Breath sounds in the clinical assessment of airflow obstruction

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Bohadana, A. B., Peslin, R., and Uffholtz, H. (1978). *Thorax*, 33, 345-351. Breath sounds in the clinical assessment of airflow obstruction. In a group of 34 inpatients showing varying degrees of airflow obstruction we studied the relationship between breath sound intensity (BSI) and abnormalities of lung function. The BSI was evaluated by chest auscultation to provide a score, in a manner similar to that described by Pardee *et al.* (1976), and was found to correlate closely with indices of airflow obstruction or their logarithms such as specific conductance ($r=0.759$), maximal expiratory flow at 50% of vital capacity ($r=0.790$), forced expiratory volume in one second ($r=0.768$), and forced expiratory volume to vital capacity ratio ($r=0.860$). Correlations with lung volumes, although statistically significant, were weaker. Multiple correlation studies showed that BSI score correlated independently with indices of both airflow obstruction and lung distension.

In our experience, BSI score can be useful not only in the detection but also the quantification of airflow obstruction, although its predictive power is impaired in subjects with associated restrictive disorders. It can also fail to detect mild, pure airflow obstruction.

It is a current concept that the severity of functional impairment in patients with airflow obstruction is poorly related to physical signs and symptoms. Indeed, although patients with moderately severe airflow obstruction usually exhibit several symptoms and signs, assessment of obstruction on a clinical basis can be misleading and subjects with severe functional impairment may be entirely overlooked, whereas normal subjects may be misdiagnosed (Schneider and Anderson, 1965). Other studies have emphasised the relative unreliability of symptoms and signs in the evaluation of airflow obstruction, acute or chronic. Thus McFadden *et al.* (1973) found that dyspnoea and wheezing were inaccurate in detecting severe functional impairment in subjects with acute asthma. More importantly, evidence of considerable functional derangement during recovery was present in asymptomatic subjects. Godfrey *et al.* (1970) examined the correlation between several symptoms and signs and functional measurements in patients with stable, chronic airflow obstruction, and found that forced expiratory time (which can be looked on more as a physiological test) was the only sign that directly reflected the obstruction; all the others were probably related to factors such

as secondary effects of the obstruction, age, or the duration of the illness.

Pardee *et al.* (1976) suggested recently, however, that breath sound intensity (BSI) heard at the chest could be useful in the clinical evaluation of airflow obstruction. It seems evident from their data that a clearly reduced BSI is invariably associated with an abnormal forced expiratory volume in one second (FEV₁). They also suggested that, when combined with other symptoms and signs, BSI may be an even better predictor of ventilatory function.

Considering its simplicity and potential usefulness, we decided to investigate BSI further to provide information about its possible relationships with measurements of lung function other than FEV₁ and to determine more precisely the limitations of its use in daily clinical practice.

We have therefore examined groups of adult inpatients and correlated BSI with pulmonary function measurements.

Methods

SUBJECTS

Thirty-four adult inpatients were examined by one

of us (ABB). They were unknown to the observer, and practically all were referred to our department by their private physician because of respiratory complaints. They were examined immediately after arrival and no clinical criteria of selection were adopted, except for those who had undergone pneumonectomy. This would, theoretically, enable us to see patients with different degrees of obstruction with or without associated restrictive disorders. Nevertheless, patients incapable of performing the respiratory manoeuvres correctly, thereby preventing a good BSI evaluation, were rejected from the study. During the experiments only two subjects did not meet this requirement.

The mean age (years $\pm$ SD), height (cm $\pm$ SD), and weight (kg $\pm$ SD) were 52.1 ($\pm$ 13.4), 166.4 ($\pm$ 5.1), and 69.1 ($\pm$ 14.5) respectively. There were 24 men and 10 women of whom 11 were smokers (10 men and 1 woman), 13 were ex-smokers (11 men and 2 women) and 10 non-smokers (3 men and 7 women).

BSI EVALUATION

BSI evaluation was carried out on seated patients in a fashion similar to that described by Pardee *et al.* (1976). Sound intensity was noted on a grading scale, which was essentially the same as theirs, as follows: 0—absent breath sound, 1—barely audible breath sound, 2—faint but definitely audible breath sound, 3—normal breath sound, and 4—louder than normal breath sound.

