Fourth International Conference

on Lung Sounds

第4回 国際肺音学会

September 20, 21, 1979

Northwestern Memorial Hospital

Chicago, Illinois

Conference Steering Committee:

Leslie H. Capel	- London
David Cugell	- Chicago
Shoji Kudoh	- Tokyo
Robert G. Loudon	- Cincinnati
Raymond L.H. Murphy, Jr.	- Boston

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FOURTH INTERNATIONAL CONFERENCE ON LUNG SOUNDS

NORTHWESTERN MEMORIAL HOSPITAL

CHICAGO, ILLINOIS

PROGRAM

Thursday, September 20, 1979

Registration		8:30 am
Welcome ----------David Cugell		9:00 am
Introduction ----------Dan E. Olson		9:15 am
Session A	9:30 am - 12:00 Noon	
Lunch	12:30 pm - 1:30 pm	
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Friday, September 21, 1979

Session C	9:00 am - 12:00 Noon	
Business Meeting	12:00 Noon	
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SESSION A

CHAIRMAN: FORBES DEWEY

Session A

Chairman: Forbes Dewey

9:30 am	The origin of crackles in the lung: an alternate hypothesis	J. Fredberg
10:00 am	Flutter of a collapsible channel: wheezing and flow limitation	J. Grotberg S. Davis
10:30 am	Acoustic modeling of lung sound transmission using bond graphs	P. Krumpe D. Margolis
11:00 am	Response of normal lungs to excitation with wide-band acoustic noise	L. Mockros J. Lewis J. Jacobs D. Cugell
11:30 am	Cough - a lung sound?	R. Loudon

THE ORIGIN OF CRACKLES IN THE LUNG: AN ALTERNATE HYPOTHESIS

J. Fredberg

Forgacs has hypothesized that crackles arise from "explosive" equalization of gas pressure between two compartments of the lung when a closed section of airway separating them opens. This leads to the question, under what conditions may collapsed airways open? To elucidate the mechanics of airway closure and opening I have investigated the influence of surface and elastic forces in the equilibrium configurations of compliant tubes obstructed by liquids. A variety of discrete equilibrium configurations can be identified, and transitions between equilibrium states may occur. In the case of linearly elastic airway walls with stiffness k and resting diameter D, and liquids of surface tension σ, the equilibrium states depend upon the dimensionless parameter $C=8\sigma/kD$. Fully collapsed states occur when C is greater than unity, which is most likely at low lung volumes, especially for small airways in regions of reduced elastic recoil at the base of the lung. From its maximum at end inspiration C falls most rapidly during early inspiration, suggesting that most transitions from collapsed to open states would occur during that breathing phase. Furthermore, this leads to the alternate hypothesis that crackles arise from the rapid release of elastic strain in the collapsed airway wall and surrounding parenchyma as the airway passes between meta-stable equilibrium states. The change in static stress levels in the parenchyma associated with airway opening is roughly $2\sigma/D$, or 130 dB re 20μPa for 1mm airways. For such an airway 1-2 cm (10-20 diameters) from the pleura the change in stress level at the pleura can be predicted from St. Venant's principle (stress$\sim 1/r^2$) to be some 30-40 dB less, or 90-100dB. Dynamic equalizations of gas pressure differences may occur as well, but it is not necessary to postulate them.

This research was supported by the NHLBI, Grant No. 1-R01-HL23318-01

uniform tubes

$$D/D_0 = \frac{1 \pm \sqrt{1-C}}{2}$$

$$C = \frac{8\sigma}{kD_0}$$

Non uniform tubes

Normal values
$k \sim 10^5$ dyne/cm^2
σ

FLUTTER OF A COLLAPSIBLE CHANNEL: WHEEZING AND FLOW LIMITATION

J. B. Grotberg*
S. H. Davis**

Airway wheezing is mathematically modelled by an inviscid, incompressible fluid flowing through an infinite, two-dimensional, flexible channel that has either resistance to bending or elastance. The linearized equations are solved for traveling wave oscillations of the wall and fluid that depend on the wavelength, fluid velocity, geometry, and the fluid and wall properties.

Initially, stable waves travel both upstream and downstream. however, at a large enough fluid speed, called the stasis velocity, the upstream wave reverses direction. The stasis velocity corresponds to flow limitation and is consistent with previous one-dimensional, steady models of this phenomenon. Our dynamical model redefines flow limitation as the absence of upstream energy fluxes instead of the absence of upstream waves. The stasis velocity is a measure of volume flow rate. Changes in the model parameters that correspond to asthma, chronic bronchitis, emphysema, tumor impingement and hyperbaric atmospheres predict decreases in the limited flow.

