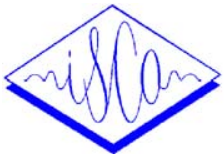


Speech Prosody 2004
Nara, Japan
March 23-26, 2004

Physiological Factors Causing Tonal Characteristics of Speech: from Global to Local Prosody

Kiyoshi Honda

ATR Human Information Science Laboratories, Kyoto, Japan
honda@atr.jp

Abstract

Voice fundamental frequency (F0) determines the tonal quality of vowels, and its rise and fall comprise part of prosody in speech. This seemingly simple linear function results from highly complex physiological factors and thus lacks definitive explanations of the causal mechanisms. This report reviews previous studies and recent discoveries regarding the causal factors of F0 patterns and discusses possible explanations for lexical accent, local F0 fluctuations, and phrasal declination. A special focus is placed on the following topics. (1) Historical arguments on the two actions of the cricothyroid joint, rotation and translation, for stretching the vocal folds: whether they both actually exist and how they contribute to F0 patterns is revisited with MRI observations. (2) Causal mechanisms of the so-called micro-prosody, i.e., F0 fluctuations due to voicing and vowel articulation: whether such local prosodic patterns are automatically derived from the relevant anatomical structure or derived from deliberate efforts of a speaker to enhance speech perception is discussed based on EMG data.

10.21437/SpeechProsody.2004-171

1. Introduction

Phonological models of prosody and physical bases of F0 changes suggest that the rules governing F0 patterns in speech are rather simple. However, the underlying mechanisms generating such simple characteristics are surprisingly complex. This is because numerous laryngeal and extra-laryngeal mechanisms are involved in controlling F0 during speech. Furthermore, the same mechanisms are used for other controls in voicing or articulation and they interact with each other in a complex manner. Many studies have been conducted to reveal physiological mechanisms of F0 control, while they are not detailed in current textbook chapters except for a few representative ones. This report summarizes the anatomical and physiological basis of laryngeal mechanisms for F0 control based on the author's experience through previous electromyographic (EMG) studies and recent magnetic resonance imaging (MRI) experiments.

2. Gross Anatomy of the Larynx

Figure 1 depicts the anatomy relevant to the mechanisms of F0 control. The laryngeal framework consists of the hyoid bone, the thyroid cartilage, and the cricoid cartilage. The thyroid cartilage articulates to the cricoid cartilage with bilateral slender arms of the inferior horns. The cricoid cartilage has a triangular shape in the lateral view with a thin arch anteriorly and a wide plate facing the cervical spine. In addition to direct and indirect muscular force, the cricoid cartilage receives force from the trachea and the cervical spine.

The laryngeal muscles are divided into two groups: the intrinsic laryngeal muscles that interconnect the laryngeal cartilages and the extrinsic laryngeal muscles that support the hyoid-larynx complex externally. The primary function of the intrinsic muscles, shown in Figure 1(a), is the adduction and abduction of the glottis by the adductor muscles (the thyroarytenoid, the lateral cricoarytenoid, and the transverse arytenoid) and the abductor muscle (the posterior cricoarytenoid). The second function of the intrinsic muscles is the actions of the cricothyroid joint for respiration, deglutition, and F0 control. The extrinsic laryngeal muscles are numerous according to their functional definition, as illustrated in Figure 1(b). The function of the extrinsic muscles is the movement of the larynx in vertical and horizontal directions. The actions of these muscles also cause various interactions between the larynx and the supra-laryngeal articulators.

3. Laryngeal Mechanisms of F0 Control

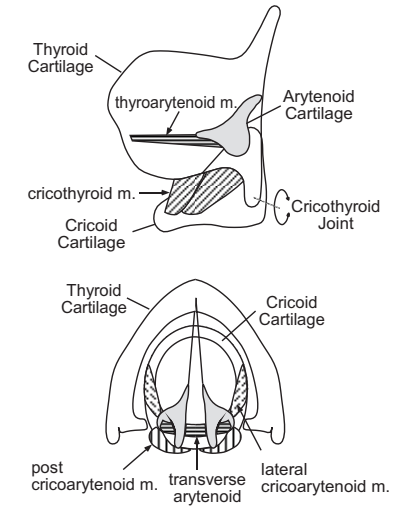
It is well known that the principal biomechanical factor in the regulation of F0 is the tension of the vocal folds, which is regulated mainly by the vocal fold lengthening through the action of the cricothyroid joint. In the case of the vibration of a string, longer vibrating materials produce a lower frequency of vibration, when the tension is constant. This analogy explains developmental changes in F0 from infants to adults, while it does not apply to the changes in vocal fold vibration within a speaker. The longitudinal stretch of the vocal folds causes a reduction of the mass per unit of length in addition to an increase in vocal fold tension. This effect overcomes the lengthening of vibrating material, resulting in a higher frequency of vibration.

