SEVENTH INTERNATIONAL CONFERENCE
ON LUNG SOUNDS

OCTOBER 7 & 8, 1982

University of California, Davis
School of Medicine

Veterans Administration Hospital
Martinez, California
U.S.A.

PRESENTED BY

International Lung Sounds Association
STEERING COMMITTEE

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David Cugell, M.D.  Chicago, Illinois
Sadamu Ishikawa, M.D.  Boston, Massachusetts
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Raymond L.H. Murphy, M.D.  Boston, Massachusetts
SEVENTH INTERNATIONAL CONFERENCE ON LUNG SOUNDS
Martinez, California

PROGRAM

Thursday, October 7, 1982

Registration ........................................... 8:30 a.m.
Opening Remarks, Dr. P. Krumpe ......................... 9:00 a.m.
Welcome, C. Nixon, Director
Martinez V.A. Medical Center ........................ 9:05 a.m.
Keynote Address, Dr. M. Mori .......................... 9:10 a.m.
Session A - Drs. J. Fredberg & Y. Ploysongsang,
Moderators ........................................... 9:20 a.m. - 12:00 p.m.
Lunch .................................................. 12:00 p.m. - 1:00 p.m.
Session B - Drs. W. Ball & N. Gavriely,
Moderators ........................................... 1:00 p.m. - 4:30 p.m.
Cocktails & Buffet .................................... 7:00 p.m.

Friday, October 8, 1982

Session C - Drs. R. Mikami & S. Ishikawa,
Moderators ........................................... 9:00 a.m. - 11:45 a.m.
Lunch .................................................. 11:45 a.m. - 12:45 p.m.
Business Meeting ..................................... 12:45 p.m. - 1:00 p.m.
Session D - Drs. S. Kudoh & R. Loudon,
Moderators ........................................... 1:00 p.m. - 3:20 p.m.
Cracklefest ........................................... 3:20 p.m. - 4:00 p.m.
Summary, G. Lillington ............................... 4:00 p.m. - 4:30 p.m.
SESSION A

Moderators: J. Fredberg & Y. Ploy-Song-Sang

9:20 am Airway to Pleura Sound Transmission In Excised Lung
D. Rice

9:40 am Sound Wave Propagation in a Liquid-Lined Channel
J. Grotberg
G. Kriegsmann
W. Grayhack

10:00 am What Makes the Lung Behave as a Low-Pass Filter?
N. Honda
M. Mori
K. Kinoshita
S. Koike
S. Murao

10:20 am Analysis of Acoustic Transmission of Respiratory System, Preliminary Data
N. Shioya
Y. Takezawa
S. Kudoh
A. Shibuya
R. Mikami

10:40 am Coffee Break

11:00 am Acoustic Transmission of the Pulmonary System
V. Goncharoff
J. Jacobs
D. Cugell

11:20 am Sound Transmission Profile in Experimentally Induced Pulmonary Edema
S. Ishikawa
M. Simonelli
A. Kenler
R. Johnston
D. Donovan
B. Johnston
M. Pavesi
K. MacDonnell

11:40 am Further Implications of the Stress Relaxation Quadrupole Model of Crackle Generation
J. Fredberg
S. Holford
E. Del Bono
R. Murphy

12:00 pm Lunch
The time it takes a sound impulse introduced into the trachea to travel from the carina to the pleura was measured at many pleural locations in several excised horse and dog lungs. The lungs were inflated to TLC with several gases. The delay time was always greater than it would have been if the sound had travelled the full distance at the free field sound velocity in the inflating gas. This excess time delay was not dependent upon airway length.

Airway sound must propagate some distance in the parenchyma in order to be observed on the pleural surface. Since the speeds of sound propagation are known, the sound path length in each medium can be determined from transit time measurements.

The data suggest that the transition from airway to parenchymal propagation occurs in airways 1 to 2 mm in diameter. Since transition point location is likely to be sensitive to airway characteristics, early detection of small airway disease may be possible using time delay measurements.

Supported in part by NIH, NHLBI, 1R23 HL211199
Sound transmission through the airways has been modelled in the past by using wall characteristics appropriate for flexible tubes, but oscillatory gas flow characteristics that have been derived from rigid tube acoustics. In addition, the liquid lining, prevalent in the tracheobronchial network, has been ignored. In order to account for these effects, we have developed a mathematical model of a two-dimensional, infinitely long channel with flexible, liquid-lined walls. The liquid is considered viscous and compressible. Surface tension is neglected and the channel wall is visco-elastic. By employing the equations of motion we seek travelling wave solutions of the form $e^{i(kx-ft)}$. The propagation constant $k = k_R + ik_I$ is a complex number whose real part (wave number) and imaginary part (decay coefficient) are analyzed as a function of frequency, $f$, and the remaining parameters. The phase velocity and group velocity are also examined and can be much lower than the free-space sound speed.
Low-pass filtering action of the lung has been well known, for which, however, no adequate explanation has been given so far. The purpose of this study is to calculate transmission characteristics of the lung on a theoretical basis and compare the results with those actually measured. Because the acoustic impedance of the bronchial wall is at least 20 times larger than that of the air, the sound generated in the airway does not transmit across the airway but travels within the airway. Since the geometry of the airway is much smaller compared to the wavelength of the lung sound, each bronchus and its bifurcation may be regarded as acoustic inertance and acoustic capacitance respectively. Assuming the airway to branch in symmetrical dichotomous manner and the structure distal to the 17th generation to be spherical (d = 7 mm), we calculated the input acoustic impedance of the subglottal airway ($Z_0$): $Z_0 = j(9.21 \times 10^{-3} - 60.5/\omega)$, where $j = \sqrt{-1}$ and $\omega$ = angular frequency. Calculated transmission characteristics agreed well (within 5 dB) with those of in vivo measurements (Buller and Dornhorst (1956)) and with those actually measured in our airway model. We suggest that the structure of the airway is primarily responsible for the low-pass filtering action of the lung.
Acoustic transmission of the respiratory system was studied using a sinusoidal sound wave (100 - 1000 Hz) introduced into the oral cavity. Intensity of the sounds was analyzed at four locations; at the mouth, over the trachea, at the apical and at the basal position of the chest.