The oscillating wall becomes unstable at a critical fluid speed which is greater than the stasis velocity. These unstable oscillations, called flutter, are implicated as the mechanism of wheezing because their amplitudes can be large (audible) and they can be self-sustained when the wall damping (energy dissipation) is included. Stable waves die out with damping. The stability of an airway (critical fluid speed) is decreased when the model parameters simulate the above lung pathologies.

The pitch of the wheeze (flutter frequency) depends on the ratio of wall mass to fluid mass among other parameters. The model predicts no discernible pitch change with He-O_2 mixtures versus air since the ratio is dominant ($\sim 10^3$) in either case. The model also provides a rationale for determining which airway generation is wheezing based on its pitch.

Since the critical velocity is greater than the stasis velocity, our model shows wheezing is always associated with flow limitation but flow limitation is not necessarily accompanied by wheezing. This idea can explain the absence of wheezing in patients who are flow limited. A wheeze often transiently increases in pitch toward the end of expiration as fluid speeds decrease. The model predicts that the oscillations would change from unstable to stable and that stable frequencies, although transient, increase as the fluid speed continues to decrease.

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Biol. J. Vol 9. 1969

ACOUSTIC MODELING OF LUNG SOUND TRANSMISSION USING BOND GRAPHS

P.E. Krumpe
D.L. Margolis

We have developed a computer model of regional lung sound transmission using Bond Graph Analysis (Gebbon, U.D., ASME Jol. Dyn. Syst. Meas. & Control, Vol 99, Series G, No. 2, June 1977). This approach utilizes a pictorial representation of acoustic energy storage, exchange and dissipation mechanisms to describe system dynamics. The trachea and segmental airways of both lungs were represented by 21 sets of differential equations, while the peripheral airways were represented by lumping their resistance and compliance effects. The frequency response of the system is 8500 Hz.

We used morphometric values for the segmental airways reported by Weibel (1963). This Bond Graph Model assumed symmetrical airway branching, equal regional volumes of each lung segment, uniform rates of inflation from residual volume, and predicted more or less uniform sound attenuation in each lung segment.

Next we attempted to validate the Bond Graph Model's predictive capability by measuring random noise transmission from trachea to lung. The integrated sound energy from 60 to 2000 Hz was displayed on a real time spectral obtained over the lung by data obtained over the trachea. An increased amount of attenuation of high frequency components (above 200 Hz) was apparent in the lung base compared to the lung apex in 3 normal upright subjects. These differences were lung volume dependent. We therefore concluded that regional differences in transmission properties are present in normal lungs.

As a result of these experimental data we have modified our Bond Graph Model to include non-uniform time regional constants, asymmetric branching angles of airways, and differences in segmental volume.

This Bond Graph Model will be used to predict sound transmission characteristics of patients with abnormal lung mechanics.

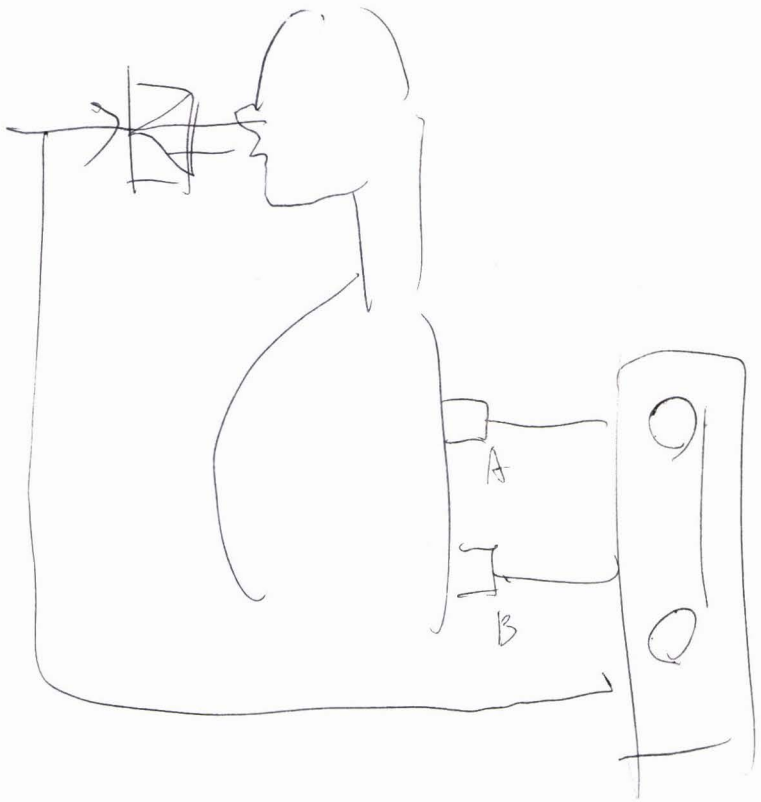
RESPONSE OF NORMAL LUNGS TO EXCITATION WITH WIDE-BAND ACOUSTIC NOISE

L. F. MOCKROS
J. D. LEWIS
J. E. JACOBS
D. W. CUGELL

Wide-band acoustic noise is driven into the mouth of subjects, modulated by the airway system, and received on the thoracic surface with one or more microphones. The received signal, or signals, is cross-correlated with the input noise and the resulting correlogram represents the impulse response of the intervening "black box", the lungs. The technique and the special instrumentation were described at previous Conferences on Lung Sounds.