There are other intra-laryngeal mechanisms of F0 control that are rather obscure in the literature. They include the regulation of effective length of vibration by medial compression forces and the regulation of vocal fold stiffness by contraction of the vocalis muscle. All of these tension control mechanisms directly interact with subglottal air pressure to determine the dimensions of sound, i.e. frequency, intensity, and quality.

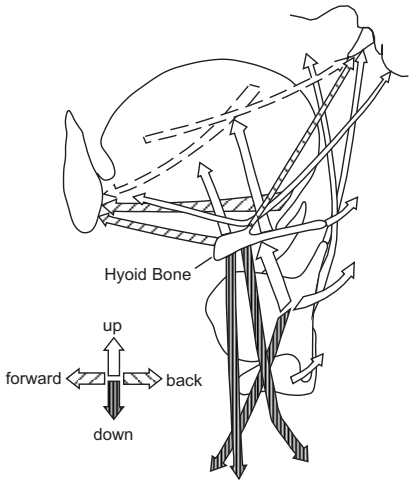
3.1. Rotation and translation of cricothyroid joint

The cricothyroid joint is the main framework for F0 control, and any force applied to this joint can influence F0. Many textbooks have stated that the joint has two degrees of freedom: rotation and translation, as shown in Figure 2. The rotation of the joint has been confirmed by many studies using x-ray [1]. With regard to translation, there are two opposing views: a negative view from a morphological investigation of the ligaments that firmly stabilize the joint [2], and a positive view from X-ray observations of a singer's larynx [3].

The author's group recently examined movements of the cricothyroid joint using high-resolution MRI with a custom laryngeal coil [4]. Figure 3 shows the data from a male speaker,



(a) Intrinsic Laryngeal Muscles



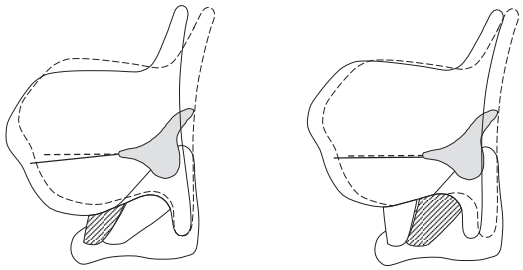
(b) Extrinsic Laryngeal Muscles

Figure 1: *Gross anatomy relevant to F0 control.*

indicating that the joint exhibits both rotation and translation. Between two phonation sessions with fundamental frequencies of 120 Hz and 180 Hz, joint rotation was 5 degrees and translation was 1.25 mm. Since joint translation takes place parallel to the longitudinal axis of the vocal folds, it can stretch the vocal folds more effectively than rotation at a cost of higher efforts against elasticity of the vocal folds. As discussed later, this preliminary observation suggests that translation may be a determinant factor for generating F0 patterns, while rotation may be involved in producing a spontaneous increase of vocal fold tension in voicing.

3.2. Cricothyroid muscle

The cricothyroid (CT) is the largest muscle among the intrinsic muscles, and its action directly acts on the cricothyroid joint. The CT has two anatomical subdivisions: the pars recta (straight part) and the pars obliqua (oblique part). It has been reported that these two subdivisions produce rotation and translation of



(a) Rotation (b) Translation

Figure 2: *Rotation and translation of the cricothyroid joint. The pars recta and oblique of the cricothyroid muscle have been assumed to cause rotation and translation of the joint, respectively.*

the cricothyroid joint, respectively.

