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Lung Sound Analysis in the Diagnosis of Obstructive Airway Disease

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Key Words

Lung sounds · Vibration energy of lung sounds · Obstructive airway disease

Abstract

Background: Dyspnea is prevalent and has a broad differential diagnosis. Difficulty in determining the correct etiology can delay proper treatment. Non-invasively obtained acoustic signals may offer benefit in identifying patients with dyspnea due to obstructive airway disease (OAD). ***Objectives:*** The aim of this pilot study was to determine whether patients with acute dyspnea due to OAD had distinguishing features when studied with a computerized acoustic-based imaging technique. ***Methods:*** Respiratory sounds from patients with dyspnea due to OAD (n = 32) and those with dyspnea not due to OAD (n = 39) were studied and compared with normal controls (n = 16). ***Results:*** In patients without OAD and in controls, the ratios of peak inspiratory to peak expiratory vibration energy values (peak I/E vibration ratio) were remarkably similar, 6.3 ± 5.1 and 5.6 ± 4, respectively. For the OAD patients, the peak I/E vibration ratio was significantly lower at 1.3 ± 0.04 ($p < 0.01$). In the patients without OAD and the controls, the ratios of inspiratory time to expiratory time (I/E time ratio) were again similar, 1.0 ± 0.1 and 0.99 ± 0.11, respectively. For the OAD patients, the I/E time ratio was significantly lower at 0.72 ± 0.19 ($p < 0.01$). ***Conclusions:*** This modality was useful in identifying patients whose dyspnea was due to OAD. The ability to objectively and non-invasively measure these differences may prove clinically useful in distinguishing the operant physiology in patients presenting with acute dyspnea.

Introduction

Dyspnea is a common symptom and is one of the top reasons why patients visit a hospital emergency department (ED) in the United States. More than 4 million patients present to EDs with dyspnea each year [1, 2]. The most common causes of dyspnea are acute and chronic cardiac and respiratory illnesses. Obstructive airway disease (OAD) is also widely prevalent; in the United States, asthma affects more than 22 million persons, and an estimated 10.2 million (5.9% of the adult population) are reported to have chronic obstructive pulmonary disease [3, 4]. For the patient with acute dyspnea, differentiation between OAD and other pathologies (non-OAD) is essential for proper management. Symptoms of OAD and non-OAD overlap; diagnostic difficulty has been reported, particularly in the pre-hospital setting [5, 6]. The capacity to rapidly triage patients has a beneficial impact upon outcome in the ED [7, 8], and there is no single test which can be used to diagnose and distinguish the cause of dyspnea [2–4]. A rapid non-invasive strategy which would help in diagnosis might be of clinical value.

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Respiratory sounds are generally considered to provide clinically relevant information about patients with and without OAD [9–12]. However, the detection and analysis of lung sounds with a stethoscope has limitations. It is based upon the subjective analysis of lung sounds and is not generally recorded or subjected to rigorous quantitative analysis [13, 14]. Over the years, there have been various attempts to acquire, computerize and refine acoustic data to better detect and monitor pulmonary abnormalities [15]. The application of computer technology has provided new insights into acoustic mechanisms and new measurements of clinical relevance [15, 16]. Breath sounds contain more information about anatomy and physiology than a stethoscope alone can collect and analyze [15–17].

This was a pilot study whose aim was to investigate the feasibility and applicability of a new technology using respiratory sound (vibration) measurements in patients presenting to the ED with acute dyspnea, and to determine how the images and numerical data in patients with OAD differed from data from patients without OAD.

Methods

Study Design and Subjects

The study protocol was approved by the institutional review board, and informed consent was obtained from all patients and healthy volunteers. Consecutive patients, 18–85 years of age, who presented to the ED with acute dyspnea were enrolled. The study group consisted of patients who were admitted due to this chief complaint. Based on discharge diagnosis, patients were then divided into 2 groups: (1) the OAD group, patients whose dyspnea was ultimately determined to have been due to asthma and/or chronic OAD, and (2) patients for whom the cause of dyspnea was ultimately determined to be other than OAD (non-OAD). The designations OAD and non-OAD were made by the attending physician in each case based upon history, physical examination, radiology, laboratory values and physiologic testing. Patients ultimately diagnosed with multifactorial dyspnea including both OAD and other non-OAD conditions were excluded from the analysis. Patients with hemodynamic instability or those who were deemed unable to be seated without assistance were also excluded. Healthy subjects with no known cardiopulmonary disease and normal chest radiographs (as per official report) were enrolled as a control group.

