

SIXTH INTERNATIONAL CONFERENCE
ON LUNG SOUNDS

第6回 国際肺音学会

OCTOBER 1 & 2, 1981

THE FAULKNER HOSPITAL
BOSTON, MASSACHUSETTS
U.S.A.

PRESENTED BY
THE INTERNATIONAL LUNG SOUNDS ASSOCIATION

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SIXTH INTERNATIONAL CONFERENCE ON LUNG SOUNDS

BOSTON, MASSACHUSETTS

PROGRAM

Thursday, October 1, 1981

Registration		8:30 am
Opening Remarks	-----Dr. R.L.H. Murphy, Jr.	9:00 am
Welcome	-----Dr. D. Fairfax, President Faulkner Hospital	9:05 am
Introduction	-----Dr. P. Krumpe	9:10 am
Session A	9:20 am - 12:30 pm	
Lunch	12:30 pm - 1:30 pm	
Session B	1:30 pm - 5:00 pm	
	Cocktails and Buffet	7:00 pm

Friday, October 2, 1981

Session C	9:00 am - 11:40 am	
Business Meeting	11:40 am - 12:00 Noon	
Lunch	12:00 N - 1:00 pm	
Lung Sounds Nomenclature	--Dr. David Cugell	1:00 pm
Session D	1:20 pm - 3:40 pm	
Cracklefest	3:40 pm - 4:00 pm	
Summary of the Conference	--Dr. C. F. Dewey	4:00 pm

Session A

Moderators: R. Mikami and S. Ishikawa

- | | | |
|----------|---|--|
| 9:20 am | The Lung as a Filter | N. Gavriely
Y. Palti |
| 9:40 am | Sound Transmission Characteristics of the Airway and their Effect on the Waveform of Crackles and Wheezes | N. Honda
M. Mori
K. Kinoshita
H. Morinari
T. Shiraishi
S. Koike |
| 10:00 am | Propagation of Intrabronchial Spark Sounds in the Canine Lung Thoracic System | S. Kudoh
N. Miyasaki
N. Nagayama
A. Shibuya |
| 10:20 am | Estimation of in Vivo Regional Time Constants of the Lung in Man by Using Breath Sounds | Y. Ploysongsang |
| 10:40 am | Coffee Break | |
| 11:00 am | Grand Rounds | R. Loudon and
Panel |
| 12:30 pm | Lunch - Hallowell Hall | |

THE LUNG AS A FILTER

N. Gavriely
Y. Palti

A filter may be characterized by its transfer function, which relates its output to its input as a function of frequency. To calculate the transfer function, both the input and the output signals should be known. When the amplitude of the input signal is constant over a wide range of frequencies the power spectrum of the output signal represents the transfer function of the filter.

Near the site of their generation, breath sounds have a spectral pattern of random noise. The spectrum of transmitted sounds picked up over the chest wall therefore estimates the transfer function of the transmitting matter, i.e. the lung and the chest wall. The power spectrum of normal thoracic breath sounds was found to have an exponential decrease of power as frequency increases with a 12 dB/oct slope. Such a pattern is compatible with a simple passive filter model of sound transmission.

The small dimensions of the lung ultrastructure relative to wave lengths of breath sounds allow approximation of the lung as continuous, homogenous matter, the transfer of which may therefore be represented by the mean mechanical admittance per unit volume - Y:

$$Y = 1/Z = R^2 + (\rho\omega - 1/c\omega)^2^{-\frac{1}{2}}$$

Where Z - Impedance, R - mean frictional resistance, ρ - means specific mass of the lung, C - compressibility, ω - angular frequency.

For $R = 40 \text{ g x cm}^{-3} \text{ x s}^{-1}$ and $\rho = 0. \text{ g x cm}^{-3}$ ($1/\omega c$ omitted for $\omega c \gg 1$) a 3 dB point is found at 60 Hz with a 6 dB/oct pressure attenuation slope, corresponding to 12 dB/oct for sound intensity.

Since thoracic breath sounds represent the transfer function of the lung, which is in turn a simple function of mean frictional resistance, compressibility and specific mass, spectral analysis of breath sounds may be useful in the assessment of mechanical properties of the lung which are otherwise accessible only by invasive procedures.

SOUND TRANSMISSION CHARACTERISTICS OF THE
AIRWAY AND THEIR EFFECT ON THE WAVEFORMS
OF CRACKLES AND WHEEZES

N. Honda
M. Mori
K. Kinoshita
H. Morinari
T. Shiraishi
S. Koike

Lung sounds were recorded by placing two microphones about 4 cm apart in the same lung field. Fifteen patients, five with pulmonary fibrosis, four with bronchial asthma, two with bronchiectasis, and two with pulmonary edema were studied. Within the frequency range of 0.1 KHz to 8 KHz, the response of the two microphones and the recording system were equal. When corresponding waveforms of the same crackles were compared, they were different both in shape and timing. Generally, waveforms of larger amplitude preceded those of smaller amplitude and this delay was more prominent in bronchiectasis than in pulmonary fibrosis. With the increase of delay the amplitude became smaller and the shape of the waveform became more round. When corresponding waveforms of a wheeze were compared, they were the same in frequency but different in phase so that we could observe Lissajous loops.

