Assessing the Physiological and Behavioural Evidence for the Role of Kinesthesia in Speech Production

Évaluation des preuves physiologiques et comportementales liées au rôle de la kinesthésie dans la production de la parole

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Abstract

The current view in sensorimotor control is that "One can only control what one senses" (McCloskey & Prochazka, 1994, p. 69). This statement summarizes extensive research in limb motor control, which indicates sensorimotor integration underlies the coordination of goal-related movements. In this paper, evidence for the specific role of kinesthesia in speech production is assessed by reviewing results of physiological and behavioural studies. This evidence indicates that speech movements are encoded by oral kinesthetic receptors and this kinesthetic information appears to be transmitted to cortical sensorimotor regions. Further, behavioural studies suggest that oral kinesthesia contributes to adaptations observed in perturbation studies and also that jaw kinesthesia may have an active role in jaw opening gestures. It is suggested that for highly skilled movements such as speech production, kinesthetic information is necessary for predictive control and facilitation of automaticity, rather than continuous error feedback. Finally, evidence suggesting an association between speech disorders and oral kinesthetic deficits is presented. It is suggested that kinesthesia has a fundamental role in speech motor control and that clinical management of speech disorders may be enhanced by consideration of kinesthesia.

Abrégé

L'avis courant au sujet du contrôle sensorimoteur est que « l'on ne peut contrôler que ce que l'on peut sentir » (McCloskey et Prochazka, 1994, p. 69). Cet énoncé résume toute la recherche effectuée dans le domaine du mouvement des membres et du contrôle moteur, qui indique que l'intégration sensorimotrice gouverne la coordination des mouvements ayant un objectif précis. La présente étude évalue les preuves liées au rôle spécifique de la kinesthésie dans la production de la parole en examinant les résultats d'études sur la physiologie et le comportement. Ces preuves révèlent que les mouvements pour produire la parole sont codés par des récepteurs kinesthésiques et que cette information kinesthésique semble être transmise aux régions sensorimotrices du cortex. Par ailleurs, les études sur le comportement laissent entendre non seulement que la kinesthésie orale contribue à certaines adaptations observées dans les études de perturbation, mais aussi que la kinesthésie de la mâchoire peut avoir un rôle actif à jouer dans les gestes d'ouverture de la mâchoire. Dans le cas des mouvements hautement spécialisés, comme la production de la parole, il est suggéré que l'information kinesthésique est nécessaire pour le contrôle prévisionnel et la facilitation de l'automatisation, plutôt que la rétroaction continue des erreurs. Enfin, on présente ici des observations qui supposent une association entre les troubles de la parole et les déficits kinesthésiques oraux. Il est suggéré que la kinesthésie joue un rôle fondamental dans le contrôle moteur de la parole et que le traitement clinique des troubles de la parole peut être amélioré grâce à la kinesthésie.

Key words: kinesthesia, speech production, speech disorders, internal models, tendon vibration

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Most complex goal related movements, including speech, that are made to achieve a particular behavioural goal invariably involve the coordination of multiple joints or articulators. As such, understanding the physiological basis of coordination will contribute to our understanding of the processes underlying the control of these movements. Research over the past thirty years has confirmed that intact kinesthetic feedback appears necessary for the successful and efficient coordination of complex multi-articulatory movements (Cordo et al., 1995; Ghez, Gordon, Ghilardi, Christakos & Cooper, 1990; Sanes, Mauritz, Dalakas, & Evarts, 1985). Although most of this research has either involved limb or trunk movements or has used animal models, a number of studies indicate that kinesthesia also has a active role in speech motor control (Abbs, Gracco, & Cole, 1984; Johansson, Trulsson, Olsson & Abbs, 1988; Loucks & De Nil, 2000).

This review paper will address five issues concerning the role of kinesthesia in speech motor control:

1. What is kinesthesia?

2. Is there physiological evidence for speech-related kinesthesia?

3. Does kinesthesia contribute to movement coordination?

4. What significance does kinesthesia have for theoretical questions in speech production research?

5. Is there a connection between speech disorders and kinesthetic deficits?

For further discussions of these issues, the reader is referred to a number of excellent reviews of the physiology of somatosensation and kinesthesia (McCloskey, 1978; Prochazka, 1996), its role in movement control (Prochazka, 1996), and its role in speech/oral motor control (Abbs, 1997; Kent, Martin, & Sufit, 1990).

What is Kinesthesia?

Kinesthesia, typically defined, is the sensation of position and movement of those parts of the body (articulators/joints) moved by striated muscle (e.g., knowing where your arm is in space and where it is moving). This definition, however, does not capture the full scope of this basic physiological mechanism because kinesthesia encompasses a group of sensations related to position and movement of an articulator: muscle sensations related to force level, heaviness, and muscle stiffness/tension); sensations for timing muscle contractions; and, sensations of integrated body posture (Gandevia, 1996). These sensations arise from kinesthetic inputs, which are afferent signals from somatosensory receptors, and from motor command signals, which are corollary discharges and efference copies. Collectively, kinesthetic inputs and motor command signals are referred to as kinesthetic signals. Further, kinesthetic signals are capable of evoking percepts but this is not an exclusionary criterion, as kinesthetic signals can also be processed subconsciously, particularly for control of rapid skilled movements (Gandevia, 1996).

Proprioception is the more general mechanism that refers to all sensations of position and movement of the limbs, trunk, and oral articulators that are encoded by sensory receptors (Prochazka, 1996). Proprioception does not encompass motor command signals nor is perception entailed. Physiologically, the same afferent signals generate sensations of proprioception and kinesthesia so the mechanisms clearly overlap. In a functional sense, accessibility to perception and consciousness suggests that kinesthetic inputs and motor command signals are processed cortically (kinesthesia incorporates proprioceptive signals that reach the cortex). On this basis, kinesthetic sensations of integrated body posture (e.g., perception of the whole limb in reference to the body or integrated vocal tract postures) may indicate that kinesthetic inputs are encoded in cortical somatosensory maps (Gandevia, 1996).

Kinesthetic inputs arise from the sensory information transmitted from somatosensory receptors in muscles, joints, and skin to cortical somatosensory areas. Although there has been a longstanding debate over which somatosensory receptors actually contribute to kinesthesia, it is generally considered that muscle spindles, joint receptors, cutaneous mechanoreceptors, and golgi tendon organs generate kinesthetic inputs (Gandevia, 1996; McCloskey, 1978). The somatosensory receptors that generate kinesthetic inputs within the orofacial complex conform to these types of receptors; however, their exact morphology, density, and distribution differs from the limbs and trunk (Dubner, Sessle, & Storey, 1978). Muscle spindles are present in the jaw closing muscles, lingual muscles, levator palatine and in very low densities in certain intrinsic laryngeal muscles, but are not present in the facial muscles and most intrinsic laryngeal muscles (Dubner et al., 1978; Rowlerson, 1990; Sanders, Han, Wang, & Biller, 1998). Both rapidly adapting (RA) and slowly adapting (SA) cutaneous mechanoreceptor types provide rich sensory innervation throughout the facial skin, oral mucosa, and laryngeal mucosa (Dubner, Sessle & Storey, 1978). Golgi tendon organs (GTO) and joint receptors are only present in the masseter and temporomandibular joint respectively (Capra & Dessem,1992). Somatosensory signals from receptors in the oral tract and face pass through the trigeminal sensory complex to the thalamus, and then to

the facial somatosensory cortex and cerebellum (Capra & Dessem, 1992; Dubner et al., 1978). Laryngeal sensation is conducted through the superior laryngeal nerve to the solitary nucleus and trigeminal complex (Capra, 1995). Other sensory receptors in the oral tract and larynx appear to respond to changes in air pressure/flow and could potentially provide reliable signals of speech kinematic and kinetic events, but their role is beyond the scope of this paper (see Davis, Bartlett, & Luschei, 1992).

