INTERNATIONAL LUNG SOUNDS ASSOCIATION

Toledo, OH October 8 and 9, 2010

2010

The 35th

International Conference on Lung Sounds

University of Toledo Medical Center

THE 35th

INTERNATIONAL CONFERENCE

ON

LUNG SOUNDS

Presented by

The International Lung Sounds Association

October 8 – 9, 2010

Toledo, Ohio

FINAL PROGRAM AND ABSTRACTS

LIST OF ILSA CONFERENCES

No.	Date	Place	Local Organizer(s)
1.	October, 1976	Boston, MA	Raymond L.H. Murphy, Jr.
2.	September, 1977	Cincinnati, OH	Robert Loudon
3.	September, 1978	New Orleans, LA	William Waring
4.	September, 1979	Chicago, IL	David Cugell
5.	September, 1980	London, England	Leslie Capel & Paul Forgacs
6.	October, 1981	Boston, MA	Raymond L.H. Murphy, Jr.
7.	October, 1982	Martinez, CA	Peter Krumpe
8.	September, 1983	Baltimore, MD	Wilmot Ball
9.	September, 1984	Cincinnati, OH	Robert Loudon
10.	September, 1985	Tokyo, Japan	Riichiro Mikami
11.	September, 1986	Lexington, KY	Steve S. Kraman
12.	September, 1987	Paris, France	Gerard Charbonneau
13.	September, 1988	Chicago, IL	David Cugell
14.	September, 1989	Winnipeg, Canada	Hans Pasterkamp
15.	October, 1990	New Orleans, LA	David Rice
16.	September, 1991	Veruno, Italy	Filiberto Dalmasso
17.	August, 1992	Helsinki, Finland	Anssi Sovijarvi
18.	August, 1993	Alberta, Canada	Raphael Beck
19.	September, 1994	Haifa, Israel	Noam Gavriely
20.	October, 1995	Long Beach, CA	Christopher Druzgalski
21.	September, 1996	Chester, England	John Earis
22.	October, 1997	Tokyo, Japan	Masahi Mori
23.	October, 1998	Boston, MA	Sadamu Ishikawa
24.	October, 1999	Marburg, Germany	Peter von Wichert
25.	September, 2000	Chicago, IL	David Cugell
26.	September, 2001	Berlin, Germany	Hans Pasterkamp
27.	September, 2002	Helsinki, Stockholm	Anssi Sovijarvi

No.	Date	Place	Local Organizer(s)
28.	September, 2003	Cancun, Mexico	Sonia Charleston, Ramon Gonzales Camerena & Tomas Aljama Corrales
29.	September, 2004	Glasgow, Scotland	Ken Anderson & John Earis
30.	September, 2005	Boston/Cambridge, MA	Raymond L.H. Murphy, Jr.
31.	September, 2006	Halkidiki, Greece	Leontios Hadjileontiadis
32.	November, 2007	Tokyo, Japan	Shoji Kudoh
33.	October, 2008	Boston, MA	Sadamu Ishikawa & Raymond L.H. Murphy,Jr.
34.	September, 2009	Haifa, Israel	Noam Gavriely
35	October 2010	Toledo, Ohio	Dan E. Olson

ORGANIZATION

President:	Dan E. Olson, M.D., Ph.D.
Chairman:	Raymond L.H. Murphy, Jr., M.D.
Steering Committee:	Sadamu Ishikawa, M.D. Raymond L.H. Murphy, Jr., M.D. John Earis, M.D. Masato Takase, M.D. Jukka Rasanen Shoji Kudoh, M.D. Noam Gavriely, M.D.

HISTORY OF THE INTERNATIONAL LUNG SOUNDS ASSOCIATION

In October 1976, the First International Conference on Lung Sounds was held in Boston, MA. The objectives of this conference were defined as follows:

"Studies of lung sounds have been reported with increasing frequency in recent years. This conference is convened to provide an opportunity for exchange of ideas and experience among those who have an active interest in the subject. Clinicians, physiologists, engineers and perceptual psychologists can each contribute towards a better understanding of what lung sounds mean. They will have a better chance of doing so after talking together."

"We hope that comparisons of methods of recording, analyzing and describing lung sounds will reduce ambiguity. We hope that discussions about work in progress may prevent unnecessary duplication of effort. We hope that investigators will save time and avoid some mistakes by learning what others have done."

Enthusiasm generated by this conference has continued, and annual meetings have been held since. These annual conferences have typically occurred over a period of two to three days being devoted to presentation of papers with discussion, and a half day being devoted to a workshop. Attendance at the conferences has averaged about 60. This is the 35th annual meeting.

Co-founders: Robert G. Loudon and Raymond L.H. Murphy, Jr., M.D.

GENERAL INFORMATION

Conference Venue:

Friday, October 8, 2010	University of Toledo Health Science Campus Dana Conference Center Toledo, Ohio
Saturday, October 9, 2010	University of Toledo Health Science Campus Dana Conference Center Toledo, Ohio
Local Organizing Committee:	Dan E. Olson, M.D., Ph.D. Erin Chesher, Administrative Secretary
Address:	International Lung Sounds Association Raymond L.H. Murphy, Jr., M.D. 1153 Centre Street Boston, MA 02130
Telephone Number: **Fax Number:** **E-Mail**:	617 983-7000, x4436 617 524-4419 rlmurphy@partners.org
Registration:	Registration will be held at the University of Toledo Health Science Campus Dana Conference Center
Registration Fees:	Registration/membership fees will be $75 per person. Resident and student fees will be $25 per person. **NOTE**: NO CREDIT Cards will be accepted
Certificate of Attendance:	Participants, duly registered, will receive a certificate of attendance upon request.
CME:	Category II
Social Evening/Reception:	Friday, October 8, 2010 Maumee Bay Lodge & Conference Center Transportation provided

FINAL

PROGRAM

Friday, October 8

8:30 **Registration**

9:00 **Welcoming Address:**

Jeffrey Gold, M.D.
Chancellor and Executive Vice President
for Biosciences and Health Affairs
Dean of the College of Medicine

Dan Olson, M.D., Ph.D.
Professor of Medicine
Pulmonary, Critical Care
and Sleep Medicine

Scientific Session A

Chairpersons: Sadamu Ishikawa, M.D. & Noam Gavriely, M.D.

