ELEVENTH INTERNATIONAL CONFERENCE
ON LUNG SOUNDS

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SEPTEMBER 17-19, 1986

VETERANS ADMINISTRATION MEDICAL CENTER
LEXINGTON, KENTUCKY

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ELEVENTH INTERNATIONAL CONFERENCE ON LUNG SOUNDS
HARLEY HOTEL
LEXINGTON, KENTUCKY

PROGRAM

Wednesday, September 17, 1986

Workshop - Problems in Lung Sound Nomenclature
Moderators: Raymond Murphy and David Cugell
Signal Processing Demonstration
Y. Ploysongsang

Thursday, September 18, 1986

Registration -----------------------------------------------
Opening Remarks - Steve Kraman ------------------------------
Keynote Address - Robert Baughman -----------------------------
Session A  David Rice & Peter Krumpe, Chairmen -------------
Photograph -----------------------------------------------------
Lunch -------------------------------------------------------------
Session B, Part I Masashi Mori & Marc Desmeules, Chairmen ----
Special Lecture - Japan Revisited - David Cugell -------------
Session B, Part II -----------------------------------------------
Cocktails and Buffet at the Spindletop --------------------------

Friday, September 19, 1986

Session C Shoji Kudoh & Steve Kraman, Chairmen -------------
Debate:
Resolved: The clinical value of lung sounds in 1986 is minimal
Pro: Frank Davidson
Con: Wilmot Ball
Moderator: Y. Ploysongsang

Lunch -------------------------------------------------------------
Business Meeting -----------------------------------------------
Session D Robert Loudon & Gerard Charbonneau, Chairmen ------
Cracklefest ------------------------------------------------------
Summary - N.K. Burki ---------------------------------------------
SESSION A
David Rice and Peter Krumpe, Chairmen

9:15 - 9:35  The relationship between forced expiratory flow and tracheal sounds  M. Mori

9:35 - 9:55  Can the flow rate be evaluated from breath sounds?  G. Charbonneau

9:55 - 10:15  Tw/Ttot estimated from simultaneous neck and chest-wall recordings  R. Baughman

10:15 - 10:35  COFFEE BREAK

10:35 - 10:55  Detection of tracheal stenosis by means of frequency analysis of tracheal sounds  M. Yonemaru

10:55 - 11:15  The determination of the resonant frequency of the respiratory system above 50 Hz  S. Kraman

11:15 - 11:35  Pulsed sound in the lung: Reflections, multiple delays and interference  D. Rice

11:35 - 11:55  Reduction of heart sounds from recorded lung sounds by the adaptive filtering technique  V. Iyer

11:55 - 12:10  PHOTOGRAPH

12:10 - 1:30  LUNCH
THE RELATIONSHIP BETWEEN FORCED EXPIRATORY FLOW
AND TRACHEAL SOUNDS

M. Mori
M. Ohno
M. Iguchi
T. Hisada
H. Kino
S. Koike

To see the relationship between the flow and tracheal sound we did
simultaneous measurement of forced expiratory flow and tracheal
sound in thirteen subjects, eleven normal and two with obstructive
lung disease. Total of twenty measurements were subjected to the
analysis. The sound signals were recorded on an FM tape recorder
(TEAC, XR510), and later band-pass filtered (0.08-2 kHz),
digitized (sampling rate:5kHz) and displayed. Waveforms of the
forced expiratory flows and the tracheal sounds were compared and
power spectra of tracheal sounds were obtained by FFT (1024 or 512
points fast Fourier transform). In thirteen out of twenty
measurements the time the amplitude became maximum was delayed by
about 30 ms compared to the flow. In the spectral analysis of
tracheal sounds we observed, as the flow decreased, development of
line spectrum in each subject. The frequency of this line
spectrum ranged between 0.419 to 1.084 kHz and there was a
positive correlation between this frequency and peak flow
(r=0.855). The origin of these line spectra are most likely
vortices in the upper airway developed during forced expiration,
and the results support our theory that the formation of vortices
is the primary reason for the development of flow limitation.
CAN THE FLOW RATE BE EVALUATED FROM BREATH SOUND?

G. Charbonneau
M. Sudraud
G. Soufflet

We recorded lung sound and flow-rate for 7 normal subjects (3 male and 4 female). Sound was picked up at the trachea with a sensitive microphone held in a small probe. Flow-rate was measured at the mouth using a Fleisch #3 pneumotachograph. Subjects were told to breathe during about 30 sec. with an increasing peak flow-rate starting from apnea to around 2 l/s. Both sound and flow-rate were directly digitized (i.e. without temporary analogic recording) at a sampling rate of 5120 Hz. Sound and flow are then divided in 128 sample blocks. For each block the frequency spectrum is computed using the fast Fourier transform. Frequency spectrum depends on the flow-rate in many ways. Different observations which will be discussed have led us to compute on each spectrum the following formula:

$$S = K^* \frac{F_{mean}}{1 + \frac{A}{A_{mean}}}$$

where A and K are constant, F mean and A mean are respectively the mean frequency and the mean amplitude of the spectrum computed on a 128-sample block.