An equally important aspect of kinesthesia involves the motor command signals - corollary discharges and efference copies. These kinesthetic signals have been consistently recognized in thinking on motor control and speech production, but have been debated more vigorously than kinesthetic inputs and have been less amenable to empirical study (Gandevia, 1996). Kinesthetic signals have acquired a new prominence, however, through recent applications of the internal model concept(s) from robotics to motor control (see Section 4 of this article, What significance does kinesthesia have for theoretical questions in speech production research?; Blakemore, Wolpert, & Frith, 1998; Guenther, Hampson, & Johnson, 1998).

A corollary discharge is the general term for signals derived from motor commands that are sent to sensory areas (Gandevia, 1996). A primary role for corollary discharge is to estimate the *sensory* outcome of an action, in order to *cancel* redundant sensory signals generated by the movement (reafference), thereby, allowing only the most relevant information to influence the movement. Sensory cancellation also enables the system to distinguish whether a sensory input is externally or internally generated. For example, corollary discharges during eye tracking allow the visuomotor signal to determine if the environment is in motion or the eye is in motion. Sensory cancellation is also likely responsible for the nontrivial observation that it is very difficult to tickle oneself – expected sensation cancels self-generated sensation- and how the auditory system distinguishes self-generated speech from others' speech signals. An efference copy is an exact copy of a motor command. Knowledge of a motor command allows the motor system to estimate the outcome of the command. The evidence for efference copies (but also generally for corollary discharges) comes from studies of the perception of heaviness or movement effort and perception of movement timing (see Gandevia, 1996 for a thorough review of motor command signals).

Is There Physiological Evidence for Speech-Related Kinesthesia?

In line with the first sense of kinesthesia, evidence for somatosensory encoding of movement and position has been obtained through physiological recordings of afferent neuron activity during movements. Somatosensory receptors can actually encode the amplitude, direction, and velocity of movement (Edin & Abbs, 1991; Gandevia, 1996; Grill & Hallett, 1995; Prochazka & Gorassini, 1998). Studies of somatosensory areas in the central nervous system also indicate that somatosensory afferent neurons in the brainstem, thalamus, and cortex encode kinematic parameters (Lenz et al., 1990; Lin, et al., 1994a, b; Mima et al., 1997; Ro & Capra, 1995). The demonstration that kinematic information is processed in the somatosensory system is suggested to be evidence that: a) sensory signals indicating movement position and movement velocity are the physiological substrate of kinesthesia and b) the signals are ostensibly available for motor control. The following section will address direct and inferential evidence indicating that oral somatosensory receptors can encode speech movement patterns and that this information is relayed to the central nervous system.

Oral Somatosensory Receptor Function

Research on orosensory physiology to date indicates that cutaneous mechanoreceptors and muscle spindles encode oral/speech movement kinematics (Johannson et al., 1988a, b; Nordin & Hagbarth, 1989; Nordin & Thomander, 1989; see also Appenteng, K., 1990; Larson, Finocchio, Smith, & Luschei, 1983; and Ro & Capra, 1995 for oral sensory afferent recordings in animal models). Relating the activity of these receptors to kinesthetic perceptions would thereby establish a physiological basis for speech kinesthesia. Although joint receptors and GTOs may contribute to oral/speech kinesthesia, their role has not been studied.

Cutaneous Mechanoreceptors

Cutaneous mechanoreceptors are present throughout the vocal tract and appear to have very similar physiological properties to mechanoreceptors found in the hand (Dubner et al., 1978; Edin, Essick, Trulsson, & Olsson, 1995; Nordin & Hagbarth, 1989; Trulsson & Johansson, 2000). Traditionally, it was thought that cutaneous mechanoreceptors were predominantly involved in tactile perception and had a minimal or negligible kinesthetic role (McCloskey, 1978). Recently, however, it has been demonstrated that these receptors can encode kinematic signals (Edin & Abbs, 1992; Edin & Johansson, 1995; Gandevia, 1996). The availability of kinesthetic signals from orofacial cutaneous mechan-

oreceptors indicates that speech kinesthesia would be far more robust than if kinesthetic signals were available only from other receptor types such as muscle spindles.

Direct evidence for cutaneous mechanoreceptor contributions to kinesthesia comes from microneurography, which is an experimental technique used in humans to identify somatosensory receptors and record their activity. Microneurography recordings indicate that cutaneous mechanoreceptors can encode the direction and velocity of finger movements (Edin & Abbs, 1992). In this study, finger flexion movements activated a sensory neuron with a receptive field on the dorsal surface of the hand. Afferent activity increased with flexion and fell off during extension. This afferent activity was evoked when the finger movement stretched the skin within the receptive field of the neuron (Edin & Abbs, 1991; Edin, Essick, Trulsson, & Olsson, 1995). This movement-related sensory signal could presumably inform the brain on the dynamic position of the finger. Remarkably, if the skin overlying a neuron's receptive field is stretched in a manner that resembles the movement, but without any actual movement, the evoked afferent activity is very similar to the movement-evoked activity (Edin, 1992). This demonstrates that it is the somatosensory receptor that generates the kinesthetic information, rather than the corresponding movement.

These findings aid in the interpretation of microneurographic recordings during speech production. In two studies, recordings were made from cutaneous mechanoreceptors with facial receptive fields while participants produced short utterances (Johansson et al., 1988; Nordin & Thomander, 1989). The afferent activity of these neurons closely corresponded to labial speech events during syllable production. As shown by Johansson et al., multi-unit afferent activity and single unit afferent activity increases in relation to lower lip movements for the production of bilabial stops. The afferent activity appeared to be maintained during the stop gap and release for /p/, then fell off with production of the following vowel. Based on such patterns of movement-evoked afferent activity during both hand and speech movements, Connor and Abbs (1998a, b) have developed a computational model for labial kinesthesia. The predictions of the models are that facial skin stretch caused by lower lip movement is sufficient for accurate kinesthetic encoding of lower lip kinematics during speech (Connor & Abbs, 1998a). Together, the microneurography and modelling research provide intriguing evidence that cutaneous mechanoreceptors appear capable of encoding ongoing speech movements.