What causes such differences? In our speculation most of them are explainable by the transmission characteristics of sound in the airway. We constructed an airway model and confirmed our hypothesis by a model experiment.

PROPAGATION OF INTRABRONCHIAL SPARK SOUNDS
IN THE CANINE LUNG-THORACIC SYSTEM

S. Kudoh
N. T. Miyasaki
N. Nagayama
A. Shibuya

Previously, we speculated that crackle and intrabronchial spark sounds on the chest wall surface appeared to be surface waves because of the late arrival of the sounds and the apparent reverse dispersion of the wave form. In this paper, for the purpose of clarifying the direction of sound propagation in the chest wall and approximate speed of the sounds in the lung-thoracic system, intrabronchial spark sounds were recorded on each layer of the living canine chest wall; skin, pectoralis major muscle, intercostal muscle and parietal pleura, and arrival time of the front part of the waves and the phase differences among each location were analyzed.

The results were as follows:

- 1) arrival time was well correlated with the direct distance from the sound source which measured by x-ray films, as well as the distance from the presumed epicenter
- 2) the front part of the waves was considered to be a direct wave from the sound source because of the direction of the vector of the wave number in the chest wall
- 3) speed of sound of the entire lung-thoracic system was estimated to be 50-70 m/sec approximately. Moreover, propagation speed in the chest wall was suspected to be very slow in comparison with speed of sound in the ultrasonic range, 1500 m/sec.

ESTIMATION OF IN VIVO REGIONAL TIME CONSTANTS OF THE LUNG
IN MAN BY USING BREATH SOUNDS

Y. Ploysongsang

Breath sounds were recorded from 19 healthy non-smokers and 15 relatively asymptomatic smokers. Positions of recording were: 5 cm from the top of the right lung in the anterior mid-clavicular line (M5), four areas 20 cm from the top of the right lung and 7.5 cm apart for each area (M20a, M20b, M20c and M20d). The sounds were recorded in pairs (M5 vs M20a, M20b vs M20a, M20c vs M20a and M20d vs M20a) on the x and y axes of a storage oscilloscope while the subjects were breathing small breaths (200-300 ml) at FRC. Both in normals and smokers, the M5 breath sounds led the M20a breath sounds. In the non-smokers, the phase differences of breath sounds between M20b vs M20a, M20c vs M20a and M20d vs M20a ranged between 2-3 degrees; whereas in the smokers, these values ranged between 7-9 degrees ($P < 0.01$). Using RC electrical analogs of the lung, it was found that, in the non-smokers, the mean time constant of M5 was 235 ± 21.1 milliseconds (ms) ($\bar{X} \pm \text{SEM}$), and that of M20a, M20b, M20c and M20d was 470 ± 36.7 ms; whereas in the smokers, these values were 335 ± 27.1 ms and 710 ± 80.3 ms respectively. There was also a larger degree of scattering of time constants in the smokers. It is concluded that (i) the ventilation of different regions of the lung is more out of phase in the smokers than in the non-smokers, (ii) the ventilation in the upper part of the lung leads that in the lower part of the lung, (iii) the regional time constant increases from the top to the bottom of the lung probably due to the effect of the gravity, (iv) the inhomogeneity of the time constant is greater in the smokers than in the non-smokers, (v) the regional time constant is generally larger in the smokers than in the non-smokers.

Session B

Moderator: J. Mead

- | | | |
|---------|--|--|
| 1:30 pm | Invited Physiology Lecture: "Pulmonary Pressure-Flow Relationships | R. Ingram |
| 2:15 pm | Inspiratory Crackles are Increased by Surfactant Inactivation | P. Krumpe
R. Kilpatrick |
| 2:35 pm | The Direction of the Initial Deflection (DID) in the Waveform of Bubbling Rales, Fine Crackles and Pleural Friction Rubs | H. Ogasawara
M. Matsuzaki
M. Munakata
Y. Minami
Y. Homma
M. Murao |
| 2:55 pm | Effect of PEEP on Crackles in Normals | P. Workum
R. Murphy
F. Davidson |
| 3:15 pm | Coffee Break | |
| 3:30 pm | Stress Relaxation Quadrupole Model of Crackle Generation: Theory | J. Fredberg
S. Holford |
| 3:50 pm | Stress Relaxation Quadrupole Theory of Crackle Generation: Results | S. Holford
J. Fredberg |
| 4:10 pm | Theory of Breath Sounds: Validation Studies | J. Hardin
J. Seiner
J. Patterson
J. Levasseur
W. Tribble, Jr. |

INSPIRATORY CRACKLES ARE INCREASED BY SURFACTANT INACTIVATION

P. E. Krumpe
R. L. Kilpatrick

Crackles are thought to result from a sudden equalization of pressure which occurs when a closed airway "pops open". If the number of crackles during lung inflation reflects the number of airways closed during the previous deflation, we reasoned that experimental conditions favoring airway closure should produce more crackles. We, therefore, measured inspiratory crackles in 8 excised bovine lobes during quasistatic inflation under controlled experimental conditions.

