SUBGLOTTAL PRESSURE BEHAVIOR IN SINGING AND SPEECH

Johan Sundberg, Dept. of Speech Communication and Music Acoustics, Royal Institute of Technology, Box 70014, S-10044 Stockholm, Sweden

ABSTRACT

Subglottal pressure is determined by muscular forces, elasticity forces, and gravitation and represents the major control parameter for vocal loudness. In neutral speech subglottal pressure is generally rather constant, while in emotive speech it is quite variable. In singing it is varied also with F0. As subglottal pressure affects pitch, singers need to learn a virtuosic breath control to stay in tune.

INTRODUCTION

Lately, several advances have been made in our understanding of subglottal pressures in singing. After the pioneering investigations by Proctor, Mead, and associates [1], summarized in Proctor [2], important contributions on breathing kinematics have been made by Hixon and associates [3]. During the eighties, the author had the privilege of carrying out a series of investigations of singers' breathing together with the neurologist Curt von Euler and the late phoniatrician Rolf Leanderson [4], [5], [6]. Here, the anatomy and physiology of the breathing apparatus will first be reviewed, and then certain characteristics of subglottal pressures in singing and speech will be described.

THE BREATHING APPARATUS

Overpressures of the air below the glottis, henceforth *subglottal pressure*, is produced by decreasing the volume of the rib cage. There are three different forces that influence this pressure: muscular forces, elasticity forces and gravitation. The main muscular forces are exerted by the *intercostal muscles*, the *diaphragm*, and the *abdominal wall muscles*. The intercostals join the ribs. The external intercostals widen the rib cage by lifting the ribs, and so provide an inspiratory muscle force. The internal intercostal muscles decrease the rib cage volume. The diaphragm is an inhalatory muscle inserting into the lower contour of the rib cage. When contracting, it is flattened so that the floor in the rib cage is lowered, and lung volume is increased. With the body in an upright position, the diaphragm muscle can be restored to its upward-bulging shape by means of the abdominal wall muscles. By contracting, these muscles press the abdominal content upward, into the rib cage, so that the diaphragm, the floor in the rib cage, moves upward and the lung volume is decreased. Consequently, the abdominal wall muscles are exhalatory.

The external and internal intercostals represent a paired muscle group producing inspiratory and expiratory forces. The diaphragm and abdominal wall the represent a similar paired muscle group for inhalation and exhalation. In costal breathing, the intercostals are used for respiration, and in ventricular breathing the diaphragm and abdomen are used as respiratory muscles. Mostly a combination of costal and abdominal breathing is used.

The volume of the abdominal content cannot be altered appreciably. Therefore, when the diaphragm contracts, it presses the abdominal content downward which, in turn, presses the abdominal wall outward. If the abdominal wall remains flat during inspiration, this means that only the intercostal muscles were used. An expansion of the abdominal wall during phonation is not necessarily a sign of diaphragmatic activation. It may equally well result from the increased

lung pressure that is required for phonation, because an overpressure in the lungs is transmitted downward through the diaphragm so that the subglottic pressure exerts a pressure on the abdominal wall. By contracting the abdominal wall muscles, this expansion can be avoided.

Apart from these muscular forces, there are also *elasticity* forces. The magnitude of these forces depends on the amount of air contained in the lungs, or the lung volume. The lungs always attempt to shrink, somewhat as rubber balloons, when hanging inside the rib cage. They are prevented from doing so by the fact that they are surrounded by a vacuum. The lungs therefore exert an entirely passive expiratory force which increases with lung volume. This force corresponds to a pressure that may amount to around 20 cm H_2O after a maximum inhalation and after a deep exhalation, it is only a few cm H_2O.

If the rib cage is forced to deviate from its rest volume, e.g., because of a contraction of the intercostal muscles, it strives to return to a smaller volume. Therefore, also the rib cage produces elastic forces. At high lung volume a passive expiratory force is generated that may produce an overpressure of about 10 cm H_2O. Conversely, if the rib cage is squeezed by the expiratory intercostal muscles, it strives to expand again. After a deep costal exhalation, the resulting passive expiratory force may produce an underpressure of about -20 cm H_2O.

