thalamic regions that project to the APA or the motor cortex. Of the cortical areas concerned with movement, the pallidonnigral system is most directly connected to the SMA. The pallidonnigral–thalamo–SMA–MI pathway is considered by some (Evarts, 1985; Schell & Strick, 1984) to possibly mediate the transcortical reflex (discussed later) which is thought to be active during manipulation and speech.

The organization of the pallidum may offer some clues regarding variable effects of therapeutic lesions delivered to the posteroverentral segment of the GPi. It was previously believed that the output of the basal ganglia was directed to a single region of the thalamus which in turn projected to a single area of cerebral cortex (Kemp & Fowell, 1970). This notion has been replaced by the contemporary view that the striatum and pallidum are organized into several discrete channels, each of which is somatotopically organized and projects to a separate cortical motor area.
(Alexander, DeLong, & Strick, 1986; Middleton & Strick, 1996, 1997; Strick, Hoover, & Mushiake, 1993). Using retrograde transneuronal transport of herpes simplex virus type 1 (HSV-1), Hoover and Strick (1993) found that spatially separate zones of GPi in cebus monkey project as discrete channels to the primary motor cortex, supplementary motor area, and the ventral premotor area by way of the thalamic subnucleus ventralis lateralis pars oralis (VLo). Furthermore, these discrete channels that link the caudate, putamen, GPi, and GPi to cortical motor areas may be somatotopically organized (DeLong, Crutcher, & Georgopoulos, 1985; Strick, Dum & Picard, 1995) although there is some evidence to indicate a redistribution of somatosensory representations of body parts in primate putamen (Flaherty & Graybiel, 1991). The discrete organization of GPi outputs may provide some clues on the pathophysiology of Parkinson's disease, especially where it concerns the relative distribution of tremor, rigidity, and akinesia. According to Hoover and Strick (1993), the cardinal symptoms of PD may reflect variable involvement of individual GPi output channels. Given the organizational features of circuits connecting the basal ganglia with cortical motor areas, it appears likely that subtle differences in the location of the therapeutic lesion associated with posteroventral pallidotomy could produce a wide range of changes in motor status among limb and/or vocal tract systems depending upon which GPi output channels are actually interrupted or modified.

**Cerebello-Thalamocortical Relations**

Cerebellothalamic projections originate from all deep cerebellar nuclei, including the dentate, interpositus, and fastigial (Asanuma et al., 1983b; Asanuma, Thach, & Jones, 1983c; Brooks & Thach, 1981). Recent application of the herpes simplex virus type I has enhanced our understanding of the cerebello-thalamocortical pathways. There are at least three major cerebellothalamic systems that ultimately influence separate cortical areas, including APA (PMC), MI, and prefrontal cortex (PFC) (Middleton & Strick, 1997). Each cortical field receives input from a distinct region of the dentate nucleus. One system, located in rostral portions of the deep nuclei, projects largely to VPLo, which in turn influences the motor cortex (Sasaki, Jinna, Gemb, Hashimoto, & Mizuno, 1979; Sasaki, Kawaguchi, Oka, Sakai, & Mizuno, 1976; Schell & Strick, 1984). A second system, located in caudal portions of the deep nuclei, projects somatotopically to medial portions of ventrolateral thalamus in area X, and on to the PMC (Asanuma et al., 1983c; Brooks & Thach, 1981). Therefore, “face” efferents from caudal regions of the deep nuclei projecting to area X of the thalamus have access to neurons in the face representation of the motor cortex.

**Synthesis on Subcortico-Thalamocortical Systems**

The cerebellum, thalamus, and certain subcortical nuclei form parallel pathways to motor and premotor cortical areas that are hypothesized to contribute to the programming of skilled movement and the sequencing of motor tasks. These are summarized in Figures 2-12 and 2-13. Outputs from the cerebellum and basal ganglia are thought to make up at least six "channels" of parallel systems of subcortical efferents which project to localized areas of the thalamus. These in turn project, in a segregated fashion, to motor and premotor areas of the cerebrum (Hoover & Strick, 1998; Middleton & Strick, 1997; Schell & Strick, 1984). One parallel pathway originates in the caudal portions of the deep cerebellar nuclei and most directly influences the PMC. A second pathway originates in SNpr and GPi with direct access to SMA. The third pathway originates in rostral portions of the deep cerebellar nuclei and most directly influences the primary motor cortex. The obvious question is whether individual output channels send the same information to the cortical areas they innervate? From their physiological recordings, Middleton and Strick (1996) suggest that individual output channels are con-
Concerned with distinct aspects of motor and/or cognitive behavior. For example, the output channels in dentate and globus pallidus that project to MI may be concerned with specific movement-control parameters. Other output channels such as those projecting to premotor cortex are likely concerned with the guidance of sequential movements based on external cues. Output channels from the cerebellum that influence the prefrontal cortex may be involved in more cognitive tasks, such as working memory. Clearly, these findings lead to the prediction that even minor variations in the extent and location of lesions within the sensorimotor regions of the globus pallidus and deep cerebellar nuclei can produce impairments in motor behavior or cognitive function (Barlow, Iacono, Paseman, Biswas, & D'Antonio, 1998; Middleton & Strick, 1996).

Additional evidence in support of the subcortical "output channels" is available from lesion studies. For example, given the anatomic relation between the basal ganglia and the SMA, it logically follows that some of the movement disorders associated with basal ganglia dysfunction might be mediated by the SMA. Akinesia, associated with basal ganglia disorders (Denny-Brown, 1980), also results from lesions involving the SMA in humans (Damasio & Van Hoesen, 1980; LaPlane, Talairach, Meining, Bancaud, & Ongogolo, 1977).

Lesions involving the PMC can result in a complex disorder of learned skilled movements or apraxia (Geschwind, 1965; Heilman, 1979). The anatomic relation between the cerebellum and the PMC suggests the possibility of cerebellar involvement in the motor func-

**Figure 2-12.** Sensorimotor output channels in globus pallidus internal (GPI). The topographic trends in the localization of output channels related to the cortical motor areas are indicated at the top and bottom of the diagram. (Redrawn with permission from J. E. Hoover and P. L. Strick, 1993, "Multiple Output Channels in the Basal Ganglia," Science, 259, 819-821. Copyright 1993 American Association for the Advancement of Science.)
Cortical Area

M1 → PMv → PFC

Thalamic Nu.

VPLo → X → MD/VLc

Cerebellum

DN → DN → DN

Dorsal ← Rostral

Ventral → Caudal

Figure 2-13. Output channels in the dentate. Regions of the motor cortex (MI), premotor cortex (PMv), and prefrontal cortex (PFC) are each the target, via the thalamus, of projections that are distinct regions of the dentate. The topographic trends in the localization of output channels related to these cortical areas are indicated at the top and bottom of the diagram. (Redrawn with permission from Middleton & Strick, 1997.)

tions of the frontal lobe. Future studies might search for "apraxic-like" symptoms following lesions which involve caudal cerebellar efferent systems.

Pathways Transmitting Central Programs to MI

In addition to association areas of the cerebral cortex, the cerebellum and basal ganglia are potential sources of central programs reaching MI (Arbib, 1981). The planning and management role of the motor program is relatively unimpaired in the performance of Holmes' cerebellar patients, despite the gross impairment of the feedforward component of the pointing control system(s).

The lateral cerebellum is important in events occurring just before and after movement onset. Long-term goals are probably subserved by secondary motor areas or by the basal ganglia. As reviewed earlier in this chapter, cerebellar projections to the thalamus are directly relayed to MI via the thalamic nucleus VPLo, while regions of the thalamus receiving inputs from the globus pallidus project in large part to the premotor cortex (supplementary motor area and lateral area 6), areas which in turn project to MI via cortico-cortical connections. Therefore, the initiation of movement involves prefrontal, temporal, and parietal association cortex projections via striatum and globus pallidus to VL-VA and hence to premotor cortex (Brooks, 1979). This sequence implies that discharges of the premotor cortex would precede (for centrally programmed movement) activity in MI or deep cerebellar nuclei. Therefore, during the initial phases of a centrally pro-
grammed movement, it is possible for cortico-
comotoneurons in MI to be influenced by
cells in the premotor area (via corticocortical
inputs) and from the cerebellum (via the cere-
bello-thalamo-cortical pathway). It is also
possible for activity to occur in the dentate
nucleus of the cerebellum prior to discharge
of neurons in MI (via projections from pre-
motor cortex-pontine nuclei-dentate) as ob-
served by Thach (1975) in the case of ballistic
arm movements in monkey. This particular
example illustrates the importance of prior in-
struction in selecting the appropriate neural
pathways for reflex and intended movements.

A study examining the effects of cerebel-
lar cooling on MI responses to limb displace-
ments has provided further evidence for sep-
ate pathways underlying the reflex and
intended components of MI discharge (Vilis,
Hore, Meyer-Lohmann, & Brooks, 1976). MI
neuron responses to limb displacements con-
sist of an early reflex phase of discharge (20–
50 ms) followed by a second phase of re-
sponse (50–100 ms). This second phase cor-
responds to what has been referred to above
as the intended or centrally programmed
component of MI output. Cerebellar nuclear
cooling did not affect the early component of
MI feedback-dependent response. Cerebellar
nuclear cooling did, however, decrease the
second phase of MI activity, thereby implicat-
ing a role for the cerebellum in the central
programming of movement.

Response Properties of MI

Activity of Corticomotoneurons in MI  The
motor cortex is an important structure, serv-
ing as an important relay station for other
areas of the cerebral cortex involved in the
initiation, planning, and programming of
speech movements (Eccles, 1977). The py-
ramidal cells of the motor cortex are strategi-
cally situated in the speech movement control
system. In addition to functioning as a sum-
mring point for a variety of prefrontal and
somatosensory inputs, the pyramidal cells of the
motor cortex also provide a direct output
channel from the brain to the lower motoneu-
rons that are mapped to muscles of the vocal
tract, including the tongue, lips, jaw, pharynx,
larynx, and chest wall. Therefore, special con-
sideration is given in the following para-
graphs to the neurophysiology of the motor
cortex in relation to fine motor control.

Most of what is known about the electro-
physiology of the primary motor cortex is the
result of work conducted on limb motor sys-
tems. Where possible, reference will be made
to vocal tract systems. The majority of neu-
rons recorded in the hand area of primate MI
manifest changes in discharge frequency cor-
related with wrist load and the pattern of
muscle contraction, as opposed to changes in
wrist position (Tvarts, 1968). MI firing pat-
terns more strongly correlated with force, in-
cluding the rate of force change (Smith,
Hepp-Reymond, & Wyss, 1975), than with
other movement parameters (Humphrey,
Schmidt, & Thompson, 1970). Averaging the
electromyographic activity following the dis-
charge of MI corticomotoneurons that covary
with flexion-extension movements of the
wrist allows for the classification of cortico-
motoneuron (CM) cells into one of four basic
types, phasic-tonic (59%), tonic (28%), phasic-
ramp (8%), and ramp (5%) (Fetz, Cheney, &
German, 1976). Overall, most PTNs recorded
from MI manifest a stronger relation to the
rate of force change than to static force.

Another prominent feature of MI PTNs
is an increased activity level when the force
changes about the wrist are limited to small
steps between 0.1 and 1.0 N. It has been hy-
pothesized that force encoding by MI cells
involves the recruitment of additional cortical
cells as the demands for greater absolute
force levels are incorporated into the motor
program (Hepp-Reymond, Wyss, & Anner,
1978). The evidence in favor of this force en-
coding scheme is based on the fact that (1)
the linear covariation of the firing rate with
the force is limited to a certain force operat-
range, (2) the time constants for changes in
discharge frequency with force show a large
interneuronal variability, and (3) a few neu-
rons coding force are active only above a cer-
tain threshold force level (Hepp-Reymond et
al., 1978).
The behavior of CM cells projecting to jaw muscles is consistent with observations reported for the hand and wrist. During a controlled biting task in monkeys, involving steady forces ranging from 1 N to 70 N, there is a clear relation between the rate of maintained discharge and the force output of jaw-closing muscles for 25% of the cells tested (Hoffman & Luschei, 1980). Investigation of MI neuron activity during chewing and operantly conditioned biting revealed that most CM cells for the jaw failed to exhibit modulation with chewing. This observation is very important because it points to a greater role for MI in controlling operantly conditioned or learned movements as opposed to automatic movements using the same muscles (Ervasti, 1981a, 1981b; Phillips & Porter, 1977).

Recent studies by Georgopoulos and colleagues (Caminiti, Johnson, & Urbano, 1990; Caminiti, Johnson, Galli, Ferraina, & Burnod, 1991; Georgopoulos, Kalaska, Caminiti, & Massey, 1982; Georgopoulos, Kettner, & Schwartz, 1988; Kalaska, Cohen, Hyde, & Prud'homme, 1989; Schwartz, Kettner, & Georgopoulos, 1988) have discovered that MI neurons are capable of directional encoding. They found that the activity of some MI neurons was maximal for preferred directions, but broadly tuned around this axis. This characteristic of spatial tuning means that a large number of MI neurons are active for any given direction of movement. Concerning the issue of tuning, Georgopoulos and his colleagues (Georgopoulos, DeLong, & Crutcher, 1983) proposed that MI neurons are processed as a composite to produce a population vector which can then be used to accurately predict movement direction in two-dimensional as well as three-dimensional space. The presence of directionally sensitive neurons is not unique to MI, but appears to be a common feature of cortical and subcortical regions that are anatomically connected to MI, including premotor cortex (Caminiti, Johnson, Galli, Ferraina, & Burnod, 1991; Kurata, 1993; Weinrich & Wise, 1982), deep cerebellar nuclei (Fortier, Kalaska, & Smith, 1989), postcentral and parietal cortex (Kalaska, 1988; Kalaska, Caminiti, & Georgopoulos, 1983), and possibly basal ganglia (Georgopoulos et al., 1983).

