ILSA

International
Lung Sounds Association

37th Annual Conference
Charter House
Mayo Clinic, Rochester, MN
October 4.-5. 2012

Conference Program

MAYO
CLINIC

Dear Participants of the 37th ILSA meeting,

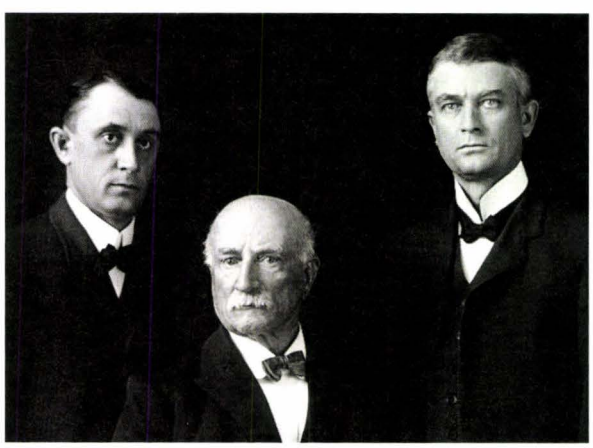
It is my distinct pleasure to welcome you to Mayo Clinic in Rochester, Minnesota.

For more than a century, people from all walks of life have found answers at Mayo Clinic, the first and largest integrated, not-for-profit group practice in the world. Doctors from every medical specialty work together to care for more than a million people each year, joined by common systems and a philosophy of "the needs of the patient come first."

From its humble beginnings in the cornfields of southeastern Minnesota, Mayo Clinic has opened its doors to visiting physicians and scientists from around the globe. It is in this collaborative spirit that I extend a warm welcome to the members of the International Lung Sounds Association.

Regards,

Jukka Räsänen



Drs. Charles H. Mayo, William W. Mayo and William J. Mayo
Founders, Mayo Clinic

From the Organizers…..

Welcome to Rochester!

Thank you for participating in the 37th Annual Conference of the International Lung Sounds Association held in the Downtown Campus of Mayo Clinic Rochester. The presentations will be held at the Charter House Auditorium within walking distance of any downtown hotel. Please see attached map for location.

The Rochester International Airport has shuttle service to the downtown area. The drive is approximately 20 min. Please sign up for transport at the baggage claim area of the airport.

On Thursday, October 4th, all conference participants are invited for a cocktail hour followed by dinner at the home of Jukka and Gina Räsänen, 6011 Countryview Court NW in Rochester. Transportation will be provided and can be accessed at the Eastern entrance to the Kahler Grand Hotel, as marked on the map.

For questions or assistance please call (+1.507) 269.5787

We are looking forward to a great meeting!

ILSA 2012
Scientific Program

Thursday, October 4th

8:30	***Registration and light breakfast***		
9:00	Opening remarks	*Sadamu Ishikawa, MD*	*USA*
9:05	Welcome	*Jukka Räsänen, MD*	*USA*
9:10	Mayo Clinic - A brief history	*Gina Vitali-Räsänen, BS*	*USA*
9:30	History of the Stethoscope	*Hugh Smith, MD*	*USA*
10:00	History of the International Lung Sounds Association	*Raymond Murphy, MD*	*USA*
10:30	The Bill & Melinda Gates Foundation and Technology Needs in Developing Countries	*Deborah Burgess, PhD*	*USA*
11:00	***Lunch***		
12:00	Sound in Anesthesia and Critical Care: Current Practice and Future Needs	*Michael Nemergut, MD*	*USA*
12:30	Pulmonary Acoustics in Acute Lung Injury	*Jukka Räsänen, MD*	*USA*
13:00	Four Decades in Search of the Elusive Rale	*Raymond Murphy, MD*	*USA*
13:30	Crackle Morphology in Acute and Chronic Lung Disease	*Noam Gavriely, MD*	*Israel*
14:00	***Coffee break***		
14:30	Acoustic Biomarkers of Chronic Obstructive Lung Disease	*Raymond Murphy, MD*	*USA*
14:50	Flow Measurement at the Mouth Significantly Changes Lung Sound Amplitude Measurement	*Raymond Murphy, MD*	*USA*
15:10	Ready to Eat?! Sound and Watermelon Maturity	*Gregory Schears, MD*	*USA*
18:00	***Cocktail hour*** *and*	*tel. 507 269-5787*	
19:00	***Dinner*** *at the home of Jukka Räsänen and Gina Vitali-Räsänen, 6011 Countryview Court NW, Rochester 55901*		

Friday, October 5th

8:00	***Assembly and light breakfast***		
8:30	Lung Sound Indices in the LongTerm Management of Bronchial Asthma	*Yukio Nagasaka, MD*	*Japan*
8:50	Diagnosis of Obstructive Sleep Apnea from Awake Tracheal Breath Sounds	*Zahra Moussavi, PhD*	*Canada*
9:10	Evolution of Crackles in a Patient with Idiopathic Pulmonary Fibrosis on Pirfenidone Therapy	*Sadamu Ishikawa, MD*	*USA*
9:30	Characteristics of Breath Sounds when Asthmatic Patients Have Wheezes or Rumbling Rhonchi	*Michiko Tsuchiya, MD*	*Japan*
9:50	***Coffee break***		
10:10	Effect of Respiratory Flow Measurements with a Pneumotachograph on Tracheal Breath Sounds	*Zahra Moussavi, PhD*	*Canada*
10:30	Cardiac Response to Respiration	*Sadamu Ishikawa, MD*	*USA*
10:50	Closing remarks	*Jukka Räsänen, MD*	*USA*
11:00 - 12:00	***Tour of the Downtown Mayo Clinic Facilities***	*Gina Vitali-Räsänen, BS*	*USA*