Crackles were defined as discontinuous bursts of sound energy above 2000 Hz and were recorded from a stethoscope glued to the visceral pleura of 8 excised bovine lobes. Volume history of inflations were controlled as follows. The lobes were first inflated to TLC ($P_L = 30$ cm H₂O) until all crackles ceased, next deflated to end expiratory transpulmonary pressures (P_{LEE}) between -10 and +10 cm H₂O, and then slowly reinflated to TLC. Crackles were counted during quasistatic reinflation and were correlated with inspiratory and expiratory compliance (C_{LI} , C_{LE}) and hysteresis ratio (HR).

Crackles were completely absent when degassed lobes were inflated with saline. Thus crackles require the presence of an air-fluid interface for their production. During inflation with air, the prevalence of crackles was dependent on the volume history of the preceding deflation. Inspiratory crackling increased as the end expiratory transpulmonary pressure (P_{LEE}) of the prior breath decreased. Compared to baseline conditions (B), the bronchial administration of 2 cc of saline (S) or aqueous Renografin-65 (R) produced only slight increases in crackles and no changes in either C_{LI} , C_{LE} , or HR. However, in all 8 lobes the bronchial administration of 2 cc of kerosene (K) resulted in a significant increase in crackles during reinflation from $P_{LEE} = 0, 2, 5, 7$ and 10 cm H₂O ($p < 0.5$ for all changes by multivariate analysis). Subdivision of lung volume and bronchographic morphology (using R as contrast material) were unchanged by K. However, C_{LI} decreased, while C_{LE} and hysteresis ratio increased ($p < .05$).

These data demonstrate that surfactant is inactivated by K (as has previously been reported by Menkes, et al, JAP 46:67-73, 1971). We conclude that the increased crackles after K indicate increased airway closure due to higher surface forces after surfactant inactivation. Furthermore, crackle measurements under controlled conditions can be used to study airway instability.

THE DIRECTION OF THE INITIAL DEFLECTION (DID)
IN THE WAVEFORM OF BUBBLING RALES, FINE CRACKLES
AND PLEURAL FRICTION RUBS

H. Ogasawara
M. Matsuzaki
M. Munakata
Y. Minami
Y. Homma
M. Murao

We investigated the direction of the initial deflection (DID) in the waveform of rales by time-expanded waveform analysis using our phonopneumograph. All the recorded lung sounds were classified into bubbling rales, fine crackles and pleural friction rubs, etc., according to our tentative criteria of the rales. In total, 1,134 bubbling rales, which may be consistent with coarse crackles in ATS classification, 753 fine crackles, and 911 pleural friction rubs (FR) were analyzed.

DID was upward in $93.0 \pm 4.0\%$ ($m \pm SD$) of inspiratory bubbling rales and $95.4 \pm 3.2\%$ of fine crackles, while downward in $93.2 \pm 10.9\%$ of expiratory bubbling rales. In FR, DID distribution showed wide variance.

To explain these clinical observations, we undertook the following two experiments: 1) chemical paste was infused into a vinyl tube, and was bubbled by a syringe attached to one end. DID of bubbling sounds recorded at both ends was opposite each other. 2) Two microphones (Mic A, Mic B) were placed on a vinyl plate (5 mm in thickness) 20 cm apart. DID of pulse sounds generated by rubbing the back of the plate A to B around the middle point of two microphones was upward in side A and downward in side B, B to A, vice versa.

In bubbling rales, DID mechanism could be explained by a sudden change of airway pressure due to rupture of a bubble. In FR, these experimental and clinical observations were well explained by theory of the earthquake, concerning to an initial motion of P (primary) wave which shows a quadrant type of distribution around the hypocenter (sound source).

We concluded that DID mechanisms of bubbling rales and FR were proved experimentally and theoretically, and that DID analysis might be an important clue to elucidate the generating mechanism of lung sounds.

THE EFFECT OF CONTINUOUS POSITIVE AIRWAY PRESSURE
ON THE PREVALENCE OF CRACKLES

P. Workum
F. Davidson
R. Murphy, Jr.

A proposed mechanism for the generation of crackles is the explosive opening of collapsed airways caused by the sudden "equalization of pressure upstream and downstream of the closed section of the airways" (1). Continuous positive airway pressure (CPAP) has been used to improve oxygenation in critically ill patients presumably because it prevents airway closure. We hypothesized that if CPAP prevents airway closure it should also decrease the number of crackles observed during chest auscultation. Consequently, we measured the number of crackles in five apparently normal subjects at the right anterior base during tidal breathing and a slow inspiration from residual volume (RV). We repeated the measurements when the subjects were placed on 5 centimeters of CPAP. We also measured the RV and functional residual capacity (FRC) of each subject at atmospheric pressure and five centimeters of CPAP. The results of the prevalence of crackles, FRC and RV, at atmospheric and atmospheric plus five centimeters water pressure will be presented.

