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THE PHYSIOLOGY OF STRESS

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INTRODUCTION

Although "stress" has been observed in many of the world's languages, its physical correlates have been widely studied only in European languages and of these, one language, English, has been the most extensively investigated. Therefore, the points made in the following review of the physiological correlates of stress should not be taken as necessarily applying to any language outside this restricted range. Moreover, I will make no attempt to give systematic coverage to those aspects of the phonetics of stress and accent covered in some of the extensive reviews already available, e.g., Vanderslice (1968), Broad (1968), Lehiste (1970), Ohala (1970), Benguerel (1970), Gårding (1973), Netsell (1973), van Katwijk (1974), and Di Cristo (1975). I will instead concentrate on some of the more controversial aspects of the subject.

EARLY VIEWS ON STRESS

Early assumptions on the articulatory correlates of what was called "stress" in such languages as English, German, and Russian generally had it that stressed syllables were produced with greater articulatory, especially expiratory, force and thus greater intensity than unstressed syllables (Sweet 1890, 1911, Passy 1895, Jones 1960, Grammont 1965). This type of accent system, so-called (among other things) "expiratory accent" or "accent of intensity," was held to be different from the type of accent found in such languages as Swedish, Serbo-Croatian, and Chinese, which was called "pitch accent". The latter type was supposedly realized by distinct pitch[1] modulations (Roudet 1910). The term "stress" was generally reserved for the first type of accent system. It was also allowed that stressed syllables were generally longer than unstressed syllables (Parmenter and Treviño 1935, Muyskens 1931).

There were, however, a few who thought a pitch change was an essential element of "stress"; Stetson (1928) cites Mitford (1804), Coleman (1914), Abas (1925), and Morris (1925). Stetson himself, while allowing that there might be a pitch change on accented syllables, thought that even so it would be a secondary effect of the increased expiratory force since

> ... the heavy stroke of the accent involves the chest pressure and is apt to change the pitch because the laryngeal musculature is often affected by tensions in the other musculatures of speech (141).

However, he cites no evidence supporting this claim (of the special sensitivity of the laryngeal muscles to other muscles' contractions) and I know of none even today.

Some of the earliest objective analyses of speech supported the minority view on the role of pitch in the production of stressed syllables. Muyskens (1931) and Scott (1939), for example, found significant pitch variations on stressed syllables and the latter even claimed:

> ...there seems to be a strong indication that stress [i.e., intensity] unaided, is not very efficient as a distinguishing feature of English...

Some influential research on the physiological correlates of stress was that conducted by Stetson (1928) who presented evidence (e.g., records of oral and subglottal air pressure and chest movements) that there were separate expiratory pulses ('breath pulses') for each spoken syllable as well as extra heavy pulses for stressed syllables. Many of Stetson's recording techniques were rather primitive and some of his experimental techniques problematic (for example, the fact that many of his syllables showing discrete breath pulses also happened to be almost isolated utterances) and many of his findings were not replicated by Ladefoged (1962, 1967) and his colleagues or by Lieberman, Griffiths, Mead, and Knudson (1967), both using more modern techniques. Nevertheless, Ladefoged did find a momentary increase in the activity of the expiratory muscles (sampled via electromyography) and in the subglottal air pressure during and sometimes immediately before a stressed syllable, whether emphatically stressed or not. He calibrated the effect that variations in subglottal pressure, Ps^2, would have on both intensity (Ladefoged and McKinney 1963) and Fo (Ladefoged 1963). The Ps was found to be directly related to the major variations in intensity of voice (independent of those due to the resonance characteristics of the vocal tract). However, the major part of the Fo variations including those on stressed syllables, were accomplished by the larynx via changes in the tension of the vocal cords.

RESULTS OF MODERN RESEARCH

As a prelude to the discussion of subsequent work on the physiology of stress it may be appropriate to mention some of the extensive experimental data on the perceptual correlates of stress. Using such techniques as synthetic speech, Bolinger (1958), Lawrence (1953), and Fry (1955, 1958, 1960, 1965) found that in English there was actually a hierarchy of cues that listeners used to identify stress on a syllable; listed in the approximate order of greater to lesser importance: pitch modulation, duration, intensity, segmental quality (including especially vowel quality, but probably also such things as degree of aspiration, etc.). As for pitch modulation, it was shown that it was changing pitch, not necessarily higher pitch, which served most effectively to signal stress.

Acoustic analysis of natural speech (again in English) generally reinforced the findings from these perceptual studies (e.g., Bolinger 1958, Lieberman 1960, Lehiste and Peterson 1961, Morton and Jassem 1965, Lisker and Abramson 1967). Generally similar results were obtained from other languages identified as having stress: Polish (Jassem, Morton, and Steffan-Batóg 1968), German (Isačenko and Schädlich 1966), Czech (Janota and Liljencrants 1969, Ondráčková 1972, Janota and Ondráčková 1975), Dutch (van Katwijk and Govaert 1967, van Katwijk 1969). (It should be acknowledged, however, that quite different results from the above have also been obtained on occasion. Fonagy (1958), for example, reports that in his studies of Hungarian stress increased respiratory muscular activity is a more reliable correlate of stress than variations in intensity, Fo, or duration.)