The cross-correlation between the input noise and the signal received at the third intercostal space on a young healthy subject indicates the transmitted signal is dominated by energy at about 600 Hz. The signals received at the fifth, seventh and ninth intercostal spaces not only show a dominant signal at the 600 Hz frequency but also a dominant signal at about 2500 Hz. For general screening purposes, the signal received at the ninth intercostal space has proved convenient and useful. The 2500 Hz signal, or "A" wave, generally has a maximum at a delay time of about 0.4 ms and the 600 Hz signal, or "B" wave, generally has a maximum at a delay time of about 1.6 ms. A recent test of 39 young (ranging in age from 18 to 31 years), healthy male subjects indicates distinct differences between subjects who smoke and those who had never smoked. All correlograms with signals received at the ninth intercostal space indicate the dominance of a 600 Hz signal. The 2500 Hz signal appears in almost all records of non-smokers, but rarely appears in those of subjects who smoke.

A fairly simple mathematical model is being used to help interpret these correlograms. The geometry used in the model is the asymmetric airway system proposed by Fredberg and Hoenig (1978). The only addition to their geometry is a short tube representing the pharynx. The analysis consists of calculating the transfer function and is based on solving linearized, one-dimensional fluid dynamic equations for steady oscillatory flow in a complex dentritic structure. The calculation method is essentially that described as an impedance analysis in Streeter and Wylie (1967). It consists of prescribing terminal impedance conditions, adding the impedance of the first airway generation, matching boundary conditions at the proximal junction, proceeding up the system through the next generation and so on until the mouth is reached. The technique reduces the governing partial differential equations to an algebraic algorithm. The complicated dentritic airway system can be investigated for its input impedance, resonant frequencies and transfer function. A novel aspect of the present algorithm is the inclusion of mucus layer dissipation.


A
B

The longest-path geometry of Fredberg and Hoenig (1978) was used to model the path from the mouth to the ninth intercostal space. The calculations suggest dominant frequencies at about 900 Hz and 3000 Hz, reasonably similar to the observations with the noise correlator unit. Further tests with the model lung, cutting off the upper generations, suggest the lower frequency is generated in the trachea and large bronchi and the higher frequency is generated in the middle generations. Thickening the mucus layer in the model lung, with attendant increased dissipation, produces a loss of the high frequency wave. Subjects who smoke are likely to have thickened mucus layers and the lack of the high frequency wave in the correlograms may be due to this dissipation mechanism. Calculations using a shorter path model indicate the dominant low frequency wave, but the high frequency wave is absent. With the transducer placed near the apex of the lung at the third intercostal space, the correlogram also failed to indicate a dominant high frequency wave.

Fredberg, J.J. and Hoenig, A. Mechanical response of the lungs at high frequencies. Journal of Biomechanical Engineering, ASME, 100: 57-66, 1978.

Streeter, V.L. and Wylie, E.B. Hydraulic Transients. McGraw-Hill, New York, 101-113, 1967.

SESSION B

CHAIRMEN: R. MIKAMI AND S. ISHIKAWA

Session B

Chairmen: R. Mikami and S. Ishikawa

1:30 pm	Is "crackle" a surface wave? ---- Wave form of intrabronchial spark sounds and "crackle" on the chest wall	S. Kudoh S. Sasaki A. Shibuya N. Aisaka I. Ono S. Shirai R. Mikami
2:00 pm	Difference of crepitation and bubbling sounds in experimentally induced lung edema	Y. Homma Y. Minami M. Matsuzaki S. Fujiya M. Murao
2:30 pm	Analysis of crackles by zero-crossing method	A. Shibuya N. Aisaka S. Kudoh S. Shirai R. Mikami
3:00 pm	The origin of crackles	M. Mori K. Kinoshita H. Morinari T. Shiraishi A. Koike S. Murao
3:30 pm	Status of the ATS Lung Sound Nomenclature Report	D. Cugell
4:00 pm	Cracklefest (Râlefest) A recital of strange sounds	Various contributors

