Pneumothorax detection using pulmonary acoustic transmission measurements

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Abstract—*Pneumothorax is a common clinical condition that can be life threatening. The current standard of diagnosis includes radiographic procedures that can be costly and may not always be readily available or reliable. The objective of this study was to investigate the hypothesis that pneumothorax causes detectable pathognomonic changes in pulmonary acoustic transmission. An animal model was developed whereby 15 mongrel dogs were anaesthetised, intubated and mechanically ventilated. A thoracoscopic trocar was placed into the pleural space for the introduction of air and confirmation of a ~30% pneumothorax by direct visualisation. Broadband acoustic signals were introduced into the endotracheal tube, while transmitted waves were measured at the chest surface. Pneumothorax was found consistently to lower the pulmonary acoustic transmission in the 200–1200 Hz frequency band, whereas smaller transmission changes occurred at lower frequencies ($p < 0.0001$, sign test). The ratio of acoustic energy between low-(< 220 Hz) and high-(550–770 Hz) frequency bands was significantly different in the control and pneumothorax states ($p < 0.0001$, sign test). This implies that pneumothoraces can be reliably detected using pulmonary acoustic transmission measurements in the current animal model. Further studies are needed to investigate the feasibility of using this technique in humans.*

Keywords—*Acoustics, Audible frequency, Diagnosis, Pneumothorax, Dogs*

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1 Introduction

1.1 *Pneumothorax*

Pneumothorax (PTX) refers to air accumulation in the pleural space between the lung and the chest wall. This is a relatively common and potentially lethal condition that can arise spontaneously or as a complication of pulmonary pathology or trauma (BAUMAN *et al.*, 2001). For example, PTX can be caused by spontaneous rupture of small alveoli or blebs, progression of inflammatory diseases, penetrating (e.g. knife or bullet) or blunt (e.g. motor vehicle accident) chest trauma and complications of diagnostic or therapeutic procedures. More specifically, PTX rates are about 5% and 20% for central venous line insertions and percutaneous transthoracic lung biopsies, respectively (DESPARS *et al.*, 1994). Furthermore, PTX occurs in 5–15% of mechanically ventilated patients (HAAKE *et al.*, 1987).

A recent consensus conference of the American College of Chest Physicians has estimated that there are more than 20 000 pneumothoraces annually in the USA, with an associated managing cost of about US13×10^7 (BAUMAN *et al.*, 2001). However, because PTX is potentially life threatening, can be fast paced and is usually easily treatable, physicians are loath to miss the diagnosis. Hence, the number of diagnostic procedures to evaluate suspected PTX may be several times higher than actual PTX incidents.

1.2 *Current diagnostic methods and missed diagnosis*

Currently, PTX diagnosis involves a combination of history, physical examination and chest imaging. The latter is typically chest radiography (CXR), with computed tomography (CT) performed to investigate more obscure cases. Although a combination of physical examination, history and CXR is usually sufficient for the diagnosis, it can be difficult to obtain adequate positioning and end-expiratory timing in critically ill patients, thereby leading to misdiagnosis. For example, one study found that 30% of 112 PTX in 88 critically ill patients were not detected by routine CXRs (TOCINO *et al.*, 1985). Another study of 3500 autopsies found 12 unsuspected or untreated tension pneumothoraces, with ten of these cases being on mechanical ventilation at the time of death (LUDWIG and KIENZLE, 1978). In one prospective study of adult MICU patients, 6% developed a PTX during their ICU stay, and 32% were not correctly diagnosed at initial presentation (KOLLEF, 1991). Additionally, PTX misdiagnosis in trauma patients can

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also occur, as PTX can be present in the absence of visually observable severe chest wall injury (MELLER *et al.*, 1984; CIRAULO *et al.*, 1994).

1.3 *Objectives and achievements*

The central hypothesis of the current study is that PTX leads to characteristic acoustic transmission changes in the pulmonary system. Such methodology may lead to inexpensive devices providing accurate, portable, rapid and safe PTX diagnosis. The objective of the work presented here is to provide initial data on the acoustic transmission changes associated with PTX in a dog model. Achievements of the study include

(*a*) determination of useful acoustic signatures of PTX; e.g. PTX was found to cause a significant drop in acoustic transmission between the trachea and the chest surface
(*b*) finding the optimum frequency bands where that effect is most prominent
(*c*) identifying a single parameter that completely separated the control and PTX states.