Intensity of the sounds over the trachea and the chest wall increased during the inspiratory phase and decreased during the expiratory phase. In contrast, the intensity at the mouth decreased during inspiration and increased during expiration. It is believed that opening and shutting of the glottis significantly influenced sound transmission from the oral cavity to the trachea.

Sound intensity on the chest, which was normalized by intensity over the trachea, showed rapid attenuation with increasing frequency. The attenuation of sound intensity at the basal position was also observed at the apical position. Frequency response was not significantly different at differing lung volumes.
ACOUSTIC TRANSMISSION OF THE PULMONARY SYSTEM

Vladimir Goncharoff
John E. Jacobs
David W. Cugell

We measured the acoustical impulse response of the lungs and thoracic cage over the frequency range 500 Hz to 10KHz. This was achieved by introducing wideband noise through the mouth and detecting transmitted sound with a microphone placed on the chest wall. The impulse response, as well as frequency amplitude and phase responses were obtained using analog correlation and digital signal processing. This measurement technique has been found to be accurate by testing a specially designed electro-acoustical transducer, and comparing with results obtained from other testing methods.

In general, test results on twenty-two patients show no consistent differences between healthy subjects and subjects with obstructive lung disease in the frequency range 500 Hz to 4 KHz. However, recent tests on five subjects in the frequency range 4 to 10 KHz indicate at least 12 dB/oct. greater dropoff of transmitted signal with increasing frequency in healthy subjects as compared to those with severe obstruction. A phenomenological model will be described that attempts to explain these results.

This work supported in part by NIH Grant #5 RO1 HL25787 and funds from the Monte and Maxine Monaster Foundation.
Artificially induced lung sounds were recorded over central airways (trachea and peripheral airways) during mechanical ventilation in mongrel dogs before and after induction of severe pulmonary edema with oleic acid.

Lung volume and mean airway pressure were kept at the same level and three levels of frequencies were used for ventilation: 15, 120, and 1700 breaths per minute.

The sound source was the ventilatory device, situated at the proximal end of the endotracheal tube. Recorded sounds were transcribed on the photographic paper at a speed of 100 mm/sec., and the amplitude of the sound waves was measured manually from the paper tracing.

During low frequency ventilation, no significant change in sound amplitude was noted before and after induction of pulmonary edema at either central or peripheral airways.

At the higher frequencies (120 and 1700) breaths per minute, the changes in the difference between amplitude length of the central and peripheral airways from pre to post induction of the pulmonary edema was predominately a result of amplitude changes in the central airways.

At the ventilatory frequency of 120 breaths per minute, a significant decrease in sound amplitude was noted in the central airway after the induction of pulmonary edema while at the ventilatory frequency of 1700 breaths per minute, an increase in sound amplitude was observed. There was no significant change in peripheral airway sound at either frequency.

The possible mechanisms of these findings and clinical implication will be discussed.

This investigative work was supported in part by BioMedical Research Development Grant, NIH. 1-508 BR 09223 and the Asthma Foundation.
The stress relaxation quadrupole hypothesis of crackle generation accounts for many heretofore unrelated observations, and leads further to several surprising predictions. In particular, the stress relaxation quadrupole model predicts(accounts for:

1. The existence of initial deflections of both polarities
2. The existence of expiratory crackles
3. A preponderance of inspiratory over expiratory crackle observations
4. A preponderance of positive over negative initial deflections for inspiratory crackles
5. For expiratory crackles the converse of #4 above
6. Simultaneous positive and negative initial deflections for the same crackle at different observation positions
7. Fine crackles being more localized on the chest than coarse crackles

These and other aspects of the model will be discussed in light of existing data.
SESSION B

Moderators: W. Ball & N. Gavriely

1:00 pm Invited Physiology Lecture: "Lung Water and Lung Sounds"  
N. Staub

1:45 pm Validation of Breath Sound Theory  
J. Seiner  
J. Hardin  
J. Patterson  
J. Levasseur  
W. Snell

2:10 pm Spectral Analysis of Breath Sounds in Newborn Infants  
H. Pasterkamp  
F. Leahy  
W. Gibson  
R. Fenton  
V. Chernick

2:30 pm Frequency Analysis of Healthy Children's Breath Sounds  
S. Manabe  
J. Sakamoto  
Y. Saka  
T. Igarashi  
T. Yasuda  
Y. Yoshida  
Y. Ueda  
S. Kudoh  
T. Shibuya

