PERIPHERAL FEEDBACK MECHANISMS IN SPEECH PRODUCTION MODELS?

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In this paper we are concerned with the problem of whether or not peripheral feedback mechanisms can be used in the control of the muscles during speech. Since most human skeletal muscles, including the cranial muscles, contain proprioceptors known as "muscle spindles" (Cooper 1960, Granit 1970), it is reasonable to hypothesize that feedback mechanisms utilizing such devices may play a direct role in normal speech production. In fact, this proposal has been made by a number of speech production theorists (Ladefoged and Fromkin 1968, Öhman 1967, Tatham 1969, MacNeilage 1970). Nevertheless, there exists no direct evidence that such mechanisms are used during speech. It is quite possible that the muscle spindles in cranial muscles are used to regulate the slower 'tonic' activity of these muscles, but are not employed in regulating the faster 'phasic' activity characteristic of speech.

Ladefoged and Fromkin (1968) reported a pilot experiment designed to gather data on this topic. They suggested that if quick-acting peripheral feedback mechanisms operate in the control of the speech muscles, and if we assume that it is the intention of the speaker to achieve a particular target configuration of the articulators associated with a particular segment, then a mechanical interruption of normal articulatory activity should result in a short-latency increase in the muscle activity controlling the articulator, produced as compensation for the mechanical interruption. Any increase in muscle activity with a latency smaller than about 50 milliseconds could only be attributed to the action of a peripheral feedback mechanism. Ladefoged and Fromkin's experiment involved interruption of labial activity by a mechanical device, and they examined the electromyographic activity of the *orbicularis oris* muscle to determine if the muscle activity had increased just after interruption of labial closure. This experiment did not provide any interpretable results, however, and no conclusions could be drawn concerning the role of peripheral feedback mechanisms in speech. In this study, we essentially replicated Ladefoged and Fromkin's experiment, with some methodological changes.

A special device was constructed to interrupt labial activity during normal speech. Two thin bars were placed between the subject's lips, the upper bar being controlled by a small DC motor. At certain points during the labial closure, the upper bar could be moved upward, exerting a moderate force, lasting about 100 msec, against the subject's lip. The electromyographic activity of the upper *orbicularis oris* muscle was obtained using wire electrodes inserted into the muscle. The electromyographic (EMG), audio, and a DC signal indicating the exact point of interruption were recorded simultaneously on an instrumentation recorder. The EMG signals for labial consonants typically consisted of an interference pattern, with about four to six motor units firing.

The EMG signals were analyzed by converting all positive spikes in the signal above the noise level into pulses of equal amplitude and duration. The resulting pulse train was fed into a simple RC integrator. The integrator has a fast rise time (3 msec) and a slow decay time (about 90 msec). Calibration determined that the smoothed output of the integrator was linear with respect to pulse frequency over a range of 25 to 1500 pps, well within the range of EMG spike frequencies encountered in our data. We used this method of analysis since we were interested in observing whether or not there was an increase in EMG spike frequency just after interruption. Such an increase could be caused by an increase in frequency of firing of the motor units already in use, or by recruitment of additional motor units.

One subject pronounced a series of VCV words containing bilabial stops with different combinations of the vowels /i/, /a/, and /u/. On random trials, the experimenter enabled the interrupter, effecting a counteraction of the closure gesture of the upper lip for the labial consonant. In addition to these VCV words, the subject also pronounced several long steady-state rounded vowels such as /u:/ and /o:/. The interrupter was triggered manually at random points during these vowels. As a further condition, the subject was also asked to make some slow closure gestures of the lips while not phonating, and the interrupter was again triggered manually at random points during these gestures.

The raw EMG waveform and the integrator output were carefully examined for all gestures and VCV syllables, normal and interrupted. (In this paper we are relying on a qualitative rather than quantitative analysis of the data.) In over 95% of the trials on which the syllables were interrupted, we found that the frequency of the spikes in the EMG did not increase significantly during the 100 msec period following interruption. Compare Figures 1 and 2. Figure 1 shows the uninterrupted articulation of the item /apa/, while Figure 2 shows the item /ipa/, with the point of interruption indicated by the arrows. These results are typical of those we observed throughout the experiment.