2 Materials and methods

All experiments were approved by the Institutional Animal Care and Use Committee and carried out in the operating theatre of the Comparative Research Center of Rush-Presbyterian-St Luke's Medical Center. A schematic diagram of the experimental setup is shown in Fig. 1. Fifteen mongrel dogs under general anaesthesia with endotracheal intubation were studied in the left lateral decubitus (right-side-up) position, with their skin closely shaved. A ventilator delivered a tidal volume of $20\,ml\,kg^{-1}$ at a rate of $12\,breath\,min^{-1}$. Before each acoustic test, the ventilator was briefly stopped for 15 s at end expiration, and acoustic waves were introduced into the endotracheal (ET) tube by a speaker*. The speaker was driven by a broadband signal from a dynamic signal analyser† and a power amplifier‡. The speaker input contained all frequencies in the 20–1600 Hz range, with uniform amplitude within ±1 dB. The sound pressure level (measured using a sound level metre¶ in the ET tube was about 110 dB.

An electronic stethoscope§ was used to measure the transmitted acoustic waves in the right mid-clavicular line at the level of the third rib. The static load applied on the stethoscope was kept at 250–300 g, as monitored by a load cell‖ connected to a digital voltmeter. This load was chosen because it provides enough coupling to the chest surface without significantly affecting stethoscope performance. The stethoscope output was recorded on an analogue audio tape recorder# for 15 s during each acoustic test. To help ensure accurate identification of experimental data, the details of each experiment as it was performed were recorded on a separate audio track using a Lavalier microphone.

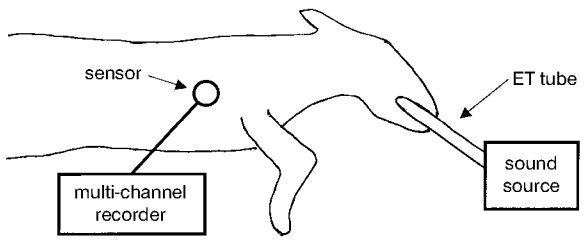

Fig. 1 *Schematic diagram of experimental setup. Sound is introduced through endotracheal tubes into respiratory system, while acoustic signal is measured at chest wall surface*

Following baseline measurements, the PTX state was created by insertion of a 5 mm thoracoscopic trocar** into the pleural space via a small incision in the seventh intercostal space in the right mid-axillary line. The obturator was removed, leaving the thoracoscopic cannula in place. A video endoscope was inserted through a trocar, confirming the control state. Air was then introduced via the cannula using a syringe attached to a three-way stopcock to produce approximately 30% PTX. The percentage of PTX was calculated based on air pocket volume, where a 100% PTX was defined as the condition corresponding to atmospheric air pocket pressure at full expiration. The thoracoscope was inserted intermittently to confirm the location and degree of PTX. This was followed by performance of the acoustic tests, as described earlier. In addition, the spectra of the acoustic input and electronic stethoscope output were monitored on-line with a laptop computer using an analogue-to-digital card†† and digital signal-processing software‡‡. A low-pass anti-aliasing 4th-order filter¶¶ with a cutoff of 1500 Hz was used in addition to the internal low-pass filter (cutoff = 2000 Hz) integrated into the electronic stethoscope.

During post-processing, the sensor output was digitised at $4096\,samples\,s^{-1}$ for 15 s. Each data set was divided into 120 overlapping segments (250 ms each, overlap = 50%). Using the fast Fourier transform (FFT), the spectrum of chest wall sounds was calculated for each data segment after windowing with the Hanning window. The mean spectral values were determined by the averaging of results from all segments at each frequency (4 Hz resolution) from 0 to 2048 Hz. Note also that each spectral estimate was determined from about 120 realisations, which led to a reasonable (9%) spectral uncertainty at the 95% confidence interval.

These spectra were utilised to distinguish between the control and PTX states. Because each spectrum was described by a large number of values (one for each of the 512 frequency bins), parameter reduction was sought. The study results suggested that the energy ratio between two frequency bands could serve as a single diagnostic parameter. Such ratios were calculated as the total energy in one band divided by the total energy in the second band, where the total energy in any frequency range was calculated as the sum of the spectral energy values within that range.

To optimise the choice of frequency bands, two variable-width spectral windows were defined as a stationary window and a moving window. The former was located at the low-frequency region (starting at 0 Hz), and the latter was allowed to sweep through the entire frequency range without overlapping with the first window. For each possible window width and location, the energy ratio was calculated for each of the 15 subjects, in both the control and PTX states. As the objective of this analysis was best to separate the control and PTX states, optimum frequency bands were chosen as the ones that maximised energy ratio differences between the two states. This was followed by the choosing of appropriate thresholds that separated the control and PTX states. Statistical-significance calculations were performed using the sign test and the Wilcoxson signed rank test. A p-value less than 0.05 was used to determine significance.

