

REGULATION OF INTENSITY AND PITCH IN CHEST VOICE

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ABSTRACT

The simultaneous control of fundamental frequency and intensity of phonation was investigated. Simultaneous measures were obtained of laryngeal muscles and the respiratory system. An inverse relation between intensity and activity of vocalis and cricothyroid was found at high pitch chest voice

INTRODUCTION

Studies of the simultaneous control of fundamental frequency (Fo) and intensity of phonation have dealt with either the intrinsic laryngeal muscles [1,2,3,4] or with the respiratory system [5,6]. In one study [7] subglottal pressure (Ps) was measured simultaneously with electromyographic (EMG) activity of intrinsic and extrinsic laryngeal muscles for singing. The purpose of the present study is to reexamine the simultaneous control of Fo and intensity in speech. To that end simultaneous recordings of speech, electroglottogram (EGG), Ps, lung volume, and EMG activity of cricothyroid (CT), vocalis (VOC) and sternohyoid (SH) were obtained while the subject sustained the vowels /e/ and /i/. These physiological signals were chosen because they are believed to be important in the control of both intensity and pitch. No open vowels were used because jaw opening may influence the intensity of the radiated sound and the EMG-activity of certain laryngeal muscles.

METHOD

Speech Material

The subject was a male native speaker of Dutch. He was instructed to breathe deeply and to phonate a vowel as long as possible at a constant Fo. This task was repeated five times for each of twelve different conditions: 2 vowels (/e/ and /i/), 3 intensities (soft, normal and loud) and 2 frequencies (low and high), making a total of 60 phonations. The intensity level (IL) of the soft utterances was approximately 7 dB below the normal IL, and the IL of the loud utterances was approximately 5 dB above the normal IL.

The audio signal was led to a pitch-extractor, and the subject could see the Fo level on an oscilloscope. Both target levels for Fo, viz. a low pitch level of 116 Hz and a high pitch level of 160 Hz, were indicated on the oscilloscope screen. In this way the subject could control his Fo.

Recording and Processing of Data

Subglottal air pressure was recorded using a Millar pressure transducer, which was inserted pernasally and fed through the glottis into the trachea. The pressure measurement was calibrated by recording the signal while the subject held lung pressures of up to 20 cm H₂O against a water-filled U-tube manometer [8]. The catheter, situated in the posterior commissure of the glottis, did not have a noticeable effect on phonation. The perimeter of the thorax and abdomen were measured with a Resptrace inductive plethysmograph. Lung volume was calculated from the weighted sum of these two signals. Special calibration manoeuvres yielded the two weighing factors. The EMG activity was recorded with hooked-wire electrodes [9]. All electrodes were inserted percutaneously. Correct electrode placement was confirmed by monitoring an oscilloscope during various functional manoeuvres [9].

The physiological signals, the audio signal, an octal code and a timing pulse were recorded on a one inch, 14-channel instrumentation recorder [10]. The processing of the data was done with the Haskins Laboratories EMG data processing system [11]. The voice signal was sampled and digitized at a 10 kHz rate, Ps and EGG at a 5 kHz rate, and EMG, chest and abdomen signals at a 200 Hz rate. The mean glottal flow (Ig) was obtained by taking the derivative of the lung volume. The vocal intensity was evaluated from the audio signal. The intensity levels were calculated relative to the lowest measured IL. The Fo was derived from the EGG in order to verify whether the Fo of the utterances remained roughly at the target levels. The EGG signal was also used to obtain the open quotient (OQ), which is defined as the time during which the glottis is open divided by the time of one vibratory cycle. The signals of six physiological quantities (viz. Ps, Ig, IL and EMG activity of VOC, CT and SH) were used for further processing.