Acoustic biomarkers of Chronic Obstructive Lung Disease

Andrey Vyshedskiy[1], Andy Eaton[2], Brian Leaker[2], and Raymond Murphy[1]
[1]Brigham and Women's / Faulkner Hospitals, Boston, MA, USA; [2]Queen Ann Street Medical Center, London, UK

Objectives: The goal of this study was to determine lung sounds-derived biomarkers that distinguished Chronic Obstructive Pulmonary Disease (COPD) patients from age-matched controls and to see if these biomarkers were helpful in detecting early effects of smoking.

Methods: We used a multichannel lung sound analyzer (Stethographics Model STG-1602) that provides acoustic data from multiple sites on the chest wall to derive over 100 acoustic biomarker candidates. Sixteen biomarkers based on timing, frequency, amplitude, and adventitious sounds were statistically different between COPD (90 patients) and control (90) patients recruited in Boston, MA (test set). These biomarkers are:

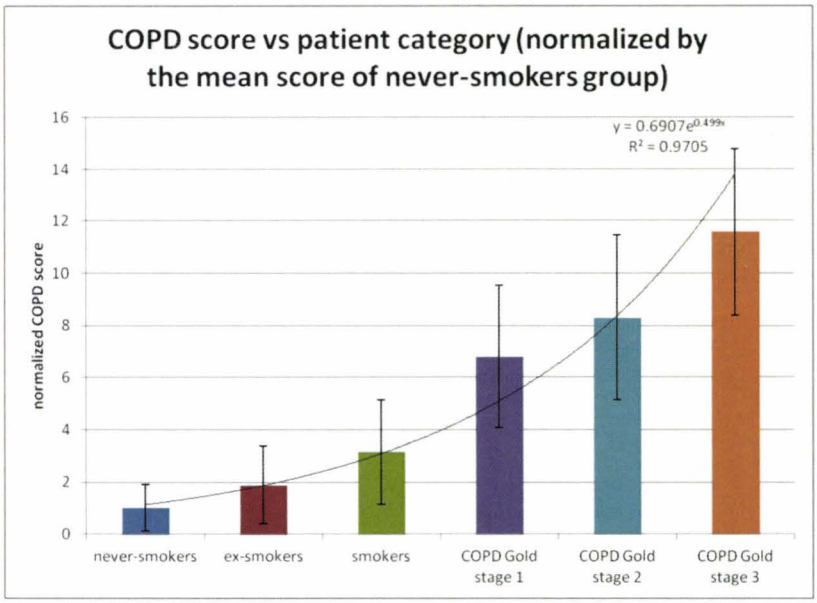
1	Inspiratory crackle rate	9	Average lag of chest channels compared to the trachea
2	Expiratory crackle rate	10	Inter-channel asynchrony at the beginning of inspiration
3	Inspiratory wheeze and rhonchi rate	11	Inter-channel asynchrony at the end of inspiration
4	Expiratory wheeze and rhonchi rate	12	Lead time-integrated amplitude
5	Ratio of the duration of inspiration to the duration of expiration	13	Lag time-integrated amplitude
6	Ratio of peak inspiratory amplitude to peak expiratory amplitude	14	Dynamic range
7	Ratio of low frequency energy to high frequency energy (R4)	15	Amplitude of inspiratory chest sounds
8	Average lead of chest channels compared to the trachea	16	Slope of the chest versus tracheal sound

After examining the frequency distribution of the test set the scoring system was developed. Each of the 16 parameters was evaluated and a score was assigned based on the value of the individual parameter. The total Acoustic COPD Score was calculated as the sum of the individual score for each parameter. The Acoustic COPD Score was then evaluated in four groups of patients recruited in London, UK (control set):

1. COPD (60 patients, GOLD Stages 1-2), who are current smokers with at least a 10 pack-year smoking history;
2. Current smokers (60 patients) with at least a 10 pack-year smoking history and age-matched to the COPD cases;
3. Ex-smokers (60 patients) with at least a 10 pack-year smoking history who have not smoked for at least one year and age-matched to the COPD cases;
4. Never-smokers (60 patients) who were age-matched to the COPD cases.

Results: Acoustic COPD score was significantly greater for COPD, smokers, and ex-smokers groups compared to never-smokers ($p<0.0001$ for all pair-wise comparisons with never-smoker group).

Conclusions: This study showed that measurable differences exist between the lung sound patterns of COPD patients, smokers, ex-smokers and age-matched never-smokers. Lung sounds derived biomarkers are helpful in diagnosing COPD and in detecting the early effects of smoking.



Flow measurement at the mouth significantly changes lung sound amplitude measurement

Andrey Vyshedskiy[1], Andy Eaton2, Brian Leaker[2], and Raymond Murphy[1]

[1]Brigham and Women's / Faulkner Hospitals, Boston, MA, USA; [2]Queen Ann Street Medical Center, London, UK

Objectives

The goal of this study was to determine the effect of flow measurement at the mouth on the measurement of lung sound amplitude in normal subjects.