*Model DA-1, ElectroVoice, Buchanan, MI, USA
†Model 35670, HP, Loveland, CO, USA
‡Model PM-125, Carver Professional, Portland, OR, USA
¶Catalogue number 33-2055, Radio Shack, Fort Worth, TX, USA
§Model 04-1060, Labtron Electromax, Hauppage, NY, USA
‖ELF-T3E-2L, Entran, Fairfield, NJ, USA
#Portastudio 424, Teac Corp., Japan
**Model 355, Endopath Dilating Tip, Ethicon, Cincinnati, OH, USA
††Card 500, National Instruments, Austin, TX, USA
‡‡Matlab, MathWorks, Natick, MA, USA
¶¶MX 350 Sound Craftsmen, Santa Ana, CA, USA

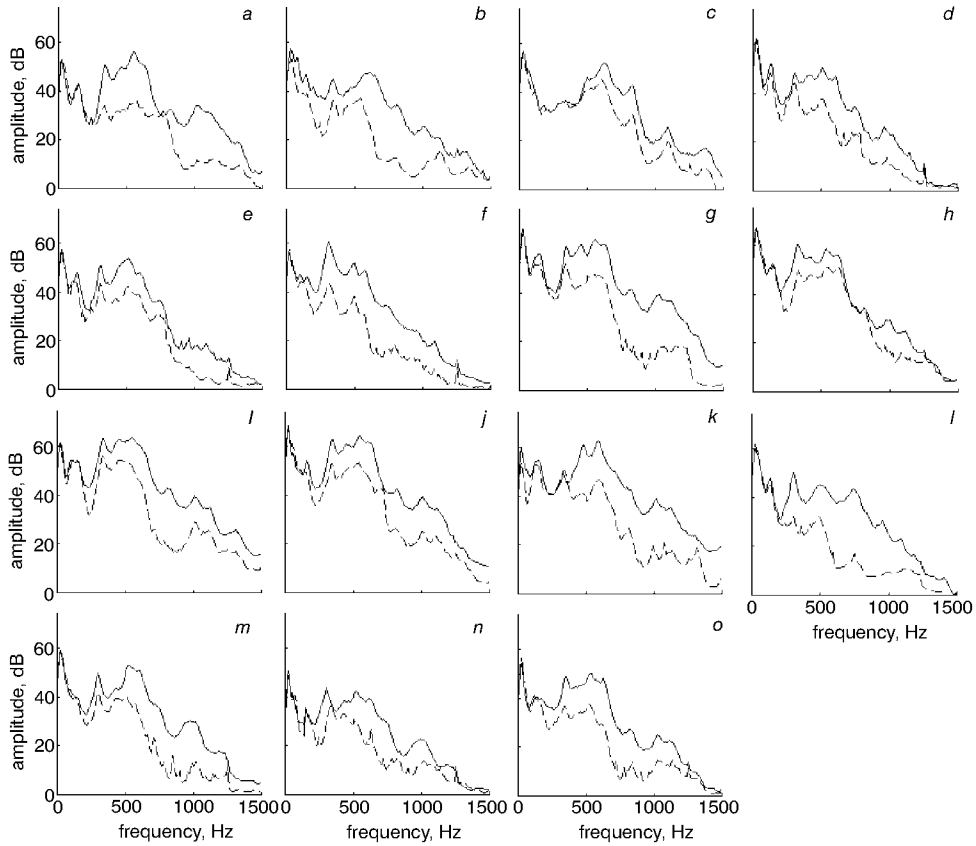

Fig. 2 *(a)–(o) Spectra of sounds transmitted from mouth to chest wall of 15 animals for (—) control and (---) 30% PTX states. Large drop is apparent in acoustic transmission due to PTX at frequencies above ~300 Hz, compared with small change in amplitude in 0–250 Hz range ($p = 0.00006$). Note that amplitude axis is logarithmic, so that changes are a factor of 10–100*

3 Results

The spectra of chest wall sounds are shown in Fig. 2 for each of the 15 individual subjects, in both the control and the PTX states. So that we can more easily visualise overall trends, Fig. 3 shows the mean spectra of all 15 animals, with the 95% confidence intervals indicated by the error bars.