Perhaps the ultimate verification of the correctness of the above findings, especially the primary importance of pitch and duration as the essential cues for stress, has been the fact that highly successful synthetic speech has been generated by rules based on these findings (Mattingly 1966, 1968, Rabiner 1968, Matsui, Suzuki, Umeda, Omura 1968, Umeda 1976).

Given these results in the perceptual domain, it is not surprising that the more recent physiological investigations of stress have confirmed that all the characteristics of the speech signal shown to be perceptually important are accomplished actively by the articulators in the vocal tract (and the muscles which serve them) which are responsible for producing those features. For example, cine x-ray studies show greater extent and force of articulator movement (and consequently greater duration) when stressed as opposed to unstressed syllables are produced (Broad 1968, Harris, Gay, Scholes, and Lieberman 1968, Kent and Netsell 1972). This is, however, mainly true of what would be classified as emphatically stressed syllables, less so of normal, non-emphatic, non-contrastive, word stress.

F_O VARIATIONS IN STRESS: IS THE LARYNX OR LUNGS RESPONSIBLE?

The only moderately controversial topic in the whole area of the physiology of stress is that surrounding the question of how the pitch variations associated with stress are controlled, specifically, whether by the laryngeal or the pulmonic systems. This controversy was precipitated by the publication in 1967 of the M.I.T. doctoral dissertation of Philip Lieberman in which it was claimed that except for the terminal pitch rise in yes-no questions, the pitch variations in speech were regulated primarily by the subglottal air pressure which, in turn, was controlled by the pulmonic system.[3]

The basis for his claim was recordings (from three speakers) of the acoustic speech signal and Ps during a variety of utterances. A sample of his data is given in Figure 1. In this data he noted that there was generally a close temporal coincidence between a momentary Fo rise on stressed syllables (marked 'A' in Figure 1) and a momentary increase in Ps (marked 'A'') and likewise the fall in Fo at the end of declarative sentences and the fall in the Ps (not manifested in this figure).
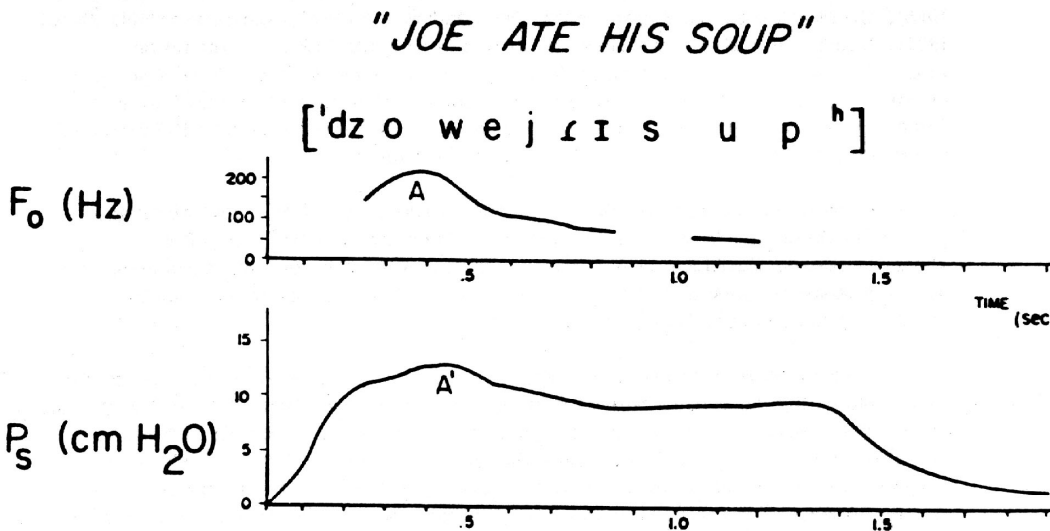
Figure 1.   Sample of data obtained by Lieberman (1967). Top: Fundamental frequency
(Fo); bottom: subglottal pressure (Ps). The data shows the close temporal
coincidence of the momentary rises in Fo (A) and Ps (A's).

(Such observations, of course, had been made before by others who recorded Ps variations in speech, e.g., Smith 1944.) Lieberman concluded from this that the Ps changes caused the Fo variations; he assumed the laryngeal muscles played no essential part in varying pitch in these cases. He measured corresponding values of Fo and Ps at points in the data where he assumed the laryngeal muscles were inactive (i.e., in declarative sentences) and arrived at "calibrations" of the effect of Ps on Fo that ranged from 16 to 22 Hz/cm H_2O.[4]

The crucial assumption in Lieberman's claims, that the laryngeal muscles maintain a constant level of activity except during the Fo rises of questions, was quite unsupported by independent evidence. Of course, at that time neither Lieberman nor anyone else had directly measured the level of activity of the laryngeal muscles during connected speech. Nevertheless up to that time almost all phoneticians and speech scientists held the view opposite to Lieberman's, viz., that the Fo of voice during speech is in all cases primarily regulated by the laryngeal muscles and that the effect of Ps on Fo was too small to account for the major part of the observed Fo variations, including those on stressed syllables (Sweet 1877, Scripture 1902, Stetson 1928, Pressman and Kelemen 1955, Ladefoged 1963, Fry 1964, Öhman and Lindqvist 1966, Zemlin 1968, Proctor 1968). There were several reasons for this dominant view:

1. It was commonly noted that the larynx moved up and down in the neck during the Fo changes in speech and it was therefore assumed that (somehow or other) the larynx contributed to these changes (Herries 1773 , Scripture 1902, Critchley and Kubik 1925).

