Pneumothorax effects on pulmonary acoustic transmission

Hansen A. Mansy,1 Robert A. Balk,2 William H. Warren,3 Thomas J. Royston,4 Zoujun Dai,5 Ying Peng,5 and Richard H. Sandler1

1Department of Mechanical and Aerospace Engineering and Department of Pediatrics, University of Central Florida, Orlando, Florida; 2Division of Pulmonary and Critical Care Medicine, Rush Medical College, Chicago, Illinois; 3Department of Cardiovascular-Thoracic Surgery, Rush Medical College, Chicago, Illinois; 4Department of Bioengineering, University of Illinois at Chicago, Chicago, Illinois; and 5Department of Mechanical Engineering, University of Illinois at Chicago, Chicago, Illinois

Submitted 23 February 2015; accepted in final form 26 May 2015

Mansy HA, Balk RA, Warren WH, Royston TJ, Dai Z, Peng Y, Sandler RH. Pneumothorax effects on pulmonary acoustic transmission. J Appl Physiol 119: 250–257, 2015. First published May 28, 2015; doi:10.1152/japplphysiol.00148.2015.—Pneumothorax (PTX) is an abnormal accumulation of air between the lung and the chest wall. It is a relatively common and potentially life-threatening condition encountered in patients who are critically ill or have experienced trauma. Auscultatory signs of PTX include decreased breath sounds during the physical examination. The objective of this exploratory study was to investigate the changes in sound transmission in the thorax due to PTX in humans. Nineteen human subjects who underwent video-assisted thoracic surgery, during which lung collapse is a normal part of the surgery, participated in the study. After subjects were intubated and mechanically ventilated, sounds were introduced into their airways via an endotracheal tube. Sounds were then measured over the chest surface before and after lung collapse. PTX caused small changes in acoustic transmission for frequencies below 400 Hz. A larger decrease in sound transmission was observed from 400 to 600 Hz, possibly due to the stronger acoustic transmission blocking of the pleural air. At frequencies above 1 kHz, the sound waves became weaker and so did their changes with PTX. The study elucidated some of the possible mechanisms of sound propagation changes with PTX. Sound transmission measurement was able to distinguish between baseline and PTX states in this small patient population. Future studies are needed to evaluate this technique in a wider population.

PTX is also a recognized complication during positive pressure ventilatory support (6, 10, 17, 42). Delayed PTX diagnosis and treatment, especially for patients with mechanical ventilation, may lead to PTX progression and hemodynamic instability (5, 12). PTX diagnosis involves evaluating a combination of medical history, physical examination, and chest imaging (2, 7, 13, 15, 41). History and physical examinations generally lack sensitivity and specificity (21, 27, 28).

Standard portable end expiratory upright chest X-rays are not always sufficiently sensitive to diagnose PTX (30, 37). It was also reported that 30% of 112 pneumothoraces in 88 patients who were critically ill were not detectable by routine chest radiographs (41). Another study found 12 patients with unsuspected or untreated tension pneumothoraces (10 of whom were on positive pressure ventilatory support) among 3,500 autopsies (26). In addition, chest X-rays are not always readily available and require patient positioning and timing of respiration to enhance pneumothorax detection. Consequently, chest computed tomography (CT) scans have become the gold standard for PTX detection (34). The superiority of a chest CT is, however, offset by expense, increased radiation exposure, lack of immediate availability (especially in situations of forward trauma, combat, ambulance, and other emergency or lower technology settings), and need for patient transport to the CT scanner (16, 43).

The use of thoracic ultrasonography for PTX detection has been gaining popularity and some now consider the technique to be a new gold standard with the advantages of being quick and relatively easy to perform (20). During this test, the absence of signs of visceral pleural movement (e.g., lung sliding and lung point signs) are suggestive of the presence of PTX (25). However, the absence of these signs can be observed in patients with lung fibrosis or pleural adhesion without PTX (38).

Misdagnosis or delayed diagnosis of pneumothoraces in critically ill patients may have severe consequences (2). Several studies suggested that additional tools for PTX detection and monitoring would be helpful (14, 25, 27, 28, 31, 39, 40, 44, 48).

