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Production of Tone[1]

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Real languages are not minimal redundancy codes invented by scholars fascinated by the powers of algebra, but social institutions serving fundamental needs of living people in a real world. [In trying to understand] how human beings communicate by means of language, it is impossible for us to discount physical considerations, [i.e.] the facts of physics and physiology [Halle 1954: 79–80].[2]

1. INTRODUCTION

This chapter is offered as a brief review of those aspects of tone production that may be relevant to an understanding of tonal phenomena. It is reasonable that widely attested sound patterns, tonal or not, can be explained to a great extent, if not totally, by reference to the only thing that is common to different linguistic communities, though they be geographically, chronologically, or geneologically remote from each other, namely, the physical mechanisms used in the transmission and reception of speech: the human articulatory and auditory mechanisms, including associated neurological structures. This approach has proven useful in phonology for almost a century (Passy 1890, Rousselot 1891, Haden 1938, Grammont 1965, Lindblom 1972, 1975, Ohala 1971, 1972a, 1974a, 1974b, 1975a, 1975b, 1975c, 1976, Ohala and Lorentz 1977).

It is impossible in the space of one brief chapter to give even the beginnings of a comprehensive review of the production of tone, and, in any case, there are other extensive reviews of fundamental frequency production which serve that purpose: Arnold 1961, Damste 1968, Luchsinger and Arnold 1965, Zemlin

[1] This research was supported in part by the National Science Foundation, and the Committee on Research and the Computing Center of the University of California, Berkeley.

[2] Parts of this quotation have been rearranged from the original without, I think, distorting its sense.

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1968, Ohala 1970, Sawashima 1970, Broad 1973, Netsell 1969, Dixit 1975, as well as collections such as Bouhuys 1968 and the extensive bibliography in Di Cristo 1975. I will therefore attempt the following more modest tasks: first, to review very briefly enough of laryngeal anatomy and physiology so that the later parts of this chapter and the other chapters making reference to the production of tone will be understandable even to those with little previous exposure to experimental phonetics; second, to review the more recent and more controversial issues in tone production; third, to provide an introduction to the literature in this area; and fourth, to give the reader some of the facts which have emerged from the various experimental studies on tone production and also to convey a "feel" for how research is done in this area, for it is only with an understanding of the basic research techniques that the reader will be able to evaluate on his own the evidence and claims.

1.1 Terminology

I use the terms "pitch" and "fundamental frequency" (F_0) interchangeably. Both will be taken to mean the rate of vibration of the vocal cords during voice production. When quantified, the units are hertz (Hz). Some cases of tonal contrasts which linguists have described apparently include the distinctive use of other phonetic parameters besides pitch, for example, duration, voice quality, manner of tone offset, and vowel quality. However, I will be concerned only with pitch itself, and not with these other parameters. Subglottal air pressure, P_s, will be mentioned frequently as a determinant of pitch. More properly this should be the difference between subglottal and oral air pressure, i.e., the transglottal pressure drop; however, in most cases of interest (nonobstruents) this would have approximately the same value as subglottal pressure itself, since oral pressure would be near zero. The units for P_s will be centimeters of water (cm H_2O), since the standard way of measuring pressures in the vocal tract is with a U-tube water manometer (see Figure 4). Glottal air flow will also be mentioned as a determinant of pitch. In most cases glottal air flow is directly proportional to P_s and has a similar effect on pitch. There are instances, however, where the two are not necessarily related to each other (namely, when glottal area varies or when the larynx itself moves up or down). In such complex cases the effect of one parameter on pitch may be greater than the other (see Ishizaka and Flanagan 1972).

2. REVIEW OF LARYNGEAL ANATOMY AND PHYSIOLOGY

Functionally, the larynx is a valve and a sound producer. As a valve it regulates the flow of air into and out of the lungs and keeps food and drink out of the lungs. The two functions are accomplished by a relatively complex

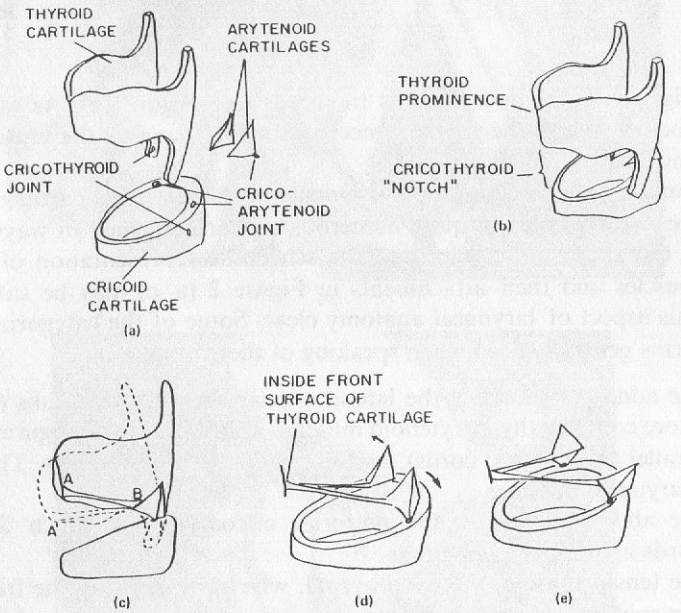


Figure 1. (a) An exploded schematic representation of laryngeal cartilages and their movements. (b) Cartilages as they are normally joined. (c) Manner of rotation of thyroid and cricoid cartilages which cause vocal cords, AB, to increase in length, $A'B$. (d) Adducted position of the vocal cords when arytenoid cartilages are tilted inward. (e) Abducted position of the vocal cords when arytenoid cartilages are tilted outwards.

arrangement of cartilages, muscles, and other tissues. The hard structure of the larynx consists of four principal cartilages: the thyroid, the cricoid, and a pair of arytenoid cartilages, as shown schematically in Figure 1 (a—exploded view—and b). The thyroid and cricoid are connected as shown and pivot about a transverse axis. The two arytenoid cartilages are connected to the cricoid cartilage via a ligamentous "hinge" and sit atop its rear rim. Each can rotate on the rim of the cricoid in such a way as to bring their front projections towards or away from the midline (see Figure 1d and e).

The two vocal "cords" or, more appropriately, the vocal folds, are basically ligaments which stretch between the inner lower front surface of the thyroid cartilage and the front faces of the separate arytenoid cartilages (see Figure 1 d and e). It is the rotation of the arytenoid cartilages which enables the vocal cords to be brought together towards the midline for voicing or breath-holding (adducted) or to be separated from each other (abducted). With the cords adducted and the arytenoid cartilages fixed with respect to the cricoid cartilage, rotation of the thyroid and cricoid cartilages with respect to each other causes the vocal cords to change their length: Bringing these two cartilages' front edges together lengthens and stretches the cords; increasing the separation of the

front edges shortens and slackens the cords (see Figure 1c). As will be discussed below, this is the primary mechanism for changing the tension of the cords and thus the pitch of voice.

The muscles which cause the movements of the laryngeal cartilages and of the larynx as a whole are quite numerous and are attached in ways that are difficult to explain fully in prose. The schematic representation of some of these muscles and their attachments in Figure 2 (a–c) may be sufficient to make this aspect of laryngeal anatomy clear. Some of the categorizations or cover terms generally used when speaking of these muscles are:

1. The adductor muscles: the lateral cricoarytenoid, the vocalis (that part of the more complex thyroarytenoid muscle—not shown in the figures—which runs parallel to the vocal cords), and the interarytenoid muscles. These close off the laryngeal opening.
2. The abductor muscle: the posterior cricoarytenoid, which draws the vocal cords apart.
3. The tensor muscle: the cricothyroid, which, by drawing the front edges of the thyroid and cricoid cartilages closer together, lengthens and tenses the vocal cords.
4. The strap muscles: the sternohyoid, sternothyroid, and thyrohyoid (also the omohyoid, not shown in the figures), which are in front of the larynx and which attach directly or indirectly to the larynx and to portions of the skeletal frame enabling them to move the larynx as a whole up or down or, within limits, forward or backward. (Such movements are most noticeable during yawning and swallowing.) Some believe that the strap muscles may also be able to affect movements of the thyroid cartilage with respect to the cricoid (see below).
5. The constrictor and other pharyngeal muscles which connect to the larynx from the rear via the pharyngeal walls and which can, like the strap

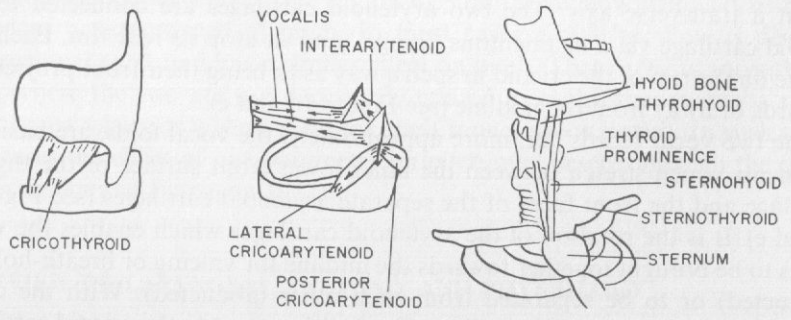
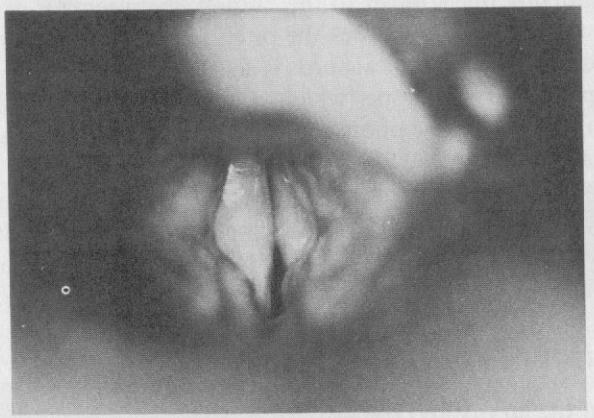


Figure 2. Schematic representation of the laryngeal muscles and the movements they accomplish: (a) cricothyroid muscle; (b) lateral cricoarytenoid, vocalis, posterior cricoarytenoid, and interarytenoid muscles; (c) strap muscles: sternothyroid, thyrohyoid, and sternohyoid muscles.


(a)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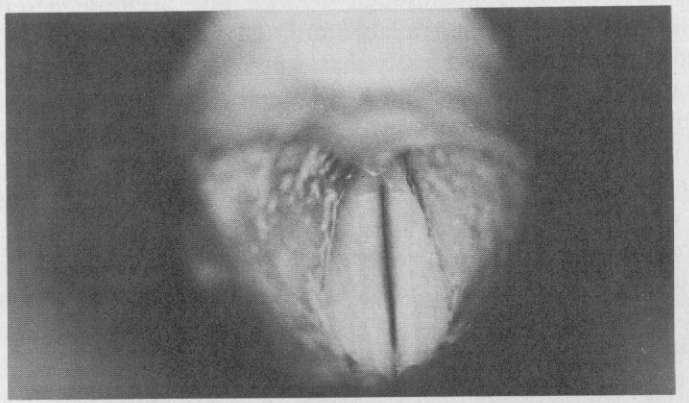
(c)

Figure 3. Photographs (by the author and Ralph Vanderslice) of the larynx taken with a laryngoscope. In these views the front of the larynx is at the top. (a) The vocal cords (the white bands) in the abducted position for normal breathing (cf. Figure 1e). The "knobby" structures at the base of the vocal cords are the fleshy covering of the arytenoid cartilages. (b) Low-pitched phonation. (c) High-pitched phonation.

muscles, affect the position of the larynx as a whole and also the closure or constrictions of the laryngeal passage giving out onto the pharynx.

The first three groups are also referred to collectively as the intrinsic laryngeal muscles, and the remaining ones as the extrinsic laryngeal muscles, although occasionally some writers include the cricothyroid in the latter category. Like most structures in the body, all of these muscles, except the interarytenoid, are symmetrically paired, one on the right and one on the left, even though it is common to speak of the cricothyroid "muscle" rather than "muscles."

It is fairly easy to detect on oneself the rotation of the thyroid and cricoid cartilages—or at least one result of it, the diminishing of the cricothyroid space—by applying one's finger to the "notch" between the cartilages (easily felt through the skin) as one raises the pitch of voice. The stretching and the abduction/adduction of the vocal cords is also relatively easy to view in oneself with the help of a laryngoscope (which is just a small mirror placed at a 45° angle on a thin handle). The photographs in Figure 3 (a–c) were taken using a homemade laryngoscope.