2. It had been a common clinical observation that paralysis of the intrinsic (or some extrinsic) laryngeal muscles frequently led to defects in the control of pitch in speech (Critchley and Kubik 1925, Luchsinger and Arnold 1965, Arnold 1961, Sonninen 1956). On the other hand, respiratory paralysis did not lead to any difficulty in pitch regulation (Peterson 1958).

3. Direct electromyographic recordings of the activity of the laryngeal muscles during steady-state phonation or during singing showed the intrinsic and extrinsic laryngeal muscles, especially the cricothyroid, to be very active during Fo variations (Katsuki 1950, Faaborg-Andersen 1957, 1965, Sawashima, Sato, Funasaka, and Totsuka 1958, Arnold 1961, Kimura 1961, Hirano, Koike, and von Leden 1967). It was therefore assumed (explicitly so by Arnold) that these results could be extrapolated to speech conditions.

4. Calibrations of the effect of Ps variations on Fo, where care was taken to insure that the vocal cords maintained a constant level of tension, were done on excised larynges as early as the first half of the 19th century (Mueller 1851) and on intact speakers by Isshiki (1959), Ladefoged (1963), and Öhman and Lindqvist (1966). In the studies involving the living subjects the procedures involved having the subject produce a steady-state vowel at a constant pitch while receiving slight pushes on the chest at unexpected moments.

These pushes produced brief involuntary increases in Ps which in turn produced brief increases in Fo. In general such studies yielded ratios of $\Delta Fo/\Delta Ps$ in the range 2 to 5 Hz/cm H2O in the pitch range used in speech. Ladefoged and Öhman and Lindqvist then applied these values to the analysis to their own records of Fo and Ps during connected speech (i.e., working with the same kind of data Lieberman did) and factored out that part of the Fo contour that could be attributed to variations in Ps. From this the major part of the observed Fo variations in speech had to be attributed to the action of the laryngeal muscles. Öhman and Lindqvist cited additional reasons for their conclusions:

> ...The ΔP change [i.e., the change in the pressure drop across the glottis] which is due to stress is always much smaller than that due to stop consonants for instance, and ...the fo changes during the stressed syllables do not correlate well with the stress-induced ΔP changes either in phase or in amplitude.

(This latter point, in fact, also applies to the data in Fig. 1).

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

Lieberman's hypotheses were quickly disproved. New evidence was obtained as well as refinements of the existing counterevidence which was known before the publication of Lieberman's provocative claims. These took several forms:

1. Vanderslice (1967) recorded the vertical movements of the larynx and Ps during connected speech and showed the former to be in better synchronization with Fo than was Ps. It is reasonable to conclude, then, that the larynx, at least in part by its vertical movements, actively participates in the control of Fo. It should be mentioned, though, that the larynx-hyoid apparatus (unlike the lower mandible, for instance) is quite complex as regards its muscular structure and attachments to the skeletal frame and accordingly has many degrees of freedom in its movements (Ohala and Hirose 1969, Ohala 1972). It is not unexpected then that there should be some inter- or even intra-subject variability as regards use of gross vertical movements of the larynx in Fo regulation. Thus, although there may be some speakers for which the vertical movements of the larynx do not show clear correlation with Fo during speech (Gandour and Maddieson 1976), this in no way undermines the conclusion that this mechanism is used by many if not most speakers during speech, especially for large variations in Fo such as occur on stressed syllables (Ohala 1972, Ewan and Krones 1974, Shipp 1975a, b, Ewan 1976).

2. Additional calibrations of the effect of Ps on Fo were done using refined techniques (including monitoring laryngeal muscle activity during the induced transglottal pressure changes) and studying a greater range of voice qualities, initial Fo levels, and intensities (Ohala and Ladefoged 1969, Ohala 1970, Lieberman, Knudson, and Mead 1969, Hixon, Mead, and Klatt 1971). A summary of the values of ΔFo/ΔPs from these and earlier studies are given in Table 1. In general, the studies using excised larynges yield higher values for this ratio than do the studies involving intact larynges, Lieberman's (1967) study excepted. This may very well be an artefact due to the excised larynx lacking normal muscle tonus (van den Berg and Tan 1959). Lieberman, Knudson, and Mead's (1969) upper limit for normal voice of 10 Hz/cm H_2O is still on the high side although it is substantially lower than the values claimed by Lieberman (1967). Curiously, Hixon, Mead, and Klatt (1971), using the same technique and one of the same subjects involved in the study of Lieberman et al. (1969), could not replicate those authors' findings.

There are, of course, methodological problems with all these calibration techniques. Nevertheless, there is little evidence to support Lieberman's claims that the Ps could be responsible for all or even most of the observed Fo variations on stressed syllables. This conclusion is not surprising since Lieberman made no serious attempt to insure that the tension of the vocal cords (as affected by the laryngeal muscles' activity) remained constant during the portions he computed Δ Fo/Δ Ps. [5]

3. It was pointed out that Ps 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 closure, reduced mean glottal area (such as happens during increases in Fo (Sonesson 1960, Ishizaka and Flanagan 1972)) or increased percentage of closed time in the glottal area function (such as occurs during increases in voice intensity) (Isshiki 1969, Ohala 1970, 1975a, 1975b, van Katwijk 1971, 1974). This is not a controversial point; it is well known among speech scientists (Stetson 1928, Peterson 1957, Isshiki 1961, 1964, Campbell, Murtagh, and Raber 1963, Yanagihara and von Leden 1966, Öhman and Lindqvist 1966, Ladefoged 1968, Koyama, Kawasaki, and Ogura 1969, Rothenberg 1968, Zemlin 1968, Benguerel 1970, Netsell 1973, Löfqvist 1975).