**Somatosensory Input to MI** There appears to be more than one route by which somatosensory information can influence MI activity. Neurons in MI receive somatosensory input from muscle spindle afferents (Albe-Fessard & Liebeskind, 1966; Mallis, Pribram, & Kruger, 1953), and cutaneous mechanoreceptors (Welt, Aschoff, Kameda, & Brooks, 1967). The functional significance of these afferent inputs that influence MI from thalamus versus primary somatosensory cortex (SI) is considered in the following paragraphs.

Neurons in thalamic nucleus ventralis posterior lateralis pars oralis (VPLo) receive short latency (6–10 ms) inputs from the arm and transmit these signals to the arm area of MI (Horne & Tracey, 1979; Lemon & van der Burg, 1979). Electrical stimulation and HRP injections confirms these connections. On the other hand, SI receives somatosensory inputs via the ventrobasal complex, which receives projections from the medial lemniscus. This latter pathway is important because neural activity in MI is dependent on dorsal column function, and lesions of the dorsal column and/or postcentral gyrus significantly diminish MI responses to somatosensory stimulation (Friedman & Jones, 1981).

These findings are supported by the fact that massive fiber projections link areas of the postcentral cortex (SI, SII, area 5) to MI. It should be noted that inputs to SI are not directly transmitted to MI; rather, a hierarchical organization within SI suggests that somatosensory information is sequentially processed prior to transmission to MI. Inputs to areas 3a, 3b, and 1, are preprocessed in areas 1, 2, and 5 for additional signal processing before transmission to area 4 of the primary motor cortex. At this point it becomes possible forlemniscal inputs mediated by SI to interact with nonlemniscal inputs mediated by the VPL nuclei of the thalamus.

The notion that large, high velocity movements and small, low velocity move-
ments may involve different control mechanisms is supported by the results of studies on motor cortex activity (Evarts & Fromm, 1978; Fromm & Evarts, 1977). Precentral neuron responses to kinesthetic stimuli are enhanced during accurate positioning and controlled fine movement, but depressed just before and during ballistic movement. Thus, there are marked changes in motor cortex responses to feedback depending upon the movement strategy at the time feedback was delivered.

As reviewed earlier in this chapter, afferent feedback is especially important in modulating motor cortex output during precise control. During such fine movements, motor cortex neurons are influenced by closed-loop negative feedback, as evidenced by their intense modulation to sensory feedback from the hand (Evarts, 1982). However, these motor cortex neurons may, depending upon the context and goals of the situation, shift from being controlled by the negative feedback pathway to being controlled by central programs that are themselves triggered by afferent stimuli.

For movements of the articulatory apparatus, it is apparent that the initial set or preparatory state of the subject is of critical significance in determining the short latency responses occurring as a result of afferent inputs during movement. Presumably, each particular form of motor behavior has an associated central state that selects the afferent inputs to be employed in controlling movement. For articulation, an analogous process is thought to occur, with the difference that a very great number of subunits of the motor program can be called up by any of a very large number of different sorts of afferent stimuli.

Summary of MI Response Properties

In summary, there are a number of different features of movement to which MI output might be related, including force level, rates of force change, position, and velocity (Evarts, 1981a, 1981b). Actions of the primary motor cortex appear to be under the influence of premotor and somatosensory areas which apparently modify MI output according to set, goal, and intention. Some of the most notable response properties of motor cortex include:

- The discharge frequencies of MI neurons change with different levels of force. The proportion of force-related neurons is high in certain classes of MI neurons (those sending axons to contact lower motoneurons) and low in other classes (those sending axons to the red nucleus).
- MI activity is related to the magnitude of muscular contraction underlying this force. Due to the length-tension properties of muscle, more muscle discharge is required to exert a given force against an external object when the muscle is short than when it is long.
- Unlike spinal cord and brainstem motoneurons, which are silent during skeletal muscle relaxation, most MI neurons are spontaneously active at rest. As the set or intention of the subject changes, there may be corresponding changes in the level of this spontaneous discharge.
- MI neurons related to force exhibit marked changes in discharge frequencies for small changes of force near the zero-force level. This has been seen for fingers, jaw, and arm. This points to the special role of MI output for controlling the early recruited portion of the motoneuron pool in fine motor control.
- MI neurons, through a composite analysis of population vectors, accurately encode the direction of movement.
- MI neurons are responsive to a variety of somatosensory inputs originating from muscle spindle afferents, joint receptors, and cutaneous mechanoreceptors.
- Lemniscal somatosensory information is preprocessed by well-defined areas in the postcentral gyrus (areas 3, 1, 2, 5) before transmission to MI.
- Non-lemniscal inputs to MI are mediated by the thalamic nucleus ventralis posterior lateralis.
**Transcortical Reflexes**

Corticomotoneurons (CM) are located in motor areas of the cerebral cortex and project to lower motor neurons in the brainstem and spinal cord. CM cells can be activated and modulated by peripheral afferent input emanating from deep receptors (Strick & Preston, 1978; Tanji & Wise, 1981), cutaneous afferents, and postcentral gyrus outputs (Evarts, 1985). This form of MI activation is known as a transcortical reflex. It should be noted that the transcortical reflex was originally referenced to an output from motor cortex CM cells rather than to any particular component of a muscle response (Phillips, 1969). For the transcortical reflex, the phylogenetically recent corticomotoneuronal projection represents the efferent limb.

In primates, CM cells have two phases of activity following a limb displacement. The first (20 ms latency) phase is analogous to a segmental stretch reflex that is dependent on the direction of limb displacement, while the second phase (40 ms latency) depends on motor set. These two phases of motor cortex output have different properties and different functional roles. The first phase follows the rules of a closed-loop negative feedback control system and is especially important in the maintenance of postural stability, whereas the second phase operates according to open-loop principles and is considered important in goal-directed movements that occur from a variety of starting positions using multiple articulators such as the system for speech production (Evarts, 1985). Therefore, the properties of motor cortex CM responses are quite different, depending on the extent to which one or the other of these two control modes is dominant. For a more complete discussion of the dynamic processes involved in closed-loop and open-loop modes in MI, refer to Wiesendanger (1981) and Wiesendanger and Miles (1982). As to the pathways mediating the transcortical reflex, the recent papers by Asanuma et al. (1983a, 1983b, 1983c) consider the current state of knowledge and summarize evidence showing that there are several routes (corticocortical and thalamocortical) over which sensory inputs may activate MI in the closed loop mode.

**NEURAL CONTROL OF LARYNGEAL-VOCALIZATION SYSTEMS**

**Respiratory Control System**

The respiratory system provides the air source for sound production during speech and participates in a variety of other behaviors including quiet and forced breathing and expulsive reflexes. The demands of these varied motor tasks are altered by such factors as postural orientation and gravitational load (Hixon, Mead, & Goldman, 1987). In order to deal with these varying demands, the neural control of the respiratory system is complex, with interactions between phylogenetically newer mechanisms (voluntary motor control) and phylogenetically older pathways (e.g., central pattern generators for automatic breathing). Unfortunately, only a preliminary understanding of the neurophysiology of speech breathing is available. A brief discussion of the mechanical constraints and neural control mechanisms underlying these behaviors is presented in the following sections. The interested reader is referred to Hixon (1987, especially Chapters 1 and 4) and von Euler (1982) for a more detailed treatment.

**Mechanical Considerations and Musculature**

Both active and passive forces play a role in the process of breathing. The passive forces include the elasticity of the lungs, rib cage, and abdominal wall, and the surface tension of the thin liquid coating of the alveoli. For each degree of lung inflation or deflation, these passive forces of the respiratory mechanism combine to produce a unique alveolar pressure. The functional relation between the two is known as the “relaxation curve.” In the absence of muscular effort or a blocked air-
way, air flows between the lungs and the atmosphere until the lungs contain a "functional residual capacity" of air for which alveolar pressure just balances atmospheric pressure.

Several thoracic and abdominal muscles actively change lung volume, thereby altering alveolar pressure and/or respiratory air flow. The diaphragm moves the lower wall of the thoracic cavity downward, leading to lung expansion. The intercostal (and other thoracic) muscles alter lung volume by changing the configuration of the rib cage. In addition, various abdominal muscles, acting indirectly via the abdominal contents, raise and lower the bottom wall of the thoracic cavity. Usually, subsets of these different muscle groups are activated differentially during quiet breathing and speech.

**Quiet Metabolic Breathing**

The main function of metabolic breathing is the efficient exchange of oxygen and carbon dioxide to maintain optimal levels of blood gases and extracellular pH. Normally, this is accomplished by a stereotyped breathing pattern occurring periodically twelve or more times per minute. Starting at the resting expiratory capacity, the lungs are inflated by about one-sixth the available inflation volume. Exhalation then returns the lungs to their original volume (Hixon, Goldman, & Mead, 1973). The most important muscular contributor to this process is the diaphragm (von Euler, 1982). Its activity increases progressively during inspiration, then rapidly decreases at the end of inspiration, although some residual activity is maintained during a portion of the expiratory cycle to counteract the elastic recoil of the lungs and the rib cage. Of lesser importance to quiet breathing are the external and parasternal intercostals (von Euler, 1982), while the remaining thoracic and abdominal muscles have minimal involvement in most circumstances, but can be recruited when deeper or more forceful breathing is required (Hixon et al., 1973).

**Speech Breathing**

During speech, the goal of maintaining blood gas and pH balance is relaxed, such that major departures from the homeostatic set points of these variables can be observed (von Euler, 1982). Considerably higher lung inflation volumes are typically used. In addition, air pressures and flows for speech production fluctuate considerably, both with slowly varying linguistic features such as general loudness and prosody and more rapidly varying demands such as phonemic articulation and syllabic stress. Even the relatively simple task of producing a steady state vowel requires expiratory air pressures and flows which depart at almost all lung volumes from those produced by passive relaxation. Thus, carefully graded muscular effort must be continuously delivered to produce the required air stream. The addition of rapidly varying varying features such as phonemic and prosodic structure clearly places additional demands on respiratory muscle control.

As a result, muscle activity during speech varies markedly from that during quiet metabolic breathing (see Hixon, 1987, for detailed treatment), even to the extent of relying on a different set of muscles. The diaphragm generally ceases EMG activity at the onset of expiration, and remains inactive during the remainder of the utterance. By contrast, both inspiratory and expiratory intercostals show much increased involvement. This increased functional role during phonation may rely heavily on the much richer muscle spindle innervation of the intercostal muscles relative to the diaphragm. The abdominal muscles can also be significantly involved in speech production, depending on the speaker's posture relative to gravitational forces.

**Brain Systems for Respiratory Control**

Respiratory control appears to involve at least two separable neural systems. The first is a central pattern generator which is responsible for producing optimal metabolic breathing.
The second provides control during vocalization and speech, to some extent by overriding metabolic breathing systems. Thus, inferences about mechanisms of speech motor control from study of the metabolic control system are quite limited (von Euler, 1982). Unfortunately, data are sparse concerning the actual workings of the speech breathing control system.

**Respiratory Motoneurons**

Motoneurons (and associated spinal interneurons) which control the respiratory musculature are found distributed across most cervical and thoracic segments of the spinal cord, projecting to their muscles through various peripheral nerves. These neurons form the final common pathway for both metabolic and speech respiratory control. In addition, these cells also take part in various expulsive, postural, and segmental reflexes.

**Metabolic Breathing System**

The central pattern generator for quiet, metabolic breathing is shown in Figure 2–14 (Cohen, 1981; Kalia, 1981; von Euler 1977, 1980, 1982). Two important pools of neurons (I-alpha and I-beta) are found in the nucleus tractus solitarius (NTS). Both cell groups show an increasing firing rate during inspiration, feed-back from the respiratory center (Pco₂) since (Pco₂) from the pulmonary venous blood.

![Figure 2–14. Respiratory central pattern generator. Major functional components and connections thought responsible for generating quiet metabolic breathing. Excitatory connections are indicated by solid lines and pluses; inhibitory connections by dotted lines and minuses. Anatomical entities presumed responsible for these functions are indicated in parentheses. Also depicted (dashed lines) are sources and targets of descending influences that modulate pattern generating activity. PAG: periaqueductal grey; PBM: nucleus parabrachialis medialis; Bot. C.: Botzinger complex; NTS: nucleus of the solitary tract; Iα & Ib: cell types within NTS. (Based on Von Euler, 1980.)](image-url)
tion, but respond differentially to sensory feedback. Although these neurons receive lung and airway sensory feedback via the vagus nerve (Kalina, 1981), their cyclic inspiratory activity does not require these inputs, since they also integrate chemical information (Pco₂, Po₂, and pH). Descending influences from cortical and bulbo-ponsine centers also appear to be present.

A second, less studied group of respiratory-related neurons includes the Botzinger Complex, nucleus para ambiguous (NPA), and nucleus retroambigualis (NRA). The function of these neurons is not well understood. The Botzinger Complex has been demonstrated to fire during expiration, and has been speculatively implicated in the role of switching off the inspiratory activity of NTS neurons.

Finally, the nucleus para brachialis medialis (NPBrm) has been identified as the "pneumotactic center." It contains three spatially segregated types of respiratory neurons through which neural activity appears to cycle: (1) inspiratory units, (2) units that fire across the inspiratory-expiratory transition, and (3) expiratory units (Hugelin, 1977). A lesion of this nucleus results in abnormally deep and prolonged inspirations.

**Neural Control of Respiration During Vocalization**

The basic pattern generator described above is probably involved in respiratory control during vocalization, although there are few data that directly address the issue. In addition, vocalization respiratory control is thought to involve numerous vocalization related brain structures, including anterior cingulate cortex, periaqueuductal grey, cerebellum, and direct and indirect motor cortical projections to the spinal motoneurons. Direct motor cortical control of the respiratory motoneurons appears to achieve greatest significance in human speech control. As discussed below, the effects on vocal behavior of stimulating and lesioning these regions, together with their vocalization-related neural firing, are beginning to be understood. However, the role during speech of these different regions in controlling the respiratory apparatus, per se, remains largely a matter of speculation.

