Journal of Sound and Vibration (1998) **213**(2), 377–382

AN ACOUSTIC MODEL OF NOISE PRODUCTION IN THE HUMAN BRONCHIAL TREE UNDER FORCED EXPIRATION

V. I. Korenbaum and A. A. Tagiltsev

Institute of Physics and Information Technologies of Far Eastern State University, 25 *Uborevich Street*, *Vladivostok* 690600, *Russia*

J. V. Kulakov

Vladivostok State Medical University, 2 *Ostryakov Street*, *Vladivostok* 690600, *Russia*

A. S. Kilin

Vladivostok Divisional Railway Hospital, 25 *Verhneportovaya Street*, *Vladivostok* 690003, *Russia*

and

H. V. Avdeeva and I. A. Pochekutova

Institute of Medical Climatology and Rehabilitation S.B. of R.A.M.S., *Sadgorod*, *Vladivostok* 690025, *Russia*

(*Received* 19 *November* 1997)

1. INTRODUCTION

Tracheal auscultation of forced expiratory (FE) noises was offered in reference [1] as a probable method for medical diagnostics of obstructive lung diseases (i.e., diseases resulting in bronchial permeability failures); however the complexity of this process acoustic picture caused the author's pessimism. Nevertheless, experimental confirmations [2–4] of diagnostic importance of expiratory noises, accompanying FE, analysis have appeared recently. This work represents an attempt at specification of an FE noise producing acoustic model.

2. ACOUSTIC MODEL

It is known that the origin of respiratory noises is connected with turbulence of air flows [1, 5–8]. However, the quantitative description of mechanisms of noise production in a bronchial tree has not been developed yet. An approach used for acoustic noise of ventilation systems [9] was offered in reference [10]. On the basis of these ideas it is possible to formulate the following acoustic model for the noise production during FE.

The noises, registered during FE, are classified according to the nature of origin into aerodynamic and auto-oscillatory producing mechanisms. At the beginning of FE the broadband noise of a turbulent boundary layer, having a spectrum close to white noise in a band of frequencies of about 50–450 Hz is found in the trachea for healthy adult persons.

Increase in expiratory flow rate causes consecutive turbulization of flow, beginning in the trachea, and appearance of considerably more powerful, than previous, noise of turbulent flow (indicated by 1, 2 in Figure 1), defined by broadband spectral maxima in the field of frequencies about 200 Hz (trachea) (f_1) and 300–400 Hz (principal bronchi) (f_2).

0022–460X/98/220377 + 06 $25.00/0/sv971490

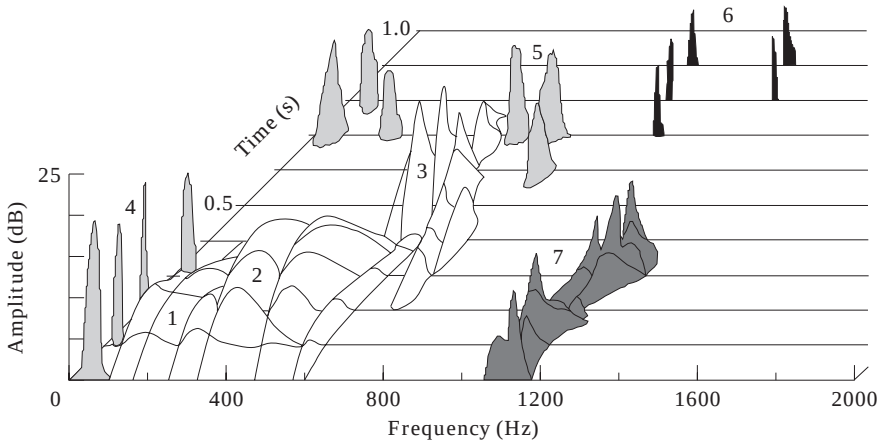

Figure 1. Forced expiratory noise waterfall spectrum scheme: 1, Broad-band spectral maximum of noise of turbulent flow in trachea; 2, broad-band spectral maximum of noise of turbulent flow in principal bronchi; 3, narrow-band spectral maximum of vortex, shedding noise; 4, low frequency discrete spectral components of auto-oscillatory noise; 5, middle frequency discrete spectral components of auto-oscillatory noise; 6, high frequency discrete spectral components of auto-oscillatory noise at the end of forced expiration; 7, high-frequency discrete spectral components of auto-oscillatory noise at the beginning of forced expiration.

