Volume-dependent variations of regional lung sound, amplitude, and phase

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Kiyokawa, Hiroshi, and Hans Pasterkamp. Volume-dependent variations of regional lung sound, amplitude, and phase. J Appl Physiol 93: 1030–1038, 2002. First published May 10, 2002; 10.1152/japplphysiol.00110.2002.—Acoustic imaging of the respiratory system demonstrates regional changes of lung sounds that correspond to pulmonary ventilation. We investigated volume-dependent variations of lung sound phase and amplitude between two closely spaced sensors in five adults. Lung sounds were recorded at the posterior right upper, right lower, and left lower lobes during targeted breathing (1.2 ± 0.2 l/s; volume = 20–50 and 50–80% of vital capacity) and passive sound transmission (±0.2 l/s; volumes as above). Average sound amplitudes were obtained after band-pass filtering to 75–150, 150–300, and 300–600 Hz. Cross correlation established the phase relation of sound between sensors. Volume-dependent variations in phase (±1.5 ms) and amplitude (±11 dB) were observed at the lower lobes in the 150- to 300-Hz band. During inspiration, increasing delay and amplitude of sound at the caudal relative to the cranial sensor were also observed during passive transmission in several subjects. This previously unrecognized behavior of lung sounds over short distances might reflect spatial variations of airways and diaphragms during breathing.

respiratory mechanics; acoustics; respiratory sounds; pulmonary ventilation; signal processing; computer assistance

SIGNIFICANT ADVANCES HAVE BEEN MADE in the physiological imaging of pulmonary airways by using computer tomography and magnetic resonance techniques (5). Less well developed is the diagnostic application of computerized lung sound analysis, but recent efforts at standardizing measurements and reporting (33) reflect the growing interest in respiratory acoustic techniques to assess pulmonary ventilation.

Because lung sounds originate in large- and medium-size airways and are transmitted to the chest surface through air spaces, parenchyma, and chest wall, they offer an indirect way to the imaging of pulmonary ventilation. Lung sound generation is affected by lung volume and by the velocity and direction of airflow (9, 18). Regional ventilation (19) and closing volume (27) influence lung sound intensity. Individual anatomic variations within the chest cavity affect sound transmission from mouth to chest surface (17), and the density of pulmonary parenchyma impacts on the propagation and attenuation of sound (30, 31).

In humans, breath sound intensity decreases with induced bronchial narrowing (3, 23). In dogs, there is increased sound transfer with induced pulmonary edema (8). It is also well known that changes of lung sounds occur when air or fluid collects in the pleural space (6). Although auscultation offers a subjective method to detect such variations, the use of objective respiratory acoustic measurements is more promising for reliable and reproducible detection of regional changes.

The rapid development of computer technology has made it possible to acquire, process, and store lung sound signals from multiple sites and visualize regional acoustic changes during respiration with relative ease. Kompis et al. (13) used an array of 16 sensors in a recent study to compute acoustic images of the thorax from lung sounds in children and adults. With only two closely spaced sensors, we found that the respiratory phase affected the time difference (Δt) of lung sounds between both recording sites in healthy adults (12). These preliminary observations suggest that more changes occur in lung sounds within narrow regions on the chest surface than had been previously recognized. We hypothesized that these changes in lung sounds over short distances may relate to changes in lung volume and possibly to changes in transmission rather than generation of sounds within the chest. The objectives of our study were, therefore, 1) to measure volume-dependent variations of sound amplitude and phase between two closely spaced sensors; 2) to investigate the effects of lung volume, recording site, and sensor distance; and 3) to compare observations on lung sounds with those obtained during passive sound transmission.

MATERIALS AND METHODS

Five healthy nonsmokers, ages 21–50 yr, gave their consent to participate in this study. The project was approved by the Health Research Ethics Board of the University of Manitoba. Lung function testing was performed according to...
American Thoracic Society (ATS) guidelines (1) and by using the reference values of Morris (22). All subjects had normal static and dynamic pulmonary function (Table 1). Measurements were made with the participants seated inside a body plethysmograph (V6200 Autobox, SensorMedics, Yorba Linda, CA) (Fig. 1). Three recording sites were chosen on the posterior chest (right upper (RU) at the level of T4, right lower (RL) and left lower (LL) at the level of T8, 5 cm lateral to the spine) to represent areas with different magnitudes of change in lung volume and airway spatial configuration during breathing. At these recording sites, two contact sensors (EMT 25C, Siemens Canada, Mississauga, Ontario) were attached in craniocaudal alignment with 33 (narrow) or 43 mm (wide) center-to-center distance with double-sided adhesive tape rings (Double-Stick discs, model 2181, 3M Health Care, St. Paul, MN). Each sound signal was amplified and then filtered to avoid aliasing (custom-made amplifier and analog 6th order Bessel low-pass filter, cut-off at 2.5 kHz). Signals were sampled at 10,240 points/s with 12-bit resolution by using data acquisition hardware (PCI-1200, National Instruments, Austin, TX) on a personal computer (IBM-PC300 PL, IBM Canada, Markham, Ontario). Flow and volume signals from the plethysmograph input/output interface (POD Box, SensorMedics) were simultaneously acquired and then down-sampled to 320 points/s.