Auscultation was performed in the following chest zones, bilaterally—upper anterior, midaxillae, and posterior base. Subjects were asked to perform fast, deep inspirations from residual volume to generate as loud a sound as possible while breathing through the mouth. Whenever necessary they were taught to breathe without making too much noise at the mouth. They were allowed to rest between the examination of two zones whenever they looked tired and showed a tendency not to mobilise their best inspiratory flows. The final BSI score was obtained by taking the sum of the values of each individual zone, and care was taken to note the latter separately, since an identical final BSI score in different subjects was not incompatible with considerable interzonal variations. Possible final BSI score ranged from 0 to 24.

In a preliminary study BSI inter-observer variability was determined by the separate examination of 20 unknown inpatients and outpatients independently by two of us (ABB and HU) at five-minute intervals. Both observers used the same stethoscope. A good general agreement was arrived at by both examiners despite a slight

tendency for one of them (ABB) to overestimate BSI, as compared to the other (Fig. 1). There was some disagreement when, in a given subject, scores from the individual zones were compared. Discrepancies between observers were noticed in 25 zones (out of 120) and ranged from 1 point (24 cases) to 2 points (1 case).

Among the possible reasons underlying these differences one must be remembered because its role may be important—namely, slight differences between the examiners' levels of sound reference. Taking this into account it seems wiser to consider as abnormal a BSI ≤ 16 rather than < 18 .

Fig. 1 Correlation between BSI score as evaluated by two observers independently (observer 1, ABB; observer 2, HU). Slope of regression of y on x (not represented) is not significantly different from zero at 5% level.

PULMONARY FUNCTION TESTS

Before undergoing pulmonary function tests, subjects were allowed a short period of rest.

Lung volume, and its subdivisions, and airway resistance were measured using a constant-volume body-plethysmograph (Fenyves et Gut Medizinische Elektronik). Airways resistance was measured during quiet breathing, converted to its reciprocal, the conductance, and expressed as specific conductance ($SGaw = Gaw/FRC$). Functional residual capacity (FRC) was determined by the method of Dubois *et al.* (1956). Total lung capacity (TLC) was obtained by adding the inspiratory capacity. Results were expressed as a percentage of the pre-

dicted values of Amrein *et al.* (1970). Maximal expiratory flow-volume curves were obtained when the subjects performed several (four at least) forced vital capacity manoeuvres while sitting in the body-box that now communicated through a metal screen with the exterior. Pressure drop through the screen was proportional to flow, whose integrated signal allowed, after corrections for gas compressibility, recording of volume. Flow at the mouth was measured with a calibrated Fleisch no. 3 pneumotachograph and displayed against volume on an x-y recorder (Bryans). Flows were measured at 50% of the vital capacity (VC) and the results expressed as a percentage of the predicted values of Knudson *et al.* (1976). After another short period of rest, spirometric measurements were carried out using a 9-litre watersealed spirometer (G. Boullitte). From the best of at least three slow inspiratory or expiratory vital capacity manoeuvres (Hutchinson *et al.*, 1973) and three forced expiratory manoeuvres, the following indices were derived: slow VC, FEV₁, and FEV₁/VC.

After correction to BTPS, values were expressed as a percentage of the predicted values of the

European Coal and Steel Community (1971), except for FEV₁/VC ratio, which was expressed as the observed value.

Results

No significant correlations were found between BSI score and age, weight, or height. Although they were not very close, correlations between the BSI score and the lung volumes were statistically significant (Fig. 2).

When we plotted the BSI score against several indices of obstruction or their logarithms, a strong correlation emerged whatever the index and, whereas the relationship was linear when the BSI score was plotted against FEV₁ and FEV₁/VC, it was curvilinear when we used indices such as V max₅₀ VC and SGaw (Fig. 3). This implies that whereas the BSI score begins to deteriorate at relatively high values of FEV₁ and FEV₁/VC ratio it does so only at lower values of V max₅₀ VC and SGaw.

It was also interesting to test, by multiple linear regressions, whether BSI score was better explained by lung distension and airway obstruction

BSI Score

Fig. 2 Correlations between BSI score and lung volumes. Solid lines represent regression of y on x, dashed lines represent regression of x on y.