Muscle spindles. For the past thirty years, muscle spindles have been considered the primary contributors to kinesthesia (McCloskey, 1978). However, a broader view is required given the findings reported in the previous section (Gandevia, 1996). Muscle spindles are complex intramuscular sensory receptors that encode muscle stretch. Basically, this information is used by the sensorimotor system to resolve the position and velocity patterns of the joint to which they are attached (for a review see Gordon & Ghez, 1991). Joint movement stretches the antagonist muscle(s) activating both the primary and secondary afferents of the muscle spindles. The spindles produce a burst of activity that encodes both the displacement of the joint to a new position and the velocity of the movement. As muscle spindles are present in almost all skeletal muscles, the somatosensory system can receive information on the position and movement of almost all joints in the body. As previously mentioned, the problematic issue for understanding muscle spindle activity in speech kinesthesia is that facial muscles and intrinsic laryngeal muscles generally lack spindles (Dubner et al., 1978). Nonetheless, legitimate inferences can be made for speech because research indicates that oral muscle spindle behaviour in muscles such as the masseter parallels limb muscle spindles (e.g. the monosynaptic jaw-stretch reflex; Dessem, Donga, & Luo, 1997; Dubner et al., 1978; Larson et al., 1983; Weber & Smith, 1987). This would suggest that masseter muscle spindles and tongue muscle spindles could encode the articulatory position and velocity of their respective articulator. The role of muscle spindles in kinesthesia will be discussed in more detail in the section below on tendon vibration.

Central processing of oral kinesthetic information during speech production. In a single study, thalamic activity in the ventral posteromedial nucleus (VPm – orofacial thalamic sensory area) arising from speech production was recorded during surgery for Parkinson's disease (McClean, Dostrovsky, Lee, & Tasker, 1990). In five participants, cutaneous mechanoreceptors with labial receptive fields responded consistently during labial contact and labial movements associated with the bilabial plosive /p/. Neurons in VPm with lingual receptive fields increased activity during tongue movements for vowels and the alveolar plosive /t/ in two participants. Significantly, the patterns of the VPm responses during speech production resemble the results found in the speech microneurography studies reported above, which suggests orofacial kinesthetic information arising from speech movements is preserved at the level of the thalamus.

Another line of emerging physiological evidence comes from positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) studies. Both methods allow for the noninvasive spatial localiza-

tion of brain regions in humans where blood flow shows relative increases or decreases during the performance of a task(s) (Papanicolaou, 1998). Although the evidence is strictly inferential at this time, speech and oral motor tasks produce significant cerebral blood flow (rCBF) increases in brain motor areas that are implicated also in kinesthetic processing (speech studies - Bookheimer, Zeffiro, Blaxton, Gaillard, & Theodore, 2000; Reicker, Ackermann, Wildgruber, Dogil, & Grodd, 2000; sensorimotor activation - Mima et al., 1997; Korvenoja et al., 1999; Mima et al., 1999; Naito, Ehrsson, Geyer, Zilles, & Roland, 1999). Not surprisingly, such tasks produce activation in the primary oral sensory cortex, which suggests oral sensory information is transmitted to the cortex (Lotze, Seggeweis, Erb, Grodd, & Birbaumer, 2000; see also Lin, Murray, & Sessle, 1994a, b for afferent recording from orofacial SI in monkeys during an oral motor task). Speech production by healthy right-handed individuals also activates the right lateral cerebellum (Reicker et al., 2000). This has particular significance since the cerebellum is known to receive extensive projections from somatosensory receptors that encode kinesthetic information and cerebellar disease is known to produce kinesthetic and motor control deficits (Grill, Hallett, Marcus, & McShane, 1994; Grill, Hallett & McShane, 1997). Although the significance of these neuroimaging findings for speech related kinesthesia is still by association, the common cerebral areas that are active during speech production and kinesthetic tasks, support the position that somatosensory information influences cortical and subcortical activation during speech production.

Evaluation of physiological evidence. The current scope of physiological evidence for speech kinesthesia shows that speech/oral movements are encoded peripherally by oral somatosensory afferents and centrally by thalamic and cortical sensory neurons. This evidence corresponds to known somatosensory pathways documented for animal models and human limb movements. More importantly, this evidence indicates that kinesthetic inputs are available to the central nervous system during speech production. The caveat to these findings is that it is not clear how or if this peripheral kinesthetic information is actually used for speech motor control. To strengthen the argument for speech kinesthesia, it must be shown that afferent signals can modulate the ongoing production of speech movements.

Does kinesthesia contribute to movement coordination? To demonstrate the role of kinesthesia in speech motor control or in general movement motor control, the researcher must be able to experimentally manipulate somatosensory activity while simultaneously recording movement. The various methodologies employed to manipulate kinesthesia encompass deafferentation studies, perturbation studies, and tendon vibration.

Deafferentation

Recent evidence confirming the fundamental role of kinesthesia in motor control has come from studies of patients with sensory neuropathies. These diseases destroy the large sensory afferents that carry all kinesthetic and tactile signals (below the neck) while force production (strength), pain sensation, and temperature sensation are spared. Systematic studies indicate these patients essentially lack somatosensation: they lack stretch reflexes, cannot perceive the positions and movements of their limbs, and show profound discoordination (Ghez et al., 1990; Ghez & Sainburg, 1995). The three most relevant findings from these studies are:

1. The patients showed large errors in pointing movements made without visual feedback and considerable drift in limb position after finishing the movement. This indicates their motor system did not have access to position or movement information (Ghez et al., 1995; Sanes et al., 1985).

2. They could not effectively couple or coordinate two or more joints for functional movements, (e.g., reaching, cutting bread; Ghez, & Sainburg, 1995). This study showed they were not able to compensate for joint interaction torques.

3. In pointing movements where vision of the hand was excluded, the patients showed errors in orienting the starting hand position towards the target.

The second two points suggest that kinesthetic information has a predictive role in movement contol in that kinesthetic information was necessary prior to movement onset to compensate for biomechanical interactions and to adjust articulatory trajectory. This predictive function suggests kinesthetic information has a role in movement planning. As will be discussed below, this evidence has been used to suggest that kinesthesia is necessary to maintain internal models of movement control (Gordon, Ghilardi, & Ghez, 1995; Ghez et al., 1995). (Although case reports of trigeminal sensory neuropathies are largely descriptive and somewhat anecdotal, there are indications this neuropathy is associated with speech and swallowing impairments [Auger & McManis, 1990; Lecky, Hughes, & Murray, 1987]).

A different line of research has employed local anaesthetics to suppress somatosensory information from an articulator/limb while that limb is engaged in a movement task. There is a voluminous body of research, including speech research, using this approach that encompasses widely differing theoretical approaches,

highly varying tasks, and longstanding criticisms. The central criticisms are: a) not all sensory information may be suppressed, so residual sensory information coming from the articulator may contribute to task performance; b) the anaesthesia may dampen efferent as well as afferent function; and c) the loss of a mechanism does not provide direct information on how that mechanism functions normally. (This criticism is also relevant for the peripheral neuropathy studies; however, the experiment control and clear results of those studies support the interpretations.) These criticisms are especially relevant for the studies assessing the effects of local anaesthesia on speech production (Abbs, Folkins, & Sivarajan, 1976; Borden, 1979). In these studies, there was some indication that anaesthesia did perturb speech kinematics and production, but acoustic/perceptual analyses were not very sensitive to these effects (Abbs et al., 1976; Putnam & Ringel, 1976). Although the results do not support a role for continuous somatosensory feedback in speech production, they may speak more to the contribution of oral somatosensation to adaptive control of speech production. The mature speech motor systems of adults likely do not depend on constant feedback to maintain accurate production, whereas the less mature systems of children may require more consistent and accurate feedback to maintain control. Preliminary evidence for this comes from the finding that oral anaesthesia interfered more with speech production in four-year-old boys than in adults (Borden, 1976).