1. Forgacs P. Crackles and Wheezes, Lancet 1967: 7/22, 203-205.

STRESS RELAXATION QUADRUPOLE MODEL OF CRACKLE GENERATION: THEORY

J. J. Fredberg¹
S. K. Holford²

In modeling the crackle phenomenon we considered the simplest set of circumstances consistent with a plausible physical argument, expressed as a point source in an ideal medium. The point source, a quadrupole, represents the stress field in lung parenchyma in the neighborhood of a collapsed airway, and its time course when the airway opens. For the sake of simplicity we have characterized the dynamics of the airway opening event by a single time constant. The ideal medium, lung parenchyma, was modeled as a homogeneous linearly elastic lossless non-dispersive continuum of infinite extent undergoing infinitesimal strains. Despite its simplicity, this elementary model leads to predictions of crackle waveforms, spectral densities, and spatial intensity distributions which go far beyond data available in the literature, which are consistent with the data that do exist, and consistent with qualitative features of crackle phenomena observed in chest auscultation. Until more data are available and critical experiments carried out this model may provide a useful working hypothesis and a convenient organizational framework for interpretation of discrete sound emissions from lung parenchyma.

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Supported by Grant HL23318 from the National Heart, Lung,
and Blood Institute

STRESS RELAXATION QUADRUPOLE THEORY OF CRACKLE GENERATION: RESULTS

S. K. Holford
J. J. Fredberg

Using the stress relaxation quadrupole model for crackles (rales), we calculated the pressure response in the surrounding medium resulting from the opening of collapsed airways of diameter $d = 0.5$ to 5 mm. We calculated the outputs of both an ideal blocked force transducer and an air-coupled microphone. We studied the temporal, spectral, and spatial characteristics of model crackles and observed the effects of varying airway diameter.

The crackles produced by the quadrupole model and simulated transducer filter were similar to crackles observed in patients using air-coupled microphones. The waveform shapes varied with airway size (d), ranging from shapes similar to fine crackles for small d to shapes similar to coarse crackles for large d . Successive zero-crossing intervals of model crackles increased with time. This transition from fast to slow response was most notable for small airway sizes. Model crackles changed in shape as the radius (r) and angle (θ) of the observation point varied relative to the source airway. The most important angular effect was the polarity reversal of the crackle waveform at an angle of 90 compared to the waveform at 0. As radius increased, both the amplitude of the crackles and their zero-crossing intervals decreased.

The frequency spectra of predicted crackles at the transducer output had a peak at about 200 Hz independent of airway size. The predicted high frequency content was greater for crackles from small airways.

Crackle waveform energy was nonuniformly distributed in the volume surrounding the airway. Crackles were transmitted best to regions equatorial with respect to the airway axis. Constant energy contours calculated on the chest surface had an ellipsoid appearance. The predicted extent of the area of crackle observability on the chest was greater for larger airways, implying a connection between crackle waveform shape and chest surface distribution.

Supported by Grant HL23318 from the National Heart, Lung, and Blood Institute and by the Institute of Occupational and Environmental Health.

THEORY OF BREATH SOUNDS: VALIDATION STUDIES

J. C. Hardin
J. M. Seiner
J. L. Patterson, Jr.
J. E. Levasseur
W. Tribble, Jr.

We previously developed a theory of breath sound production based on the oscillation of orbiting vortices within the bronchial passages. This predicted the expiratory frequency of sound as: $f_n = 0.212 (V_n/D_n) (D_n/D_{n+1})^2$, where V_n and D_n are respectively the mean axial velocity and diameter in the n th order of bronchi. We have tested this theory in a series of experiments in an anechoic chamber with a numerically machined model bifurcation with a 4 mm ID parent tube and 3.4 mm ID daughter tubes. Airflow was let into the model through straight tubes, and was controlled over a Reynolds number range of 2000 to 8000. The simulated breath sounds were subjected to narrow band spectral analysis.

The frequencies predicted by theory were within 25% of the measured frequencies for the fundamental tone. Significant nonlinearity was, however, observed and the findings indicated that the inherent non linear behavior was due to variations in the position of formation of the vortices within the elliptical zone of transition between parent and daughter tubes. Serial cross-sections through a Batson's #17 corrosion compound injection of dog lung bifurcations showed a peaking of cross-sectional area at the bifurcation, implying sequential deceleration and acceleration of the flow through the junctional region. Studies were also done with water flowing through the model, while colored dye was instilled through minute orifices into the central stream of each daughter tube. The information from these anatomic and flow studies, including studies with flexible simulated airways, is being utilized to refine our original theoretical analysis.