S may be considered as an evaluation of the flow-rate each 50 ms. Plotted versus the measured value of the flow-rate, S shows a linear relationship. This feature can be used as an almost instantaneous evaluation of the flow-rate, or it is possible to compute the mean of S over consecutive 128-sample blocks. This leads to calculating the mean of the flow-rate during 100 ms, 200 ms, ...800 ms. Of course, the longer the time window the better the correlation between computed flow and real value. This correlation can reach 0.96 in the best conditions.
Previous work in our laboratory has demonstrated that the level of airway obstruction in asthmatics is related to the estimated $\text{Tw}/\text{Itot}$, determined by random sampling analysis of lung sounds (Chest 1985; 88:364-368). In our previous work, we used a microphone over the right anterior chest. Others have reported the utility of listening over the trachea to evaluate wheezing in asthmatics. We therefore compared the lung sounds recorded by a stethoscope over the right anterior chest wall to those heard over the trachea of five asthmatics who were studied as part of a sleep study of nocturnal asthma. The sounds were recorded directly on a tape recorder and subsequently played back and analyzed using the previously described technique. Briefly, five minute segments were analyzed by taking 50 random samples of 250 msec in duration of the time amplitude signal. Each sample was analyzed using the Fast Fourier Technique (FFT) and the frequency spectrum from 0 to 1000 Hz was calculated. Each spectrum was analyzed for the presence or absence of a peak at a frequency between 150 and 1000 Hz. The number of spectrums with peaks divided by the total number of spectrums analyzed (50) was the estimated $\text{Tw}/\text{Itot}$. The estimated $\text{Tw}/\text{Itot}$ for both the chest and neck recordings was calculated. Comparisons were then made of the 52 five minute segments of sound for the five patients analyzed. There was a good correlation ($r=.78$, $p<0.001$) between the estimated $\text{Tw}/\text{Itot}$ ratios from the neck and chest. We conclude that either chest or neck positions may be used to record lung sounds in asthmatics for subsequent estimated $\text{Tw}/\text{Itot}$ analysis.
DETECTION OF TRACHEAL STENOSIS BY MEANS OF FREQUENCY ANALYSIS OF TRACHEAL SOUNDS

M. Yonemaru
T. Kawashiro
T. Yokoyama
K. Kikuchi

Thyroid cancer occasionally protrudes into tracheal lumen or invades recurrent nerve. In these clinical conditions, changes in tracheal sounds due to tracheal or laryngeal stenosis are often recognized. The aim of the present study is to investigate the difference in frequency content between tracheal sounds of healthy subjects and those of patients with tracheal stenosis. Tracheal sounds and mouth flow were simultaneously recorded from healthy subjects and patients with tracheal stenosis caused by thyroid cancer. Patients' tracheal sounds were recorded before and after surgical relief of tracheal stenosis. Inspiratory flow was 0.2 l/sec. Signals of tracheal sounds were digitized at a sampling rate of 10 kHz with a precision of 12 bits. The FFT was applied to data tables of 1,024 points. Above 500 Hz, the highest spectral peak of stenosed tracheal sounds (-19±8 dB, n=12) was significantly greater than that of non-stenosed tracheal sounds both of healthy subjects (-44±9 sV, n=25) and of operated patients (-35±9 dB, n=17). The results indicate frequency analysis of tracheal sounds is applicable to detection of tracheal stenosis caused by thyroid cancer.
The natural mechanical resonant frequencies of the respiratory system are of interest to physiologists because of their relevance to respiratory mechanics and to the phenomenon of high frequency ventilation. Most studies have evaluated the lung at frequencies no higher than 50 Hz. Because the normal sounds made by the lungs during breathing have frequency components of up to 500 Hz, it is reasonable to assume that resonances, manifest by enhanced acoustic transmission, may extend above 50 Hz. The present study was designed to evaluate the lung for resonant frequencies between 50 and 1200 Hz. Five healthy subjects were studied. Sound was introduced through an acoustic driver and mouthpiece and was picked up from the neck overlying the trachea and four locations over the posterior chest wall (upper and lower chest, bilaterally). Resonant frequencies were determined by calculation of the transfer function between signals detected at the trachea and each of the four chest locations. The input signal was a 25 Hz square wave. A square wave was used instead of the more conventional white noise source in order to concentrate more acoustic energy at lower frequencies where the resident thoracic noise is greatest and thereby improve the signal/noise ratio (S/N). The harmonics, at 25 Hz intervals, cover the rest of the frequency range of interest. Preliminary experiments demonstrated that a 25 Hz square wave signal provided a S/N at the chest wall) of approximately 7 dB greater (between 50 and 200 Hz) than a pseudorandom white noise source of similar input power. The transfer functions were measured and compared with the subjects breathing air and again after breathing a mixture of 80% helium and 20% oxygen (He-02). Results: on air, all transfer function plots showed one or more peaks between 50 and 167 Hz with variable roll-off of transmission up to 400 Hz. Virtually no energy was detected above this frequency. For the group, the mean frequency of the major amplitude peak was 81 Hz (range: 50 to 167 Hz) at the upper chest wall locations and 64 Hz (range: <50 to 75 Hz) at the lower. With He-02, the transfer function plots were similar or nearly identical to those using air and showed no consistent differences in shape or frequency of the amplitude peaks of the transfer functions. Conclusions: The human adult respiratory system appears to contain acoustic resonances at one or more discrete frequencies up to approximately 160 Hz. In these subjects transmission of sound was very poor above 400 Hz up to the maximum studied frequency of 1200 Hz.
We sent sound pulses with frequencies between 60 and 1000 Hz into the mouth and recorded them over the trachea and on the chest. Reflections within the trachea increase the duration of the tracheal pulse. The frequency of the tracheal pulse was raised when the driving frequency was less than 200 Hz and lowered when the driving frequency was greater than 250 Hz. Signals recorded on the chest are often suppressed at their beginning, center, or end. The time delay of the chest signal can change 100% (jump) with a microphone position change of 5 mm. These jump points move with changes in lung volume or posture. These observations indicate that there are both fast and slow modes of sound propagation in the lung and that adjacent regions of the lung can transmit signals which are delayed differently and interfere with one another. Care must be taken when using either random or continuous wave input signals because the above effects are hidden and may cause negative time delay estimates from cross correlation. On the other hand, shape analysis of the leading edge of the pulses provides repeatable estimates of time delay. At 250 Hz the patterns of delays appear to be linked to the segmental and subsegmental bronchi.
REDUCTION OF HEART SOUNDS FROM RECORDED LUNG SOUNDS
BY THE ADAPTIVE FILTERING TECHNIQUE