In a thin person's neck it is possible to see the outline of some of the strap muscles, particularly the sternohyoid—especially if the individual, lying supine, attempts to lift up his head. There is as yet no easy way to visualize the other muscles, even with a laryngoscope.

3. BRIEF HISTORY OF RESEARCH ON PITCH REGULATION

The role of the larynx and trachea in sound production has been recognized for millenia (Allen 1953), although detailed and accurate descriptions of laryngeal anatomy were not available until the work of Vesalius (1543). And it was not until even more recently that experimental evidence was offered to show that the vocal cords produce the sound of voicing by means of their vibration, which in turn interrupts (i.e., "chops up") the air stream passing through the glottis into the vocal tract proper, and that it was lengthening and consequent tensing of the cords which changed the rate of vibration, i.e., the pitch of voice (Ferrein 1741, Willis 1833).

The great German physiologist, Johannes Müller, more than anyone else can be credited with putting our knowledge of laryngeal physiology on a firm empirical basis, in that not only did he verify (and quantify) many earlier claims about the action of the larynx in sound production, but he also uncovered many new details as well (Mueller 1851). Many of his studies were done using freshly excised human larynges, sometimes with most of the rest of the vocal tract attached (see Figure 4). By adding known weights to strings attached (via pulleys) to various parts of the larynx, he simulated the action of certain laryngeal muscles, and by blowing into a tube connected to the trachea (the exact pressure of which was measured by a U-tube water manometer), he simulated the pulmonic air flow. He showed that although the primary mechanism for raising pitch was the tensing of the vocal cords via the cricothyroid

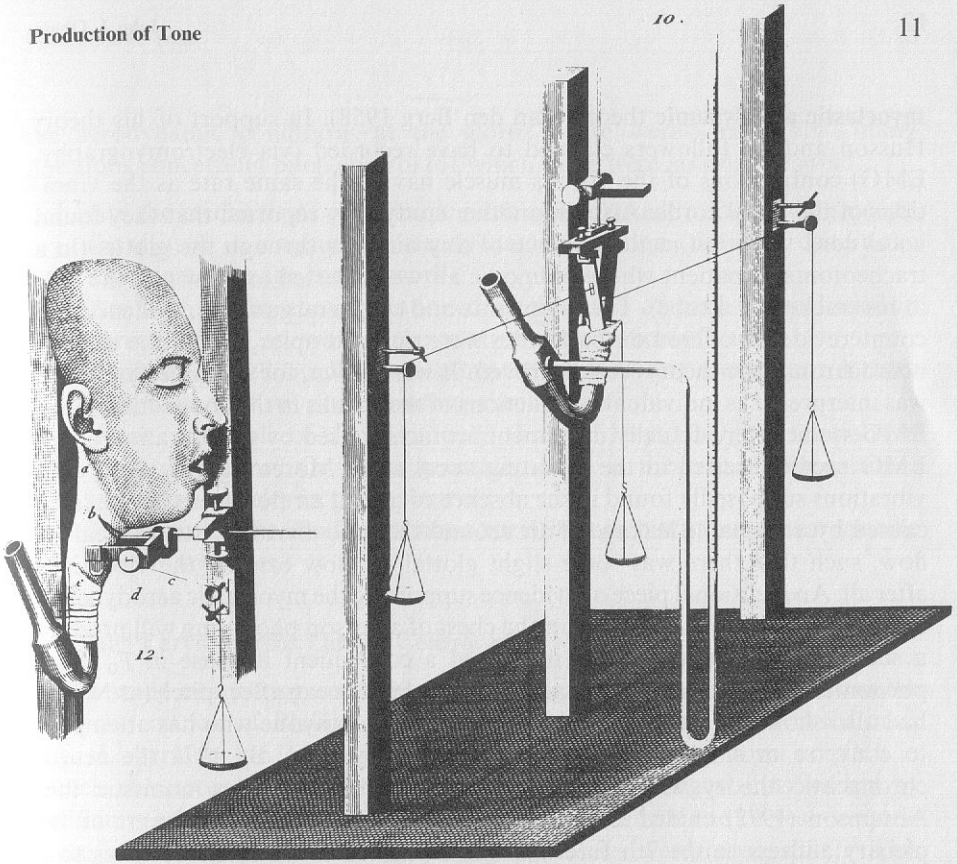

Figure 4. The laboratory set-up Müller used in his studies with excised larynges to quantify the effect of muscular pull and air flow on various parameters of the voice, including pitch (from Mueller 1851).

muscle, it was also possible to change pitch by varying P_s; in this case pitch varied at the rate of about 4.5 Hz/cm H_2O.

Although Müller was not the first to use this technique, he was perhaps the first to use it carefully and to get reliable quantitative data from it. Experimentation with excised larynges has continued to yield important data which increase our understanding of laryngeal mechanisms used in speech (e.g., van den Berg and Tan 1959, Furukawa 1967, Anthony 1968, Baer 1975).

Of course, excised larynges cannot duplicate every aspect of the living larynx, and this leaves open the possibility that studies based on them exclusively may have missed some very fundamental aspects of normal laryngeal behavior. At least Raoul Husson thought so. In his 1950 dissertation he challenged the prevailing views on the mechanisms of vocal cord vibration and pitch regulation by claiming that each vibration of the cords was accomplished by a separate contraction of the adductor muscles, i.e., for a F_0 of 400 Hz these muscles would supposedly contract at the rate of 400 twitches/sec. This was called the **neurochronachtic** theory; the traditional view was called the

myoelastic aerodynamic theory (van den Berg 1958). In support of his theory Husson and his followers claimed to have recorded (via electromyography, EMG) contractions of the vocalis muscle having the same rate as the vibrations of the vocal cords. Also, in another study they reported that they found vocal cord vibration in the absence of any air flow through the glottis (in a tracheotomized patient whose pulmonic air was diverted out through the hole in his trachea by a tube). The arguments and counterarguments, evidence and counterevidence offered on both sides were quite complex, but in the end the neurochronachtic theory was disproved. It was shown, for example, that what was interpreted as individual contractions of the vocalis in the above-mentioned EMG studies were actually movement artifacts caused by the vibration of the EMG needle inserted in the vibrating vocal cord. Moreover, the vocal cord vibrations supposedly found in the absence of glottal air flow were shown to be caused by a probable leakage of air around the tube diverting the tracheal air flow, such that there was some slight glottal air flow causing the vibrations after all. An additional piece of evidence supporting the myoelastic aerodynamic theory was the fact that a push on the chest of a person phonating will produce a sudden involuntary increase in P_s and a consequent increase in F_0. This phenomenon, which shows that aerodynamic factors can affect pitch (as Müller had also shown on excised larynges), is familiar to anyone who has attempted to converse or sing while driving over a bumpy road. By 1971 the neurochronachtic theory was judged long dead and so much a nonissue that Abramson (1972) chided Lafon for bothering to devote a good part of his plenary address at the 7th International Congress of Phonetic Sciences to a refutation of Husson's claims.

Insofar as a model, whether implemented mechanically or by a computer program, represents a manifestation of a theory, it can be said that all important aspects of the myoelastic aerodynamic theory of vocal cord vibration have been successfully duplicated and, in that sense, verified by a number of very successful vocal cord models (Wegel 1930, Ishizaka and Flanagan 1972, Flanagan, Ishizaka, and Shipley 1975).

In retrospect, Husson's theory seems highly unlikely, given what was known at the time about laryngeal physiology and about muscular contractions in general. Nevertheless some of the most imaginative and technologically advanced studies on laryngeal physiology were made as a result of the dispute between the two theories (see, e.g., Fabre 1957, Isshiki 1959, van den Berg and Tan 1959, Faaborg-Andersen 1957, 1965). This is consistent with the view that two (or more) clearly defined competing theories can be very beneficial to a scientific field (Feyerabend 1968, Derwing 1973).

4. RECENT ISSUES IN LARYNGEAL PHYSIOLOGY

Although the earlier research indicated that the pitch of voice could be varied both by adjustments in the tension of the vocal cords and by changes in the aerodynamic conditions at the glottis, there were—inevitably—many questions concerning pitch control for which there were no clear answers:

1. Are the mechanisms of dynamic pitch control used in speech the same as those used in singing and steady-state phonation (the only conditions investigated in the earlier work)?
2. Are both of the above-mentioned mechanisms of pitch control used in speech, and, if so, what is the relative contribution of each?
3. Is pitch lowered simply by relaxing the pitch-raising muscles, or is there a separate (active) pitch-lowering mechanism?
4. What is the role of the commonly observed larynx height variations in pitch variations? Or, more generally, what is the role of the extrinsic laryngeal muscles in pitch control?
5. What causes the small systematic variations in pitch found near specific speech segments?

4.1 The "Larynx versus Lungs" Controversy

One study which triggered a detailed investigation of these questions was the MIT dissertation of Philip Lieberman (1967b) and other articles based on it (e.g., Lieberman 1967a, 1968). Briefly, he claimed that variation of pitch in speech was accomplished not by the action of the laryngeal muscles but by variations in P_s (except in the case of the pitch rise at the end of yes–no questions, where he allowed that the laryngeal muscles were responsible).[3] This claim was based on recordings from three speakers of the voice signal and the P_s during various types of utterances. His data were similar to the top two parameters shown in Figure 7 (the third parameter in the figure will be considered later). Such records (and many that were published previously, e.g., Smith 1944, Ladefoged 1963) often show a certain synchronization between the momentary pitch rise on the stressed syllable (cf. *bombed* in the second sentence) and a momentary increase in the P_s function. Likewise, the terminal falling pitch such as that in Sentence 1 in the figure was observed to coincide with falling P_s. Lieberman had no records of the activity of the laryngeal muscles and assumed that in general they maintained a constant level of tension in the vocal cords, except, of course, in the case of the terminal rises on yes–no questions, such as that in the third sentence in the figure, where there is

[3] Lieberman claimed that this was the "archetypal" manner of pitch control in speech. He also allowed that "alternate articulatory maneuvers" could be used to vary pitch, for example, use of the laryngeal muscles. So formulated, his hypothesis is nonempirical, since there is no conceivable data that could falsify it (see also Ohala and Ladefoged 1970, Ohala 1970). For example, Lieberman considered his hypothesis that P_s causes F_0 as verified even though he himself admitted that a majority of his subjects, two out of three, gave evidence of using these so-called "alternate articulatory maneuvers."

no correlation between pitch and P_s and thus where P_s cannot be invoked as a cause of the pitch change. He also obtained simultaneous values of F_0 and P_s at various points in his data where he assumed that the laryngeal muscles maintained a constant level of activity and thus arrived at "calibrations" of the effect of P_s on F_0 that ranged from 16 to 22 Hz/cm H_2O.[4]

Lieberman intended his "P_s causes F_0" hypothesis to apply to pitch changes on stressed syllables and sentence-final elements. He did not apply it to tone or to pitch accent (such as occur in Chinese and Japanese) and in fact, called for further research to decide how pitch was varied in such cases. He did suggest, however, that the shape of "allotones" that appear on stressed syllables or at the end of sentences in Chinese are due to the same factors which shape pitch variation in nontonal languages, i.e., P_s variations. There have also been some attempts by others to explain certain aspects of tonal sound patterns by reference to Lieberman's hypothesis, e.g., terracing in African tone languages (Painter 1971, 1974).

Although no one had yet made direct recordings of the activity of the laryngeal muscles during speech when Lieberman's dissertation appeared, the dominant opinion among phoneticians and speech scientists was not in agreement with his claims. Rather, they thought that the pitch of voice during speech was in all cases, yes–no questions or not, controlled by the laryngeal muscles and that the effect of P_s on pitch (certainly real enough, as Müller had shown a century ago) was too small to account for the major part of the observed pitch variations (Sweet 1877, Scripture 1902, Stetson 1928, Wegel 1930, Pressman and Kelemen 1955, Ladefoged 1963, Öhman and Lindqvist 1966, Zemlin 1968, Proctor 1968). There were a number of reasons for this belief:

1. Larynx height follows pitch variations.

It was commonly observed that the larynx moves up and down in the neck during the pitch changes in speech and singing, and it was thus assumed that in some way the larynx contributed to these pitch changes (Herries 1773, Scripture 1902, Critchley and Kubik 1925).