Methods

We used a multichannel lung sound analyzer (Stethographics Model STG-1602) that provides acoustic data from multiple sites on the chest wall to record lung sounds with and without a flow measuring device (pneumotach) attached at the mouth. Patients were asked to breathe deeper than normal for 20 seconds (with and then without the pneumotach). Sound loudness was assessed by calculating root mean square (RMS) of the inspiratory sound at each chest site and then averaging between the fourteen chest sites.

Results

Measuring flow at the mouth reduced chest acoustic RMS in 32 of 66 subjects (49%). The reduction ranged from 2% to 60% (mean 27%). In another 32 patients measuring flow at the mouth increased chest acoustic RMS. The reduction ranged from 3% to 197% (mean 52%). Only in two subjects measuring flow at the mouth was not associated with a change in lung sound amplitude.

Conclusions

Sound loudness (measured as RMS) is proportional to air flow. In a given patient doubling of the airflow results in doubling of the RMS. In most subjects in this study measuring airflow at the mouth resulted in a significant change in measured RMS. Therefore we conclude that the pneumotach altered airflow. Furthermore the direction of change was unpredictable. In approximately half of the subjects the mere fact of inserting a pneumotach into the mouth reduced airflow and in the other half it increased airflow. Accordingly it is difficult to interpret the clinical or physiologic significance of RMS measurements made when a pneumotach is in the mouth.

We can only speculate why addition of a pneumotach changed airflow. A pneumotach increases flow resistance. Since patients only control breathing effort, it is possible that patients who attempt to conserve breathing effort end up with a reduction of airflow. On the other hand, patient who attempt to conserve airflow, end up overcompensating and, as a result, actually increase their airflow.

In any case, researchers thinking of adding a pneumotach to their protocol have to keep in mind that addition of a pneumotach unpredictably changes the measured sound amplitude.

Lung sounds indices in the long-term management of bronchial asthma.

Yukio Nagasaka[1], Michiko Tsuchiya[1], Takuma Minami[1], Terufumi Shimoda[2], Shohei Yasuda[3], Katsumi Murakami, [4] Chizu Habukawa[5]

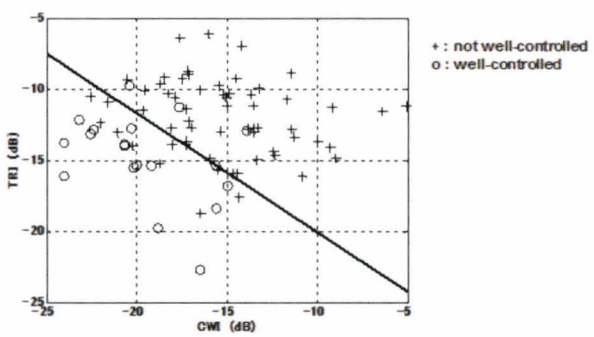
1. Dept. of Pulmonary Medicine, Rakuwakai Otowa Hospital 2. Dept. of Clinical Research National Fukuoka Medical Center 3. Dept. of Medicine, Horiguchi Seikeigeka Hospital 4. Dept. of Pediatrics, Kinki University Sakai Hospital 5. Dept. of Pediatrics, National Minami Wakayama Medical Center

Many studies of acutely induced airway narrowing showed that an increase of frequency or power of breath sounds were more sensitive sound parameter than wheezes. Thus, changes of lung sounds are expected to be good indicator of airway narrowing or control of asthma. We evaluated if differences in breath sounds may reflect the control status of asthmatic children.

Eighty asthmatic children (aged 10.3 ± 3.0 years) underwent breath sound analysis when they had no overt asthmatic symptoms, including wheezing and asthmatic fit. Asthma control was assessed by Asthma Control Test™ and divided into well controlled (scores were perfect) (n=19) and not well controlled (scores were not perfect) (n=61). Breath sounds were recorded using two sensors, located on right anterior chest and trachea. Then chest wall sound transmission index (CWI) and tracheal sound index (TRI) were calculated. A new parameter, breath sound index (BSI) was calculated from a 2-dimensional diagram of CWI and TRI. As acoustic transfer characteristics of chest and tracheal sound correlated well with the body size (Habukawa, Jpn J Clin Physiol 2011), we standardized the data according to this estimation.

There was a significant difference of CWI and TRI between well-controlled and not well-controlled groups ($p < 0.001$). BSI had better discrimination accuracy between the two groups ($p < 0.001$) than CWI or TRI alone. Sensitivity and specificity of BSI on asthma control were 83.6% and 84.2%, respectively.

Although our results are still preliminary, it will be possible to evaluate asthma control by a new index calculated from breath sound analysis.



Diagnosis of obstructive sleep apnea based on spectral features of tracheal breath sounds during wakefulness

Davood Karimi and Zahra Moussavi
Biomedical Engineering Program, University of Manitoba, Winnipeg, MB R3T 5V6, Canada

Rationale - Obstructive sleep apnea (OSA) is massively underdiagnosed mainly due to the time consuming and high-cost nature of the current assessments. This study aimed at developing a new technique for OSA diagnosis based on short recordings of tracheal breath sounds during wakefulness.