BSI Score

Fig. 3 Correlations between BSI score and four indices of obstruction or their logarithms.

rather than by each of these factors taken alone. For this, we selected functional indices that were not significantly correlated between themselves, that is TLC for distension and $\dot{V}$ max₅₀ VC or SGaw for obstruction. As shown in Table 1 the multiple regressions are highly significant with r values substantially higher than those obtained from simple regression (Figs. 2 and 3); all slope coefficients are significantly different from zero. BSI score may, therefore, be considered as independently correlated to both distension and obstruction.

Discussion

The loudness of breath sounds heard at the chest depends mainly on two factors, both or either of which can modify the BSI—namely, the sound intensity at the site of generation and the transmission properties of the aerated lung and chest wall. Although an extensive review of the mechanisms governing both factors is beyond the scope of this study, some physiological considerations are necessary for a better understanding of our results.

Table 1 Multiple linear correlations between BSI score and indices of airflow obstruction and lung distension

x	y	$r_{x,y}$	P_1	a	b	c	see	$r_{z,xy}$	P_2
TLC % pred	log $\dot{V}$ max 50% CV (% pred)	-0.095	$P > 0.1$	9.74	-0.055	7.77	1.73	0.862	< 0.001
TLC % pred	log SGaw (% pred)	-0.271	$P > 0.1$	7.07	-0.036	7.19	2.09	0.790	< 0.001

x, y = independent variables.

$r_{x,y}$ = correlation coefficient of the independent variables. P_1 = corresponding statistical significance.

a, b, c = coefficient of the multiple regression $Z = a + bx + cy$ where Z is the BSI score.

see = standard error of the estimate of the regressions.

$r_{z,xy}$ = correlation coefficients of the multiple regressions. P_2 = corresponding statistical significance.

SOUND GENERATION

It is generally accepted that, in healthy subjects, breath sounds are generated in the central airways as a result of turbulent airflows (Forgacs *et al.*, 1971; Banaszak *et al.*, 1973). Indeed, there is good evidence that turbulence exists in many points of the tracheobronchial tree, even during quiet breathing. Thus Olson *et al.* (1972) using casts of human airways were able to show that turbulence is present in the trachea and proximal bronchi at flows as low as 200 ml s^{-1} . On the other hand, calculations by Pedley *et al.* (1977) show that, for values of inspiratory flows around 2 l s^{-1} , turbulence is present in the trachea and may persist up to the fourth or fifth bronchial generation. Because sounds generated by turbulence are poorly transmitted however, their intensity is greatly decreased, and it is doubtful whether they are heard at the chest wall (Olson, 1973). Recent work by Ploy-Sang-Song *et al.* (1977) suggests that vesicular breath sounds are not simply transmitted sounds from central airways but are generated more peripherally (in sites between bronchioles 3 mm diameter and alveoli) and are related to non-turbulent, density-dependent airflow.

Regardless of the site of sound generation and of its precise production mechanisms, BSI heard at the chest is influenced by the magnitude of the inspiratory flow as well as by the number of patent airways leading to the zone of auscultation. The influence of flow on BSI score can be easily demonstrated, and we did so in a group of five healthy, well-trained subjects from the laboratory. Each subject was asked to perform a series of inspiratory manoeuvres at constant flows while breathing through a pneumotachograph. Flows were kept constant with the aid of an oscilloscope. The examiner (ABB) was not aware of the flows being generated, which were freely chosen by the subjects from five preset values ranging from 0.5 l s^{-1} to 3 l s^{-1} . As in the patients, BSI score was also assessed during maximal inspiratory flows. As shown in Fig. 4, BSI score was found to increase linearly with the increasing flows. This agrees well with the observations of Leblanc *et al.* (1970), although these authors' experiments were somewhat different, as breath sound intensity and inspiratory flows were recorded graphically at given lung volumes.