U.K. Iyer
P.A. Ramamoorthy
H. Fan
Y. Ploysongang

Auscultation of the chest, owing to its simplicity and non-invasive nature, is an attractive diagnostic method used by physicians. To increase its usefulness and objectivity in pulmonary diagnosis, there is growing interest in lung sound analysis, using time or frequency domain signal processing techniques. The sounds at the chest wall are, however, contaminated by incessant heart sounds, which interfere in the diagnosis based on, and analysis of, lung sounds. The problem with the conventional and commonly used method of linear fixed high pass filtering, to eliminate the heart sounds, is the spectral overlap of the heart and breath sounds in approximately the 50-150 Hz region. The stopband edge for the high-pass filter becomes a matter of unscientific compromise between retaining the low frequency spectrum of breath sounds and eliminating the heart sounds, and the breath sound information below the chosen edge is lost in the filtering process. Time-variant digital adaptive filtering, using a reference signal (ECG), where the coefficients of the filter are updated and based on the Least Mean Square error criterion as the signal is being filtered, is found by us to reduce the heart sounds considerably (70-90%) with minimal effects on the breath sounds. The theoretical basis and the implementation details of such a scheme are presented in the paper. A finite impulse response (FIR) filter, with 300 taps, is used as the adaptive filter and a modified ECG signal ("augmented ECG") is used as the reference signal. The superiority of the new method over traditional high-pass filtering is noted in the results on five studied subjects.
SESSION B

Masashi Mori and Marc Desmeules, Chairmen

1:30 - 1:50  Foam: An acoustic model of the lung   D. Rice

1:50 - 2:10  Production mechanism of crackles in experimental lung edema   K. Tanimura

2:10 - 2:30  Does the type and number of crackles relate to histologic evidence of airway opening?   F. Davidson

2:30 - 2:50  The application of digital filters for the analysis of crackles; detection of crackles and elimination of heart sounds   M. Ono

2:50 - 3:15  Japan Revisited - SPECIAL LECTURE   D. Cugell

3:15 - 3:35  COFFEE BREAK

3:35 - 3:55  New techniques of detecting adventitious sounds in pulmonary edema dogs   Y. Ploysongsang

3:55 - 4:15  Do fine crackles occur exactly in the "late" period?   H. Kusaka

4:15 - 4:35  Spectral characteristics of fine and coarse crackles   H. Ogasawara

6:30  COCKTAILS AND BUFFET
An aqueous solution of gelatin will form a foam with specific gravity ranging between 0.2 and 0.4 and pore size ranging between 0.1 and 0.3 mm. This foam approximates macroscopic properties of lung parenchyma except that the gas is trapped in closed pores. The gas trapping provides mechanical stability. Sound propagates through this foam at speeds independent of frequency and equal to speeds measured in parenchyma (25m/s) (Rice, JAP 54:304, 1983). Airways can be formed in the foam and may be lined with unfoamed gelatin. Auscultation with turbulent flow in an airway finds sound similar to bronchial breath sounds immediately above the airway, and sound similar to vesicular breath sounds further away. Spectral analysis of these sounds indicates that the foam strongly attenuates frequencies of 1 KHz and above. The airways exhibit dispersive sound propagation. Speeds ranging from a minimum of 40 m/s at 50 Hz to free field speeds at >10 KHz were measured in a 6 mm airway. With steady flow, obstructing the airway by finger pressure on the overlaying foam yields stable wheezes. Crackles are simulated by decreasing airway pressure with a sudden release. Positive initial deflections are recorded in radial directions and negative ones from axial directions, in agreement with theory (Fredberg & Holford, J Acoust Soc Am 1983; 73:1036). This model may be useful for testing lung sound theories because the structure is simple and the effects of individual variables can be studied in isolation.