Subglottal pressure is affected also by *gravitation*. In an upright position, the abdominal content is pulling the diaphragm downward and hence produces an inhalatory force. In supine position, gravitation strives to move the abdominal content into the rib cage and so produces an exhalatory force.

As the elasticity forces are both exhalatory and inhalatory, depending on lung volume, there is a particular lung volume, at which these passive forces are equal. This lung volume value is called the *functional residual capacity* (FRC). As soon as the lungs are forced to depart from FRC by expanding or contracting, passive forces try to restore the FRC volume.

REGULATION OF SUBGLOTTAL PRESSURE

Above we have seen that subglottic pressure is dependent on the activity in different respiratory muscles plus the lung volume dependent passive elasticity forces, plus the posture dependent influence of gravitation. The muscular activity required for maintaining a constant subglottic pressure is dependent on the lung volume because the elasticity forces of the lungs and the rib cage strive to raise or to lower the pressure inside the lungs, depending on whether the lung volume is greater or smaller than the functional residual capacity, FRC. When the lungs are filled with a large quantity of air, the passive exhalation force is great, and it generates a high pressure. If this pressure is too high for the intended phonation, it can be reduced by a contraction of inhalatory muscles. The

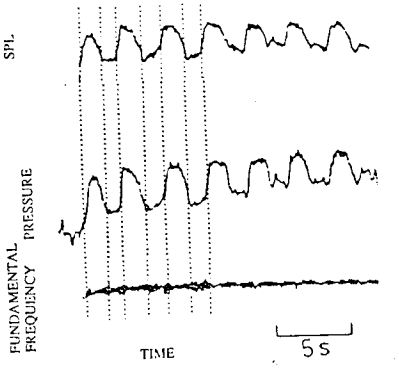

Figure 1. Variation of subglottal pressure during variation of vocal loudness. The top curve shows sound level, the middle curve esophageal pressure, and the bottom curve F0.

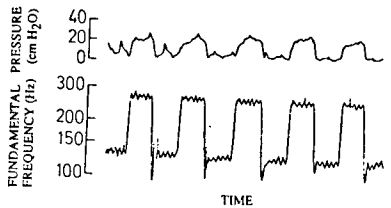

Figure 2. Variation of subglottal pressure with pitch. The graph shows a registration from a professional baritone singer performing a sequence of alternately ascending and descending octave intervals. The top curve shows esophageal pressure captured by a GELTEC pressure transducer, and the bottom curve F0. The wiggles in the esophageal pressure reflect pulse beats.

need for this activity then gradually decreases as the lung volume decreases, reaching zero at the lung volume where the elasticity forces provide the target pressure. Beyond this point the muscles of exhalation must take over more and more, thus compensating for the increasing inhalatory recoil force of the increasingly compressed rib cage.

When we speak, we generally use rather small lung volumes, typically just above FRC [7], [8]. Under these conditions, the elasticity forces are not very strong. In singing, larger portions of the vital capacity are frequently required. Thus long phrases may be initiated at very high lung volumes and end when the lungs nearly depleted [7]. Under these conditions, the elasticity forces are considerable.

Subglottal pressure in singing

Ideally, subglottal pressure is measured by inserting a fine needle into the trachea, obviously a rather intrusive method. However, it can be measured also as the mouth pressure during [p]-occlusion [9], [10], [11].

As mentioned, subglottal pressure is the main physiological parameter for variation of vocal loudness. Figure 1 illustrates this. It shows the sound level and the underlying subglottal pressure in a singer who alternates between *subito forte* and *subito piano* at constant pitch. Sound level and subglottal pressure change quickly and in synchrony between two rather stationary values such that square-wave-like patterns emerge.

In singing, variation of subglottal pressure is required not only for loudness variation but also with pitch [12]. When we increase pitch, we stretch the vocal folds. It seems that stretched vocal folds require a higher driving pressure than more lax vocal folds [13]. Figure 2 illustrates this pitch dependence in terms of a recording of a singer performing a series of alternating rising and falling octave intervals. It can be observed that the higher pitch was produced with a much higher pressure than the lower pitch. The wrinkles in the pressure curve represent the singer's heart beats and the undulations in the F0 curve correspond to the vibrato. Figure 3 illustrates the combined dependence of subglottal pressure on loudness and pitch in a singer. As subglottal pressure affects