**Laryngeal System**

The larynx occupies a unique position in the vocal tract. Its activity must be coordinated with the respiratory system to produce a frequency modulated sound source during vocalization. At the same time, its activity must coordinate with the rapidly acting articulatory structures of the upper airway during phonemic articulation (Barlow, Netsell, & Hunker, 1986). It can thus be considered a microcosm of the entire speech production system. This, together with the relatively homologous structure and function of the larynx across mammalian species, may make it a particularly interesting system to study in trying to understand the neural mechanisms of vocal control.

**Effects of Different Laryngeal Muscles**

The various laryngeal muscles serve a variety of functions, both in terms of their effects on glottal closure and in the rapidity with which they can make their adjustments to the glottis. The influence of the more important of these muscles is summarized below.

**Pure Abductor/Adductors: PCA and IA**

Posterior cricoarytenoid (PCA) muscle activity appears to best correlate with vocal fold abduction. These muscles contract during respiration (especially the inspiratory phase), and have minimal activity during phonation (Dickson & Maue-Dickson, 1982). By contrast, the interarytenoid (IA) muscles are active mainly during glottal closure, and thus serve the antagonistic function to the PCAs. IA EMG activity generally remains relatively constant during phonation and does not vary in relation to changes in fundamental frequency (F₀).

**Mixed Function: TA, LCA, and CT**

In the case of the thyroarytenoid (TA), the lateral cri-
coarytenoid (LCA), and the cricothyroid (CT) muscles, EMG activity is related to both vocal fold adduction and increases in fundamental frequency (Baer, 1981; Hast & Globus, 1971; Hirano, Vennard, & Ohala, 1970; Larson & Kempster, 1983). The vocalis muscle, defined as the medial portion of the TA (Perlman, Titze, & Cooper, 1984), is an intrinsic laryngeal muscle involved in the control of F0 intensity, and register in phonation (Atkinson, 1978; Baer, Gay, & Niimi, 1976; Hirano, Ohala, & Vennard, 1969; Shipp & McGlone, 1971). Twitch contraction times for the vocalis muscle are very fast, ranging from 10 to 32 ms in the dog and cat (Alipour-Haghghi, Titze, & Durham, 1987; Hast, 1967; Hirose, Ushijima, Kobayashi, & Sawashima, 1969; Martensson & Skoglund, 1964). The cricothyroid, also characterized as a fast twitch muscle (Hast, 1966), correlates most consistently with fundamental frequency changes (Sapir, McLean, & Luschei, 1984; Shipp, 1982). TA activity correlations with F0 tend to vary considerably between subjects, while LCA appears to be more strongly correlated with adduction than F0 control (Shipp, 1982). A composite summary of intrinsic laryngeal muscle activity as a function of task is shown in Figure 2–15.

Other F0 Control: Extrinsic Muscles and P0. Certain extrinsic laryngeal muscles also may play a role in determining voice fundamental frequency. These muscles can raise and lower the larynx from its resting position, and thus indirectly produce F0 changes (Shipp, 1982). In addition, changes in subglottal air pressure (P0), partially under control of the lower respiratory musculature, can produce small F0 alterations (Hixon, Mead, & Klatt, 1971; Shipp, 1982).

Peripheral Afferent Innervation and Reflexes

Several different types of receptors provide afferent information from the larynx. Included are rapidly and slowly adapting corpuscular receptors located in the subglottic mucosa, corpuscular receptors located within the laryngeal joint capsules, and a limited number of muscle spindles and many spiral nerve endings wrapped around muscle fibers within the laryngeal muscles. Sensory information from these receptors is conveyed mainly through the internal branch of the superior laryngeal nerve, then the vagus nerve, to NTS.

It has been suggested that this sensory information plays an important role in reflexive modulation of vocal fold adduction during speech (Wyke, 1983). However, subglottal air pressure changes do not produce reflexive laryngeal EMG activity during electrically stimulated vocalizations in anesthetized cats (Garrett & Luschei, 1987), even though such reflexes can be produced by mechanical stimulation (Garrett & Luschei, 1987; Kirchner & Suzuki, 1968). In addition, neither unilateral nor bilateral transection of the superior laryngeal nerve internal branch produces much change in the acoustic structure of electrically elicited monkey calls (Jurgens & Kirzinger, 1985). Thus, the relevance of these reflexes during speech and other vocal behavior is open to question.

Extrinsic reflexes also act on the laryngeal musculature. Inputs from a variety of pulmonary receptors evoke reflex contractions thought to serve mainly non-vocalization protective functions (Wyke, 1983). However, involvement of these reflexes in vocal control cannot be ruled out, as information from the lungs can provide powerful control of the expiratory drive and laryngeal adduction during both normal expiration (Green & Neil, 1985) and electrically elicited vocalization (Garrett & Luschei, 1987). In addition, short latency inputs from auditory centers may provide the means for acoustically based adjustments of the vocal tract during phonation (Barlow, 1980; Burnett, Freedland, Larson, & Hain, 1998; Cole & Abbs, 1983; McLean, 1977; Wyke, 1983).

CNS Nuclei, Pathways, and Physiology

Laryngeal Motoneurons The various extrinsic and intrinsic laryngeal muscles are differentially innervated. The motoneurons
controlling the laryngeal, as well as pharyngeal and esophageal musculature, are located in a long, ill-defined brainstem cell column which includes the nucleus ambiguus (NA) and the retrofacial nucleus (RFN) (Davis & Nai, 1984; Lawn, 1966a, 1966b; Miyazaki, 1982; Schweizer, Ruebsamen, & Ruehle, 1981; Yoshida, Miitumasa, Hirano, Morimoto, & Kanaseki, 1987; Yoshida, Miyazaki, Hirano, & Kanaseki, 1983; Yoshida, Miyazaki, Hirano, Shin, Totoki, & Kanaseki, 1982). A summary of this organization is presented in Figure 2-16 (Yoshida et al., 1983). Esophageal and pharyngeal constrictor muscles are represented in the rostral half of the cell column, while motor neurons controlling the laryngeal muscles (except cricothyroid) are found in the caudal half of the column (Yoshida et al., 1983). The cricothyroid muscle (CT) is represented by ventrally located cells in the caudal portions of the rostral half of NA. There is also partial segregation of neural input to different muscles in the dorsoventral and mediolateral dimensions, as well.

**Physiological Studies** Although much remains to be determined, activity in NA and its associated cell column has been studied during both breathing and conditioned vocalizations in animals. During quiet breathing, four types of NA neurons have been reported. These included motoneurons (as identified by antidromic stimulation of the laryngeal nerves) active during inspiration and expiration, inspiratory interneurons which send axons into the spinal cord, and interneurons without any obvious respiratory function (Delgado-Garcia, Lopez-Varneo, Serra, & Gonzalez-Baron, 1983). Zeal and Larson (1987) identified three types of laryngeal motor neurons in the TA region of NA. The most common type was recruited only during swallowing and conditioned vocalization; the other two types were involved in expiratory...
Figure 2-16. Functional organization of laryngeal motoneurons: A: Line drawing of the location of HRP labeled cells in the caudal brainstem and the cervical cord of cats following HRP injection of the nodose ganglion (a thickening of the vagus nerve near the brainstem in which all vagal fibers innervating the vocal tract are present). SO: superior olivary nucleus; FN: facial nucleus; RFN: retrofacial nucleus; AM: nucleus ambiguus; Hyp: hypoglossal nucleus; DMV: dorsal motor vagus nucleus; RAM: retroambigualis nucleus; AN: accessory nucleus. (Redrawn and adapted from Miyazaki, 1982.)

B: Schematic diagram of the level of labeled cell columns for the nodose ganglion, the recurrent laryngeal nerve, and the muscles in the rostrocaudal direction. The level in the brainstem is indicated with the shape of the hypoglossal nucleus (Hyp); obex, OB: the superior olivary nucleus (SO), and inferior olivary nucleus (IO). (Redrawn from Yoshida, Miyazaki, Hirano, & Kanaseki, 1983.)

C: Outline of somatotopic arrangement of the labeled motoneurons for each muscle in transverse plane of the nucleus ambiguus at three levels (I, II, III) which are indicated in Figure 2-7B. NG: nodose ganglion; RLN: recurrent laryngeal nerve; STP: stylapharyngeal muscle; CeP: cephalopharyngeal or superior constrictor muscle; P: hypopharyngeal or middle constrictor muscle; TP: thyropharyngeal or inferior constrictor muscle; CP: cricopharyngeal muscle; CE: cervical esophagus muscle; ME: middle part of thoracic esophagus muscle; LE: lower part of thoracic esophagus muscle; LVP: levator veli palatini muscle; CT: cricothyroid muscle; PCA: posterior cricoarytenoid muscle; TA: thyroarytenoid muscle; LCA: lateral cricoarytenoid muscle; IA: interarytenoid muscle. (Redrawn from Yoshida, Miyazaki, Hirano, & Kanaseki, 1983.)

and inspiratory respiration, respectively. In the associated nucleus retroambiguus, unit activity was typically related to respiration, but could also be related to vocalization and orofacial muscle activity (DeRosier, West, & Larson, 1988). Additional study is needed of how these and other laryngeal motor neurons act to control vocal output.
**Brainstem Nuclei** Limited data are available concerning projections to laryngeal motoneurons from higher brain centers. Yoshida et al. (1985) and Kobler (1983) have studied this issue using HRP transport from NA/RFN in the cat and bat, respectively. Although numerous brainstem regions were labeled in these studies, including contralateral NA cell columns, three regions had the most consistent inputs to NA/RFN. These were the NTS, which is a primary target for sensory feedback from the larynx, the nucleus parabrachialis (NPB), which is involved in the central pattern generator for respiration and may perform second order sensory processing on somatosensory information from the larynx, and a small area of the peri-aqueudal grey (PAG) near the inferior colliculus. As discussed below, the NPB and PAG have been implicated in vocalization control by physiologic techniques. However, both NTS and NPB have identified roles in metabolic respiration; their involvement in vocalization control per se needs to be confirmed physiologically.

Also of interest is the finding that certain brain regions, include parts of the lateral pontine and medullary reticular formation, project to multiple motor nuclei responsible for effectors at different levels of the vocal tract (Thoms & Jurgens, 1987). Such regions may provide a mechanism for coordinating the function of different vocal tract systems during vocalization.

**Physiological Recording Studies** The activity in these brain regions, especially during vocal behavior, has only begun to be studied. Probably best characterized are neurons in a small portion of PAG, which do not have activity correlated with respiration, but which fire during the interval immediately preceding vocalization onset. This neural activity is typically unrelated to vocalization intensity. However, for at least a few units, there appears to be preferential firing preceding certain types of vocalizations (Larson, 1985; Larson & Kistler, 1984; see also Sandrew & Poletti, 1984).

The parabrachial nucleus in mammals shares close anatomical relations with other vocalization controlling brainstem structures, including the cingulate cortex, PAG, NTS, and nucleus retroambiguus, and possibly the nucleus ambiguus which is known to innervate laryngeal muscles. The NPB has also been identified as the pneumotaxic center, manifesting strong modulation of neural activity during respiration in the anesthetized cat. Most NPB units tend to be active during periods between conditioned vocalizations (Farley, Barlow, & Netsell, 1992; Farley, Barlow, Netsell, & Chmelka, 1992). Some NPB units are active during vocalization, while others are suppressed. These findings suggest that the parabrachial region’s involvement in vocal control is very complex with probable convergence of respiratory, acoustic, and sensorimotor features of vocalization.

**Stimulation and Lesion Studies** Most studied of the brainstem vocalization-related nuclei is the PAG. Stimulation of small loci in or near PAG can elicit bilateral, relatively normal sounding vocalizations in both anesthetized monkeys (Larson, 1985; Magoun, Atlas, Ingersoll, & Ranson, 1937) and cats (Garrett & Luschei, 1985; Larson, Wilson, & Luschei, 1983; Magoun et al., 1937), together with vocal tract EMG activity at all levels of the airway (Larson, 1985). Such stimulation can also alter ongoing calls in awake, vocalizing monkeys (Larson, 1985). Stimulus intensity appears to control loudness of the elicited calls (Larson, 1985); by contrast, stimulus frequency is only weakly correlated with vocal fundamental frequency (McGline, Richmond, & Bosma, 1966). In monkeys, more dorsal anterior stimulating sites produce clear calls such as “coos,” while stimulating more posteriorly produces rougher calls (i.e., “barks”) (DeRosier, Ortega, Park, & Larson, 1987). Recent reports that monkey calls can be produced by microinjections of excitatory amino acids into the active region of PAG suggest that these effects cannot be attributed to stimulation of fibers of passage (Carrié, Dampney, & Bandler, 1987; Jurgens & Richter, 1986; Richter & Jurgens, 1986).

PAG destruction is reported to produce mutism in both humans (Botez & Barbeau,
1971) and cats (Adametz & O’Leary, 1959). In monkeys, both unilateral and bilateral lesions of NTS seriously disrupt the acoustic structure of electrically elicited vocalizations, while destruction of NPB has no discernible effect (Jurgens & Kirzinger, 1985).

**Neocortical Areas—Sensorimotor Cortex**

Human primary somatosensory and motor cortices are generally thought to play a major role in laryngeal control during speech, and their damage to result in major loss of phonatory control (see Barlow et al., 1986, for review). There is some indication that lesions of different lateral precentral regions produce different deficits (Abbs & Welt, 1985). However, for non-humans, sensorimotor cortex apparently plays a much smaller role in vocalization control. There is a relative lack of direct motor cortex projections to NA in all but anthropoid apes and humans (Kuypers, 1958a, 1958b, 1958c, 1964), although direct projections appear to be present to nuclei projecting to NA, including NTS, PAG, and NPB (Jurgens & Pratt, 1979; Kuypers, 1958b, 1958c). Lesions of either somatosensory or motor cortical regions also appear to have little effect on non-human vocalizations (Jurgens & Kirzinger, 1985). Likewise, motor cortex stimulation can produce vocal output in anthropoid apes (Dusser de Barenne, Garol, & McCulloch, 1941) and humans (Penfield & Rasmussen, 1950), but typically does not do so in other species, although laryngeal EMG activity may be affected (Clark & Luschei, 1973; Craggs, Rushton, & Clayton, 1976; Hoffman & Luschei, 1980; Larson et al., 1983; McGuinness et al., 1980).