In accordance with reference [10] a formula for frequency of turbulent flow noise spectral maximum f may be written as

$$f \approx 0{\cdot}2V/(2^i d_i^3), \tag{1}$$

where V is the instantaneous volumetric expiratory flow rate (EFR), i is the ordinal number of a bronchial tree generation, and d_i is the diameter of the ith generation bronchus. Using characteristic EFR meanings, usually determined by computerized spirometry, for the spectral components described above one finds from formula (1) the approximate equations:

$$f_1 \approx 2 \times 10^5 \times \mathrm{PEFR}/d_0^3(\mathrm{Hz}), \qquad f_2 \approx 10^5 \times \mathrm{PEFR}/d_1^3(\mathrm{Hz}), \tag{2, 3}$$

where PEFR is the peak volumetric flow rate (litres/second), d_0 is the diameter of a trachea (mm) and d_1 is the averaged diameter of a principal bronchus (mm).

During development of functional expiratory stenosis, accompanying FE [10] such significant linear flow rates are reached in the region of the 2nd–5th bronchial tree levels that shedding of vortices may occur. The latter takes place on ''turbulizators'', the role of which bronchial tree bifurcations probably execute. This effect is characterized by appearance of a powerful narrowband (discrete) spectral maximum, denoted 3 in Figure 1, f_3 being in a range of frequencies about 400–600 Hz (heard as a wheezing sound). During FE this discrete component can be displaced upwards in frequency, which, most likely, reflects a zone of the maximal resistance shift from lobar to segmentary and subsegmentary bronchi as a result of expiratory stenosis development. According to the authors' observations this effect is marked among the majority of healthy adult persons performing FE with the required intensity.

The expression for the frequency of a spectral maximum f_3 of the vortex shedding in agreement with reference [10] may be also presented as equation (1). Taking into consideration that the vortexes' tearing off is connected with the expiratory stenosis, observable after near 1/3 forced vital capacity of the lungs (FVC, the meaning which is also standardly determined by computerized spirometry) expiration and, by using the

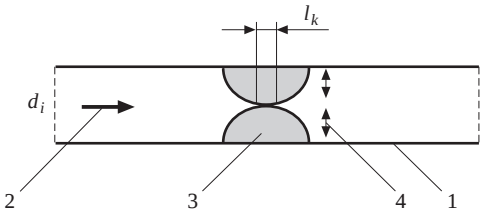

Figure 2. Bronchial wall tissue closing oscillation scheme: 1, Bronchial wall; 2, air flow direction; 3, bronchial wall tissue protuberances; 4, longitudinal direction of tissue protuberance oscillations.

closest standard EFR meaning for this FE stage, for the frequency f_3 (at least at the beginning of the stage) it is possible to write approximately

$$f_3 \approx 5 \times 10^4 \times \mathrm{EFR}_{25}/d_2^3(\mathrm{Hz}), \tag{4}$$

where $\mathrm{EFR}_{25}(1/\mathrm{s})$ is the instantaneous volumetric expiratory rate at a level of 25% of FVC expiration, and d_2 is the lobar bronchus averaged diameter (mm).

Due to the idealization of the FE volumetric expiratory rate curve (representing it as close to a rectangular triangle, that proved to be correct for healthy persons) an approximate estimation becomes also possible for temporary FE parameters of healthy persons: the FE noise process duration as a whole,

$$T \approx 2 \times \mathrm{FVC}/\mathrm{PEFR}, \tag{5}$$

and the duration of the vortex shedding,

$$t \approx (2/3) \times \mathrm{FVC}/\mathrm{EFR}_{50}, \tag{6}$$

where EFR_{50} is the instantaneous volumetric expiratory rate at a level of 50% of FVC expiration (which is the closest to the average EFR meaning of characteristic values for vortex shedding stage).