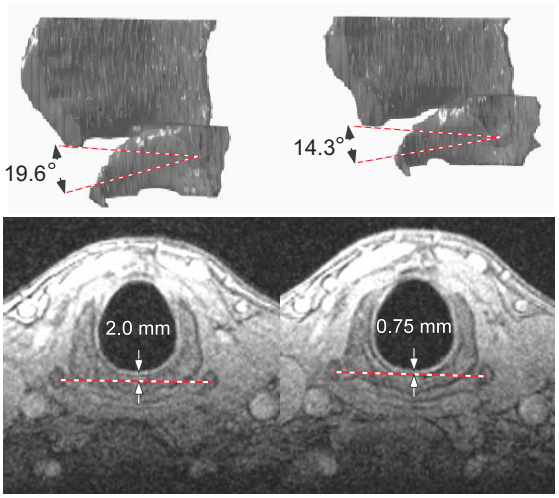
The EMG studies reporting CT activity in the rise of F0 are numerous [5], [6], [7], [8], [9]. Among them, Atkinson's correlation study appears most systematic [8]. This study examined EMG activities of the intrinsic and extrinsic laryngeal muscles for rises and falls of F0 in short utterances, with the results showing a higher correlation of the intrinsic muscles (CT in particular) vs. F0 and a lower correlation of the extrinsic muscles vs. F0. This work has been the basis of the general understanding among researchers: increase of CT activity determines F0 rises, while diminution of CT activity along with activity of F0 lowering muscles regulates F0 falls.

Whether the two parts of the CT function differently in F0 control is a question that has not so far been examined. The author's experience [10] [11], as shown in Figure 4, suggests that the pars recta shows a stationary level of activity for voicing regardless of F0 variation and that the pars obliqua demonstrates a linear relationship with F0, being more active for higher F0. This result may indicate that the pars recta acts with other intrinsic muscles to initiate voicing and that the pars obliqua determines slower changes in vocal fold tension.

3.3. Other intrinsic mechanisms

The rest of the intrinsic muscles function primarily for adduction (closing) and abduction (opening) of the glottis. There are three adductors (the thyroarytenoid, the lateral cricoarytenoid, and the transverse arytenoid) and one abductor (the posterior cricoarytenoid). It can be assumed that these muscles contribute to F0 control to a certain extent by their various effects on the parameters regulating F0.

The thyroarytenoid (TA) muscle is anatomically an antagonist to the CT because its contraction can produce joint rotation to contribute to vocal fold shortening. Functionally, however, its role in F0 control is not straightforward. Contraction of the TA does not only act on the joint, but also changes the property of the deep layer of the vocal folds. Many EMG observations of the TA have indicated that its activity increases with F0 [5], [6], [7], [8], [9], despite its potential F0 lowering effect. Fink [12] believes that the TA may develop considerable tension during active isometric contraction, depending on the initial length. Titze et al. [13] show evidence that F0 is raised by TA contraction in a low range of F0. In several studies that measured muscle response time, the TA is observed to be the fastest muscle [14], [15]. This suggests that the TA may produce a rapid F0 change in certain segmental adjustments.



(a) 120 Hz (b) 180 Hz

Figure 3: *Rotation (top) and translation (bottom) of the joint observed by MRI. The top figure shows the right half of the 3D image of the cartilages. Note that the cricoid cartilage rotates and translates as seen in the location of its arch. The bottom figure shows the transverse images of the larynx sectioned at the cricothyroid joint. The inferior horns of the thyroid cartilage indicated by the line translate forward in high F0.*

The lateral cricoarytenoid (LCA), the smallest muscle in the larynx, is believed to have some F0-raising effect through the medial compression force on the vocal processes. The length of the vibrating portion of the vocal folds can be changed by an increase in medial compression force even when the anatomical length of the vocal folds is the same [16], [10]. A tight compression of the left and right vocal processes causes a forward shift of the posterior end of vocal fold vibration, which results in shorter effective length of vibration. This is a control used for the length of vibration, independent of the other tension control mechanisms.

It has been speculated that the posterior cricoarytenoid (PCA), the only abductor muscle of the larynx, stretches the vocal folds by pulling the arytenoid cartilage backwards [17]. This effect needs to be maintained by other components because excessive force of PCA contraction can cause glottal opening. The adductor muscles, such as the transverse arytenoid muscle, appear to contribute to the action of the PCA by moving the arytenoid cartilages dorsocranially along the cricoarytenoid joints.

4. Extra-laryngeal Mechanisms of F0 Control

The muscles and the rigid structures supporting the laryngeal framework assist F0 control by their effect on the cricothyroid joint and intra-laryngeal tissue. This function of the extrinsic laryngeal muscles is described as "the external frame function" by Sonninen [3]. Several hypotheses have been proposed to account for F0 control mechanisms by the external forces.