2:50 pm Three Dimensional Contour Mapping of Lung Sounds in Normals  
R. Dosani  
S. Kraman

3:10 pm Coffee Break

3:30 pm Tracheal Sound Recordings in Monitoring of Sleep-Disordered Breathing  
J. Cumminskey  
G. Guilleminault  
P. Krumpe  
C. Williams

3:50 pm Differential Phonopneumography in the ICU  
P. Krumpe

4:10 pm Lung Sound Mapping  
R. Murphy  
E. Del Bono
Validation studies on our breath sound theory were conducted with three Y-tubes precision-machined in Plexiglas blocks, with 70° branch angles and lateral curvatures at the bifurcation equal to 7.5 x the radius of the parent tube (average in human lung). The parent/daughter I.D.s of Y-tubes were: 4.6/4.0, 4.0/3.4, 2.4/2.0 mm. Narrow band sound spectra were recorded in the anechoic chamber during "expiratory" flow.

Sharply defined tones were recorded over the Reynolds number (Re) range of 2300 to 6500 (diameter of parent tube was used in Re calculations). The data indicate the correctness of the original Hardin theory in ascribing breath sounds to the interaction of laminar vortices, with resulting vortex orbiting that generates pressure variations, i.e., sound. No other source of tones was evident over the Re range studied. Frequency of sound was more closely related to \( \sqrt{\text{Re}} \) than to Re itself.

The original Hardin theory, based on inviscid analysis, is being modified to account for viscous effects that produce variation in boundary layer thickness with varying stream velocity. Theoretical analysis for the case of non-equal flow rates in the daughter tubes (certain airway diseases) has indicated the possibility of chaotic behavior of the laminar vortices, with production of more broadband sound than in the equal flow condition.
SPECTRAL ANALYSIS OF BREATH SOUNDS
IN NEWBORN INFANTS

H. Pasterkamp*
F. Leahy
W. Gibson
R. Fenton
V. Chernick

To determine whether breath sounds in newborns are different from those described in older subjects, we used fast Fourier transform and power spectra analysis in 10 newborn infants and compared early, mid, and late phases of inspiration and expiration. Subjects, corrected for gestational ages, ranged from 1 - 20 days, weighed 2.4 - 3.9 kg., and were all free of respiratory disease. Analysis was limited to 50 - 1000 Hz. Average power and peak power were computed in the 100 - 400 Hz range, and the spectral upper limit was defined as the frequency where power attenuated to 10% of peak power. All infants showed peak frequencies below 250 Hz in inspiration and below 200 Hz in expiration. The spectra were within 600 Hz during inspiration and 500 Hz during expiration for 9/10 cases. Early and mid phases of inspiration had similar or greater average power than the same phases of expiration in 8/10 cases, whereas end-expiration power was equal to or greater than in end inspiration for 8/10 cases. Thus, frequency spectra of breath sounds in newborn infants are similar to those seen in older subjects. However, considerable power is still present during the late expiratory phase. It will now be of interest to study the influence of disease on neonatal breath sounds.

*Supported by a grant from the Manitoba Lung Association
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<th>Value</th>
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<td>0.000 +/− 0.01</td>
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<tr>
<td>2</td>
<td>1.000 +/− 0.01</td>
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A frequency analysis was conducted to study the characteristics of children's breath sounds during the transition period from childhood to adulthood. Sixty-nine healthy children, 39 boys and 30 girls, between the ages of 3 and 15, and taller than the minimum height of 90 cm were studied. A recording was made, using a Sony-ECM-150 microphone, placed on the chest between the second and fourth intercostal space of the right midclavicular line of each child while in a sitting position.

The respiratory airflow and amplified second and fourth intercostal sounds were inputed into the data recorder. The recorded breath sounds were band passed between 100-3000 Hz, and analyzed by FFT on an inspiratory phase of 512 msec. The peak frequency (PF) and height (less than 150 cm) showed an inverse linear correlation. Children with a height of more than 150 cm showed an almost constant PF regardless of any further increase in height. The PF at the second intercostal space always showed higher values than that at the fourth intercostal space. There was a tendency towards an inverse linear correlation between the PF and chest circumferences ranging from 50 cm to 75 cm. For chest circumferences exceeding the 75 cm limit, there was also a constant PF. It was concluded that the transition to adult breath sounds ends at approximately age 12 or when growth surpasses 150 cm and chest circumference exceeds 75 cm.
THREE DIMENSIONAL CONTOUR MAPPING OF
LUNG SOUNDS IN NORMALS

Razak A. Dosani
Steve S. Kraman

We have produced three dimensional contour maps of lung sound amplitude at the left and right posterolateral chest wall by means of a new technique (flow-corrected phonopneumography).

We studied 10 nonsmokers (ages 24 to 48 years) with normal spirometry and no history of lung disease. The posterior chest wall was marked with an 18 x 18 cm grid consisting of 100 points. A microphone with a 14 mm chest piece was placed on each point and the sound of a single inspiration was recorded while the subject breathed through a pneumotachograph. This was repeated for each of the 100 points on each side. The sound signals and pneumotachograph output were then processed to generate an airflow corrected lung sound amplitude index for each point. This information was then assembled into a contour map of lung sound intensity.

