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# Quantitation of Wheezing in Acute Asthma\*

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Pulmonary sounds were recorded before and after bronchodilator treatment in 20 patients with acute attacks of asthma. Analysis of the sounds showed that improvement in the forced expiratory volume in one second was associated with the following two changes in the sound signal: (1) the proportion of the respiratory cycle occupied by wheeze  $(T_{\nu}/T_{\rm tot})$  ratio) was reduced from 86 percent to 31 percent on average; and (2) the sound frequency of the highest pitched wheeze was also reduced, from a mean of 440 Hz to 298 Hz.

uring an acute attack of asthma, auscultation of the patient's lungs is likely to be repeated many times. The severity of the attack, the improvement or deterioration, and the response to treatment are judged by several criteria, such as the patient's symptoms, physical findings, pulmonary function tests, and blood gas analysis; but repeated auscultation plays an important part in assessment. Auscultation is simple, is readily available, requires only a stethoscope, and entails no risk or inconvenience to the patient. Its value is diminished by difficulty in interpreting the sounds we hear. Their complexity strains our memory. Uncertainty about which characteristics are important leads to a dependence on overall impressions. This might with advantage be replaced by concentration on a few significant elements, if these could be identified.

Methods are now available for the recording, analysis, and quantitation of pulmonary sounds. These methods allow measurement of the physical correlates of these perceived sounds and separation of the elements which combine to form them. We therefore studied pulmonary sounds in patients before and after bronchodilator treatment of acute asthma, in order to identify which characteristics changed with reduction in airflow obstruction.

Our objective was to identify those features of the wheezing sounds themselves which changed as a result of effective bronchodilator treatment. We did not, in this study, make any effort to relate these sounds to airflow rates, to pulmonary volumes, or to respiratory rate, all of which are likely to change with relief of airflow obstruction. Asthmatic subjects presenting in an acute attack were studied, which required a simple portable method of recording sounds without delaying treatment.

Patients with a known history of asthma who presented with an acute exacerbation to the emergency room at the University of Cincinnati University Hospital were invited to participate in the study. If they chose to do so after being informed of its purpose and design, their written consent was obtained. In addition to the routine history and physical examination and other tests considered to be indicated by the responsible physician, those admitted to the study had pulmonary function tests, and pulmonary sounds were recorded on magnetic tape in standard fashion.

Pulmonary function tests were performed using a 9-L water-sealed spirometer (Collins). The forced vital capacity (FVC), forced ex-

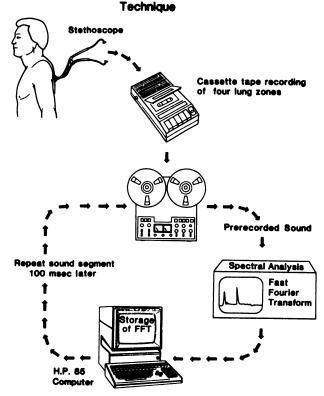


FIGURE 1. Pulmonary sounds are auscultated with electronic stethoscope over four quadrants of chest. Pulmonary sounds are recorded on portable cassette recorder. These recordings are rerecorded on computer-controlled tape recorder. A 250-msec segment of sound is played into spectrum analyzer. Sound is analyzed using Fast Fourier Transform (FFT) technique over spectral range of 1 to 1,000 Hz. Transformed data are stored, and next segment of sound is analyzed. Sound segments are 100 msec apart, giving 60 percent overlap of analysis. After 25 to 30 segments are analyzed (2.5 to 3.0 seconds, time of respiratory cycle), plot of wave forms is produced, as shown on Figures 2, 4, and 5.

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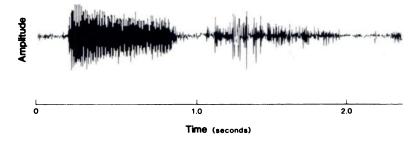
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MATERIAL AND METHODS

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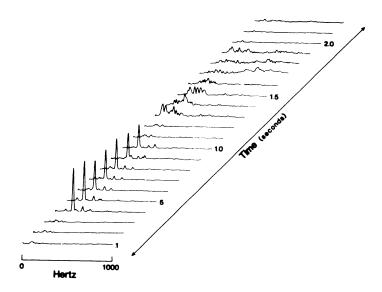