Supported by HL30359.
PRODUCTION MECHANISM OF CRACKLES IN EXPERIMENTAL LUNG EDEMA

K. Tanimura
Y. Homma
H. Ogasawara
H. Kusaka
H. Ukita
N. Denzumi
Y. Kawakami

Last year, we preliminarily reported that crackles heard in the experimental edema of canine lung lobes might be generated by sudden opening of obstructed small airways. In this study, we attempted to define the kind of crackles which occurred in the experimentally induced lung edema by using Fast Fourier Transformation (FFT) analysis and histologically demonstrate lung edema in various conditions by rapid freeze method. Lung edema was produced by perfusion of whole blood. We used isolated lung lobe (mainly left lower lobes) from 9 mongrel dogs. As a control, we also used 4 canine lung lobes without perfusion. The excised lobe was ventilated manually in an airtight hard lucite box. Crackles were picked up by an electret condenser microphone from the inside of the capsule attached to the pleural surface of the lobe. Capsule position was restricted within 3 to 5 cm under the middle of the lobes. We simultaneously recorded total airflow at the airway opening (V), transpulmonary pressure (differential pressure between airway opening and box pressure, Ptp), and lung sounds. And we studied the pathological findings of three conditions: 1) control; without perfusion, 2) early stage of edema; venous pressure 3 to 5 cmH2O at 5 to 10 minutes after the perfusion, 3) late stage of edema; venous pressure 13 to 15 cmH2O at 40 minutes after the perfusion. After FFT analysis, the peak frequency of crackles in control, early stage of edema, and late stage of edema were respectively: 677±150, 595±150, and 545±99 Hz (mean±SD). Thus, the frequency of each crackle changed to lower as the degree of edema increased. That is, coarse crackles may be added to fine crackles with time. Microscopically, no edema of the lung was revealed in control, mainly interstitial edema in early stage, and fluid-filled alveoli in late stage, respectively. In this study, the relationship between lung sound generation and Ptp was confirmed to be same as the results we reported previously. From these results, the production mechanism of crackles in lung edema will be discussed.
Crackles are thought to be due to the opening of previously closed small airways. If each closed airway produces one crackle when it opens, the number of crackles heard over a particular area of lung should be the same as the number of airways present provided: 1) all airways were initially collapsed and 2) all airways were subsequently open after expansion to produce crackles. We counted the number of crackles produced when pig lung segments were inflated from collapse to full inflation. We also counted airways histologically from tissue sampled from beneath the recording microphone. The results as well as the problems in accurately assessing crackle counts will be discussed.
In the analysis of lung sounds, it is important to detect crackles and avoid noises such as heart sounds. So far the commonly used methods are visual approach and the use of high-pass filters. To provide more accurate and objective method, we designed two types of digital filters, one for the separation of crackles and the other for the elimination of heart sounds. The crackle filter, which is to detect and separate crackles from normal breath sounds, is composed of an autoregressive prediction filter and a non-linear function. We applied this filter to lung sounds containing crackles and succeeded in separating crackles (non-stationary signals) and normal breath sounds (quasi-stationary signals). The discrimination of heart sounds from crackles, however, was not complete, so we used a heart-sound filter we designed by modifying the adaptive noise canceller proposed by Widrow et al. We believe these filters are useful in the computer-aided analysis of lung sounds.
NEW TECHNIQUES OF DETECTING ADVENTITIOUS SOUNDS IN PULMONARY EDEMA DOGS

Y. Ploysongsang
V.K. Iyer
P.A. Ramamoorthy

Pulmonary edema was induced in five dogs by an intravenous infusion of Ringer's lactate solution. Lung sounds recorded during the increasing pulmonary edema were studied with the objective of detecting pulmonary edema at a stage early enough to avoid potential fatality and to implement effective treatment. Specialized spectral estimation techniques were applied to analyze the recorded sounds looking for early abnormal (adventitious) lung sounds (wheeze and crackles). The Maximum Entropy (ME) spectral estimation techniques was used to identify and study wheezes in early pulmonary edema, since the method is known to be sensitive to harmonic signals like wheezes. Short term spectral analyzes, using Fourier and ME methods, were used to study the spectral variations of lung sounds over the breath cycle, and also to identify the phases of the cycle where the adventitious sounds occurred which may have some physiological and clinical relevances. Wigner Time-Frequency (WTF) analysis was used to study and detect early crackles, which are known to be short non-stationary bursts of energy. The WTF analysis is known to be sensitive to short non-stationaries. Results of the investigation showing the superiority of the ME technique over the conventional FFT in frequency resolution, and that of the WTF technique over the short-term time frequency analysis in time resolution will be presented for 5 dogs.
DO FINE CRACKLES OCCUR EXACTLY IN "LATE" INSPIRATORY PERIOD?

H. Kusaka
Y. Homma
H. Ogasawara
K. Tanimura
H. Ukita
N. Denzumi
Y. Kawakami

Fine crackles have widely been believed to occur characteristically in late inspiratory period. But in patients with interstitial lung disorders, we occasionally note fine crackles not in late inspiratory period. In the present study, we analyzed the inspiratory crackles from 10 patients with idiopathic pulmonary fibrosis (IPF) and 12 patients with collagen vascular disease (CVD), in terms of the timing in inspiratory period when fine crackles occurred. The lung sounds and air flow in 5 breaths of each subject were recorded simultaneously by our phonopneumogram reported previously. The percent levels within the whole inspiratory period where the initial crackle appeared were determined (initial crackle level). The percent levels where the median out of all crackles was positioned were also determined (median crackle level). In the study of initial crackle (IC) level, 50 and 60 samples were picked up in IPF and CVD patients, respectively. And in the study of median crackle (MC) level, 738 and 628 inspiratory crackles were sampled in IPF and CVD patients, respectively. Respiratory function test (%VC, FEV1.0/FVC, %DLCO, %DLCO/VA) were also done.

The obtained results were:

1) The IC level appears in 45% and 43% of the inspiratory period, in the both IPF and CVD patients, respectively.

2) The MC level is 65% and 62% of the inspiratory period in both IPF and CVD patients, respectively.

3) The more %DLCO decreased, the earlier the initial crackle appeared in inspiratory period.

From these results, we conclude that in both IPF and CVD patients, fine crackles occur not in late inspiratory but in mid inspiratory period.
SPECTRAL CHARACTERISTICS OF FINE AND COARSE CRACKLES

H. Ogasawara
Y. Homma
K. Tanimura
H. Kusaka
N. Denzumi
H. Ukita
Y. Kawakami

Fine and coarse crackles are divided mainly by their pitch, but spectral analysis of these crackles and their variation in the clinical condition were not fully determined. We studied the frequency characteristics of these crackles and their relationship to the respiratory functions of the diseases hearing those crackles. Fine crackles of 17 patients with interstitial lung diseases (idiopathic pulmonary fibrosis (IPF), 8 patients, collagen vascular disease (CVD) 9 patients, and coarse crackles of 10 patients with type B chronic obstructive pulmonary disease (COPD) were examined. Ten crackles of each patient were first filtered by cutting at 60 Hz, and 512 data points of a crackle were digitalized at 50 kHz using a 12-bit analog-to-digital converter. Peak and maximal frequencies, that is frequency at 1/100 of peak power, were determined by fast Fourier transform (FFT) and were investigated as to their relationship to the respiratory functions.