Methods - Tracheal breath sounds were recorded from 90 individuals during nose and mouth breathing at their tidal flow rate in supine and upright postures. The apnea-hypopnea index (AHI) of the subjects was determined through the full-night polysomnography. Several spectral features were computed from the power spectrum density of the sound signals. These features were computed from different frequency bands: 150-450 Hz, 450-600 Hz, and 600-1200 Hz and 150-1200 Hz. The differences of these features between supine and upright breathing and between nose and mouth breathing were also calculated. Data from 60% of the non-OSA (AHI<5) and severe OSA subjects (AHI>30) were used to select the most significant features that were statistically different between these two groups; data from the rest of the subjects were used to evaluate the classification accuracy. Then, an exhaustive algorithm was used to select the best combination of 3 characteristic features for diagnostic classification. The classification method employed in this study was an ad hoc method based on voting from the individual 3 features.

Results - The best features included spectral centroid over both low (150-450 Hz) and high (600-1200Hz) frequency bands, and signal's average power over 600-1200 Hz. These features, extracted from different breathing maneuvers and differences between the maneuvers, formed the best feature set. Having investigated many feature sets, overall, we found that that the spectral features of breath sounds are correlated with AHI, and maybe used for OSA diagnosis. The detailed classification results of the best 3 feature set for the population of this study are shown in Table 1. If the goal is defined as diagnosing anybody with AHI>15, then the proposed method's sensitivity and specificity are 84% and 86%, respectively (Table I).

Conclusions - The results indicate that power spectral features of breath sounds collected during wakefulness have the potential to be used for OSA diagnosis. The accuracy of the method decreases when classifying into four classes with no gap between the AHI values, as expected. It is likely that the classification error can be reduced by matching for anthropometric parameters such as age, BMI, and gender.

TABLE 1. Classification Results

True/Assigned	AHI<5	5<AHI<15	15<AHI<30	AHI>30
AHI<5	**27**	6	1	2
5<AHI<15	10	**8**	3	2
15<AHI<30	4	1	**6**	3
AHI>30	0	0	0	**17**

EVOLUTION OF CRACKLES IN A PATIENT WITH IDIOPATHIC PULMONARY FIBROSIS ON PIRFENIDONE THERAPY

S.Ishikawa(1),S. Izumi(2),S.Kudoh(3),A.Vyshedskiy(4),R,L.Murphy(5),P.LaCamera(6)and A.Ernst(7)

(1,6,7) Pulmonary & Critical Care, Steward St Elizabeth's Medical Center, Boston, MA, U.S.A
(2) Dept. of Resp. Med., National Center for Global Health, Tokyo, Japan
(3) Fukujuji Hospital, Tokyo, Japan
(4,5) Brigham & Women's/ Faulkner Hospital, Boston, MA, U.S.A.
sadaishi@massmed.org

We have been following a patient, diagnosed as idiopathic pulmonary fibrosis(IPF) by lung biopsy for 2 years. On the first consultation(01-04,2010), this 68 year old Japanese male engineer, with a history of heavy smoking, was evaluated because of "crackles" heard on routine physical. He claimed he was able to climb 4 flights without coughing.

On auscultation,dry crackles were heard on right base. Peak flow,500L/min, FEV1 2.56, FVC 3.04L. Lung sounds recordings were made with Murphy's STG16, which showed crackles on both bases, but of low intensity. Therefore, close observation was decided.

Six months later(06-29-2010) he returned, claiming shortness of breath on stairclimbing. He had bilateral crackles, 1/3rd up on left, and 1/5th up on right. Lung sounds recordings showed an increase of crackles, intensity and distribution. FEV1 decreased to 2.15, FVC 2.84L.
Transbronchial lung biopsy of right middle lobe showed suggestive lesion of IPF. Steroid therapy was started. Because of further deterioration on steroid therapy, Pirfenidone was prescribed(11-25-2010). He returned to Boston a month later(12-24-2010). Lung sounds recording showed high intensity of crackles on both lower zones.

On Pirfenidone therapy for over a year, he returned to Boston for 4th visit(01-16-2011). At that point he was able to climb stairs without difficulty. On examination, chest was remarkably clear,crackles were heard only at the left base, and right side was clear. Lung sounds recording confirmed presence of inspiratory and expiratory crackles of lower intensity on left, right side was clear. FEV1 improved to 2.84, with FVC 3.08L.

Evolution of crackles coincided improved Vital capacity as well as his physical capacity. It is worth considering beneficial effect of Pirfenidone for IPF, although this is only one case. Lung Sounds recording was a valuable guide for this patient's clinical course.

Characteristics of breathe sounds when asthmatic patients have wheezes or rumbling rhonchi.

Michiko Tsuchiya[1], Yukio Nagasaka[1], Takumi Minami[1], Terufumi Shimoda[2], Katsumi Murakami, [3] Chizu Habukawa[4]

1. Dept. of Pulmonary Medicine, Rakuwakai Otowa Hospital 2. Dept. of Clinical Research, National Fukuoka Medical Center 3. Dept. of Pediatrics, Kinki University Sakai Hospital, 4. Dept. of Pediatrics, National Minami Wakayama Medical Center

Bronchial breath sounds are signs of airway inflammation in stable asthmatic patients (ILSA 2006), and we described breath sounds numerically by their power and frequency (ILSA 2011). In this study, we measured power and frequency of breath sounds when wheezes or rumbling rhonchi were heard.

Sixty eight cases of mild to moderate (Step2 & 3) adult asthmatics were studies when they had no acute symptoms. Patients breathe rather freely while recorded lung sounds.

We measured highest frequency of inspiratory (HFI) and expiratory (HFE) lung sounds and average sound power of low frequency range (LF: 100 to 195 Hz) and compared power ratio of expiratory and inspiratory breath sounds (E/I LF).