2. Different consequences of laryngeal versus pulmonic paralysis.

Clinical observations suggested that paralysis or other loss of the use of some of the laryngeal muscles commonly resulted in defective use of pitch in speech (Critchley and Kubik 1925, Sokolowsky 1943, Sonninen 1956, Arnold

[4] As I have pointed out elsewhere (Ohala 1977a), the values Lieberman gives for $\Delta F_0/\Delta P_s$ are questionable even in the absence of data on laryngeal activity. My own analysis of his data showed that if he had indeed followed the measurement procedures he specified in his book, the values would be more like 28–33 Hz/cm H_2O. These are absurdly high values, of course, and clearly reveal that the assumption behind the measurements, namely, that the laryngeal muscles maintained a constant level of activity, is false. The skeptical reader may, if he cares to, check this for himself, since all the relevant raw data are published in Lieberman 1967b.

1961, Luchsinger and Arnold 1965). On the other hand, respiratory paralysis apparently did not result in any defect in the use of pitch in speech (Peterson 1958).

3. Electromyographic studies of laryngeal function.

Direct EMG recordings during steady state phonation showed the laryngeal muscles, especially the cricothyroid, to be very highly correlated with the pitch level produced (Katsuki 1950, Faaborg-Andersen 1957, 1965, Sawashima, Sato, Funasaka, and Totsuka 1958, Arnold 1961, Kimura 1961). Hirano, Koike, and von Leden (1967) also found the sternohyoid active for low pitch and extremely high pitch. It was therefore assumed that these results could be extrapolated to speech conditions. It should be mentioned here that as a technique for gathering data on muscle activity, EMG is rather simple in concept but extremely difficult to use in practice, especially on the muscles of the larynx. It consists of inserting small electrodes (thin needles or wires) into the muscle of interest and then amplifying and recording the small spikelike voltage variations they pick up from the contracting muscle. The chief problem is making sure the electrodes end up in the intended muscle, which is particularly difficult in the case of the larynx since so many muscles are crowded into such a small space. For example, we may guess that the "cricothyroid" muscle which Zenker (1964) found to be active during low pitch and during jaw opening—an unusual pattern for this muscle—was actually the sternohyoid muscle for which these patterns are more expected. The sternohyoid lies over the cricothyroid (see also Faaborg-Andersen 1964). Nevertheless, used carefully it can give us reliable information on the activity of specific muscles, as is evident from the similar results obtained in independent studies (see below).

4. Calibrations of the extent of P_s influence on F_0.

Calibrations of the effect of P_s on F_0, where some care was taken to ensure that the vocal cords maintained a constant level of activity, yielded values of $\Delta F_0/\Delta P_s$ (i.e., rate of change of pitch with respect to the change in P_s) of 2–5 Hz/cm H_2O in the pitch range used in speech. The calibrations of $\Delta F_0/\Delta P_s$ were obtained by the push-on-the-chest technique described above. Figure 5 (data from Öhman and Lindqvist 1966) shows the kind of raw data obtained from such investigations. The close temporal coincidence of the F_0 and P_s curves is a reasonable assurance that the F_0 change was caused in a purely automatic way by the P_s increase and not by the speaker reacting to the push on the chest. Figure 6 shows how the data can be analyzed, by plotting the F_0 variation as a function of the P_s variation. The slope of such plots, then, equals $\Delta F_0/\Delta P_s$. In Figure 6 the slope is 2.9 Hz/cm H_2O. Having obtained such calibrations Ladefoged (1963) and Öhman and Lindqvist (1966) applied them to their records of P_s and F_0 during connected speech in order to factor out those pitch variations that could be attributed to P_s. By far the major part

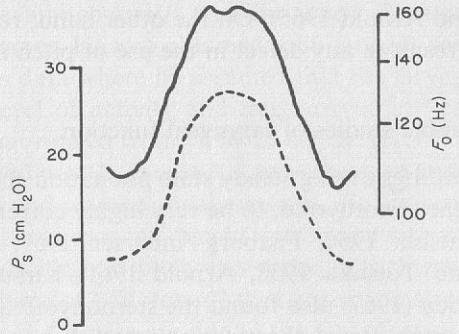

Figure 5. Synchronized variations in pitch, F_0 (solid line), and subglottal air pressure, P_s (broken line) during a sudden push on the speaker's chest (redrawn from data given by Öhman and Lindqvist 1966).

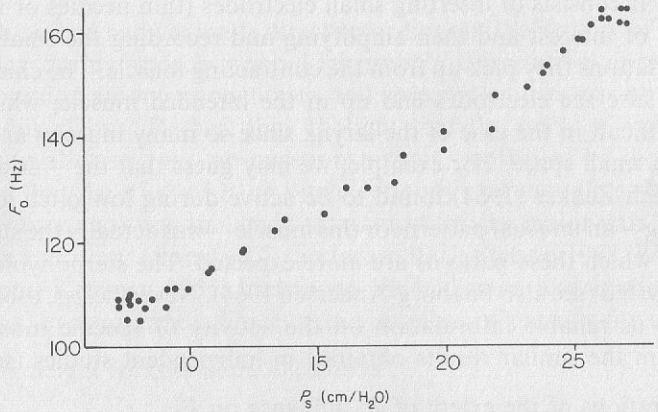

Figure 6. Variations in fundamental frequency, F_0, plotted as a function of variations in subglottal air pressure, P_s, during a sudden push on the speaker's chest (data from Figure 5). From such data it can be deduced that P_s can influence F_0 at the rate of about 2.9 Hz/cm H_2O (the slope of the line passing through the data points as determined by the least squares method).

of the recorded pitch variations had to be attributed to factors other than P_s, i.e., to laryngeal factors. For example, for a stress-related pitch rise of 50–100 Hz—quite common in normal speech—no more than 5–10 Hz of the rise could be due to P_s variations. Öhman and Lindqvist also commented that "the f_0 changes during stressed syllables do not correlate well with the stress-induced [P_s] changes either in phase or in amplitude [p. 4]."

Lieberman did not review most of this evidence and made no attempt to reconcile his claims with the fraction he did review except to dismiss Öhman and Lindqvist's findings as applying to singing not to speech.

Although Lieberman (1968) declared his hypotheses "verified," the overwhelming bulk of research done on the topic since 1967 has not supported his

innovative claims and has instead supported the traditional view of the primacy of the larynx in pitch control. New evidence was obtained, as well as refinements and elaborations of the existing types of data. These were:

1. Larynx height correlates with pitch.

Vanderslice (1967) recorded vertical laryngeal movements and P_s during connected speech and showed the former to be better correlated with pitch than was P_s. Quantitative data showing good correlation between larynx height and pitch were obtained by many investigators (Ohala 1972b, Ewan and Krones 1974, Ewan 1976, Shipp 1975a, 1975b, Shipp and Izdebski 1975, Kakita and Hiki 1976). There is undoubtedly some individual variation in this, however, since Lindqvist, Sawashima, and Hirose (1973) and Gandour and Maddieson (1976), each studying a single subject, failed to find substantial correlation between these two parameters. Looking at all the relevant studies, however, there are still more cases where the correlation was found than where it was not. It is reasonable to conclude, then, that the larynx, at least in part by its vertical movements, actively participates in the control of pitch in speech whether it is found on pitch falls or pitch rises, on nonterminal or sentence-terminal elements.

2. Further calibrations of $\Delta F_0/\Delta P_s$.

Additional calibration of the effect of P_s on pitch were made using refined techniques, including monitoring the activity of the laryngeal muscles during the induced pressure changes (Netsell 1969, Ohala and Ladefoged 1970, Ohala 1970, Lieberman, Knudson, and Mead 1969, Hixon, Mead, and Klatt 1971, Baer 1976, Okamura, Gould, and Tanabe 1976).[5] Table 1 summarizes these and earlier such determinations of $\Delta F_0/\Delta P_s$. The studies employing excised larynges generally yield higher values than do those involving living subjects, Lieberman's (1967b) study excepted, of course. This is probably due to the excised larynges lacking normal muscle tonus (van den Berg and Tan 1959). Lieberman *et al.*'s (1969) upper limit of 10 Hz/cm H_2O is substantially lower than that originally claimed by Lieberman. Curiously, Hixon *et al.* (1971), using the same technique and one of the same subjects involved in the study of Lieberman *et al.*, could not replicate those earlier findings, i.e., did not find values as high as 10 Hz/cm H_2O. Although there may be methodological problems with all of these calibration techniques, there is scant evidence from them to support Lieberman's claim that P_s could be responsible for all or even most of the observed pitch variations in speech. It follows, then, that other factors, presumably the laryngeal muscles, must be causing the pitch changes.

[5] Okamura *et al.* (1976) do not report their results in a way which allows them to be converted to Hz/cm H_2O. However, their data seem much like that of Hixon *et al.* (1971).

TABLE 1

Values of $\Delta F_o/\Delta P_s$ (in Hz/cm H_2O) from Various Studies

Source	Normal voice	High pitch and falsetto
Mueller (1851)[a]	4.3 ~ 4.5	10 ~ 16
Isshiki (1959)	3.3	
van den Berg and Tan (1959)[a]	5 ~ 13	17 ~ 20
Ladefoged (1963)	5	
Öhman and Lindqvist (1966)	2.9	
Furukawa (1967)[a]	8[b]	
Anthony (1968)[a]	6 ~ 8	
Lieberman, Knudson, and Mead (1969)	3 ~ 10	9 ~ 18
Netsell (1969)	3.5 ~ 9.4	
Ohala and Ladefoged (1970)	2 ~ 4	7 ~ 10
Ohala (1970)	2 ~ 8	
Hixon, Mead, and Klatt (1971)	2 ~ 4	
Baer (1976)	3 ~ 5	
Lieberman (1967)	16 ~ 22	

[a] Used excised larynges.
[b] Average slope of one F_o versus P_s plot.

3. P_s not independent of laryngeal activity.

It was pointed out (by Isshiki 1969, Ohala 1970, 1975a, 1976, van Katwijk 1971, 1974) that P_s variations can be caused in part by changes in glottal and oral impedance, i.e., by anything which would cause reduced air flow, e.g., obstruent closures, reduced mean glottal area, such as occurs during pitch increases (Sonesson 1960, Ishizaka and Flanagan 1972), or increased percentage of closed time in the glottal area function, such as occurs during voice intensity increases (Sonesson 1960, Flanagan 1965). This point has long been well known among speech scientists (Stetson 1928, Peterson 1957, Strenger 1958, Isshiki 1964, Yanagihara and von Leden 1966, Öhman and Lindqvist 1966, Ladefoged 1968, Zemlin 1968, Benguerel 1970, Netsell 1973).

A related fact which no doubt reflects the increase of glottal impedance with increasing pitch is that the minimum pressure drop necessary to maintain voicing is greater for high pitch than for low pitch (Mueller 1851, Isshiki 1959).

Thus, not only is it improbable that P_s variations could cause much of the observed pitch change in speech, it is probable that to some extent the P_s variations are themselves caused by laryngeal activity.

For this reason it is not surprising that good correlations are occasionally found between speech intensity (which is unquestionably causally correlated with P_s) and pitch (Lieberman 1967a, Zee and Hombert 1976), but, again, rather than the correlation implying that one of the parameters causes variation in the other, it reflects the fact that both are heavily influenced by a third parameter, laryngeal activity.