IS "CRACKLE" A SURFACE WAVE? ---- WAVE FORM OF INTRABRONCHIAL SPARK SOUNDS AND "CRACKLE" ON THE CHEST WALL

S. Kudoh
S. Sasaki
A. Shibuya
N. Aisaka
I. Ono
S. Shirai
R. Mikami

In this study we analyzed the wave forms of intrabronchial spark sounds on the chest wall, which were generated by electric spark discharge at a needle gap inserted into the canine bronchus. In free air space, the peak level of sound pressure was approximate 110 dB at 1 cm distance. Duration of spark sound by the naked electrode was 70 usec, but it was 0.7 msec when the electrode was covered with vinyl tube. The duration of wave form on the chest wall from the intrabronchial spark sound was 10 msec or more. This wave form consisted of three or four different phases. The first phase, which synchronized with the timing of discharge, was considered to be caused by mixing of electric wave on spark discharge. The second phase, which could not be recognized in some cases, was estimated to be the direct wave, i.e., probably P-wave, from the source. The third phase was the maximum wave not only in duration but in amplitude. Reverse dispersion was seen in this phase as well as in wave forms of crackles. We estimate that this third phase had the general characteristics of a surface wave originated from an impulsive event in the elastic body. The physical meaning of the fourth phase, in which damped oscillation was frequently seen, could not be revealed.

We could estimate that the wave form of crackle was not originated directly from an impulsive event in the lung, but had a characteristic of surface wave as the third phase.

DIFFERENCE OF CREPITATION AND BUBBLING SOUNDS IN EXPERIMENTALLY INDUCED LUNG EDEMA

Y. Homma
Y. Minami
M. Matsuzaki
S. Fujiya
M. Murao

We have clinically investigated the occurrence mechanism of the pulmonary discontinuous sounds or rales by means of sound spectrograph (SG) and Fourier transform technique, and reported that the rales might be classified into two main groups with different mechanisms. The one is crackles which contain velcro rale and crepitation with acoustically resonant character produced in the certain condition where the vibration of surrounded tissue wall is limited and the other is bubbling with acoustically explosive character produced by the explosion of the bubbles in the airways.

To ascertain this event, the sounds produced in canines with experimental lung edema were investigated. After the anesthetization by nembutal, positive end expiratory pressure (PEEP) breathing was maintained. Then, a large amount of saline was dripped and Alloxan (50 mg/kg) was additionally injected intravenously. During this operation, lung sounds were recorded on audio-tape from the surface of chest wall and then analyzed by sound spectrograph or Fourier transform analysis. It was noted that the sounds recorded in early stage of the experiment were resonant sounds or crepitation supposed to be produced by the lung congestion where the movement of the lung tissue wall was limited, and the sounds in late stage of the experiment after removal of PEEP were explosive sounds or bubbling supposed to be produced by explosion of the bubbles exudated into the airways.

These results may suggest that the rales should be classified into resonant sound or crackle and explosive sound or bubbling.

ANALYSIS OF CRACKLES BY ZERO-CROSSING METHOD

A. Shibuya
N. Aisaka
S. Kudoh
S. Shirai
R. Mikami

In an attempt to classify crackles by objective analytical method, the initial deflection width in time-expanded wave form or the frequency spectrum by FFT or sound spectrogram have been used.

We have recently reported that reverse dispersion was found in the wave forms of crackles. In this study, we investigated the dispersion curve on wave forms in each kind of crackle by the zero-crossing method, in which the relationship between the period on every successive zero-crossing point and the elapsed time from the initial deflection was evaluated. The dispersion curves could be distinguished between fine crackles, medium crackles and coarse crackles.

The zero-crossing method was considered to be useful to classify crackles especially by computer system.

THE ORIGIN OF CRACKLES

M. Mori
K. Kinoshita
H. Morinari
T. Shiraishi
A. Koike
S. Murao

By waveform analysis and model experiments we tried to determine the origin of crackles (rales). Crackles were recorded from six patients, four with tuberculosis and two with chronic bronchitis. By waveform analysis, in which the time scale was expanded 1,600 times, we observed in the waveforms of crackles initial "high-pitched segments" with characteristics of shock waves and subsequent "low-pitched segments" with characteristics of damped sinusoids. We speculated that the former was a shock wave caused by an abrupt opening of the airway and that the latter was an oscillation caused by this shock wave exciting a resonator in the lung. To test our hypothesis we experimentally excited a resonator by a shock wave and obtained waveforms similar to those of crackles. We suggest that the characteristics of the initial segments are primarily determined by the opening pressure of the airway in which crackles are formed and that the attenuations of the subsequent segments are primarily determined by the quality factor (Q) of the resonator in the lung. The equivalent quality factors of the lung, which we calculated from the waveform of crackles, were about 3 to 6.