Session C

Moderator: J. Fredberg

- | | | |
|----------|--|---|
| 9:00 am | The Origin of Wheezes | M. Mori
N. Honda
K. Kinoshita
H. Morinari
T. Shiraishi
S. Koike |
| 9:20 am | Flutter of a Flexible Channel Conveying a Compressible, Viscous Fluid | J. Grotberg
E. Reiss |
| 9:40 am | Time-Frequency Spectrums of Respiratory Sounds in Asthmatic Patients | Y. Maeda
K. Nitta
Y. Yui
T. Shida |
| 10:00 am | Wheezes in Asthmatic Patients: The Number of Wheezes and its Relation with Breathing Maneuver and Severity of Airway Obstruction | T. Takezawa
F. Shirai
S. Sawaki
R. Mikami
S. Kudoh
A. Shibuya
N. Aisaka |
| 10:20 am | Coffee Break | |
| 10:40 am | Wheezing in Asthma and its Response to Bronchodilators | R. Baughman
R. Loudon |
| 11:00 am | Margins of Spectral Variability of Lung Sounds | C. Druzgalski |
| 11:20 am | Vesicular Lung Sound Amplitude Mapping by Flow-gated Phonopneumography | S. Kraman
D. O'Donnell |
| 12:00 N | Lunch - Hallowell Hall | |

THE ORIGIN OF WHEEZES

M. Mori
N. Honda
H. Morinari
T. Shiraishi
S. Koike
K. Kinoshita

Wheezes are believed to be generated by a regular vibration of the airway wall. However, what causes such a regular vibration has not been clearly understood. We studied waveforms of wheezes recorded from four patients with asthma. Wheezes were composed of sinusoidal waves and their harmonics, fundamental frequencies ranging between 0.1 KHz to 1.0 KHz. Like frequency modulated signals the frequency changed, sometimes as much as 100 Hz, with the development of a wheeze. We divided the waveform of a wheeze into three segments, early (build-up), middle, and late (decay). Characteristically, both build-up and decay of a wheeze were exponential. In an oscillatory system, such a positive exponential growth indicates presence of a negative resistance by means of feedback. In asthma a feedback loop is established if the natural frequency of the narrowed bronchial wall and that of the adjacent lung are equal and the feedback signal is in phase. In conclusion, we suggest wheezes are caused by a feedback excitation.

FLUTTER OF A FLEXIBLE CHANNEL CONVEYING A
COMPRESSIBLE, VISCOUS FLUID

J. B. Grotberg^{1,2}
E. L. Reiss¹

A mathematical model of flow through a two-dimensional, flexible channel is examined for its stability characteristics. At the previous conference, this airway model was shown to lose stability by partial collapse (static divergence) when the conveyed fluid was inviscid and incompressible. The collapsed state was stable to periodic disturbances which decay to zero because of wall damping; no flutter was predicted. The non-linear collapse also provided a mechanism for flow limitation when the fluid non-linearity of convective acceleration was included.

Currently we consider two important fluid properties: compressibility and viscosity. The former decreases the critical fluid speed slightly in these subsonic flows. The latter is treated as a friction factor, commonly used in pipe flows. For small fluid friction of either compressible or incompressible flow, we show that the channel loses stability by flutter, not collapse. Since these oscillations are unstable, they will grow to large audible amplitude, providing insight into the wheezing and flow limitation mechanisms. The functional dependence of the flutter frequency, F , for incompressible flow is

$$F = \frac{f}{g} \times \frac{(R_0 \gamma)^{\frac{1}{2}}}{2\pi R_w h} \times (E + \left(\frac{2\pi}{\gamma}\right)^4 D)^{\frac{1}{2}} \times \left(\tanh\left(\frac{2\pi b}{\gamma}\right)\right)^{-1}$$

where f , g , R_0 , R_w , h , $2b$, D , E , and γ are the fluid damping, wall damping, fluid density, wall density, wall thickness, channel depth, bending resistance, elastance, and wavelength, respectively. The direct relation to wall elasticity and inverse relation to wall mass are expected. However, the damping ratio, f/g , is a surprising determinant of the motion. We shall discuss the frequency shift of large amplitude waves and flow limitation in the non-linear theory.

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²

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TIME-FREQUENCY SPECTRUMS OF RESPIRATORY SOUNDS
IN ASTHMATIC PATIENTS

Y. Maeda
K. Nitta
Y. Yui
T. Shida

Changes in respiratory sounds in the asthmatic patient will be illustrated with their time-frequency spectrum in bird's-eye view representation.

For this research, several respiratory sounds of normal volunteers and asthmatic patients were recorded in a cassette-type tape recorder using a condenser-type microphone, and stored into a computer memory through A/D conversion (a 16-bit A/D converter was used). Digitalized data samples of respiratory sounds were transformed into time-frequency domain by applying a series of FFT (Fast Fourier Transform) process with a certain time interval, and represented in time-frequency spectrum. The number of data samples at each FFT process was 1024 and the length of the time interval was given by shifting 400 data samples.

The results were as follows:

- 1) for asthmatic patients, several notable humps which correspond to spectral peaks can be observed in the time-frequency spectrum of their respiratory sounds
- 2) those humps appear during expiratory phase, and locate in the frequency band under 2KHz. At the beginning of expiratory phase there rises a simple hump under 1.5 KHz and it branches toward plural humps
- 3) the frequencial location of above mentioned humps are in integral relationship. It suggests that the humps are formed mainly by whistling rale which is a kind of harmonic oscillation.