Although intra-subject variability is evident, three overall trends can be seen in Fig. 2 and 3. First, the higher-frequency sounds are less well transmitted from the airways to the chest wall for both states. Secondly, the low-frequency sounds appear to be least affected by the presence of PTX. Finally, in each of the 15 subjects, high frequencies (>200 Hz) suffered greater amplitude attenuation in the PTX state.

These trends suggested that the energy ratio between two different frequency bands may be a useful parameter for PTX diagnosis. The frequency-band choice was optimised, yielding frequency windows of 550–770 and 0–220 Hz. Fig. 4 shows the energy ratios for the control and PTX states in all

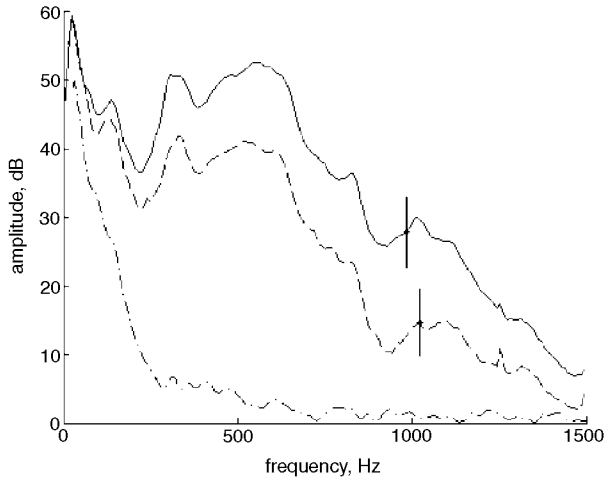

Fig. 3 *Mean spectra for (—) control and (---) PTX states. In addition to general trend of decreasing amplitude with frequency, there is significant drop due to PTX of at least 20 dB for frequencies between 300 and 1200 Hz. (-·-·) Background noise*

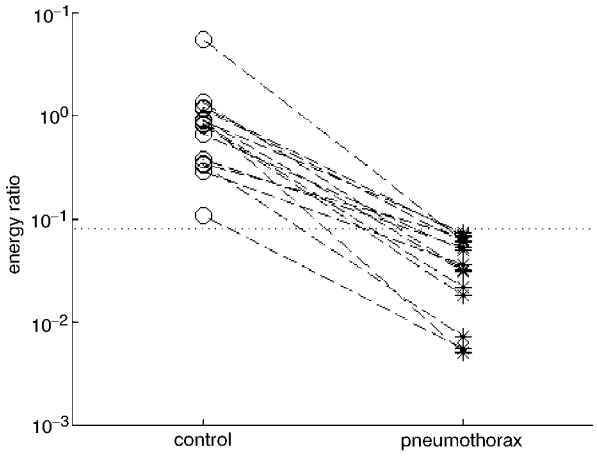

Fig. 4 *Ratio between acoustic energies of high-(550–770 Hz) and low-(0–220 Hz) frequency bands of sounds transmitted from mouth to chest wall for (○) control and (*) PTX states. (---) Connects same animal data points. Note that, in every case, ratio was higher for controls than for pneumothoraces and that (. . .) threshold value of 0.08 seemed to separate completely two states*

animals. This ratio was found to be lower in each of the PTX states ($p = 0.00006$, Wilcoxson signed rank test). It can also be seen that all control energy ratios were greater than 0.10, whereas those of each PTX state were less than 0.06 ($p = 0.00006$, sign test).

4 Discussion

The current study investigated the spectral changes in the acoustic transmission of the pulmonary system due to PTX. To measure these changes, sound was introduced at the mouth and measured at the chest wall surface. These measurements suggested that the acoustic barrier created by PTX hindered sound transmission. The study identified a single acoustic parameter that appeared to separate the control and PTX conditions, suggesting possible utility for PTX diagnosis. Advantages of the method include speed, accuracy, lack of radiation, safety, easy of use and low cost; its disadvantages include be the need for an external sound source.