We determined the HFI and HFE as the point that intensity of lung sound was higher than -50 dBm, and lasting for more than 0.2 sec.. Wheezes were determined by auscultation and sound spectrogram. Rumbling rhonchi were determined by auscultatory finding.

Wheezes were observed in eight cases and rumbling rhonchi were observed in ten cases. Breath sound of patients who wheezed showed higher HFE, HFI and E/I LF than those of without wheezes. LSI and frequency was not different between patients with or without rumbling rhonchi.

Patients who wheezed had modest generalized airway narrowing beside critically narrow airways that produce wheezes. Presence or absence of rumbling rhonchi did not affect LSI or frequency; implying bronchial hyper-secretion did not co-exist with generalized airway narrowing. We conclude that airway narrowing and bronchial hyper-secretion were independent phenomenon of airway inflammation.

	E/I LF (mean+/- SE)	**HFE**	**HFI**	**HFE/HFI**
Wheezes (-)	0.34 +/- 0.032	293 +/- 12.9	460 +/- 15	0.63 +/- 0.023
Wheezes (+)	0.43 +/- 0.077	404 +/- 41.8	515 +/- 39.8	0.69 +/- 0.043
Rumble (-)	0.35 +/- 0.047	305 +/- 14.7	462 +/- 15.9	0.64 +/- 0.024
Rumble (+)	0.33 +/- 0.089	312 +/- 31.8	485 +/- 30.5	0.62 +/- 0.039

Effect of respiratory flow measurement with a pneumotachograph on tracheal breath sounds*

Davood Karimi and Zahra Moussavi

Abstract— **Respiratory air flow and breath sounds are often measured simultaneously in research studies on human respiratory system. The goal of this study was to investigate the effects of respiratory flow measurement with a pneumotachograph on the spectral features of tracheal breath sounds recorded simultaneously. Tracheal breath sounds were recorded from eight male subjects in two experimental sessions: with and without a pneumotachograph. The recordings were performed for both tidal and deep breathing. Power spectral density (PSD) of the each inspiration and expiration phases of breath sound signals was estimated. Several features were extracted from the estimated PSD and the effect of the use of pneumotachograph on the value of these features was examined using paired t-test. The results show that the use of a pneumotachograph had no significant effect on the spectral features of the breath sounds during deep breathing but has significant effect during tidal breathing, particularly in expiration phase.**

I. Introduction

Breath sounds and respiratory flow are used extensively in studying respiratory system function and disorders [1-3]. Even though respiratory flow can be measured or estimated by indirect methods, for example by measuring chest or abdominal movements [4] or by estimating based on tracheal respiratory sounds [5,6], direct measurement of respiratory flow by means of a pneumotachograph is more reliable [4]. However, there is a concern that the use of a pneumotachograph may affect the subject's breathing pattern. For example, a previous study has shown that the use of a mask and mouthpiece plus a nose-clip or only a mask significantly increases the tidal volume [7].

The goal of this study was to investigate whether the use of a pneumotachograph to measure respiratory flow would affect the spectral characteristics of the breath sounds measured simultaneously over trachea.

II. Methods

A. Data recording

Tracheal breath sounds were recorded from 8 subjects (all male, aged 23 to 34 years) in two experimental sessions:

1. breathing through a mouth piece pneumotachograph,
2. mouth breathing without a pneumotachograph

In each recording, the subject, while sitting upright, breathed at their normal flow rate (tidal) as well as deep breathing. The order of these two trials, i.e. tidal breathing and deep breathing was changed randomly from subject to subject. For each of the two flow rates, the trial consisted of five complete breathing cycles (inspiration and expiration). To have a controlled flow rate, the subjects were instructed for breathing by hand moving up and down with the same speed through the experiment. This ensured that the duration of the breath remained the same throughout the recording, and that during deep breathing not only the volume but also the flow rate increased compared to tidal breathing. The subjects were asked to wear a nose clip during both experiments to ensure that they breathed only through their mouth. The sounds were collected by an ECM77B Sony microphone embedded in a small chamber allowing 2 mm space between the microphone and the skin, placed over the suprasternal notch of trachea with a double sided adhesive tape. The signal from the microphone was band-pass filtered (0.05-5000 Hz), amplified, and recorded on a laptop computer at a sampling rate of 10240 Hz.

B. Analysis

The inspiratory and expiratory phases of each breath sound signal were first separated. This was done manually by plotting the logarithm of variance of the sound signal, which is a function of flow rate (Figure 1).