Results were as follows:

1) Peak frequencies of fine and coarse crackles were 481±95 (mean±SD) and 273±45 Hz respectively, and maximal frequencies were 1193±339 and 557±100 Hz, respectively.

2) There was no difference in spectral analysis of fine crackles between IPF and CVD patients.

3) Peak frequency of fine crackles was lower in patients with decreased diffusing capacity and that of coarse crackles was higher in patients with severe airflow limitation.

These results showed that clear-cut separation of fine and coarse crackles might be possible by spectral analysis, and that frequency variation of these crackles might demonstrate the severity of the disease.
SESSION C

Shoji Kudoh and Steven Kraman, Chairman

9:00 - 9:20 Characterization of discontinuous adventitious lung sounds in cystic fibrosis
J. Kanga

9:20 - 9:40 Value of adventitious lung sounds analysis in Farmer's lung disease (FLD)
M. Desmeules

9:40 - 10:00 Crackles in sarcoidosis and in fibrosing alveolitis
R. Loudon

10:00 - 10:20 Objective correlations of medium crackles
M. Desmeules

10:20 - 10:40 COFFEE BREAK

10:40 - 11:00 Crackle measurements in COPD, interstitial fibrosis and congestive heart failure
R. Murphy

11:00 - 11:20 Observer variability in interpretation of voluntary cough of asthmatics and bronchitics
S. Ishikawa

11:20 - 12:00 DEBATE: The clinical value of lung sounds in 1986 is minimal
PRO: F. Davidson
CON: W. Ball
Y. Ploysongsang

LUNCH

1:00 - 1:15 BUSINESS MEETING
CHARACTERIZATION OF DISCONTINUOUS ADVENTITIOUS LUNG SOUNDS IN CYSTIC FIBROSIS

J. Kanga
S. Kraman

Discontinuous adventitious lung sounds are frequently heard in patients with cystic fibrosis. These sounds probably represent accumulation of secretions in bronchiectatic airways. Since auscultatory findings play an important role in the evaluation and follow-up of these patients, more objective criteria in reporting lung sounds are desirable. We recorded lung sounds from 7 patients with cystic fibrosis on whom clinical auscultation revealed discontinuous adventitious lung sounds. The patients ranged in age from 4 to 22 years (mean 12 years), and ranged in height from 92 to 160 cms (mean 128 cms). The recordings were made on the first day of hospitalization for pulmonary exacerbation. A total of 154 discontinuous adventitious lung sounds were analyzed among this group by the method described by Holford (1,2). By this method each crackle is displayed on an oscilloscope and the width of the initial deflection (IDW) and the duration of the first two cycles (2CD) are measured. Our results revealed an IDW of 0.82±0.26 msecs.; and a 2CD of 5.06±1.44 msecs. (Mean+S.D.). By the Classification system of Holford these adventitious lung sounds are medium crackles, falling between the dimension of coarse and fine crackles as recorded by us in other patients. Discharge recordings were available on 4 patients. All patients showed clinical improvement at the time of the second recording. Comparison of the IDW and 2CD with those obtained during the first recording showed a significant change in the IDW in all patients, and the 2CD in 1 patient only. The IDW increased in 3 subjects and decreased in one. This preliminary work classifies the discontinuous adventitious lung sounds in patients with cystic fibrosis as medium crackles. Significant change in the IDW is seen with clinical improvement, although the direction of the change is inconsistent. Lung sound analysis may offer another tool for the assessment and follow-up of lung disease in patients with cystic fibrosis.


VALUE OF ADVENTITIOUS LUNG SOUNDS ANALYSIS IN FARMER'S LUNG DISEASE (FLD)

M. Desmeules
Y. Cormier
M. Laviolette
J. Belanger
R. Murphy

Lung sounds were recorded and analyzed in 27 patients with FLD. Group A consisted of 6 acute cases of FLD while group B included 21 ex-FLD who still had radiological, biological or functional signs of the disease. Inspiratory crackles were observed in all group A and 12/21 group B patients. The number of crackles increased significantly during a deep inspiration from 10±18 (Mean±S.D.) to 27±151, but not in group B: from 10±14 to 14±13 (P<0.05). Crackles measurements gave a mean initial deflection width of 0.96±0.16 ms in group A and 0.99±0.13 ms in group B; values for 2 cycle duration were 4.94±0.64 ms and 5.04±0.64 ms respectively. Wheezing was observed in only 2 subjects during quiet breathing. Inspiratory squawks were observed in 7 patients. They had a mean duration of 90±86 ms. A "triggering" crackle with an average frequency of 374±91 Hz immediately preceded 40% of the squawks. Patients with squawks had a smaller vital capacity than other patients (p=0.01). Adventitious lung sounds analysis provides information that may be useful for diagnosis and follow-up of FLD.