Recording Procedure

Respiratory sound data were acquired on the day of presentation to the ED. All recordings were obtained with the subjects in a seated position. Respiratory sounds were captured using a vibration response imaging device (Deep Breeze™, Or-Akiva, Israel). This is a non-invasive computerized acoustic-based imaging technique that displays the geographic distribution of vibration energy of respiratory sounds throughout the respiratory cycle [16, 17]. With this technique, 36 sensors (2 arrays, 1 array over each lung) are adhered to the patient's back by a computer-controlled low vacuum and used to record respiratory vibrations. Subjects are instructed to take deep, comfortable breaths during 20 s of recording. The recorded signal is amplified and filtered to include frequencies between 150 and 250 Hz in order to minimize cardiac and other non-respiratory vibration frequencies. High-energy artifacts from background noise due to patient movement against matrix framework are occasionally encountered and easily identified in the image. These images were excluded from analysis.

Data Analysis

Recorded lung sounds (measured as vibration energy) can be displayed in different formats. First, the input from the sensors can be displayed as a video made by serial 2-dimensional images (fig. 1). In this format, an image very similar to a ventilation scan is generated, with depiction of the relative spatial distribution of sound intensities. Increased sound intensity is represented as an increase in darkness. Each frame of the video represents 0.17 s worth of data. For each respiratory cycle, there are 2 vibration peaks, 1 during inspiration and 1 during expiration. The inspiratory peak will be referred to as the peak inspiratory vibration (PIV), and the expiratory peak will be referred to as the peak expiratory vibration (PEV) [16]. A graphed linear summation of total vibration energy is generated in addition to the 2-dimensional image (fig. 1, below 2-dimensional images) and was used to choose breath cycles. Numerical data of peak vibration energy during inspiration and exhalation were obtained using proprietary software. The ratios of PIV/PEV (peak I/E vibration ratio) and the ratios of inspiration time to exhalation time (I/E time ratio) during each respiratory cycle were calculated and then averaged for each patient.

Three groups were analyzed and compared: OAD, non-OAD and normal controls. Three methods of analysis were used. The first was visual description of the videos. The second was analysis of the linear graphic material to determine inspiratory and respiratory time. The third was the analysis of numerical vibration energy data, PIV and PEV.

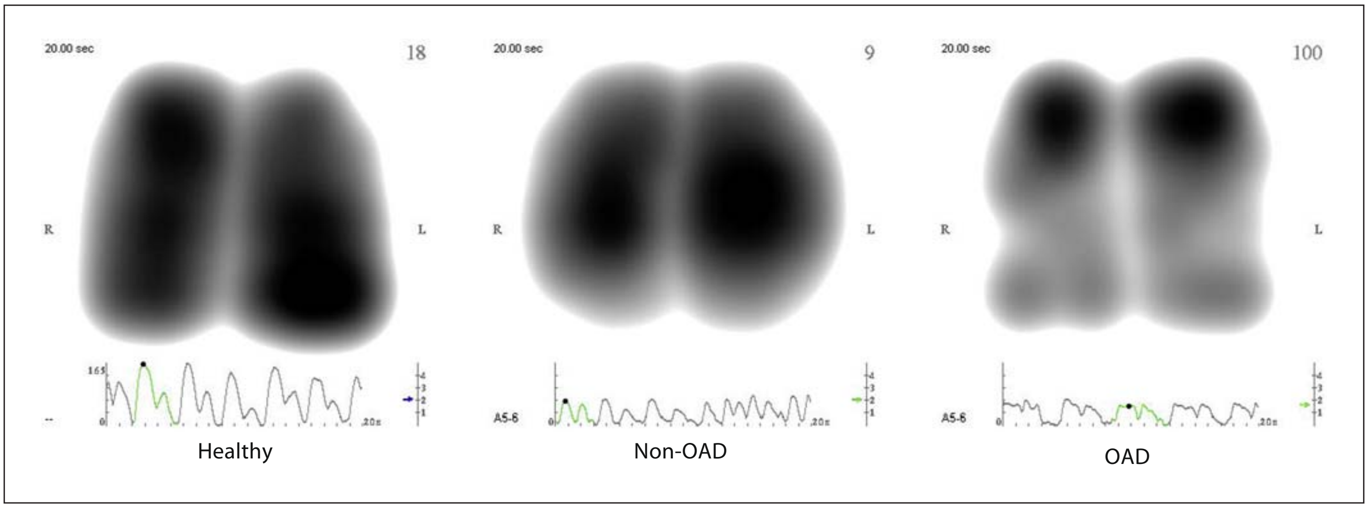
Statistical Analysis

Wilcoxon signed rank tests for paired and unpaired data (SPSS 11.5, SPSS Inc., Chicago, Ill., USA) were used to analyze the data. The mean ± standard deviation is reported. p values <0.05 were considered statistically significant.