The extrinsic laryngeal muscles are divided into two anatomical groups: the supra-hyoid muscles and the infra-hyoid muscles ("strap muscles", shown by the dark arrows in Figure 1(b)). Most of the muscles have attachments to the hyoid bone. The hyoid bone is a unique structure because it is gently connected by a muscular and ligamentous network, without any articulations, to the rigid structures. The position of the hyoid bone is determined by the conditions of the other rigid structures and the equilibrium of the forces in the entire system.

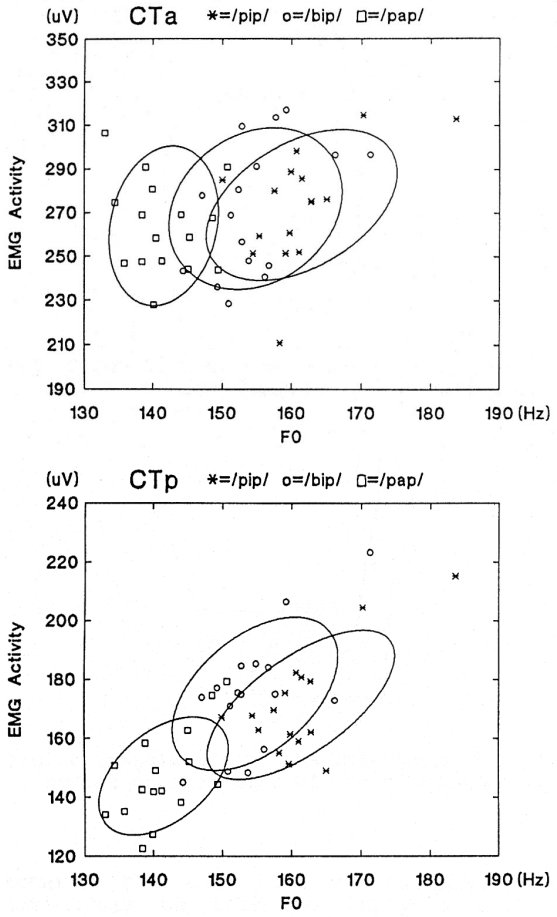


Figure 4: *EMG activity of the two parts of the cricothyroid muscle in voicing. The data were recorded from a male English speaker during repetitions of /CVC/ words. Anatomically, CTa (top) and CTp (bottom) correspond to the pars recta and obliqua, respectively. CTa shows almost constant activity in voicing regardless of F0, while CTp has a relatively high overall correlation with F0. Cited from [11].*

4.1. Extrinsic laryngeal muscles

Many researchers have investigated the possible mechanisms of F0 control exerted by the extrinsic laryngeal muscles. Among them, the sternohyoid (SH), one of the strap muscles, has been most frequently examined in relation to F0 lowering. The SH consistently shows various degrees of negative correlations (weak to moderate) with F0 during speech utterances in various languages [8], [18], [19]. In word accent, the activity of the CT is often followed by the activity of the SH [20], [21], [22]. In a sentence, the SH shows increased activities in the lower F0 regions, mostly near the end of a phrase or sentence.

The sternothyroid (ST) and the thyrohyoid (TH) muscles have shown contradicting results. The sternothyroid (ST) exhibits activity correlated with F0 changes in speech utterances

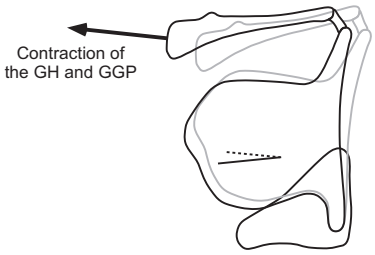


Figure 5: *Advancement of the hyoid bone and its effect on the cricothyroid joint. The hyoid bone is pulled forward by the geniohyoid muscle for F0 raising and by the genioglossus for high-vowel articulation.*

[23], [24], [8] and in singing [25] [26], while the muscle does show a synchronous increase in activity with the CT in vibrato [27]. The thyrohyoid (TH) muscle is located between the hyoid bone and the thyroid cartilage, and the contraction of this muscle causes approximation of these two rigid structures. The TH shows activity that follows both F0 pattern and jaw opening [23].

The supra-hyoid muscles have also been examined for their roles in F0 control. The geniohyoid (GH) muscle is known to show F0 related activity [28]. Figure 5 shows that the function of the GH is to pull the hyoid bone forward. The advancement of the hyoid bone applies a force to the thyroid cartilage to rotate along the cricothyroid joint in the direction that stretches the vocal folds [29]. The tongue muscles are also involved in hyoid bone movements. The posterior genioglossus (GGP) muscle, which runs parallel to the GH, also shows increased activity at F0 rises.