FIGURE 2. Time-amplitude wave form is shown at top. Burst of activity is characteristic for wheeze. Same sound segment was analyzed as described in Figure 1. Each line represents 250-msec sound, analyzed over spectral range from 1 to 1,000 Hz. Line 1 represents beginning of expiration. On line 4, peak is identified at 250 Hz. This peak corresponds to wheeze heard on auscultation and seen on time-amplitude wave form. Peak is seen on line 4 through 10. This means wheeze was present for 700 msec. Total duration of breath was 2.4 seconds, so T<sub>w</sub>T<sub>tot</sub> is 0.292. Amplitude of peak can be measured, and this is linearly related to intensity of sound. We can measure frequency, intensity, and duration of wheezes.

piratory volume in one second (FEV<sub>1</sub>), and peak expiratory flow rate were measured and were accepted as valid only if replicate measurements varied by less than 10 percent.

Pulmonary sounds were recorded using an electronic stethoscope (3M) and a portable cassette recorder (GE model 3-5140A), as shown in Figure 1. Auscultation was performed over four areas posteriorly: the right apex, left apex, right base, and left base. Cardiac sounds could not be heard in these areas. At least five tidal breaths were recorded in each area at each testing period.

Patients were then treated with a sympathomimetic bronchodilator, either 0.3 ml of a 1/1,000 solution of epinephrine subcutaneously or 0.5 mg of terbutaline as an aerosol. The patients were reexamined 15 to 30 minutes after treatment, and of pulmonary function tests and recording of pulmonary sounds were repeated.

Pulmonary sounds from the same site before and after bronchodilator treatment were compared, choosing the site with the best signal-to-noise ratio. The pulmonary sounds to be analyzed were rerecorded on one channel, and an electronic signal to mark the beginning of expiration was recorded on another channel of a seventrack remote-control FM tape recorder (Ampex model PR50). The beginning of expiration could be identified easily by ear from the recorded sound signal. Segments of the sound signal, each 250 msec in duration, were analyzed in sequence, starting with the beginning of expiration and continuing with segments each starting 100 msec later than its predecessor and thus overlapping it by 60 percent. At least 25 segments were analyzed for each respiratory cycle, depending upon its duration. Each segment was digitized at 2,048 samples per second, and the frequency spectrum from 0 to 1,000 Hz, with a band-width of 6 Hz, was obtained using Fast Fourier Transformation (Hewlett-Packard spectrum analyzer 3582A). The frequency domain data were stored in the controlling computer (Hewlett-Packard 85) for subsequent graphic display.

Figure 2 shows an example of the method of display, with the corresponding time-amplitude plot for comparison. Twenty-five

segments of sound, each 100 msec later than the preceding segment, are shown from lower left to upper right. Each segment is shown as a sound spectrum, from 0 to 1,000 Hz on a linear scale horizontally. Power at each frequency is represented vertically, also on a linear scale.

The sound signal was monitored by ear as it was being analyzed. It was noted that when wheezes were audible, sharp peaks were present in the sound spectra of the corresponding segments. Examples of these peaks are seen in Figure 2 in the segments from 0.4 to 1.0 second. The height of each peak is related to the sound intensity at that frequency for that segment, and thus the intensity,

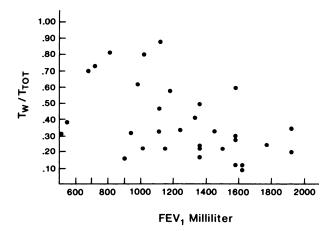


FIGURE 3. Linear relationship between  $FEV_1$  and duration of wheeze over total breath cycle is shown. Relationship is significant (r=0.459; p<0.01). Two values with  $FEV_1$  of less than 600 ml have shorter wheeze than expected. This may represent quiet chest of severely ill asthmatic subject. If we consider only those patients with  $FEV_1$  of more than 600 ml, r=0.593 (p<0.001).