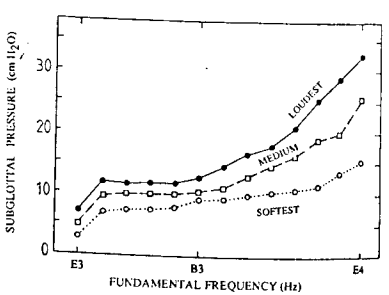

Figure 3. Illustration of the dependence of subglottal pressure on pitch and loudness. Pressures were captured as the oral pressure during [p]-occlusion for the tones in ascending chromatic scales sung at low, middle and high vocal loudness by a professional tenor. From Cleveland & Sundberg, [12].

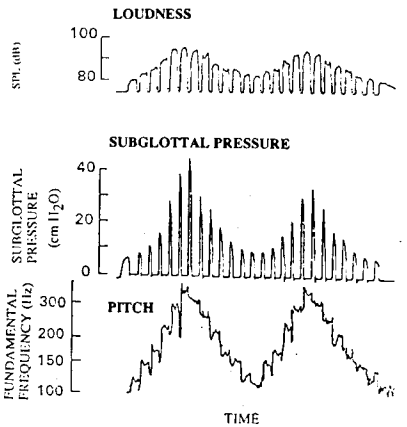

Figure 4. Pitch and loudness dependent variation of subglottal pressure in singing. The top, middle and bottom curves represent sound level, oral pressure during [p]-occlusion, and F0 in a professional baritone singer performing an exercise with an ascending triad on the pitches of a major tonic chord up to the duodecime followed by a descending dominant seventh triad.

pitch, an error in the subglottal pressure is manifested not only as an error in loudness, but also as an error in pitch. Therefore singers must tune their subglottal pressure quite accurately. Accordingly, one finds very well-formed subglottal pressure patterns in proficient singers. Figure 4 illustrates this. It shows the pressures produced by a baritone singing an ascending triad on the tonic chord and a descending triad on the dominant seventh chord. Note that the singer did not give the top pitch the highest pressure. Instead, the peak pressure is given to the first note after the top note. At this note the new dominant chord appears which would represent the musical peak of this phrase. Consequently, the singer gives this note the main stress [14].

The skill required for an accurate reproduction of this exercise is obviously very high, and it is even greater if the tones are sung staccato rather than legato. In staccato, the vocal folds must open the glottis during the silent segments. For this to be possible without wasting air, subglottal pressure must be reduced to zero during the silent intervals between the tones. As a consequence, the singer has to switch from the target value, that was required for the pitch, down to zero during the silent interval, and then up to the new target value which is different from the previous one. A failure to reach the target pressures is manifested as a pitch error. This pitch error becomes quite substantial in loud singing, particularly at high pitches. From the point of view of breath and pitch control, this exercise is clearly virtuosic.

Subglottal pressure in speech

Subglottal pressure during speech has been studied in several investigations (for an excellent overview, see Ohala, 1990 [15]). Earlier it was believed that in speech each syllable was produced with a subglottal pressure peak. However, this was not confirmed in later investigations. Rather, subglottal pressure has been found to be rather smooth and constant, at least in neutral speech. Occasional peaks occur but are presumably caused by downstream variations in flow resistance, e. g., during consonant production [15]. In emphatic or emotive speech, on the other hand, subglottal pressure peaks are frequently observed. An example comparing the same subject's neutral and emphatic speech is shown in Figure 5. In neutral speech, void of emphatic stress, it seems sufficient to signal stress by F0 gestures and syllable duration while in emphatic and emotional speech also subglottal pressure is recruited.

In normal speech, changes in overall vocal loudness are generally associated with shifts in overall F0; the louder the speech, the higher the mean F0. This

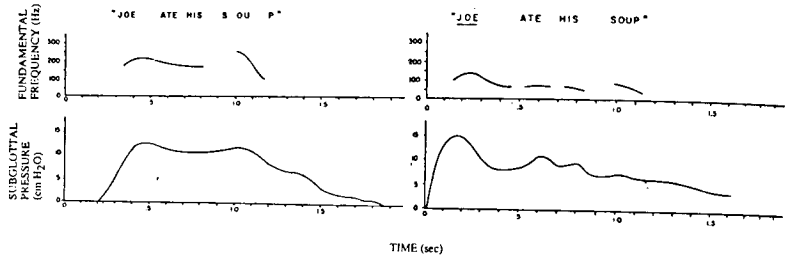

Figure 5. F0 and subglottal pressure (upper and lower curves) during neutral and emphatic speech. The underlined words were emphasized. Emphasis is realized by increase of subglottal pressure. After Lieberman [16].