Nonetheless, slow potential shifts, usually thought to be related to motor system preparation, can be recorded from animal motor cortex immediately preceding vocalization onset (Szirtes, Marton, & Urban, 1977). In addition, motor cortical stimulation can affect laryngeal activity during vocal behavior elicited by PAG stimulation (Larson et al., 1983). Sensorimotor cortex thus may play a non-obligatory, supporting role in non-human laryngeal control.

**Other Neo-Cortical Areas**

Several other cortical areas are thought to participate in control of vocal production in humans, although their role in laryngeal control per se has not been extensively studied. Stimulation of supplementary motor areas in humans can produce vocal output (Penfield & Welch, 1951). In monkeys, lesions of homologs of Broca’s, Wernicke’s, and other association areas implicated in human speech apparently have little effect (Sutton, Larson, & Lindeman, 1974).

**Limbic Areas—Anterior Cingulate Gyrus**

In contrast to sensorimotor areas, anterior portions of the cingulate gyrus appear to play a major role in animal vocalization control, especially in monkeys (Jurgens, 1983). These areas are highly connected with other cortical and subcortical regions thought to be involved in emotion and/or vocal control (Jurgens, 1983). Normal-appearing vocalizations can be elicited by stimulating anterior cingulate regions (Jurgens & Ploog, 1970; Jurgens & Pratt, 1979; Robinson, 1967a, 1967b; Smith, 1945), although excitatory amino acid injections appear to be without effect (Jurgens & Richter, 1986; Richter & Jurgens, 1986). These stimulation effects appear to be relatively independent of reinforcement potential (Jurgens, 1976), suggesting that the vocalizations produced are not merely sequelae to altered emotional state. While lesions of anterior cingulate do not normally disrupt the ability to vocalize spontaneously (Franzen & Myers, 1973; Kirzinger & Jurgens, 1982; Trachy, Sutton, & Lindeman, 1981), they are reported to interfere with performance of conditioned vocalization tasks in monkeys (Aitken, 1981; Sutton et al., 1974; Trachy et al., 1981). Bilateral anterior cingulate lesions in humans often produce a loss of volitional control of emotional intonation (Jurgens & von Cramon, 1982), and sometimes lead to major or complete deficits in speech articulation ability (Botez & Barbeau, 1971).

**Other Limbic Regions**

Stimulation of many other limbic areas, including the amygdala,
the lateral, dorsomedial, and periventricular hypothalamic areas, and the mediodorsal thalamus can produce vocalizations in monkeys. However, in many of these regions, the calls are probably a secondary consequence of emotional, motivational, or pain/pleasure states evoked by the stimuli (Jurgens, 1976; Ploog, 1981). These regions may thus influence vocal control indirectly via connections with structures such as anterior cingulate cortex and periaqueductal grey.

**Cerebellum** The cerebellum appears to play a role in vocal tract coordination in both humans and animals. In humans, lesion of certain cerebellar regions lead to “ataxic dysarthria,” a syndrome characterized by excessive fluctuations in fundamental frequency and intensity, along with reduced lip and jaw movement velocities during speech production (Barlow et al., 1986; Hirose, Kiritani, Ushijima, & Sawashima, 1978; Kent & Netsell, 1975; Kent, Netsell, & Abbs, 1979). Electrical stimulation of the recurrent laryngeal nerve produces evoked potentials in the paramedian lobes and ansiform lobes adjacent to the vermis (Lam & Ogura, 1952; Larson, Sutton, & Lindeman, 1978). In animals, lesions of the cerebellum and/or deep cerebellar nuclei do not alter the frequency of vocalization, but can affect cell structure, including the intensity, fundamental frequency, and duration of the vocalization, as well as more complex interactions between these features (Larson et al., 1978). These observations are generally consistent with a cerebellar role in coordination, but not initiation, of vocal behavior.

**Basal Ganglia** Basal ganglia damage results in a variety of speech defects involving the laryngeal system, including breathlessness, roughness, tremor, hoarseness, deficits in F0 control, and losses in the ability to rapidly open and close the vocal folds (Barlow et al., 1986). These effects are thought to occur indirectly through alterations of motor cortex function. Though currently lacking, studies of basal ganglia effects on non-human vocalizations could be interesting, since they might provide information on motor cortex involvement in animal vocal control.

**Thalamus** As indicated in earlier sections, thalamic centers, especially VL and VPM, form integral parts of the motor control systems involving basal ganglia, cerebellum, and motor cortex. Thus, these nuclei undoubtedly participate in human speech motor control. However, their involvement in non-humans can again be questioned, since VPM thalamus appears to be noncritical for electrically elicited monkey calls (Jurgens & Kirzinger, 1985).

### Summary: Multiple Control Systems and Homology

Figures 2–17a and 2–17b include schematics of the major systems thought to be involved in the neural control of the larynx and chest wall. The primary motoneurons represent a final common pathway for descending influences, and in addition participate in relatively direct reflexes involving these vocal tract structures. Auditory and somatosensory feedback are also indicated, although in less detail than descending control systems.

Three major descending systems common to all mammalian species are distinguished on the basis of their relatively separable functional roles. The first, the central pattern generator for quiet, metabolic breathing, can function alone, but is strongly influenced by higher control centers. The second, “emotional vocalization” system, is composed mainly of limbic and brainstem regions and is considered responsible for initiation or production of nearly all animal vocalizations. A third, “general motor” system, includes structures which probably subserve more volitional or operant motor behaviors. While this last system has been studied extensively in connection with limb and orofacial control, its role in laryngeal and respiratory system control remains sketchy.

Two systems relatively unique to humans are also depicted. The first of these is an elab-
oration of association cortical areas concerned with language function. The second, subserv- ing fine speech motor control, is the direct projection from motor cortex to the primary motoneurons.

Obviously, many aspects of these functional systems, including basic data about connectivity and physiological activity, remain to be understood. At present, some of these matters can only be addressed by invasive neurobiological studies which can be performed only in animal models. Unfortunately, questions can be raised concerning the applicability of animal data to human speech production mechanisms. Animal vocalizations appear to be controlled mainly by more prim-
ITIVE, emotive systems such as anterior cingulate cortex and periaqueductal grey. In contrast, direct motor cortical control is thought to dominate human speech production, while more primitive systems have lesser involvement or may even be "gated out" (Myers, 1976; Netsell, 1982).

Nonetheless, data from animal models may have substantial impact on practical issues involving brain trauma. A role for the emotional vocalization system cannot be ruled out during normal speech production. In addition, an abnormal involvement of more primitive systems in speech production as a consequence of head injury may be the source of some observed speech deficits. Finally, the emotional vocalization system may play an especially prominent role in the development of speech and language. If this role were understood, it might form a basis for remedial training following head trauma.
NEURAL CONTROL OF SUPRALARYNGEAL SYSTEMS

Upper and Lower Lip Control Systems

The lips are composed of approximately 10 muscles originating on the bony surfaces of the maxilla and mandible. Nearly all muscles make insertion into the integument surrounding the mouth, and therefore, are remarkably different from most other skeletal muscles (Blair, 1986; Kennedy & Abbs, 1979). There is essentially no fascia within the labial muscles and no tendons. The fibers of the muscle oribicularis oris (OO) form the major bulk of the upper (UL) and lower (LL) lips. It has been described as a sphincter-like muscle with the fibers generally coursing from one corner of the mouth to the other and whose action contributes to compression and rounding of the lips (Gray, 1977; Kennedy & Abbs, 1979; Muller, Abbs, Kennedy, & Larson, 1977; Muller, Milekovic, & MacLeod, 1984). Assisting the OO for lower lip closure is the mentalis (MENT) muscle, which is optimally situated to provide a high degree of mechanical advantage in achieving forceful lower lip elevation (Barlow & Rath, 1985). The complex of lower perioral muscles does not appear to be controlled homogeneously. Instead, there may be independent activation of individual interdigitated muscles (e.g., oribicularis oris inferior and depressor labii inferior), or fractionated activation of fibrils within a muscle (Blair, 1988).

The differences between the upper and lower lips also extend to their fine motor control. The surfaces of the lips are displaced at movement velocities in the range of 5 to 20 cm per second during speech. The relative contribution of each lip during speech production can be significantly different. For example, during the production of [p], [b], and [m], the lower lip covers a trajectory that is approximately twice that of the upper lip (Barlow, 1984; Hughes & Abbs, 1976; Kuehn & Moll, 1976; Netsell, Kent, & Abbs, 1980; Sussman, MacNeilage, & Hanson, 1973). To maintain temporal synchronization with the upper lip, peak velocity for the lower lip is approximately twice that of the upper lip (Barlow, 1984; Netsell et al., 1980). The lower lip dental contrast for [f] production is obvious evidence that the UL and LL can be activated separately.

Peripheral Neural Elements

The muscles and connective tissues of the human vocal tract are richly endowed with a variety of specialized sensory elements, including muscle spindles, Golgi tendon organs, joint receptors, and mechanoreceptors. These sensors selectively transduce or convert mechanical energy (resulting from passive and active movement and force) into a neural signal that is encoded by the central nervous system. An in-depth review of the anatomic and physiologic response properties of these receptor types is beyond the scope of this chapter. See the following references for information concerning proprioceptors (Houk & Rymer, 1981; Matthews, 1981), and mechanoreceptors (Barlow & Farley, 1989; Chambers, Andres, von During, & Iggo, 1972; Darian-Smith, 1966; Halata & Munger, 1983; Janig, 1970; Johansson, 1976; Johansson & Vallbo, 1976; Mountcastle, 1980a, 1980b; Munger, 1971; Munger & Halata, 1983).

The organization of somatic sensory receptors is equally unique for this musculature. Morphologic equivalents of muscle spindles and Golgi tendon organs, which mediate proprioceptive information in limb muscles, have not been found in facial muscles (Folkins & Larson, 1978; Kadanoff, 1956; Lovell, Sutton, & Lindeman, 1977; Stal, 1994). However, examination of the unique linkage between muscle and skin provides an alternative anatomy for proprioception in the face. The attachments of the perioral muscles are made by small fascicles that can be widely distributed (Blair & Smith, 1986). Because of this unique relation between skin and muscle, nerve endings and mechanoreceptors become logical candidates to mediate proprioceptive information. Several tissue-mechanoreceptor...
configurations have been identified in human skin that appear capable of providing proprioceptive information (displacement, velocity, force, rate of force change, acceleration) (Johansson, 1976; Johansson & Olsson, 1976). Reflex sensitivity to small mechanical inputs applied to the perioral region indicates a high degree of sensorimotor coupling.

**Mechanosensitivity of the Perioral System**

Structures common to speech and mastication include the mouth, tongue, anterior teeth, lips, and jaw. Since these motor behaviors use the same body parts, they probably use information from the same sensory receptors, although the importance of the various types of sensory afferents and the way in which this information is used may be quite different (Lund et al., 1982). The work of Lund and his colleagues provides a basis for speculating about the patterns of sensory inputs generated during speech. First, the vast majority of mucosal and cutaneous afferents seem to be silent in the absence of movement. According to Lund, most skin and intraoral afferents are not excited by small local distortions of the skin but only by direct contact with their receptive fields. This observation is consistent with the relatively high reflex sensitivity of perioral muscles to mechanical stimulation of the labial mucosa (Barlow, 1988a, 1988b). Conceivably, no signals would be sent from the surface of the closing lower lip or protruding tongue until they touch another surface. In contrast, hair afferents could provide a continuous velocity signal during speech.

Recently, psychophysics has been applied to the study of somatic sensory processing of mechanical inputs to human perioral skin (Barlow, 1987; Essick, 1991; Essick, Affricca, Aldershoft, Nestor, Kelly, & Whitseel, 1988; Essick, Dolan, Turvey, Kelly, & Whitseel, 1990; Essick, Franzen, & Whitseel, 1988; Essick, McGuire, Joseph, & Franzen, 1992; Essick & Whitseel, 1985; Essick, Whitseel, Dolan, & Kelly, 1989; Fucci, 1972; Mountcastle, LaMotte, & Carli, 1972; Verrillo, 1966a, 1966b, 1966c). Inspection of mechanical frequency detection thresholds of human perioral skin reveals that the distribution and typing of mechanoreceptors present in the lips and surrounding skin is remarkably different from the afferents that innervate the glabrous surfaces of the hand (Barlow, 1987). As shown in Figure 2–18, threshold functions for facial skin sites to sinusoidal mechanical stimulation lack the characteristic “dip” at 250 Hz that is correlated with the pacinian corpuscular afferent. The significance of this difference in mechanosensitivity between hand and perioral structures may relate to their respective kinematic roles in motor control.

An elaboration of mechanosensory specialization in the face was revealed by Essick and colleagues (Essick et al., 1988) through the use of more complex mechanical stimuli. Subjects were instructed to psychophysically scale the velocity of an external probe moving across the skin at a relatively constant force. Velocity-tuning was most apparent in facial skin as opposed to the skin of the arm or hand (Essick, Starr, Dolan, & Affricca, 1987). The optimal velocity range in the face is 4 to 8 cm/sec. These psychophysical estimates of velocity detection may bear direct relation to velocity characteristics of perioral structures during speech since the majority of lip and jaw movements occur within this velocity range. Future studies might attempt to relate the velocity of tissue shear in this region of the face during speech to the psychophysical estimates of velocity-tuning to an external probe. Based upon these insightful studies, it seems likely that spatiotemporal integration associated with cutaneous and subcutaneous motion perception may be an important signal source encoded by the brain for the maintenance and timing of vocal tract movements for speech.

**Real-Time Modulation of Speech-Orofacial Motion Sense**

The functional activities of the oral system, including speech, eating, sucking, emotional
expression, all require a high degree of coordination among the many muscles involved. The oral system is not only highly coordinated but also extraordinarily flexible and adaptive. The same apparatus used for speech, eating, and smiling can switch rapidly from one activity to another, even do two at the same time. Within any activity, whether it is speech or sucking, the coordination and adaptation is remarkably rapid, generally on the order of tens of milliseconds. These adaptations are reflected as co-articulatory effects and are consistent with a neuromotor system that plans at a goal level with lower level effectors that monitor and adjust the synergy to meet the goals.