Analysis of these maps revealed:

1. Marked intersubject variation, some subjects having sounds four times as loud as others at comparable sites.
2. Frequent intrasubject variability in amplitude at adjacent locations.
3. Consistently decreased amplitude over the scapulae.

These results show in graphic form that the amplitude of the inspiratory lung sound is dependent not only on airflow (as shown shown in previous studies) but also on other undefined factors relating to sound production, transmission, or both.
Lung sound amplitude contour maps of 10 healthy male subjects. The amplitude indices of several of these maps have been increased by the degree noted (ie. X 1.5) to allow better appreciation of the contour.
TRACHEAL SOUND RECORDINGS IN MONITORING OF SLEEP-DISORDERED BREATHING

Joseph M. Cummiskey
C. Guilleminault
Peter Krumpe
C. Williams

Sleep-disordered breathing (SDB) is defined as a cessation or reduction of airflow at the mouth or nose for greater than 10 seconds. Despite having numerous methods for monitoring airflow, none is satisfactory on its own. Pattern reading of polysonmographs are used to tabulate SDB. We examined the usefulness of tracheal sounds and ear oximetry compared to the standard thermistors impedance plethysmograph (strain gauges) and ear oximeter in 10 sleep apnea syndrome patients.

Tracheal sounds were monitored by the use of a Littman stethoscope diaphragm taped onto the manubrium sternum. The stethoscope diaphragm was coupled to a one-quarter inch microphone. The signal was filtered (above 200 Hz) using a 24 decibel per octave filter. The signal was rectified and integrated to display a sound envelope. The output gain on the amplifier was adjusted for each patient under standard conditions.

The table shows the number of sleep disordered breathing events per hour during sleep (mean ± SD) measured by thermistors, strain gauges and ear oximeter compared to breath sounds and ear oximetry in 10 sleep apnea syndrome patients, and the mean length in seconds of respiratory events.

<table>
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<th>With Desaturation</th>
<th>Mean Length of Respiratory Event (Seconds)</th>
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<tr>
<td></td>
<td>Apnea</td>
<td>Apnea &amp; Hypopnea</td>
</tr>
<tr>
<td>Thermistors, strain</td>
<td>14 ± 6</td>
<td>10 ± 4</td>
</tr>
<tr>
<td>gauges &amp; ear oximeter</td>
<td></td>
<td>26 ± 3</td>
</tr>
<tr>
<td>Breath sounds &amp;</td>
<td>16 ± 5</td>
<td>7 ± 3</td>
</tr>
<tr>
<td>ear oximeter</td>
<td></td>
<td>22 ± 2</td>
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P N.S. N.S. < 0.05

There was no significant difference in the number of respiratory events associated with desaturation recorded during sleep by the two sets of methods. The duration of respiratory events was significantly longer when recorded by thermistors and strain gauges than by breath sounds. Breath sound recordings can be substituted for thermistors and impedance plethysmography in monitoring for sleep disordered breathing.
DIFFERENTIAL PHONOPNEUMOGRAPHY IN THE I.C.U.

Peter Krumpe

In order to evaluate the use of regional lung sounds to monitor regional airway dynamics, I graphically recorded the integrated (10 millisecond time constant) rectified and filtered (200-600 Hz) breath sound intensity (IB) over homologous lung segments in I.C.U. patients receiving mechanical ventilation. Peak IB and the time delay between right and left peak IB during inspiration were measured and the effects of varying ventilator flow rate, tidal volume and PEEP were studied.

In three "normal" patients (essentially negative x-ray examinations) peak IB amplification over homologous chest areas was adjusted to be similar at 0 PEEP. Breath sounds were homophonous and time delays between R and L IB were absent (< 10 milliseconds). IB increased as inspiratory flow was increased (between 50 and 100 l/min), decreased as PEEP was increased (between 0 and 15 cm H2O), and was uninfluenced by increasing tidal volume (at constant flow from .500 up to 1.50 L).

Two patients with regional pathology were studied (one had extrinsic tumor compression of the left lower lobe bronchus and the other had a flail segment of the lateral chest wall). In these patients peak IB was delayed by 200 milliseconds over the affected region, thus documenting a longer time constant for inspiration of the underlying lungs.

The amplitude of IB decreased less over the area of extrinsic airway compression than over its homologous lung region as PEEP was increased, suggesting a fixed airway obstruction in this area.

By contrast the amplitude of IB decreased more over the flail chest segment than its homologous area as PEEP was increased, suggesting that airway narrowing due to low lung volume under the unstable chest wall segment was preferentially improved by PEEP.

These data demonstrated that differential phonopneumography is possible in I.C.U. ventilator patients and suggests that regional lung sounds can provide useful information about underlying regional dynamics.
Lung sound intensity, timing of adventitious sounds, and duration of inspiration in relation to the duration of expiration were observed at 48 designated locations over the chest wall and at the mouth of 34 patients. Notations were made on a prescribed form after each observation at each site providing a visual display of the auscultatory findings or "lung sound map." The accuracy of the technician making these observations had been assessed previously in studies of inter- and intra-observer variability and by comparison of the technician's findings with results of time expanded waveform analysis. This accuracy averaged 90% (Kappa .7 to .8).

Three chest physicians interpreted 40 lung sound maps from these 34 patients without knowledge of the results of chest x-rays, pulmonary function tests or other clinical information. Their interpretations agreed with the clinical diagnoses in 75%, 72.5%, and 55.0% of cases respectively.