Using the voice onset as a line-up point the six physiological signals of the five repetitions were averaged, resulting in an averaged signal for each quantity and each condition. Because of an artefact in the recordings of the EMG activity of the SH some of the tokens had to be discarded, but of each utterance at least four tokens remained for processing and averaging. For reasons that will be mentioned below, the mean value between 2 and 10 seconds after voice onset was calculated for these average signals. So the final measures are mean values for each of the six quantities in each of the twelve conditions.

RESULTS AND DISCUSSION

The duration of the sustained vowels varied between 10 and 20 seconds. While phonating the EMG-activity of VOC and CT, the Ps, Ig, IL and Fo were approximately constant. The EMG-activity of SH had a peak value immediately before the onset of phonation. Collier [12] and Hirose and Sawashima [13] also observed SH activity just before voice onset and assumed that the SH helps in preparing the larynx for the "speech mode." The peak value of SH activity depended on the frequency and intensity of the vowel that had to be produced. The largest peak values were recorded in the low frequency - high intensity condition, while the peak was almost absent in the high frequency - low intensity condition. In all utterances the EMG activity of the SH had levelled off to a more or less constant value 2 seconds after voice onset. Therefore for each measured physiological quantity the mean value was calculated from 2 to 10 seconds after voice onset, as mentioned above.

The mean values of the average signals are shown in the Figures 1 to 4. For each repetition the mean value between 2 and 10 seconds after voice onset is also given. The results are analyzed by making three different comparisons for the relevant physiological quantities.

1. /e/ vs. /i/

A comparison is made between the data of the vowels /e/ and /i/. Both are closed vowels and therefore the jaw opening was roughly the same. The major distinction between the two vowels is a difference in their formants, caused by a different vocal tract shape. This did not result in big dissimilarities between the recorded signals, but some differences did occur.

Ps. At equal intensity levels the Ps was always slightly higher when the vowel /i/ was produced.

CT. For the low Fo condition the activity of the CT was about the same for both vowels, but for high Fo the activity of the CT was less for the vowel /i/. This can be a compensation to keep Fo constant, because an increased Ps could raise the Fo

VOC and SH. These two muscles showed more activity for the vowel /i/ when phonating at low Fo, and approximately the same activity when phonating at high Fo.

2. Fo regulation

For the same phonetic condition and intensity, the signals recorded at low and high pitch voice are compared.

VOC and CT. From the Figures 1 to 4 it can be seen that the activity of VOC and CT was substantially higher in high-pitch chest voice than in low-pitch chest voice. This confirms previous findings that the VOC and the CT are the primary muscles in regulating Fo, especially in chest voice [3,4].

SH. The activity of the SH decreased with increasing frequency, a result also found by Ohala [14] and Collier [12]. The Figures 1 to 4 show that the decrease of SH activity was more obvious at the high IL.

Ps. Across different fundamental frequencies Ps was almost the same. This is contradictory to the general belief that Fo and Ps are positively related [12,15]. In this case the Fo

is not raised by increasing Ps, but probably by an appropriate adjustment of the activity of CT, VOC and SH.

3. intensity regulation

Since the vocal intensity is also a function of the acoustic impedance of the vocal tract, comparison of intensity is only done between two states in which the shape of the vocal tract is approximately the same, i.e. when the subject produced the same vowel.

Ps. Intensity was always positively related to Ps. This is consistent with the results obtained by Isshiki [5] and Baer [7].

Ig. The glottal flow was more or less constant for different intensities. The EGG recordings revealed that the OQ decreased with increasing intensity. Therefore, although Ps increased with increasing intensity, Ig could remain fairly constant [5]. In the chest register glottal flow is not dominant in controlling intensity; apparently, the form and spectral content of the flow pulses are more important.

SH. A positive relation between intensity and the EMG activity of the SH was found.

VOC and CT. At low chest voice no significant change in the EMG-activity of the VOC and CT as a function of intensity were found. Gay et al. [4] also found that muscle activity, of all five intrinsic laryngeal muscles, remained relatively steady across changes in vocal intensity.