Unfortunately, we were not able to evaluate the influence of flow on BSI score successfully in patients because, in general, they were not able to generate constant flows often and long enough to allow a good BSI score assessment. Also, their relatively small inspiratory flows did not allow us to study BSI in a range of flows as wide as in

Fig. 4 BSI score at several constant inspiratory flows in five healthy subjects. At a flow of 0.5 l s^{-1} breath sounds were inaudible in basal regions in all subjects.

normal subjects. Nevertheless, there is no reason to believe that in diseased subjects BSI would not be flow-dependent. In fact we think that decreased inspiratory flows, probably associated with a diminished number of patent airways, is one of the major factors determining the decrease in the BSI score found in our subjects with airflow obstruction. Thus it is not surprising that good correlations were observed between the BSI score and various indices of obstruction, although the latter were obtained during forced expirations.

SOUND TRANSMISSION

As recently emphasised by Ploy-Sang-Song *et al.* (1977) sound transmission is important and is considered separately from the sound intensity at the source only for the sake of clarity; indeed they are closely related and BSI at the chest wall will depend on how they interact. Thus the sound must travel from the site of generation to the point of auscultation over the chest through the aerated lung tissue, which acts as a selective band-pass filter (Banaszak *et al.*, 1973). In conditions such as lung distension the distance to be travelled by the sound increases, and consequently sound energy and BSI will decrease. If one considers that linear distances in the lungs change as the cube root of the volume changes, when the lung volumes increase by a factor of three (a rather exceptional finding), the distance between the site of sound generation and the point of auscultation will increase by only 45%. This does not mean necessarily that BSI score will decrease in the same proportion. Indeed, calculations using the multiple linear regressions show that, in the above circumstance, the BSI score will be severely decreased

even if flows are kept in the normal range. In clinical practice increases in lung volume by a factor of two or three are less common than decreases in flow by similar factors. Because lung distension was less pronounced than decrease in flows, its contribution to a diminished BSI score was smaller in most of our patients: to take the average figures in the group, the increase in TLC (112.9% pred) would be responsible for a decrease in BSI score of less than one point, while the decrease in $\dot{V}_{\max_{50}}$ VC (24.6% pred) would lead to a decrease of around four points.

It should be noted that while transmission effects add to those of decreased flows in subjects with lung distension they can counterbalance the effects of low flows in subjects with associated restrictive disorders. An illustration is given in Table 2 where two subjects with lung distension and restrictive disease (histiocytosis X) and who show similar degrees of airflow obstruction are compared. Using the multiple regression equations (Table 1) the expected BSI score difference (2.5 points) agreed well with the observed one, suggesting that the latter is mainly due to differences in lung volumes and consequently in sound transmission properties. Although it is possible that the transmission effect is mostly a matter of longer or shorter distance between the site of sound generation and the point of auscultation, the quality of the lung parenchyma may also influence the BSI. It is well known from clinical practice and from published reports (McKusick *et al.*, 1955) that sound-conducting properties are enhanced by consolidated lung tissue and that in such circumstances bronchial breath sounds can be heard at the chest.

Table 2 Comparison of two patients with restrictive disorder and lung distension who show similar degrees of airflow obstruction

Indices	Subject with restrictive disorder	Subject with lung distension
BSI score	18	15
TLC (% pred)	69	135
RV (% pred)	118	248
FEV ₁ (% pred)	45.4	59.5
SGaw (% pred)	53	53.4
$\dot{V}_{\max_{50}}$ VC (% pred)	20.4	36.2

BSI differences between two subjects with similar degree of airflow obstruction but different lung volumes can be accounted for by transmission properties (see text).

Breath sound intensity as evaluated in this study can be used as a valuable clinical means of assessing airflow obstruction. It appears to be quite specific, and notation using a grade rating scale contributes to reduce inter-observer disagreement

as pointed out by Pardee *et al.* (1976). Nevertheless, a short period of training seems desirable if comparable data are to be collected by different physicians. It must also be remembered that both physicians' and patients' motivation is most important if a good BSI evaluation is to be obtained and the examiner must be sure that the patient is generating his best flows. BSI sensitivity seems to be only moderate, and it can fail to detect mild pure airflow obstruction as well as obstruction in subjects with associated restrictive disorders.

The BSI score should obviously not replace functional measurements, but it can be extremely useful when a lung function laboratory is not available or even when, despite a good laboratory, the patient cannot for some reason undergo repeated functional assessment. Furthermore, it can be very useful in medical teaching as it is related to several physiological and pathophysiological aspects of the respiratory system.

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