Table 1-Data from 12 Patients with Wheeze after Treatment

Case	Before Bronchodilators			After Bronchodilators				
	FEV,		Highest Frequency, Hz	FEV <sub>1</sub>	T_/T <sub>tot</sub>	Highest Wheeze, Hz	Change	
		$T_{w}/T_{tot}$					$\Delta FEV_1$	$\Delta T_{\star}/T_{tot}$
1	0.55	39	460	0.90	16	330	±0.35	-23
2	0.68	70	310	1.15	22	210	$\pm 0.47$	<b>-48</b>
3	0.72	72	420	0.98	62	220	±0.26	-10
4	0.81	81	580	1.19	58	300	$\pm 0.38$	-23
5	0.94	32	260	1.36	24	320	$\pm 0.42$	- 8
6	1.02	80	290	1.36	23	280	$\pm 0.34$	-57
7	1.11	47	430	1.50	22	250	±0.39	- 25
8	1.11	88	470	1.58	28	330	$\pm 0.47$	-60
9	1.24	33	710	1.58	12	470	$\pm 0.34$	-21
10	1.36	50	400	1.58	30	390	$\pm 0.22$	<b>-20</b>
11	1.53	41	400	1.77	25	250	±0.24	- 16
12	1.58	60	550	1.92	35	230	±0.34	- 25
Mean	1.05	58	440	1.41*	30*	298†		
SD	0.38	20	128	0.31	15	76		

<sup>\*</sup>Compared to before bronchodilators, p < 0.001.

frequency, and duration of wheezes can be measured. As the duration of a respiratory cycle is likely to be increased with relief of an asthmatic attack, the duration of wheezing was expressed as a proportion of the respiratory cycle duration  $(T_w/T_{to})$ . In the example in Figure 2, this ratio is 0.7/2.4 seconds, or 0.29.

Comparisons of observations before and after treatment were tested for statistical significance using Student's paired t-test.<sup>2</sup> The least-squares technique was used for correlation. Differences with a less than 5 percent probability of resulting from chance were

regarded as statistically significant.

#### RESULTS

Twenty patients, with a mean age of 43 years, were studied. Eighteen of the 20 were female. All 20 had audible wheezing before treatment; 12 of them still had some wheeze after treatment. All patients showed improvement in their FEV<sub>1</sub>; the mean value for the

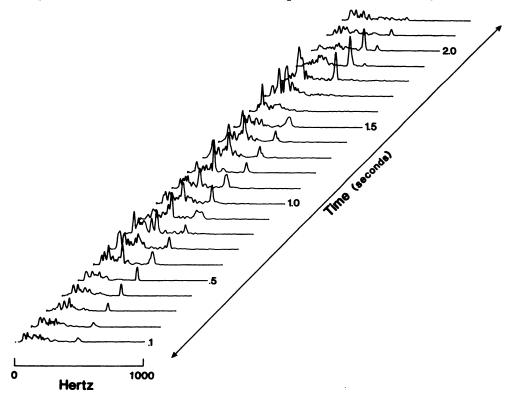


FIGURE 4. Complex set of wheezes in asthmatic subject in acute bronchospasm. Line 1 represents beginning of expiration. Wheezing was heard on inspiration and expiration, and several peaks are identified, even at same moment in breath cycle (polyphonia). This patient has wheezing in 19 of the 22 segments making up her breath cycle. Her  $T_{\nu}/T_{tot} = 1.9$  second/2.2 second = 0.86. Highest pitch of any wheeze is 500 Hz.

<sup>†</sup>Compared to before bronchodilators, p <0.01.

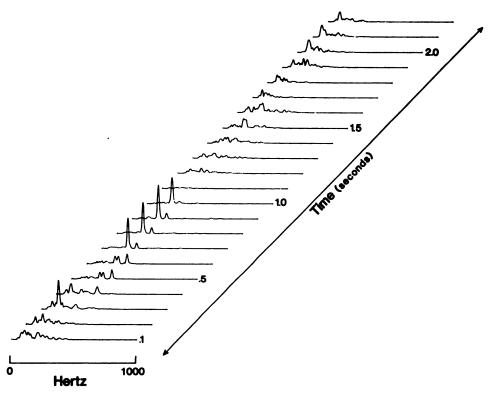


FIGURE 5. Same patient as in Figure 4 after bronchodilators. Line 1 marks beginning of expiration. Time of wheezing is now 0.8 second of a total of 2.6 seconds (T<sub>e</sub>/T<sub>test</sub> = 0.31). Highest frequency is now 300 Hz.

group rose from  $1.15\pm0.39$  L (mean  $\pm$  SD) before treatment to  $1.49\pm0.43$  L after treatment. Peak expiratory flow rates improved from  $150\pm67$  L/sec to  $192\pm66$  L/sec. The probability that either improvement resulted from chance was less than 1:1,000 (p<0.001).