9:20 - 9:40 OBJECTIVE COUGH FREQUENCY IN INTERSTITIAL PULMONARY FIBROSIS
A Key, K Holt, A, J.A. Smith, J Earis

9:40 - 10:00 AUTOMATED ANALYSIS OF CRACKLES IN PATIENTS WITH INTERSTITIAL PULMONARY FIBROSIS
B Flietstra, N Markuzon, A Vyshedskiy, R Murphy

10:00 - 10:20 CRACKLES IN IDIOPATHIC PULMONARY FIBROSIS (IPF)
A Key, K Holt, J Earis

10:20 - 10:40 STATISTICAL ANALYSIS OF FORCED EXPIRATORY WHEEZES ORIGIN IN HEALTHY
V Korenbaum, M Safronova, A Dyachenko, I Pochekutova

10:40 - 11:10 **Break**

11:10 - 11:30 ARE "RHONCHI" JUST A LOW PITCH WHEEZES DENOTING BRONCHIAL NARROWING OR RUMBLING SOUNDS DENOTING RETAINED BRONCHIAL SECRETION?
Y Nagasaka, S Yasuda, K Murakami, H Kiyokawa

11:30-11:50 WHEEZING DURING SLEEP
Kanjwal S, D Olson

12:00-12:30 **Photo**

12:30-1:30 **Lunch**

Scientific Session B

Chairpersons: Angela Key, M.D. & John Earis, M.D.

1:30 - 2:30	**Lecture:** **Fluid Mechanics in the Airways and the Physics of Sound** Dan E. Olson, M.D., Ph.D.
2:30 - 2:50	SPECTRAL ANALYSIS OF BREATH SOUNDS IN NON-WHEEZING ASTHMATIC CHILDREN M Takase
2:50 - 3:10	INCREASED ASYNCHRONY OF THE TIMING OF LUNG SOUNDS IN PATIENTS WITH CHRONIC OBSTRUCTIVE LUNG DISEASE A Vyshedskiy, R Murphy
3:10 - 3:30	**Break**
3:30 – 3:50	MULTICHANNEL TRANSMISSION OF SOUND IN THE HUMAN RESPIRATORY SYSTEM V Korenbaum, A Nuzhdenko, A Tagiltsev, A Kostiv, A Dyachenko, N Lopatkin
3:50 - 4:10	LUNG SOUND CHARACTERISTICS OF PATIENT WITH ACUTE LUNG INJURY – ADULT RESPIRATORY DISTRESS SYNDROME (ARDS) S Ishikawa, G Hayes, L Kenney, A DeGorodo, V Pinto-Plata
4:10 - 4:40	THE HISTORY OF JAPANESE LUNG SOUNDS RESEARCH S Kudoh, M Takase, Y Nagasaka, M Munakata, Masashi, Mori
4:40-5:00	**Business Meeting**
5:30	**Bus transportation to Maumee Bay Resort for Dinner**
8:30	**Bus leaves Maumee Bay Resort for The Toledo Hilton Hotel**

Saturday, October 9

Scientific Session C

Chairpersons: Masato Takase, M.D. & Shoji Kudoh, M.D.

9:00 - 9:50	**Lecture:** **The Tale of an Old Stethoscope: From Bampton (1816-65) to the British Thoracic Society** John Earis, M.D. & Helen Earis, M.D.
9:50 - 10:10	CRACKLE PITCH AND RATE DO NOT VARY SIGNIFICANTLY DURING A SINGLE EXAMINING SESSION IN PATIENTS WITH PNEUMONIA, CONESTIVE HEART FAILURE AND INTERSTITIAL PULMONARY FIBROSIS A Vyshedskiy, S Ishikawa, R Murphy
10:10 – 10:30	AIRFLOW-BREATH SOUND CORRELATION USING CUGELL'S FORMULA (1996) E Shapiro, N Gavriely
10:30 - 11:00	**Break**
11:00 – 11:20	VALIDATION OF ANAUTOMATIC WHEEZE DETECTOR E Weizel, Y Genis, A Avrahami, E Dekel, J Tenenbaum-Katan, I Kroin, S Godfrey, N Gavriely
11:20 – 12:00	**Lecture:** **Four Decades in Search of the Illusive Rale (Crackle)** Raymond Murphy, M.D. DS.c.
12:00 - 1:00	**Lunch**
1:00 – 2:00	Steering Committee Meeting

SCIENTIFIC SESSION A

CHAIRPERSONS:

Sadamu Ishikawa, M.D. & Noam Gavriely, M.D.

OBJECTIVE COUGH FREQUENCY IN INTERSTITIAL PULMONARY FIBROSIS

Angela Key[1,3], Kimberley Holt[1,3], Andrew Hamilton[2], Jaclyn A. Smith[2], and John Earis[1,3]

[1] University Hospital Aintree, Liverpool, UK

[2] Respiratory Research Group, University of Manchester, Manchester, UK

[3] University of Salford, Salford, Greater Manchester, UK.