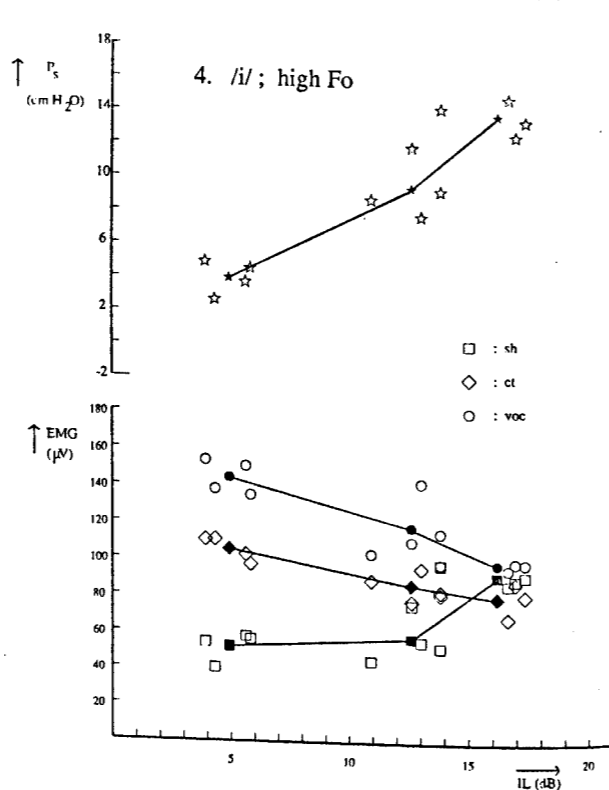
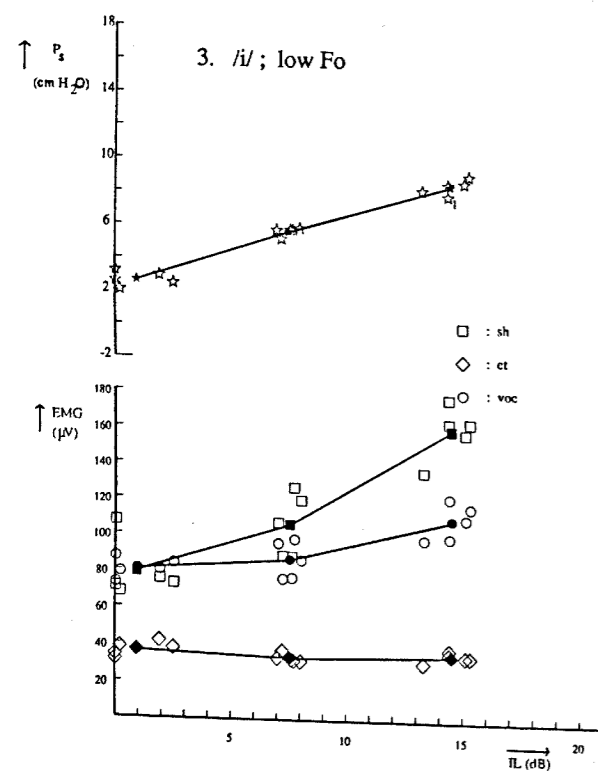
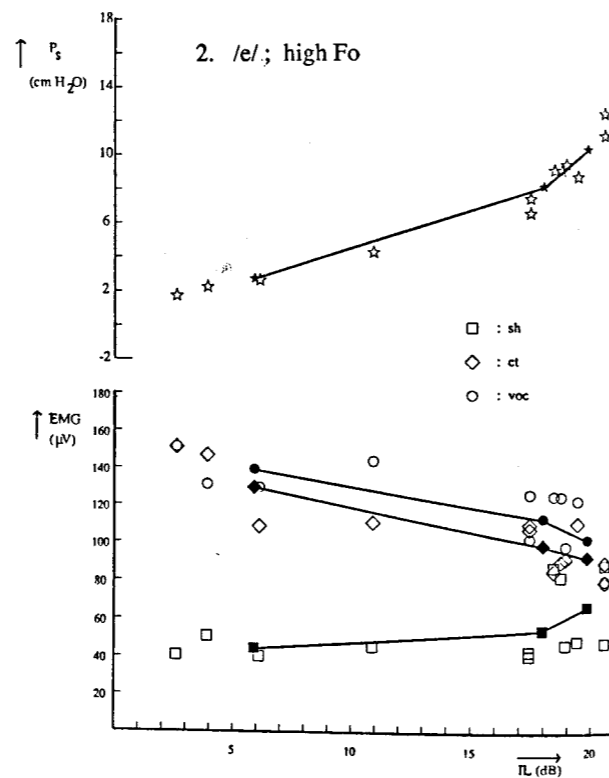
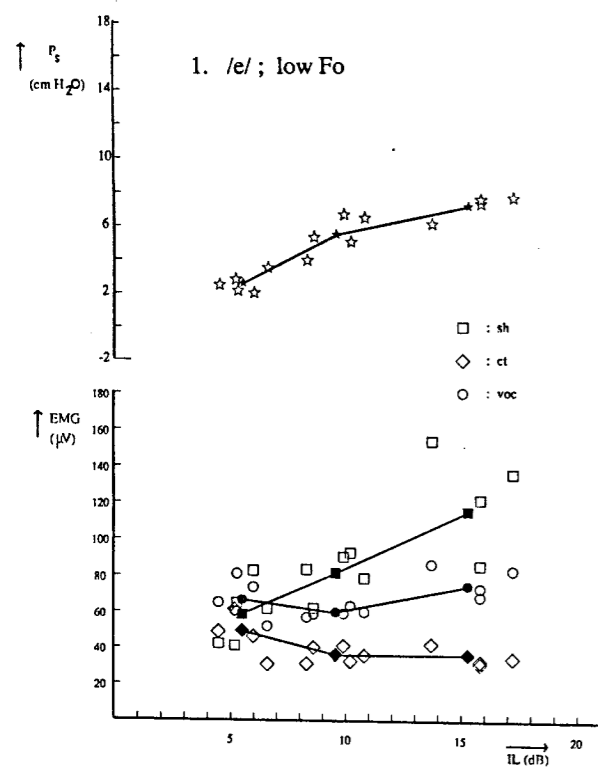
At high pitch chest voice a negative relation between intensity and the EMG activity of VOC and CT was found. The compensatory mechanism is necessary to keep Fo constant, because some of the factors that increase intensity also increase Fo.