Additional observations on reviewing the lung sound maps include:

1. over the trachea the expiratory sound was perceived to be equal or longer in duration than inspiration in all patients.

2. over the chest wall inspiration was perceived to be shorter than expiration in most patients. When expiration was longer than inspiration in more than a few central sites, symptomatic bronchial asthma was usually present.

3. In the upper half of the chest, expiration equalled inspiration more often than at the base.

4. Sound intensity estimates were difficult to evaluate. The intensity at the mouth was increased in asthmatic patients as reported by Forgacs. Decreased intensity at the mouth was more difficult to evaluate. The expiratory sound was commonly absent at the bases. When absent at locations higher than the bases, chronic obstructive lung disease was frequently present.
5. Fine end inspiratory crackles when few in number were difficult to evaluate in terms of diagnostic specificity.

6. Patients with bronchitis and classical early medium crackles also had fine end inspiratory crackles; patients with interstitial fibrosis and many pan inspiratory or late inspiratory fine crackles also often had medium or coarse crackles. Whether this was because multiple diseases were simultaneously present or whether it represented the spectrum of sounds in a given disease was often difficult to assess.

7. Two patients with interstitial fibrosis who responded to therapy showed a marked decrease in number of positive sites for crackles.

Our preliminary analysis of lung sound maps suggests that they are an inexpensive but useful method for assessing respiratory status and documenting the course of some lung illnesses. In addition, by requiring a systematic tabulation of auscultatory findings with minimal loss of information due to memory, they point out areas where additional study is necessary.
SESSION C

Moderators: R. Mikami & S. Ishikawa

9:00 am Variation of the Frequency Distribution of Breath Sounds with the Flow Rate
C. Charbonneau
J. Racineux
M. Sadraud
E. Tuchais

9:20 am Lung Sound Monitoring in Asthmatics
R. Baughman
R. Loudon

9:40 am Tracheal Auscultation in the Differentiation of Whistling Sounds Heard at the Chest
H. Husodo

10:00 am Subsonic Flapping Flutter
J. Grotberg
E. Reiss

10:20 am Coffee Break

10:40 am The Effect of Mild Airways Obstruction on the Site of Origin of Lung Sounds
S. Kraman

11:00 am Special Lecture on Acoustics: "The Perception of Localization of Sounds"
R. Efron

11:45 am Lunch
VARIATION OF THE FREQUENCY DISTRIBUTION
OF BREATH SOUNDS WITH THE FLOW RATE

G. Charbonneau
J. Lacineux
M. Sudraud
E. Tuchais

We recorded breath sounds at the trachea of 11 normal subjects and 12 clinically identified as asthmatic subjects at the flow rate .51/s and 11/s. The recorded signal is digitized at 5120 Hz and analyzed using Fast Fourier Transform.

For each subject, the digitized sound is divided into 1024 sample blocks shifted from 128 to 128 samples and the frequency spectrum is computed. The sum term to term of all these spectra is calculated by separating inspirations and expirations at the two flow rates. Both for normal and asthmatic cases, increasing the flow rate induces stronger components of high frequency, especially in the expiratory spectra. There seems to exist a critical flow rate (variable from one subject to another); above it high frequency components become stronger in the spectra.

For each shifted frequency spectrum, we compute the mean frequency F.M. over the range 60 to 1260 Hz. Plotted versus time with the flow rate and the slope of the flow rate, this curve presents similar variations to the slope of the flow rate. This indicates that the air acceleration is an important parameter for the breath sounds production. In these curves, we can also notice that the peak values of the F.M. curves mostly occur in the middle of the expiration while the minimum values appear at the end of the inspiration.
We have previously shown that duration of wheeze over the total breath cycle (Tw/Ttot) correlated with the level of obstruction. This was most apparent in the individual patient studied before and after bronchodilators. We have developed a system to scan lung sound recordings for three minutes and then estimate the Tw/Ttot.

We studied 10 asthmatics in acute bronchospasm with reduced forced expiratory volumes in one second (FEV₁) and wheezing. A microphone was strapped to the patient's back and lung sounds were recorded for 20 - 30 minutes on a portable tape recorder for subsequent analysis.

Lung sound recordings were played back into a Hewlett-Packard 3582A Spectrum Analyzer which analyzed 250 msec of sound and performed a frequency amplitude plot over a 0 to 1000 Hz frequency range. Fifty segments were randomly taken from a three minute segment of lung sound recording. The 250 msec sound segments were analyzed for the presence of one or more peaks with a frequency greater than 200 Hz which we call wheezes from our previous work. The number of segments with wheezes over the total number analyzed was assumed to be an index of Tw/Ttot. We confirmed our previous observation that the Tw/Ttot correlated with the degree of airway obstruction (FEV₁) (R = 0.68, p < 0.05).

The Tw/Ttot of all subjects were different by the method of analysis of variance (\( F = 52.6, P < 0.001 \)). The coefficient of variation for individual patients ranged from 5.8 - 19.3%.

