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MODELING THE MECHANISM OF THE VESICULAR SOUND GENERATION

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Current research presents a mathematical model of the mechanism of vesicular sound generation, based on the assumption of significant contribution of the alveolar walls vibration in the generation of noise during the act of respiration. The walls of the alveoli are presented in the form of membranes, and we show that when the membranes are periodically tensed, transverse oscillations of the membranes will occur, causing sound vibrations in the parenchyma. The characteristics of the composite noise signal, which is formed during the simultaneous excitation of a representative ensemble of membranes with different geometric and mechanical characteristics, are determined. Based on the analysis of the signal, its shape, spectrum and fractal properties is found to be close enough to the shape, the spectrum and the fractal properties of the real vesicular breath sound.

KEY WORDS: mathematical model, alveoli, vesicular sound, membrane, fractal model signal, singularity spectrum, parenchyma

Представлено математичну модель для механізму генерації везикулярних звуків дихання. В основу моделі покладено припущення про суттєвий внесок у загальний рівень звуків складових, що пов'язані з коливаннями стінок альвеол у процесі дихання. Стінки алвеол у легенях моделюються як пружні мембрани. Показано, що при періодичній зміні натягу в мембрані виникають поперечні коливання, які і є джерелом звуку в паренхімі. Запропоновано оцінку характеристик композитного звукового сигналу, що формується за рахунок суперпозиції випромінювання ансамблю мембран різної геометрії та з різними механічними властивостями. Аналіз характеристик змодельованого сигналу, таких як форма, спектр, фрактальна структура, дає достатньо підстав для висновку про адекватне моделювання реальних везикулярних звуків дихання.

КЛЮЧОВІ СЛОВА: математична модель, альвеоли, везикулярні звуки, мембрани, фрактальна модель сигналу, спектр сингулярності, паренхіма

Представлена математическая модедь для механизма генерации везикулярных звуков дыхания. В основу модели положено предположение о существенном вкладе в общий уровень звуков составляющих, которые связаны с колебаниями стенок альвеол в процессе дыхания. Стенки альвеол в легких моделируются как упругие мембраны. Показано, что при периодическом изменении натяжения возникают поперечные колебания, которые и являются источником звука в паренхиме. Предложена оценка характеристик композитного сигнала, который формируется за счет суперпозиции излучения ансамбля мембран различной геометрии и с различными механическими свойствами. Анализ характеристик модельного сигнала, таких как форма, спектр, фрактальная структура, дает достаточные основания для вывода об адекватном моделировании реальных везикулярных звуков.

КЛЮЧЕВЫЕ СЛОВА: математическая модель, альвеолы, везикулярные звуки, мембраны, фрактальная структура сигнала, спектр сингулярности, паренхима

INTRODUCTION

Research in [1], which is based on the use of traditional and original methods of recording and processing of vesicular and tracheal breath sounds in healthy people, including their fractal analysis, finds the nature of vesicular and tracheal noises to be different. It is shown that vesicular sound is likely to be generated as a result of periodic stretching of parenchyma during respiration, whereas tracheal noise, as already well-known, is generated due to pressure pulsationson the inner surface of the trachea due to unsteady air flow in the glottis. However, the question about the mechanism of the energy transformation, stored in the lung parenchyma due to its deformation, into the sound energy continues to be controversial.

At the same time, back in 1961, prominent Soviet clinician A.A. Kovalevsky [2] suggested that the deformation of the parenchyma in the act of respiration should cause fluctuations in the alveolar walls, and these oscillations in turn excite the sound vibrations in the lung parenchyma. Later, another prominent clinician A.Y. Gubergrits [3] fully agreed with Kovalevsky's assumptions. The authors of the article "Breath sounds" in the Large Medical Encyclopaedia [4] also believe that the vesicular sound generated in the alveoli themselves mainly due to alveolar wall oscillations, resulting from elastic tension of the

alveoli when they are tensed during inspiration and when the strain is relieved during expiration. We are also impressed with this assumption, and thus the purpose of this paper is to show, at least in theory, that the proposed by clinicians mechanism of vesicular breath sound generation in the lungs is quite believable.

1. THEORETICAL MODEL

First of all, let us recall some of the physical and geometric characteristics of the parenchyma. Parenchyma is the biological tissue of the lungs, having mainly a honeycomb structure [5] (in the first approximation resembling foam). It is these cells which are randomly oriented air-filled irregular polyhedrons with the maximum size of the order of $L \approx 300 \cdot 10^{-6}$m that are called alveoli. The amount alveoli in human lungs is approximately 600 million. Alveolar walls are quite thin (about $h \approx (6...10) \cdot 10^{-6}$m) and contain tiny blood vessels (capillaries). The density of the walls of the biological tissue is close to the density of water, i.e. $\rho \approx 10^3$kg / m^3. The surface of the alveolar walls is wetted with a thin layer of liquid which tends to flatten alveoli as a result of surface tension. However, the layer of bio cells lining the wall surface excretes a particular substance, surfactant, which reduces the surface tension of the liquid and thus ensures stability of the alveolar shape [5].