The processes by which the nervous system monitors the effects of active muscle contraction on tissue configuration and externally imposed displacement is known as motion sense, including kinesthesia and proprioception. Kinesthesia refers to the sense perception of movement, and secondarily to muscle sense (Greek, Kinesis, motion + aisthesis, sensation). The second part of this definition could be expanded to include proprioception (Latin, Proprius, one’s own, + capio, to take); however, deep muscle sense is usually associated with the activity of muscle spindles and Golgi tendon organs (GTOs) common to limb antigravity muscles.

**Role of Afferents in Guiding Movements**

To be able to carry out such adaptive coordination, the nervous system is presumed to monitor the consequences of motor output in order to adjust the patterns to maintain the goal. For example, in the control of limb movements and posture, vision often plays an important role in targeting and guiding the reaching limb, or in visual stabilization of posture. Sensory information derived from pro-
proceptors (muscle spindles, GTOs), joint receptors, and mechanoreceptors located within skin and muscle provide a wealth of information about motion sense and position of muscle systems during movement. A number of cutaneous and muscle receptors exhibit their greatest sensitivity to small amplitude input signals (Knibestol, 1975; Knibestol & Vallbo, 1970; Goodwin, Hulliger, & Matthews, 1975; Matthews & Stein, 1969; Poppele & Bowman, 1970). Furthermore, the gain of some reflexes is greatest in response to small displacements (Cooker, Larson, & Luschei, 1980; Hoffer & Andreassen, 1981; Marsden, Merton, Morton, Adam, & Hallett, 1978).

**Neural Encoding of Mechanosensory Events in the Orofacial System**

For orofacial movements, vision is not as dominant a factor, at least for ongoing motor control. Therefore, the nervous system must rely on auditory, proprioceptive, kinesthetic, and cutaneous information. The orofacial complex is densely innervated with a wide variety of cutaneous, musculocutaneous, muscle, and joint afferents. Indeed, direct human microneurographic recordings from orofacial afferents have confirmed that the sensory flow conducted along trigeminal pathways is abundant and closely related to the movements, forces, and tissue contacts associated with speech, chewing, lip closure, and experimentally delivered perturbations to skin surfaces in and around the oral opening (Johansson, Trulsson, Olsson, & Abbs, 1988; Johansson, Trulsson, Olsson, & Westberg, 1988; Nordin & Hagbarth, 1989; Nordin & Thomander, 1989).

Nordin and Hagbarth (1989) described the response characteristics of 84 low threshold mechanoreceptive afferents innervating facial hairy skin or glabrous lip sampled with microelectrodes from the human infraorbital nerve. Units were classified as slowly adapting (SA) or rapidly adapting (RA), with small or large receptive fields. Of particular interest in this report was the discovery of units whose activity patterns were influenced by tangen-

tial skin stretch and voluntary contraction of facial muscles. Innervation density was highest near the corner of the mouth and on the upper lip. SA units with large receptive fields were spontaneously active and presumed to be Ruffini endings. As suggested by Nordin and Hagbarth (1989) the dual function as extero- and proprioceptors demonstrated for cutaneous mechanoreceptors in the face raises the question as to why no tactile sensations or skin paraesthesia are aroused by the afferent barrage caused by facial movements. Some of these issues are considered in more detail by Chapman (1994). In addition to different patterns of receptor activation, at least two central mechanisms should be considered including afferent or surround inhibition (Darian-Smith, 1965), and corollary discharge in which the motor command preceding contraction of facial muscles may influence the interpretation of afferent inflow (McCloskey, 1981).

Recent experiments conducted in our laboratory confirm the reported depression of tactile sensations during low-level force generation (Andreatta & Barlow, 1998). This is an interesting observation when compared to findings on the modulation of reflex activity in which excitability of the segmental, trigemino-facial pathway increases significantly as reflected in R1 amplitude over the same range of active forces (~ 0–0.5 N). So, how is it that segmental loop activity increases while perception of vibrotactile inputs is diminished? Moreover, Nordin and Thomander (1989) reported that a sinusoidal vibratory stimulus delivered to the face was highly effective in driving populations of motor units. The ease with which entrainment occurs combined with the reflex modulation experiments suggests that the segmental pathway is facilitated, whereas sensory flow mediated by the trigemino-thalamo-cortical pathway is gated from conscious perception. Apparently, the transmission of tactile inputs is diminished or gated during active force, an hypothesis supported in a review by Chapman (1994).

In a related study, Nordin and Thomander (1989) completed infraspinacular microelectrode recordings from the human infra-
orbital nerve close to the infraorbital foramen. Multunit activity from low threshold mechanoreceptive afferents was recorded during tactile stimulation, vibration, and facial movements. Sixty-six (66) receptive fields corresponding to different fascicles were mapped with tactile stimuli on facial hairy skin and the glabrous surface of the upper lip. The smallest receptive fields were located on the upper lip (<2 cm²) with nerve conduction velocities of approximately 30 ms. Skin indentation evoked dynamic ON- and OFF- responses and a less pronounced static discharge. Stretching of the skin was accomplished by tugging on the free end of tape stuck to the skin outside the receptive field. Consistent with Nordin and Hagbarth (1989), dynamic changes in facial muscle contraction and lateral skin stretching evoked ON- and OFF- responses. Vibratory mechanical stimuli included an 8 mm probe driven at 10–50 Hz at 4 mm amplitude. Afferent double-peaked responses to an oscillating probe applied to the perioral region induced synchronous EMG activity of several motor units during sustained lip protrusion. Vibratory inputs from 14–50 Hz were highly effective in entraining primary trigeminal afferents and OOS EMG bursts with a conduction delay of approximately 11–14 ms. The latency of the entrained perioral motor units is consistent with perioral R1 segmental characteristics (Barlow & Bradford, 1996).

The dynamic sensitivity to minor variations in contraction and stretching was very high during normal facial movements associated with speech. These observations are consistent with infraorbital recordings made in cats using a servo controlled linear motor to deliver a tangential mechanical shear stimulus to the angle of the rima oris (Barlow, Farley, & Spangler, 1989).

Dynamic stretch sensitivity to both the onset and release of tangential stretch was common to all multunit recordings. This high sensitivity to lateral stretch would cause the size of the fascicular field to be overestimated. The high stretch sensitivity of mechanoreceptors in the face is not surprising given the course and termination of muscle fibers into the integument, basement membrane of skin, and connective tissues surrounding muscle. Active contraction of facial muscles, even very small gestures, was an effective stimulus of mechanoreceptors proximal to the active muscles. The high sensitivity of mechanoreceptive afferents in the face to movement reinforces the observation made by Bosma (1970) almost 30 years ago in which he stated "the mouth's sensory experiences are generated principally by its own actions, and its actions are responsive to its sensory experiences" (p. 550).

It may be concluded that several types of mechanoreceptors both in hairy and glabrous skin and in the red zone of the lips can contribute to facial kinesthesia and motor control by signaling small variations in stretching and contraction. A graphical representation of receptive fields identified on the human perioral skin for single cutaneous afferents is shown in Figure 2–19. The rapidly conducting mechanoreceptive afferents (type Aβ, 6–12 μ diameter, 35–75 ms) present in perioral tissues are capable of encoding position, velocity, acceleration, load dynamics, as well as directional changes in tissue conformation and spatial contact areas (Barlow, 1987, 1991, 1998; Barlow & Farley, 1989; Nordin & Thomander, 1989; Rath & Essick, 1990). In fact, Nordin and Thomander (1989) have suggested that the presence of a specialized slowly adapting mechanoreceptor, termed "pseudo-Ruffini," provides the capacity for proprioception in the lips. The evidence from these experiments suggests that the orofacial sensorium is well suited to encode the consequences of movement to be used by the CNS in establishing dynamically assembled neuronal groups or maps of orofacial space, driven by multimodal and temporal correspondence between perception and action.

The notion that mechanosensory units in the skin can encode parameters of strain for movement perception is also supported in work by Edin and Johansson (1995) on finger movements. They discovered that strain-sensitive mechanoreceptors in the skin proximal to the joints of the fingers provide afferent information that can be used by the CNS for movement perception and execution. In
Receptive fields of single cutaneous afferents 
reported in the human face


In certain situations, these afferent signals have precedence over signals generated in mechano receptive muscle afferents with respect to movement perception and fine motor control. Skin strain, occurring around the lips during movement, is an effective stimulus for cutaneous mechanoreceptor activity (Connor, 1997; Connor & Abbs, 1998). Using an artificial neural network, Connor and Abbs (1998) found that multiple segments of skin strain are predictive of lip movement and strongly suggest that perioral cutaneous mechanoreceptors are fully capable of encoding proprioceptive information during speech and other skilled movements (see Figure 2–20).

In a recent study, intrafascicular multi-unit and single unit activity was sampled from mechanoreceptive afferents from the human lingual nerve with perimucosally inserted tungsten microelectrodes (Trulsson & Essick, 1997). Using nylon filaments and blunt glass probes to deliver mechanical stimulation to
sory neurons in the thalamus and cerebral cortex. McLean, Dostrovsky, Lee, and Tasker (1990) encountered single units in the VPM of the human thalamus during stereotaxic surgery that were responsive to conformational changes in perioral tissues during speech movements and mechanical stimulation of the face. Rate and amplitude encoding of servovcontrolled mechanical inputs to the lip of the cat has been recorded in sensorimotor cortex (Barlow & Spangler, 1985). Obviously, these findings complicate the mechanisms by which mechanosensory inputs are gated from perception since the fidelity of speech movement-related afference appears to be well preserved by neurons in the VPM thalamus.

**Sensorimotor Regulation of Speech**

Coordination and adaptation among orofacial muscle systems is remarkably rapid, generally on the order of tens of milliseconds. Introduction of external loads or rapid perturbations to the sensorium is a powerful experimental method for indexing changes in the excitability of segmental pathways and inferring the dynamic moment-to-moment control properties of the neuromotor system. This technique has been applied to limb (Evarts, 1981) and orofacial systems (Abbs & Gracco, 1984; Andreatta, Barlow, Biswas, & Finan, 1996; Andreatta, Barlow, & Finan, 1994; Barlow & Bradford, 1996; Barlow, Finan, Bradford, & Andreatta, 1993; Gracco & Abbs, 1985, 1988; Kelso, Tuller, V-Bateson, & Fowler, 1984). External loads can take the form of relatively long duration inputs (~100–300 ms) which alter the trajectory of articulatory movements during speech or very brief (~4 ms) punctate mechanical inputs with short rise/fall times. The former have proven useful in characterizing patterns of load compensation while paradigms using the punctate mechanical stimuli have increased our understanding of segmental mechanisms, including descending control of trigemino-facial pathways during active force tasks.

The work of Gracco and colleagues utilized long duration mechanical loads during

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**Figure 2-20.** Correspondence between actual lower lip speech movements and output from an artificial neural network. (Reprinted with permission from Connor, 1997.)

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the dorsal surface of the mucosa of the tongue, these investigators were able to isolate and study the physiology of 33 single mechanoreceptive afferents. Of these 33 units, 22 were superficial units manifesting a strong response in the presence of cutaneous mechanical stimuli. Fourteen (14) of these superficial units were classified as rapidly adapting type I and the remaining 8 were either SA I or SA II's. Eleven (11) of the 33 mechanoreceptive afferents were deep units, all of which had SA characteristics. Seven (7) of 11 were spontaneously active. All 11 of these deep units reliably encoded tongue position without direct contact of their receptive fields. The superficial units responded vigorously when the tongue was moved to bring the receptive field into physical contact with other intramural structures.

The encoding of mechanoreceptive afferent activity in trigeminal pathways appears to be preserved by second and third order sen-
speech. Loads of approximately 250 ms in duration, with rise times of 5 or 15 ms to endpoints of approximately 0.35 or 0.55 N, were delivered during the production of a lip closing gesture. These perturbations significantly changed upper and lower lip displacement, movement time, and closing velocity (Gracco & Abbs, 1985). Due to the complexities of the orofacial anatomy and central representation, it is clear that much remains to be learned about the sensorimotor control of both speech and nonspeech activities.

Modulation of Mechanically Evoked Perioral Reflexes During Static and Dynamic Force in Humans

A closely related perioral muscle response component used to study the sensorimotor organization of trigemino-facial system during motor control involves facial reflexes (Larson, 1977; Netsell & Abbs, 1977). Investigators have typically used a brisk mechanical input to the perioral region, or electrical stimulation of trigeminal nerve branches to evoke the reflex (Ekborn, Jernelius, & Kugelberg, 1952; Gandiglio & Fra, 1967; Kugelberg, 1952; Larson, Folkins, McLean, & Muller, 1978). Of particular interest to speech neurophysiologists is the exquisite sensitivity of the perioral system to innocuous mechanical stimuli (Bratzlavsky, 1979; Lund et al., 1982). Displacing a smooth metal disk in a gliding motion over the surface of the upper lip also produces potent reflex effects in the lower lip muscles (Smith et al., 1985; Smith, McFarland, Weber, & Moore, 1987; Weber & Smith, 1987). McLean and Smith (1982) generated consistent perioral reflex responses using a 0.5 mm stretch applied to the corner of the mouth. Larson et al. (1978) used a 1.0 and 3.0 mm stretch to evoke the perioral reflex. Recent studies have been successful in generating reflex responses in perioral muscles during fine force control using low amplitude taps (100 microns) applied to the mucosal surfaces of the lip (Barlow, 1988a, 1988b; Barlow & Netsell, 1986b, 1986c; Netsell & Barlow, 1986). The functional properties of this sensorimotor process are considered in greater detail in the following sections.