SESSION C

CHAIRMAN: WILLIAM W. WARING

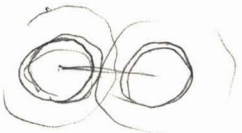
Session C

Chairman: William W. Waring

9:00 am	A technique for the determination of site of production of lung sounds	S. Kraman
9:30 am	Peak frequency of breath sounds recorded at homologous loci on the chest wall	J. Schreiber W. Anderson M. Wegmann W. Waring
10:00 am	Rhonchi and airflow dynamics in asthmatic patients	S. Ishikawa P. Workum S. Holford R. Murphy, Jr.
10:30 am	Relationship between breath sounds amplitude and bronchospasm	B. Tiep M. Belman M. Trippe
11:00 am	Central to peripheral sound transmission in excised lung	D. Rice
11:30 am	Sub-miniature audio transducers	M. Killion
12:00 Noon	Business meeting: Chairmen: R.G. Loudon R.L.H. Murphy, Jr.	

A TECHNIQUE FOR THE DETERMINATION OF SITE OF PRODUCTION OF LUNG SOUNDS

S.S. Kraman



A method of analyzing lung sounds recorded from the chest wall has been evaluated in a pilot study. The method involves the recording of sounds from two separate areas of the chest wall (2 cm horizontal separation) using identical microphones, amplifiers and filters. The phase of one of these signals is then inverted and then mixed with the other (non-inverted) signal and displayed on a storage oscilloscope. This is termed the cancellation signal. The amount of cancellation that occurs as a result of summing the two signals is determined by comparing it to the amplitude of both signals when mixed without phase inversion (non-cancellation signal). The amplitude of the cancellation signal is divided by that of the non-cancellation signal to give a number (lung sound cancellation index) that is equal to or less than 1.0. Sounds originating far from the chest wall and therefore a similar distance from each microphone, arrive in phase and result in greater cancellation (after phase inversion of one of the signals) than sounds originating close to the chest wall and therefore, nearer to one microphone than the other.

Twenty-three people were chosen at random and studied by this method as well as by spirometry. A negative correlation was found between the inspiratory lung sound cancellation index and the % predicted maximal mid-expiratory flow ($r = -0.74$), the forced expiratory flow at one second corrected for forced vital capacity ($r = -0.70$), and the % predicted of the forced expiratory flow at one second ($r = -0.58$). All significant at $p = <0.001$.

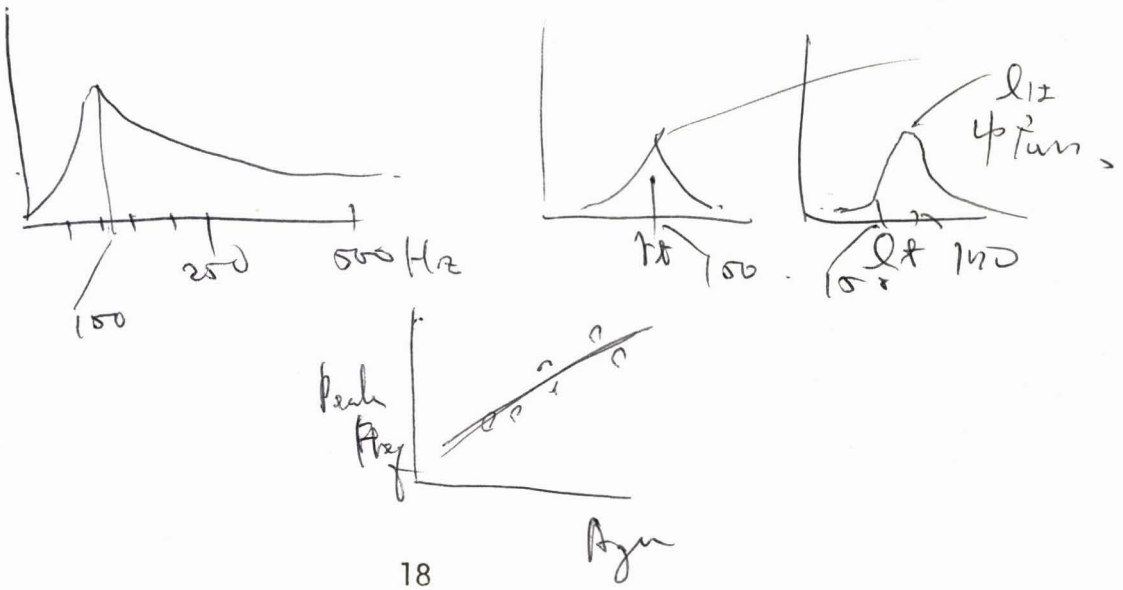
These preliminary results suggest that the site of generation of the inspiratory vesicular sounds moves toward the chest wall with progressive degrees of airway obstruction.