WHEEZES IN ASTHMATIC PATIENTS: THE NUMBER
OF WHEEZES AND ITS RELATION WITH BREATHING
MANEUVER AND SEVERITY OF AIRWAY OBSTRUCTION

Y. Takezawa
F. Shirai
S. Sawaki
R. Mikami
S. Kudoh
A. Shibuya
N. Aisaka

Thirty three patients with asthmatic attacks were examined to clarify the relationships between the number of wheezes included in one respiratory cycle and breathing maneuver, and severity of airway obstruction. We analyzed wheezes simultaneously recorded at three locations, i.e. on the left and right sides of the chest and on the neck over the trachea, by a phonopneumographic method using a sound-spectrograph. The patients were seated upright and were studied during three different breathing maneuvers: quiet breathing, maximal voluntary ventilation (MVV maneuver), and slow deep breathing (VC maneuver). A total of 102 wheezes in one respiratory cycle were detected in 21 patients during the quiet breathing; 108 wheezes in 32 patients during the MVV maneuver; and 168 wheezes in all of 33 patients during the VC maneuver. The number of wheezes for each patient during the VC maneuver was significantly correlated with FEV_1/FEV predicted % ($r = 0.69$), but there was no such correlation with either the quiet breathing or the MVV maneuver. We concluded that the VC maneuver was the most effective method of detecting wheezes.

WHEEZING IN ASTHMA AND ITS
RESPONSE TO BRONCHODILATORS

R.P. Baughman
R.G. Loudon

Bronchial asthma is usually accompanied by wheezing. To correlate the characteristics of wheezing with the patient's level of respiratory dysfunction and response to bronchodilators, we studied patients with a spontaneous acute asthmatic attack who were treated with a sympathomimetic bronchodilator. Pulmonary function tests were performed. Lung sounds were observed by an electronic stethoscope and recorded on a portable cassette recorder. Observations were made over both apices, both bases, the larynx, and the mouth. Comparison was made of the lung sounds at the same site before and after bronchodilators using the Hewlett-Packard 3582A spectrum analyzer. Two hundred and fifty millisecond segments of the sound wave form for a complete respiratory cycle were analyzed using a fast fourier transform (FFT) technique and a spectral range of 1 to 1000 Hz. By plotting overlapping segments, we were able to quantitate frequency, duration, number, and intensity of wheezes. We compared these values with the forced expiratory volume in the first second (FEV_1). In general, the more obstructed the patient, the longer the duration of the wheeze. This relationship broke down at low flow rates (FEV_1 less than 600 cc).

We also studied the response to bronchodilators. There was no correlation between the frequency, duration, or intensity of the initial wheeze and the change in FEV_1 after bronchodilator. Eight of nine patients showed a significant response in the FEV_1 . Six of the eight lost their initial wheeze. In the other two, the frequency content of the wheeze fell, along with its duration and intensity.

MARGINS OF SPECTRAL VARIABILITY OF LUNG SOUNDS

C. K. Druzgalski

The determination of a spectral composition of lung sounds allows one quantitative differentiation of normal and pathologic auscultatory signs. This diagnostic differentiation is relatively simple in acute or chronic states. However, in multiple disorders or states with occasionally occurring manifestation of the diseased process, or initial stages of a disease the variability of the spectrum of lung sounds defines the sensitivity required to detect a respiratory disorder. For this reason, studies of spectral content variability were conducted including numerical determination of absolute and normalized frequency spectra and their statistical analysis in normal and asthmatic patients at various levels of ventilation.

The experimental procedures included recording and storage of lung sound data and corresponding respiratory airflow, respiratory volume, and movement of the chest wall on an eight-channel tape recorder. The sound data included recording of lobar as well as tracheal and oral respiratory sounds at controlled levels of ventilation. The detailed spectral composition of the sound was determined in the frequency range from 0 to 2000 Hz using real-time processing. Numerical characterization includes spectral magnitude determination in 200 Hz or smaller frequency windows. In addition, predominant frequencies in specific frequency bands were recorded. The data base developed which consists of about 300 numerical values for each studied subject was cross-correlated within studied groups of subjects as well as within the data collected for each subject for different levels of ventilation. Statistical analysis includes determination of mean value and standard deviation of spectral bands as well as segmental curve fitting analysis for studied groups of subjects. Results obtained show clear numerical correlation and define the range of spectral variability of lung sounds and the degree of their dependence on ventilatory conditions for each group of sounds defined as lobar, tracheal, and oral as well as margins of their dynamic range.

It is believed that information derived will improve utilization of quantitative methods, real-time spectral analysis in particular, in lung sound data analysis and define the extent of spectral analysis applicability in clinical practice.

Supported by NHLI Grant HL 25801

VESICULAR LUNG SOUND AMPLITUDE
MAPPING BY FLOW-GATED PHONOPNEUMOGRAPHY

S. Kraman
D. O'Donnell

The relative amplitude of the inspiratory component of the vesicular lung sound at different sites on the chest wall has never been systematically determined in great detail due to the difficulty in repetitively and precisely controlling the inspiratory air flow of the subject. This study was designed to provide very detailed amplitude maps of vesicular sound at precisely controlled airflow rates.