ABSTRACT

Objectives:

Cough is a common presenting symptom in patients with Idiopathic Pulmonary Fibrosis (IPF). This study measured cough rates in IPF patients and investigated the association between cough and measures of health related quality of life and subjective cough assessments. In addition, IPF cough rates were related to measures of physiological disease severity and compared to cough rates in health and other respiratory conditions.

Methods:

Nineteen IPF patients, mean age 70.8 years ±8.6, five female (26.3%) were studied. Subjects performed full pulmonary function testing, 24 hour ambulatory cough recordings, completed a cough related quality of life questionnaire (Leicester Cough Questionnaire) and subjectively scored cough severity with a visual analogue scale. Ambulatory cough recordings were manually counted and reported as number of coughs per hour.

Results:

The 24hr cough rates were high (median 9.4, range 1.5-39.4), with day time rates much higher than night time (median 14.6, range 1.9-56.6 compared to 1.9, range 0-19.2, $p=0.003$). Strong correlations were found between objective cough frequency and both the VAS (day $r=0.80$, $p<0.001$, night $r=0.71$, $p=0.001$) and LCQ ($r=-0.80$, $p<0.001$), but not with measures of pulmonary function. Cough rates in IPF were higher than healthy subjects ($p<0.001$) and asthma patients ($p<0.001$) but similar to patients with chronic cough ($p=0.33$).

Conclusion:

This study confirms objectively that cough is a major, very distressing and disabling symptom in IPF patients. The strong correlations between objective cough counts and cough related quality of life measures suggest that in IPF patient's, perception of cough frequency is very accurate.

Automated Analysis of Crackles in Patients with Interstitial Pulmonary Fibrosis

Flietstra, B., MS, Markuzon, N., PhD, Vyshedskiy, A., PhD, and Murphy, R., MD
Massachusetts Institute of Technology, The Charles Stark Draper Laboratories, and The Faulkner Hospital

Abstract

Background
The crackles in patients with interstitial pulmonary fibrosis (IPF) can be difficult to distinguish from those heard in patients with congestive heart failure (CHF) and pneumonia (PN). Misinterpretation of these crackles can lead to inappropriate therapy.

Purpose
The purpose of this study was to determine whether the crackles in patients with IPF differ from those in patients with CHF and PN and to determine if computerized lung sound analysis is helpful in distinguishing IPF from these conditions.

Materials and Methods
We studied 39 patients with IPF, 95 with CHF and 123 with PN using a multichannel lung sound analyzer (Stethographics, STG16). Recordings were made at 14 sites over the chest. Crackle features were extracted from the time amplitude plots of these recordings and were analyzed using machine learning methods including neural networks and support vector machines.

Results
The IPF crackles had distinctive features and distribution over the chest that allowed them to be separated from those in patients with PN with a sensitivity of 0.82, a specificity of 0.88 and an accuracy of 0.86. They were separated from those of CHF patients with a sensitivity of 0.77, a specificity of 0.85 and an accuracy of 0.82.

Conclusion
Distinctive features are present in the crackles of IPF that help separate them from the crackles of CHF and PN. Automated analysis based on crackle features was seen to be accurate in distinguishing IPF patients from those of CHF and PN. The information used in the study can be obtained by computer analysis at the bedside. This has the potential of aiding clinicians in diagnosing IPF more easily and thus helping to avoid medication errors.

CRACKLES IN IDIOPATHIC PULMONARY FIBROSIS (IPF)

Angela Key[1], Kimberley Holt[1] and John Earis[1].

[1] University Hospital Aintree, Liverpool, UK.

Objectives: Crackles are an important diagnostic feature of Idiopathic Pulmonary Fibrosis (IPF). This study measured the number of inspiratory and expiratory crackles per litre (CPL) and investigated the association between CPL (total number, upper zones and lower zones) and measures of physiological disease severity. In addition the timings of the inspiratory and expiratory crackles during maximal manoeuvres were assessed.

Methods: Fourteen IPF patients mean age 68.5 years (± 8.1), two female (14%) were studied. Subjects performed full pulmonary function testing and underwent crackle recordings. Inspiratory crackles were recorded from a slow inspiratory capacity (SIC) and expiratory from a slow vital capacity (SVC) using the Stethographics Inc. equipment with a modified microphone array. Eight microphones were placed anatomically across the thorax and one at the trachea. The numbers of inspiratory and expiratory crackles per litre were derived from Stethographics Inc. validated algorithm.

Results: The number of inspiratory CPL (median 19.55, range 5.16-85.49) were greater than the number of expiratory CPL (median 8.25, range 1.61-24.27), p=0.001. The CPL were predominantly found in the lower zones (inspiratory p=0.002, expiratory p=0.002). The distribution of crackles throughout the SVC was relatively even (p=0.194), whilst the SIC demonstrated higher numbers of crackles were found between 25-50% of the manoeuvre time (p=<0.001). Inspiratory CPL demonstrated strong correlations with measures of pulmonary function, especially in the upper zones (FEV_1 p=0.001, FVC p=0.007, TLC p=0.006 and DLco p=0.001). Whereas, expiratory CPL demonstrated correlations between pulmonary function and the lower zones (FEV_1 p=0.009, FVC p=0.04, TLC p=0.018 and DLco p=0.036).