We conclude that: 1) our method of analysis is workable, 2) the Tw/Ttot of individual patients is reproducible, 3) the Tw/Ttot of various patients are different, reflecting the different degrees of airway obstruction.
TRACHEAL AUSCULTATION IN THE DIFFERENTIATION OF WHISTLING SOUNDS HEARD AT THE CHEST

H. O. Setiono Husodo

High-pitched sounds heard at auscultation of the chest can roughly be divided into two groups. One group has the following characteristics:

1. It is heard mostly during inspiration
2. Its place in the respiratory phase is not constant
3. It will disappear or change its location at the chest after forced expiration (coughing) or even after a change of body position
4. It is not associated with forced expiration
5. The sound is often interrupted
6. Distribution at the chest is patchy, random and not constant
7. It is more audible over the chest during auscultation than during auscultation over the trachea

This group represents the high-pitched sounds supposed to be caused by tenacious mucous in the bronchial lumen and can be called the bronchitic squeaking sounds.

The other group shows the following peculiarities:

1. It is heard exclusively during expiration
2. The respiratory phase is constant
3. It is intensified or provoked by repeated forced expiration and rapid respiration
4. It is always accompanied by forced expiration
5. The sound is continuous
6. It is audible over the whole lung and is continuous
7. It is relieved by bronchodilators
8. It is more audible on tracheal auscultation than on auscultation over the chest

This group of sounds is supposed to be generated at narrowed bronchial lumens caused by bronchial wall alterations as found in bronchial asthma. It is properly termed the "wheeze."

Bronchitic squeaking sounds are better heard over the chest while wheezing is best heard over the trachea.
SUBSONIC FLAPPING FLUTTER

J.B. Grotberg
E.L. Reiss

This presentation serves as an update of our theory, discussed at the last meeting, for flow induced oscillations of a model airway, i.e., wheezing. A flexible, two-dimensional channel with visco-elastic walls loses stability by flutter when a viscous gas flows through at the critical flutter speed, $V_F$. The oscillations have a critical wavelength, $L_F$, and a critical flutter frequency, $F$, which are determined by the analysis. The dependence of these values on the remaining parameters corresponding to wall and gas properties is discussed. Experimental data from the literature will be interpreted in terms of the flutter theory and a sample calculation of $V_F$, $L_F$, and $F$, appropriate for airways, will be shown.

\[
F = \frac{a V_F}{L_F}
\]
\[
V_F = \frac{((E_b/R_f)(1+Bk^4)\tanh k)}{(m a^2 k^2 \tanh k + k (1-a)^2))^{3/2}}
\]
\[
L_F = \text{determined from minimum of } V_F(k)
\]

\[
a = \frac{G}{(G + M k \tanh k)}
\]
\[
2b = \text{channel depth}
\]
\[
B = \frac{D}{(E b^4)}
\]
\[
D = \frac{Y h^3}{(12(1-n^2))} \text{ wall bending stiffness}
\]
\[
E = \text{wall elastance}
\]
\[
f = \text{fluid damping coefficient (\(0.003\) plane Poiseuille flow)}
\]
\[
G = \frac{f}{g}
\]
\[
h = \text{wall thickness}
\]
\[
k = 6.28b/L
\]
\[
L = \text{wavelength}
\]
\[
m = \text{fluid viscosity}
\]
\[
M = \frac{R_w h}{(R_f b)} \text{ mass ratio}
\]
\[
n = \text{Poisson's ratio}
\]
\[
R_f = \text{fluid density}
\]
\[
R_w = \text{wall density}
\]
\[
Y = \text{elastic modulus of wall}
\]
THE EFFECT OF MILD AIRWAYS OBSTRUCTION ON
THE SITE OF ORIGIN OF LUNG SOUNDS

S. S. Kraman

The site of origin of the inspiratory vesicular lung sound is not precisely known. Recent studies support the notion that this sound emanates from the larger airways where turbulence is known to exist. In order to determine whether the site of lung sound generation changes in mild airways obstruction, a study was designed using a technique that yields a comparative estimate of the site of lung sound origin. Forty-four subjects without known lung disease between the ages of 19 and 53 years (mean 29 years) and with smoking histories of 0 to 65 years (mean 8 pack years) were studied by spirometry, He-air V/V loops and single breath N2 washout. In addition, the degree of similarity between inspiratory lung sounds recorded by two microphones spaced 1 to 8 cm apart at the lung bases was determined by a flow-gated, automated sound subtraction technique. This sound subtraction intensity index (SII) was then plotted against the pulmonary function testing results (% of predicted) and analyzed by linear regression.

The best correlations were between the SII at 2, 5, and 6 cm intermicrophone separation at the right lung base and the % forced expiratory volume at one second (FEV1/FVC, \( r = 0.436, P < 0.01 \)), FEV1 (\( r = -0.386, P < 0.01 \)) maximal mid-expiratory flow (FEF25-75, \( r = -0.390, P < 0.01 \)), peak flow at 75% FVC (\( V_{\text{max}} \) 75% VC, \( r = 0.290, P < 0.01 \)), and \( V_{\text{max}} \) 50% VC (\( r = 0.371, P < 0.02 \)). There was no statistically significant correlation between any lung parameters and the forced vital capacity, difference in flow at 50% FVC on 80% He vs air, closing volume, slope of phase 3 of N2 washout, volume of isoflow, smoking pack years, age or sex. These correlations indicate that the sounds recorded by the two microphones became less similar (suggesting that the site of origin of the inspiratory vesicular sound moved toward the peripheral airways) in mild airways obstruction. Interestingly (and unexplained), there was no significant correlation between any pulmonary function parameter and any lung sound parameter measured at the left lung base.
SESSION D