There are several relevant studies of anaesthetic effects on finger movements (Ferrell & Smith, 1988; Gandevia & McCloskey, 1976). In one study, the participants' threshold for detection of finger movement was determined in separate conditions: a) joint and cutaneous mechanoreceptors were suppressed with anaesthesia; b) muscle spindles were suppressed by disengaging finger muscles; and, c) a control condition (i.e., normal kinesthetic sensibility; Gandevia & McCloskey, 1976). Elimination of either kinesthetic input caused an increase in the movement threshold compared to the control condition indicating that each of the receptors contribute to kinesthesia. The findings highlight the general principle that intact kinesthesia, particularly that of body posture, depends on the integrated contribution of all the somatosensory receptor types.

Perturbation Studies

Movement perturbation paradigms have been used extensively in speech motor control research, and perhaps are the most widely noted contributions from speech research to the general motor control literature. The perturbation is used to alter the oral sensorimotor environment during speech articulation. If compensations to the perturbation are highly functional and rapid, it suggests that oral sensation (i.e., kinesthesia) is available for online sensorimotor control over speech movements. The two standard types of perturbations used in speech research are classified as static and dynamic perturbations.

Static perturbations

In static perturbation studies, either fixation of the jaw is induced through a bite block or vocal tract morphology is altered through insertion of an artificial palate. Typically, comparisons of speech variables are made with and without these oral prostheses in place. The variables of interest are usually whether acoustic patterns and the perceptual quality of a particular speech segment changes following the perturbation, and the latency for the perceptual quality to normalize.

In older studies, researchers reported that acoustic patterns and perceptual quality of vowels were not affected by the bite block perturbation (Kelso & Tuller, 1983; Lindblom, Lubker, & Gay, 1979). It was emphasized that the successful compensations were present on the first production. This may suggest that somatosensory information (kinesthetic & tactile) can be used to predictively adjust the speech system regarding state changes prior to actual speech production under the altered state. This is viewed as evidence that the speech system maintains robust internal models that permit immediate and flexible accomodations to changing conditions (Guenther et al., 1998; Perkell et al., 1997).

Recent studies, however, show that while highly acceptable speech can be produced with the bite block, significant alterations in the acoustic patterns and perceptual quality of both consonants and vowels can occur (Flege, Fletcher, & Homeidan, 1988; McFarland & Baum, 1995). McFarland and Baum (1995) have shown compensation for the bite block is often not complete, but that participants improve their production (particularly for the vowels) after a conversational practise period with the bite block in place. The training effects suggest that multi-modal sensory feedback, kinesthetic, tactile, and auditory, – are likely contributing to any compensations, but the loss of jaw flexibility still impedes complete compensation.

Artificial palates also cause considerable acoustic and perceptual distortion in the production of certain speech segments (Hamlet, & Stone, 1978; McFarland, Baum, & Chabot, 1996). Interestingly, McFarland et al. (1996) showed that the pattern of speech perturbations differs from the bite block effects, as vowels may be less affected than consonants (one exception is the /i/ vowel

where tongue palatal contact occurs). For example, the fricative /s/ is distorted considerably (acoustically and perceptually) along with some effects on stop consonants. The differences in effects between the bite block and artificial palate studies are consistent with the differences in their production: Vowels are more affected by loss of jaw motion while fricatives and stops are more affected by changes in oral tract morphology. Also, improvement in production with the artificial palate does take place, but considerable practise is required, high individual variability is present, and compensation may not be complete (Hamlet & Stone, 1974; McFarland et al., 1996). As per the bite block studies, kinesthetic feedback (also tactile and auditory) is likely necessary to make accommodations to the perturbation so that speech production can proceed, but that biomechanical/structural changes can still exceed sensorimotor adaptability in some participants (Savariaux, Perrier, & Orliaguet, 1995). The observed improvement with practise suggests that sensory feedback may facilitate compensation through a motor learning process (McFarland et al., 1995, 1996).

The interpretation of static perturbation studies regarding sensory compensation is facilitated when speech physiological measures are recorded along with acoustic and perceptual measures. For example, kinematic data would clearly aid in the interpretation of how the compensations occur (see also Flege et al., 1988; Hamlet & Stone, 1978). In one important bite-block study, cineradiographic recordings of tongue movements were obtained during vowel production in the unperturbed and perturbed conditions (Gay, Lindblom, & Lubker, 1981). The authors report that perceptual quality of the vowels did not change across the conditions, whereas tongue kinematic profiles showed much greater amplitude and considerably altered patterns in the bite block condition. It appears that rapid reorganization of tongue control allowed for a considerable degree of motor equivalence, adaptation, and predictive control of speech production.

Dynamic perturbations

Dynamic perturbations are used to transiently impede movement execution. Speech perturbations typically involve a mechanical force that transiently resists jaw or lower lip movements during a jaw/labial closing gesture. Most studies have successfully applied perturbations of known force during specific phases of oral closing gestures. Two important finding were that participants rapidly compensated for the perturbation on the first perturbation trial and that the perturbations either minimally affected of did not affect the perceptual quality of the speech gesture - even though certain perturbations were of a large amplitude. Participants showed rapid kinematic and muscular compensation by increasing the amplitude of other articulatory movements such as upper lip movement and oribicularis oris superior activation to accomplish a bilabial speech goal (/p/ or /b/) - or increased genioglossus activation to achieve alveolar contact (/z/; Abbs et al., 1984; Kelso, Tuller, Vatikiotis-Bateson, & Fowler, 1984). These gesture-specific compensations occur within 40-100 ms of the perturbation, which exceed orofacial reflex latencies, but are not extremely rapid. Functional compensations over the same time course have also been seen in laryngeal movements following dynamic labial perturbation (Saltzman, Lofqvist, Kay, Kinesella-Shaw, & Rubin, 1998; Shaiman, 1989), which indicates this compensatory mechanism is available to structures with loose/minimal biomechanical coupling. Owing to the rapidity and functionality of these compensations, it was suggested that a feed-forward mechanism generated the response based on somatosensory information related to the perturbation (Abbs & Gracco, 1984). In this view, orofacial sensorimotor linkages allow for a predictive relationship between an ongoing movement and an upcoming movement. The speech motor system is considered to receive online afferent information that it uses to correct upcoming speech gestures (Gracco, 1997).

Evaluation

Together, the static and dynamic perturbation studies reveal an inherent flexibility in the speech motor system. The evidence for a predictive role of sensory information (kinesthetic & tactile) in these compensation is important for three reasons: a) The system is not restricted to closed-loop feedback for compensation; b) The availability of sensory information at such short latencies means that predictive compensation can potentially occur at planning stages or during online production; c) An internal representation or internal model of the task likely functions to transform the predicted sensory errors into appropriate motor commands.

The real caveat for understanding the role of kinesthesia in the perturbation studies is that compensations were often practise-dependent, subject-dependent and often incomplete. When practise is provided, it is apparent that both auditory and somatosensory feedback will be available for compensation. This is just one example where the roles of both auditory and kinsthetic information must be integrated to provide a full explanation. In fact auditory-kinesthetic-tactile integration in speech may be the appropriate reference for speech production as the visuo-kinesthetic-tactile integration is for reaching movements. The integrated sensory information may be necessary to adapt the previously mentioned

internal model to the altered vocal tract. It is also relevant to note that recent findings suggest somatosensory feedback relevant to an ongoing movement is enhanced in novel movement conditions such as perturbations, and could function to facilitate adaptation (Knecht, Kunesh, Buchner, & Freund, 1993; Prochazka, 1989; Staines, Brooke, & McIlroy, 2000).