Conclusions: **Crackles are predominantly basal inspiratory crackles in IPF and occur in high numbers between 25-50% of the SIC manoeuvre time. CPL showed strong correlations with pulmonary function, particularly the inspiratory CPL in the upper zones and the expiratory CPL in the lower zones. Thus suggesting that in IPF inspiratory crackles in the upper zones and expiratory crackles in the lower zones develop during more advanced disease.**

STATISTICAL ANALYSIS OF FORCED EXPIRATORY WHEEZES ORIGIN IN HEALTHY

V. Korenbaum*[1,2], PhD, prof., M. Safronova[1], A. Dyachenko[3,4], PhD, prof., I. Pochekutova[1], MD, PhD
[1] - V.I. Il'ichev Pacific Oceanological Institute FEB RAS; [2] - Medical Physics Department of Far Eastern Federal University, [3] - State Scientific Center – Institute of Medical Biological Problems RAS;
[4] - A.M. Prokhorov General Physics Institute RAS.

Background: An origin of forced expiratory wheezes (FEWs) remains not quite clear even in healthy. The hypothesizes are known on FEWs formation: flow-induced bronchial wall oscillations and particularly dynamic flatter (Gavriely et al., 1987), vortex shedding on bronchial tree bifurcations (Hardin & Patterson, 1979; Korenbaum et al., 1997-2009) or bronchial/tracheal lumen occlusion (Hardin & Pope, 1992; Lyubimov et. al., 2010; Vovk et al., 2002) or inner corrugated surface (Stoneman et al., 2008). An origin of FEWs in bronchial tree levels may not be identified unambiguously.
The objective is statistical analysis of localization of FEWs sources by bronchial tree levels.
Method: The studied sample included 25 healthy subjects, performed forced exhalation (FE) maneuver with room air (a), helium-oxygen (h) and krypton-oxygen (k) gas mixtures being different in density. FE noises were recorded above trachea and above symmetrical basal areas of right and left lungs. Spectrograms were calculated. Two independent experts subjectively identified in each spectrogram: paths of the most powerful mid-frequency (MF) FEWs (400–600 Hz) and paths of early and late high-frequency (HF) FEWs (above 600 Hz). They evaluated percentage of spectral similarity (PSS, %) of FEW's recorded on the left and the right basal areas of lungs, based on identity of their frequency, duration and amplitude. The 1-st expert estimated PSS value only between single pairs of FEWs being of maximal power, while the second expert considered all pairs of FEWs, being in the limit of dynamic range -35 dB from maximal amplitude of a spectrogram.
Results: Averaged PSS indexes are listed in the Table. An interpretation was based on the assumption that PSS should be near 100% if FEWs were produced in trachea (a common part of respiratory path). Alternatively, when FEWs were produced in more distal levels of bronchial tree, PSS value should be essentially decreased.

Gas mixture	The 1-st expert			The 2-nd expert		
	MF FEWs	Early HF FEWs	Late HF FEWs	MF FEWs	Early HF FEWs	Late HF FEWs
a	84.4	89.6	83.1	55.2	16.2	14.7
h	90	79.5	88.9	36	11.1	12.4
k	70.3	91	83.9	32.1	11.2	12

Mainly PSS values (Table) considerably differ from 100%, in particular for the 2-nd expert that forces to assume not only trachea, but also more distal levels of bronchial tree involvement in FEWs formation. For MF FEWs of maximal power (the 1-st expert), PSS values differ between all three gas mixtures ($p = 0.0103$, Kruskal-Wallis ANOVA by Ranks test). Analogous data of the 2-nd expert are near statistical significance ($p = 0.063$). However paired comparison between (h) and (k) reveals significant differences for both experts ($p = 0.006$, $p = 0.032$, Mann-Whitney U Test). Thus in (h) all MF FEWs sources are localized closer to the mouth, while in (k) more distal bronchi are involved. It may be explained by smaller density of (h), which makes possible more substantial dynamic compression of airway lumen during FE. In particular for MF FEWs of maximal power (the 1-st expert) in (h) sources are localized in vicinity of trachea, while in (a) and (k) principal and probably lobar bronchi are involved. As for vortex shedding mechanism the picture is in concordance with the extrapolated Hardin&Patterson's prediction (Korenbaum, 2009).
Conclusions: FEWs sources are localized not only in trachea. MF FEWs localization by levels of bronchial tree is dependent on gas density being more distal for heavier gas.

The study was partially supported by RFBR grants 09-08-00105, 05-08-18171.

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Are "rhonchi " just a low pitch wheezes denoting bronchial narrowing or rumbling sounds denoting retained bronchial secretion?

Yukio Nagasaka, Shohei Yasuda
Department of Medicine, Kinki University Sakai Hospital
Katsumi Murakami
Department of Pediatrics, Kinki University Sakai Hospital
Chizu Habukawa
Department of Pediatrics, National Hospital Organization Minamiwakayama Medical Center
Hiroshi Kiyokawa
Den-en-chofu Respiratory Clinic, Tokyo

The term "rhonchi" has been the cause of a lot of confusion because sometimes this term is used just as low pitch wheezes which have whistling characters and sinusoidal structures in their time-expanded wave-forms. In other cases, the same term is used to denote the rumbling or snoring sounds which have a more complex form than simple sinusoidal in their time-expanded wave forms and indicates retained secretion in bronchi. The sound analysis of rumbling "rhonchi" is difficult because this has a low frequency of less than 100 Hz and may be masked by noise especially when recorded in our daily practice; the outpatient departments or our hospital wards.

This discrepancy in the terminology of lung sounds makes a lot of confusion in our daily practice in the management of respiratory diseases. When we write "rhonchi" in charts of our patients, some nurses, physiotherapists (PTs) or even physicians understand that this patient has bronchospasm and may need bronchodilator medications. Other nurses, PTs and physicians may take that this patient has retained secretion and needs.

We will raise this problem and present sounds of both low pitch wheezes and rumbles for our hope that this presentation will trigger the discussion and may reach conclusion on this diverse terminology.