(Supported by the Quebec Lung Association and the Chaire de Pneumologie, Laval University).
Crackles on chest auscultation are described as characteristic of several interstitial lung diseases, e.g. fibrosing alveolitis and asbestosis, but are described as rare, or are not mentioned, in sarcoidosis. All patients with sarcoidosis or fibrosing alveolitis seen by the pulmonary service at the University of Cincinnati over a four-week period were examined by the authors together. Standard auscultatory sites (R&L base, R&L apex, and mouth) and methods for recording the profusion, character, and timing of crackles were agreed on; findings were recorded independently without discussion. All of the eleven patients with fibrosing alveolitis, but only one of the seventeen patients with sarcoidosis, were noted by both observers to have crackles at two or more of the five auscultatory sites. Agreement between the observers on profusion of crackles (on a four-point scale of 0, 1, 2, 3) occurred in 112 of 140 comparisons; 22 comparisons showed a difference of one point, 4 of 2 points, and 2 of 3 points. Comparisons of character (fine or coarse) when both observers noted crackles at the same site showed 45 concordant and 29 discordant pairs. Major discrepancies in timing were rare. Our results show a more clearcut difference between sarcoidosis and fibrosing alveolitis than one previously reported study (Epler, Carrington, and Gaensler, Chest 1978; 73:333-339), in part because of the populations studied and in part because of the criteria used for presence of crackles. The disparity in physical findings between sarcoidosis and fibrosing alveolitis is marked, and of potential diagnostic importance.
OBJECTIVE CORRELATIONS OF MEDIUM CRACKLES

M. Desmeules  
E. Del Bono  
R.L.H. Murphy

The term medium crackles was omitted from the ATS/ACCP classification because (1) it was not clear that auscultators could reliably agree on the observation of medium crackles and 2) no clear correlations of this term with objective crackles measurements were proven. Three experienced observers independently listened to lung sound recordings of 33 patients with crackles. Quality of crackles was graded on a scale of 1 to 9 with the convention that grades 1-2-3 corresponded to fine, grades 4-5-6 to medium and grades 7-8-9 to coarse crackles. The score reached by each individual observer in the 3 categories of crackles agreed 7 out of 9 times. Time-expanded wave form analysis was performed on each of the 340 crackles. Mean results for initial deflection width (IDW), 2-cycle duration (2-CD) as well as the mean crackle score (mean CR score) appear on table I.

<table>
<thead>
<tr>
<th>No. Pts.</th>
<th>Mean CR Score</th>
<th>IDW</th>
<th>2-CD</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>2.60 (Fine)</td>
<td>0.65</td>
<td>4.75</td>
</tr>
<tr>
<td></td>
<td>± 0.70</td>
<td>± 0.10</td>
<td>± 0.76</td>
</tr>
<tr>
<td>13</td>
<td>4.62 (Medium)</td>
<td>0.80</td>
<td>5.32</td>
</tr>
<tr>
<td></td>
<td>± 0.77</td>
<td>± 0.27</td>
<td>±1.04</td>
</tr>
<tr>
<td>10</td>
<td>7.20 (Coarse)</td>
<td>1.06</td>
<td>5.97</td>
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<tr>
<td></td>
<td>± 0.42</td>
<td>± 0.28</td>
<td>±0.47</td>
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</tbody>
</table>

p < 0.01

Mean CR scores correlated positively with IDW and 2-CD. But individual scores failed to separate medium from coarse crackles in 2 instances on both IDW and 2-CD.

From these findings we conclude that:

1) The existence of a good agreement between crackles quality at auscultation and objective measurements such as IDW and 2-CD is confirmed.
2) Pooling the observations of 3 auscultators gives a better correlation for fine, medium and coarse crackles with IDW and 2-CD.
3) It is reasonable to keep the term medium crackles in the nomenclature.
We have previously reported that the crackles observed in seven patients with interstitial fibrosis had shorter initial deflection widths and shorter two cycle durations than were observed in the crackles of patients with chronic obstructive lung disease (COPD). We have increased the numbers of such patients to 20 in each category and have found essentially similar results. Crackles waveforms in congestive heart failure, however, showed no significant differences in IDW or 2CD than patients with COPD as illustrated below:

<table>
<thead>
<tr>
<th></th>
<th>COPD</th>
<th>IF</th>
<th>CHF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>IDW</td>
<td>1.11</td>
<td>.23</td>
<td>0.69</td>
</tr>
<tr>
<td>2CD</td>
<td>6.71</td>
<td>1.32</td>
<td>4.85</td>
</tr>
<tr>
<td>ZC</td>
<td>5.23</td>
<td>1.42</td>
<td>4.26</td>
</tr>
</tbody>
</table>

The timing of crackles of CHF, however, differed from those of COPD in that 93% of the CHF crackles as compared to only 15% of the COPD crackles were pan inspiratory or mid to late inspiratory. Furthermore, the crackles measured in patients with CHF tended to be transient and to change the value of their IDW and 2CD whereas in COPD these measurements remained constant. Objectively verifiable differences exist in the crackles of these three conditions.
Lung sounds were recorded over the central airway with an electronic stethoscope while subjects, in sitting position, voluntarily coughed. These recordings were then transcribed, using computer, onto paper at the speed of 21.5 mm/sec. There was good concordance between the auditory and visual analysis. Thirty bronchitics, 30 asthmatics and 30 normal subjects were studied. These 90 subjects recorded sounds and tracings were mixed and coded 1 to 90. Five physicians were asked to listen to the taped sounds and subsequently shown the tracings of each case. They were asked to score A for Asthmatics, B for Bronchitics, or N for Normals. Prior to this testing, typical examples of voluntary cough sounds and tracings, according to the average period of each cough and wave form characteristics of normals, bronchitics, and asthmatics, were demonstrated to them. Observer variability and agreement were determined. Observer agreement was fairly good. Observer variability and its influence on the pulmonary function will be discussed.
SESSION D

Robert G. Loudon and Gerard Charbonneau, Chairmen

1:15 - 1:35  Comparison of subjective description and automated characterization  H. Pasterkamp

1:35 - 1:55  Airflow phenomena in relation to breath sound generation  J. Seiner

1:55 - 2:15  Lung sound analysis by fast Walsh transform  D. Daien

2:15 - 2:35  Effect of helium on breath sounds in asthma  M. Desmeules

2:35 - 2:55  Coffee Break

2:55 - 3:15  Characteristics of power spectrum averaged, flow standardized breath sounds in normal and asthmatic subjects  M. Desmeules