Eight healthy non-smoking males were studied. Each breathed through a heated Fleisch pneumotachygraph. Lung sounds were recorded using a microphone with a plastic chest piece of 1.5 centimeters diameter. The sounds were recorded in two cm steps along the following lines, bilaterally: vertically, anteriorly, six cm from the mid sternum, from clavicle to abdomen (series A); vertically, posteriorly, six centimeters from the spine, from the level of T1 to the base, with the scapulae moved laterally out of the way (series B); horizontally, from the mid sternum to the spine at the level of but not following the anterior fifth intercostal space (series C). The sound amplitude within a 100 ms gate beginning at an airflow rate of 1.3 l/s was automatically determined by computer. The sound amplitude of each breath vs its position in each series was then compiled in graphic form. The results revealed much intersubject variability. The overall trends were: series A) amplitude decreasing with distance from the clavicle; series B) amplitude increasing with distance from T1 and a peak at the lung bases and series C) approximately equal amplitude throughout. Comparison of our findings with anatomical descriptions of chest wall thickness at similar sites to those used in our study suggests that chest wall thickness may play a role in determining lung sound amplitude as does the distribution of ventilation. However, other, as yet undefined factors, also seem to be involved.

Session D

Moderator: W. Ball

- | | | |
|---------|--|---|
| 1:00 pm | Lung Sounds Nomenclature | D. Cugell |
| 1:20 pm | Low Frequency Breath Sounds | R. Urquhart
S. Banham
C. Godley
J. MacLeod
F. Moran |
| 1:40 pm | Sound Transmission Profile in Experimentally Induced Pulmonary Edema | S. Ishikawa
F. Landry
M. Ahluwalia
R. Johnston
D. Donovan
B. Johnston
K. MacDonnell |
| 2:00 pm | Recording and Analysis of Normal and Abnormal Lung Sounds | M. Sudraud
C. Charbonneau
J. Racineux
E. Tuchais |
| 2:20 pm | Recording and Analysis of Lung Sounds in Surgical Specimens and Postmortem Human Lungs | R. Murphy
A. Aaronson
E. Del Bono
B. Weinstein |
| 2:40 pm | Respiratory Adventitious Sounds in Silicosis - A Correlative Study with Physiological and Radiological Abnormalities | M. Munakata
H. Ogasawara
M. Matsuzaki
Y. Minami
M. Inoue
Y. Homma
M. Murao |
| 3:00 pm | Coffee Break | |
| 3:20 pm | Investigation of Amplitude Heterophony in a Presumed Normal Subject | S. Kraman
D. O'Donnell |
| 3:40 pm | Cracklefest | |
| 4:00 pm | Summary of Conference | C. F. Dewey |

LOW FREQUENCY BREATH SOUNDS

R.B. Urquhart
S.W. Banham
C.C. Godley
J.E.S. Macleod
F. Moran

Lung sounds from subjects with (a) asbestosis, (b) cryptogenic fibrosing alveolitis, (c) cardiac failure and (d) normal lungs were recorded using an FM tape recorder. The electret microphone allowed recordings to be made over a wide frequency range. A microphone enclosure was designed to reduce interference from ambient sound, and thus allow recordings to be made in a general hospital ward.

The recordings were digitized by computer and the fast fourier transform was used to obtain spectra. Systematic differences between the shapes of spectra in different groups were apparent (over the low frequency end of the spectrum). Pattern recognition techniques were used to extract information from the spectrum and to construct a scatter diagram. (The scatter diagram was constructed using the two directions of greatest variance in the data set.) The scatter diagram indicated the following groups: (1) asbestosis patients and cryptogenic fibrosing alveolitis patients, (2) cardiac failure patients, (3) normal subjects.

Further investigation gave evidence to suggest that these spectral differences were due to changes in breath sound rather than to adventitious sound. These results may have implications for diagnosis since alteration in breath sound has not hitherto been recognized as important in the diseases studied (1,2).

Subsequent research has concentrated on a more detailed comparison of breath sound in asbestosis with that in normal subjects. In this research, a more sophisticated approach, again by spectral analysis and pattern recognition, has been developed.

It is felt that low frequency breath sounds could provide a source of information complementary to that provided by high frequency events such as crackles.

1. R.B. Urquhart, J. McGhee, J.E.S. Macleod, S.W. Banham and F. Moran. The Diagnostic Value of Pulmonary Sounds: A Preliminary Study by Computer-Aided Analysis, in press. Computers in Biology and Medicine.
2. N. Gavriely and Y. Palti. Breath sounds in emphysema and lung fibrosis - deviations from the normal spectral curves. 5th International Conference on Lung Sounds, London 1980.

SOUND TRANSMISSION PROFILE IN EXPERIMENTALLY
INDUCED PULMONARY EDEMA

S. Ishikawa
F. Landry
M. Ahluwalia
R. Johnston
D. Donovan
B. Johnston
K. MacDonnell

Artificially induced lung sounds were recorded over central airway (trachea) and peripheral airway (uppermost part of the lung on left decubitus position i.e. right lower lobe) during mechanical ventilation in mongrel dogs before and after induction of pulmonary edema with oleic acid. The sound source (solenoid valve) was attached to the proximal end of the endotracheal tube. The solenoid valve was activated with a frequency of 480 cycles per minute and a "jet" of oxygen (20~30 ml) was injected into the endotracheal tube. Recorded sounds were transcribed on the photographic paper with a speed of 100 mm/sec and the amplitude and decay period of the sound wave were measured manually from the paper tracing. The amplitude and decay period of the sound wave recorded increased linearly from outside, central and peripheral airways, in normal lung and in lung with mild degree of pulmonary edema. The slope of amplitude and decay period change became less in lung with moderate degree of pulmonary edema and its relationship between central and peripheral airway was reversed in severe pulmonary edema. The possible mechanisms of this finding and clinical implications will be discussed.