4. Electromyography of laryngeal muscles during speech.

Electromyographic studies of the activity of the laryngeal muscles during speech in a variety of languages (tone, "pitch accent," nontone) showed that they were centrally involved in pitch changes, no matter what type of pitch change was involved. (Ohala and Hirano 1967, Öhman, Mårtensson, Leanderson, and Persson 1967, Fromkin and Ohala 1968, Hirano, Ohala, and Vennard 1969, Hirano and Ohala 1969, Netsell 1969, Lieberman, Sawashima, Harris, and Gay 1970, Ohala 1970, 1972b, Garding, Fujimura, and Hirose 1970, Simada and Hirose 1970, 1971, Erickson and Abramson 1972, Sawashima, Kakita, and Hiki 1973, Atkinson 1973, Fischer-Jørgensen 1974, Collier 1975, Maeda 1975, Shipp 1975a, Erickson and Atkinson 1976, Atkinson and Erickson 1976, Erickson, Liberman, and Niimi 1976). In agreement with the earlier findings for singing, the cricothyroid muscle was found to be the primary force for raising pitch although the lateral cricoarytenoid and vocalis also assisted. In addition, in many studies the sternohyoid and sternothyroid were shown to be active during pitch lowering. (The involvement of the strap muscles in pitch change is not surprising, of course, given the evidence cited above that larynx height varies with pitch.) Typical EMG records are shown in Figures 7, 8, and 9. The pitch and P_s parameters in Figure 7 were discussed earlier; the third parameter shown is the cricothyroid activity. It can be seen that in the

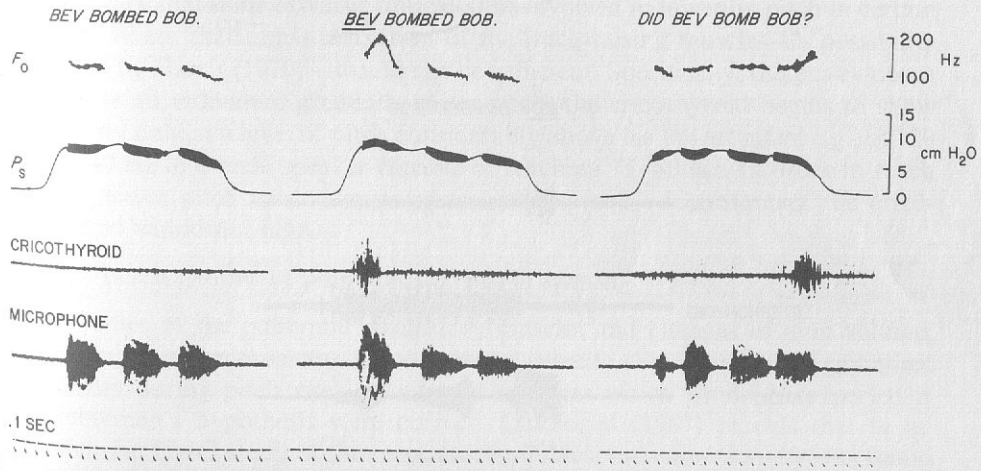

Figure 7. From the top: fundamental frequency (F_0), subglottal air pressure (P_s), cricothyroid activity, microphone signal and .1 sec time standard during three utterances.

first sentence, where there is no pitch rise, this muscle is not active. However, considerable activity is evident during the pitch rises in the other two sentences. Moreover, the degree of activity during the terminal rise in the third sentence, the question, where the involvement of the laryngeal muscles has never been questioned, is of about the same magnitude as the degree of activity associated with the pitch rise on the stressed syllable in the second sentence, where Lieberman would have predicted the laryngeal muscles would show no change in level of activity.

Figure 8 shows F_0 and the activity of the cricothyroid and the sternohyoid muscles. Again, during the rise in pitch on *Bev* the cricothyroid is active, but during the lowered pitch after the rise the cricothyroid is inactive and the sternohyoid shows increased activity. Since the sternohyoid attaches at its upper end to the hyoid bone, it is also occasionally involved in such segmental activities as jaw opening and tongue retraction (Ohala and Hirose 1969). This has led some researchers to discount the role of the sternohyoid in pitch control and to suggest that its activity in such records may be associated only with segmental gestures (Lieberman 1970, Harris 1970). This issue is easily resolved by recording the activity of this muscle during pitch changes in the absence of any segmental gestures, e.g., while humming. Figure 9 (from Ohala 1972b) shows data gathered under such conditions. It shows the sternohyoid active during the lowering of pitch and during the maintenance of low pitch. This finding was recently replicated by Atkinson and Erickson (1976).

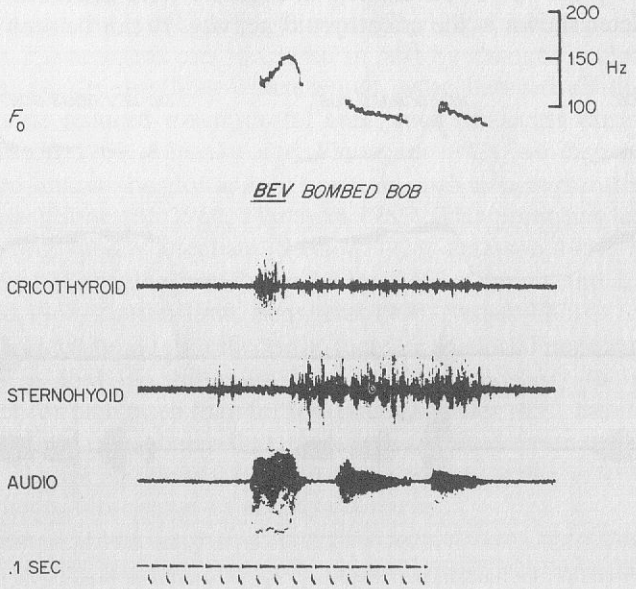

Figure 8. From the top: fundamental frequency (F_0), cricothyroid activity, sternohyoid activity, microphone signal (audio), and .1 sec time standard during the utterance "**Bev** bombed Bob."

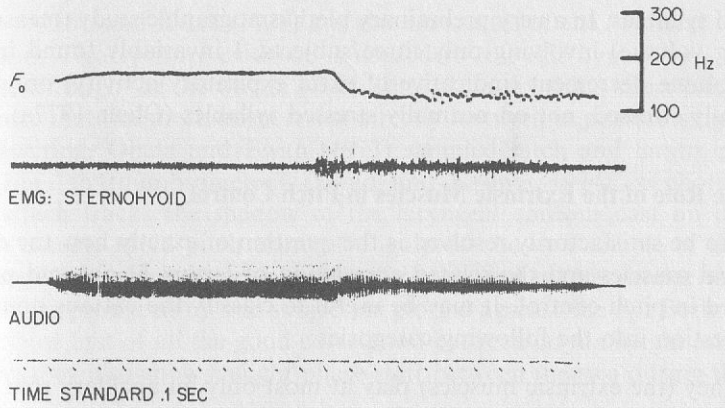

Figure 9. From the top: fundamental frequency (F_0), sternohyoid activity, microphone signal (audio), and .1 sec time standard during a sudden voluntary change in F_0 while the subject phonated with jaw closed.

I should emphasize that the sternohyoid and sternothyroid muscles, although certainly involved in pitch lowering, are not necessarily the only or even the primary muscles responsible for it. This conclusion is based on the fact that these muscles often show no marked activity in speech until after a pitch contour has begun to lower (Sawashima, Kakita, and Hiki 1973, Erickson and Atkinson 1976). Other muscles may be involved in initiating the fall. The main point is that laryngeal muscles do contribute to pitch lowering in speech.

Although there are still reasonable grounds to dispute the involvement of any particular extrinsic muscle in pitch lowering during speech, it would appear necessary that **some** extrinsic muscle(s) be involved in lowering pitch in certain cases where the simple relaxation of the pitch-raising muscles—a possibility raised by Harris (1970)—would not be sufficient. Specifically, there is evidence that as an individual gradually raises pitch, the cricothyroid begins to show activity only at a level of pitch considerably above his lowest pitch, e.g., at 120 or 140 Hz in a male speaker capable of reaching 75–80 Hz. In order to reach such lower pitch levels—quite common in speech—relaxation of the cricothyroid would not help.

5. Investigations of pulmonic activity in speech.

Studies of the pulmonic respiratory muscles and changes in lung volume, although still quite scanty, failed in many cases to show the kind of pulmonic activity during pitch rises on stressed syllables which would be expected if Lieberman's hypothesis were correct. Ladefoged (1967) reports that in an extensive EMG study of the respiratory muscles, increased activity was found on stressed syllables, whether these were emphatically stressed or not. However, Munro and Adams (1971) and van Katwijk (1971, 1974) failed to replicate this finding, except that van Katwijk did find increased activity on emphatically

stressed syllables. In a very preliminary plethysmographic study (measurement of lung volume) involving only three subjects, I invariably found increased lung volume decrement (indicative of extra expiratory activity) only on emphatically stressed, not on normally stressed syllables (Ohala 1977a).

4.2 The Role of the Extrinsic Muscles in Pitch Control

Yet to be satisfactorily resolved is the question of exactly how the extrinsic laryngeal muscles and the related correlation of larynx height and pitch are involved in pitch control. It may be useful to classify the various opinions on this question into the following categories:

1. They (the extrinsic muscles) play at most only an ancillary role in pitch regulation.
2. They play a central role by changing (lengthening and/or shortening) the vocal cords.
3. They play a central role in some other way than by changing vocal cord length.

Sokowlowsky (1943) and Faaborg-Andersen (1964), among others, have espoused (1). Sokowlowsky feels the extrinsic muscles move the larynx about in the neck in order to allow the intrinsic muscles to do their task more efficiently. However, he does not go into detail on this point. It is also the "received opinion" among voice teachers that singers should not shift the position of their larynx to change pitch (Luchsinger and Arnold 1965). This is undoubtedly true, but it need not apply to ordinary speech.

The second view has been advocated by (among others) Sonninen (1956, 1968), Zenker and Zenker (1960), Zenker (1964), Lindqvist (1972), and Erickson, Liberman, and Niimi (1976). Zenker and Zenker suggest a variety of ways the extrinsic muscles could change vocal cord length, whereas the others generally focus on a specific muscle or group of muscles which might perform the task: Sonninen on the sternothyroid, Lindqvist on the pharyngeal constrictors, and Erickson *et al.* on the supralaryngeal muscles which attach to the hyoid bone. All of these hypotheses are quite promising and require further research to test them.

In 1972 I presented evidence for the third position, suggesting that it was the **vertical**, not simply the anterior–posterior tension of the vocal cords which could affect pitch. Conceivably the vertical stretching of the soft tissues of the larynx may cause the vocal cords to be free of overlying tissues (the false vocal cords) and in that way allow freer and more rapid vibration. Another possibility is that vertical stretching thins and vertical compression thickens the cords, thereby affecting their rate of vibration. Stevens (1975) suggests that the vertical shifts of the larynx may create changes in the vocal cord properties which would affect their sensitivity to P_s. Shipp and Haller (1972), Shipp

(1975a, 1975b), and Ewan (1976) are also advocates of the "vertical tension" hypothesis. Clearly more evidence is needed to test this hypothesis.

Whatever might be the way in which vertical larynx shifts affect pitch, there is evidence that this mechanism is less directly involved in pitch raising than in pitch lowering. Ohala and Ewan (1973) sampled pitch and larynx position (using the "thyroumbrometer" (Ewan and Krones 1974), a photoelectric device which tracks the shadow of the laryngeal contour cast on it) while subjects execute rapid pitch changes. Figure 10 shows a sample of the data. In Figure 11 the average larynx height (from several tokens similar to that in Figure 10 as produced by one subject) is plotted against the F_0. Both of these figures show first of all the good correlation between larynx height and pitch; however, they also show a slight phase shift between the two during the pitch

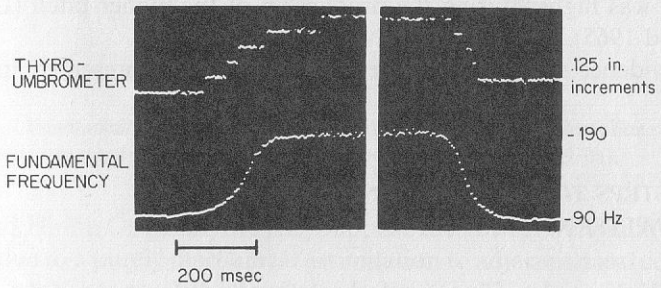

Figure 10. Synchronized variations in fundamental frequency (F_0) and larynx height (transduced by the thyroumbrometer) during a sudden F_0 increase (on the left) and a sudden F_0 decrease (on the right) (from Ohala and Ewan 1973).

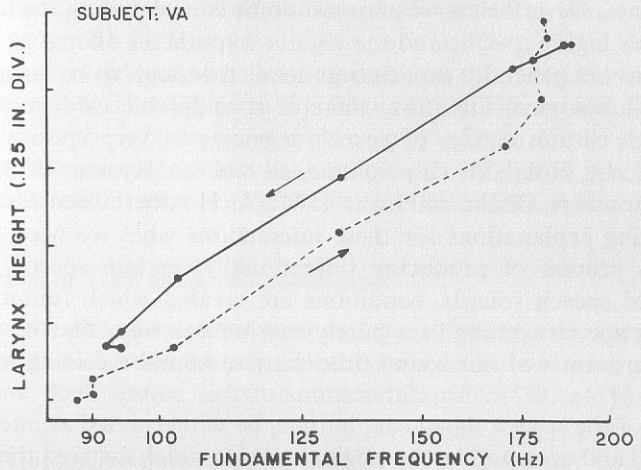

Figure 11. Larynx height plotted as a function of fundamental frequency, F_0, during rapid changes in F_0 (from tokens similar to that in Figure 10) (from Ohala and Ewan 1973).

rise—i.e., after the target high pitch is reached during the rising contour, the larynx continues to rise. In the case of the falling contour, the two parameters are more closely synchronized. Similar records presented by Kakita and Hiki (1976) show the same thing. This may mean that the cricothyroid is the primary pitch raiser, with secondary (and somewhat delayed) assistance from the muscles that raise the larynx; for pitch lowering, though—at least for rapid drops in pitch—the larynx height mechanism may be primary.