3:15 - 3:35  Acoustic analysis of short wheezes  Y. Koyama

3:35 - 4:05  CRACKLEFEST

4:05 - 4:35  SUMMARY
We presented 10 examples of tape recorded breath sounds from asthmatic patients to 40 health professionals, 10 in each group of residents, nurses, staff physicians and physiotherapists. Each recording was approximately 20 sec in duration. The examples were selected to include a variety of normal and adventitious tracheal lung sounds. Automated spectral characterization provided data on wheezing pitch, intensity and duration. All participants performed the test twice, at least two weeks apart, listening to the sound examples through earphones from an electronic stethoscope. Their subjective descriptions showed significant differences in terminology between groups and individuals, but much less variation within subjects. This likely reflects individual teaching experiences in lung sound terminology. When asked to characterize the sound examples according to the recommended ATS nomenclature and to wheezing characterization as used by Forgacs, the participants showed greater intraobserver variability than in their spontaneous descriptions. A standardized nomenclature is important in the assessment of pulmonary disease by acoustical signs. Spectral characterization of breath sound teaching tapes may prove valuable to achieve this goal.
We have utilized a large scale 3-order airway model (I.D.s 3.18/2.54/1.91 cm, branch angle 70°) together with nebulized kerosene, and a 4-watt argon-ion laser to improve the resolution of airflow patterns. Both steady and sine wave airflow were used. Inspiratory airflow patterns were recorded on 3/4 inch videotape. In the monoplanar configuration of the model, airflow follows roughly a "C" or "S" course in the airways. The associated vortices at the orifices of the 3rd order showed major differences. The centers of the 2 vortices at the S-orifice were displaced away from the central diameter of the airway. At the C-orifice the vortices were nearer the diameter line but a small secondary vortex was present in the periphery. The major vortex axis at the C-orifice differed by 116° from that on the S-side, probably by virtue of effects on the "swirl" of the vortex system. Breath sounds generated by vortex orbiting will be altered by these "C-S phenomena". We believe that these are explicable by momentum/viscosity interaction and by large-scale vortex phenomena in a curved pipe described by Dean and boundary layer small scale vortices described by Taylor-Gortler. At the instant of airflow reversal by the pump from inspiration to expiration the vortex pattern at the S-orifice performed a sudden reversal to a smaller mirror image. Asymmetry of carina axes - the normal situation in a three dimensional lung appreciably alters the tones at a given Reynolds number.
LUNG SOUND ANALYSIS BY FAST WALSH TRANSFORM

D. Daian
H. Pasterkamp
R. Fenton

The application of computer-aided techniques in the analysis of respiratory sounds has traditionally employed power spectral analysis by fast Fourier transformation (FFT). Although the usefulness of this approach is undebatable, its applicability under certain clinical situations, i.e. automated quantification of wheezing at the bedside, is hampered by the excessive computation time required by FFT, which consists of complex multiplications and additions. In contrast, fast Walsh transform (FWT), which has been successfully applied in the processing of other biological signals such as electroencephalograms, uses only real additions and subtractions for spectral computation. We have compared the computation time for breath sound spectra, and the accuracy of automated wheeze detection by spectral characteristics, both by FFT and FWT transformation. Normal and continuous adventitious sounds, recorded over lung and/or trachea, were analyzed. FWT reduced spectral computation time by a factor of 2.54. This is an advantage over FFT, which in our laboratory currently takes about 8 times real time for spectral computation. However, automated wheeze detection by our criteria (FENTON et al, IEEE Trans Biomed Eng 1985) was not as reliable with FWT as compared to FFT spectra. This may limit the usefulness of FWT for lung sound analysis, unless wheeze detection schemes can be developed that are based on properties unique to the Walsh domain.
CHARACTERISTICS OF POWER SPECTRUM AVERAGED, FLOW STANDARDIZED BREATHE SOUNDS IN NORMAL AND ASTHMATIC SUBJECTS

M. Desmeules E. Verreault C. Gagnon
A. Tremblay Y. Trottier B. Tousignant

Quantitative analysis of breath sounds in asthma is limited by the inter-and intra-breath variability of wheezing; this presumably results from variation in the actual pressure, flow and/or volume prevailing in each respiratory cycle. Power spectrum averaging (PSA) provides a more accurate estimate of the true spectrum of mixed random signals. PSA was used to analyze breath sounds in 7 normal (N) and 6 asthmatic (A) subjects. We sampled an average of 10 sound segments [3-16] starting at a target flow of 0.5 l/s during tidal inspiration (I), expiration (EX) and forced expiration (FE). Mean results are summarized in Table I.