Results

Patients

A total of 71 patients and 16 healthy subjects were enrolled in the study. Of the patients, 32 had OAD and 39 were classified as non-OAD. Eight patients were excluded from analysis due to diagnoses of both OAD and non-OAD etiologies of dyspnea. Auscultation findings at the time of the recordings and the treating physician's final diagnoses are listed (table 1).



Color version available online

Fig. 1. Representative vibration energy images. **a** Healthy volunteer. The images for the normal patients showed that right and left lungs viewed together have peripheral smooth, rounded and uninterrupted contours. Planar distribution, area, size and intensity of the right and left lung images are similar and encompassed the entire imaging field. **b** Non-OAD patient (a representative patient with congestive heart failure). The image is also smooth and rounded, but for this patient, the image is smaller in size, likely due to pulmonary edema, particularly at the bases. **c** OAD patient. In this patient, with chronic obstructive pulmonary disease, different areas of each lung demonstrated peaks in sound energy at different times. Because of this asynchrony, the contours of the lung periphery on the 2-dimensional images are not smooth, but have a 'bumpy-lumpy' appearance.

Dynamic and Static Image Features

Peak inspiratory images for each of the study groups are presented in figure 1 (online supplement video 1, www.karger.com/doi/10.1159/000178023). The static and dynamic images had differences as described below.

In all subjects, the dynamic images 'build and fade' twice for every respiratory cycle, once for inspiration and again for exhalation. In the healthy control subjects, the maximal vibration intensity in inspiration is visually greater than that in exhalation. Sound patterns develop synchronously in both lungs. Right and left lungs viewed together have peripheral smooth, rounded and uninterrupted contours. Planar distribution, area, size and intensity of the right and left lung images are similar. The 2-dimensional sound images for the non-OAD patients were visually similar to those of the healthy controls.

In the subjects with OAD, the dynamic images showed asynchrony within each lung and between lungs; different areas of each lung demonstrated peaks in sound energy at different times. Because of this asynchrony, the contours of the lung periphery on the 2-dimensional images are not smooth, but have a 'bumpy-lumpy' appearance ('disco' lung).

Table 1. Subject characteristics

Characteristics	Healthy (n = 16)	Non-OAD (n = 39)	OAD (n = 32)
Demographic information			
Age, years	49 ± 11	47 ± 15	49 ± 19
Gender			
Male	15	21	16
Female	1	18	16
Auscultation results			
Crackles	0	29	7
Wheezes	0	5	21
Final diagnosis			
CHF	0	22	0
COPD	0	0	12
Asthma	0	0	20
Pneumonia	0	7	0
Pleural effusion	0	2	0
Upper tract infection	0	4	0
Pulmonary embolism	0	1	0
Esophageal reflux	0	1	0
Pneumothorax	0	1	0
Bronchitis	0	1	0

Values are the mean ± standard deviation, or the number of patients.

CHF = Congestive heart failure; COPD = chronic obstructive airway disease.

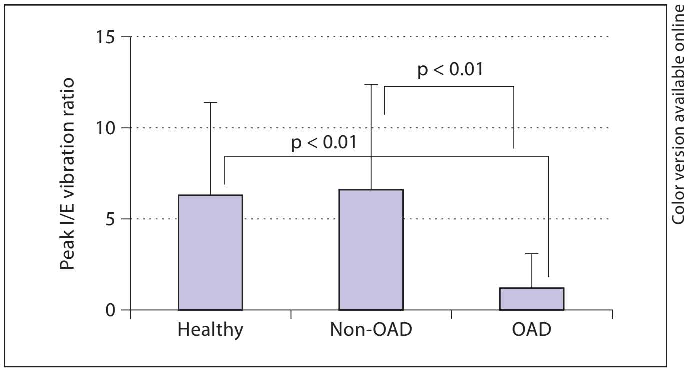


Fig. 2. I/E peak vibration ratio in OAD and non-OAD.