5. F0 Control Mechanisms for Accent, Intonation and Microprosody

This section attemps to explain how tonal units of prosody such as word accent and intonation are realized by the physiological mechanisms. Although answering the question is difficult because many arguments exist and real data are diverse, the following descriptions should help update phonological and physiological models of F0 generation.

5.1. Segmental control of F0

Segmental F0 changes, as in word accent, are primarily controlled by activities of the laryngeal muscles. The abovementioned mechanisms can roughly account for the segmental control of F0. Contraction of the cricothyroid CT muscle stretches the vocal folds, increasing their tension and decreasing their mass per length, and thus produces F0 rises. Relaxation of the CT resets vocal fold tension and results in F0 falls. In addition, some of the strap muscles, located in front of the neck, show activities in F0 falls, and the sternohyoid SH muscle is known to be an antagonist to the CT. Many EMG studies on word accent have shown reciprocal activity of CT and SH; in high-to-low accent, CT activity is followed by SH activity, and vice versa.

5.2. Global control of F0

In an utterance, F0 tends to fall gradually toward the end of a phrase or sentence. This declination of F0 has been attributed to a natural downdrift of the subglottal pressure during phonation rather than laryngeal muscle activities. This is because the subglottal pressure curve in a sentence shows a pattern resembling declining F0 curves, while the EMG pattern of the laryngeal muscles does not [30]. The CT tends to show a burst of activity only at an accent nucleus or emphasis, and becomes almost silent in low F0 portions in a sentence. The lack of muscular activity during declination has been discussed, leading to a simplified explanation: local F0 events are produced by laryngeal muscles and phrasal declination is caused by subglottal air pressure P_s. This hypothesis has a critical problem, however. The rate of F0 change with respect to P_s (r.f.p.) is roughly in the range of 2-6 Hz/cmH_2O [31], which is too small to explain the range of F0 in a sentence. According to Maeda [24], the magnitude of the baseline fall is between 20 Hz and 40 Hz. Assuming that the r.f.p value is about 5 Hz/cmH_2O and the P_s fall is 3 cmH_2O, the F0 drop due to the P_s fall is only 15 Hz. Therefore, the P_s fall alone cannot account for the fall of the baseline. Then, a question arises as to what mechanisms are involved in regulating vocal fold tension during global F0 changes.

Vertical laryngeal movement may be an active factor for phrasal declination because the larynx position tends to be lower in lower F0. EMG studies on the strap muscles have focused on the presumed relationship between vertical larynx movement and F0 change. For example, Kakita and Hiki [32], by measuring the vertical movements of the larynx and the EMG of the strap muscles, found high correlations among F0, vertical laryngeal movement, and activity of the strap muscles. They made an anatomical model based on their data, in which the TH and the ST control the vertical position of the larynx. The vertical dislocation of the cricothyroid joint is hypothesized in the model to account for the relationship between vertical laryngeal movement and vocal fold length.

Our recent studies have proposed a plausible explanation of the causal relationship between vertical laryngeal movements and F0 change [33], [34]. These studies used magnetic resonance imaging (MRI) to measure the movements of the hyoid-larynx complex during sustained phonation at various fundamental frequencies. In the data, the cricoid cartilage rotates with the vertical laryngeal movements as it slides along the anterior curvature (lordosis) of the cervical spine. The angle of the posterior plate of the cricoid cartilage remains parallel to the tangential line along the curvature of the cervical spine as the position of the cricoid cartilage changes. Figure 6 illustrates the chain of the mechanisms from vertical laryngeal movements to the change in vocal fold length. Considering the shape of the cervical lordosis, this mechanism seems to be more effective for F0 lowering than for F0 raising and supports F0 control in the low F0 range where CT activity is no longer available.

5.3. Local F0 fluctuation

In addition to accent and intonation, local fluctuations of F0 play a role in signaling segmental information. This is often called microprosody [35], which includes F0 variation due to consonants and intrinsic vowel F0.