A clear limitation of the perturbation paradigms for assessing kinesthesia's role is the lack of specificity of the stimuli. Clearly, there are affects on oral sensation, but there is no information regarding which sensory receptors respond to the perturbation or what the magnitude of the response is. Direct monitoring of sensory changes and knowledge of the receptor types responding to the perturbation are necessary to understand the role of kinesthesia.

Tendon Vibration

The tendon vibration paradigm addresses these shortcomings by allowing for specific manipulation of muscle spindle activity during functional movements. (See also Andreatta, Barlow, Biswas, & Finan, 1996 for a labial perturbation paradigm that targets labial cutaneous mechanoreceptors specifically.) The application of a vibratory stimulus to a tendon selectively entrains primary muscle spindle afferents without preventing movement. Microneurographic recordings have demonstrated that tendon vibration produces quasi-linear changes in the primary muscle spindle activity over a 20-80 Hz vibration range (Cordo, Burke, Gandevia, & Hales, 1998; Roll, Vedel, & Ribot, 1989). This technique has been shown to directly affect kinesthetic awareness through either a movement illusion effect or a movement undershoot effect (depending on the task; Goodwin, McCloskey, & Matthews, 1972; Roll & Vedel, 1982; Sittig, van der Gon, & Gielen, 1987). These effects, which were established in limb movement research, have been extended to speech/oral movements.

Movement illusion. The movement illusion effect established that kinesthetic percepts can be elicited in the absence of movement. Although this is somewhat counter-intuitive, it shows that sensory input gives rise to the perceptions of position and movement. The typical protocol of movement illusion studies involves the application of vibration to a muscle tendon of a restrained limb. During the vibration period, participants consistently report that the vibrated limb is moving or has changed position - even though movement has not occurred. If participants are asked to track the perceived position of the restrained limb with the contralateral unrestrained limb, most participants move the contralateral limb over a large amplitude while the vibrated limb remains stationary (Goodwin et al., 1972). The vibration further affects movement velocity perception, because, when requested, participants will also track the velocity of the perceived movement (Sittig et al., 1987). The illusion is caused by the enhanced muscle spindle activity, which misinforms the brain that a movement is occurring. Interestingly, the magnitude of the tendon vibration effect is directly related to the vibration frequency (Calvin-Figuiere, Romaiguere, Gilhodes, & Roll, 1999; Sittig et al., 1987). The illusion effect has been found throughout the body (neck, spinal cord, elbow, wrist, knee, ankle) for both simple and complex movements, suggesting that kinesthesia is a basic mechanism.

Accordingly, a jaw movement illusion has served to demonstrate that oral kinesthesia is also highly developed. Hellsing (1978) showed that unilateral masseter tendon vibration can induce a jaw opening illusion. Tendon vibration was applied to the masseter while jaw opening movements were prevented. Eight participants produced finger tracking movements corresponding to a perception of jaw opening or they verbally reported a sensation of jaw opening. This result confirms a role for kinesthesia in jaw control and demonstrates the feasibility of tendon vibration for oral motor research.

Two recent movement illusion studies are highly relevant for this discussion since the authors reported the finding that a reliable movement illusion can be elicited by stimulating cutaneous mechanoreceptors (Collins & Prochazka, 1996; Edin & Johansson, 1995). In these studies, controlled stretches were applied to the dorsal skin of the hand while the fingers and hand were restrained from movement. As per the tendon vibration illusion, participants consistently reported that their finger was in motion or had changed position. This finding is remarkable, first because it establishes a clear perceptual basis for cutaneous mechanoreceptors in kinesthesia and, secondly, it challenges the view that muscle spindles are the predominant contributor to kinesthesia. A significant experimental challenge would involve replication of the cutaneous based movement illusion in facial muscles. This would confirm that facial movements are encoded kinesthetically despite the lack of muscle spindles.

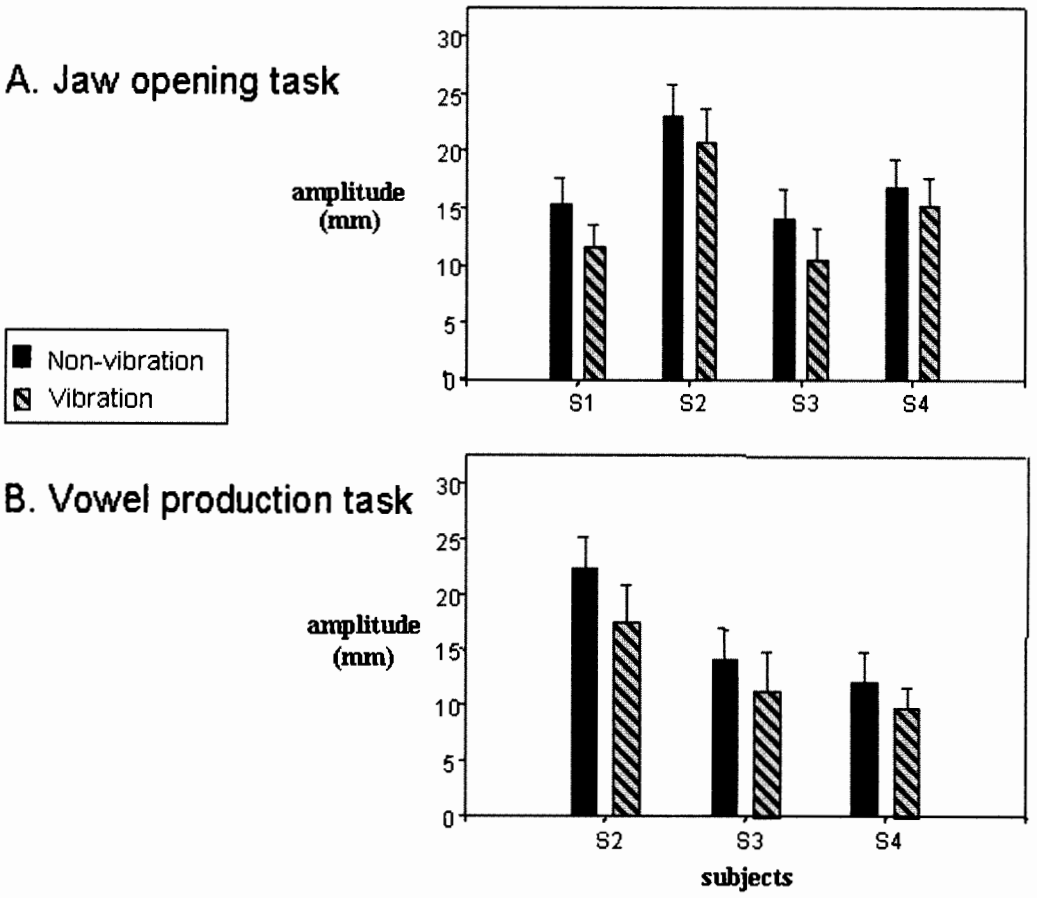
The importance of the movement illusion is that kinesthetic perceptions of position, movement, and velocity are driven by somatosensation. As muscle spindles and cutaneous mechanoreceptor stimulation can evoke this kinesthetic perception, it suggests that both receptors could contribute to speech kinesthesia. A straightforward implication may be that cutaneous-based kinesthesia from muscle groups lacking muscle spindles and muscle spindle based kinesthesia from the masseter is complimentary, thereby contributing to an integrated kinesthetic awareness of vocal tract posture.