Thus, the alveolar walls, in the absence of respiration, have some initial tension, which, however, cannot disturb the shape of the alveoli. During inhalation, parenchyma increases in volume due to workings of the intercostals and the diaphragm muscles, which increase the volume of the thorax. Naturally, the volume of the alveoli increases due to stretching of their walls. It is understood that during exhalation, this process is reversed. With this in mind, it is in the process of respiration, as stated in [2–4], that periodic stretching of the walls transforms into transverse vibrations of the alveolar walls, that in turn excite acoustic oscillations recorded on the surface of the chest as noise vesicular breathing.

We now show that the described mechanism of transformation of periodic deformation of the alveolar wall stretching into their transverse vibrations is possible. To do this, we consider a simple two-dimensional physical model that can illustrate the mechanism that leads to the excitation of transverse vibrations of the alveolar walls under their periodic extension. Since $h << L$, then it is perfectly acceptable to use a rectangular membrane, fixed on a contour (Fig. 1), as the simplest model of an alveolar wall.

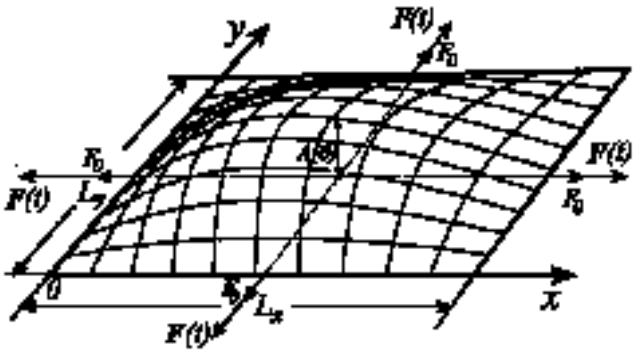

Рис. 1. Fixed contour membrane at time $t = 0$

Suppose there are a constant (time-independent) membrane tension F_0 and a small, compared with the dimensions of the membrane, initial transverse deviation of the centre of the membrane $A(0)$ at time $t = 0$. We now apply periodic tension $F(t)$ to the membrane (which will simulate the tension resulting in periodic stretching of an alveolar wall during respiration) and would like to see if this leads to the transverse vibrations of the membrane.

To solve this problem, we use a well-known differential equation of membrane oscillations [6],

$$\frac{\partial^2 w\,(x,y,t)}{\partial t^2} = c^2\,(t)\left[\frac{\partial^2 w\,(x,y,t)}{\partial x^2} + \frac{\partial^2 w\,(x,y,t)}{\partial y^2}\right], \tag{1}$$

where $w(x,y,t)$ is the transverse deflection of the membrane from the equilibrium position, $c^2\,(t) = [F_0 + F\,(t)]\,/\bar{\rho}$; $c(t)$ is the velocity of propagation of perturbations in the membrane, $\bar{\rho} = \rho h$ is the surface density of the membrane's bio tissue.

Given the above assumptions, we can write the boundary conditions as follows:

$$w\,(x,y,t) = 0; \quad 0 \le x \le L_x, \quad y = 0, \;\; y = L_y;$$

$$w\,(x,y,t) = 0; \quad 0 \le y \le L_y; \quad x = 0, x = L_x. \tag{2}$$

We shall focus on the case when the membrane oscillates only in its first mode, when there are no sections on the surface of the membrane oscillating in antiphase. Then, taking into account (2), we get

$$w\,(x,y,t) = A\,(t)\sin\left(\frac{\pi x}{L_x}\right)\sin\left(\frac{\pi x}{L_y}\right). \tag{3}$$