A related fact which undoubtedly reflects the increasing impedance of the glottis with increasing Fo is that the minimum pressure drop necessary to maintain voicing is greater for high Fo than low Fo (Mueller 1851, Isshiki 1959).

- Indirect evidence of increased glottal resistance during stressed syllables (in comparison to unstressed syllables) would be the finding of less oral air flow on stressed syllables (Ohala 1975a, 1975b). Such reductions in

Table 1. Values of $\Delta Fo/\Delta Ps$ (in Hz/cm H₂O) from various studies.

Source	Normal voice	High pitch & Falsetto
Mueller (1851)*	4.3 ~ 4.5	10 ~ 16
Isshiki (1959)	3.3	
van den Berg and Tan (1959)*	5 ~ 13	17 ~ 20
Ladefoged (1963)	5	
Öhman and Lindqvist (1966)	2.5	
Furukawa (1967)*	8**	
Anthony (1968)*	6 ~ 8	
Lieberman, Knudson, and Mead (1969)	3 ~ 10	9 ~ 18
Ohala and Ladefoged (1969) Ohala (1970)	2 ~ 4 2 ~ 8	7 ~ 10
Hixon, Mead, and Klatt (1971)	2 ~ 4	
Lieberman (1967)	16 ~ 22	

* Used excised larynges.

** Average slope of one Fo vs. Ps plot which ranged from 0 to 16 Hz/cm H₂O.

air flow, although probably not to be found on all stressed syllables, are evident in some of the published data of Klatt, Stevens, and Mead (1968; see their figure 5) and Broad (1968).

Thus not only is it improbable that the observed Ps variations could cause much of the observed Fo changes, it is probable that to some extent the Ps fluctuations were themselves caused by the laryngeal muscles as they adjusted the state of the vocal cords for Fo and intensity variations.

4. Finally, and most conclusively, the activity of the laryngeal muscles was sampled directly during connected speech using electromyography (Ohala and Hirano 1967, Hirano, Ohala and Vennard 1969, Hirano and Ohala 1969, Fromkin and Ohala 1968, Ohala 1970, 1972, Lieberman, Sawashima, Harris, and Gay 1970 Atkinson 1973, Netsell 1973, Collier 1975, Maeda 1975, Shipp 1975a, Erickson and Atkinson 1975, Kakita and Hiki 1975) and it was found that Fo was varied in speech in much the same way as it was in singing. There were muscles active for raising pitch: the cricothyroid, thyrohyoid, lateral crico-arytenoid, and vocalis; and muscles active during lowering of pitch: the sternohyoid and sternothyroid. (The involvement of the strap muscles in Fo change is not surprising given the evidence reviewed above that larynx height varies with Fo.)

Typical data is shown in Figs. 2 and 3 (from Ohala 1970). In Fig. 2 the records of Fo, Ps, activity of the cricothyroid and lateral crico-arytenoid, and the voice signal are given for three utterances. From such data it is evident that although there is a noticeable increase in Ps on stressed syllables (compare the middle sentence with the first in Fig. 2), there is also a considerable increase in the activity of the cricothyroid muscle at the same time. Fig. 3 shows an increase in the activity of the sternohyoid muscle during the lowering of Fo. (It should be mentioned, however, that the sternohyoid also participates in purely segmental gestures as well (Ohala and Hirose 1969, Ohala 1972) and may in some subjects only reveal its involvement in Fo lowering in the indirect way of not being active during high Fo.)

The increase in the activity of the cricothyroid muscles during the stressed syllable in Fig. 2 (middle sentence), where Lieberman would have predicted the change in laryngeal muscle activity to be nil, is of about the same magnitude as that during the final pitch rise in the question (right-most sentence), where the involvement of the laryngeal muscles has never been in question.