Further support for the "vertical tension" hypothesis comes from cases of bilateral cricothyroid paralysis in which patients are still able to vary pitch (although over a more restricted range, of course). In one such case, lateral X-rays taken at two different pitches revealed that there was no change in the rotation of the thyroid and cricoid cartilages with respect to each other—and thus we may presume no anterior–posterior stretching of the vocal cords—but the larynx was higher during the production of the higher pitch (Luchsinger and Arnold 1965).

More evidence relevant to these questions will be presented in the next sections.

5. FIRST STEPS TOWARD EXPLAINING UNIVERSAL TONE PATTERNS

Although the study of laryngeal physiology and the study of the way tones pattern in languages are both relatively far along, the application of the physiological research to questions in tonology is very recent and therefore somewhat primitive. There are more unanswered questions in this area than there are answered ones. Nevertheless we may take some consolation in the fact that the questions are highly specific and the various hypotheses offered as answers to the questions are generally explicit and detailed enough to be testable.

In the phonological literature, there is considerable evidence that tones interact with certain classes of speech segments in very specific ways (see Chapter III, by Hombert, this volume, as well as Hyman 1973, Hombert 1975, and Hombert, Ohala, and Ewan 1976). As Hombert discusses in Chapter III, in seeking explanations for these interactions what we need to show is that in the process of producing (intentionally) certain speech sounds or sequences of speech sounds, conditions are created which (unintentionally) affect other speech sounds in a purely mechanical way, that is, due to the physical constraints of our sound producing or sound receiving mechanisms (see Ohala 1974a, 1975b for elaborations of this point). Such unintentional distortions of the speech signal can, in time, be reinterpreted as intended parts of the signal and even outlive the original sounds which fostered their development. Thus it must have been with the nasal vowels in languages such as

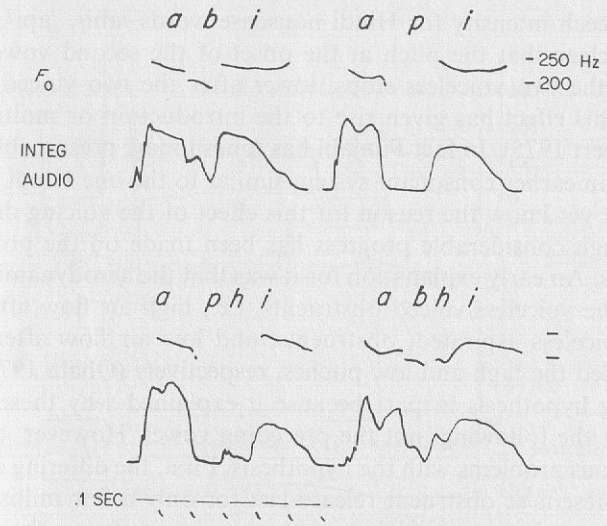

Figure 12. Fundamental frequency (F_0) and the integrated, rectified microphone signal during four Hindi nonsense words; .1 sec time standard at bottom (from Ohala 1974a).

French and Hindi. Originally we may suppose the vowels were unintentionally nasalized due to a purely mechanical assimilation to adjacent nasal consonants; when the nasal consonants dropped out the nasalization remained on the vowels.

Since the interaction between tone and segments is fully covered in Chapter III of this volume, I will restrict my discussion to two aspects of this question. The first concerns the commonly observed phenomenon that voiceless oral obstruents produce high tone (or a higher variant of a tone) on the following vowel, whereas voiced oral obstruents produce low tone (or a lower variant) on the following vowel (Haudricourt 1961, Cheng 1973). The second question I will discuss concerns the influence of vowels on pitch. There will be some overlap between my discussions here and those in Chapter III, but to insure an understanding of the earlier part of this chapter this discussion is necessary.

5.1 Interaction of Tone and Oral Obstruents

The different effects on tone produced by voiceless and voiced oral obstruents are manifested phonetically today even in nontonal languages,[6] e.g., Hindi, as is evident in the pitch curves in Figure 12. The figure shows the

[6] Hombert (1975) and Jeel (1975) provide good reviews of the literature (as well as original contributions) on F_0 differences after voiced/voiceless stops.

pitch and speech intensity for Hindi nonsense words /abi/, /api/, /aphi/, and /abhi/. It is clear that the pitch at the onset of the second vowel is slightly higher after the two voiceless stops, lower after the two voiced stops. Diachronically this effect has given rise to the introduction or multiplication of tones (Hombert 1975). In fact Punjabi has tones today, presumably due to the influence of an earlier consonant system similar to the one Hindi has.

We do not yet know the reason for this effect of the voicing distinction on pitch, although considerable progress has been made on the problem in the last few years. An early explanation for it was that the aerodynamic conditions created by the voiceless/voiced obstruents, i.e., high air flow after voiceless, especially voiceless aspirated, obstruents, and low air flow after voiced obstruents caused the high and low pitches, respectively (Ohala 1973). This was an appealing hypothesis in part because it explained why these consonants should affect the following, not the preceding vowel. However, there are apparently serious problems with the hypothesis. First, the differing aerodynamic conditions present at obstruent release last for only a few milliseconds after voicing begins, whereas the effect of these consonants on the pitch of the following vowels is known to last for 100 msec or more (Hombert 1975). Moreover, although the air flow differences between voiceless and voiced obstruents is as described above at the moment of release, there is some evidence that by the time voicing starts, glottal air flow after voiceless aspirated stops may in fact be **less** than that at vowel onset after a voiced stop. (This is evident in data published by Isshiki and Ringel 1964, van Hattum and Worth 1967.) In any case, the P_s is less at vowel onset following an aspirated stop as opposed to a voiced or voiceless unaspirated stop. This is shown in Figure 13 where the P_s and microphone signal are given for two utterances identical except for the medial stops. P_s builds up (from the level on the vowels) during the closed portions of both stops. At release of the voiced stop the P_s remains at this high level and then gradually returns to normal. After the release of the aspirated stop, however, the P_s decreases markedly (due to the rapid outflow of air) and then gradually builds back up at vowel onset. But P_s is lower at vowel onset

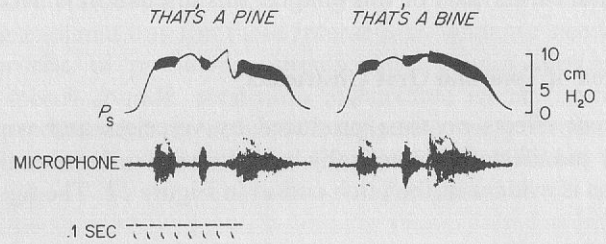

Figure 13. From the top: subglottal air pressure (P_s), microphone signal, and .1 sec time standard during two utterances (from Ohala 1975a).

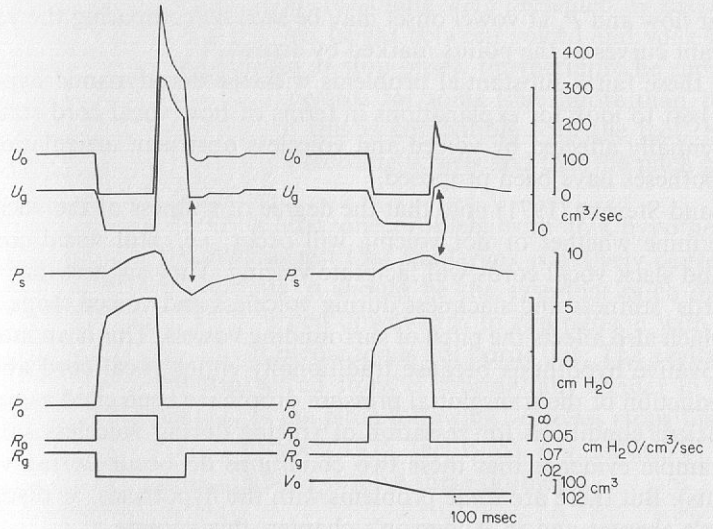

Figure 14. Simulation of aerodynamic events during two VCV utterances. On the left C = voiceless aspirated stop; on the right C = voiced stop. Upper four output parameters: oral airflow (U_o), glottal airflow (U_g), subglottal air pressure (P_s), and oral air pressure (P_o); three input parameters: oral constriction air resistance (R_o), glottal resistance (R_g), oral volume (V_o) (from Ohala 1975a).

after the /ph/ than the /b/. P_s data in other studies reveal the same basic pattern (Ladefoged 1963, Ohala and Ohala 1973).[7]

These relationships between stop type and the air flow and P_s at vowel onset are also predicted by an aerodynamic model of speech (Ohala 1975a, 1976). The input and output of the model for intervocalic voiced and voiceless aspirated stops are given in Figure 14. In the figure, the bottom three curves, oral resistance (R_o), glottal resistance (R_g), and oral volume (V_o) (not given for the voiceless aspirated stop), are the independent parameters, i.e., the input to the model and the top four curves; oral air flow (U_o), glottal air flow (U_g), subglottal air pressure (P_s), and oral air pressure (P_o) are the dependent parameters, i.e., the output of the model. Stop closure occurs when the oral resistance becomes infinite. The main difference between the two stop types is the presence versus the absence of glottal opening (reduction of glottal resistance) when the oral closure occurs. The above-mentioned relations of

[7] A further, if indirect, indication of the reality of this difference in P_s after voiced and voiceless stops can be found in the various reports that vowels have greater intensity following voiced as opposed to voiceless aspirated stops (House and Fairbanks 1953, Lehiste and Peterson 1959). This makes sense only when we realize that voice intensity is largely a function of P_s and that the P_s on vowels will vary as a function of the type of stop preceding as described above.

glottal air flow and P_s at vowel onset may be seen by comparing the values of the relevant curves at the points marked by arrows.[8]

Given these fairly substantial problems with the aerodynamic hypothesis, it seems best to look for explanations in terms of how vocal cord state could be differentially affected by voiced and voiceless obstruent articulation. Two such hypotheses have been proposed.

Halle and Stevens (1971) note that the degree of stiffness of the vocal cords can determine whether or not voicing will occur, i.e., stiff vocal cords will inhibit and slack vocal cords will facilitate voicing. They suggest that it is the vocal cords' stiffness and slackness during voiceless and voiced stops, respectively, which also affects the pitch of surrounding vowels. This is an interesting and innovative hypothesis because traditionally either vocal cord abduction or the reduction of the transglottal pressure drop have been cited as necessary and sufficient conditions for cessation of voicing during voiceless stops (and there is ample evidence that these two conditions do occur during voiceless obstruents). But there are some problems with the hypothesis, as discussed in Hombert's chapter and in Anderson's chapter, this volume.

A second hypothesis suggests itself from the fairly well-established fact that larynx height is slightly higher for voiceless than voiced stops (Jespersen 1889 Hudgins and Stetson 1935, Ewan and Krones 1974, Ewan 1976; further references on this point are given by Slis and Cohen 1969). If it is then true, as suggested above, that, other things being equal, vocal cord tension varies with larynx height, the different pitch on vowels following the different obstruents is accounted for. Presumably the lower larynx during voiced stops is a consequence of the need of the vocal tract to expand in order to accommodate more air in the oral cavity so that it can maintain the positive transglottal pressure drop necessary for voicing. In Figure 14 it can be seen that oral volume (V_o), one of the input parameters to the aerodynamic model, increases during the simulated voiced stop. This turned out to be necessary in order to maintain the glottal air flow necessary for voicing. Without this volume increase P_o would become equal to P_s (and thus the transglottal pressure drop and glottal air flow would fall to zero) within 20 msec of the stop closure onset.

[8] It may be worth mentioning that when originally working on this problem, it was the predictions of the model regarding the differences in air flow after stops which were discovered first, and only then was the literature searched to try to verify the predictions—and, as mentioned, some support was found for them. This is one function of models: to tell us what to look for, to reveal to us the logical consequences of our assumptions, so that by comparing the model's predictions with "real" data we can see whether those assumptions are reasonable or not. Some may think model making is a futile exercise since one can only get out of a model whatever one puts into it, and therefore its output ought to be known beforehand. This is not necessarily the case, however. As Simon (1969) notes, simulation studies can provide new knowledge because "even when we have correct premises, it may be very difficult to discover what they imply. All correct reasoning is a grand system of tautologies, but only God can make use of that fact. The rest of us must painstakingly and fallibly tease out the consequences of our assumptions [p. 15]."