PEAK FREQUENCY OF BREATH SOUNDS RECORDED AT HOMOLOGOUS LOCI ON THE CHEST WALL

J.R. Schreiber
W.F. Anderson
M.J. Wegmann
W.W. Waring

Boyden

Sound generated in the tracheobronchial tree is transmitted through the pulmonary parenchyma to the chest surface. The frequency of these transmitted breath sounds was studied in normal subjects by using a technique of two channel phonopneumography (differential segmental phonopneumography). The posterior chest wall was divided according to Boyden's anatomic system into 12 topographic projections of broncho-pulmonary segments, six for each lung. Two microphones were then utilized simultaneously to record transmitted breath sounds from two different but homologous regions of the posterior chest wall. The frequency components (amplitude spectra) of the recorded breath sounds were then computed and averaged by a dual channel Fast Fourier Transform processor and displayed on two storage oscilloscopes. The "peak frequency" (PF) of the breath sounds was defined as the frequency (Hz) of the breath sounds with the greatest sound intensity. The peak frequencies of breath sounds recorded from homologous regions of the right and left hemithorax were found to be remarkably similar. Peak frequencies of breath sounds recorded simultaneously from the apical and basal regions of the posterior chest wall were however, significantly different ($P < .05$); apical recordings yielded higher peak frequencies. The lowest peak frequencies were obtained from breath sounds recorded over the right and left anterior basal lung segments ($P < .001$). Peak frequencies obtained from recordings of breath sounds from the twelve bronchopulmonary segments of the posterior chest wall were then averaged for each individual in order to obtain a mean peak frequency ($\overline{PF}$). In our population $\overline{PF} \pm 1$ SD was 162 ± 22 Hz. It is suggested that peak frequency is one of the determinants of the "pitch" of breath sounds and may be of value in making pulmonary auscultation more precise.

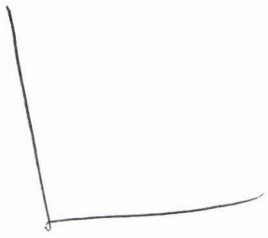


Wheezes

~~RHONCHI~~ AND AIRFLOW DYNAMICS IN ASTHMATIC PATIENTS

S. Ishikawa
P. Workum
S. Holford
R. Murphy, Jr.

Eleven clinically stable asthmatic patients (ages 19-53) were examined on two separate occasions to determine the effects of bronchodilators on the prevalence of rhonchi. Breath sounds were recorded at ten sites, including the trachea and mouth while breathing quietly and during the flow volume maneuver. Attempts were made to correlate the presence of rhonchi with airflow dynamics. The following observations were made: 1) Those who had rhonchi in many sites of sound sampling on Day One also had rhonchi on Day Two, 2) Zones where rhonchi were present in inspiratory and expiratory phase decreased in general after bronchodilator therapy and the change was more pronounced on expiratory phase of quiet breathing, 3) Those who didn't have rhonchi did not develop rhonchi following bronchodilator, 4) Flow volume maneuver intensified breath sound and detectability of rhonchi in individuals who did not have rhonchi during quiet breathing, 5) Rhonchi were detected at various zones of the lung which had an MMEFR as low as 0.3 L/second, 6) The patients who had rhonchi detected only at the central airway showed FEV_1 >70%, FEV_1/FVC >70%, and MMEFR >2 L/second, and 7) There was no direct relationship found between change in parameter of the airflow dynamics and presence of rhonchi.