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>A</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(m ± SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Int. Ampl.</td>
<td>2.10 ± 1.06</td>
<td>6.44 ± 5.96</td>
<td>NS</td>
</tr>
<tr>
<td>Mean F</td>
<td>272 ± 29</td>
<td>318 ± 36</td>
<td>*</td>
</tr>
<tr>
<td>S (dB/oct)</td>
<td>-11.8 ± 2.48</td>
<td>-8.33 ± 3.97</td>
<td>NS</td>
</tr>
<tr>
<td>r</td>
<td>0.97 ± 0.02</td>
<td>0.80 ± 0.24</td>
<td>NS</td>
</tr>
<tr>
<td>EX</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Int. Ampl.</td>
<td>0.92 ± 0.03</td>
<td>6.15 ± 5.26</td>
<td>*</td>
</tr>
<tr>
<td>Mean F</td>
<td>295 ± 34</td>
<td>279 ± 41</td>
<td>NS</td>
</tr>
<tr>
<td>S (dB/oct)</td>
<td>-9.5 ± 1.39</td>
<td>-13.2 ± 2.49</td>
<td>**</td>
</tr>
<tr>
<td>r</td>
<td>0.99 ± 0.01</td>
<td>0.92 ± 0.07</td>
<td>*</td>
</tr>
<tr>
<td>FE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Int. Ampl.</td>
<td>40.2 ± 12</td>
<td>26.2 ± 16</td>
<td>NS</td>
</tr>
<tr>
<td>Mean F</td>
<td>366 ± 35</td>
<td>328 ± 46</td>
<td>NS</td>
</tr>
<tr>
<td>S (dB/oct)</td>
<td>-5.9 ± 2.27</td>
<td>-5.8 ± 3.85</td>
<td>NS</td>
</tr>
<tr>
<td>r</td>
<td>0.88 ± 0.16</td>
<td>0.65 ± 0.22</td>
<td>*</td>
</tr>
</tbody>
</table>

* p < 0.05  ** p < 0.01

The averaged power spectra were highly reproducible in N but more variable in A. Changes related to wheezing predominated in the 300-500 Hz frequency range. During tidal I and EX the integrated sound amplitude (Int.Ampl) was higher in A than in N for comparable flow rates. During FE the difference between the 2 groups was cancelled presumably because N subjects generated much higher expiratory flow rates. Asthmatics had a higher mean weighted frequency (mean F) than N during inspiration but not during either quiet or forced expiration. Sound amplitude varied as a function of log-frequency, with a negative slope (S dB/oct) and a high correlation coefficient (r) in N. In many asthmatics however the relationship was not linear, as seen from lower values of r. In both groups the r was smaller and the slope was flatter during forced expiration. No clear relationship was seen between the severity of airways obstruction and any of the sound characteristics analyzed. However, sound amplitude was weakly correlated with the ratio Flow/FEV1.0% predicted. This supports the idea that linear air velocity in the airways is one of the factors involved in the production of wheezing. (Supported by La Chaire de Pneumologie, Universite Laval, Sainte-Foy (CANADA),
EFFECT OF HELIUM ON BREATH SOUNDS IN ASTHMA

M. Desmeules
E. Verreault
C. Gagnon
B. Tousignant

Forgacs states that breathing helium does not modify the pitch of wheezing in asthma. No experimental data have been provided to support this observation. We studied the effects of breathing 80% helium - 20% oxygen (heliox) on breath sounds in 6 asthmatic subjects. They had an FEV1.0% predicted (mean ± SD) of 25.5±12.5%. Wheezing was present at auscultation during inspiration (in 4) and during expiration in the 6 subjects. Seven normal subjects were used as control. Spectral analysis was performed on power averaged, flow standardized sound segments sampled during tidal inspiration, expiration and forced expiration. Compared to air, heliox caused a significant reduction of the mean weighted frequency in normal during inspiration: 318±36 to 301±30 Hz, and during forced expiration: 328±46 to 293±42 Hz. In asthmatics the changes were in the same direction but not significant. During quiet expiration frequency did not vary consistently in either group. Heliox caused a 35% reduction of the integrated sound amplitude in normals: 40.2±12 to 26.0±20 units (p<0.05) and less consistently during inspiration: 2.10±1.06 to 1.15±0.49. In asthmatics, the sound amplitude was reduced by 16% during inspiration and 19% during forced expiration. Breathing heliox caused a slight increase of flow rates in asthma but not in the control group.

In summary:

1. Heliox reduced the intensity of breath sounds during inspiration and expiration, in both normals and asthmatics.

2. A slight increase in flow rates may offset this effect in asthma.

3. Heliox caused either no change or a slight decrease in the mean weighted sound frequency.

The relation of these findings to the recent models proposed to explain the production of wheezing will be discussed.

Supported in part by La Chaire de Pneumonologie, Universite Laval, Sainte-Foy, Canada.

-32-
ACOUSTIC ANALYSIS OF SHORT WHEEZES

Y. Koyama
N. Shioya
N. Narita
A. Shibuya
S. Kudoh
Y. Maeda
R. Mikami

For many years, an inspiratory short continuous sound, called short wheeze, squawk or squeak, has been described, but its acoustic characteristics have not yet been studied well. The purpose of this study is to find out the acoustic characteristics of short wheezes. Inspiratory short wheezes were recorded in 9 patients with interstitial lung diseases (6 with asbestosis, 2 with collagen disease, 1 with idiopathic interstitial pneumonia), and 8 with other lung diseases (3 with bronchiectasis, 3 with pulmonary tuberculosis, 2 with diffuse panbronchiolitis). Short wheezes were picked up on the chest wall, and air flow at the mouth was recorded simultaneously. We analyzed the duration, pitch, position in inspiration, number of overtones and wave form of short wheezes, using a sound-spectrograph and a high-speed level meter.

Results were as follows:

1) Short wheezes were associated with crackles in all cases.
2) The number of short wheezes in one inspiratory phase of a case was 1.35±0.68 (means±S.D.).
3) The position in inspiration was 62.0±21.0%, and the pitch was 545±270 Hz. In interstitial lung diseases, short wheezes which occur later tended to be higher in pitch.
4) The duration of short wheezes was 86.5±53.3 ms.
5) The number of overtones of short wheezes was 3.56±2.54.
6) The length from the initial deflection to the maximum amplitude was less than 20 ms.

We conclude that short wheezes were found not only in interstitial lung diseases but also in other lung diseases, and that they were acoustically different from "wheezes".

-33
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