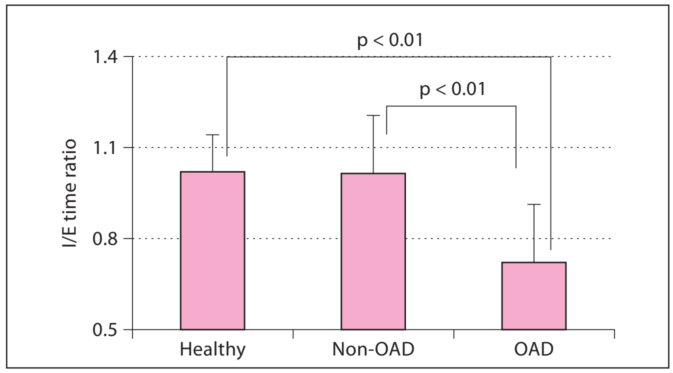


Fig. 3. I/E time ratio in non-OAD and OAD exacerbation.

Analysis of Numerical Vibration Data

The PIV and PEV values were calculated for each group and expressed as peak I/E vibration ratio for each patient. In healthy volunteers and non-OAD patients, the peak I/E vibration ratios were similar, 6.3 ± 5.1 and 6.7 ± 5.8, respectively ($p > 0.05$). In marked contrast, the peak I/E vibration ratio was 1.2 ± 1.4 in patients with OAD. This differed significantly from the other 2 groups ($p < 0.01$) (fig. 2).

The I/E time ratio during each respiratory cycle was calculated. In healthy volunteers and non-OAD patients, the I/E time ratios were similar, 1.0 ± 0.1 and 1.0 ± 0.2, respectively ($p > 0.05$). In marked contrast, the I/E time ratio was 0.7 ± 0.2 in OAD patients. This differed significantly from both of the other groups ($p < 0.01$) (fig. 3).

Discussion

This pilot study of patients presenting to the ED with acute dyspnea demonstrates the potential capacity to use this technology to differentiate patients with OAD from those without OAD at the time of presentation to the ED.

Three main findings differentiate OAD from other conditions. The first is the heterogeneity of sound distribution in OAD, which is visually evident on both the dynamic and the static images (online suppl. video 1, www.karger.com/doi/10.1159/000178023). The second is the distribution of vibration energy between inspiration and exhalation, with a significantly greater vibration energy during exhalation in the patients with OAD. The third is the presence of a significantly longer exhalation phase in OAD, leading to a change in the I/E time ratio.

Each of the above differences has a clear physiologic basis and could be evaluated with different modalities. The first, inhomogeneity of air flow, has been studied by documenting the anatomic results of the inhomogeneity [18–21]. CT and PET scanning have been used to demonstrate inhomogeneity, with a mosaic pattern considered to represent differences in regional ventilation due to regional differences in degree of air flow obstruction [18–20]. One group of authors described the use of multiple sequential chest roentgenograms which are then analyzed mathematically, with changes in pixel density representing changes in ventilation [21]. The dynamic vibration energy images obtained from this study demonstrate the same physiologic processes through an analysis of sound vibration energy, a direct correlate of the ventilation itself, instead of through an analysis of the anatomic results of variations in ventilation. The second finding, a greater sound intensity during exhalation in OAD is not a surprising finding; its auscultatory correlate is the wheezing often present in asthma or chronic obstructive pulmonary disease [22]. The greater sound intensity during expiration in OAD patients may also be due to greater turbulence and vibrating secretions. The third finding, the change in inspiratory and expiratory phases, reflects the prolongation of exhalation due to air flow obstruction.

The elegance of this breath sound analysis technique lies in its simplicity, portability and repeatability. There is, in addition, no radiation exposure. The capacity to capture sounds simultaneously throughout both lungs and to record them for computer analysis goes far beyond the capacities of a stethoscope. The instruments are more portable than most pulmonary function machines, and the efforts required for data collection are much less demanding than those for pulmonary function testing.

This acoustic-based sound analysis technique is a relatively unique imaging modality. Most imaging modalities document anatomy, whereas vibration imaging documents the geometric distribution of physiologic measurements. In this study of patients presenting to the ED with acute dyspnea, we have shown that the use of this imaging technique can help to identify those patients who have OAD and in so doing separate them both from other patients and from healthy controls. Although these results are not surprising from knowledge of pathophysiology, the ability to have a noninvasive and objective assessment of these differences may prove to be clinically useful in determining underlying physiology in patients presenting to the ED with acute dyspnea. Further studies targeting the effect of the use of this sound analysis technique information on management decisions are needed.