As a result of air flow rate decreasing, all narrowband spectral maxima 4–7 in Figure 1 observed later, unlike purely aerodynamic processes, considered above, represent auto-oscillations, connected with modulation of the air flow caused by a mechanical oscillatory system (resonator). It is possible to distinguish low frequency discrete components f_4 (it is lower than approximately 100 Hz), middle frequency discrete components f_5 (between 100 Hz and 600–700 Hz), high frequency (more than 600–700 Hz) discrete components at the end of an expiration f_6, and at the beginning of an expiration f_7.

The analysis of possible biophysical mechanisms of mechanical resonance results in the conclusion [1, 10] that it may be represented by bronchial wall mucous membrane tissue closing (see Figure 2). This effect is more characteristic for pathological condition of bronchial wall tissues (inflammation, oedema) and it is provoked by the pressing motion of the bronchial walls resulting from the expiratory stenosis. The frequency of such a mechanical resonator may be represented [10] as

$$f_0 \approx 0{\cdot}5(K/m)^{0{\cdot}5}/\pi, \tag{7}$$

where m is the effective mass and K is the equivalent rigidity. The exact meaning of f_0 depends on the bronchial wall tissue closing configuration and consequently is variable; however, its limits can be estimated with the introduction of some assumptions. One can assume, that the bronchial wall tissue protuberances (3 in Figure 2) undergo longitudinal oscillations. Note, that for small meanings l_k the tissues can undergo flexural oscillation

as a result of an air flow influence. On the other hand, for large l_k the closing zone may be impenetrable for an air flow; this is, by the way, verified by observations [11]. So, it is clear intuitively, that to produce longitudinal oscillations the value of l_k must be between these two limiting cases. Proceeding from these reasons, one can suppose that $0{\cdot}1d_i \leqslant l_k \leqslant 0{\cdot}5d_i$. Consequently the area of a closing zone, having an order $S_k \sim l_k d_i$, lies in the limits $0{\cdot}1d_i^2 \leqslant S_k \leqslant 0{\cdot}5d_i^2$. For an estimation of the rigidity of the system with longitudinal oscillations one can regard it as an equivalent rectangular prism of rubber-like material with the dimensions $a \sim d_i$, $b \sim l_k$, $h \sim d_i/2$. The rigidity of such a prism is defined as [12]

$$K \sim \mu G_z S_k / h, \tag{8}$$

where μ a tissue's shift module. The dimensionless value $G_z \sim 4$ in accordance with values of the parameters $a/h \sim 2$, $b/h \leqslant 1$ [12]. Thus, the rigidity (8) has the order $K \sim 8\mu l_k$. The mass of tissues participating in a movement can be taken as that of a cylinder with a diameter d_i and length l_k. Supposing the density of the tissues ρ to be close to that of water, one finds the formula for the equivalent mass as $m \sim 0{\cdot}25\rho l_k d_i^2$. Thus, the expression for the frequency (7) may be transformed to

$$f_0 \sim 0{\cdot}5(32\mu/\rho\pi)^{0{\cdot}5}/d_i\pi. \tag{9}$$

As for the shift module μ, this value for external soft biotissues [13] is of the order of 10^3 N/m^2. As the bronchial mucous membrane is obviously more compliant than external tissues, having an elastic membrane (skin), because of the specific data absence one can assume that 10^2 N/m$^2 \leqslant \mu \leqslant 10^3$ N/m^2. In this case the frequency f_0 may be represented as

$$f_0 \sim (1{\cdot}6 - 5{\cdot}1)/d_i. \tag{10}$$

For example, for the 10th generation bronchi ($d_{10} \sim 1{\cdot}3$ mm), $f_0 \sim$ 1200–3900 Hz, and for the 3rd generation bronchi ($d_3 \sim 5$ mm), $f_0 \sim$ 350–1000 Hz.