4.1 *General spectral features*

The spectral data of Figs 2 and 3 showed a general trend of decreased acoustic transmission with increasing frequency as a result of the increased acoustic damping, which is consistent with previous reports on wave propagation in biological tissue and systems (MANSY *et al.*, 2001; WODICKA and SHANNON, 1990; KRAMAN and AUSTRHEIM, 1983; DONNERBERG *et al.*, 1980; VON GIERKE *et al.*, 1952; OESTERICHER, 1951). An important consequence of lower signal amplitudes at high frequencies is the drop in the signal-to-noise ratio. For example, the signal-to-noise ratio was less than 0 dB at 1800 Hz and therefore data analysis was limited to frequencies <1500 Hz. The average roll-off of the acoustic spectra between 300 and 1500 Hz was about 20 dB per octave. This is higher than the reported roll-off of the acceleration signal between the extrathoracic trachea and the posterior chest of normal men (10–17 dB per octave between 300 and 600 Hz) (WODICKA and SHANNON, 1990). Note that only qualitative comparison with the latter study is warranted, because of differences in the system configuration, actuators, sensors, sensor positioning and species. It is also to be noted that, although electrical signal input was band-limited white noise, the acoustic input into the trachea was non-uniform owing to speaker characteristics and because of the acoustic impedance of the lungs.

The spectral data also showed some 'peaks and valleys'. More specifically, the spectra peaked at about 500 Hz and had a dip at about 250 Hz. The morphology of these spectra and the frequency of resonance and anti-resonance are similar to those reported in a previous dog model (DONNERBERG *et al.*, 1980) and in healthy humans (WODICKA *et al.*, 1992). For example, the former study found a peak at about 300 Hz, and the latter reported peaks at about 150 and 450 Hz and a dip at about 375 Hz. Figs 2 and 3 also suggest that the frequency of the spectral peaks and dips was not significantly altered by the presence of PTX. Similar spectral insensitivities to lung contents have been previously reported in a canine model of congested lungs (DONNERBERG *et al.*, 1980). Although investigating the possible sources of the observed resonances and anti-resonances is beyond the scope of the current study, potential mechanisms may include chest wall vibrations (i.e. chest wall mass oscillates, while the rib cage stiffness provides the elastic restoring action), airway resonance and standing waves in the chest that may be allowed to form given the small acoustic damping nature of the lung parenchyma at low frequencies (WODICKA *et al.*, 1992).

4.2 *Diminished transmitted sounds with pneumothorax*

The trend of reduced chest acoustic signals with PTX is consistent with the well-known clinical sign of diminished breath sounds over the affected lung (JARVIS, 1992). One possible reason for this amplitude drop is the acoustic impedance mismatch at the boundary of the air cavity created by PTX. We hypothesise that this mismatch is caused by the large differences in sound speed and mass density (of air compared with the parenchyma and chest wall) that can combine to create a huge jump in the acoustic impedance at the air cavity boundary. Because an acoustic impedance mismatch strongly reflects sound waves, the net effect would be a decrease in the transmitted acoustic signal amplitude. Published values of the acoustic properties of the parenchyma (WODICKA *et al.*, 1989), air (LEIGHTON, 1994) and chest wall (VOVK *et al.*, 1995) were used to estimate the energy loss coefficient T_L between any two layers as $T_L = 4\ Z_1 Z_2/(Z_1 + Z_2)^2$, where Z_1 and Z_2 are the acoustic impedance of the two layers. This simplified analysis predicted an acoustic energy loss due to PTX of 20–25 dB, which is in agreement with the spectral energy drop of up to 16 dB seen in Figs 2 and 3 in the 300–1200 Hz range. This effect was not seen at lower frequencies, as discussed below.

4.3 *Redistribution of spectral energy due to pneumothorax*

Pneumothorax can cause spectral energy redistribution secondary to the frequency-dependent acoustic transmission characteristics. As the acoustic barrier created by PTX blocks the direct route of sound transmission from the parenchyma to the chest surface, low-frequency sound waves still reach the surface efficiently on the ipsilateral side via longer pathways, as attenuation is small at low frequencies (MANSY *et al.*, 2001; WODICKA and SHANNON, 1990). Conversely, the high frequencies endure significantly higher damping as they attempt to reach the surface via the longer path around the air pocket. This could have caused the preferential high attenuation of the high frequencies seen in Figs 2 and 3. Another possible factor for the observed low-pass filtering of the PTX air pocket may be that the latter acts as a resonator (e.g. Helmholtz resonator) with increased absorption characteristics at high frequencies. The current study exploited this frequency-dependent attenuation for PTX diagnosis by calculating the ratio of acoustic energy between the high- and low-frequency bands. One possible advantage of this use of ratios, compared with using the sound energy in a single frequency band (e.g. 0–1500 Hz), is that the latter may be highly dependent on subject size and other chest structural variations.