WHEEZING DURING SLEEP

Kanjwal S, Olson D.
University of Toledo Medical Center

Background

Wheezes are continuous adventitious sounds having a musical character and lasts longer than 100 milliseconds. The presence of a wheeze may indicate airway stiffening or narrowing and is associated with asthma or other hypersensitivity of the airways. Sleep disturbances are complicated issues involving a host of factors and body systems. Asthma symptoms are thought to be a cause of sleep disturbance, but the reverse is also true. Sleep disturbances due to OSA have previously been linked to wheezing secondary to asthma.

Objectives

We assessed the temporal association between wheeze as detected by continuous, objective and quantitative monitoring and sleep events as detected by standard PSG. We further evaluated the effect of wheeze on sleep quality and wheeze induced sleep fragmentation in association to day time symptoms.

Method

We investigated the relationship of wheezes during sleep to the stage of sleep, arousals, apneas/hypopneas, and the patient's clinical predisposition for bronchospasm. This is a preliminary study assessing entire night wheeze monitoring during ten randomly chosen PSG evaluations for potential sleep apnea. Only ten patient assessments were complete at the time of this preliminary delivery and because of the multiple variables assessed plus significant technical issues, no definite conclusions can be made at this time. After informed consent, random patients who were undergoing a formal PSG for potential sleep apnea or other sleep disturbances were also monitored for wheeze sounds during sleep. Acoustic Respiratory Monitoring (ARM) device was used to record wheeze. Usual PSG monitoring was conducted for sleep fragmentation and sleep architecture.

Results

A total of ten patients (8 males and 2 females) with known sleep related breathing disorder were investigated, only one had a clinical diagnosis of asthma. Four patients were treated for GERD and may have clinical suggestions toward nocturnal regorge. Mean age was 55±12 (range 23 to 75), BMI was 35±5.Sleep structure was disturbed in 8 of 10 patients with reduced deep sleep, REM sleep and prolonged sleep latency. Wheeze sounds were evident in the majority of sleeping subjects but no obvious relationship was observed to suggest that architecture of sleep relates to the onset of wheezes. The onset of wheeze was not obviously associated with the timing of sleep stages, sleep stage changes, arousals, awakenings, leg motions, apneas/hypopneas, or other sleep disturbances. Wheezes were characterized as to time of onset, frequency of pitch, and duration. No associations were evident to sleep architecture at this very preliminary point of this study.

SCIENTIFIC SESSION B

CHAIRPERSONS:

Angela Key, M.D. & John Earis, M.D.

SPECTRAL ANALYSIS OF BREATH SOUNDS

IN NON-WHEEZING ASTHMATIC CHILDREN

Masato Takase, MD, PhD, Department of Pediatrics, Nippon Medical School Tama-Nagayama Hospital, Tokyo, Japan

Lung function tests in asymptomatic asthmatic children often indicate the presence of mild airflow limitation. Significant improvements in lung function indices in response to bronchodilator inhalation strongly suggest the diagnosis of asthma. Inspiratory breath sounds are believed to be originated from peripheral airways and measurement of their sound spectra could be an alternative for lung function testing. We have reported our studies on the spectral changes of inspiratory breath sounds in asthmatic children since 1995. The studies revealed that the power shift to higher frequency, measured by spectral edge frequency (SEF) indicates the presence of mild airway narrowing in non-wheezy asthmatic children. These findings were confirmed by other Japanese investigators using the simpler indicator, highest frequency of inspiratory sound (HFI). We would like to present the summary findings of the studies concerning spectral analysis of breath sounds in asthmatic children. Our findings suggest the importance of careful auscultation, because there are much more information other than just the presence or absence of wheezes.

Increased Asynchrony of the Timing of Lung Sounds in Patients with Chronic Obstructive Lung Disease

A. Vyshedskiy, R. Murphy,
Brigham and Women's / Faulkner Hospitals, Boston, MA, USA

Abstract

Objective

Uneven distribution of ventilation has been shown to be present in patients with COPD using a variety of complex techniques. Our objective in this study was to quantify the asynchrony of the start and end of the breath sound pattern of inspiration detected by computer in patients with COPD as compared to patients with other conditions.

Methods

A 16-channel lung sound analyzer (Stethographics Model STG1602) was used to collect 20s samples of sound during deeper than normal breathing from patients with COPD (n=101) and patients with no known cardiopulmonary disease (controls, n=379). We also studied patients with pneumonia (PN, n=118), congestive heart failure (CHF, n=92) , bronchial asthma (n=62), and interstitial pulmonary fibrosis (IPF, n=39). The difference in timing between the start of inspiration at the trachea and the start of inspiration at each of 14 chest wall sites was calculated. The asynchrony of the start of the inspiration (SA) was defined as the ratio of the mean of these starting time differences to the duration of the inspiration at the trachea. The end of inspiration asynchrony (EA) was defined similarly.

Results

The SA averaged -1±14% for controls, 0±6% for IPF, -5±12% for asthma, -6±18% for CHF, -8±15% for PN, and -16±29% for COPD. The negative sign indicates that chest microphones detected the start of sounds associated with inspiration before the start was detected by the tracheal microphone. The EA averaged 14±21% for controls, 13±13% for IPF, 12±19% for asthma, 19±21% for CHF, 17±23% for PN, and 25±33% for COPD. Asynchrony of the start and end of inspiration was significantly more common in COPD than in controls ($p<0.0001$).

Conclusion: The mechanism of the inter-channel asynchrony is unknown, but a possible explanation is regional variations in resistance and elastance. In other words in a normal subject as the chest wall moves outward on inspiration the airways dilate relatively uniformly and the lung is uniformly expanded. In COPD, the lung dilatation is less likely to be uniform and the dilatation is more apt to be asynchronous secondary to regional variations in resistance and elastance.