Moderators: S. Kudoh & R. Loudon

1:00 pm  Spectral Characteristics of Lung Sounds in a Dried, Inflated Dog Lung, A Potential Model for Lung Sound Research  
N. Gavriely

1:20 pm  A New Method for Classifying Discontinuous Adventitious Lung Sounds  
M. Matsuzaki  
H. Ogasawara  
M. Munakata  
Y. Minami  
S. Fujiya  
Y. Homma

1:40 pm  Auscultatory Percussion by the Use of a Mechanical Percussor  
M. Mori  
N. Honda  
K. Kinoshita  
H. Morinari  
S. Koike

2:00 pm  Crackles in Excised Pig Lung  
F. Davidson  
R. Murphy  
E. Del Bono

2:20 pm  Dispersion of the Lung and Heart Sounds Characteristics  
C. Druzgalski  
J. Eddleman  
A. Wilson

2:40 pm  Audio and Electronic Evaluation of Breath Sounds  
S. Ishikawa  
A. Kenler  
M. Simonelli  
B. Johnston  
J. Zebniak  
S. Maskwa  
K. MacDonnell

3:00 pm  Coffee Break

3:20 pm  Cracklefest: Auditory presentations by participants to include:  
1) Hamman Sign Mimicking  
2) Post Tussive Crackles  
3) Lung Sounds During Suctioning  
P. van Spiegel  
R. Murphy  
P. Krumpe

4:00 pm  Summary of Conference  
G. Lillington
SPECTRAL CHARACTERISTICS OF LUNG SOUNDS IN A DRIED, INFLATED DOG LUNG, A POTENTIAL MODEL FOR LUNG SOUND RESEARCH

Noam Gavriely

In order to produce a physical model for the study of lung sounds under conditions of constant lung volume and flow rate we studied the spectral characteristics of sounds generated by constant (inspiratory or expiratory) flow through a dried, inflated, and decorticated dog lung.

An excised dog lung was inflated to 30 cm H₂O and dried. Random noise (0.5 - 1000 Hz) was applied to the tracheal opening and picked up by two 21050 A HP contact sensors from the trachea and left lower lobe. The transfer function, phase and coherence were calculated to give the attenuation properties of the lung. It was found that the dried lung acts as a lowpass filter with 3 dB point around 200 Hz. The lung was then decorticated by removing the pleura allowing for constant inspiratory or expiratory flows at constant volume and flow rates. The amplitude spectra of the "inspiratory" sounds picked up over the trachea and left lower lobe were flat to 800 and 200 Hz respectively and rolled off at 24 and 12 dB/oct respectively thereafter. The coherence of the inspiratory sounds was low (< 0.5) suggesting that the surface sounds are not directly related to the tracheal sounds. The spectra of the expiratory tracheal sounds were similar to the inspiratory ones. However, the intensity of the surface sounds were reduced to 20-25% of the inspiratory ones. The coherence in the expiratory case was over 0.7 between 200 to 800 Hz suggesting direct relationships between the surface sounds and the tracheal ones. It is apparent that the dried, inflated lung preparation may be a useful model for the study of the physics of normal lung sounds.
A NEW METHOD FOR CLASSIFYING DISCONTINUOUS ADVENTITIOUS LUNG SOUNDS

Michiyuki Matsuzaki
Hideki Ogasawara
Mitsuru Munakata
Yoshitsugu Minami
Shuichi Fujiya
Yukihiko Homma

Two new indices for an objective sorting of discontinuous adventitious lung sounds (rales) were measured from time-expanded waveforms of the rales from 20 patients with fibrosing lung disease (FLD) and from eighteen patients with chronic airflow obstruction (CAO). One index is a period from the starting point of the waveform to its first peak (1/4 cycle duration: Tb) and the other is one to the third peak (9/4 cycle duration: Tf). Ten types of model rales from a lung-sound teaching tape by Murphy and a record by Ueda were also studied. To clarify pathophysiological meanings of these indices, body mass index (BMI) and some parameters for pulmonary function (VC, FEV1, DLCO, PAO2 and PACO2) were simultaneously measured and correlative study with Tb or Tf was performed.

1) These indices were easy to be measured because the values were not affected by the trend of the baseline and showed a good reproducibility. 2) On Tb-Tf two dimensional plane, crackles from FLD patients (Fcr) and those from CAO patients (Ccr) were clearly divided to form an oval-shaped area individually separated by a vertical line Tb = 0.5 msec. "Crepitant rales" (Ueda) and "fine crackles" (Murphy) located in the lower portion of Fcr area, the rest of the model crackles distributed over Ccr area widely. 3) Significant positive correlations were found between Tf and VC (p < 0.05), Tf and BMI (p < 0.05) in idiopathic interstitial pneumonia, Tb and FEV1 (p < 0.05), Tf and FEV1 (p < 0.01), and Tb and BMI (p < 0.01) in Ccr. 4) An experimental study proved that positive correlations between BMI and Tb or Tf are due to a filtering effect on higher frequency components proportional to the thickness of transmission media. 5) Correlations between spiographic data and these two indices were discussed in relation to mechanisms of crackle generation and transmission. 6) Our indices seem to have better specificity for differentiating rales than IDW method by Murphy. Because of simplicity of measuring and correlation with some functional parameters, these indices have a greater advantage for clinical application.
AUSCULTATORY PERCUSSION BY THE USE
OF A MECHANICAL PERCUSSOR