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References

1 McCaig LF, Nawar EW: Division of Health Care Statistics National Hospital Ambulatory Medical Care Survey: 2004 emergency department summary. Adv Data 2006;372:1–29.

2 McCord J, Nowak RM, McCullough PA, Foreback C, Borzak S, Tokarski G, Tomlanovich MC, Jacobsen G, Douglas W: Ninety-minute exclusion of acute myocardial infarction by use of quantitative point-of-care testing of myoglobin and troponin I. Circulation 2001;104:1483–1488.

3 National Heart, Lung and Blood Institute, National Asthma Education and Prevention Program. Expert panel report 3: guidelines for the diagnosis and management of asthma. Full report 2007. http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm.

4 Mannino DM: COPD: epidemiology, prevalence, morbidity and mortality, and disease heterogeneity. Chest 2002;121:121S–126S.

5 Hoffman JR, Reynolds S: Comparison of nitroglycerin, morphine and furosemide in the treatment of presumed pre-hospital pulmonary edema. Chest 1987;92:586–593.

6 Wuerz RC, Meador SA: Effects of prehospital medications on mortality and length of stay in congestive heart failure. Ann Emerg Med 1992;21:669–674.

7 Trzeciak S, Rivers EP: Emergency department overcrowding in the United States: an emerging threat to patient safety and public health. Emerg Med J 2003;20:402–405.

8 Chalfin DB, Trzeciak S, Likourezos A, Baumann BM, Dellinger RP, for the DELAY-ED study group: Impact of delayed transfer of critically ill patients from the emergency department to the intensive care unit. Crit Care Med 2007;35:1477–1483.

9 Congestive heart failure and pulmonary edema. www.emedicine.com/EMERG/topic108.

10 Braunwald E, Zipes DP, Libby P: Heart Disease: A Textbook of Cardiovascular Medicine, ed 6. Philadelphia, Saunders, 2000.

11 Loudon HG, Murphy RLH: Lung sounds; in Crystal RG, West JB, Barnes PJ, Cherniack NS, Weibel ER (eds): The Lung. New York, Raven Press, 1991, pp 1011–1019.

12 Schreur HJ, Vanderschoot J, Zwinderman AH, Dijkman JH, Sterk PJ: Abnormal lung sounds in patients with asthma during episodes with normal lung function. Chest 1994;106:91–99.

13 Loudon RG: The lung exam. Clin Chest Med 1987;8:265–272.

14 Ertel PY, Lawrence M, Brown RK, Stern AM: Stethoscope acoustics. 1. The doctor and his stethoscope. Circulation 1966;34:889–898.

15 Pasterkamp H, Kraman S, Wodicka GR: Respiratory sounds. Advances beyond the stethoscope. Am J Respir Crit Care Med 1997;156:974–987.

16 Dellinger RP, Jean S, Cinel I, Tay C, Rajanala S, Glickman YA, Parrillo JE: Regional distribution of acoustic-based lung vibration as a function of mechanical ventilation mode. Crit Care 2007;11:R26.

17 Dellinger RP, Parrillo JE, Kushnir A, Rossi M, Kushnir I: Dynamic visualization of lung sounds with a vibration response device: a case series. Respiration 2008;75:60–72.

18 Sharma V, Shaaban AM, Berges G, Gosselin M: The radiological spectrum of small-airway diseases. Semin Ultrasound CT MR 2002;23:339–351.

19 Sung A, Naidich D, Belinskaya I, Raoof S: The role of chest radiography and computed tomography in the diagnosis and management of asthma. Curr Opin Pulm Med 2007;13:31–36.

20 Tgavalekos NT, Tawhai M, Harris RS, Musch G, Vidal-Melo M, Venegas JG, Lutchen KR: Identifying airways responsible for heterogeneous ventilation and mechanical dysfunction in asthma: an image functional modeling approach. J Appl Physiol 2005;99:2388–2397.

21 Tanaka R, Sanada S, Okazaki N, Kobayashi T, Fujimura M, Yasui M, Matsui T, Nakayama K, Nanbu Y, Matsui O: Evaluation of pulmonary function using breathing chest radiography with a dynamic flat panel detector: primary results in pulmonary diseases. Invest Radiol 2006;41:735–745.

22 Chronic obstructive pulmonary disease: Management of chronic obstructive pulmonary disease in adults in primary and secondary care. 2004. www.nice.org.uk/CG012NICEguideline.