The perioral reflex is classically described as a two-component excitatory EMG response consisting of an early (R1, 10–15 ms) non-adapting component, and a late (R2, 30–45 ms) component that adapts rapidly. The neural pathways mediating the early component (R1) are thought to be restricted primarily to the brainstem and involve at least two synapses (Figure 2–21) although recent experiments clearly indicate that the amplitude of R1 can be modulated by concurrent activation of the motor cortex. The R2 circuitry is not well understood; however, ablation of area 3a in monkeys has been shown to abolish this component of the perioral reflex (Larson, 1977). In the past, perioral reflexes were characterized as simple, unitary, nonspecific, and primarily responsive to innocuous inputs. Contemporary techniques using servocontrolled mechanical stimulation methods have revealed the dynamics of perioral reflex action to be rather complex, and bound by the properties of the stimulus, subject state (experience and conditioning), and task dynamics of the CNS/muscle system at the time of perturbation. This segmental reflex is also under descending control. With this realization, the mechanically evoked perioral response becomes an important and sensitive indexing probe of facial motoneuron excitability. With appropriate stimulus paradigms, it is possible to reveal both subthreshold and suprathreshold effects on facial lower motoneurons during speech and nonspeech behaviors. The use of brief (punctate, 4 ms rise/fall) perturbations (as previously used in our laboratory) or longer duration mechanical perturbations (used by Gracco & Abbs, 1985; 5 ms rise/fall–250 ms duration) actually represent a small sample of the stimulus continuum that can be used to explore the response continuum of sensorimotor action in the lower face. Of course, there is the added complexity of spatial configuration (contactor area, edges, number of receptive field inputs) of the labial sensory experience that combine with the temporal features to reveal an additional constellation of sensorimotor response dynamics that must be
considered in a model of orofacial motor control (Barlow, 1991; Barlow & Bradford, 1996). The term "reflex" as applied to the study of orofacial sensorimotor integration has the tendency to constrain our conceptualization of how mechanosensory information is encoded and used by the motor system. The graded nature of "reflex" actions following perturbation is matched only by the graded nature of inputs to lower motor neurons originating from neuronal groups in cerebral cortex which of course are continuously under modification during evolving behavior. This is one hallmark of an adaptive system. In essence, the range of stimulus-related activity observed from single/multi-unit EMG records actually reflects the modulation of dynamically assembled neuronal groups.

A number of experiments involving punctate mechanical stimuli delivered under a variety of muscle activation conditions have demonstrated that mechanosensory information can influence the firing patterns of lower motor neurons in the brainstem (Andreata, Barlow, Biswas, & Finan, 1996; Andreata et al., 1994; Barlow, 1990, 1991; Barlow, Andreata, Finan, & Biswas, 1995; Barlow & Bradford, 1996; Barlow et al., 1993).

Mechanosensory Flow During Force Statics

The spatiotemporal organization and specificity of the mechanically evoked, short latency perioral response (R1) was sampled
from a group of 9 normal adult humans (Barlow & Bradford, 1996). Perioral reflex activity was sampled during passive and active static force conditions (0.10 N) in the presence of servocontrolled mechanical inputs to lip vermilion. Results confirmed that the sensorimotor apparatus of the lower face is very responsive to low level mechanical inputs and highly dependent on several factors including input site (upper vs. lower lip), amount of glabrous tissue stimulated (contactor array size), and task dynamics (passive vs. active subject-generated lip force).

A composite record of R1 modulation for these 9 normal subjects is given in Figure 2-22 to illustrate the robust relation between contactor array size and R1 amplitude as a function of subject-generated lip force for mechanical inputs delivered to the left side of the lower lip vermilion. Each token slice, represented along the Z-axis within each surface plot, represents the average IEMG response to 64 stimuli. The test protocol delineating token slice number and associated experimental condition is given in the table above the IEMG plots. The progression of test conditions within each muscle surface plot is the same, with the foreground block representing R1 activity with lip muscles at rest (0.0 N) and contactor arrays systematically changed in configuration from 2, 6, 10, to 14 points. Two control conditions (motor on, no contact with the face) are plotted between force conditions to demonstrate the absence of stimulator induced artifact in the IEMG signals. The background block of slices for each muscle panel clearly shows potentiated R1 activity that is modulated with contactor array size during active, subject-generated lip compression hold force of 0.10 Newton. The steplike increase in baseline IEMG for all muscles is the result of the active force condition. Notice the growth of R1 with successively larger contactors with perioral muscles at rest and during the active force condition. Stimulus onset is indicated at time zero on the X-axis. Stimulation site is indicated by the star symbol. R1 activation is clearly greater ipsilateral to the stimulus, and tends to be larger at the homonymous recording site. The degree of muscle response specificity varies according to the stimulus input site.

**Sensorimotor Specificity**

McClean and Smith (1982) reported that mechanical stimulation of the skin of the upper or lower lip produces reflex responses of local sign in both inferior and superior divisions of the orbicularis oris muscle. For example, stimulation of remote structures (e.g., the chin) does not evoke the early reflex response in orbicularis oris inferior motor units. Localization of the reflex is defined as **response specificity** (Netsell & Barlow, 1986). The high degree of response specificity in these experiments is likely due to the physiologic nature of the mechanical stimulus and the nature of the motor task performed by subject. The fact that different populations of labial motor units have different reflex responses to mechanical stimulation at one site, suggests that trigemino-facial projections are muscle specific within the sensory and motor nuclei of the brainstem (Barlow & Netsell, 1986a, 1986c; McClean & Smith, 1982). This observation is consistent with the hypothesis that the neural control of small populations of perioral facial muscle motoneurons may, at times, operate in an independent mode (Blair, 1988).

Thus, several features of the orofacial sensorium are encoded by primary trigeminal mechanoreceptive afferents, including afferent gain, specificity, locus, and spatial summation. These inputs are presumed to serve higher order sensory relays in the development of a central representation and dynamic conformational map of perioral space. These sensorimotor features encoded by trigeminal afferents are presumed to play an important role in motor learning and maintenance of oromotor control during speech, suck, mastication and swallow, and gesture.

**Mechanosensory Flow During Force Dynamics**

The potential for phase-dependent sensorimotor adjustments during the production of
LOWER LIP INPUT SERIES

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<tr>
<td>2</td>
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<td>4</td>
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<td>10</td>
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Figure 2–22. The relation between contactor array size and R1 amplitude as a function of subject-generated lip force for mechanical inputs delivered to the left side of the lower lip vermilion. 0 is shown in the IEMG wireframe surface plots at four recording sites (OOS-Right & Left, OOI-Right & Left). Each token slice, represented along the Z-axis within each surface plot, represents the average IEMG response to 64 stimuli. The test protocol delineating token slice number and associated experimental condition, is given in the table above the IEMG plots. The progression of test conditions within each muscle surface plot is the same, with the foreground block representing R1 activity with lip muscles at rest (0.0 N) and contactor arrays systematically changed in configuration from 2, 6, 10, to 14 points. Two control conditions (motor on, no contact with the face) are plotted between force conditions to demonstrate the absence of stimulator-induced artifact in the IEMG signals. The background block of slices for each muscle panel clearly shows potentiated R1 activity that is modulated with contactor array size during active, subject-generated lip compression hold force of 0.10 N. The step-like increase in baseline IEMG for all muscles is the result of the active force condition. (Adapted from Barlow & Bradford, 1996.)
force ramps at different rates of recruitment using muscles surrounding the mouth was examined in a recent experiment (Andreatta et al., 1996). Servo-controlled mechanical inputs were delivered to the upper lip vermilion at prespecified force thresholds during force ramps to an endpoint of 1 N in 5 young adults. The rate of force recruitment was either 1 N/s or 4 N/s. As shown in Figure 2–23, the R1 amplitude growth function derived from EMG signals taken at the OOI and OOS recording sites was nearly monotonic for the 1 N/s recruitment condition. However, virtually identical mechanical stimuli produced a very different R1 growth function for the 4 N/s recruitment condition. R1 amplitude was significantly greater at the beginning of the force ramp when compared to the slower ramp condition. As shown in Figure 2–24, the R1 growth function associated with the 4 N/s force rate peaked at the 50% force level, and attenuated near the termination phase (80%) of the force ramp. Apparently, the dynamics of muscle activation influence how mechanoreceptive afferent signals are integrated into the stream of motor output. The experiment just described clearly demonstrates one method for indexing these moment-to-moment changes occurring at the level of the facial nucleus.

Carefully modulating the mechanically evoked perioral response in the face provides an objective tool for indexing excitability changes among neuronal groups among brain stem and cortical circuits in a human model. These pathways are undoubtedly involved in the emergence of orofacial motor skills such as sucking, swallowing, smile, and speech. This notion is consistent with observations in upper limb motor function suggesting that the transition of simple reflex processing to more elaborate forms of sensorimotor actions for voluntary reactions is fundamental to motor skill acquisition (Bawa, 1981). This is a
hallmark feature of biological systems in that behaviors of increasing complexity are derived from relatively simple precursors.

Concerning the dynamics of perioral reflex action, Smith and her colleagues (1985) suggest "this reflex pathway is not suppressed during phonation or speech. However, the response appears to be suppressed or absent because the amplitude of the observed response depends upon the activation levels of the various muscles of the lower lip and, therefore, indirectly on the nature of the gesture the subject is instructed to produce" (p. 148). This finding supports an earlier notion by McClean and Smith (1982) in which they emphasized that the question of perioral reflex amplitude (i.e., modulation) during speech and vocalization may require evaluation in terms of the specific muscle groups that are active.

Reducing the complexities of speech into subsets of constituent force and movement tasks is another strategy used by a number of investigators to study facial sensorimotor control mechanisms. Physiologic examination of sensorimotor integration during the production of motor features (e.g., force recruitment, force recruitment) common to neonates, infants, children, and adults has been valuable in specifying attributes that are preferentially encoded (selected) in the development of attractors or stable modes of oromotor control for a variety of behaviors including sucking, swallowing, and speech. For example, stimulus evoked activity in the orbicularis oris muscle is significantly related to the rate and phase of force recruitment when the target endpoint force is held constant (Andreatta et al., 1996; Barlow et al., 1995). This feature of sensorimotor control is likely to show a developmental course and be manifest in a variety of oromotor behaviors involving similar levels of force.

**Short-Term Adaptation**

Following the presentation of consecutive stimuli to the face of the cat, Lindquist and Martensson (1970) noted a substantial de-
crease in the amplitude of early reflex component between the first and second tap. In physiological systems, this property is known as short-term adaptation and is defined as the rapid exponential decay in the evoked motor response immediately following stimulation. This phenomenon should not be confused with fatigue, which is defined as the slow decline in response that occurs with continuous stimulation over a period of minutes (Harris, 1977; Kiang, Watanabe, Thomas, & Clark, 1965; Smith, 1973, 1977; Young & Sachs, 1973).

As it turns out, the human perioral reflex manifests short-term adaptation to repeated stimulation (Barlow & Netsell, 1986b). Preliminary experiments indicate that the pattern of early component short-term adaptation for the MENT recording site appears to be remarkably different from either of the OOI placements. The degree and nature of short-term adaptation for a particular muscle recording site is hypothesized to reflect an aspect of temporal resolution for somatic-motor signal processing. As yet, the significance of this phenomenon for speech motor control remains unknown.

**Neuronal Groups and Pattern-Generating Circuitry**

Neural circuits in the spinal cord and brainstem play an important role in motor coordination. Segmental reflexes provide the nervous system with a set of elementary patterns of coordination that can be activated either by sensory stimuli or by descending signals from the brainstem and cerebral cortex. Most brainstem reflexes, including the perioral reflex, have relatively complex circuits (compared to stretch reflex). Although a few reflexes have primarily local actions on single muscles, most coordinate the actions of groups of muscles. With the exception of the jaw reflex, most reflex pathways are polysynaptic, having one or more interneurons interposed between the sensory and motor neurons (Gordon, 1991). This feature allows descending signals as well as other afferent inputs to modify the expression of the reflex, including the activation of networks of interneurons for the timing of reflex components. Experiments involving sucking, force dynamics, and speech permit study of descending influences on reflex excitability. Divergent and convergent connections in reflex pathways play an important role in the spatial organization of reflex behaviors, determining which sensory inputs are enhanced or suppressed and which muscles contract or relax. Other types of connections determine the temporal organization of reflexes. For example, reverberating circuits, closed circuits of interneurons that re-excite themselves, are responsible for some reflexes that outlast the stimulus. And finally, more complex temporal patterns are produced by networks of interneurons called central pattern generators (CPG).

**Entrainment**

Another method of investigating the regulatory effects of afferent feedback involves the activation of mechanoreceptors in a manner that mimics natural movement. For example, in the walking system of the cat, hip extension and unloading of leg extensor muscles appear to control the transition from stance to swing. Cyclic mechanical stimulation of the hip joint or extensor musculature in reduced cat preparations results in synchronization of natural motor patterns with the externally applied periodic signal (Andersson & Grillner, 1983; Conway, Hultborn, & Kiehn, 1987; Kriellaars, Brownstone, Noga, & Jordan, 1994). This phenomenon is termed "entrainment" and is defined as the synchronization of an endogenous oscillator to external periodic events (Glass & Mackey, 1988; Kriellaars et al., 1994; Pavlidis, 1973). For a given stimulus with fixed amplitude and period, a stable phase relationship between the stimulus and oscillator must exist to satisfy the conditions for entrainment. In the present discussion, the external periodic event refers to a repetitive mechanical stimulus that is capable of generating synchronous activity in mechanoreceptive afferents. One potential effect of brief peripheral
stimulation is resetting of the CPG motor pattern and modulation of the cycle phase. Central neuronal networks that demonstrate entrainment or resetting effects require the integration of afferent information into the pattern generating circuitry.