Data which tend to support this reasoning were presented by Ewan (1976), who found the difference in larynx height between voiced and voiceless stops to be greater at stop release than at stop onset. Furthermore he showed that this difference in larynx height persists for some time (more than 100 msec) into the following vowel. All of this is compatible with the fact that stops affect the pitch of following not preceding vowels and that the pitch perturbations last for over 100 msec.

There is, unfortunately, at least one problem with this hypothesis, too. Namely, given that the data suggest that the larynx is actively depressed for voiced stops vis-à-vis sonorants, it should be the case that pitch on vowels after voiced stops is also perturbed downwards in comparison with that for sonorants. However, this latter prediction is not borne out: The available data suggest that pitch following voiced stops is substantially similar to that following sonorants and that it is the pitch following voiceless stops that is perturbed upwards (Lea 1972, Hombert 1975, Jeel 1975). A great deal of research is currently going on to attempt to resolve these problems.

5.2 The Influence of Vowels on Pitch

It has been noted for over 50 years that, other things being equal, the average pitch of vowels shows a systematic correlation with vowel height, that is, the higher the vowel, the higher the pitch (Crandall 1925, House and Fairbanks 1953, Lea 1972). The difference in pitch between high and low vowels may be as much as 25 Hz. Although such a pitch perturbation is of the same order of magnitude as that caused by consonants, there are remarkably few instances in which vowels are claimed to have triggered the development of tones. I suspect the reason for this lies in the constraints of the auditory system and not in the articulatory system (see speculations on this in Hombert, this volume, and Hombert *et al.* 1976). Nevertheless the mechanism causing this pitch perturbation is of interest since it may provide additional evidence on some of the hypothesized ways pitch can be changed other than by action of the intrinsic laryngeal muscles.

Two hypotheses are currently entertained as explanations for this effect. One, the "coupling" hypothesis, suggests that during the production of relatively close, constricted vowels, the acoustic impedance of the vocal tract is high enough to "dictate" to the vocal cords the frequencies they can vibrate at, namely, at or very close to the resonant frequencies of the vocal tract. Since high, close vowels have low first resonances—closest to the F_0 of voice—they would be expected to have the greatest effect on pitch (Lieberman 1970, Atkinson 1973). An early mathematical model of the vocal cords' vibration constructed by Flanagan and Landgraf (1968) did reveal such interaction between vocal cord vibration and the vocal tract, but in later, more sophisticated models this phenomenon was far less evident (Ishizaka and Flanagan 1972).

According to the "tongue pull" hypothesis (Ladefoged 1964, Lehiste 1970), the tongue, in articulating a high close vowel, somehow pulls on the vocal cords and causes greater tension in them, thus raising the pitch slightly. Ohala and Eukel (1976) attempted to test the two hypotheses by measuring the effect on the average pitch of vowels when speakers utter them with and without small wooden blocks propping open their jaws. Earlier research by Lindblom and Sundberg (1971a) showed that speakers can utter acoustically normal high vowels even with their jaws propped open by compensatorily bunching their tongues up more and thus presumably increasing the pull of the tongue on the structures it is connected to, including the larynx. Thus the test allows one of the hypothesized causal factors, tongue pull, to vary while the other causal factor, vocal tract impedance, remains constant. An enhancement of the pitch difference between high and low vowels when speaking with propped open jaw would support the tongue-pull hypothesis; no such enhancement would support the coupling hypothesis.

In fact, the pitch difference was enhanced. This was taken as support of the tongue-pull hypothesis, and therefore may be taken as further support for the hypothesis discussed above that the pitch of voice can be controlled in part by varying the vertical tension of the vocal cords. (See Ohala and Eukel 1976 for more details of the test and for further arguments in support of the conclusion.)[9]

5.3 Falling versus Rising Tones

There are intriguing reports in the tonal literature that falling tones behave differently than rising tones, namely, that they are more numerous in tonal inventories of languages (Cheng 1973), that the pitch interval between the tonal sequence low–high is more likely to be reduced than the interval between a high–low sequence (Hyman 1973a), that falling contours must cover a greater pitch range than a rising contour in order to be perceived with a given level of "prominence" (Black 1970, 't Hart 1975). (For further evidence of such asymmetries see also Hombert 1975.) The perceptual data may simply reveal listeners' "knowledge" of an articulation-caused asymmetry between rising and falling pitch contours, or it could be the reverse: The asymmetry may be of auditory origin, and speakers may modify their articulation to accommodate the listener.

[9] To keep this review as balanced as possible, I should note that objections have been raised against the tongue-pull hypothesis, too. One of these is that larynx height is in certain cases **inversely** related to vowel height, just the opposite of what would be expected if the tongue were pulling on the larynx during high vowels (Ladefoged, DeClerk, Lindau, and Papcun 1972, Atkinson 1973). It is not feasible to review the details of this minor dispute here. Arguments can be found in Ohala 1972b, 1977b, and Ohala and Eukel 1976 as to why this counterevidence to the tongue-pull hypothesis may be irrelevant to this issue.

Some studies by Ohala and Ewan (1973) and Sundberg (1973), although they do not reveal the exact causes of the effect, do suggest that it is at least articulatory in nature. (This does not rule out the possibility that it is both articulatory and auditory in nature.) Briefly, both studies showed that speakers are able to produce a falling pitch over a given pitch interval much faster than a rising pitch over the same interval. (In Sundberg's study this was true only of speakers with no voice training.) This effect is evident in Figure 10, which is from the study by Ohala and Ewan.

Of course, we should be cautious in attempting to apply these results to speech and to tone production, but they may indicate that since falling tones can be produced faster than rising tones, they make better tonal contrasts—are perceptually more salient—and for that reason are found in greater number in languages. Also, since they can be accomplished quicker, they might be less likely than rising tones to "spill over" onto the next syllable. This is an area where more research is needed.

5.4 Downdrift

Most (all?) languages exhibit a gradual fall in pitch from the beginning to the end of an utterance, that is, over a stretch of speech that has been called variously a "phonological phrase," a "breath group," or a "syntagm." (This need not apply to questions, however.) In many tone languages this results in successive tones becoming phonetically lower and lower in pitch until, at the end of the phrase, the high tones could be phonetically as low or even lower than the low tones at the beginning of the phrase. This is called "downdrift" in African languages but is evident in nontone languages as well (Collier 1972, Weitzman 1970). Breckenridge (1977) has, in fact, recently demonstrated the auditory reality of downdrift (or the "declination effect," as she calls it) in English. Listeners judged the peaks of two pitch prominences in a synthesized sentence to be equal even though the second was a few hertz lower than the first. The causes or origins of downdrift are not known, but it is possible to consider the following three hypotheses for it.

Maeda (1975) suggests that the larynx progressively lowers during a single breath group as a result of its linkage to the sternum which should lower as lung volume decreases, and that given the correlation between larynx height and pitch, this movement should cause a gradual lowering of pitch. Ewan (1976) showed, however, that the larynx normally moves upwards during expiration (on which speech is superimposed), not downwards.

One might also suppose that a gradual reduction in P_s due to the pulmonic system's having to work harder (against the elastic recoil force of the lungs and thorax) to maintain a positive P_s as lung volume decreases might cause a gradual lowering of pitch. This hypothesis encounters the same difficulty noted

above for Lieberman's hypothesis, namely, that the magnitude of the observed downdrift usually exceeds what could be accounted for by P_s variations.

A third hypothesis—one which I personally believe in—is that the effect is due to active laryngeally caused changes in vocal cord tension, and that it is not an "automatic" effect at all, but is purposeful. This is superficially reasonable because the gradual pitch decrement in utterances serves a useful linguistic purpose in signaling clause and sentence boundaries. We might say (with apologies to Voltaire) that if downdrift were not purposeful to start out with, speakers would soon enough make it so since it is so useful in speech. Moreover, there is some evidence in favor of this hypothesis, namely, that the rate of pitch decrement is inversely proportional to the length of the utterance it spans (Weitzman 1970, Hirose 1971, Collier 1972, Silverstein 1976). This would be a very amazing finding if the slow pitch fall were a purely mechanical effect determined by decreasing lung volume or the like—in which case the rate should be about the same no matter how long the utterance—on the other hand, it is a very reasonable finding if it is actively and purposefully controlled by the speaker. This still leaves open, of course, the question of how this apparently universal pattern originated.

REFERENCES

Abramson, A. C. 1972. Discussion of plenary address by J.-C. Lafon. In A. Rigault and R. Charbonneau (Eds.), *Proceedings of the 7th International Congress of Phonetic Sciences*. The Hague: Mouton. Pp. 25–26.

Allen, W. S. 1953. *Phonetics in ancient India*. Oxford Univ. Press.

Anthony, J. K. F. 1968. Study of the larynx II. *Work in Progress, 2*, 77–82.

Arnold, G. E. 1961. Physiology and pathology of the cricothyroid muscle. *Laryngoscope, 71*, 687–753.

Atkinson, J. E. 1973. Aspects of intonation in speech: Implications from an experimental study of fundamental frequency. Unpublished Ph.D. dissertation, University of Connecticut.

Atkinson, J. E., & Erickson, D. E. 1976. The function of strap muscles in speech: Pitch lowering or jaw opening. *Journal Acousical Society of America, 60*, S65.

Baer, T. 1975. Investigation of phonation using excised larynxes. Unpublished dissertation, MIT.

Baer, T. 1976. Effects of subglottal pressure changes on sustained phonation. *Journal of the Acoustical Society of America 60*, S65.

Benguerel, A. P. 1970. Some physiological aspects of stress in French. *Natural Language Studies*. University of Michigan Phonetics Laboratory, *4*.

van den Berg, Jw. 1958. Myoelastic-aerodynamic theory of voice production. *Journal Speech and Hearing Research, 1*, 227–244.

van den Berg, Jw., & Tan, T. S. 1959. Results of experiments with human larynxes. *Practica Oto-Rhino-Laryngologica, 21*, 425–450.

Black, J. W. 1970. The magnitude of pitch inflection. In B. Hala, M. Romportl, & P. Janota (Eds.), *Proceedings of the 6th International Congress of Phonetic Sciences, Prague, 7–13 September 1967*. Prague: Czechoslovak Academy of Sciences. Pp. 177–181.

Bouhuys, A. (Ed.). 1968. Sound production in man. *Annals of the New York Academy of Sciences, 155*(1).

Breckenridge, J. 1977. The declination effect. *Journal of the Acoustical Society of America, 60*, S90.

Broad, D. J. 1973. Phonation. In F. D. Minifie, T. J. Hixon, & F. Williams (Eds.), *Normal Aspects of Speech, Hearing, and Language*. Englewood Cliffs: Prentice-Hall. Pp. 127–167.

Cheng, C. C. 1973. A quantitative study of tone in Chinese. *Journal of Chinese Linguistics, 1*, 93–110.

Collier, R. 1972. *From pitch to intonation*. Ph.D. dissertation, University of Louvain.

Collier, R. 1975. Physiological correlates of intonation patterns. *Journal of the Acoustical Society of America, 58*, 249–255.

Crandall, I. B. 1925. The sounds of speech. *Bell System Technical Journal, 4*, 586–622.

Critchley, M., & Kubik, C. S. 1925. The mechanism of speech and deglutition in progressive bulbar palsy. *Brain, 48*, 492–534.

Damste, P. H. 1968. X-ray study of phonation. *Folia Phoniatrica, 20*, 65–88.

Derwing, B. L. 1973. *Transformational grammar as a theory of language acquisition*. Cambridge University Press.

Di Cristo, A. 1975. Soixante-et-dix ans de recherches en prosodie. *Travaux de l'Institut de Phonétique d'Aix-en-Provence, Etudes phonétiques, 1*.

Dixit, R. P. 1975. Neuromuscular aspects of laryngeal control: With special reference to Hindi. Unpublished Ph.D. dissertation, University of Texas.

Erickson, D., & Abramson, A. S. 1972. Electromyographic study of the tones in Thai. *Status Reports on Speech Research*, Haskins Laboratories, *SR-31/32*, 231–236.

Erickson, D., & Atkinson, J. E. 1976. The function of the strap muscles in speech. *Status Reports on Speech Research*, Haskins Laboratories, *SR 45/46*, 205–210.

Erickson, D. M., Liberman, M. Y., & Niimi, S. 1976. The geniohyoid and the role of the strap muscles in pitch control. *Journal of the Acoustical Society of America 60*, S63.

Ewan, W. G. 1976. Laryngeal behavior in speech. Unpublished Ph.D. dissertation, University of California, Berkeley.

