SOME PHYSIOLOGIC CORRELATES OF VOICE FREQUENCY CHANGE

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The role of the intrinsic laryngeal muscles in posturing the vocal folds to effect frequency change has played an important part in every theory or speculation about voice production. Although there are still some exceptions, most phoneticians, and others interested in voice physiology, have accepted the basic principles of the myoelastic-aerodynamic theory of voice production as described by van den Berg.

The data to be presented here are measurements on 16 normal, young adult male volunteer subjects who sustained phonation at five frequency points in their modal to falsetto range. EMG data were obtained by inserting bipolar hooked wire electrodes to the thyroarytenoid, interarytenoid, posterior cricoarytenoid and cricothyroid muscles while simultaneously sampling sub-glottal pressure with an intratracheal catheter and measuring air flow through a pneumotachograph.

The simultaneously recorded electromyographic (EMG), aerodynamic and voice signals were tape-recorded as subjects sustained phonation at their 10, 30, 50, 70 and 90 % points of their total modal-to-falsetto frequency range. Phonation was produced at the 25 % and 75 % intensity levels at each frequency. The recorded signals during a two-second segment of each phonation task were digitized using an RMS integration at a sampling rate of 10 per second. Each subject performed a physiological calibration maneuver involving inspiring air quickly and singing an upward diatonic scale beginning in the lower portion of the modal register to the top of the modal register. The maximum voltage value obtained for each muscle during this calibration maneuver was assigned the number 100 and all further measures throughout the experimental procedure were converted to a percentage of that value. Thus, each muscle's activity for a given task could be compared to a high value generated during the physiologic calibration maneuver.

Figure 2 shows the group data for frequency change as an average of the two intensity levels at each frequency point. It can be seen that the posterior cricoarytenoid muscle, represented by bar No. 1, maintains a low level of activity across the total frequency range and apparently plays no role in altering voice frequency. The same lack of correspondence can be seen in the activity pattern of the interarytenoid muscle, which though operating at a relatively higher level to the physiologic cali-

PHYSIOLOGICAL MECHANISMS FOR VOICE FREQUENCY INCREASE

REGISTER		MYOELASTIC-AERODYNAMIC (van den Berg)	EMG-AERODYNAMIC STUDY (Shipp, McGlone)
MODAL (Chest)	Primary:	Vocalis activity	Thyroarytenoid (vocalis) and cricothyroid activity
	Secondary:	Increase airflow Interarytenoid activity	No trend in airflow No change in interarytenoid
FALSETTO	Primary:	Vocalis relaxation Cricothyroid activity	Thyroarytenoid (vocalis) activity Cricothyroid activity

Fig. 1. Physiological mechanisms for voice frequency increase.

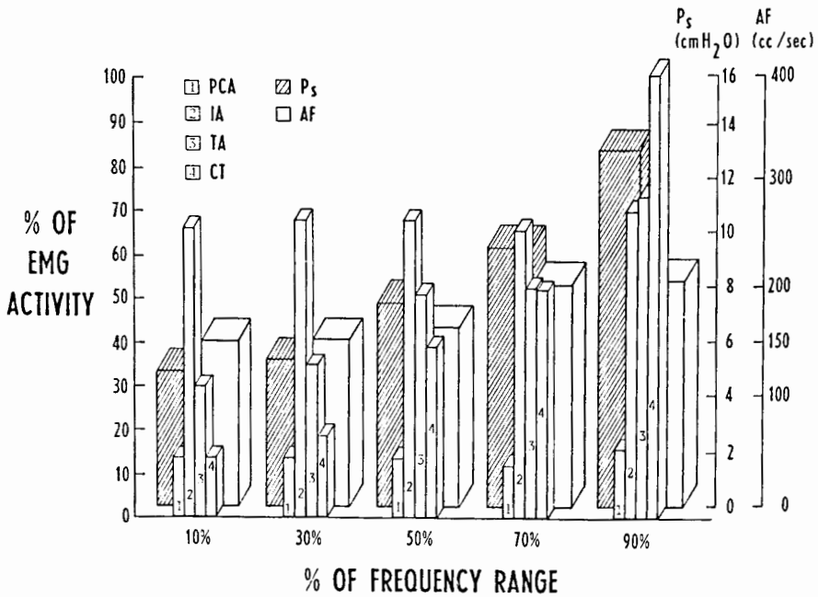

Fig. 2. Physiologic pattern of voice frequency change.

bration, is uncorrelated with voice frequency change. The histogram indicates that it is the last two muscles, the thyroarytenoid and cricothyroid muscles, that increase their activity levels systematically with increased voice frequency. It appears that in the lower part of the modal or chest register there is little cricothyroid activity to offset the relatively higher thyroarytenoid contraction. From the 10 % through the 90 % frequency point, both muscles increase in activity with the cricothyroid increasing at a faster rate. Sub-glottal pressure, as has been found in most other studies, also increases steadily with vocal frequency whereas air flow appears to be unrelated to frequency change.