covariation has been analyzed, revealing high degrees of correlation [17]. The average growth was found to be about 0.4 semitones per dB increase of equivalent sound level. From a measured sound level change, it is possible to roughly estimate the underlying increase in subglottal pressure; a doubling of subglottal pressure leads to an increase in sound level of approximately 9 dB [18], [13]. The effect of a subglottal pressure increase on pitch can also be estimated: on the average, a 1 cm H_2O rise in subglottal pressure results in a F0 increase of about 4 Hz [19], [20]. According to Gramming & al. [17] the postulated pressure increase could indeed explain all of the increase in F0. Thus, the

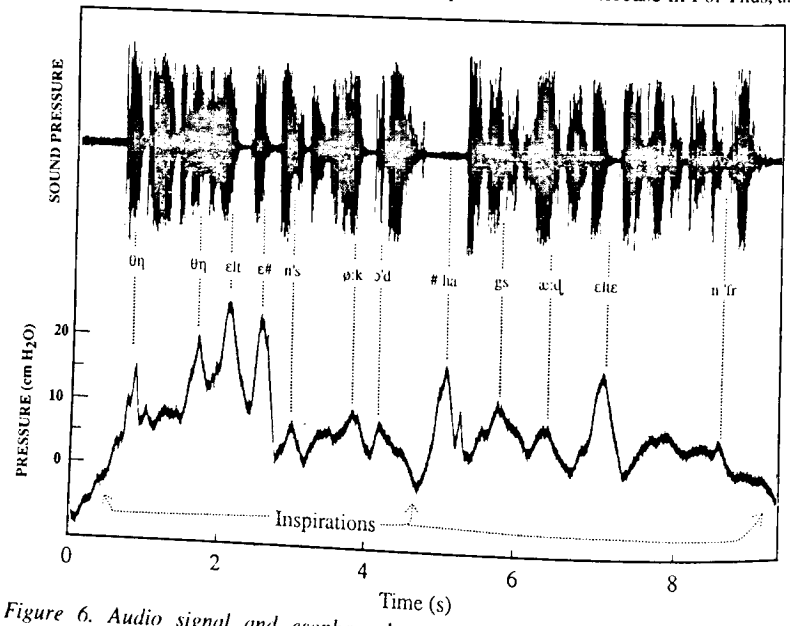

Figure 6. Audio signal and esophageal pressure (upper and lower curve) of a professional actor reading a poem in Swedish as during a stage performance with an audience. The sounds produced during the pressure peaks are shown between the curves. From Sundberg & al [21].

subjects did not seem to bother to use the pitch-raising musculature in order to raise their voice pitch in loud reading. Instead, the pitch just increased passively because subglottal pressure was raised.

Figure 6 shows a graph from an investigation of a professional actor [21]. The esophageal pressure was measured while the subject read a poem as during a theatre play. Several short subglottal pressure peaks can be seen, unexpectedly appearing during the production of voiced consonants. It seems difficult to explain these high pressure peaks as the results of downstream variations of airflow resistance. A more plausible explanation is that they were deliberately produced with the purpose to increase the audibility of the consonants. Unfortunately, only one actor was studied.

CONCLUSIONS

There are great differences in the subglottal pressure behavior in speech and singing. In neutral speech, subglottal pressure is used mainly for control of the overall vocal loudness and is thus basically constant. In singing, subglottal pressure is tailored with regard to both pitch and loudness and must therefore be varied within wide limits. As pitch may change at intervals of 200 ms or shorter in singing, subglottal pressure must be changed quickly. Furthermore, as a change in subglottal pressure affects F0, singers also need to match the target subglottal pressures quite accurately. In speech, loudness and pitch are typically interdependent, so that a rise in loudness is associated with a rise in mean F0. In neutral speech a narrow range of lung volumes just above FRC is used and hence the elasticity forces contributing to subglottal pressure are moderate. In singing these forces represent an important factor, since wide ranges of lung volumes are used. Thus, the demands raised on the breathing apparatus are much higher in singing than in speech.