Ewan, W. G., & Krones, R. 1974. Measuring larynx movement using the thyroumbrometer. *Journal of Phonetics, 2*, 327–335.

Faaborg-Andersen, K. 1957. Electromyographic investigation of intrinsic laryngeal muscles in humans. *Acta Physiologica Scandinavica, 41*, Suppl. 140.

Faaborg-Andersen, K. 1964. Electromyography of the laryngeal muscles in man. In D. W. Brewer (Ed.), *Research potentials in voice physiology*. State Univ. of New York. New York: University Publishers, Inc. Pp. 105–123.

Faaborg-Andersen, K. 1965. *Electromyography of laryngeal muscles in humans: Technics and results*. Basel: Karger.

Fabre, P. 1957. Un procédé électrique percutane d'inscription de l'accolement glottique au cours de la phonation: Glottographie de haute fréquence. Bulletin, Académie Nationale de Médecine, *141*, 66–69.

Fant, G. 1960. *Acoustic theory of speech production*. The Hague: Mouton.

Ferrein, A. 1741. De la formation de la voix de l'homme. *Mémoires d'Académie Royale des Sciences, 51*, 409–442.

Feyerabend, P. K. 1968. How to be a good empiricist: A plea for tolerance in matters epistemological. In P. H. Nidditch (Ed.), *The philosophy of science*. Oxford Univ. Press. Pp. 12–39.

Fischer-Jørgensen, E. 1974. Electromyographic investigation of Danish consonants, stress, and stød. *Annual Report of the Institute of Phonetics, Univ. of Copenhagen, 8*, 203–206.

Flanagan, J. L. 1965. *Speech analysis, synthesis, and perception*. New York: Academic Press.

Flanagan, J. L., Ishizaka, K., & Shipley, K. L. 1975. Synthesis of speech from a dynamic model of the vocal cords and vocal tract. *Bell System Technical Journal, 54*, 485–506.

Flanagan, J. L., & Landgraf, L. 1968. Self-oscillating source for vocal tract synthesizers. *IEEE Transactions on Audio, 16*, 57–64.

Fromkin, V. A., & Ohala, J. 1968. Laryngeal control and a model of speech production. *Preprints of the speech symposium, Kyoto*. (Reprinted in *UCLA Working Papers in Phonetics, 10*, 98–110.)
Fujimura, O., & Lindqvist, J. 1971. Sweep-tone measurements of vocal-tract characteristics. *Journal of the Acoustical Society of America, 49*, 541–558.
Furukawa, M. 1967. A study of the mechanism of phonation using excised larynges [in Japanese]. *Oto-Rhino-Laryngological Clinic, Kyoto, 60*, 145–181.
Gandour, J., & Maddieson, I. 1976. Measuring larynx height in Standard Thai using the cricothyrometer. *UCLA Working Papers in Phonetics, 33*, 160–190.
Garding, E., Fujimura, O., & Hirose, H. 1970. Laryngeal control of Swedish word tone: A preliminary report of an EMG study. *Annual Bulletin*, Research Institute of Logopedics and Phoniatrics, *4*, 45–54.
Grammont, M. 1965. *Traité de phonétique* (2nd ed.). Paris: Librairie Delagrave.
Greenberg, J. H. 1970. Some generalizations concerning glottalic consonants, especially implosives. *International Journal of American Linguistics, 36*, 123–145.
Haden, E. G. 1938. The physiology of French consonant changes. *Language Dissertations, 26*.
Halle, M. 1954. Why and how do we study the sounds of speech? In H. J. Mueller (Ed.), *Report of the 5th Annual Round Table Meeting on Linguistics and Language Teaching*. Washington, D.C.: Georgetown Univ. Pp. 73–83.
Halle, M., & Stevens, K. N. 1971. A note on laryngeal features. *Quarterly Progress Report*, Research Laboratory of Electronics, MIT, *101*, 198–213.
Hanson, R. J. 1975. Fundamental frequency dynamics in VCV sequences. Paper delivered at 8th International Congress of Phonetic Sciences, Leeds, August.
Harris, K. S. 1970. Physiological aspects of articulatory behavior. *Status Reports on Speech Research*, Haskins Laboratories, *SR 23*, 49–68.
't Hart, J. 1975. Discriminability of the magnitude of pitch movements in speech-like signals. Paper delivered at 8th International Congress of Phonetic Sciences, Leeds, August.
Haudricourt, A. G. 1961. Bipartition et tripartition des systèmes de tons dans quelques languages d'Extrême Orient. *Bulletin de la Société Linguistique de Paris, 56*, 163–180.
Herries, J. 1773. *The elements of speech*. London. (Reprinted: Meuston: The Scolar Press, 1968.)
Hirano, M., Koike, Y., & von Leden, H. 1967. The sternohyoid muscle during phonation. *Acta Oto-Laryngologica, 64*, 500–507.
Hirano, M., & Ohala, J. J. 1969. Use of hooked-wire electrodes for electromyography of the intrinsic laryngeal muscles. *Journal of Speech and Hearing Research, 12*, 362–373.
Hirano,, M., Ohala, J. J., & Vennard, W. 1969. The function of laryngeal muscles in regulating fundamental frequency and intensity of phonation. *Journal of Speech and Hearing Research, 12*, 616–628.
Hirose, M. 1971. On Japanese pitch accent: An acoustic analysis. *Monthly Internal Memorandum*. Phonology Laboratory, Berkeley, May, pp. 16–40.
Hixon, T. J., Mead, J., & Klatt, D. H. 1971. Influence of forced transglottal pressure changes on vocal fundamental frequency. *Journal of the Acoustical Society of America, 49*, 105.
Hombert, J. M. 1975. Towards a theory of tonogenesis: An empirical, physiologically, and perceptually-based account of the development of tonal contrasts in language. Unpublished Ph.D. dissertation, University of California, Berkeley.
Hombert, J. M., Ohala, J. J., & Ewan, W. G. 1976. Tonogenesis: Theories and queries. *Report of the Phonology Laboratory*, Berkeley, *1*, 48–77.
House, A. S., & Fairbanks, G. 1953. The influence of consonant environment upon the secondary acoustical characteristics of vowels. *Journal of the Acoustical Society of America, 25*, 105–113.
Hudgins, C., & Stetson, R. H. 1935. Voicing of consonants by depression of larynx. *Archives Néerlandaises de Phonétique Expérimentale, 11*, 1–28.
Husson, R. 1950. Etudes des phénomènes physiologiques et acoustiques fondamentaux de la voix chantée. *Revue Scientifique, 88*, 67–112; 131–146; 217–235.

Hyman, L. M. (Ed.). 1973a. Consonant types and tone. *Southern California Occasional Papers in Linguistics, 1*.
Hyman, L. 1973b. The role of consonant types in natural tonal assimilation. In L. Hyman (Ed.), Consonant types and tone. *Southern California Occasional Papers in Linguistics, 1*, 151–179.
Ishizaka, K., & Flanagan, J. L. 1972. Synthesis of voiced sounds from a two-mass model of the vocal cords. *Bell System Technical Journal, 51*, 1233–1268.
Isshiki, N. 1959. Regulatory mechanism of the pitch and volume of voice. *Oto-Rhino-Laryngological Clinic*, Kyoto, *52*, 1065–1094.
Isshiki, N. 1964. Regulatory mechanism of voice intensity variation. *Journal of Speech and Hearing Research, 7*, 17–29.
Issihiki, N. 1969. Remarks on mechanisms for vocal intensity variations. *Journal of Speech and Hearing Research, 12*, 669–672.
Isshiki, N., & Ringel, R. 1964. Air flow during the production of selected consonants. *Journal of Speech and Hearing Research, 7*, 233–244.
Jeel, V. 1975. An investigation of the fundamental frequency of vowels after various Danish consonants, in particular stop consonants. *Annual Report of the Institute of Phonetics, Univ. of Copenhagen, 9*, 191–211.
Jespersen, O. 1889. *The articulations of speech sounds represented by means of analphabetic symbols*. Marburg in Hessen: N. G. Elwert.
Kagaya, R., & Hirose, H. 1975. Fiberoptic electromyographic and acoustic analyses of Hindi stop consonants. *Annual Bulletin*, Research Institute of Logopedics and Phoniatrics, *9*, 27–46.
Kakita, Y., & Hiki, S. 1976. Investigation of laryngeal control in speech by use of thyrometer. *Journal of the Acoustical Society of America, 59*, 669–674.
Katsuki, Y. 1950. The function of the phonatory muscles. *Japanese Journal of Physiology, 1*, 29–36.
van Katwijk, A. 1971. Subglottal pressure and linguistic stress. *IPO Annual Progress Report*, Eindhoven, *6*, 31–34.
van Katwijk, A. 1974. Accentuation in Dutch: An experimental study. Ph.D. dissertation, Utrecht Univ.
Kimura, N. 1961. Electromyographic study of the extrinsic muscles of the larynx. *Oto-Rhino-Laryngological Clinic*, Kyoto, *54*, 481.
Ladefoged, P. 1963. Some physiological parameters in speech. *Language and Speech, 6*, 109–119.
Ladefoged, P. 1964. *A phonetic study of West African Languages*. Cambridge Univ. Press.
Ladefoged, P. 1967. *Three areas of experimental phonetics*. Oxford Univ. Press.
Ladefoged, P. 1968. Linguistic aspects of respiratory phenomena. In A. Bouhuys (Ed.), *Sound production in man. Annuals of the New York Academy of Sciences, 155*(1), 141–151.
Ladefoged, P. 1971. *Preliminaries to linguistic phonetics*. Chicago Univ. Press.
Ladefoged, P., DeClerk, J., Lindau, M., & Papcun, G. 1972. An auditory–motor theory of speech production. *UCLA Working Papers in Phonetics, 22*, 48–75.
Lea, W. A. 1972. Intonational cues to the constituent structure and phonemics of spoken English. Ph.D. dissertation, Purdue Univ.
Lehiste, I. 1970. *Suprasegmentals*. MIT Press.
Lehiste, I., & Peterson, G. E. 1959. Vowel amplitude and phonemic stress in American English. *Journal of the Acoustical Society of America, 31*, 428–435.
Lieberman, P. 1967a. Intonation and the syntactic processing of speech. In W. Wathen-Dunn (Ed.), *Models for the perception of speech and visual form*. Cambridge, Mass.: MIT Press. Pp. 314–319.
Lieberman, P. 1967b. *Intonation, perception, and language*. Cambridge, Mass.: MIT Press.
Lieberman, P. 1968. On the structure of prosody. *Zeitschrift für Phonetik, Sprachwissenschaft, und Kommunikationforschung, 21*, 157–163.
Lieberman, P. 1970. A study of prosodic features. *Status Reports on Speech Research*, Haskins Laboratories, *SR-23*, 179–208.
Lieberman, P. 1975. *On the origins of language*. New York: Macmillan.