All subjects were in their chest or modal register at the ten and thirty percent frequency levels and were always in falsetto register at their 70 % and 90 % frequency points. It was between the 30 and 50 or between the 50 and 70 % frequency points that individual subjects shifted vocal register.

These group data allow a comparison to be made between the muscle data obtained and the theoretical formulations for frequency change as proposed by van den Berg. In making these comparisons, we have made two assumptions that appear to us to be valid; one is that the chest register is comparable to what we have termed the modal register, and second, that when van den Berg speaks of the vocalis muscle, this term is interchangeable with thyroarytenoid muscle. Van den Berg has proposed that in the modal or chest register the primary means for increasing voice frequency is by increasing activity in the vocalis muscle whereas our data show a concomitant increase in both the thyroarytenoid and cricothyroid muscle. A secondary means he has proposed for increasing frequency is to increase the air flow and also to increase interarytenoid muscle activity. Our data show no relationship between air flow and frequency and, further, that interarytenoid muscle activity remains relatively invariant across the frequency range. In the falsetto register, van den Berg postulated a different mechanism for frequency change in which the vocalis muscle was completely relaxed while cricothyroid muscle activity provided the necessary tensions along the vocal ligament for altering frequency. Data from our study, rather than demonstrating major shifts in intermuscle contraction patterns for the two registers, show a gradual, continuous increase in cricothyroid-thyroarytenoid interaction to raise frequency regardless of the register in which phonation is produced.

It should be noted that the data presented represent the way in which most subjects alter voice frequency; variations on this pattern were evident in some individual subjects but no consistent pattern emerged other than the one presented here.

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DISCUSSION

HOLLIEN (Gainesville, Fla.)
It has been shown that contraction of the crico thyroid *m.* cannot increase the distance between the thyroid and arytenoids enough to account for the amount of vocal fold lengthening that has been empirically demonstrated. Obviously, some intrinsic *m.* posteriorly in the larynx must operate to pull the arytenoids post. up their articulation with the cricoid. Yet your data show no appropriate firings related to frequency increase. Could you comment on this?

SHIPP
I can only state that of the four intrinsic laryngeal muscles sampled, only the thyroarytenoid and cricothyroid muscles increase their activity levels coincident with fundamental frequency rise throughout the vocal range. If research shows that these forces are insufficient to explain the magnitude of the arytenoid cartilage movement, we may well look at extrinsic laryngeal muscle contribution to position the cartilages at the extremes of the normal frequency range.

MACNEILAGE (Austin, Tex.)
I would like to ask Dr. Hollien what is the evidence that the amount of cord lengthening observed in phonation cannot be accounted for by the action of the cricothyroid increasing the distance between the thyroid and the cricoid cartilages.

HOLLIEN
Basically, the physics of this mechanism would predict (as Negus and others have pointed out) that the very limited distance between the thyroid and the cricoid — and the probable action of the C-T muscle within this space — would permit only an extremely small (perhaps 2-3 mm) variation in the arytenoid-thyroid distance; and examination of anatomical specimens would appear to verify such a contention. Yet, it has been demonstrated that the vocal folds vary in length as much as 7-8 mm — or more if registers other than the modal are considered (Hollien, *Journal of Speech and Hearing Research* 3 [1960], Hollien and Moore, *Journal of Speech and Hearing Research* 3 [1960], Hollien, in *Proceedings of the International Association of Logopedics and Phoniatrics* [1962] and Hollien, Brown and Hollien, *Folia Phoniatrica* 23 [1971]).

Evidence of a more direct nature can be found in Damste, Hollien, Moore and Murry, *Folia Phoniatrica* 20 [1968]. Here, the mean crico thyroid distance varied only 2.0 mm as a function of frequency, yet the several measures of vocal fold length varied (means) from 3.5 - 5.3 mm. In other words, a C-T shift of about 10 % 'resulted' in a T-A shift of 30 % — a seemingly mechanical impossibility. Further, the difference in data here are confirmed by the four studies cited above as they report mean vocal fold lengthening to be as great as 50 %. Hence, the C-T relationship alone cannot account for the observed magnitudes of vocal fold lengthening and it is postulated that other mechanisms operate to assist in this laryngeal activity.

SMITH (Hamburg)
Fåborg Andersen showed a decrease of activity when the voice proceeded from chest to falsetto. In your recording at the posterior muscle you see a decrease of activity at just this transition. Then follows an augmentation, i.e., the posterior muscle fulfills its work in keeping the arytenoids in position, when the cricothyroid contracts.

SHIPP
The slight increase in mean PCA activity between the 50 % and 70 % frequency point is not statistically significant. We have not seen in upward or downward, continuous or discrete frequency change, any posterior cricoarytenoid activity that was at all related to frequency.