In Figure 3, the relationship between  $FEV_1$  and  $T_w/T_{tot}$  is shown for all cases in whom a wheeze was identified, 20 patients before therapy and 12 patients with persistent wheeze after therapy. There is a significant correlation, with a correlation coefficient of 0.46 (p<0.01). It will be noted that the two patients with  $FEV_1$  less than 600 ml had less wheeze; the correlation coefficient is 0.59 if these two patients are excluded. There is no significant correlation between  $T_w/T_{tot}$  prior to treatment and the percent increase in  $FEV_1$  after treatment.

Changes in FEV<sub>1</sub> and in  $T_{\omega}/T_{tot}$  are shown in Table 1 for the 12 patients with wheeze after treatment. They had a statistically significant increase in FEV<sub>1</sub> (p<0.001), a significant decline in  $T_{\omega}/T_{tot}$  (p<0.001), and a significant reduction in the highest measured sound frequency, from a mean for the group of 440 Hz to 298 Hz (p<0.01). An example of the change for one patient is shown in Figures 4 and 5. Before treatment (Fig 4), a wheeze was present both during expiration (0.1 to 1.5 seconds) and inspiration (1.8 to 2.1 seconds), giving a  $T_{\omega}/T_{tot}$  ratio of 0.86. After treatment, Figure 5 shows the peak corresponding to wheeze heard only during expiration, from 0.3 to 1.0 second; the  $T_{\omega}/T_{tot}$ 

ratio has fallen to 0.31.

The intensity of wheezing and the simultaneous presence of wheezes of different pitch (polyphonic wheezing) can be readily examined by this technique. There was no correlation between these characteristics and the  $FEV_1$  or the response to therapy in the patients studied.

#### **DISCUSSION**

Wheezing sounds are a well recognized and frequently obvious physical finding in patients with asthma.3.4 Dodge and Burrows5 found that 20 percent of a general population sample was aware of wheezing at one time or another, but only 6.6 percent were diagnosed as having asthma. Patients with chronic airflow obstruction who were referred for pulmonary function tests were shown by Marini and colleagues to have a greater probability of responding favorably to bronchodilators if wheezing was heard during the expiratory phase of unforced deep breathing. They defined a wheeze as a continuous musical sound that commenced at any time during expiration, and wheezing was scored on an arbitrary scale. Forgacs<sup>7</sup> observations on wheezing sounds heard clinically and heard experimentally using excised bronchi led him to propose the toy trumpet as a musical instrument analogue of the wheezing airway. Rapid linear airflow through severely narrowed airways appears necessary for the production of these sounds, and Forgacs<sup>7</sup> suggests that apposition of the bronchial walls may be necessary for their initiation. Grotberg and Davis<sup>8</sup> have developed mathematical models which are based upon an interaction between vibrating airway walls and the flowing air column in sound production.

Objective measurement of the pulmonary sound signals makes it easier to decode them and to relate their characteristics to changes in structure and function. Analysis of these sound signals has been made easier by technical advances such as sound spectrum analysis. This has been applied to vesicular and bronchial breath sounds by Gavriely et al, who noted a peak in the frequency spectrum which was associated with wheezing. Charbonneau et al<sup>9</sup> showed a higher mean frequency in spectra recorded at the larynx from patients with asthma than in spectra from normal subjects.

Our method of analysis allows more exact measurement of changes (for example, those resulting from treatment) in terms both of frequency content and of timing in the respiratory cycle. By making observations at the same site, differences due to sound conduction from site of origin to the surface of the chest wall are minimized. While the changes in wheezes with relief of obstruction cannot as yet be explained in terms of specific changes in airway characteristics, a modest beginning has been made in the description of relationships. Potential advantages for our method of analysis are suggested by the correspondence. These

measured changes in the objective, recorded sound signal can in turn be related to the subjective perceived changes in sound. The attributes of wheezing most likely to be altered by effective bronchodilatation (T\_/T<sub>tot</sub> and pitch) are indicated for the consideration of the interested auscultator.

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