Sound transmission in the respiratory system has been studied by many investigators (23, 24, 27, 33, 46, 47). Although some studies have suggested that properties of the thoracic structures can significantly affect transmission (47), other studies in healthy volunteers (24) reported no changes in peak amplitude or frequency with changes in lung volume or resident gas. More recent investigations (9, 18, 22, 27, 28, 33, 36, 46) have demonstrated that certain respiratory system changes have acoustic correlates. This suggests that only certain (and

Address for reprint requests and other correspondence: H. A. Many, 12760 Pegasus Drive, Rm 308, Dept. of Mechanical and Aerospace Engineering, Univ. of Central Florida, Orlando, FL 32816 (e-mail: hansen.mansy@ucf.edu).

8750-7587/15 Copyright © 2015 the American Physiological Society http://www.jappl.org

PNEUMOTHORAX IS AN ABNORMAL ACCUMULATION OF AIR IN THE PLEURAL SPACE THAT CAN RESULT IN LUNG COLLAPSE (3, 4, 17). PNEUMOTHORAX (PTX) IS A RELATIVELY COMMON DISORDER THAT IS POTENTIALLY LIFE-THREATENING, YET TREATABLE WITH TIMELY DIAGNOSIS. MORE THAN 20,000 CASES OF PTX ARE REPORTED EACH YEAR IN THE UNITED STATES, WITH AN ESTIMATED COST OF >$130,000,000 (4). PTX CAN DEVELOP SPONTANEOUSLY OR IT CAN RESULT FROM TRAUMA OR INVASIVE PROCEDURES (34). SPONTANEOUS PNEUMOTHORACES ARE EITHER PRIMARY (I.E., THOSE OCCURRING WITHOUT OBVIOUS CAUSE OR UNDERLYING LUNG DISEASE) OR SECONDARY, WHICH DEVELOP IN PATIENTS WITH UNDERLYING LUNG DISEASE (4, 34). PRIMARY SPONTANEOUS PTX INCIDENCE IS ESTIMATED TO BE 7.4/100,000 IN MEN AND 1.2/100,000 IN WOMEN (29), Whereas SECONDARY SPONTANEOUS PTX INCIDENCE IS ABOUT 6.5/100,000 IN MEN AND 2.0/100,000 IN WOMEN (27). TRAUMATIC PTX IS A COMMON ENCOUNTER WITH CHEST INJURY AND OCCURS IN ABOUT 20% AND 40% OF PATIENTS WITH BLUNT AND PENETRATING TRAUMA, RESPECTIVELY (11).
not all) acoustic variables may correlate with respiratory conditions and, hence, a high degree of care in data collection and analysis need to be taken to identify the useful acoustic correlates of individual pulmonary conditions.

In the current study we hypothesize that the presence of air in the pleural space causes measurable changes in the acoustic properties of the chest structures. Because the acoustic properties of air are significantly different from those of the chest wall tissue and the lungs, acoustic impedance mismatch will occur at the boundaries of the pleural air pocket. This mismatch will impose acoustic barriers to sound waves and, consequently, cause acoustic transmission changes. This scenario was found to cause sound transmission reduction with PTX in earlier animal studies (25, 31). Acoustic changes, if proven to correlate with the presence of PTX, may serve as an adjuvant patient-monitoring tool with potential operational advantages that include being radiation-free, low-cost, providing rapid test results, and having potential utility for continuous patient monitoring. Although this potential may prove useful in the future, the authors stress that the objective of this study is to document possible acoustic correlates of PTX and not to test a clinical tool, which would be a possible objective of future investigations.

**MATERIALS AND METHODS**

This study was approved by the Rush University Medical Center Institutional Review Board. Patients were enrolled in the study after providing consent. Subjects were recruited from those who were to undergo video-assisted thoracic surgery (VATS) as part of their normal care, during which a PTX is induced as a usual part of the procedure. Patients were sedated and intubated in the usual manner with a double-lumen endotracheal (ET) tube. Patients rested on their nonoperative side with the operative side up, and were ventilated at the normal rate and tidal volume as deemed appropriate by the attending anesthesiologist.