CONCLUSIONS

First of all, it appears to be possible to maintain a constant subglottal pressure during a prolonged utterance, regardless of the decreasing volume of the lungs. Also Fo and IL can be kept constant without the need of apparent actions of CT, VOC or SH. Thus there seems to be no reason to assume that the often observed declination in speech is an involuntary effect of the decreasing lung supply.

The findings of this study are in agreement with those of previous studies: VOC and CT are the primary muscles in regulating Fo, IL is positively related to Ps, and Ig is more or less constant for different intensities in the chest register.

Müller found that an increase in vocal intensity without an associated rise in Fo had to be accompanied by a decrease in CT activity [16]. Rubin also speculated on a decrease in intensity of contraction of the CT with increasing loudness, if Fo is to remain constant [17]. Hirano actually measured that CT activity changes often varied inversely with the vocal intensity [1,2]. In the present study it was found that the increasing Ps can be compensated, not only by a decrease of CT activity, but also by a decrease of VOC activity. Further study is needed to explain why this compensation mechanism only occurred at high pitch voice.



Figures 1, 2, 3 and 4. In these figures the P_s (cm H₂O) and the EMG activity (μ V) of SH (\square), CT (\diamond) and VOC (O) are plotted as a function of IL (dB). The open symbols represent

the mean values of the tokens, and the closed symbols represent the mean values of the averaged signals (for further explanation see text).

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