The frequency at the onset of voicing after the release of stop closures varies according to the preceding consonant: voiced stops are followed by low F0, while voiceless stops are followed by high F0. This reflects the state of the vocal folds during the closure, and acts, in addition to VOT, as an important auditory cue to segmental identity [36], [37]. As for its causal mechanism, various accounts have been proposed. The most common is the indirect influence of larynx height. The position

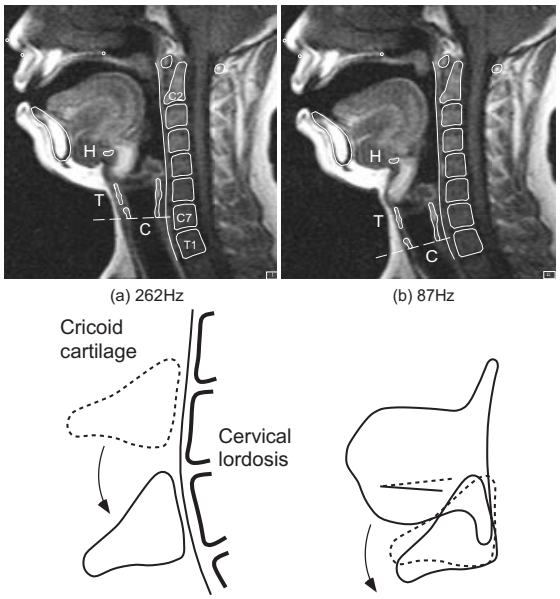


Figure 6: *Vertical larynx movement and its effect on F0. As the larynx is lowered, the cricoid cartilage moves downward keeping its posterior plate parallel to the curvature of the cervical spine, and thus rotates to shorten the vocal folds.*

of the larynx and hyoid bone are lower for voiced stops than for voiceless stops [38], and thus F0 is lower for voiced stops. This account seems reasonable because larynx lowering for a voiced stop allows glottal airflow during stop closure of the vocal tract [39]. Another account is derived from the activity of the CT muscle in voicing: the CT is used to apply a sudden stretch to the vocal folds to halt vocal fold vibration for voiceless stops [40], and thus F0 at voicing is higher after voiceless stops. Although this account proposes a reasonable mechanism for F0 variation due to voicing, a different account is also possible from the viewpoint of perceptual requirement: speakers intend to produce higher F0 after voiceless stops to realize the auditory cue of the sounds. Since the pars recta is recorded in many EMG studies, this part of the CT may contribute to the local F0 pattern at voicing (but see Figure 4).

Intrinsic vowel F0, a language-universal phenomenon for high vowels to have higher F0, is known as a vocal manifestation of tongue-larynx interaction. This phenomenon has led speech researchers to search for its causal mechanisms [41]. A contemporary view of the underlying mechanism is the biomechanical coupling between the larynx and the supra-laryngeal articulators. The classical "tongue pull" theory [42] has been refined by a few physiological studies [43] [29]. The tongue deformation for high-vowel articulation produces forward movement of the hyoid bone, which applies a force to the cricothyroid joint to stretch the vocal folds. Contrary to this biomechanical explanation, the intrinsic vowel F0 may be a deliberate production to enhance vowel identification [44]. As evidence, EMG studies have revealed the contribution of CT activity to intrinsic vowel F0 [45] [46]. Although the vowel-dependent F0 variation in speech is not large enough to interact with prosody, it should provide an additional feature to complete vowel-specific tonal characteristics and contributes to the expansion of auditory vowel space by changing the F0-F1 relationship.

6. Summary

This report described contemporary issues of physiological mechanisms of F0 control. Although experimental evidence so far is not complete and needs to be accumulated by further studies, the physiological mechanisms deriving the units of speech prosody may be summarized as follows.

- Local fluctuations: The CT (pars recta?) contributes to F0 patterns of voicing, and the tongue-larynx interaction causes vowel-dependent F0 variation.
- Lexical accent: The CT (pars obliqua?), along with other intrinsic and strap muscles, determines F0 patterns of lexical accent.
- Phrasal pattern: The extrinsic laryngeal muscles in coordination with the respiratory muscles regulate F0 patterns in phrasal declination.

7. Acknowledgement

This research was supported as part of "Research on Human Communication" with funding from the Telecommunications Advancement Organization of Japan.

8. References

[1] Fink, B.R.; Demarest, R.J., 1978. *Laryngeal Biomechanics*. Cambridge, Mass: Harvard University Press.

[2] Mayet, V.A.; Mündnich, K., 1958. Beitrag zur Anatomie und zur Funktion des M. Cricothyroideus und der Cricothyreoidgelenke. *Acta Anat.*, 33, 273-288.