Clinical Implications: A long-term goal of studies with multichannel lung sound analyzers is to provide useful diagnostic information at the bedside. The increase in the inter-channel asynchrony together with other features of COPD, such as decreased amplitude of sound and relatively prolonged expiratory phases can help provide evidence that COPD is present. This can be done using a simple test that requires little patient cooperation.

MULTICHANNEL TRANSMISSION OF SOUND IN THE HUMAN RESPIRATORY SYSTEM

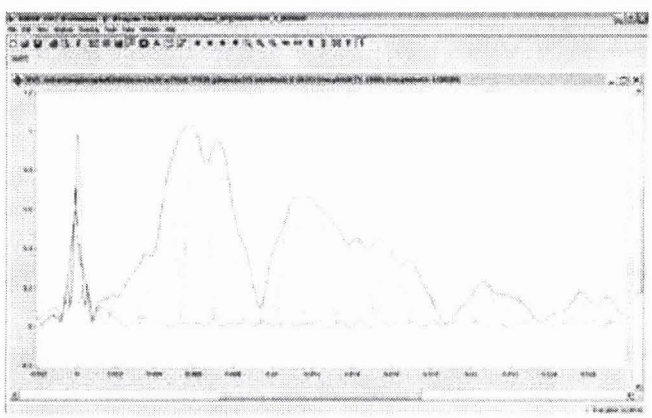
V. Korenbaum*[1,2], PhD, prof., A. Nuzhdenko[1], PhD, A. Tagiltsev[1], PhD,
A. Kostiv[1], PhD, A. Dyachenko[3,4], PhD, prof., N. Lopatkin[2]

[1] - V.I. Il'ichev Pacific Oceanological Institute FEB RAS; [2] - Medical Physics Department of Far Eastern Federal University, [3] - State Scientific Center – Institute of Medical Biological Problems RAS;
[4] - A.M. Prokhorov General Physics Institute RAS.

Background: Sound transmission in a human respiratory system represents one of the most complicated questions of respiratory acoustics. A complexity of the respiratory path structure has already caused assumptions on existence of several ways of sound transmission from the mouth to a chest wall (Buller, Dornhorst, 1956; Nemerovsky, 1981; Korenbaum et al., 1998, Bergstresser et al., 2002). Korenbaum et al., 2008 by means of complex sounding signals convolution experimentally revealed in the small sample two sound transmission mechanisms being involved simultaneously.

The objective is a study of these mechanisms in expanded sample of subjects and analysis of their dependence on filling respiratory system by various gas mixtures.

Method: The studied sample included 25 healthy subjects, performed slow exhalation maneuver with room air, helium-oxygen and krypton-oxygen gas mixtures which had various density and sound velocity but approximately the same adiabatic bulk modulus. Phase manipulated (three 511 symbol m-coded sequences with carrying frequency 200, 300, 750 Hz, general duration 2.5 s and frequency sweep (80-1000 Hz, duration 20 s) direct and reverse signals were injected into mouth by means of the installation (Korenbaum et al., 2009, 2010). Signals transmitted to two chest wall sites (above bottom part of trachea, and above basal area of right lung) were recorded by light accelerometers. A convolution procedure was carried out by calculation of cross-spectrum of synchronous fragments of electric copy of sounding signal with responses received by sensors. Subsequent inverse Furrier transformation, capture of the module, and Hilbert transformation were made (figure).



Results: At least two signal arrivals (global maxima) are recognized in each subject convolution curve recorded above basal area of lung for any gas mixture being similar in time for all types of signals (figure). The main arrival is recognized above trachea, which is accompanied by the second arrival sometimes. The first arrivals times above trachea in the sample are statistically dependent on gas mixture ($p<0.001$, Kruskal-Wallis ANOVA by Ranks) for all signals. The first arrivals times above basal area of lung are also dependent on gas mixture ($p<0.05$). However, the second arrivals times above basal area of lung are independent on gas mixture ($p>0.05$). Thus we may speculate that the main arrival above trachea and the first arrival above basal area of lung, being dependent on filling gas sound velocity, are transmitted through airways lumen at least in part of their path. Alternatively the second arrival recorded above basal area of lung, being independent on filling

gas sound velocity, seems to be purely transmitted through lung parenchyma. Really sound velocity in lung parenchyma is weakly responsible on gas phase density, and is controlled by tissue density and gas adiabatic bulk modulus (Bergstresser et al., 2002), being approximately the same for studied gases.

Conclusions: The simultaneous existence of at least two various ways of sound transmission in human lungs, distinguished on resulting sound velocity of mechanical oscillations, is experimentally proved in expanded sample of subjects for three types of signals used. Obtained dependences of arrival times on sound velocity in gas mixture allow connecting the first of this ways with air-structural transmission and the second one with pure structural transmission.

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LUNG SOUNDS CHARACTERISTICS OF PATIENT WITH ACUTE LUNG INJURY – ADULT RESPIRATORY DISTRESS SYNDROME (ARDS)

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This is a 65 year old smoker who has had 'ARDS' twice in the past (year 20012 and 2006), which was managed by us, with a conventional accepted therapeutic modality. (Intubation, Positive End Expiratory Pressure (PEEP), Ventilation with 100% oxygen) and recovered.