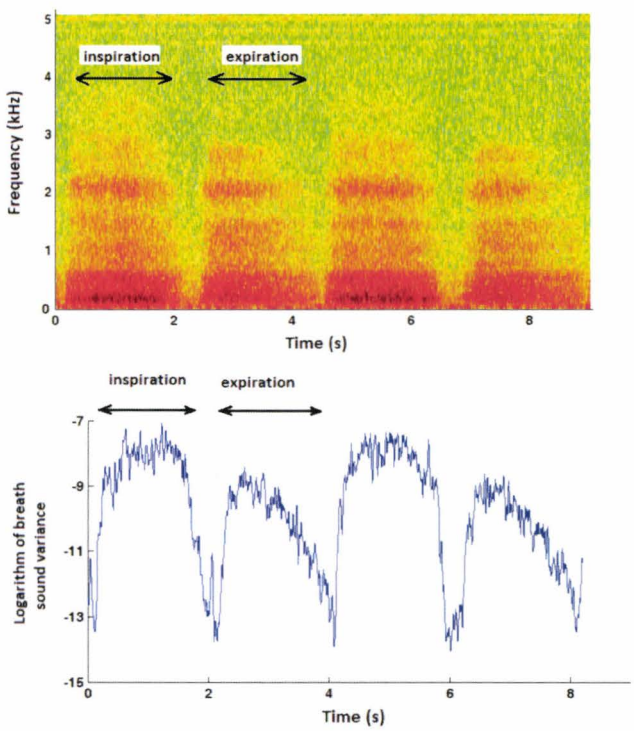


Figure 1. The spectrogram of an example breath sound (top) and the logarithm of sound variance used to separate the phases (bottom).

*Resrach supported by NSERC and TRLabs, Winnipeg, Canada.

Z. Moussavi is with the Electrical and Computer Engineering Department, University of Manitoba, Winnipeg, MB, R3T-5V6 Canada, ph: 204-474-7023; fax: 204-261-4639; e-mail: moussavi@ee.umanitoba.ca.

D. Karimi is with the Electrical and Computer Engineering Department, University of Manitoba, Winnipeg, MB, R3T-5V6 Canada.

To ensure that the sound signal was stationary, only the part of each inspiration or expiration phase for which the flow rate was at least 60% of the maximum flow for that phase was selected. For each respiratory phase, the following steps were performed in sequence to compute the desired features:

1. In order to minimize the effect of plausible differences in respiratory flow between the trials, the signals were normalized by dividing each signal by its standard deviation.

2. The power spectral density (PSD) of the signal was estimated in the frequency range of 150-1200 Hz using the Welch method [8] with a Hamming window of length 80 ms and 50% overlap. The choice of the window length was made by trial and error. Windows of length 80 ms provided a good compromise between the smoothness of the estimated PSD and its frequency resolution.

3. From the estimated PSD, the following features were computed.

- Signal's power over four frequency bands
- Spectral centroid - Eq. 1; it identifies the location of major peaks.

$$\frac{\sum f.P(f)\Delta f}{\sum P(f)\Delta f} \tag{1}$$

In equation (1) and the following equations, $P(f)$ is the estimated PSD and f is the frequency.

- Spectral bandwidth - Eq. 2 ; it gives a measure of the spread of frequency components of the signal around the spectral centroid

$$\frac{\sum (f-SC)^2.P(f)\Delta f}{\sum P(f)\Delta f} \tag{2}$$

- Spectral flatness – Eq. 3; it quantifies how tone-like a signal is, as opposed to being noise-like.

$$\frac{(\prod P(f))^{\frac{1}{f_u-f_l}}}{\frac{1}{f_u-f_l}\sum P(f)\Delta f} \tag{3}$$

- Crest factor - Eq. 4; it is another measure of tonality of the signal

$$\frac{\max(P(f))}{\frac{1}{f_u-f_l}\sum P(f)\Delta f} \tag{4}$$

In this equation, f_u and f_l represent the upper and lower limits of the given frequency band.

Spectral flatness and crest factor were computed only for the entire frequency band of 150-1200 Hz. Signal's power, spectral centroid, and spectral bandwidth were computed for the band of 150-1200 Hz as well as for three sub-bands: 150-450 Hz, 450-600 Hz, and 600-1200 Hz. The choice of sub-bands was primarily based on a visual inspection of the difference between the PSDs. In addition, the relative signal power for each of these three sub-bands was computed by dividing the signal power in that sub-band by the signal power in the band of 150-1200 Hz. As mentioned before, each breath sound signal contained three to five complete breath cycles. After computing the features for each inspiration and expiration phases, they were averaged among different breaths to obtain one feature value for inspiration and one for expiration.

Therefore, a total of 136 features were computed for each subject, 78 from the recording with pneumotachograph and 78 from the recording without pneumotachograph. Paired t-test was used to compare these 78 pairs of features to examine if the use of pneumotachograph had a significant effect on the value of each feature. Outliers, defined as feature values that were more than two standard deviations from the mean value were discarded before performing the t-test. The null hypothesis for the paired t-test was that the value of the feature was equal between the two recordings.

III. Results

Figures 2 to 5 show the estimated PSD for different experimental conditions. The plots shown in these figures are averages of the estimated PSD for all eight subjects.

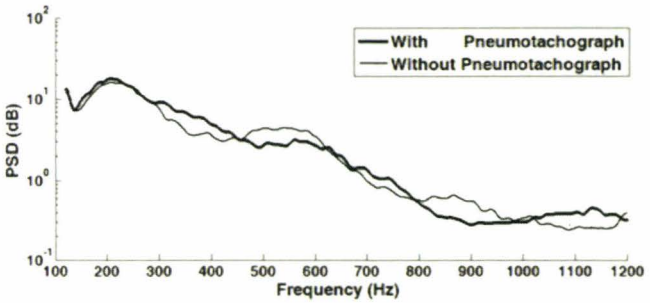


Figure 2. Estimated PSD for inspiration phase of tidal breathing.