One phenomenon which was noted in approximately 60% of the interrupted trials was inhibition of the EMG activity, commencing (on the average) 5 msec after interruption, and lasting from 30 to 60 msec. Figure 3 shows the item /apa/ with the interruption point indicated by the arrows. The inhibition can be seen quite clearly.

Inhibition was very rare during the steady-state rounded vowels and the non-speech closure gestures, and compensatory increase in frequency of firing in the EMG did not occur at all in these cases.

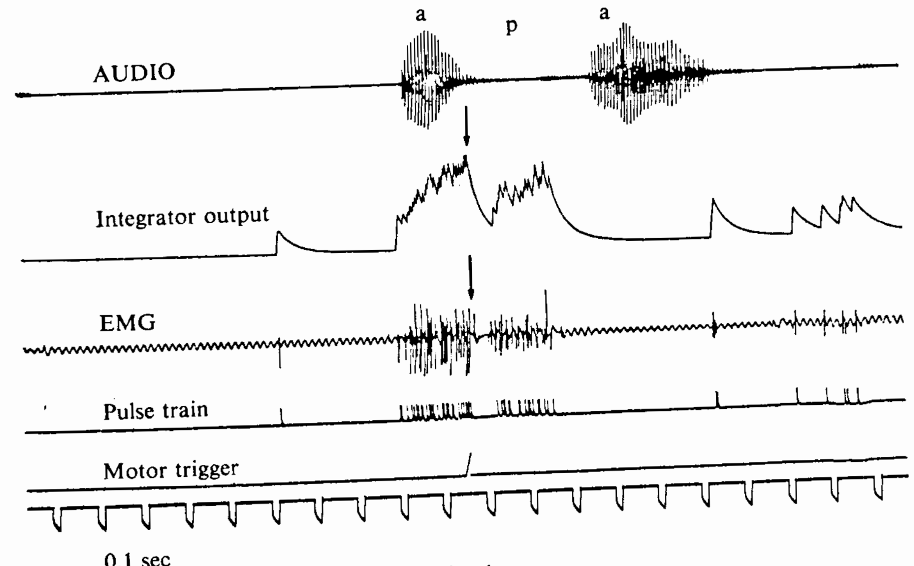

Fig. 1.

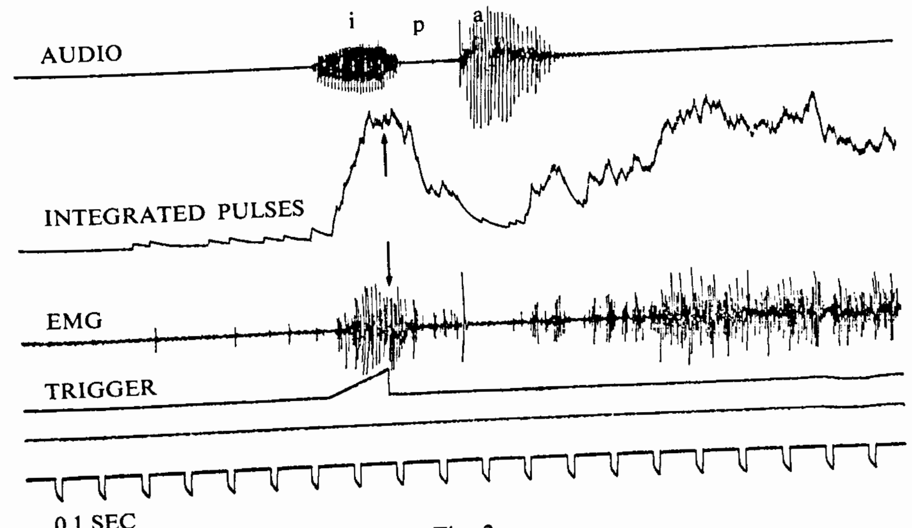

Fig. 2.