Lieberman, P., Knudson, R., & Mead, J. 1969. Determination of the rate of change of fundamental frequency with respect to subglottal air pressure during sustained phonation. *Journal of the Acoustical Society of America, 45*, 1537–1543.
Lieberman, P., Sawashima, M., Harris, K. S., & Gay, T. 1970. The articulatory implementation of breath group and prominence: Cricothyroid muscular activity in intonation. *Language, 46*, 312–327.
Lindblom, B. 1972. Phonetics and the description of language. In A. Rigault & R. Charbonneau (Eds.), *Proceedings of the 7th International Congress of Phonetic Sciences, Montreal, 22–28 August 1971*. The Hague: Mouton. Pp. 63–98.
Lindblom, B. 1975. Experiments in sound structure. Plenary address, 8th International Congress of Phonetic Sciences, Leeds, August.
Lindblom, B., & Sundberg, J. 1971a. Neurophysiological representation of speech sounds. *Proceedings of the 15th World Congress of Logopedics and Phoniatrics, Buenos Aires*.
Lindblom, B. E. F., & Sundberg, J. E. F. 1971b. Acoustical consequences of lip, tongue, jaw, and larynx movement. *Journal of the Acoustical Society of America, 50*, 1166–1179.
Lindqvist, J. 1972. A descriptive model of laryngeal articulation in speech. *Quarterly Progress and Status Reports*. Speech Transmission Laboratory, Stockholm, *2–3/1972*, 1–9.
Lindqvist, J., Sawashima, M., & Hirose, H. 1973. An investigation of the vertical movement of the larynx in a Swedish speaker. *Annual Bulletin*, Research Institute of Logopedics and Phoniatrics, *7*, 27–34.
Luchsinger, R., & Arnold, G. E. 1965. *Voice–Speech–Language*. Belmont: Wadsworth.
Maeda, S. 1975. Electromyographic study of intonational attributes. *Progress Report*, Research Laboratory of Electronics, MIT, *115*, 261–269.
Mueller, J. *Manuel de physiologie*. 1851. (Trans. from German by A.-J.-L. Jourdan, 2nd ed.) Paris: Chez J.-B. Baillière.
Munro, R. R., & Adams, C. 1971. Electromyography of the intercostal muscles in connected speech. *Electromyography, 11*, 365–378.
Netsell, R. 1969. A perceptual-acoustic-physiological study of syllable stress. Ph.D. dissertation, Univ. of Iowa.
Netsell, R. 1973. Speech physiology. In F. D. Minifie, T. J. Hixon, & F. Williams (Eds.), *Normal Aspects of Speech, Hearing, and Language*. Englewood Cliffs: Prentice-Hall. Pp. 211–234.
Ohala, J. J. 1970. Aspects of the control and production of speech. *UCLA Working Papers in Phonetics, 15*.
Ohala, J. J. 1971. The role of physiological and acoustic models in explaining the direction of sound change. *Project on Linguistic Analysis Reports*, Berkeley, *15*, 25–40.
Ohala, J. J. 1972a. How to represent natural sound patterns. *Project on Linguistic Analysis Reports*, Berkeley, *16*, 40–57.
Ohala, J. J. 1972b. How is pitch lowered? *Journal of the Acoustical Society of America, 52*,124.
Ohala, J. J. 1973. The physiology of tone. In L. Hyman (Ed.), Consonant types and tone. *Southern California Papers in Linguistics, 1*, 1–14.
Ohala, J. J. 1974a. Experimental historical phonology. In J. M. Anderson & C. Jones (Eds.), *Historical linguistics II: Theory and description in phonology*. Proceedings of the 1st International Conference on Historical Linguistics, Edinburgh, 2–7 September 1973. Amsterdam: North Holland. Pp. 353–389.
Ohala, J. J. 1974b. Phonetic explanation in phonology. In A. Bruck, R. A. Fox, & M. W. LaGaly (Eds.), *Papers from the parasession on natural phonology*, Chicago Linguistic Society. Pp. 251–274.
Ohala, J. J. 1975a. A mathematical model of speech aerodynamics. In G. Fant (Ed.), *Speech communication: Proceedings of the Speech Communication Seminar, Stockholm, 1–3 August 1974* (Vol. 2): *Speech production and synthesis by rule*. Stockholm: Almqvist & Wiksell. Pp. 65–72.

Ohala, J. J. 1975b. Phonetic explanations for nasal sound patterns. In C. A. Ferguson, L. M. Hyman, & J. J. Ohala (Eds.), *Nasalfest: Papers from a symposium on nasals and nasalization*. Stanford: Language Universals Project. Pp. 289–316.
Ohala, J. J. 1975c. Conditions for Vowel devoicing and frication. *Journal of the Acoustical Society of America, 58*, S39.
Ohala, J. J. 1976. A model of speech aerodynamics. *Report of the Phonology Laboratory*, Berkeley, *1*, 93–107.
Ohala, J. J. 1977a. The physiology of stress. In L. M. Hyman (Ed.), *Studies in stress and accent. Southern California Occasional Papers in Linguisrics, 4*, 145–168.
Ohala, J. J. 1977b. Speculations on pitch regulation. *Phonetica, 34*, 310–312.
Ohala, J. J., & Eukel, B. W. 1976. Explaining the intrinsic pitch of vowels. *Journal Acoustic Society of America, 60*, S44.
Ohala, J. J., & Ewan, W. G. 1973. Speed of pitch change. *Journal of the Acoustical Society of America, 53*, 345.
Ohala, J. J., & Hirano, M. 1967. Studies of pitch change in speech. *UCLA Working Papers in Phonetics, 7*, 80–84.
Ohala, J. J., & Hirose, H. 1969. The function of the sternohyoid muscle in speech. *Reports of the Autumn 1969 Meeting of Acoustical Society of Japan*. Pp. 359–360. (Reprinted: *Annual Bulletin*, Research Institute of Logopedics and Phoniatrics, 1970, *4*, 41–44.)
Ohala, J. J., & Ladefoged, P. 1970. Further investigation of pitch regulation in speech. *UCLA Working Papers in Phonetics, 14*, 12–24.
Ohala, J. J., & Lorentz, J. 1977. The story of [w]: An exercise in the phonetic explanation for sound patterns. *Proceedings 3rd Annual Meeting of the Berkeley Linguistics Society*, 577–599.
Ohala, M., & Ohala, J. J. 1973. The problem of aspiration in Hindi phonetics. (In Hindi) In *Hindi Bhashavigyan Ank*, Delhi: Central Hindi Directorate, Ministry of Education and Social Welfare, Government of India. Pp. 67–72. (Trans into English and reprinted in *Project on Linguistic Analysis Reports*, Berkeley, 1972, *16*, 63–70; *Annual Bulletin*, Research Institute of Logopedics and Phoniatrics, 1972, *6*, 39–46.)
Öhman, S., & Lindqvist, J. 1966. Analysis-by-synthesis of prosodic pitch contours. *Quarterly Progress and Status Reports*, Speech Transmission Laboratory, Stockholm, *4/1965*, 1–6.
Öhman, S., Mårtensson, A., Leandersson, R., & Persson, A. 1967. Crico-thyroid and vocalis muscle activity in the production of Swedish tonal accents: A pilot study. *Quarterly Progress and Status Reports*, Speech Transmission Laboratory, Stockholm, *2–3/1967*, 55–57.
Okamura, H., Gould, W. J., & Tanable, M. 1976. The role of the respiratory muscle in phonation. In E. Loebell (Ed.), *XVIth International Congress of Logopedics and Phoniatrics, Interlaken 1974*. Basel: Karger. Pp. 351–356.
Painter, C. 1971. Archetypal breath-groups and the motor theory of speech perception: Evidence from a register tone language. *Anthropological Linguistics, 13*, 349–360.
Painter, C. 1974. On the relationship between grammatical units, tonal units, and physiological constraints on the respiratory system in GWA. *Zeitschrift fur Phonetik, Sprachwissenschaft, und Kommunikationforschung, 27*, 302–319.
Passy, P. 1890. *Etudes sur les changements phonétiques*. Paris: Librairie Firmin-Didot.
Peterson, G. E. 1957. Breath stream dynamics. In L. Kaiser (Ed.), *Manual of phonetics*. Amsterdam: North Holland. Pp. 139–148.
Peterson, G. E. 1958. Some observations on speech. *Quarterly Journal of Speech, 44*, 402–412.
Pressman, J., & Kelemen, G. 1955. Physiology of the larynx. *Physiological Reviews, 35*, 506–554.
Proctor, D. F. 1968. The physiologic basis of voice training. In A. Bouhuys (Ed.), *Sound production in man. Annuals of the New York Academy of Sciences, 155* (1), 208–228.
Rousselot, L'Abbé. 1891. *Les modifications phonétiques de langage*. Paris: H. Welter.
Sawashima, M. 1970. Laryngeal research in experimental phonetics. *Status Reports on Speech Research*, Haskins Laboratories, *SR-23*, 69–115.

Sawashima, M., Kakita, Y., & Hiki, S. 1973. Activity of the extrinsic laryngeal muscles in relation to Japanese word accent. *Annual Bulletin*, Research Institute of Logopedics and Phoniatrics, *7*, 19–25.

Sawashima, M., Sato, M., Funasaka, S., & Totsuka, G. 1958. Electromyographic study of the human larynx and its clinical application [in Japanese]. *Journal of the Oto-Rhino-Laryngological Society of Japan*, *61*, 1357–1364.

Scripture, E. W. 1902. *Experimental phonetics*. New York: Scribners.

Shipp, T. E. 1975a. Vertical laryngeal position during continuous and discrete vocal frequency change. *Journal of Speech and Hearing Research*, *18*, 707–718.

Shipp, T. E. 1975b. The apparent functions of vertical larynx position for phonation. Paper delivered at 8th International Congress of Phonetic Sciences, Leeds, August.

Shipp, T. E., & Haller, R. H. 1972. Vertical larynx height during vocal frequency change. *Journal of the Acoustical Soceity of America*, *52*, 124.

Shipp, T., & Izdebski, K. 1975. Vocal frequency and vertical larynx positioning by singers and nonsingers. *Journal of the Acoustical Soceity of America*, *58*, 1104–1106.

Silverstein, R. O. 1976. A strategy for utterance production in Hausa. *Studies in African Linguistics*, Suppl. 6, 233–241.

Simada, Z., & Hirose, H. 1970. The function of the laryngeal muscles in respect to the word accent distinction. *Annual Bulletin*, Research Institute of Logopedics and Phoniatrics, *4*, 27–40.

Simada, Z., & Hirose, H. 1971. Physiological correlates of Japanese accent patterns. *Annual Bulletin*, Research Institute of Logopedics and Phoniatrics, *5*, 41–49.

Simon, H. A. 1969. *The science of the artificial*. Cambridge, Mass.: MIT Press.

Slis, I. H., & Cohen, A. 1969. On the complex regulating the voiced–voiceless distinction I & II. *Language and Speech*, *12*, 80–102; 137–155.

Smith, S. 1944. *The Danish stød*. Copenhagen: Kaifer.

Sokowlowsky, R. R. 1943. Effect of the extrinsic laryngeal muscles on voice production. *Archives of Otolaryngology*, *38*, 355–364.

Sonesson, B. 1960. On the anatomy and vibratory pattern of the human vocal folds. *Acta Oto-Laryngologica*, Suppl. 156.

Sonninen, A. 1956. The role of the extrinsic laryngeal muscles in length adjustment of the vocal cords in singing. *Acta Oto-Laryngologica*, Suppl. 130.

Sonninen, A. 1968. The external frame function in the control of pitch in the human voice. In A. Bouhuys (Ed.), Sound production in man. *Annals of the New York Academy of Sciences*, *155* (1), 68–89.

Stetson, R. H. 1928. *Motor phonetics*. Amsterdam: North Holland.

Stevens, K. N. 1975. Modes of conversion of airflow to sound, and their utilization in speech. Plenary address, 8th International Congress of Phonetic Sciences, Leeds, August.

Strenger, F. 1958. Mesure de la pression d'air sous-glottique, de la pression acoustique, et de la durée de la prononciation des différents sons de langage suédois au cours de la phonation. *Journal Français d'Oto-Rhino-Laryngologie Audio-Phonologie et Chirugie Maxillofaciale*.

Sundberg, J. 1973. Data on maximum speed of pitch changes. *Quarterly Progress and Status Reports*, Speech Transmission Laboratory, Stockholm, *1973/4*, 39–47.

Sweet, H. 1877. *A handbook of phonetics*. Oxford: Clarendon Press.

Vanderslice, R. 1967. Larynx vs. lungs: Cricothyrometer data refuting some recent claims concerning intonation and archetypality. *UCLA Working Papers in Phonetics*, *7*, 69–79.

van Hattum, R. J., & Worth, J. H. 1967. Air flow rates in normal speakers. *Cleft Palate Journal*, *4*, 137–147.

Vesalius, A. 1543. *De humani corporis fabrica* and [*The*] *epitome* [*of Andreas Vesalius*]. Basel. [*Epitome* trans. into English by L. R. Lind. Cambridge, Mass.: M.I.T. Press, 1969.]

Wegel, R. L. 1930. Theory of vibration of the larynx. *Bell System Technical Journal*, *9*, 207–227.

Weitzman, R. S. 1970. Word accent in Japanese. *Studies in the Phonology of Asian Languages*, Univ. of Southern California, *11*.

Willis, R. 1833. On the mechanism of the larynx. *Transactions of the Cambridge Philosophical Society*, *4*, 323–352.

Yanagihara, N., & von Leden, H. 1966. The cricothyroid muscle during phonation. *Annals of Otology, Rhinology, and Laryngology*, *75*, 978–1006.

Zee, E., & Hombert, J. M. 1976. Intensity and duration as correlates of F_0. *Journal of the Acoustical Society of America*, *60*, S44.

Zemlin, W. R. 1968. *Speech and hearing science*. Englewood Cliffs: Prentice Hall.

Zenker, W. 1964. Questions regarding the function of external laryngeal muscles. In D. W. Brewer (Ed.), *Research potentials in voice physiology*. State Univ. of New York. Pp. 20–29.

Zenker, W., & Zenker, A. 1960. Über die Regelung der Stimmlippenspannung durch von aussen eingreifende Mechanism. *Folia Phoniatrica*, *12*, 1–36.