This time (Dec. 2009), we were able to manage her in the same RICU (Respiratory Intensive Care Unit) without intubation and ventilatory support by respiratory (initial PO2 was on 48 mmHg on room air). We have achieved and maintained sufficient oxygenation (Sa02 98%), with a high flow oxygen via nasal cannula. With appropriate supportive care, her condition improved rapidly. After 4 days of intensive therapy, she was no longer in distress. Oxygenation was sufficient (Sa02 98%) with only 2 l/min of nasal cannula oxygen. Therefore, we were able to record her lung sounds without interference of noises from the environment (primarily due to air forcing in and out of the lung by the respirator). Lung sounds recordings were made by Murphy's STG 16 during the peak of ARDS (Dec. 30, 2009) and upon recovery (January 3, 2010). At the peak of ARDS, she was in distress, tachypneic with very brief inspiration with prolonged expiratory phase of breathing. She was full of rhonchi with numerous crackles in the lung. On recovery, her lungs showed inpsiratory carckles on upperzones, and inspiratory and expiratory crackles on the lower zones. Rhonchi were no longer present. I have been following this woman who had severe ARDS developed at age 48 and recovered by conventional therapy, which included intubation and PEEP ventilation with 100% oxygen. She was self extubated upon her recovery and survived. During 20 years of follow up, her pulmonary function gradually improved towards normal.

In 12 years her total lung capacity became 70% of predicted value with normal diffusing capacity. However, crackles persisted (inspiratory on bases and expiratory on upper zones). My examination on September 7, 2010 (20 years after the episode of ARDS) revealed she no longer had any crackles in the lung.

The History of Japanese Lung Sounds Research

Shoji Kudoh1), Masato Takase2), Yukio Nagasaka3), Mitsuru Munakata4), Masashi, Mori5) 1) Double-Barred Cross Hospital (Fukujuji Hospital), Japan Anti-Tuberculosis Association, 2) Nippon Medical School, 3) Kinki University, 4) Fukushima Medical University, 5) Towa Hospital

The resent Japanese lung sounds research started from the study on fine crackles of pulmonary fibrosis in the 1970's because the research group on pulmonary fibrosis was established supported by Ministry of Health and Welfare. KudohI first attended 2nd ILSA conference in Cincinnati, 1977 when Dr. Murphy' s "Time-Expanded Wave-Form Analysis" was published in NEJM. by Murphy.

Japanese Lung Sounds Association was established in1983 to prepare for 10th ILSA conference by Prof. Mikami in Tokyo, 1985. During the conference, we had an important and first international symposium on lung sounds nomenclature including US, UK, Germany, France and Japan. The contents were published in the Journal of the Japan Medical Association (1985; 94:2050-55) and in the Chest (1987; 92:342-345). After the conference, Japanese lung sounds nomenclature on auscultation which had followed German classification until then for a long time, but it changed after the conference.

Japanese Lung Sounds Conference has continued annually since 1983. During the past 275 years, we have discussed many subjects themes on lung sounds including methodology of analytic simethod, s, clinical problems, nomenclature, snoring, and cough sounds, etc.

and We performed ILSA conference had tree times of ILSA conference in Japan, i.e., 10th (1985, Mikami R), 22ed (1997, Mori M) and 32ed (2007, Kudoh S).

We had an international educational symposium "Aassessment of Llung Aauscultation" following the 32ed ILSA conference in Tokyo. More than two hundred participants including , i.e., physiciansdoctors, nurses and physical therapists attended the symposium. This year, Munakata gave a lecture entitled "Art and Science of Pulmonary Auscultation" at the annual meeting of Japan Society for Medical Education. The eEducation is an important future direction of ILSA as well as science and technology on lung sounds.

SCIENTIFIC SESSION C

CHAIRPERSONS:

Masato Takase, M.D. & Shoji Kudoh, M.D.

Crackle pitch and rate do not vary significantly during a single examining session in patients with pneumonia, congestive heart failure, and interstitial pulmonary fibrosis

Vyshedskiy, Andrey, PhD, Ishikawa, Sadamu, MD, and Murphy, Raymond, MD, Brigham and Women's / Faulkner Hospitals, Boston, MA.

BACKGROUND:
The purpose of this study was to determine the variability of crackles during a single examining session as assessed by a computerized lung sound analyzer (STG).

METHODS:
Using a 16-channel STG we examined the crackle pitch (CP) and crackle rate (CR) of 49 patients with pneumonia (PN), 52 with congestive heart failure (CHF), and 18 with pulmonary fibrosis (IPF). Patients were instructed to perform breathing maneuvers in the following sequence: normal breathing, deep breathing, cough several times followed by deep breathing, and a vital capacity maneuver (VCM), followed by a deep breathing maneuver.

RESULTS:
CP variability expressed as a percentage of the average CP was relatively small in all conditions and in all maneuvers: PN=11%; CHF=11%; IPF=7%. CR variability was also relatively small: PN=31%; CHF=32%; IPF=24%. Compared to the 1st deep breathing maneuver (100%), the average CP did not change significantly following coughing (PN=100%; CHF=103%; IPF=100%), the VCM (PN=100%; CHF=92%; IPF=104%), or during quiet breathing (PN=97%; CHF=100%; IPF=104%). Similarly, the average CR did not change significantly following coughing (PN=105%; CHF=110%; IPF=90%) or the VCM (PN=102%; CHF=101%; IPF=99%). However during normal breathing the CR was significantly lower in PN (74%, $p<0.0001$) and significantly higher in IPF (147%, $p<0.05$) than it was during deep breathing. In patients with CHF, the average CR during the normal breathing was not significantly different from the CR during the 1st deep breathing maneuver (108%).

CONCLUSION:
Crackles in all three conditions were surprisingly stable. Neither CP nor CR changed significantly from breath to breath or from one deep breathing maneuver to another even when these maneuvers were separated by the cough and the VCM.