[3] Sonninen, A., 1968. The external frame function in the control of pitch in the human voice. *Ann. New York Acad. Sci.*, 68-90.

[4] Takano, S.; Honda, K.; Masaki, S.; Shimada, Y.; Fujimoto, I., 2003. High-resolution imaging of vocal gesture using a laryngeal MRI coil and a synchronized imaging method with external triggering. In *Proc. Spring Meeting, Acoust. Soc. Jpn.*, 291-292. (in Japanese)

[5] Arnold, G.E., 1961. Physiology and pathology of the cricothyroid muscle. *Laryngoscope*, 71, 687-753.

[6] Hirano, M.; Ohala, J.; Vennard, W., 1969. The function of laryngeal muscles in regulating fundamental frequency and intensity of phonation. *J. Speech and Hearing Research*, 12, 616-628.

[7] Gay, T.; Strome, M.; Hirose, H.; Sawashima, M., 1972. Electromyography of the intrinsic laryngeal muscles. *Ann. Otol. Rhinol. Laryngol.*, 81, 401-409.

[8] Atkinson, J.E., 1978. Correlation analysis of the physiological features controlling fundamental frequency. *J. Acoust. Soc. Am.*, 63, 211-222.

[9] Shipp, T.; Doherty, E.T.; Morrissey, P., 1979. Predicting vocal frequency from selected physiological measures. *J. Acoust. Soc. Am.*, 66, 678-684.

[10] Honda, K., 1983. Variability analysis of laryngeal muscle activities. In *Vocal Fold Physiology: Biomechanics, Acoustics, and Phonatory Control*, I. Titze; R. Scherer (eds.). Denver: The Denver Center for the Performing Arts, 286-297.

[11] Honda, K., 1988. Various laryngeal mechanisms in controlling the voice fundamental frequency. *J. Acoust. Soc. Am., 84*, Suppl. 1, S82.

[12] Fink, B.R., 1962. Tensor mechanisms in the human larynx. *Acta Otol. Rhinol. Laryngol.*, 71, 591-600.

[13] Titze, I.R.; Luschei, E.S.; Hirano, M., 1989. Role of the thyroarytenoid muscle in regulation of fundamental frequency. *J. Voice*, 3, 213-224.

[14] Sawashima, M., 1974. Laryngeal research in experimental phonetics. In *Current Trends in Linguistics*, A.T. Sebeok (ed.). The Hague: Mouton, 2303-2348.

[15] Larson, C.R.; Kempster, G.B.; Kistler, M.K., 1987. Changes in voice fundamental frequency following discharge of single motor units in cricothyoid and thyroarytenoid muscles. *J. Speech and Hearing Research*, 30, 553-558.

[16] Broad, D.J., 1973. Phonation. In *Normal Aspects of Speech, Hearing, and Language*, F.D. Minifie; T.J. Nixon; F. Williams (eds.). Englewood Cliffs N.J.: Prentice-Hall, 127-167.

[17] Hollien, H., 1983. In search of vocal frequency control mechanisms. In *Vocal Fold Physiology*, D.M. Bless; J.H. Abbs (eds.). San Diego: College-Hill Press, 361-367.

[18] Erickson, D., 1993. Laryngeal muscle activity in connection with Thai Tones. *Ann. Bull. RILP*, 27, 135-149.

[19] Simada, Z.; Hirose, H., 1970. The function of the laryngeal muscles in respect to the word accent distinction. *Ann. Bull. RILP*, 4, 27-40.

[20] Erickson, D.; Baer, T.; Harris, K.S., 1983. The role of the strap muscles in pitch lowering. In *Vocal Fold Physiology*, D.M. Bless; J.H. Abbs (eds.). San Diego: College-Hill Press, 281-285.

[21] Simada, Z.; Niimi, S.; Hirose, H., 1991. On the timing of the sternohyoid muscle activity associated with accent in the Kinki dialect. *Ann. Bull. RILP*, 25, 39-45.

[22] Sugito, M., 2003. Timing relationships between prosodic and segmental control in Osaka Japanese word accent. *Phonetica*, 60, 1-16.

[23] Sawashima, M.; Kakita, Y.; Hiki, S., 1973. Activity of the extrinsic laryngeal muscles in relation to Japanese word accent. *Ann. Bull. RILP*, 7, 19-25.

[24] Maeda, S., 1976. *A characterization of American English Intonation*. Ph.D. Thesis, Cambridge, Mass: MIT.