To summarize the evidence reviewed: the magnitude of the effect of Ps on Fo is not great enough to account for the major part of the observed Fo changes on stressed
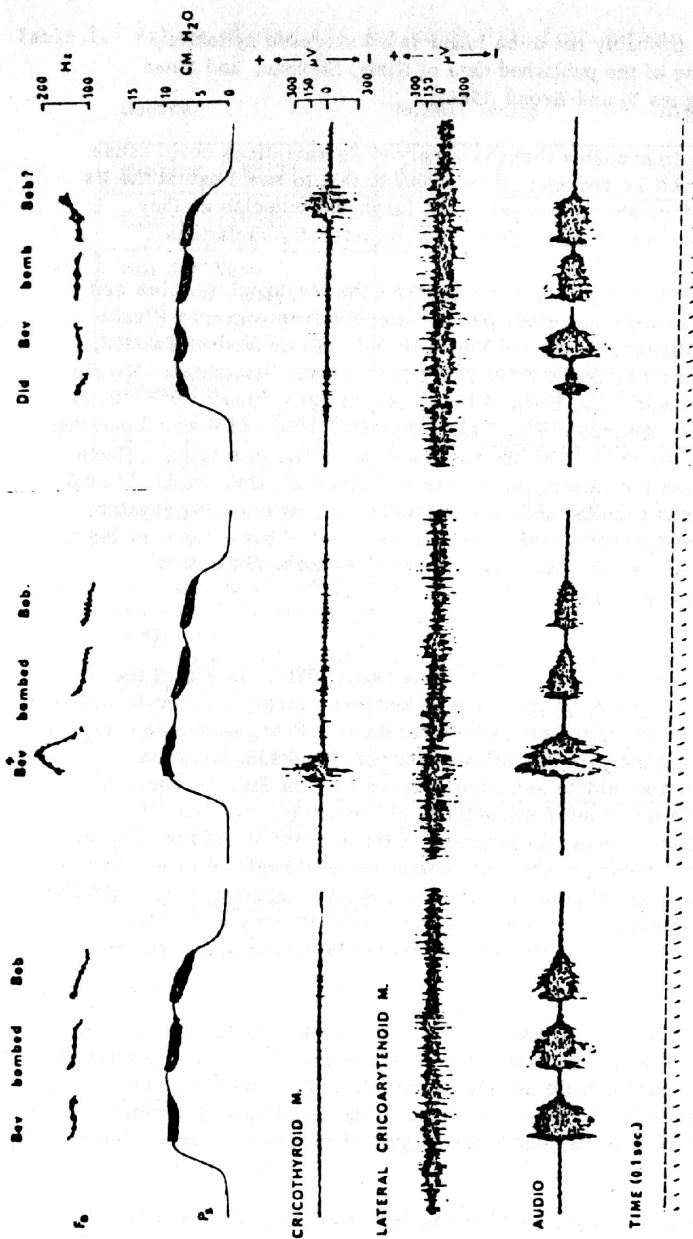
Figure 2. From top: fundamental frequency (Fo), subglottal pressure (Ps), electro-myographic signal from the cricothyroid muscle, electromyographic signal from the lateral crico-arytenoid muscle, and microphone signal of voice. The figure shows how these parameters vary during the three utterances (from left to right) "Bev bombed Bob," "BEV bombed Bob," and "Did Bev bomb Bob?" (nonsense sentences constructed to have mostly labial and voiced consonants and non-high vowels).
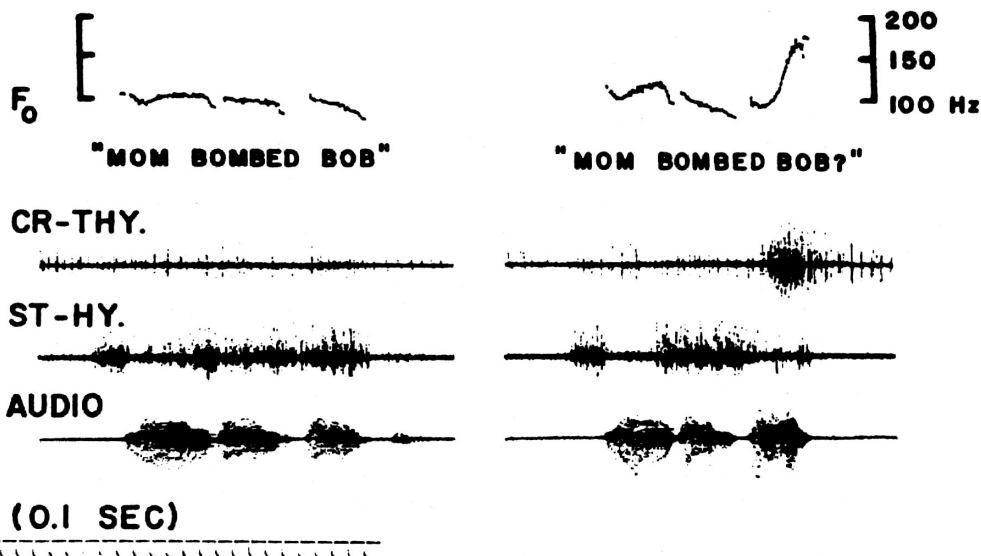
Figure 3. From top: fundamental frequency (Fo), electromyographic signal from the cricothyroid muscle, electromyographic signal from the sternohyoid muscle, and microphone signal of voice. The utterances are: "Mom bombed Bob" (on the left) and "Mom bombed Bob?" (on the right). As the two utterances are phonetically identical except for the Fo contour, the observed changes in the muscle activity from one utterance to the other must be due to these muscles' involvement in Fo regulation, not segmental gestures. The cricothyroid shows increased activity during increases of Fo, no activity during lowering of Fo; conversely, the sternohyoid shows, in addition to participation in segmental gestures (jaw opening for labial consonant + low vowel sequences), increased activity for Fo lowering, lessened activity for Fo rises.

vowels (nor the fall in Fo at the end of declarative sentences); some of the Ps variations, in fact, are probably dependent upon the action of the larynx itself, not the pulmonic system; there is indirect and direct evidence that the laryngeal muscles cause the Fo variations in speech. The answer to the question, so epigrammatically phrased by Vanderslice (1967), 'is it the larynx or lungs that controls pitch?', is the larynx.