The experimental setup is shown in Figure 1. A sound source was connected to the ventilator tubing via a T-connector, which was attached to the circuit a few centimeters from the proximal separation of the channels of the double-lumen endotracheal tube. The T-connector added a negligible volume (~3 ml) of dead air space to the ventilator circuit. The sound source was made of an electromagnetic speaker (Harman Kardon, Mill Valley, CA) that was installed in a sealed polyvinyl chloride plastic chamber that was isolated from the ventilator circuit via a viral and bacterial filter (Airlife; Allegiance Healthcare, McGaw Park, IL). This system was made air-tight to ensure negligible airflow in the tubing that connects to the sound source. The excitation signal amplitude did not significantly contribute to room noise because it was minimally noticeable in the operating room. Similar excitation levels were used in previous studies (1, 8, 27). Breathing was not interrupted during sound recording.

An electronic stethoscope (model 04-1060; Labtron Electromax, Hauppauge, NY) was used to record sounds at the chest surface at the intersection of the midaxillary and nipple lines. This location was approximately 5–10 cm from the incision site, where the thoracoscope was to be inserted. The stethoscope was enclosed in a sterile bag before placement over the chest surface and was held in place by the attending surgeon or fellow. To measure transpulmonary acoustic transmission, sounds sensed by the stethoscope were recorded for 20 s using a digital recorder (model D888; Korg, Japan) while band-limited white noise (50-2,000 Hz) was introduced into the ET tube. Sound recording was performed before PTX was induced. Surgery was then started, and a PTX was created in the usual manner. After the presence of a PTX was confirmed using the thoracoscope, the scope was removed and the incision was covered by Tagaderm to avoid varying the PTX air volume during the breathing cycle. The stethoscope was then placed in the same position and sounds were again recorded for 20 s while the sound source introduced the band-limited white noise into the ET tube. During each sound recording, the electronic stethoscope output was monitored using the digital recorder. The amplifier for the input signal was kept at the same setting throughout each experiment. The recorder saved the acoustic data in the “wav” file format for post processing. Saved files were transmitted to a computer via the recorder USB port. Sound files were postprocessed using a digital signal-processing software package (Matlab; Mathworks, Natick, MA). The sound files contained 20 s of data sampled at 8,192 Hz, resulting in about 164,000 sample for each data file. The data processing protocol included band-pass filtering (50-1,600 Hz pass band) and spectral analysis using Fast Fourier Transform followed by smoothing the spectral curves by a moving average filter.

To calculate the power spectral density of the measured signal \( x(j), j = 1, 2, 3, \ldots, N \) that contains \( N \) points, we first calculate the discrete Fourier transform from:

\[
X(k) = \sum_{j=1}^{N} x(j) e^{-2\pi j (k-1) / N}, \quad k = 1, 2, 3, \ldots, N/2 + 1
\]

where \( X(k) \) is the complex Fourier Transform and \( k \) is the index of frequency bins.

The power spectral density, \( P_{xx} \), is then estimated from:

\[
P_{xx}(k) = 10 \log_{10} \left( \frac{2}{N F_s} \ast \left( |X(k)| \right)^2 \right); \quad k = 2, 3, 4, \ldots, N/2
\]

and

\[
P_{xx}(k) = 10 \log_{10} \left( \frac{1}{N F_s} \ast \left( |X(k)| \right)^2 \right); \quad k = 1, N/2 + 1
\]

where these \( k \) values correspond to the zero and Nyquist frequencies, respectively, and \( F_s \) is the sampling frequency.

Spectral smoothing was performed through a moving average approach, which was implemented using a finite impulse-response
filter. The filter output was calculated from the discrete convolution formula:

\[ y(n) = \sum_{i=0}^{M} b_i x(n - i) \]

where \( y \) and \( x \) are the filtered and unfiltered spectra, \( b_i \) is the filter weight (i.e., impulse response at the \( i \)th instant), and \( M \) is the filter order. In the current study, \( M \) was chosen as 5, and \( b_i = 1/M \). This filtering was performed in the upward (i.e., from low to high frequency) and then in the downward (high to low frequency) directions to avoid shifting the spectral peaks.