Masashi Mori
Norinari Honda
Kojiro Kinoshita
Hajime Morinari
Shigeo Koike

Auscultatory percussion was first introduced by Guarino over 25 years ago (J Kansas Med Soc 75: 193-194, 1974). The procedure is simple. The examiner directly percusses over the manubrium while listening with the stethoscope at both lung fields to detect differences in sound transmission. To make the study more objective we tried simultaneous recordings of the transmitted signals by microphones placed at both lung fields. In normal subjects the signals transmitted almost symmetrically but in patients with lung diseases the transmission was often asymmetrical. However, we found it difficult to reproduce the same input signals by manual percussion. For this reason we made a mechanical percussor by which we can produce pulse-like signals of the same quality. To study transmission characteristics of the lung, white noises or vocal sounds have been used as input signals. However, a significant portion of white noises applied at the mouth is reflected by the structures in the upper airway and vocal sounds are narrow-band signals compared to the percussion sounds. We conclude, therefore, that auscultatory percussion is a better and more practical method for the study of transmission characteristics of the lung.
CRACKLES IN ISOLATED SEGMENTS OF EXCISED PIG LUNGS

Frank Davidson
Elizabeth Del Bono
Raymond Murphy

We studied the transmission, waveform characteristics and histologic correlations of crackles in excised pig lung. Lung segments were inflated by Swan Ganz catheter while recordings were made simultaneously over adjacent and opposite sides of the inflated segment with microphones 3 cm in diameter. Crackles were heard over the inflated segment but not from the immediately adjacent microphone. The direction of the initial deflection of associated crackles noted from microphones on opposite sides of the lung were similar. Waveforms of these crackles at the initiation of inflation showed similar patterns at each microphone whereas at the end of the inflation the patterns differed suggesting that the crackles were occurring first at a distance from each microphone and subsequently closer to one as compared to the other.

The histologic characteristics were examined from areas of lung over which crackles were heard as well as over areas where crackles were inaudible. Lung specimens were frozen at known points on pressure-volume curves. Alveolar size and the number of airways open, partially open, and or closed were estimated for each specimen. At high lung volumes when no crackles were present most airways were open. At low lung volumes where the lung would crackle if inflated airways were more commonly closed. Crackle hysteresis was observed. Namely inflationary crackles occurred when the lung was being inflated but not at the same volumes when the lung was being deflated. At the mid point of inflation when crackling was occurring, 29% of airways were open and 71% were closed or partially closed. Due to inadequate sample size we were unable to show that the state of the airways differed at a similar volume during deflation when there were no inspiratory crackles. This will require further study. In any case, excised pig lung appears to be a useful model for studying the mechanism of production of crackles.
Respiratory, as well as cardiovascular sounds, contribute to the definitive composition of the acoustical signals recorded over the chest wall. The degree of each component contribution depends on the location of the recording side as well as on peculiar characteristics of these sounds. Thus, the presence of cardiovascular sounds, due to their relatively high intensity and similarity in spectral properties, obscures objective determination of respiratory sounds characteristics.

In order to overcome this limitation, a computational method of correlation between spectral characteristics of lobar respiratory sounds and respiratory airflow and/or volume and electrocardiogram was developed. This information can be viewed on a monitor at selectable compression rate and selectable number of sound spectra per respiratory cycle and ECG waveform. Further, consecutive sound spectra and/or spectra selected in reference to specific phase of respiration or electrocardiogram can be used to obtain averaged characteristics or assess the variation of spectral characteristics in these selected phases. Thus, the utilization of the difference in periodic character of the respiratory and cardiovascular systems allows one to obtain averaged spectral characteristics attributable to respiratory or cardiovascular sounds.

The application of a personal computer to perform this task and its interface with instrumentation monitoring physiological data and performing spectral analysis make it very flexible in addition to a large and convenient data storage and retrieval capabilities. This approach in sound data analysis should improve assessment of acoustical signals detected over the thorax.
During quiet breathing, the breath sounds of healthy non-smokers and patients with lung diseases were recorded using an electronic stethoscope and a stereo tape recorder. The central airway (trachea) along with six peripheral sites was monitored. The recordings were then transcribed onto photographic paper at a speed of 100 mm/sec. Sound amplitude was measured manually from the tracings.

From the recorded lung sounds, breath sound intensity (BSI) evaluation was conducted by two observers. The sound intensity was noted on a grade rating scale (0-4), very similar to the one employed by Pardee et al (1976).

Standard pulmonary function tests were performed following breath sound recordings.

- There was much inconsistency with actual amplitude length and BSI score even among healthy nonsmokers.

- Between the two evaluators, inconsistent and different BSI scores were obtained.

- In the central airway of a healthy non-smoker, the amplitude length of an inspiration was smaller than that of its corresponding expiration. In the peripheral airway of a healthy nonsmoker, we found the reverse to be true.

- In healthy nonsmokers the relationship between inspiration and expiration is virtually the same when recording with or without the diaphragm. However, the amplitude and intensity score without the diaphragm appears to be lower than those scores with the diaphragm in healthy nonsmokers.

- Correlation between sound intensity and ventilatory function will be discussed.

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