Substituting (2) into equation (1) we obtain the equation for the amplitude of the oscillations $A(t)$. Assuming for simplicity that $L_x = L_y = L$, we have

$$\frac{d^2 A\,(t)}{dt^2} + 2\left(\frac{\pi}{L}\right)^2 \frac{F_0 + F\,(t)}{\bar{\rho}} A\,(t) = 0; \tag{4}$$

$$x = L/2; \quad y = L/2.$$

In order to proceed with solving equation (3), let us define the initial conditions as

$$A\,(0) = 0.1L; \quad \frac{dA\,(0)}{dt} = 0 \tag{5}$$

Here the value of the initial deviation $A(0)$ is chosen taking into account the inequality $A(0) << L$. Now we need to set the value of the constant tension of the membraneF_0. Unfortunately, unlike the linear dimensions of the alveolar walls and their density, there is no specific information about the magnitude of tensionF_0that we could find in the literature available to us. Therefore, in order to estimate it, even roughly, we used the equation relating the first Eigen frequency f_1 of the square membrane with its geometric and mechanical characteristics [6]:

$$f_1 = \frac{1}{\sqrt{2}L}\sqrt{\frac{F_0}{\bar{\rho}}}, \tag{6}$$

from where

$$F_0 = 2f_1^2 L^2 \bar{\rho}. \tag{7}$$

We know from experimental data [1-4,7-9] that the main energy of vesicular sound lies approximately in the 50 Hz to 250 Hz range. Assuming, for example, an average frequency of $f_1 = 100$ Hz and using the above characteristics of the alveolar walls as well as formula (6), we can easily demonstrate that the tension F_0can be of the order of $\sim 2 \cdot 10^{-5}$ N / m.

Next we need to set a variable tension $F(t)$ of the membrane. For simplicity, we choose a periodic function

$$F\,(t) = \frac{\bar{F}}{2}\left(1 + \sin\left(2\pi\tilde{f}t - \pi/2 + \varphi\right)\right), \tag{8}$$

which will model the tension impacting an alveolar wall during respiration. Here fixed frequency $\tilde{f} = 0.345$ Hz (period $\tilde{T} = 1/\tilde{f}$ is thus 2.9 s) approximately corresponds to the real frequency of respiration, φ is the initial phase and $\bar{F}$ is the amplitude. Note that function $F(t)$ does not go negative for any value of t.

We now need to set specific values for parameters $F_0, \bar{\rho}, L$ and φ. It is apparent that if we assign some specific numbers to these parameters, we can simulate the mechanism of oscillation excitation only in one alveolar on one frequency. In reality, as we know from [2–4], walls of multiple alveoli vibrate simultaneously. These alveoli have different shapes, sizes, initial tensions and, consequently, different Eigen frequencies. In addition, the alveolar walls in the parenchyma obviously cannot oscillate synchronously with the same phase. Moreover, the distance from different alveoli to the point where the sound is registered, is different, which causes a different phase delay. It is precisely due to the superposition of this multitude of oscillations with different frequencies and phases that a noise signal characteristic to vesicular breathing is generated.

In order to take this into account, at least to some extent, we proceed as follows. We assume that we have some fairly representative ensemble of membranes. To use some specific numbers, assume their quantity M to be 250. Each time we compute a numerical solution of the equation (4), we choose parameters F_0,$\bar{\rho}$,$L$and φrandomly within the following ranges[1]:
$3 \cdot 10^{-6} \le F_0 \le 60 \cdot 10^{-6}$ N/m,
$4 \cdot 10^{-3} \le \bar{\rho} \le 16 \cdot 10^{-3}$ kg/m^2,
$2,5 \cdot 10^{-4} \le L \le 3,5 \cdot 10^{-4}$m, $0 \le \varphi \le \pi$.
Let $\bar{F}$be $6 \cdot 10^{-6}$ N/m. Then the total number of numerical solutions of the equation (4) will be $M = 250(m = 1, 2, 3....M)$. As a result, we get 250 different values of amplitudes of the membrane centre deviations, which we average out and then assume that the sound pressure developed in the environment (i.e., in the parenchyma) at some time t_i is proportionate to this average amplitude.

We computed the numerical solution of the differential equation (4) over the observation time interval equal to 32 seconds. At a sampling frequency $f_d = 2048$ Hz the number of partitioning points of the observation interval was $N = 59440$. Accordingly, the set of values $t_i = i/f_d (i = 1, 2, ..., N)$ defined the current discrete point of the observation time interval. Thus, the equation (4) was solved 250 time sat each point t_i. Thereafter, at each time interval point $t_i$we computed average amplitude of the membrane centre deviation.

$$\tilde{A}(t_i) = \frac{1}{M}\sum_{m=1}^{M} A_m(t_i). \tag{9}$$

Finally, let us point to one very important fact. As shown in [7], the lung parenchyma has the property of significantly absorbing sound vibrations propagating there. Moreover, the level of absorption increases as the frequency of sound oscillations increases. One can roughly assume that the presence of absorption leads to the decrease in the level of sound oscillations as the frequency increases proportionate to $\sim 1/f^2$. In order to account for this property of the parenchyma, the calculated signal $\tilde{A}(t)$ was passed through a low