THE PULMONIC CONTRIBUTION TO STRESS

Even though it is clear now that any pulmonic contribution to the Fo change observed on stressed syllables must be small   this does not rule out the possibility of a pulmonic contribution to other aspects of stress, in particular, increases in intensity. Ladefoged (1963, 1967, 1968) citing his own extensive electromyographic investigations of the activity of the expiratory muscles during speech, maintains that there is a momentary increase in expiratory activity during or slightly preceding each stressed syllable, whether emphatically stressed or not. Van Katwijk (1974), however, obtained surface electromyographic recordings of the activity of some of the expiratory muscles during speech (of two native speakers of Dutch) and found appreciable increases in expiratory muscular activity only on emphatically stressed syllables, not on conversationally stressed syllables.

It is difficult to evaluate such electromyographic records by themselves, however, for two reasons: First, there is no simple way of estimating the magnitude of lung volume decrement such bursts of expiratory muscle activity cause and, after all, it is only by decreasing the lung volume at a faster-than-normal rate that these muscles can have any influence on Ps. It is possible these fluctuations in the level of expiratory muscle activity produce only negligible variations in Ps. Second, it is difficult to know why the expiratory muscles show increased activity near stressed syllables. Aside from the pulmonic system's possible role in the production of individual speech segments or stressed syllables, we do not know whether its long-term function is to maintain a constant pressure in the lungs or to maintain a constant rate of lung volume decrement, or some combination of the two. If its task is to maintain a steady rate of lung volume change, then the momentary increase in expiratory activity on stressed syllables could simply be a compensatory reaction to the increased glottal and supraglottal resistance to air flow expected during the production of stress--which increased resistance would momentarily slow the rate of lung volume decrease. If this were so, the increased activity of the pulmonic muscles would not, strictly speaking, be an independent feature of stress, it would be a feature dependent upon glottal and supraglottal activity.

It would be helpful to obtain some independent measure of the pulmonic system's contribution to Ps changes during speech. This can be done most directly by recording variations in lung volume during speech. Such records can be obtained using a plethysmograph such as has recently been constructed in the Phonology Laboratory at Berkeley.

Ours is a whole–body pressure plethysmograph. It consists of 1 and 2/3 50-gal. oil drums welded together and tightly fitted with a plexiglass lid. The subject stands in the plethysmograph and breathes and speaks through a face mask mounted in the side of the lid (see Fig. 4) which vents to the atmosphere. As the flange of the face mask creates an air-tight seal around the subject's nose and mouth and as the rest of the drum is air-tight, there is a fixed mass of air inside the plethysmograph. Changes in the lung volume during breathing and speaking (see chest profile in solid and broken lines in Fig. 4) therefore cause corresponding changes in the air pressure inside the plethysmograph. These pressure changes are sampled continuously through a port in the side of the plethysmogrph, transduced by a sensitive pressure transducer, and recorded on tape along with the voice signal for later analysis.

Preliminary studies have been done with three speakers (including myself): 2 English speakers and 1 Swedish speaker. Using speech samples that would eliminate or minimize jaw movement (in order that the only volume changes recorded by the plethysmograph would be those of the chest wall), the following results have been obtained (illustrated in Figs. 5 and 6):

1. There are relatively large rapid decreases in lung volume during moments of high air flow, e.g., during aspiration, [h], and fricatives. See Figs. 5b, c, and d. These presumably represent a passive collapse of the lungs due to the rapid flow of air out of the lungs and the consequent decrease in lung pressure (= Ps). This contrasts with the case of sonorants (nasals, laterals, etc.) where the rate of lung volume decrement is the same as that for surrounding vowels. See Fig. 5a.

2. There were also moments of lesser–than–normal lung volume decrement during periods of reduced air flow, i.e., during the closure phase of some stops. See arrows in Fig. 6a and b.

3. There were momentary greater–than–normal decreases in lung volume during emphatically stress syllables. See portion delimited by broken vertical lines in Fig. 6b.

4. There need not be any obvious change in the rate of lung volume decrement on non–emphatically stressed syllables. This is easiest to see in sentences consisting entirely of sonorants since the segments themselves will cause no perturbation in the rate of air flow out of the lungs. See portion delimited by broken vertical lines in Fig. 6a and also note lack of change on stressed vowel in Fig. 5a.

In general, these results are completely compatible with those obtained by van Katwijk (1974) in his electromyographic study of the expiratory muscle activity in the speech of two Dutch speakers.