Movement undershoot. The other relevant tendon vibration effect is the movement undershoot phenomena. In these studies, single-joint or multi-joint movements are performed while vibration is applied to an antagonist tendon. Participants consistently report that their limb position during vibration is more extended than its actual position or that limb movement velocity is higher than its actual velocity. For example, Inglis and Frank (1990) demonstrated that when vibration (95 Hz) was applied to the triceps during contraction of the biceps, the amplitude and velocity of bicep flexion movements was reduced significantly compared to movements without vibration. This marked undershoot effect is consistently present when tendon vibration is applied to the antagonist muscle, but is not present when vibration is applied to the agonist (Inglis & Frank). The undershoot effect further confirms the role of muscle spindles in kinesthetic perceptions of position and velocity and shows that kinesthesia has systematic, short latency effects on movements.

In two recent studies, the movement undershoot effect was observed in jaw opening movements. The first experiment comprised two separate movement conditions: nonspeech jaw opening movements and vowel production (Loucks & De Nil, 2000). Tendon vibration (90 Hz) was applied randomly on 50% of the jaw movement trials. Figures 1a and 1b shows the peak jaw opening amplitude for both tasks and conditions. In both the speech and nonspeech tasks, jaw opening amplitude was significantly lower in vibration trials compared to nonvibration trials. Importantly, the undershoot effect was present for all participants. This appears to be the first extension of the tendon vibration paradigm to ongoing speech and oral movements.

A follow-up study investigated the effect of tendon vibration on the spatial accuracy of jaw opening movements (Loucks & De Nil, 2000). Eight healthy male participants were trained to make accurate jaw-opening movements to a fixed target (18 mm) in the absence of visual feedback. Tendon vibration was applied randomly

Figure 1
Effect of unilateral masseter tendon vibration on jaw opening peak amplitude: a) Non-speech task; and, b) Vowel production task.



on 50% of the trials. Figures 2a and 2b shows the mean peak amplitude and peak velocity for all participants in both conditions. In the nonvibration condition, the mean jaw displacement approached the target displacement. In the vibration condition, jaw opening amplitude decreased significantly indicating the undershoot effect was present. A nonsignificant reduction in peak velocity is also present. These findings effectively replicate the oral movement condition of the first study in a larger sample and with increased experimental control.

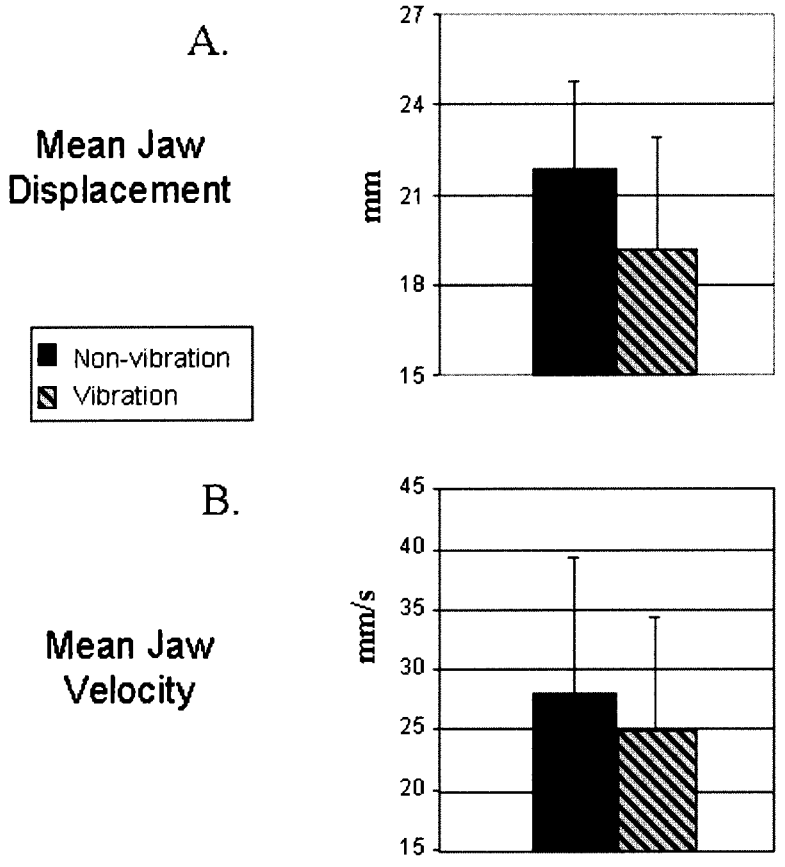
The masseter tendon vibration studies have shown that masseter muscle spindle contributions to oral and speech kinesthesia parallel findings in the general motor control literature (Cordo et al., 1998; Inglis & Frank, 1990; Sittig et al., 1987). Given the feasibility of masseter tendon vibration for research in speech kinesthesia, it is expected that this paradigm can be extended to the study of multi-articulatory speech gestures.

What significance does kinesthesia have for theoretical questions in speech production research?

The empirical studies reviewed here are considered to provide strong evidence for kinesthesia as an integral factor in motor control. Although the evidence for the role of kinesthesia in speech motor control is more preliminary, it is reasonable to infer an important role for kinesthesia. This has been recognized by the explicit inclusion of kinesthetic information in theories of motor control and speech production (Guenther et al., 1998; Kent, Adams, & Turner, 1997; Neilson & Neilson, 1987). A full review of these theories is outside the scope of the paper, but the ideas discussed in this section emphasize several aspects of kinesthesia that have particular significance for theoretical questions.

First, a dichotomous view of motor control or speech production as either open-loop and closed-loop is viewed as untenable (Desmurget & Grafton, 2000; Keele, 1986;

Figure 2
Effect of unilateral masseter tendon vibration on jaw opening peak amplitude and peak velocity.



Kent et al., 1997). Rather both peripheral feedback signals and open-loop motor commands are typically integrated in motor control. One bridge between these mechanisms is a feed-forward system in which kinesthetic information on current articulatory position and velocity (initial conditions) is used to adjust upcoming motor commands. This mechanism also allows the system to adapt ongoing movements to changes in the movement context. Evidence for this feed-forward role of kinesthesia was discussed in the sections describing the deafferented patients and speech perturbations. Arguably, this predictive role is the new scope of kinesthesia that holds the most possibility for inclusion of kinaesthetic information in theories/models of skilled movement, motor learning and movement disorders.

One theoretical framework that allows for predictive role is the internal model concept. An internal model is a neural representation that mimics the input/output characteristics of the motor system allowing for predictive control over movement (Kawato, 1999). Very generally, a forward internal model predicts the outcome of a movement and an inverse internal model predicts the motor command needed to make the movement. Forward and inverse internal models are regarded as capturing essential elements of sensory to motor transformations. In terms of kinesthetic contributions to movement control, it is thought that corollary discharges produced by a forward model can predict the kinesthetic outcome of a movement prior to movement execution. The motor command required to produce a desired movement is the efference copy generated by an inverse model. The corollary discharge and efference copy of the motor command could then be compared during motor planning to determine if the motor command can actually produce the desired movement outcome. If not, internal corrections can be made to the motor plan. In this way, these kinaesthetic signals constitute a form of internal feedback for correction of motor commands during the planning process. The strongest evidence for the predictive capacity of internal models comes from grip force studies in which normal participants can easily predict the grip force needed for stable grasp control (Flanagan & Wing, 1997). As stated previously, several current computational speech models also posit that internal models are operative in the predictive control of speech production (Guenther et al., 1998).