The comparison of these values obtained with observable narrowband maxima f_4–f_7 (see Figure 1) indicates that only a part of the discrete spectral components, mainly f_6, f_7 and partially f_5, may be explained with the help of the mechanism considered.

Discrete components with lower frequencies may result from flexural oscillations of mucous membrane sections or viscid secretion pressed into bronchial lumen (as a result of the expiratory stenosis), which is more characteristic for a pathological than normal condition of bronchial walls. Appreciably changing of the oscillation type to flexural (having approximately the same mass) results in a 1–2 order drop of the rigidity K and a 3–10 times decrease of f_0 (7), which seems to be enough to explain the appearance of components f_4, f_5 by action of this mechanism in respiratory channels of the 0–5th generations. However, frequency components f_4, f_5 can be also explained by means of the subsonic flutter effect (flexural fluctuation of bronchial walls without the tissues closing), detailed in reference [8], that is possible with a normal bronchial walls condition too.

3. EXPERIMENTAL TECHNIQUE

To estimate the reliability of the above-stated model experiments for which two independent groups of persons were selected were carried out. The first group (24 persons) consisted of subjects who had not been attributed with bronchial permeability failures according to medical data. Values of the parameters T, t, f_1, f_2, f_3 were experimentally determined for persons of this group. The second group (20 persons) was mixed, consisting of seven subjects healthy according to medical data and 13 patients having been attributed

with bronchial permeability failures. Discrete spectral component f_4–f_7 presence was determined experimentally for persons of the second group.

To define experimentally the FE noise spectral–temporary characteristics, an electret microphone having a stethoscope head (diameter 20 mm) was mounted on a subject's lateral tracheal wall. The subjects were in the sitting position with noseclips. While performing FE the signals received by a microphone were recorded with an AM tape recorder (transfer performance deviation less than 4 dB in the band of frequencies between 30 Hz and 2 kHz). Signal processing was performed by a computer of IBM PC type equipped with a system of data collecting (digitizing at 6 kHz). Separating of noise process into time intervals, spectral analysis in the frequency band up to 2 kHz (fast Fourier transformation, 512 spectral points, weighting by a Hamming window), and displaying as a waterfall spectrum were performed off-line by means of suitably designed software.

The actual values of the flow–volumetric parameters were determined by means of a computerized pneumotachograf ⟪Discom-21⟫ (Japan).

4. RESULTS AND DISCUSSION

The averages and standard deviations of the theoretical and experimental values of T, t, f_1, f_2, f_3 for the first group surveyed are represented in Table 1. Note, that the substitution of not only actual flow-volumetric parameters, but also bronchial diameters values for each person is necessary in formulae (1–6) for obtaining the true acoustic parameters; however this is difficult in practice. For this reason the averaged statistical values of the diameters [10] are used. This assumption, naturally, allows one to compare agreement of the theoretical forecasts and experimental data only as regards averaged values in each group. According to the results of Table 1 a good agreement between theoretical and experimental values for the parameters f_2, f_3, t, as regards averaged values over the group and satisfactory agreement for parameters T, f_1 are observed, which indicates the developed acoustic model's adequacy for a part of FE noises of aerodynamic origin.

For the second group surveyed the agreement between discrete frequency f_4–f_7 components appearance and the bronchial permeability failure presence confirmed by clinical methods was established. The distinctive feature of FE noise among all 13 patients with bronchial permeability failures in fine respiratory channels was steady observation of discrete frequency f_6 components at the end of an expiration. The time of appearance of this discrete component corresponds to the known physiological effect [10] of fine respiratory channels blocking during FE; this confirms the above-stated model of sound production by means of longitudinal oscillations of bronchial wall closings.

The narrowband spectral components f_4, f_5 were marked in the second group both among diseased patients, and among some persons considered to be healthy; this also is in accordance with the picture described above.