4.4 *Necessity of evaluating baseline for each subject*

The study results suggest that baseline control state measurements may not be needed for diagnosis of PTX using the proposed technique, as the energy ratio of certain frequency bands seemed adequately to distinguish between the PTX and control states. This is advantageous, as baseline measurements may not always be available. However, there are two common situations in which baseline measurements will be easily available and, hence, can be used to increase test accuracy further. The first involves patients with unilateral pneumothoraces (seen in about 98% of cases (ATHANASSIADI *et al.*, 1998)), where the contralateral hemithorax can serve as a control. The second is when sequential or on-line patient monitoring is performed. In these cases, initial (non-PTX) data will be typically available, enabling patients to serve as their own controls.

4.5 *Detection of small pneumothoraces*

Ideally, a PTX detection method should reliably detect all clinically significant pneumothoraces, which are typically thought to be those >15% (SAHN and HEFFNER, 2000). Small pneumothoraces can also be dangerous as they can progress to complete lung collapse and can produce 'tension physiology' with cardiovascular collapse (LANCEY, 1999). The current study found that 30% pneumothoraces could be reliably detected using pulmonary acoustic transmission measurements. This suggests that the minimum detectable PTX using this model is somewhere less than 30%. It is expected that smaller air collections will be detectable, especially after further method enhancements. Optimisations may include signal-processing development and optimum sensor choice and placement. Such expectations are realistic, as acoustic coupling dramatically drops after the first pulling away of lung parenchyma from the chest wall, creating an acoustic impedance mismatch. The separation of these two surfaces occurs at the highest point (owing to gravity), which is where chest wall sensors can best be placed. Future human studies can implement multiple sensors (especially given the potential for loculation and adhesions). One use limitation, however, may be pulmonary bullae, which result in air pockets within the lung, as those may have similar acoustic effects to PTX. Further studies would be required to investigate those effects.

4.6 *Sensor properties and positioning*

The current study used an electronic stethoscope gently placed at the chest surface to measure the transmitted sounds. The stethoscope characteristics were tested in an earlier pilot study (MANSY *et al.*, 1999), where broadband noise was generated in a phantom, while phantom surface vibrations were measured successively using the stethoscope and a calibrated laser Doppler vibrometer[§§]. Such measurements suggested that the stethoscope output was proportional to surface displacements with a flat response (±1.5 dB, standard deviation) in the 70–2000 Hz range (Fig. 5). This is consistent with the manufacturer claims of flat response up to about 2000 Hz. Note that using a transducer with a non-flat response will affect the measured spectra but will not affect the primary conclusion of the study, namely, that PTX causes a significant drop in acoustic transmission between the trachea and the chest wall.

Although the sensor position was not systematically varied in the current study, pilot data and computer simulations (ZHANG *et al.*, 2001) suggested that exact position matching is not necessary. In addition, sensor position matching among animals can only be approximate because of animal anatomic variability. The relatively large stethoscope diameter (about 4 cm) further contributes to the lowering of the importance of exact sensor positioning.

To develope diagnostic devices that rely on the approach presented, evaluation of different sensor positions and levels of PTX would be needed. Recent studies using computer models carried out by our group (ZHANG *et al.*, 2001) suggested that increasing the size of PTX tends to increase sound amplitude drop, and that sensors can be optimally located closest to the suspected PTX position. Validation of such trends in animals and humans is left for future studies, and clinical application of the technique may require the use of multi-sensors to increase sensitivity and specificity.

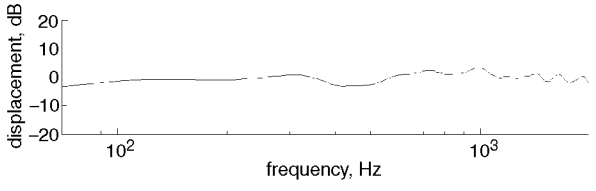

Fig. 5 *Surface displacement measured by electronic stethoscope used at different frequencies. Relatively flat response can be seen. Reference surface displacement was measured by calibrated laser Doppler vibrometer*

[§§]CLV 800, Polytec, Auburn, MA, USA

5 Conclusions

This study investigated the effect of PTX on sound transmission from the airway to the chest wall, and the results suggest possible clinical utility. The following acoustic trends were observed in the current model:

(i) increased attenuation of higher-frequency (200–1200 Hz) sounds occurred with PTX;
(ii) transmission of low-frequency (< 200 Hz) sounds appeared to be minimally affected by PTX;
(iii) separation between PTX and control states was achieved through use of spectral energy ratios.

As such a methodology may lead to inexpensive devices providing accurate, portable, rapid and safe PTX diagnosis, further investigations appear warranted.

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