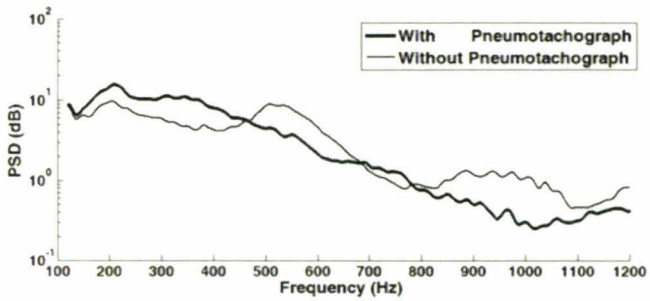


Figure 3. Estimated PSD for expiration phase of tidal breathing.

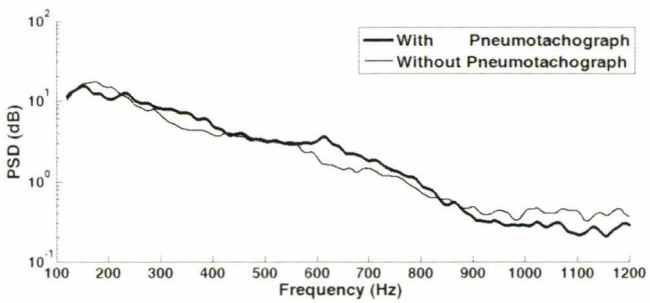


Figure 4. Estimated PSD for inspiration phase of deep breathing.

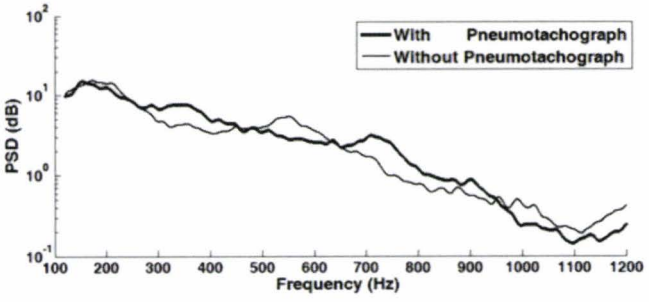


Figure 5. Estimated PSD for expiration phase of deep breathing.

Paired Student's t-test showed that, at 95% confidence level, a total of 11 features were statistically different between the two recordings. Interestingly, all of these features belonged to tidal breathing. Moreover, and equally interesting, 10 of these features belonged to the expiration phase. The following is the list of these 11 features:

From expiration phase of tidal breathing:

1. Spectral bandwidth of 150-450 Hz band
2. Spectral bandwidth of 150-1200 Hz band
3. Spectral centroid of 450-600 Hz band
4. Spectral centroid of 450-1200 Hz band
5. Signal power in 150-450 Hz band
6. Signal power in 150-1200 Hz band
7. Relative signal power in 150-450 Hz band
8. Relative signal power in 450-600 Hz band
9. Relative signal power in 600-1200 Hz band
10. Spectral flatness of 150-1200 Hz band

From inspiration phase of tidal breathing:

11. Spectral centroid in 450-600 Hz band

IV. Discussion

The results of the analysis clearly show that the use of pneumotachograph did not have a significant effect on the PSD of breath sound signals during deep breathing. However, the pneumotachograph did have a significant effect on the PSD of the breath sounds in tidal breathing. These results can also be seen visually in the PSD plots shown in Figures 2 to 5.

It may be possible to justify these observations based on the physics of breathing. Practically, the pneumotachograph acts as an additional resistance in the upper airway. When the respiratory air flow is high, the pressure drop in the upper airway and trachea is much higher and the effect of the pneumotachograph becomes less pronounced in comparison. However, in tidal breathing, the pressure drop across the upper airway and trachea is lower and the effect of the resistance created by the pneumotachograph becomes more significant. Therefore, there is no surprise that the results show a significant effect for the use of a pneumotachograph during tidal breathing but not during deep breathing.

The results also showed that the effect of pneumotachograph was much more pronounced in the expiration phase of tidal breathing. This is an expected result because inspiration is the active phase of breathing, whereas expiration is the passive phase. During inspiration, the diaphragm and intercostal muscles contract and pressure builds up in the pleural cavity, making it an active phase. Expiration, however, is the release of the pressure that has been built up in the pleural cavity and is associated with the relaxation of the diaphragm and intercostal muscles. Therefore, using the same logic as in above paragraph, it is fair to expect that the pneumotachograph will have a more pronounced effect on expiration than on inspiration.

Overall, the results suggest that in studies that require simultaneous measurement of respiratory flow and breathing sounds, measurements should be carried out at deep respiratory flow rate. At tidal respiratory flow, breathing sounds are influenced by the pneumotachograph. This is particularly more significant for the expiration phase. Therefore, if measurements are carried out at tidal breathing, inspiration phases of the breathing sounds are less affected and it is preferred to use them for analysis. It should be noted that the results of this study are based on data from eight subjects, all male, with little variability in age. This may limit generalization of the results of this study to a more diverse population.

References

[1] M. Oud, and E. Maarsingh, "Spirometry and forced oscillometry assisted optimal frequency band determination for the computerized analysis of tracheal lung sounds in asthma," Physiol Meas, vol. 25, no. 3, pp. 595–606, 2004.

[2] S. Rietveld, M. Oud, L. Rijssenbeek-Nouwens, D. Vaghi, and E. Dooijes, "Characteristics and diagnostic significance of spontaneous wheezing in children with asthma: Results of continuous in vivo sound recording," J Asthma, vol. 36, no. 4, pp. 351–358, 1999.