TABLE 1

Acoustic parameters values of FE tracheal noise for healthy persons of the first group

Parameter	f_1	f_2	f_3	T	t
Dimensions	Hz	Hz	Hz	s	s
Theoretical values (mean ± sd)	242 ± 66	389 ± 107	573 ± 152	1·13 ± 0·19	0·49 ± 0·09
Experimental values (mean ± sd)	165 ± 46	306 ± 66	514 ± 88	0·82 ± 0·19	0·55 ± 0·19

On the basis of the acoustic model and preliminary experimental data one may assume that discrete f_7 component presence is defined by longitudinal oscillations of bronchial wall closings in large respiratory channels, and results from pathologic tracheal or bronchial stenosis (it is in accordance with other conclusions [14]). However it has not been possible yet to confirm this hypothesis rigorously.

5. CONCLUSION

The model offered for noise production under FE certainly, is highly approximate; however in its framework it is possible to explain the origin of an appreciable part of the FE acoustic phenomena. The authors hope that accumulation and analysis of experimental data will allow one not only to establish better the details of the model, but also to develop a system of acoustic criteria, suitable for diagnostics of precursory symptoms of bronchial permeability failures. The authors express gratitude to all persons involved in the experiments.

REFERENCES

1. P. Forgacs 1978 *Chest* **73**, 399–405. The functional basis of pulmonary sounds.
2. H. J. Schreur, J. Vanderschoot, A. H. Zwinderman, J. H. Dijkman and P. J. Sterk 1994 *Chest* **106**, 91–99. Abnormal lung sounds in patients with asthma during episodes with normal lung-function.
3. N. Gavriely, M. Nissan, D. W. Cugell and A. H. Rubin 1994 *European Respiratory Journal* **7**, 35–42. Respiratory health screening using pulmonary-function tests and lung sound analysis.
4. V. I. Korenbaum, Ju. V. Kulakov and A. A. Tagil'tsev 1996 *Vestnik of New Medical Technologies* **3**, 87–89. New acoustic methods of research of human respiratory system.
5. F. Dalmay, M. T. Antonini, P. Marquet and R. Menier 1995 *European Respiratory Journal* **8**, 1761–1769. Acoustic properties of normal chest.
6. I. V. Vovk, K. E. Zalutsky and L. G. Krasny 1994 *Acoustical Physics* **40**, 762–767. Acoustic model of a human respiratory path.
7. N. Gavriely and D. W. Cugell 1996 *Journal of Applied Physiology* **80**, 5–13. Air-flow effects on amplitude and spectral content of normal breath sounds.
8. J. V. Grotberg, S. H. Davis 1980 *Journal of Biomechanics* **13**, 219–230. Fluid-dynamic flapping of a collapsible channel: sound generation and flow limitation.
9. M. Xekl and X. A. Muller 1980 *Directory on Technical Acoustics*. Leningrad: Sudostroenie.
10. V. I. Korenbaum, Ju. V. Kulakov and A. A. Tagil'tsev 1997 *Acoustical Physics* **43**, 78–86. Acoustic effects in the human respiratory system under forced expiration.
11. R. Beck, M. Odeh, A. Oliven and N. Gavriely 1995 *European Respiratory Journal* **8**, 2120–2128. The acoustic properties of snores.
12. V. T. Ljapunov, E. E. Lavendel and S. A. Shljapochnikov 1988 *Rubber vibrational isolators*. Leningrad: Sudostroenie.
13. E. V. Glushkov, N. V. Glushkova and E. M. Timanin 1993 *Acoustical Physics* **39**, 1043–1049. Definition of impedance and waveguide properties of biomaterials.
14. M. Yonemaru, K. Kikuchi, M. Mori, A. Kawai, T. Abe, T. Kawashiro, T. Ishihara and T. Yokoyama 1993 *Journal of Applied Physiology* **75**, 605–612. Detection of tracheal stenosis by frequency-analysis of tracheal sounds.