[25] Faaborg-Anderson, K.; Sonninen, A., 1960. The function of the extrinsic laryngeal muscles at different pitch. *Acta Otolaryngol.*, 51, 89-93.

[26] Roubeau, B.; Chevrie-Muller, C.; Guily, J.L.S., 1997. Electromyographic activity of strap and cricothyroid muscles in pitch change. *Acta Otolaryngol. (Stockh)*, 117, 459-464.

[27] Niimi, S.; Horiguchi, S.; Kobayashi, N., 1991. F0 raising role of the sternothyroid muscle: an electromyographic study of two tenors. In *Vocal Fold Physiology*, J. Gauffin; B. Hammerberg (eds.). San Diego: Singular Publishing, 183-188.

[28] Erickson, D.; Liberman, M.; Niimi, S., 1977. The geniohyoid and the role of the strap muscles. *Haskins Status Report on Speech Research*, SR-49, 103-110.

[29] Honda, K., 1983. Relationship between pitch control and vowel articulation. In *Vocal Fold Physiology*, D.M. Bless; J.H. Abbs (eds.). San Diego: College-Hill Press, 286-297.

[30] Collier, R., 1975. Physiological correlates of Intonation patterns. *J. Acoust. Soc. Am.*, 58, 249-255.

[31] Titze, I.R., 1989. On the relation between subglottal pressure and fundamental frequency in phonation. *J. Acoust. Soc. Am.*, 85, 901- 906.

[32] Kakita, Y.; Hiki. S., 1974. A study of laryngeal control for voice pitch based on anatomical model. In *Proc. Speech Communication Seminar*. Stockholm, SCS-74, 45-54.

[33] Hirai, H.; Honda, K.; Fujimoto, I.; Shimada, Y., 1994. Analysis of magnetic resonance images on the physiological mechanisms of fundamental frequency control. *J. Acoust. Soc. Jpn.*, 50, 296-304. (in Japanese)

[34] Honda, K.; Hirai, H.; Masaki, S.; Shimada, Y., 1999. Role of vartical larynx movement and cervical lordosis in F0 control. *Language and Speech*, 42, 401-411.

[35] Kohler, K.J., 1990. Macro and micro F0 in the synthesis of intonation. In *Papers in Laboratory Phonology I*, J. Kingston; M.E. Beckman (eds.). Cambridge: Cambridge Univ. Press, 115-138.

[36] House, A.S.; Fairbanks, G., 1953. The influence of consonant environment upon the secondary acoustical characteristics of vowels. *J. Acoust. Soc. Am.*, 35, 84-92.

[37] Haggard, M.P.; Ambler, S.; Callow, M., 1970. Pitch as a voicing cue. *J. Acoust. Soc. Am.*, 47, 613-617.

[38] Kent, R.D.; Moll, K.L., 1969. Vocal-tract characteristics of the stop cognates. *J. Acoust. Soc. Am.*, 46, 1549-1555.

[39] Stevens, K.N. *Acoustic Phonetics*. Cambridge, Mass: MIT Press.

[40] Löfqvist, A.; Baer, T.; McGarr, N.S.; Story, R.S., 1989. The cricothyroid muscle in voicing control. *J. Acoust. Soc. Am.*, 85, 1314-1321.

[41] Ohala, J.J.; Eukel, B.W., 1978. Explaining the intrinsic pitch of vowels. *Report of the Phonology Laboratory*. Berkeley: University of California, 2, 118-125.

[42] Lehiste, I., 1970. *Suprasegmentals*. Cambridge, Mass: MIT Press.

[43] Rossi, M.; Autesserre, D., 1981. Movements of the hyoid and the larynx and the intrinsic frequency of vowels. *J. Phonetics*, 9, 233-249.

[44] Diehl, R.L.; Kluender, K.R., 1989. On the object of speech perception. *Ecological Psychol.*, 1, 121-144.

[45] Vilkman, E.; Aaltonen, O.; Raimo, I.; Okasanen, H., 1989. Articulatory hyoid-laryngeal changes vs. cricothyroid muscle activity in the control of intrinsic F0 of vowels. *J. Phonetics*, 17, 193-203.

[46] Honda, K.; Fujimura, O., 1991. Intrinsic vowel F0 and phrase-final F0 lowering: phonological vs. biological explanations. In *Vocal Fold Physiology*, J. Gauffin; B. Hammerberg (eds.). San Diego: Singular Publishing Group, 149-158.