To quantify the effect of PTX on acoustic transmission at different frequencies, the difference in the power spectral density (PSD) between the control and PTX states was calculated. The signal energy in certain frequency bands was also calculated. This was performed by adding the PSD values in the frequency bands under consideration. Energy ratios between the different bands were calculated by dividing the energy values corresponding to the bands of interest. The spectral trends were compared using the Wilcoxon rank sum test, with a value of \( P < 0.05 \) indicating significance.

RESULTS

During the course of the study, no significant additional interventions were performed for the sole purposes of the experiment. The main extra needed steps were to connect the sound source, input sounds, and record transmitted sounds at the chest wall using an electronic stethoscope for 20 s before and after lung collapse. All patients exhibited pulmonary pathology. Out of the participating patients, 15 had lung masses and 4 had lung nodules, 1 of whom also had breast cancer. The operative side was the left side in 10 patients and the right in 9. This information was taken into consideration by the attending anesthesiologist when choosing the appropriate double-lumen ET tube type (i.e., left- vs. right-sided). Noticeable lung collapse was observed. The air space between the lung surface and chest wall around the sensor location was visually estimated to be >2 cm [which is considered a large PTX (19)] in 18 patients and appeared to be <1 cm in one patient (possibly due to pleural adhesions in the latter case).

An example of the raw and smoothed power spectral densities of the transmitted sounds is shown in Figure 1B. The spectral plot shows that smoothing did not significantly change the main spectral features. Data for other patients demonstrated similar trends.

The spectra of the control and PTX states for all subjects are shown in Figure 2A, and the mean spectra across subjects are shown in Fig. 2B, with error bars showing the 95% confidence interval. Intersubject variability is evident in Fig. 2A, but there are common trends. The first trend is a general decrease in transmitted sounds as the frequency increases for both the control and PTX states. Second, the occurrence of PTX seemed to be associated with a drop in sound amplitude, which was more noticeable in the mid-frequency (400–900 Hz) range \( (P < 0.01, \text{Wilcoxon signed rank test}) \).

Figure 3A shows the drop in acoustic spectra when lung collapse took place relative to the control state. This was calculated as the difference between the solid and dashed lines in Fig. 2A. The spectral energy for the PTX state was lower than that of the control state in most of the frequency ranges of interest, but it was higher than the control state for relatively narrow frequency values that varied among patients. The mean spectral drop with PTX is shown in Fig. 3B with error bars marking the 95% confidence interval. This figure suggests that, in the average, the spectral energy drop with PTX is more pronounced in the 400–600 Hz frequency range. The maximum and mean PSD drop in the 400–600 Hz frequency band is shown in Fig. 4. A and B, respectively, and the maximum spectral increase with PTX is shown in Fig. 4C. In this frequency range, no spectral increase with PTX was observed, suggesting that energy increases with PTX took place only outside that frequency band. Some amplitude increase with PTX was observed in computer simulations of a similar phenomenon and may be due to resonances (33) that would occur at different frequencies for different subjects as was observed in the current study.

Figure 3, A and B, shows that the PSD drop with PTX tended to be more pronounced in a certain frequency range (e.g., around 400–600 Hz) and was smaller at lower and higher frequencies. A ratio between the acoustic energy of the mid-frequency (e.g., 400–600 Hz) and low-frequency (50–250 Hz) bands was proposed for animal subjects (27) and was calculated in the current study as \( ER = \frac{\text{energy in mid-frequency band}}{\text{energy in the low-frequency band}} \). In addition, a second energy ratio between the mid- and high-frequency (1,300–1,500 Hz) bands was also calculated as \( ER = \frac{\text{energy in mid-frequency band}}{\text{energy in the high-frequency band}} \). These energy ratios are shown in Fig. 5A, which demonstrates a trend toward a reduction in energy ratios with PTX \((P < 0.01, \text{Wilcoxon signed rank test})\). The change in energy ratios with PTX is shown in Fig. 5B, which demonstrates that a drop in energy ratios in PTX states occurs relative to controls. The case with the least drop in energy ratio corresponds to the patient with the smaller air gap between the lung and chest wall.