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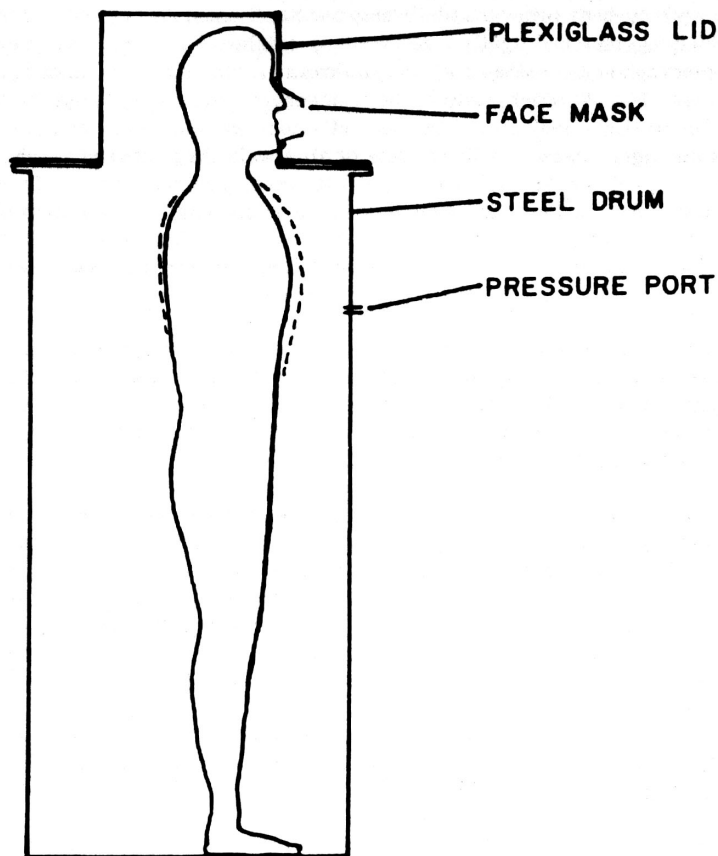
FACE MASK

STEEL DRUM

PRESSURE PORT

Figure 4.   Schematic representation of the pressure plethysmograph. The subject breathes and speaks through a face mask which makes an air-tight seal around the nose and mouth. The pressure of the air trapped inside the drum varies directly with changes in chest volume. The pressure is sampled via a port, transduced by a sensitive pressure transducer, and recorded
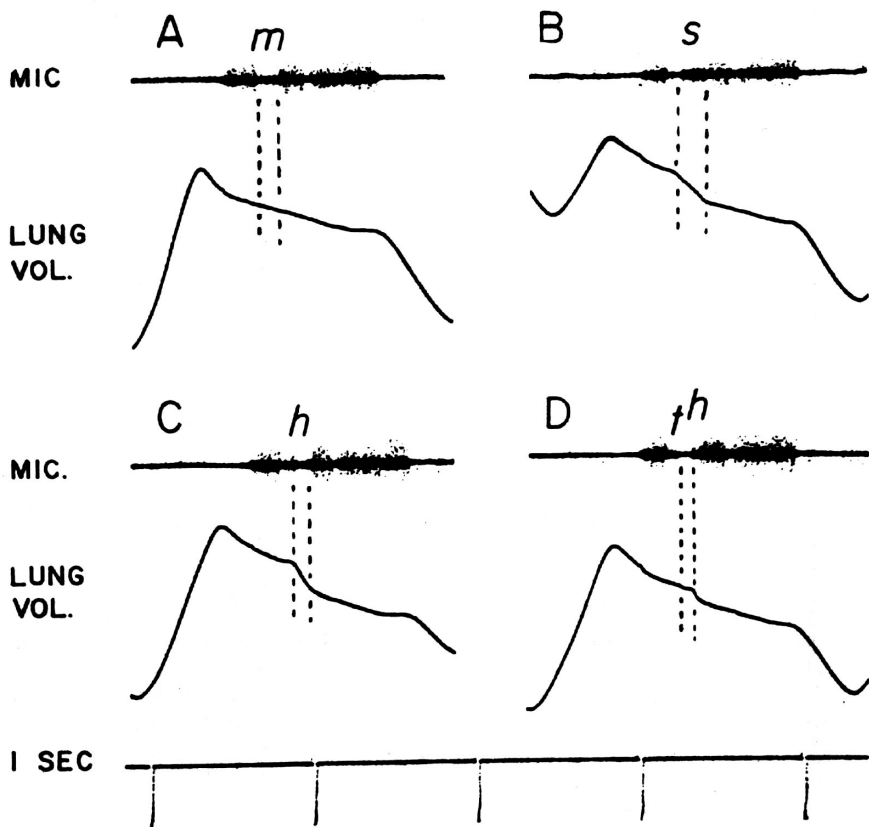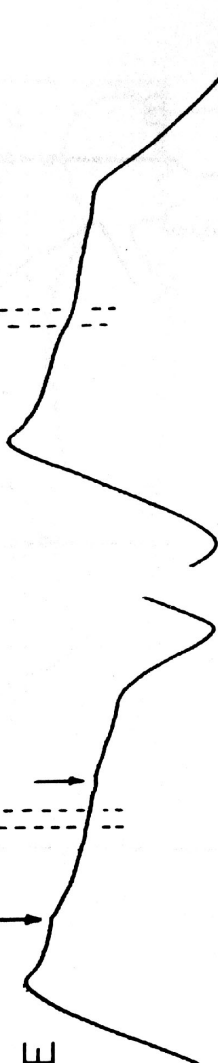
Figure 5. Representative data obtained using the plethysmograph. 5a through d show the lung volume (bottom) along with the voice signal (top) for the consonants [m, s, h, tʰ], respectively. Each of the consonants, whose onset and offset are indicated by broken vertical lines, were spoken by a male speaker of American English in the frame "deem __oon real" [dim'__unril]. (Subject LB.)

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Figure 6. Representative data obtained using the plethysmograph. 6a and b show the lung volume pattern for the utterances "deem unilluminable real" [dimʌn'ɪlumnɪblrɪl], spoken conversationally on the left and with emphatic stress on the right. The broken vertical lines delimit the stressed syllable in each utterance. The arrows mark instances of lesser-than-normal lung volume decrement during stops. (Subject LB.)