This investigative work was supported, in part, by the Dr. Maurice S. Segal Asthma Foundation Fund.

RECORDING AND ANALYSIS OF NORMAL AND ABNORMAL LUNG SOUNDS

M. Sudraud
G. Charbonneau
J. L. Racineux
E. Tuchais

The recording of breath sound is a very important problem because in most cases, analytical results depend mainly on the acoustical response of the apparatus used. We present here a new system having a perfectly flat bandpass in the range of 0-1500 Hz which covers the most significant part of lung sounds. The recorded signal is digitized with an A-D converter and then analyzed, using Fast Fourier transform (FFT), by computer.

Two kinds of analyses are performed. In both cases, FFTs are taken on 1024 sample blocks shifted from N to N samples where N is usually 128 and over a duration of about 30 seconds. On the one hand, we compute the mean frequency spectrum by separating inspirations and expirations. Normal subjects give similar spectra for inspiration and expiration indicating that the breath is acoustically reversible. While patients suffering from asthma give dissimilar ones even when the wheezes are present in both inspiration and expiration. On the other hand, we calculate the mean frequency and the mean amplitude of the spectrum between 60 Hz and 1260 Hz, for each block. We obtain thus two time-varying functions. From the amplitude curve, it is possible to determine directly and with great accuracy the starting time of each inspiration/expiration. This avoids having to record the flow rate at the mouth which modifies the sound. The frequency curves present strong differences between normal and abnormal subjects. In case of asthma, the variations reach about $\pm 80\%$ of the mean value while in normal case they do not exceed $\pm 25\%$.

RECORDING AND ANALYSIS OF LUNG SOUNDS IN SURGICAL SPECIMENS
AND POSTMORTEM HUMAN LUNGS

R. Murphy, Jr.
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B. Del Bono
B. Weinstein
F. Davidson

The accuracy of diagnostic information obtained by chest auscultation is dependent upon the correlation of specific variation in lung sounds with differing pulmonary pathologic conditions. Precise information on this subject is scant and is difficult to obtain because the exact nature of the pathology underlying the site of auscultation must often be assessed indirectly.

To study the relationship of lung sounds to pathologic changes, we made tape recordings with a microphone placed on the surface of both surgically excised and postmortem lungs. The lung preparations were ventilated by manually driving a spirometer connected by a metal or plastic tube to large airways. Recordings of these sounds were analyzed by time-expanded wave-form (TEW) and spectral analysis and compared to similar analyses of recordings made in the same subjects prior to surgery or autopsy. Subjects with a variety of diseases including chronic obstructive lung disease, interstitial fibrosis and lung cancer were examined in this fashion.

Our preliminary observations indicate that the sounds heard over excised lung tissue are quite similar to sounds heard during routine chest auscultation. Normal breath sounds and bronchial breath sounds are readily distinguished from one another. Continuous adventitious sounds (wheezes, rhonchi) appear to be heard more clearly than during usual auscultation and are flow and volume related. Fine, medium, and coarse discontinuous sounds (crackles, rales) can be detected at approximately the same sites where they are heard in the patient prior to surgery or death. The wave-form characteristics of adventitious sounds are similar in excised lungs to those observed in the intact patient. We conclude that a systematic study of lung sounds on postmortem or surgically excised lungs could provide information useful for improving non-invasive diagnosis.

Supported by Grant HL23318 from the National Heart, Lung and Blood Institute and by the Institute of Occupational and Environmental Health.

INVESTIGATION OF AMPLITUDE HETEROPHONY IN A
PRESUMED NORMAL SUBJECT

S.S. KRAMAN
D. O'DONNELL

A 36 year old male subject, previously found to have several areas of unusually loud inspiratory vesicular lung sounds over the left lung during lung sound mapping was further studied to determine the reason for this. One of the sites, located over the presumed position of the superior segment of the left lower lobe, was chosen. The amplitude at this point was twice that of the homologous segment on the right. A three dimensional amplitude map of this area revealed it to be discrete (+ 4x4 cm). The possibility of a localized increase in chest wall sound transmission was ruled out by determining that the expiratory vesicular sound as well as vocalized sounds were of normal intensity compared with the homologous segment. A ventilation scan revealed an unexpected decrease in ventilation over the segment in question. Flexible fiberoptic bronchoscopy with balloon occlusion of the left superior segment confirmed that it fed the area involved but was otherwise normal as was a bronchogram performed the same day. While we were unable to determine the reason for this localized loud inspiratory vesicular sound, we conclude that some degree of amplitude heterophony, perhaps as great as a 2:1 ratio, may be within normal limits and, in the absence of other signs of disease, should probable be disregarded or simply followed.

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