[1]Though the ranges were selected randomly, but so that, the first Eigen frequencies of the walls were to be in the range from ~ 50 to $\sim$250 Hz. Any other information about real parameters range of the F_0, $\bar{\rho}$, L and φ are absent today.

frequency filter $\Phi(f)$with a cut-off frequency of about 50 Hz, and a slope of about $\sim$ 12 dB per octave. Therefore, the final form of the above simulated signal can be represented as

$$\tilde{A}(t,f) = \tilde{A}(t)\Phi(f). \qquad (10)$$

Thus, within the framework of the adopted model, we obtained the model signal (essentially a time series), imitating, to some extent, vesicular breath sound, recorded on the surface of the thorax. In this case, the signal source is the membranes whose transverse vibrations are caused solely by their periodic tension.

2. ANALYSIS OF THE NUMERICAL RESULTS

First of all, let us see which frequency range Eigen frequencies of the membranes belong to. As an example, Fig. 2 shows the values of Eigen frequencies of the membranes obtained as a result of one random sampling of its parameters within abovementioned ranges. As is evident from the figure, the values of Eigen frequencies lie in the $\sim$40 Hz to $\sim$270 Hz range. This range of Eigen frequencies roughly corresponds to the frequency range where the major portion of the energy of vesicular sounds is concentrated.

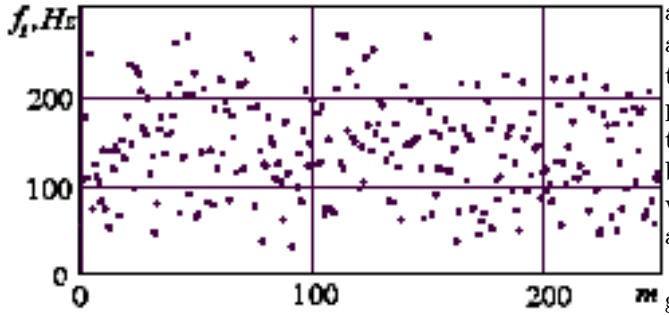

Рис. 2. One of the outcomes of random sampling of the Eigen frequencies of the membrane

Next, consider the model signal $\tilde{A}(t,f)$ obtained from calculations and simulating vesicular breath sound. We then compare it with the real signal $R(t)$ of vesicular sound recorded on the surface of the thorax of a healthy person at a point slightly below the right clavicle (see Fig. 3).

As might be expected, the overall shape of the model signal does not exactly repeat the shape of the real vesicular sound signal. The model signal does not show pronounced signal separation into phases of growth and decayof the membrane tension force (corresponding to the phases of inhalation and exhalation, which are more pronounced in the real vesicular sound signal).

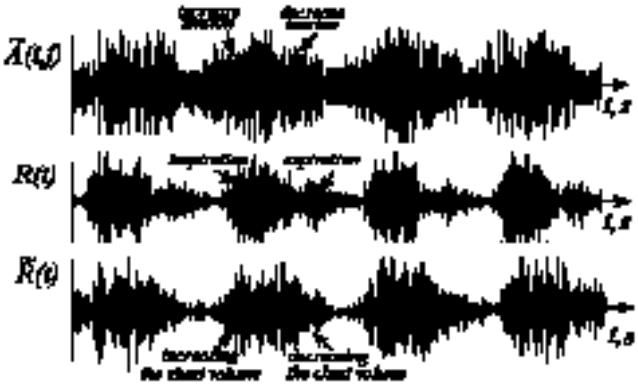

Рис. 3. $\tilde{A}(t,f)$ – the model signal; $R(t)$ – the real signal of the vesicular breath sound; $\tilde{R}(t)$ – the real signal generated by periodically increasing the chest volume, in the absence of air flow in the bronchial tree (in accordance with the method proposed in [1])

Now, referring to Fig. 4, it is interesting to compare spectra of these three signals. As can be seen, the behavior of all three spectra is virtually identical, at least in the frequency range from about 60 Hz to 300 Hz. The model signal outside this frequency range simply does not exist. The values of the Eigen frequencies of the membranes are limited by the adopted ranges of the physical parameters of the alveolar walls see Fig. 2. This fact directly indicates that the real dispersion of geometric and mechanical parameters of the alveolar walls is much wider than the one we adopted for calculations. It is also possible that during respiration it is not only the alveolar walls that oscillate, but also the alveolar duct walls and the walls of small respiratory bronchioles.