Since compensatory increase in muscular activity directly following interruption was very rarely observed in this experiment, the results can be interpreted as not offering any conclusive support for the claim that peripheral feedback mechanisms are used in speech production. Of course the results reported here are essentially preliminary, and further experiments along this line need to be carried out. Also, it is important to realize that negative results from an experiment of this sort do not demonstrate that the gamma efferent system as a whole is not employed in speech production. It does appear, however, that the neuromuscular mechanisms employed

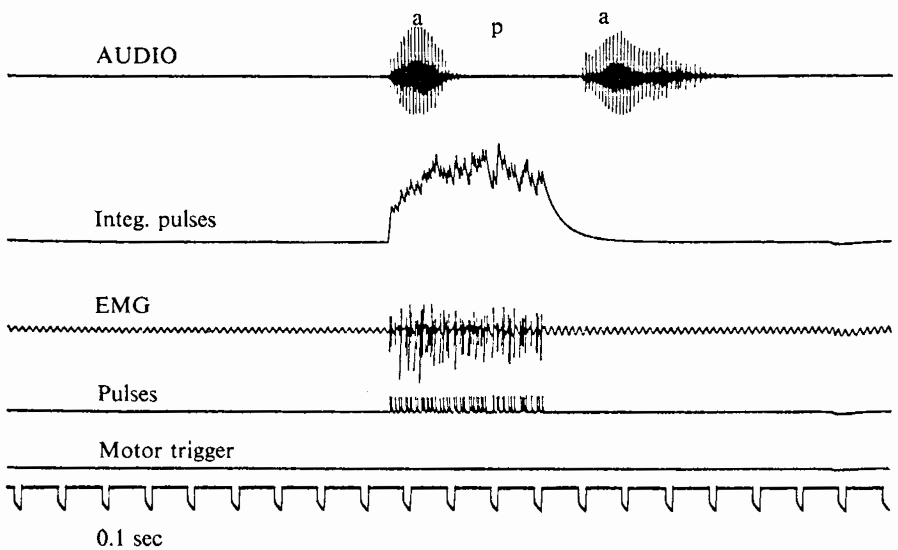

Fig. 3.

in speech are poorly adapted to respond quickly to extraneous interruptions, unlike the neuromuscular mechanisms of most of the skeletal muscles in the human body. Furthermore, it seems clear that the hypothesis that the articulators of speech are directed to achieve certain target positions (perhaps by activity of the gamma efferents), with precise alpha motor control being provided by a follow-up servo loop, should be viewed with some skepticism until it can be provided with a firmer experimental footing.

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DISCUSSION

MACNEILAGE (Austin, Tex.)
Do you have any examples where the trigger occurred during the period of increase in EMG rather than when EMG level was relatively constant? This period of increase might be more critical from the point of view of the utterance of feedback. Secondly, you remarked that the EMG interferences pattern was made up of from four to six single motor units. I am interested to know how you came to this conclusion.

SMITH
We did trigger interruptions during the period of increase. The typical result was that inhibition occurred more frequently (approximately 75% of the time), but that there was never a short-latency increase in *orbicularis oris* activity just after interruption at these times. As to the number of motor units involved, we are simply making a guess based on careful examination of the raw waveforms. We do not yet have a procedure for identifying individual motor units.

SUSSMAN (Austin, Tex.)
Until we know more about how single motor units summate to give the complex interference pattern, we must be careful on how we choose dependent variables, such as your spike frequency count, to show increased EMG activity. New single motor units may certainly be recruited without a concomitant increase in spike frequency. The summation of more single motor units may reflect a change in overall spatial configuration of the EMG and not necessarily more spikes.

SMITH
I agree completely that we need to gain more information on the make-up of interference EMG patterns in speech, and I am sure that the work that you and your colleagues are doing on this topic will prove interesting. In future research of this sort, we will look at analyses of the raw EMG waveform other than just spike frequency.

SOVIJÄRVI (Helsinki)
How do you think that you could in the future investigate the muscle activity of dental and velar sounds?

SMITH
We intend to work on a device for interrupting tongue activity in the future. We can already record EMG from the extrinsic muscles of the tongue.

FROMKIN (Los Angeles)
Are the reflexes which you mentioned similar to the data reported by Dr. L. Goldberg concerning the masseter muscle?

SMITH
As far as I know, these inhibitory periods are similar, however they are much longer. I believe Dr. Goldberg's work on the masseter muscle involved very short inhibitory periods.