DISCUSSION

The current study investigated the effects of PTX on acoustic transmission through the chest in patients with pulmonary conditions such as lung nodules and masses. To perform these measurements, controlled acoustic signals with low amplitude were introduced via an ET tube into the airways of subjects. The excitation amplitude was minimally noticeable in the operating room and did not interfere with clinical activities. Similar sound transmission measurements were used in previous studies to monitor lung conditions in which broad-band sounds are usually introduced via ET tubes. It is to be noted that the acoustic transmission properties of these tubes can affect the actual signal input to airways. A careful study (35) of sound transmission in ET tubes documented the existence of spectral peaks and valleys (at the ET tube tip) that are dependent on ET tube length and diameter changes.

In the current study the spectra of the sound delivered into the airways were calculated from the acoustic signal measured over the neck of one patient. The results showed a spectral uniformity of ±3 dB in the 100–1,600 Hz range. This spectral variability is relatively low, possibly because of the small diameter changes (<25%) in our sound-delivery ducts including the ET tube. This variability value is also comparable to the theoretical estimates at the tip of the endotracheal tube using existing theoretical estimates (35) for cases involving small diameter changes. This relatively small variability likely existed in similar ways in both the control and PTX states and may have only a minor influence on the observed PTX effect.

\[ \text{J Appl Physiol \ doi:10.1152/japplphysiol.00148.2015 \ www.jappl.org} \]
on acoustic transmission. To assess the effect of this variability more quantitatively, future studies may be performed to further document the spectral content of the sound inputted from the ET tube in both the control and PTX states.

There was no need to pause breathing during the experiment. Because sound signals were acquired without breath hold, they contained low-amplitude breath sounds. Typical breath sound amplitudes in the current study were at least 10 dB lower than the measured signals and, hence, likely had a small effect on these signals. The study subjects were those who underwent VATS, during which a lung collapse was created as a routine part of the procedure. Sound waves that reached the chest wall were measured before and after lung collapse, which was confirmed by thoracoscopy. The described sound introduction approach may be termed “active forcing” because it involves actively inputting external signals. Earlier studies have elucidated some of the useful acoustic parameters that correlated with respiratory system conditions (9, 18, 22, 27, 28, 33, 36).

The primary hypothesis in the current study is that property changes in the tissue structures along the sound transmission path cause measureable changes in the sounds measured at the chest surface. In turn, these acoustic changes may be used as an adjuvant to the methods of detecting or following the progression of certain pulmonary conditions.

Sound transmission in the respiratory system involves a set of relatively complex frequency-dependent processes including sound transmission through the airway tree; coupling to surrounding tissue; and transmission through 1) the parenchyma, 2) the PTX air pocket (if it exists), and 3) the chest wall. Earlier studies suggested that most of the low-frequency sounds (below about 600 Hz) efficiently couple from the large airways to the surrounding tissue (24). As the frequency

---

Fig. 2. A: spectra of sounds transmitted from the mouth to chest wall of all study subjects for the control (solid line) and pneumothorax (PTX) (dashed line) states. A spectral drop in acoustic transmission due to PTX can be seen at certain frequencies. B: average spectra of transmitted sounds in the control (solid line) and PTX (dashed line) states. Error bars show the 95% confidence interval.
increases, the airways become more rigid due to their mass, and the acoustic energy travels deeper into the smaller airways before coupling to the parenchyma (24). Other relevant acoustic phenomena include increased acoustic attenuation in tissue with increasing frequency, ET tube acoustics, possible resonances of thoracic structures, and reflections of sound waves at interfaces between different tissues and between tissue and PTX air (27). All these factors contribute to increasing the transmission complexity. Although each phenomenon involved can be studied separately, the current study focuses on investigating the acoustic effect of one main structural change; namely, the abnormal existence of PTX air between the lung and chest wall.

Fig. 3. A: spectral drop in acoustic transmission with PTX for all study participants. There was a relatively consistent drop in acoustic energy ($P < 0.01$, Wilcoxon signed rank sum test) for frequencies $>300$ Hz. There appears to be a smaller change in amplitude in the 0–300 Hz range. B: average drop in transmitted spectra with PTX for all study participants. Error bars show the 95% confidence interval. It appears that the drop in spectral energy with PTX is most pronounced in the 400–600 Hz range. The drop in energy was smaller outside this frequency range.