CLINICAL IMPLICATIONS:
The observation that CR is a relatively reproducible measurement during one examination provides evidence that it can be used to follow the course of cardiopulmonary illnesses such as PN, IPF, and CHF.

Title: Airflow-Breath sound correlation using Cugell's formula (1996)

E Shapira, N Gavriely, MD, D.Sc.
KarmelSonix (Israel) Ltd., Haifa, Israel.

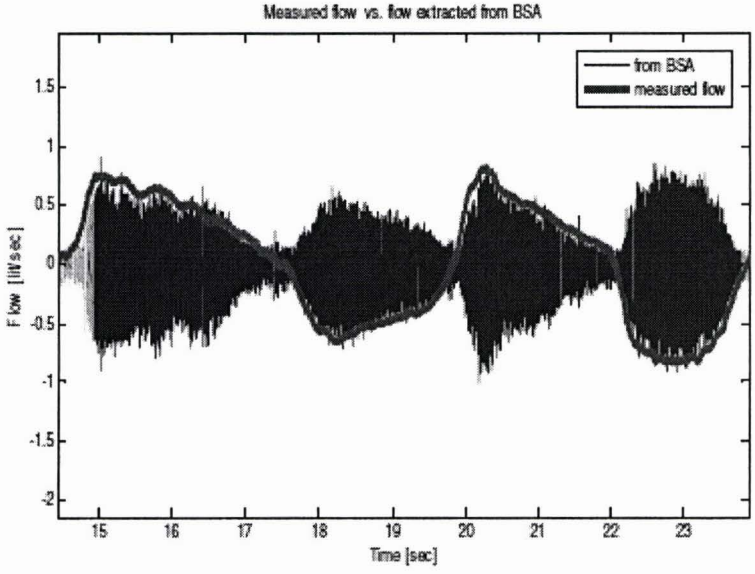
Introduction: This study describes the evaluation of the airflow rate using an acoustic technique. The advantage of this possible pulmonary function testing technique is that it does not require active patient cooperation.

Methods: The flow was measured in a quiet room using a pneumotachograph (Validyne) and was acquired using a Pulmotrack® system (KarmelSonix (Israel) Ltd, Haifa, Israel). Simultaneously the tracheal breath sounds were monitored in a different channel of the Pulmotrack®. The correlation between breath sounds amplitude and airflow rate was based on Cugell's formula:

$$BSA = a \cdot F^{b} \Rightarrow F = c \cdot BSA^{\frac{1}{b}}$$

In this equation F is the flow rate, BSA is the breath sound amplitude and a, b & c are constants.

Results: using empirical values for the constant b (different for each phase of breath) and assessing the values of the constant c, we got the following results:



The constant's values are: for expiration - b=1.68, c=$6.62e^{-5}$, for inspiration - b=1.93, c=$7.41e^{-5}$.

Conclusions: this acoustic technique of assessing the airflow rate from the breath sounds may be a useful tool for monitoring and quantification of pulmonary function.

Title: Validation of an Automatic Wheeze Detector

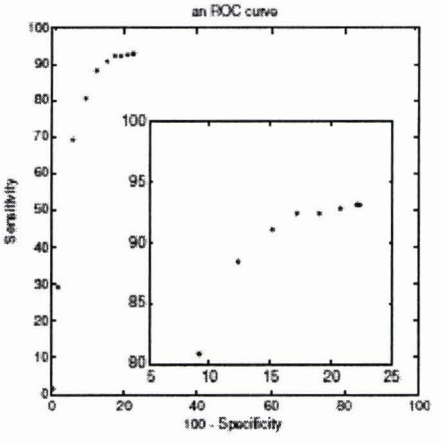
Eldad Weizel, Yulia Genis, Alon Avrahami, Eyal Dekel, Janna Tenenbaum-Katan, Isaac Kroin, Simon Godfrey and Noam Gavriely, KarmelSonix (Israel) Ltd. Haifa, Israel.

Introduction:
Automatic Wheeze Detection is needed for acoustic non-invasive monitoring of the airways in Asthma and other obstructive lung diseases. The duration of wheeze as percent of the elapsed time (Tw/Ttot) has been established as a measure of wheeze severity expressed as the Wheeze Rate (Wz%) and implemented as the Automatic Wheeze Detector Algorithm in a commercial device (PulmoTrack®, KarmelSonix Ltd, Haifa, Israel). The purpose of the present study was to calibrate the algorithm and to validate its performance in comparison with the opinion of a panel of experts.

Methods:
In this study the performance of an Automated Wheeze Detector was evaluated using a database of recordings made on 17 patients and 6 healthy subjects who participated in clinical studies in which breath sounds were recorded. The study describes an optimization method (ROC) for comparing the performance of the algorithm with that of the experts while varying parameters of the algorithm (e.g. threshold). The study also describes two methods for validating the accuracy of the Algorithm: (a) Sensitivity and Specificity analysis and (b) regression analysis of the correlation between the Algorithm determination and the consensus of a panel of 4 experts who reviewed the recordings using audio and visual inspection of the sonograms.

Results:
The results of a mixed data sample from asthma patients (pediatric and adults) and normal volunteers show Sensitivity of 85.0 and Specificity of 97.6 with a regression equation given by $AlgWz\% = 0.97*(ExpWz\%) + 1.51$ ($p<0.001$) where AlgWz% and ExpWz% are the Algorithm and Experts wheeze rate, respectively. These values were as accurate as each individual panel member.

Conclusions:
We conclude that the Automatic Wheeze Detector algorithm accurately detects wheezing in realistic clinical environments, including those that are inherently noisy.



An example of an ROC curve. Such curves were used to optimize values of the algorithm parameters.