Above, we have compared the obtained model signal and the real signal of the vesicular sound using their general enough characteristics such as waveform and spectrum. Now it is important to investigate and compare the finer structure of these signals, namely, the existence and nature of the correlated sequence of alternating members of their time series. The most expedient approach is to analysis the fractal properties of these signals. As in [1], we conduct the fractal analysis of signals based on MF-DFA (Multifractal Detrended Fluctuation Analysis) [10]. Therefore, we will not provide a detailed description of the algorithm for this method, and immediately turn to the analysis of the results.

We turn to Fig. 5, which shows all spectra of singularities for all considered signals. As can be seen, all singularity spectra are rather close to each other. This result leads to the conclusion that a rather

representative ensemble of elementary membranes whose transverse vibrations are caused by their periodic stretching can generate multifractal noise-like signal sufficiently close to the vesicular sound.

Fig. 5 also shows the three singularity spectra (curves *1'–3'*), which were obtained after randomly mixing members of series describing the signals under consideration (curves *1–3*). Mixing procedure is a standard test, which is designed to confirm the presence or absence of long-range correlations in the set of values of the time series.

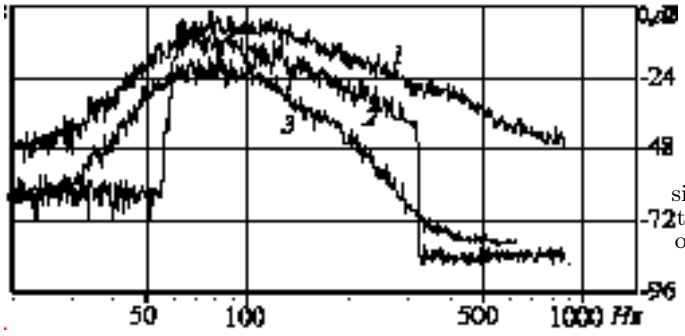

Рис. 4. Spectra of: *1* – the vesicular breath sound signal, *2* – the model signal, *3* – the signal arising due to the periodic increase in the chest volume in the absence of air flow in the bronchial tree

The most expedient approach is to analyze the fractal properties of these signals. As in [1], we conduct the fractal analysis of signals based on MF-DFA (Multifractal Detrended Fluctuation Analysis) [10]. Therefore, we will not provide a detailed description of the algorithm for this method, and immediately turn to the analysis of the results.

As follows directly from Fig. 5, mixing led to the fact that the multifractal properties of the signal disappeared and the initial series almost turned into monofractal signals with properties similar to white noise. Recall that white noise is a uniform signal with the value of the index of singularity$\alpha = 0,5$. This suggests significant influence of the original series of long-range correlations on the multifractal properties.

CONCLUSION

We proposed a model of the alveolar walls in the form of a membrane. It is shown that with its periodic tension transverse vibrations arise, which generate sound propagating into the environment (the parenchyma). A complex noise signal, which can be formed during simultaneous excitation of a sufficiently representative ensemble of membranes with different geometric and mechanical characteristics, was modelled based on the solution of the problem of parametric vibrations of the membrane. Analysis of such model signal showed that its shape, spectrum and fractal properties are close enough to the shape, spectrum and fractal properties of the real vesicular sound. This result allows us to conclude that that the vesicular sound mainly occurs due to mechanical vibrations of the alveolar walls in their periodic tension during respiration.

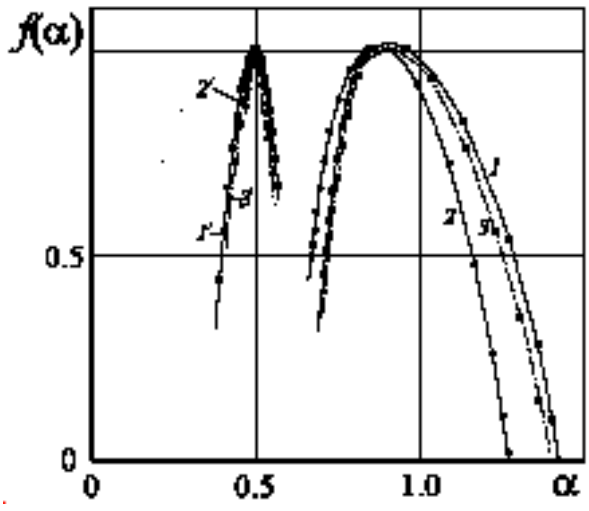

Рис. 5. Singularity spectra of: *1* – the vesicular sound signal; *2* – the model signal, *3* – the signal arising due to the periodic increase in the chest volume in the absence of air flow in the bronchial tree; *1'– 3'* – respectively for cases where the terms of the series, describing these signals, are randomly mixed

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