The measured spectra in the current study showed a general trend toward decreased amplitude with frequency, which is consistent with data from previous studies (24, 27, 46). The spectral distributions, however, varied among subjects in the control state as well as the PTX state. This may be due at least in part to differences among subjects in size and pathology. For example, the body mass index of subjects varied from 25 to 40. In addition, 15 subjects had lung masses and 4 had lung nodules, 1 of whom had breast cancer. In an earlier study that reported intersubject variability, this variability was suggested as evidence for the dependence of acoustic transmission on pulmonary structures (47). But variability can also make it more challenging to identify pulmonary conditions without baseline measurements. It is to be noted, however, that if certain acoustic signatures are found to correlate with pulmonary system changes, these features may be useful for continuously monitoring the prognosis of subjects at risk for develop-
The study results showed a general trend toward a frequency-dependent drop in sound amplitude when PTX was induced, with the largest drop in the 400–600 Hz range (Fig. 3). Similar trends were observed in dog and pig PTX models of otherwise healthy subjects (27, 33). The drop in sound amplitude is also consistent with the clinical experience of decreased sounds and tactile fremitus with PTX. Selective filtering of different frequency sounds is known to occur with some lung conditions; for example, selective enhancement of high frequencies (known as egophony) may present with lung consolidation due to fluid accumulations, which can enhance sound transmission at these frequencies.

It is worth mentioning that when PTX takes place, an air pocket is formed between the affected lung and the chest wall. At the air pocket boundary, relatively strong acoustic reflections take place due to large acoustic impedance mismatch between the air on one hand and the lung parenchyma and chest wall on the other hand. Due to these reflections, the air pocket is expected to act as a sound barrier. Reflected sounds (at the air and parenchymal surface) will travel again through the parenchyma and undergo more attenuation. Portions of the sounds that are blocked by the air pocket, however, will travel along the longer path around the pocket and further attenuate before they reach the chest surface. Acoustic damping in soft tissue is lower at low frequencies (8, 33, 45–47) and, hence, low-frequency waves will travel around the PTX air pocket more efficiently (27) (i.e., they will suffer less attenuation). The data collected in the current study consistently showed less sound attenuation with PTX below 300 Hz (Fig. 3B).

As the sound frequency increases, the air pocket blocks sound waves more efficiently (because these frequencies suffer stronger attenuation when they go around the air pocket) causing a more pronounced sound amplitude drop with PTX at these frequencies. This trend was observed in the current study for frequencies in the 300–900 Hz range ($P < 0.01$, Wilcoxon signed rank test). When the frequency further increased, the measured sound amplitudes tended to decrease (due to increased tissue damping) and the drop in their amplitudes with PTX also tended to diminish. This trend was observed at frequencies above about 1,000 Hz in the current study. The detected drop in acoustic energies and energy ratios (Figs. 4 and 5) was consistent ($P < 0.01$ for both, Wilcoxon signed rank test). This may warrant further testing of this approach in a larger patient population. If proved successful, and due to the relative ease of repeating the acoustic measurements (compared with imaging methods such as chest ultrasonography and X-ray), the described method may be helpful as a complementary tool for continuous monitoring of patients at risk for developing PTX. In that respect, candidate patient populations who may benefit from this approach include patients in intensive care units who are on positive pressure ventilation and who may be at an increased risk for developing PTX. In this case, the transpulmonary transmission measurements may be integrated in ventilators that can monitor patients and alert when this potentially lethal condition develops. Such an alert would help healthcare providers decide whether further testing or intervention is needed in light of other clinical variables.

Conclusions. This study demonstrated that there are detectable spectral changes in pulmonary acoustic transmission with PTX. The spectral changes include transmitted energy levels...
and energy ratios between different frequency bands. Using these and other clinical signs may potentially provide the basis for a rapid, safe, easy-to-use, and inexpensive bedside PTX monitoring tool. On the basis of these preliminary results, further evaluation may be warranted.

GRANTS

This study was supported by National Institute of Biomedical Imaging and Bioengineering Grant R01 EB-012142.

DISCLOSURES

H.A. Mansy and R.H. Sandler founded a company in 1997 that was awarded a patent in 2002 that is related to the technology used in this article. The patent was never sold, licensed, or commercialized. The company did not commercialize any devices that are based on this technology.

AUTHOR CONTRIBUTIONS


REFERENCES