Internal models are also relevant for this discussion because sensory feedback is required to train and update the models. Internal models are instantiated in neural network computations that mimic the learning of behaviours, such as speech production. During training, internal models require extensive feedback, but following learning, only intermittent sensory information is needed to maintain the model. As the internal model does not have access to appropriately referenced sensory information during learning, erroneous motor commands are produced. However, sensory encoding of these errors provides a training signal used to correct the internal model. Under the DIVA model (Guenther et al., 1998) and the Adaptive Model Theory (AMT; Neilson & Neilson, 1987) of speech, auditory feedback is hypothesized to be the primary sensory modality used to train the internal model and kinesthetic/tactile information is used to maintain/update the internal models. For example, kinesthetic information could maintain precise and accurate internal models for speech segment production in adults for long periods following postlingual deafness, but hearing is necessary for actual speech learning and controlling suprasegmental parameters (Burnett Freedland, Larson, & Hain, 1998; Perkell et al., 1997).

The foregoing section alluded to the critical problem of accounting for the role of sensory information in motor learning and speech development. A developmental role for kinesthesia has been relatively neglected and is poorly understood compared to knowledge of kinesthesia in the mature system. In contrast to the previous discussion of predictive control, closed loop feedback may be more important for the developing system. The closed-loop feedback signals are likely multimodal, but audition is considered the primary sensory signal for developing speech segmental control (Callan, Kent, Guenther, & Vorperian, 2000). During development, the motor system learns to associate the desired acoustic output or auditory percept (which also carries information on articulatory positions) with kinesthetic signals. The shorter latency for kinesthetic signals compared to auditory signals could then make kinesthesia more relevant for rapid modification of speech motor command signals as maturation progresses. One relevant line of research has provided evidence that a kinesthetic deficit may partially contribute to Development Coordination Disorder, although visuomotor integration is considered the primary deficit (Wilson & McKenzie, 1998).

A limitation for speech kinesthesia research is that the methodologies and interpretations of kinesthesia have primarily come from research in limb motor control and animal models. It is expected though that the specific properties of speech kinesthesia differ from what is observed in limb motor control. First, the different distributions of sensory receptors in the vocal tract and the absence of muscle afferents in many orofacial muscles indicates kinesthetic representations of the orofacial system are built up from different weightings of kinesthetic inputs relative to the limbs. Secondly, kinesthetic

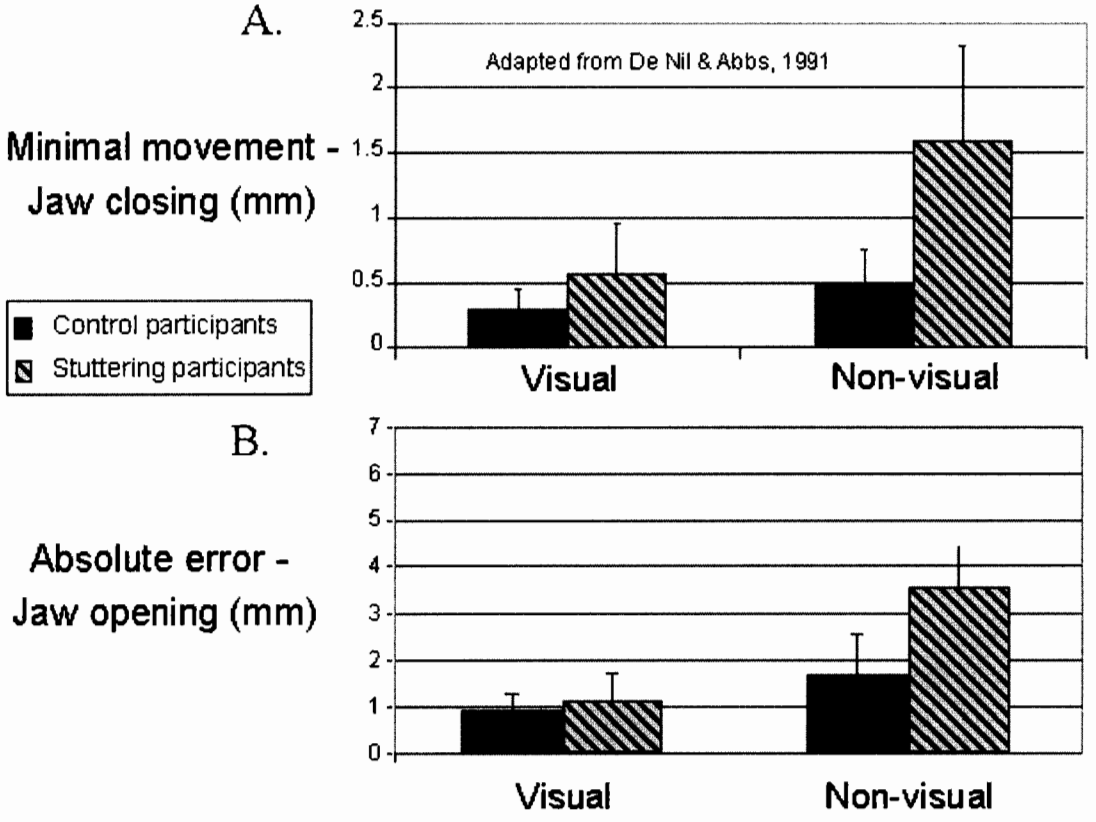
information from ongoing movement (re-afferent information) is available to the speech motor system at much shorter latencies than the limb system (Gracco & Abbs, 1985). This makes online corrections of speech production more theoretically and empirically viable than limb movements. It is anticipated that future research will continue to highlight the empirical and theoretical importance of kinesthesia in motor control and speech production. It is noted that other theoretical approaches to the role of sensory perception in motor control have used nonlinear dynamical approaches and rule based systems (Kelso, 1998; Prochazka, 1996). However, all physiologically plausible theories of speech production and development must account for the relationship between auditory information and kinesthetic information and the distinctive aspects of oral kinesthesia. Overall, the role of kinesthesia is most appropriately investigated in the context of natural multi-articulatory movements. As speech production is a prime example of multi-articulatory control, it is considered an appropriate system for kinesthetic research and speech research is expected to inform general theories of kinesthesia.

Is there a Connection Between Speech Disorders and Kinesthetic Deficits?

The study of kinesthesia should not only add to our understanding of normal speech control, but also inform current clinical approaches to diagnosing and treating speech movement disorders. A clear finding of recent motor control research is that the loss of large afferent fibres that conduct kinesthetic signals results in the severe discoordination of most functional movements (Ghez et al., 1990, 1995a, b). Only slightly less established is the finding that the discoordination of many movement disorders is associated with distur-

Figure 3

a) Minimal movement thresholds for jaw closing in adults who stutter and control participants (Adapted from De Nil & Abbs, 1991). b) Absolute error for jaw opening movements in a target accuracy task: A comparison of adults who stutter and control participants.



bances in kinesthetic integration (e.g., cerebellar ataxia, Parkinson's disease, and dystonia; Grill et al., 1994; Klockgether, Borutta, Rapp, Spieker, & Dichgans, 1995; Rickards & Cody, 1997; Rome & Grunewald, 1999). Of interest for this paper are studies showing relationships between kinesthetic deficiencies and speech motor disorders.