REFERENCES

[1] Bouhuys, A., Proctor, D., & Mead, J. (1966): "Kinetic aspects of singing," *J.Appl.Physiol.* **21**, pp. 483-496.

[2] Proctor, D. (1980): *Breathing, Speech and Song*, Springer Verlag, New York.

[3] Hixon, T. (1987): *Respiratory Function in Speech and Song*, Taylor & Francis Ltd., London.

[4] Leanderson, R., Sundberg, J., & von Euler, C. (1987): "Role of the diaphragmatic activity during singing: a study of transdiaphragmatic pressures," *J.Appl.Physiol.* **62**, pp. 259-270.

[5] Sundberg, J., Leanderson, R., & von Euler, C. (1989): "Activity relationship between diaphragm and cricothyroid muscles," *J. Voice* **3**, pp. 225-232.

[6] Sundberg, J. (1987): *The Science of the Singing Voice*, N. Ill. Univ. Press, DeKalb, IL.

[7] Watson, P. & Hixon, T. (1985): "Respiratory kinematics in classical (opera) singers," *J.Speech & Hear.Res.* **28**, pp. 104-122.

[8] Winkworth A, Davis P, Adams R & Ellis, E (in press) "Breathing patterns during spontaneous speech", to be published.

[9] van den Berg, Jw. (1962): "Modern research in experimental phoniatrics," *Fol.Phoniat.* **14**, pp. 81-149.

[10] Rothenberg, M. (1968): "The breath-stream dynamics of simple-reased-plosive production," *Bibliotheca Phonetica* No. 6.

[11] Smitheran, J. & Hixon, T. (1981): "A clinical method for estimating laryngeal airway resistance during vowel production," *J. Speech & Hear. Disorders* **46**, pp. 138-146.

[12] Cleveland, T. & Sundberg, J. (1985): "Acoustic analysis of three male voices of different quality," pp. 143-156 in A. Askenfelt, S. Felicetti, E. Jansson,

& J. Sundberg, eds.: *Proc. Stockholm Music Acoustics Conference (SMAC 83): Vol. I*, Royal Swedish Academy of Music, Publ. No. 46:1, Stockholm.

[13] Titze, I. (1989): "On the relations between subglottal pressure and F0 in phonation," *J.Acoust.Soc.Am.* **85**, pp. 901-906.

[14] Sundberg, J. (1989): "Synthesis of singing by rule," pp. 45-55 and 401-403 in (M. Mathews & J. Pierce (eds.) *Current Directions in Computer Music Research*, System Development Foundation Benchmark Series, The MIT Press, Cambridge, MA:

[15] Ohala J (1990) "Respiratory activity in speech", in W Hardcastle & A Marchal, ed.s: Speech Production and Speech Modelling, Dordrecht, NL: Kluwer Academic Publishers, 23-53.

[16] Lieberman P. (1967) *Intonation, perception, and language.* Cambridge, MA: The MIT Press, Research Monograph No 38.

[17] Gramming, P., Sundberg, J., Ternström, S., Leanderson, R., & Perkins, W.H. (1988): "Relationship between changes in voice pitch and loudness", *J. Voice* **2,** pp. 118-126

[18] Fant G. (1982): "Preliminaries to analysis of the human voice source", *Speech Transmission Laboratory, Quarterly Progress and Status Report.* No. 4, pp. 1-27. Royal Institute of Technology, Stockholm.

[19] Baer, T. (1979): "Reflex activation of laryngeal muscles by sudden induced subglottal pressure changes," *J.Acoust.Soc.Am.* **65**, pp. 1271-1275.

[20] Titze, I. (1991): "Mechanisms underlying the control of F0," pp. 129-138 in (B. Hammarberg & J. Gauffin, eds.) *Vocal Fold Phyusiology. Acoustic, Perceptual, and Physiological Aspects of Voice Mechanisms,* Singular Press, San Diego, CA.

[21] Sundberg J, Elliot N, Gramming P & Nord L (1993) "Short-term variation of subglottal pressure for expressive purposes in singing and stage speech. A preliminary investigation", *Journal of Voice 2*, 227-234.