Entrainment of rhythmic motor outputs has great potential for revealing moment-to-moment influences of mechanosensory inputs on speech motor control. There appear to be at least four centrally patterned behaviors involving components of the vocal tract: namely suck, mastication, respiration, and vocalization (vibrato). Speech has been considered by some to include a hybridization of these patterned outputs, modified in large part by afferent signals and task demands (Gracco, 1990; Grillner, 1981; Smith, 1992). Use of entrainment techniques is finding application in the study of vocal tract CPGs (Barlow, Finan, Andreatta, 1997; Finan & Barlow, 1996, 1998; Titze, 1996; Titze, Solomon, Luschei, & Hirano, 1994). The suck CPG has been the subject of study as a model for understanding how sensory and motor signals are integrated during what some might consider to be an “open loop” behavior. In neonatal guinea pigs, the CPG for sucking is located in the periaqueductal gray of the brainstem and is thought to be induced or regulated in part by a neocortical region known as the cortical sucking area (CSA) (Iriki, Nozaki, & Nakamura, 1988; Nozaki, Iriki, & Nakamura, 1986). In addition, the rhythmic patterning of lip, tongue, and jaw activity associated with non-nutritive suck is being considered as a possible transitional form of patterned orofacial output that is manifest in precursors to speech, such as canonical babbling. The properties of sensorimotor plasticity are under exploration in our laboratory using a specially designed instrument, known as an ACTIFIEER (see Barlow & Finan, 1998; Finan & Barlow, 1996, 1998). This instrument consists of 4 servo linear motors and a pacifier instrumented with 8 Ag/AgCl EMG electrodes. The ACTIFIEER can be programmed to deliver precise mechanical stimuli to the oral sensorium during sucking in order to characterize sensorimotor integration and is currently being applied in several neonatal intensive care units to study oromotor development in preterm infants at-risk for brain damage (Barlow, Dusick, Finan, Coltart, Biswas, & Denne, 1999).

**Perturbation Paradigms**

For more than four decades, the investigation of motor control in human subjects has included studies of reflex EMG activity elicited by muscle stretch or electrical stimulation (Lee & Tatton, 1978). Early studies were concerned with monosynaptic or polysynaptic, short-latency spinal reflex mechanisms. More recent investigations were concerned with long-loop or transcortical responses occurring at longer latencies (Hammond, 1955, 1956; Phillips, 1969). Since these pioneering studies, the perturbation paradigm continues to be an important tool in revealing sensorimotor properties of segmental and suprasegmental pathways in human and animal models (Desmedt, 1978).


In one of the initial studies (Folkins & Abbs, 1975), resistive loads were applied to the jaw during speech production. These loads halted the normal jaw elevation movement associated with coordinated labial-mandibular closure for [p]. Loads were initiated during the jaw closing movement associated with the production of bilabial stops, creating a situation in which bilabial closure would be disrupted if motor control were independent of peripheral feedback. In spite of
the mechanical load, subjects were able to achieve lip closure associated with the bilabial stop. Compensatory movements in both the upper and lower lips were associated with jaw loading. These authors concluded that online afferent feedback is capable of contributing to the compensatory motor reorganization of the lips and the jaw during speech production. Subsequent re-interpretation offered by Abbs and his colleagues suggested that these data appear to indicate the operation of open-loop, sensorimotor mechanisms inasmuch as compensation was absent in the jaw, where the loads were introduced, but was present in the lips. The interested reader is referred to the following references for a more detailed discussion of open-loop neural control mechanisms (Arbib, 1981; Houk & Rymer, 1981; Ito, 1975; Miles & Evans, 1979; Rack, 1981).

Gracco and Abbs (1985) reexamined the afferent contributions to the motor control of speech by applying unanticipated loads to the lower lip during the production of the bilabial /b/. Loads considered to be within the normal range of forces and movements resulted in a number of compensatory adjustments in upper and lower lip displacement, movement time, and closing velocity. Each of the 5 subjects was able to complete the speech gesture, in the presence of a perturbation, with little or no degradation of the speech output. Load timing variations within the target interval resulted in systematic changes in the site of the compensatory adjustments (upper versus lower lip) and in the magnitude of the kinematic responses. These kinematic changes appeared to reflect the dynamic nature of underlying control processes and the hypothesized mechanisms mediating autogenic (lower lip–perturbed structure) and nonautogenic (upper lip–nonperturbed structure) compensatory actions. Abbs and Gracco (1984) found that the degree of compensation is related to the magnitude of the perturbation displacement, especially for loads introduced prior to agonist muscle onset.

The compensatory responses reported by Abbs and Gracco (1984) are consistent with observations of parallel motor equivalence trade-off between the upper lip, lower lip, and jaw movements observed during speech (Hasegawa, McCutcheon, Wolf, & Fletcher, 1976; Hughes & Abbs, 1976), and observations that perturbation of the jaw induces compensation in the upper and lower lips (Folkins & Abbs, 1975). Comparable trade-offs also have been observed between the abdomen and rib cage in their synergistic contributions to speech pleural pressures (Hixon et al., 1973; Hixon, Mead, & Goldman, 1976; Hunker & Abbs, 1982). For speech motor control, it appears that feedback and feedforward processes may be important in the control of coordination among structural components along the length of the vocal tract.

**Jaw Control System**

The mandible is part of a bilateral mechanically linked musculoskeletal system that is involved in mastication, object manipulation, and speech (Luschel & Goldberg, 1981). The muscles that move the mandible include the opening muscles or flexors (digastric, lateral pterygoid, and suprathyroid), and the closing muscles or extensors (temporalis, masseter, and medial pterygoid). These muscles originate from several bones of the skull, including the maxilla, sphenoid, pterygoid, and palatine, and are heavily sheathed with fascia and organized into 3 to 5 compartmentalized planes about the temporomandibular joint. Jaw velocities during speech (2 to 10 cm/s) (Barlow, 1984) generally are slower than those typical of the lips and tongue (Kuehn & Moll, 1976). It has been well documented that muscle spindles, joint receptors, tendon organs, and a variety of mechanoreceptors in the periodontal ligament are present in the jaw system (cf. Bratlavsky, 1976; Goodwin, Hoffman, & Luschel, 1978; Harrison & Corbin, 1942; Karlsson, 1976; Luschel & Goldberg, 1981). The neural control system for the jaw appears to have multiple components, including a central pattern generator for mastication (discussed later in this chapter), reflex regulation, and "command inputs" that can modulate the activity of rhythmic movement.
or exert direct purposive control for speech and oral manipulation (Luschei & Goldberg, 1981). The following sections will briefly review some of the features of the mandibular neural control system.

**Reflexes Involving the Jaw**

A number of reflexes, possibly involved in mastication, can be evoked from mammalian jaw muscles using electrical or mechanical stimulation. The first reflex considered is the jaw stretch reflex. Jaw closing muscles are well endowed with muscle spindles, whereas the opening muscles have few. Afferents from the spindles of jaw closing muscles make monosynaptic excitatory connections, via the trigeminal mesencephalic nucleus, with the motoneurons of closing muscles (Harrison & Corbin, 1942; Lund, Richmond, Touloumis, Patry, & Lamarre, 1978). Primary and secondary muscle spindle afferents have been recorded in cats during chewing and lapping (Cody, Harrison & Taylor, 1975; Taylor, Appenteng, & Morimoto, 1981; Taylor & Cody, 1974) and in monkeys during chewing (Goodwin & Luschei, 1975).

Spindle primaries fire phasically and are most sensitive to the velocity of muscle lengthening during opening. Their activity results in part from the effects of fusimotor neurons, which are active before and during biting and rhythmical movements (Appenteng, Morimoto, & Taylor, 1980; Lund, Smith, Sessle, & Murakami, 1979; Taylor et al., 1981). Muscle spindle secondaries are mainly sensitive to length changes and their firing frequency generally is proportional to the distance between the incisor teeth during the performance of slow movements. The displacement information encoded by secondary endings, if operative in humans, may explain partially the ability of the speech motor control system to reprogram lip and tongue movements for vowel production under jaw blocked conditions.

The second major reflex considered is the jaw opening reflex. Mechanoreceptors in the periodontal ligament are strongly activated during chewing. Electrical or mechanical stimulation of the oral mucosa or direct electrical stimulation of nerves innervating the oral mucosa results in a brisk opening of the jaw. This response, better known as the jaw opening reflex (JOR), is effected by excitation of jaw opening muscles and inhibition of jaw closing muscles. The JOR is thought to be mediated by large diameter, rapidly conducting afferents in the lingual, inferior alveolar, and superior dental nerves with conduction velocities of approximately 40–60 meters/s.

Unlike reflex organization of limb muscles, there does not appear to be reciprocal inhibition of motoneurons of antagonistic muscles.

A central question concerning the role of reflexes in mastication is whether they function to modulate motoneuron activity within a central neural circuit, or whether reflexes themselves are the source for the basic pattern (Luschei & Goldberg, 1981). A model of mastication based purely on reflexes is not plausible because decerebrate cats do not masticate. Likewise, de-afferentation does not abolish the basic pattern of mastication. Therefore, the basic pattern of excitation to jaw closing motoneurons during mastication does not originate from muscle spindles (Cody et al., 1975; Goodwin & Luschei, 1975). Rather, the basic pattern of jaw movement during mastication appears to result from central mechanisms which may be activated by commands originating from the sensory-motor cortex, or modified by afferent signals conducted by the trigeminal system. The influence of spindle mediated afferent mechanisms appears to be greater for finely graded, low force control of the jaw (Goodwin et al., 1978; Lund et al., 1979).

This leads us into a discussion of jaw reflexes and load compensation. The responses to rapid loading and unloading of the jaw closing muscles are asymmetrical. Rapid stretch evokes a single response which corresponds in latency with the monosynaptic jaw jerk reflex (5–10 ms). The stretch response is followed 6–10 ms later by an increase in the velocity of closure. The velocity is dependent on EMG peak amplitude (Lamarre & Lund, 1975). Unloading of the jaw is followed by a
fall in EMG activity occurring at monosynaptic latency but with later phases of depression beginning at about 30 ms and 60 ms (Hannam, Matthews, & Yemm, 1968; Lund et al., 1982). If the long latency, presumably suprasegmental long-loop load compensatory mechanisms are functional in this system (Lamarre & Lund, 1975), they would appear to be of importance only in unloading (Lund et al., 1982).

**Load Compensation**

Although short latency load compensatory reflexes may play a role in voluntary biting and mastication, the need for them in speech production is not as evident because of the absence of large load variations (Lund et al., 1982). Further, muscle spindle sensitivity is apparently low during speech gestures involving closing movements of the jaw. A number of investigators have reported difficulty in obtaining consistent load responses in jaw closing muscles during speech (Folkins & Abbs, 1975, 1976; Lamarre & Lund, 1975; Lund, Smith, & Lamarre, 1978; Shaiman, 1989). Other investigators have found task-specific sensorimotor compensations to dynamic perturbations delivered to the jaw during productions of the final consonants in [baeb] where lip activity is need to achieve lip closure, but not during [baez] where the lip activity is not required to complete the production (Kelso et al., 1984). Shaiman (1989) reported that 0.45 N loads with a 20 ms rise time were effective in producing task-dependent kinematic adjustments in lip/jaw during [aæææ], but no labial compensation during [æææææ] in five of six subjects studied. Shaiman (1989) suggested that compensatory responses to jaw perturbations occur only when such responses are functionally necessary.

**Subcortical Mastication Pattern Generator**

The evidence in support of a central pattern generator for mastication is derived largely from experiments that have used random electrical stimulation of the cerebral cortex in the presence of a drug that selectively blocks intramuscular and extramuscular muscle fibers. Under the blocked condition, rhythmic activity is present in the efferent nerves to the jaw and tongue muscles of the rabbit (Dellow & Lund, 1971) and the cat (Nakamura, Kubo, Nozaki, & Takatori, 1976). These results suggest that the brainstem mechanism, located in the medial bulbariculcular formation, can generate a rhythmic pattern of activity without peripheral input. Lund and Rossignol (1980) suggest that the masticatory pattern generator regulates the reflex amplitude by phasically modulating the excitability of interneurons along the reflex pathway.

**Purposive Jaw Movements for Speech**

One neural structure that is consistently implicated in voluntary jaw control is the sensorimotor cortex. This is due, in large part, to its connectivity and short latency effects (8–40 ms in primates) on motoneurons of the jaw (Chase, Sterman, Kubota, & Clemente, 1973). Recent reports have shown a strong relation between the activity of neurons in the precentral motor cortex and controlled (voluntary) bite responses in monkeys. Approximately 50% of the cells studied in the face motor cortex of monkeys increased their rate of firing during the dynamic phase of the bite response (Hoffman, 1977). These corticomotorneurons are known as “phasic- tonic” cells. Another class of cells, termed “tonic,” are principally related to the static or sustained phase of voluntary bite. As described earlier in this chapter, the activity of corticomotorneurons can be modified by sensory input. Hoffman found that the majority of activated cells exhibited phase related changes in firing rate during imposed displacements of the jaw.

An evolving concept on the role of the sensorimotor cortex suggests that this cortical region functions primarily to coordinate the complex movements of the tongue, lips, and jaw during mastication and voluntary activity
(Lund & Lamarre, 1974). For example, the tongue must be carefully guided to avoid being crushed by the teeth during motor behaviors involving the jaw. Motoneurons controlling the tongue may facilitate the jaw opening muscles and provide inhibitory inputs to the jaw closing muscles (Luschei & Goldberg, 1981). The fact that the mandible can be used independently from the lips and tongue indicates that neural inputs to the jaw muscles are highly organized. At least a portion of the voluntary control system for the jaw as well as other orofacial structures is represented by neurons located within the face area of the precentral cortex. Sensory inputs originating from temporomandibular joint afferents may provide both velocity and displacement signals over the range of movement encountered in human speech and mastication (Lund et al., 1982).