Recent investigations have suggested that adults with chronic developmental stuttering show an oral kinesthetic deficit. In one study, adults who stutter and normally fluent control participants were asked to make the smallest possible movement of their jaw, tongue, lower lip, and finger in separate conditions (De Nil & Abbs, 1991). Minimal movements constitute the smallest possible perceived change in position, so perceptual resolution of these movements depends on intact kinesthesia (when other feedback modalities are not available). The task was performed in separate visual and nonvisual conditions. In the nonvisual condition, accurate performance is largely dependent on kinesthetic guidance. When visual feedback of the movements was provided, group differences were not found. However, when visual feedback was removed, the stuttering participants' minimal oral movements – jaw, tongue, and lip - were significantly larger than the control group (see Figure 3a for jaw movement data). The larger movements of the stuttering group suggest their oral kinesthetic resolution is significantly coarser than that of controls.

In a recent follow-up study, adults who stutter and normally fluent control participants were required to make highly accurate jaw opening movements to a six mm target in visual and nonvisual movement conditions (Loucks & De Nil, 2000). Accuracy in the nonvisual condition would indicate the effective use of kinesthesia to perceive jaw position. With visual feedback, both groups made highly accurate movements. When visual feedback was removed, the adults who stutter showed significantly higher movement error (i.e., less accurate movements) than the control participants (see Figure 3b). This suggests the adults who stutter could not use oral kinesthesia to make accurate jaw movements. The converging evidence from both studies suggests that an oral kinesthetic deficit is associated with chronic developmental stuttering. While recognizing the multidimensional nature of stuttering, the speech discoordination evident in stuttering may be related to limitations in the use of kinesthesia for speech movement control.

A study of oral somatosensory function in patients with Parkinson's disease employed clinical tests to determine whether they showed abnormal somatosensory function (Diamond, Schneider, & Markham, 1986). Clinical measures of jaw kinesthesia, oral tactile localization, oral cutaneous sensation, and labial two-point discrimination were used. The patients showed particularly poor performance on the jaw kinesthesia task compared to age-matched control participants. As these clinical findings for an oral kinesthetic deficit are supported by other studies documenting manual kinesthetic deficits in Parkinson's disease (Jobst, Melnick, Byl, Dowling, & Aminoff, 1997; Klockgether et al., 1995), a relationship between deficient kinesthetic processing and the speech discoordination of Parkinson's disease is indicated.

Initial evidence for a possible oral sensorimotor deficit has also been found in groups of patients with apraxia of speech or cerebellar dysarthria (McNeil, Weismer, Adams, & Mulligan, 1990). The participants were required to maintain a stable articulatory position of the upper lip, lower lip, jaw, or tongue (in separate conditions) using visual feedback. Both patient groups could not maintain static articulatory positions compared to control participants, who performed the task without difficulty. In this task both visual and kinesthetic information were available, so the inability to maintain a stable position generally suggests deficient use of sensory information for oral control. Ineffective processing of kinesthetic feedback may have contributed to performance as kinesthesia informs the motor system on both static positioning and changes in position.

Overall, the evidence from these studies is sufficient to merit continued investigation of kinesthetic deficits as an explanatory or contributing factor in speech disorders. Clinical testing of oral somatosensory function such as two-point dicrimination and vibration sensitivity has a place, particularly if a sensory neuropathy is suspected. Here the review on the merits of noninvasive sensory testing given by Kent, Martin, and Sufit (1990) provides highly appropriate guidelines. The limitations of oral somatosensory tests that are relevant for this review are that clinical tests may not be sensitive to subtle differences in somatosensory function and it's unclear how the outcomes of clinical sensory tests relate to observed impairments in motor control and therapy outcomes. Highly controlled testing of deficiencies in sensory function is often invasive or limited to sensitive experimental testing that is not feasible for most clinical settings (but consider Schnieder et al. [1986] as a systematic application of clinical sensory tests to a sensorimotor disorder). It is argued instead that appreciation of the full scope of kinesthetic contributions to motor control is just as relevant for clinical work as traditional sensory testing.

The view that all movement is actually sensorimotor in nature indicates that transformation of sensory information for motor control is an integrated process. In fact it has been shown that changes in motor control – improvement or deterioration - also affects sensory processing. Abundant evidence from studies of cortical plasticity indicates that improved motor performance and deteriorating control also change cortical sensorimotor representations (Byl, Merzenich, & Jenkins, 1996; Karni, 1995). For example, improved fluency in a group of adult stutterers who underwent intensive therapy (adaptation of the Precision Fluency Shaping Therapy; Webster, 1974) that emphasized speech motor targets also showed corresponding changes in cerebral activation in the cerebellum (De Nil, Kroll, & Houle, 2001). By inference, other therapeutic interventions that explicitly or implicitly enhance speech motor skills should also be accompanied by more effective processing of sensory information that is relevant to the task (e.g., PROMPT therapy for apraxia of speech; Hayden, 1984). In addition, intact motor control integrates sensory information from multiple modalities. While this paper has emphasized the integral role of kinesthesia, it is becoming clear that highly specific acoustic manipulations can actually produce quite rapid changes in the control of speech production (Burnett et al., 1998; Houde, & Jordan, 1998; Perkell et al., 1997). This suggests that interventions in which multi-modal sensory integration for specific speech motor goals is facilitated are more likely to be effective. The last point is that any therapy that contributes to increased automaticity of speech production likely reflects more effective sensory integration - sensory inputs shift from a corrective to a predictive function (Thoroughman & Shadmehr, 1999). In terms of current thinking in motor control, therapies that facilitate the development or consolidation of internal models would contribute to successful treatment outcomes.

Conclusion

The current view in sensorimotor research is that intact kinesthesia is necessary for movement control, and is expressed in the consensus statement that "One can only control what one senses" (McCloskey & Prochazka, 1994, p. 69). There appears to be general agreement with this viewpoint in speech motor research (i.e., that intact kinesthetic processing has a critical role in speech coordination; Abbs, 1996; Gracco, 1997; Kent et al., 1990; Perkell, 1997). This article has reviewed physiological and behavioural evidence that support this perspective. Of greater significance, however, are insights into how this basic physiological mechanism participates in the complex process of movement coordination. The speech microneurography studies provide evidence that labial cutaneous mechanoreceptors have a kinesthetic function. The perturbation studies are evidence that kinesthesia functions both rapidly and in an anticipatory manner to maintain the integrity of speech gestures (Lofqvist, 1990). Tendon vibration has shown that masseter kinesthesia contributes to vowel production in an online manner. The advantage of these methodologies is that they allow for the study of kinesthesia during ongoing multi-articulatory speech movements. This evidence has been recognized in current theories and models of motor control and it is suggested that the internal model approach captures much of the essential aspects of sensorimotor integration for motor control. A small body of literature indicates an association between kinesthetic deficiencies and speech motor disorders, but this work is preliminary. Further consideration of the role of kinesthesia in developmental speech motor control, assessment, and intervention should be goals of future research.

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