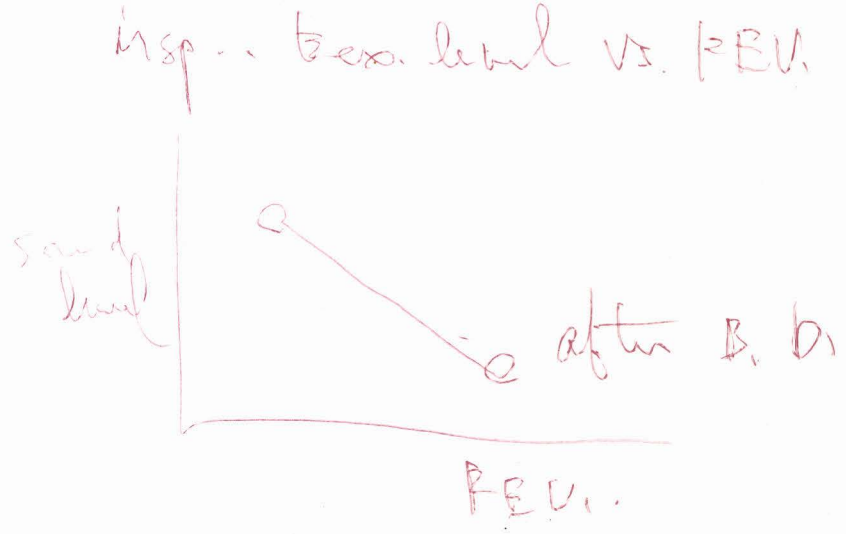


MMFR. > 0.3l

RELATIONSHIP BETWEEN BREATH SOUNDS AMPLITUDE AND BRONCHOSPASM

B.L. Tiep
M.J. Belman
M. Trippe

Biofeedback has been suggested as a useful means of therapy for asthma. We developed a wheeze biofeedback device which picks up breath sounds over the trachea and presents these sounds to the patient as a variable frequency tone or alternatively as a voltage signal. This study was designed to evaluate the validity of this device. In this experiment the output of the device was recorded from the amplitude of the breath sounds displayed on an oscilloscope. We performed breath sound amplitude measurements on 5 patients with bronchial asthma before and after the administration of aerosolized bronchodilators during quiet tidal breathing. In addition, spirometry was also performed before and after bronchodilators. Mean FVC increased from 2.52 $\pm$ .80 to 3.06 $\pm$ .86 liters and mean FEV_1 increased from 1.44 $\pm$ .71 to 1.92 $\pm$ .70 liters after bronchodilators. Concomitant with this increase in flow rates, there was a significant decrease in the amplitude of both expiratory and inspiratory breath sounds from 2.26 $\pm$ .58 to 1.14 $\pm$ .62 volts and from 1.94 $\pm$ 59 to 1.14 $\pm$ .71 volts respectively. All patients improved their FEV_1 after bronchodilators and all showed a decrease in the amplitude of breath sounds. We conclude that 1) this device is an effective and simple means of displaying information regarding intensity of breath sounds to patients and 2) an improvement in the flow rates is associated with a decrease in the amplitude of the voltage signal. We propose to use this device to evaluate the efficacy of biofeedback in the management of bronchial asthma.



CENTRAL TO PERIPHERAL SOUND TRANSMISSION IN EXCISED LUNG

D.A. Rice

Fresh lungs from dogs and horses were inflated with humidified room temperature air to a pressure of 15 cm of water. Sound, both pulsed and frequency swept, was projected down the trachea. Sound at several locations on the visceral pleura was air coupled to a sampling microphone using a conical bell. The following observations were made by comparing sound at the carina with the peripheral locations.

1) Sound was always strongly attenuated (>40dB loss) above 3kHz (dog) and 2kHz (horse). These "cut off" frequencies are comparable to maximum frequencies observed in lung sounds and in transmission studies in intact animals.

2) In the horse lungs the frequency of peak transmission (minimum attenuation) decreased from about 750 Hz to about 300 Hz as the straight line distance between the peripheral and reference microphones increased from minimum to maximum. In the dog lungs the frequency of peak transmission varied little from the mean of about 600 Hz.

3) In the horse lungs, and occasionally in the dog lungs, pleural locations were found with one or more frequencies of near zero transmission in the range 200 to 1000 Hz. When the lung is air dried these same locations show no such transmission minima but do show a decrease in delay time as much as 50%.

This work was performed at The Ohio State University, Department of Veterinary Physiology and Pharmacology, Columbus, Ohio, and supported by the National Heart Lung and Blood Institute, HL-21199.

257 cm/sec

SESSION D

CHAIRMAN: LESLIE CAPEL

Session D

Chairman: Leslie Capel

1:30 pm	Computerized analysis of late inspiratory crackles	P. Wright W. Bishop L. Capel B. McA.Sayers
2:00 pm	Crackles in the early detection of pulmonary asbestosis	F. Shirai Y. Takezawa R. Mikami S. Kudoh A. Shibuya I. Ono
2:30 pm	An investigation of the auscultatory abilities of Edinburgh medical students	A. Leitch A. Morgan R. Wilkie
3:00 pm	Observer variability in breath sound interpretation	P. Workum E. Del Bono S. Shirai K. Sada S. Holford R. Murphy S. Ishikawa
3:30 pm	Summary	W. Ball
4:30 pm	Steering Committee	

COMPUTERIZED ANALYSIS OF LATE INSPIRATORY CRACKLES

P.H. Wright
W.B. Bishop
L.H. Capel
B. McA.Sayers

Recordings of late inspiratory crackles were made from patients with cryptogenic fibrosing alveolitis. Microphones were placed at each lung base and the patients breathed through a pneumotachygraph. The resulting unfiltered sound and flow signals were recorded on an FM tape recorder (Racal Electronics). The patients all had enough crackles that some could be heard in normal tidal breathing. The patients were asked to breathe quietly through the pneumotachygraph so a series of breaths were recorded with very similar flow profiles and durations.