**Tongue System**

The tongue is capable of mass reorganization through muscle contractions of intrinsic and extrinsic muscles. Movement velocities ranging from 5 to 20 cm per second are typical during speech. A rich supply of mechanoreceptors is distributed in cutaneous and subcutaneous structures of the tongue. Muscle spindles have been found in the lingual muscles of humans (Cooper, 1953; Kubota, Negishi, & Hasegi, 1975; Nakayama, 1944; Walker & Rajagopal, 1959), and in monkeys (Bowman, 1968; Fitzgerald & Sachithananadan, 1979). However, lingual spindle afferents, derived from distal portions of the hypoglossal nerve, do not appear to project directly to the cerebellum (Bowman, 1982; Bowman & Combs, 1969a, 1969b; Egel, Bowman, & Combs, 1969; Oscarsson, 1965). Rather, lingual spindle information projects to the cerebral cortex via a fast conducting pathway synapsing in the thalamic ventral posteromedial nucleus (Bowman & Combs, 1969a, 1969b). Recently, contralateral projections have been identified in the medial portion of the pontine reticulotegmental nucleus, a precrerebellar relay nuclei (Bowman, 1982). Unlike the limbs, proprioceptive and cutaneous inputs from the tongue enter the central nervous system over separate routes. Cutaneous afferents project centrally in the lingual branch of the trigeminal nerve, whereas the proprioceptive afferents after first being incorporated in the hypoglossal nerve depart from it to enter the central nervous system over upper cervical dorsal roots (Bowman, 1971).

### Some Effects of CNS Lesions on Vocal Tract Control

Damage to certain regions of the brain, resulting from disease, developmental impairments, or traumatic brain injury, can have devastating effects on speech production. This condition is known as *dysarthria* and is defined as a speech disorder resulting from damage to neural mechanisms (sensory and/or motor) that regulate speech movements (Barlow & Netsell, 1989; Netsell, 1984a). Although dysarthria may result from lesions in many different nervous system locations, dysarthria resulting from damage to the basal ganglia or the cerebral cortex represents the two main neurologic populations that have been studied by speech neuroscientists using quantitative physiologic methods. Several of these investigations are reviewed in the following sections. Since a detailed treatment of the physiologic bases of dysarthria is beyond the scope of this chapter, the interested reader is referred to the writings of Darley, Aronson, and Brown (1969, 1975), Netsell (1982, 1983, 1984a, 1984b, 1985, 1986), and others (Yorkston, Beukelman, & Bell, 1988) for a comprehensive review.

### Lesions of Motor-Sensory Cortex

Several types of motor impairments are typically associated with precentral motor cortex lesions including paralysis, hypertonia, and spasticity (Fulton, 1949; Tasker, Gentili, Hwang, & Sogabe, 1980). Perhaps more striking are the persisting deficits in fine motor control manifest primarily in phylogenetically
advanced neural systems controlling limb (Denny-Brown, 1980; Tower, 1940; Travis, 1955) and orofacial musculature (Barlow & Abbs, 1982, 1983, 1984, 1986; Neilson & O'Dwyer, 1981; Netsell, 1982). Clinical observations suggest that the least automatic and most highly differentiated movements, including manipulation and speech, suffer first and most (Jackson, 1880). In monkeys with lesions of the precentral cortex and/or pyramidal tract, movements are slow but accurate; the inability to make fast adjustments in the distal musculature controlling the hand constitutes a permanent deficit (Gilman & Marco, 1971; Gilman, Marco, & Ebel, 1971; Lawrence & Kuyper, 1968a, 1968b; Travis, 1955). Thus, the corticospinal and corticobulbar pathways represent the main sources of descending input terminating directly on those motoneurons principally innervating the distal muscles of the hand and the complex arrangement of muscles in the vocal tract.

Recent investigation of the pathophysiology of speech subsystems in patients with lesions ostensibly involving the motor cortex has focused on the fine force and position control of the most accessible orofacial structures (Barlow & Abbs, 1984, 1986; Barlow & Burton, 1988, 1990). Similar to reports in primates, impairments in motor control include a reduction in the rate of force change associated with generating fine levels of force in the upper lip, lower lip, tongue, and jaw. These measures are highly related to impairments in movement velocity in these same structures. End point accuracy, defined as the ability to generate specified target levels for both force and position, is relatively well preserved in these structures. Moreover, the measures of orofacial force and position dynamics used in these experiments were related to impairments in speech intelligibility.

Considerably more information is needed about the nature of impaired force control and resultant movement before a cause-effect relation on speech intelligibility can be accurately specified. At this juncture, it is certainly the case that quantitative physiologic measures of force and movement control are considerably more sensitive than conventional methods in determining the distribution and nature of orofacial motor impairments that degrade fine motor performance. This fact should be of considerable value in determining the extent of damage and pattern of recovery among vocal tract structures in TBI patients.

More than 800,000 serious traumatic brain injuries (TBI) occur every year in the United States alone (Gualtieri, 1988). Of the 90% of patients who survive their injuries, a large but undetermined number of victims manifest deficits in speech and swallowing. Research aimed at mapping the relations between injury sites in the brain and speech pathophysiology is in the early stages. Studies of disordered speech motor control in TBI patients are needed which combine modern brain imaging techniques, including functional magnetic resonance imaging, computerized axial tomography, and positron emission, with basic and process-oriented studies of speech physiology. Neural imaging techniques may provide insight into the functional role of speech controlling structures in both normal and disordered speech. A significant problem facing speech scientists and clinicians is the fact that the pathophysiological response of brain tissue to impact or impulsive loading is variable in distribution and degree. Logically, a number of different types of disturbances in speech motor control may result from one individual to another. Tissue strain (compressive, tensile, or shear) resulting from traumatic brain injury may be localized (e.g., acute subdural hematoma) or distributed (diffuse axonal injury), involving the brainstem and cerebral structures (Genneralli & Thibault, 1985; Thibault & Gennarelli, 1985). A number of biological models of TBI have been developed in an attempt to duplicate the dynamic loading conditions and neuropathophysiology that occur in humans (Pickard, 1980). However, these models have found limited application to clinical studies. This is due, in part, to the vast number of environmental, biomechanical, and physiologic parameters that must be considered in developing a predictive model of experimental brain injury.
Lesions of Basal Ganglia

The temporal aspects of voluntary movements in patients with Parkinson's disease (PD) are impaired with considerable individual variation. This is due, in part, to the tremor, rigidity, and dyskinesia that accompany disease of the basal ganglia. Parkinsonian dysarthrias manifesting tremor and minimal rigidity have elongated reaction times and near normal movement times for orofacial and finger control systems (Hunker & Abbs, 1985). By contrast, in a second group manifesting rigidity with minimal tremor, reaction times were normal but movement times were elongated. These results indicate that the pathologic mechanisms involved in tremor and muscle rigidity may account, in part, for the movement aberrations of dyskinesia and bradykinesia, respectively. Hence, two distinctly different pathologic mechanisms (viz., abnormal oscillatory activity and increased muscle stiffness) may hypothetically underlie increased movement time in Parkinson's disease (also see Netsell, Daniel, & Celesia, 1975).

The delays in movement initiation common to parkinsonian patients with resting tremor may be due to an inability to initiate a voluntary muscle contraction until synchronized with the excitatory EMG phase of the tremor cycle in agonist muscles of the limbs (Hallet, Shahani, & Young, 1977), and orofacial structures (Hunker and Abbs, 1984).

Another feature of movement control following damage to the basal ganglia (parkinsonism) is a reduction of both voluntary and automatic movement, that is, hypokinesia. Similar results have been found in the monkey (Hore & Vilis, 1984). The possibility that rigidity is positively related to hypokinesia in the orofacial complex was suggested by Hunker, Abbs, and Barlow (1982). However, a recent report incorporating a larger group of PD patients indicates that while rigidity may play a part in the overall movement disability, a statistical relation between rigidity and hypokinesia is difficult to demonstrate (Caliurgi, 1987).

Lee and Tatton (1978) have suggested that parkinsonian rigidity is a manifestation of increased sensitivity in a transcortical reflex loop. They found that parkinsonian patients, unlike normal subjects, could not reduce the amplitude of the later post tendon jerk EMG response (M2). This increased feedback gain over transcortical pathways was subsequently correlated with the degree of muscle rigidity in parkinsonian patients.

Differential Impairment

Lesions of the central nervous system that disrupt speech motor control are usually characterized by differential sensorimotor impairment to vocal tract structures. The neurology of dysarthria, therefore, necessitates systematic evaluation of several components of the vocal tract including respiratory, laryngeal, velopharyngeal, and orofacial structures. The following examples illustrate some general patterns of impairment that may accompany dysarthrias of central origin.

Upper and Lower Lips Clinical observations of motor function following damage to the CNS suggest that structures normally capable of finer control manifest disproportionately greater impairment in generating precise forces and displacements (Denny-Brown, 1980; Jackson, 1980). It logically follows that the lower lip, because of its dominant kinematic role during speech, will manifest proportionately greater deficits in fine motor control following CNS damage than its counterpart, the upper lip. This pattern of impairment is shown in the following examples.

Leanderson and Persson (1972) suggested that with increased background activity and disturbed reciprocal activation, the labial muscles alter their normal coordination for speech. Hunker and Abbs (1982) extended this observation and quantified stiffness (change in force/change in displacement) in the lip muscles of parkinsonian adults with dysarthria. The perioral musculature was found to be differentially impaired,
in that the lower lip manifested the most significant deficits in range of displacement and larger increases in muscle stiffness when compared to the upper lip.

Tremors in the force or movement domain have been found in a variety of vocal tract structures including the chest wall, larynx, velopharynx, and orofacial structures. Although it has been reported that tremor frequencies are uniform among orofacial structures and the index finger within the same parkinsonian subject (Hunker & Abbs, 1983), significant deviations in frequency and amplitude may exist between the upper lip, lower lip, and index finger in Parkinson's disease. For example, for a parkinsonian adult male, the amplitude of lower lip force tremor during a 2 N contraction was approximately an order of magnitude greater than the tremor associated for a 2 N contraction performed using the upper lip (Barlow & Hunker, 1988). The difference in the magnitude of the 8.06 Hz force tremor between the upper and lower lip is an example of differential involvement in degree. In this same parkinsonian patient, the spectral peak associated with the force tremor for the lips and index finger was remarkably different, suggesting a difference in the nature of the underlying neural mechanism contributing to the motor impairment. The primary frequency component of force tremor in the index finger for this particular PD patient is centered at approximately 4.88 Hz.

Differential motor involvement of the perioral musculature also was described by Barlow and colleagues (Barlow, 1984; Barlow & Abbs, 1986) in a group of patients with a congenital form of the Upper Motor Neuron Syndrome (Landau, 1974; 1980). In particular, the ability to recruit force and achieve normal movement velocity was severely compromised in these patients. Similar results have been found in lesioned monkeys (Travis, 1955). Consistently greater deficits in fine force and position control were found in the lower lip as compared to the upper lip. The pattern and degree of these fine motor impairments were highly correlated with auditory-perceptual measures of speech intelligibility in these patients.

Jaw Congenital lesions involving the motor cortex (known as cerebral palsy or congenital spasticity) can result in a variety of negative symptoms affecting jaw control, including a slowness of movement (reduced velocity) and unsteadiness during precise movements. Jaw force instability, quantified as the variability in force output over some specified period of time, is significantly increased in this patient population, especially at lower force levels (Barlow & Abbs, 1984; 1986). Similar impairments occur in monkeys following lesions to the face area of the precentral cortex (Luschei & Goodwin, 1975).

Tongue The tongue, because of its relative inaccessibility, is more difficult to study during speech production. Therefore, recent experiments have quantified tongue forces and movements under highly controlled conditions. These studies revealed several interesting patterns of deficit that are dependent upon the site of lesion. For example, in parkinsonian patients, the instability associated with generating a steady, low-level force is disproportionately greater in the tongue than in the lips or jaw (Abbs; Hunker, & Barlow, 1983; Barlow & Abbs, 1983). In patients with the Upper Motor Neuron Syndrome, the most significant deficit in tongue force control is a reduction in the rate of force change, paralleled by a decrease in movement velocity (Barlow & Abbs, 1986). These findings are consistent with the notion that the rate of force change is encoded by corticomotoneurons.

**SUMMARY AND RESEARCH DIRECTIONS**

In general, our understanding of the neural pathways involved in the planning and execution of simple voluntary limb movements is meager at best. The flow diagram shown in Figure 2-25 (modified from Allen & Tsuka-
vocal behavior. While basic tract-tracing studies are needed, more detailed pathway analyses, including identification of neuroanatomical cell types within vocal-tract controlling nuclei and the topography of connections between nuclei and/or specific cell types, would be a much stronger guide to physiological investigations. Moreover, greater attention is needed to the question of whether tracer injections are labeling vocalization specific neural pathways rather than other neurons and fibers of passage in the same area. Anatomical tract tracing from physiologically characterized sites might be of particular utility.
HANDBOOK OF CLINICAL SPEECH PHYSIOLOGY

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FOREWORD

Steven M. Barlow is uniquely qualified to write on the physiology of normal and disordered speech production. His experience includes the design and fabrication of transducers, the development of powerful but user-friendly software, the specification of measures suited to the manifold aspects of speech physiology, the determination of disordered speech motor control, and the presentation of theoretical perspectives on the neurophysiology and motor control of speech. Barlow’s expertise is everywhere evident in the wide compass of *The Handbook of Clinical Speech Physiology*. The word *Handbook* aptly denotes the authoritative coverage of topics in this book, which sets a new and exacting standard for the application of speech physiology to the clinical arena. In many important respects, this book defines the field of clinical speech physiology. Its publication is a signal event.

An impressive accomplishment of this exciting new book is that it brings both clarity and depth to the application of physiological methods to the study of speech. The *Handbook* explains how and why various physiological measures of speech supply valuable information on the motor regulation of speech production. Readers will quickly absorb the author’s expertise and should soon be ready to join in a new era of clinical speech physiology in which laboratory methods have been carefully tailored to the demands of the scientist and the clinician alike. Barlow has had a major role in shaping this new era, and it is fitting that he should write its first essential volume. The *Handbook* invites its readers to know theory, methods, and clinical application. This book is about *understanding* and *doing*. It is written to equip the reader with the overall competence that is required to work in a clinical speech physiology laboratory.

The *Handbook* opens the door to a sophistication that was formerly reserved for the very few who worked long hours in the laboratory to acquire their skills and knowledge. Barlow’s book makes essential information readily available and guides the reader through the steps of laboratory proficiency. I say welcome to this well-crafted volume and welcome too to those readers who will practice in clinical speech physiology.

Ray D. Kent