[3] M. Yonemaru, K. Kikuchi, M. Mori, A. Kawai, T. Abe, T. Kawashiro, T. Ishihara, and T.Yokoyama, "Detection of tracheal stenosis by frequency analysis of tracheal sounds," J Appl Physiol, vol. 75, no. 2, pp. 605–612, 1993.

[4] S. Tarrant, R. Ellis, F. Flack, and W. Selley, "Comparative review of techniques for recording respiratory events at rest and during deglutition," Dysphagia, vol. 12, pp. 24–38, 1997.

[5] Yadollahi A. and Moussavi Z., Acoustical Flow Estimation: Review and Validation, *IEEE EMB Magazine*, Vol. 26, No. 1, pp. 56-61, Jan. 2007.

[6] C. Que, C. Kolmaga, L.G. Durand, S. Kelly, and P. Macklem. "Phonospirometry for noninvasive measurement of ventilation: methodology and preliminary results," J Appl Physiol, vol. 9, pp. 1515–1526, 2002.

[7] J. Ashkanazi, P. Silverberg, R. Foster, A. Hyman, J. Milic-Emili, and J. Kinney, "Effects of respiratory apparatus on breathing pattern," J. Appl. Physiol., vol. 48, pp. 577–580, 1980.

[8] J. G. Proakis, and D. K. Manolakis, "Digital Signal Processing," 4th ed., Prentice Hall, 2006.

CARDIAC RESPONCE TO RESPIRATION

Sadamu Ishikawa(1),Andrey Vyshedskiy(2),Raymond L.H Murphy(3) and Armin Ernst(4)

(1,4) Pulmonary & Critical Care, Steward St Elizabeth's Medical Center, Dept. of Medicine, Tufts University School of Medicine, Boston, MA, USA
(2,3) Brigham & Womens'/ Faulkner Hospital, Boston, MA, USA sadaishi@massmed.org

It has been said that the heart beat becomes slower on inspiration. When one takes a deeper breath, more negative pressure within the chest is generated which leads to more blood returning to the left ventricle, hence a delay of the next heart beat. We used a 2 channel ECG/ Lung Sound to simultaneouly record ECG and Tracheal Sounds. ECG('QRS') was used to identify heart beat and lung sound tracings was used to monitor breathing. All recordings were done at sitting position. Two ECG electrodes were mounted on anterior chest at a the level of 3rd intracostal space on both sides of sternum, and the 3rd electrode on the lower part of the left side of the chest. Tracheal sounds were recorded while listning by stethoscope on the neck.

After 2 or 3 regular breathings, the subject was instructed to take a deep breath and hold for few seconds. Recordings of ECG and trachael sounds were made during that period. In order to ensure reproducibility, the same maneuvor and recordings were made 4 times on each subject at one sitting. Measurements of QRS interval of 2 beats during inspiration, and QRS interval of 2 beats just before inspiration were made.

Thirty-three nonsmoking subjects showed larger 2 QRS intervals during inspiration (47 msec.) comparing to shorter, 2 QRS intervals (34 msec.), and the ratio was 1.34. Four subjects had 3 visits of 4 recording sessions. The ratios were averaged at 1.34, 1,43, 1.53,and 1.33.

Six subjects who were known to have COPD, there were no significant of 2 QRS intervals during inspiration (37 msec.) and before the inspiration (36 msec), and ratio was 1.03.

There was a clear evidence of slowing of heart beat during deep inspiration in a healthy non-smoking subject which was not observed in a subject with COPD.

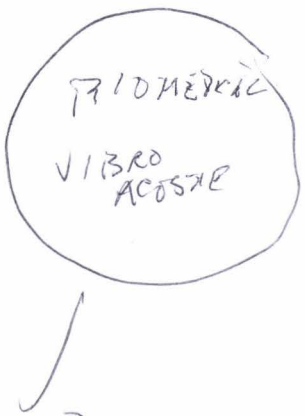
ILSA 2012

Registered participants

Last name	First	Department	Institution	City	Country
Burghess	Deborah	Global Health Technologies	Blill and Melinda Gates Foundation	Seattle, WA	USA
Gavriely	Noam		Karmel Medical	Haifa	Israel
Ishikawa	Sadamu	Pulmonary Medicine	Massacusetts General Hospital	Boston, MA	USA
Mansy	Hansen	Engineering	Rush University	Chicago, IL	USA
Moussavi	Zahra	Biomedical Engineering	University of Manitoba	Winnipeg	Canada
Murphy	Raymond	Stethographics	Stethographics	Boston, MA	USA
Nagasaka	Yukio	Pulmonary Center	Rakuwakai Otowa Hospital	Kyoto	Japan
Nemergut	Michael	Anesthesiology	Mayo Clinic	Rochester, MN	USA
Räsänen	Jukka	Anesthesiology	Mayo Clinic	Rochester, MN	USA
Schears	Gregory	Anesthesiology	Mayo Clinic	Rochester, MN	USA
Smith	Hugh	Anesthesiology	Mayo Clinic	Rochester, MN	USA
Takase	Masato	Pediatrics	Nippon Medical School	Tokyo	Japan
Tsuchiya	Michiko	Pulmonary Center	Rakuwakai Otowa Hospital	Kyoto	Japan
Vitali-Räsänen	Gina	Administration	Mayo Medical School	Rochester, MN	USA

HANSY

VIBRATION & ACOUSTIC



Pulmonary Acoustics

source propagation sensing processing