The recordings were processed at Imperial College using the computer of the Department of Electrical Engineering Applied to Medicine. The flow signal was integrated to give the inspired volume, and the sound tracings were examined after the time expansion for evidence of transient waveforms.

Transients containing high frequencies were easily visible predominantly in the late inspiratory part of the breathing cycle. No transients were seen in expiration.

An analysis is presented of the transients seen in a series of ten breaths from a patient with cryptogenic fibrosing alveolitis.

An initial impression that the crackles within a breath differ widely was not supported by analysis of waveform patterns. There was, however, a tendency for earlier crackles at lower inspired volumes, to show wider initial deflections suggesting more high frequency components were present. When the averages of transients found from successive small volumes in all ten breaths were examined this pattern change was seen to occur gradually through the breath. If this change results from filtering of sound arising at different points within the lung acting as a low pass filter it can be represented as if the earlier crackles are a filtered version of later ones.

CRACKLES IN THE EARLY DETECTION OF PULMONARY ASBESTOSIS

F. Shirai
Y. Takezawa
R. Mikami
S. Kudoh
A. Shibuya
I. Ono

In an attempt to provide improved methods of monitoring asbestos workers, the usefulness of fine crackles was studied for their value in detecting early stages of interstitial fibrosis. We investigated 270 asbestos workers and 222 subjects as a control group. The aims of our investigations were: 1) to assess the prevalence of crackles in these populations, 2) to examine the relationship of crackles to duration of exposure, 3) to quantify observer variability in detecting crackles. Observers listened to workers in each of 6 basilar chest sites during deep breathing. Workers were also examined after breath-holding. To assess observer variability 2 observers examined 74 workers chosen at random from 270 asbestos workers. The prevalence of bibasilar fine crackles during deep breathing was 14.4% in asbestos workers and 0.9% in control subjects. However during deep breathing following breathholding at low lung volumes, the rate was increased to 32.2% in asbestos workers and 4.5% in control subjects. The prevalence rose with the increasing duration of exposure. Observer agreement was greatest (100%) that crackles were present when at least one observer heard them in 3 or more sites. Agreement was least (91.9%) when they were heard in only one site. The observers also examined visual recordings of the lung sounds of 56 asbestos workers made by the time-expanded wave form plotting technique and spectral analysis.

AN INVESTIGATION OF THE AUSCULTATORY ABILITIES OF EDINBURGH MEDICAL STUDENTS

A. G. Leitch
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Disagreement about the interpretation and assessment of lung sounds is common, even with experienced observers (1-4). We have, therefore, designed a study which examines the ability of medical students to describe lung sounds and determines the effect on their performance of prior exposure to a teaching tape on lung sounds (5). A discussion session based on tape recording of actual patient lung sounds using an electronic stethoscope is an important educational feature of the study. On completion the study will also allow comparison of the performances of 2nd and 3rd year clinical students. Preliminary findings indicate considerable potential for improving overall student performance.

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OBSERVER VARIABILITY IN BREATH SOUND INTERPRETATION

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One of the problems in appreciating the significance of rales noted on chest auscultation is the lack of observer agreement on breath sound interpretation. The following study was performed to determine if the results of careful auscultation of the lungs are reproducible between observers and, therefore, reliable. Sixty-four patients were examined simultaneously by a physician using a regular stethoscope, and a laboratory technician using an electronic stethoscope while recording the breath sounds. Ten basilar sites were listened to on each patient. The tapes were subsequently listened to by the same two initial observers and two Japanese physicians to whom copies of the tapes were mailed. Inter-observer variation on the presence or absence of rales was calculated between the two on-site examiners and the four examiners who listened to the tapes. Intra-observer variation was calculated for the on-site examiners who subsequently listened to the tape replays. Observer agreement was approximately 90%. The data was further analyzed to account for chance agreement and these calculations are described. The reasons for the good observer agreement noted in this study compared to previous work is discussed. Clinical study methods for determining observer variability are presented.

Pre Clin Year
↓
1st clinical Year
(Medicine 300 h 180 h
Surgery 180 100

Gr A. Gr B.
Tapes Patients
↓ ↓
Patient Tape
↘ ↙
Discussion

Kuder:

EKG 0.71-0.84

IEG 0.53-0.88

Felson & Morgan 0.48 (unacceptable)

Fleiss 0.67-0.81

Schilling 0.76

Smylie " Attendance & total Agreement

Fowler " Statistically Significant Agr.

Hudson 0.49

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