Of course, the number of subjects studied so far with this technique is quite small, and it is possible that a simple visual analysis of the plethysmograph output is not capable of revealing small but consistent changes in the slope of the lung volume curve (computer averaging of the signals is planned for the near future). Therefore these results should be viewed cautiously. Nevertheless, the data obtained so far do not support the notion that there must always be an appreciable expiratory pulse accompanying the production of ordinary (non-emphatic) stressed syllables. For emphatic stress, however, the involvement of the respiratory system is quite apparent.

ACKNOWLEDGEMENT

FOOTNOTES

[1]I use 'pitch' and 'Fo' (fundamental frequency) interchangeably in this paper.

[2]Actually, the transglottal pressure drop which, during vowels, is usually equal to Ps.

[3]In fact, Lieberman's claims were stated with so many qualifications and occasionall; in such self-contradictory ways, that it may be seriously questioned whether they represented empirical (i.e., potentially falsifiable hypotheses at all. In this paper I have attempted to interpret his hypotheses as if they were serious scientific statements. On this point, cf. also the detailed criticisms in Vanderslice 1968, 1970, Ohala and Ladefoged 1969, Ohala 1970.

[4]Even aside from what later investigations have revealed (see below), the significance and reliability of these calibrations is questionable due to the confusion over how they were obtained. As noted in Ohala and Ladefoged (1969) Lieberman claimed to have measured the corresponding values of Fò and Ps at points in the data curves where it was

> assumed that the tension of the laryngeal muscles was
> unchanged, so that the fundamental frequency was a function
> of the subglottal air pressure [Lieberman 1967:95-6].

Nevertheless, a few pages later he remarks:

> The points in Figure 4.35 [the data in question] have a
> fair amount of horizontal dispersion, which indicates that
> the laryngeal tension is not always constant throughout the
> non-terminal portion of each breath-group [102, emphasis added].

And a few pages later:

> Quantitative calculations of the relationship between
> fundamental frequency and the subglottal air pressures
> were made... These calculations showed that the tension
> of the laryngeal muscles <u>was</u> <u>relatively</u> <u>constant</u> during
> the non-terminal portions of the breath-group [107,
> emphasis added].

As Ohala and Ladefoged commented:

> This kind of self-contradiction abounds in Lieberman's
> work; and it makes it very difficult for anybody else to
> contradict him, since he can always claim that he has
> stated the opposite to what is attributed to him... Besides
> the contradictions between these quotes there is also the
> rather strange procedure of first assuming X (i.e. the
> inactivity of the laryngeal muscles) as part of the basis
> of Y (i.e., the calculated Δ Fo/ Δ Ps ratio), and then
> later taking Y as proof of X without, as far as we can
> see, any other independent supporting evidence.

Moreover, there is evidence that these calibrations were done carelessly and
inconsistently. Though trivial by itself, this evidence is important in the context of
Lieberman's strong claims since it reduces even the prima facie plausibility of some
of these claims. In his book Lieberman presents measurements forming the basis of
the estimates of Δ Fo/Δ Ps in four figures, three of which give the individual data
from his three subjects and one of which presents the combined measurements from
all the subjects. For unexplained reasons the data in the fourth figure does not
completely agree with that in the other three although it was supposedly based upon
them. In addition, although Lieberman is (commendably) perfectly explicit about how
he did the measurements on his raw data--all relevant bits of which he published--
it turns out that a replication of these procedures fails to yield exactly the same data
points as those given in his Fo vs. Ps plots. The discrepancies are not small; they
would lead to ratios of ΔFo/ΔPs as high as 28 or 33 Hz/cm H_2O--absurdly high
values that would have clearly revealed the assumption underlying the calibration,
i.e., that the level of activity of the laryngeal muscles was constant, was erroneous.

Lieberman (personal correspondence) acknowledged the mix-up in the construction
of the figures referred to but denied that any of the data in them was unjustified: that
the data points I couldn't find were in those sentences which in his book he said he
hadn't used for the calibration, namely, the yes-no questions, where it could not
safely be assumed that the laryngeal muscles were inactive!

This is not an isolated case. His Figure 4.34, though in no way crucial to the issue under discussion, also bears no relationship to the published data it was alledgedly based on. This mix-up was also acknowledged by Lieberman (personal correspondence).

[5]Liberman (1973) repeats this fallacy in his analysis of some of my own data (presented in Fromkin and Ohala 1968 and Ohala 1970): from records of utterances showing Fo, Ps, and the activity of the cricothyroid and the lateral crico-arytenoid muscles (Fig. 2 of this paper is a sample of that data) he estimates that Fo varies as a function of Ps at the rate of 12.5 Hz/cm H_2O during periods where the two muscles recorded showed little variation in level of activity. However, Lieberman chooses to ignore the fact that in Ohala 1970 other recordings of this same subject show the sternohyoid muscle active during these kinds of pitch changes (the drop in pitch at the end of declarative sentences). Moreover, it is well known that to some extent almost all of the dozen or so intrinsic and extrinsic laryngeal muscles can play a role in regulating Fo; one cannot rule out their participation either. Lieberman seems to operate according to some extreme form of positivism: those factors not represented in the instrumental records he examines (even if they may be represented in other records) simply don't exist.

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