Subjects breathed through a pneumotachograph while observing flow and volume targets on a computer (custom-written application in LabVIEW, National Instruments). Recordings were made at two target volumes: 20–50% (low volume) and 50–80% of vital capacity (high volume), with a target flow of 1.2 ± 0.2 l/s for inspiration and expiration. During passive sound-transmission studies, target flows were <0.2 l/s to avoid the generation of lung sounds. In transmission studies, low-pass-filtered white noise (cutoff frequency 320 Hz, 24 dB/octave) generated with an acoustic driver (PD-60, Atlas Sound, Parsippany, NJ) was introduced at the mouth. Four air vent holes (7 mm diameter) on the conducting hose outside the body plethysmograph allowed slow breathing maneuvers during sound transmission. Each recording was started with a slow maximal inspiration and expiration to establish vital capacity. Subjects then performed seven complete respiratory cycles for the recording of lung sounds. During passive sound transmission, they per-

<table>
<thead>
<tr>
<th>Subject characteristics</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
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<td>4.0</td>
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</tr>
<tr>
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<td>3.5</td>
<td>3.5</td>
<td>4.3</td>
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<td>5.5</td>
<td>5.9</td>
<td>6.7</td>
</tr>
<tr>
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<td>1.5</td>
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<td>1.2</td>
</tr>
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<td>2.4</td>
<td>3.9</td>
<td>2.6</td>
<td>3.8</td>
</tr>
</tbody>
</table>

A–E, subject; FVC, forced vital capacity; FEV1, forced expiratory volume in 1 s; TLC, total lung capacity; RV, residual volume; FRC, functional residual capacity; m, male; f, female.

Fig. 1. Schematic diagram of sensor placement and recording of sounds at the chest surface. D, distance; RU, right upper; RL, right lower; LL, left lower; I/O, input/output; PC, personal computer.
formed two complete respiratory cycles at very low flows (see above). At the end of each recording, they held their breath for 5 s to allow the recording of background noise.

The recordings were obtained on two consecutive days. On the first day, lung sounds were recorded from each chest site at two lung volumes with a narrow sensor distance. The procedure was then repeated with the wider sensor distance. On the second day, lung sounds were again recorded with the narrow sensor distance and then the transmission study was performed with the sensors unchanged.

For analysis, the sound signals were separated into octave bands of 75–150, 150–300, and 300–600 Hz. The average and standard deviation of the signal-to-noise ratio for all subjects was calculated in each band. An average signal-to-noise ratio of >10 dB with a standard deviation of <50% of the mean was considered acceptable. Five respiratory cycles, excluding the first and last breath of the recording, were used for lung sound analysis. Both breath cycles with transmitted sounds were analyzed. The band-pass filtered signal was divided into 100-ms periods of 1,024 data points each. Each segment pair of signals from both sensors was analyzed for differences of amplitude and time. The amplitude difference between the sensors was ∆A. A positive ∆A indicated a larger amplitude at the caudal compared with the cranial sensor. To calculate ∆A, the root mean square of both signals was first determined by squaring of the waveforms, averaging over time, and finding the square root function. Root mean square of the caudal signal was then divided by that of the cranial signal and expressed in decibels

$$\Delta A = 20 \log \frac{\text{caudal RMS}}{\text{cranial RMS}}$$

∆A values were calculated for each 100-ms segment.

The time difference between both sensors was ∆t. A positive ∆t indicated a later arrival of sound at the caudal compared with the cranial sensor. To calculate ∆t, we used cross correlation

$$\gamma(t) = \sum_{k=1}^{N-t} x(k)y(k + t)$$

where N is number of sample points and k is discrete time. If function y(t) is a time-delayed version of function x(t), the cross correlation |γ(t)| between these two functions will become maximum at t = ∆t (10). In other words, ∆t can be estimated by determining the maximum of |γ(t)|. We calculated ∆t for each 100-ms segment. Figure 2 shows the process of filtering and segmenting the signals, of normalizing and zero padding, and of determining the maximum |γ(t)| for the shortest wavelength contained within each octave band. The latter was done to avoid phase ambiguity, i.e., matching at distances greater than one wavelength.

We determined the correlation of ∆t and of ∆A with lung volumes after volume data were down-sampled to 10 points/s. The maximum cross correlation was searched within ±½ breath cycle. We defined a positive correlation as significant when r ≥ 0.5 and a negative correlation when r ≤ −0.5. In the analysis of volume-dependent variations of ∆A and ∆t, values were excluded when flow at the corresponding segment was <0.5 l/s because acceptable lung sound amplitudes could not be expected at such low flows (7). To eliminate spurious correlations, values were also excluded when the shift relative to the lung volume signal was >0.5 s. To avoid phase ambiguity (see above), values of ∆t exceeding ±6.6 ms (at 75–150 Hz), ±3.3 ms (at 150–300 Hz), or ±1.6 ms (at 300–600 Hz) were excluded. If >20% of segments in a recording had to be removed because of these exclusion criteria (valid segments of <80%), the respective data set was considered noninformative and was excluded from further statistical analysis.

![Fig. 2. Sequence of analysis to determine the phase difference of sound between two sensors. ∆t, Difference of time.](http://jap.physiology.org/)

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**RESULTS**

We observed that the maximal change in the spatial separation of the sensors was <1 mm when subjects breathed from total lung capacity to residual volume. Acceptable lung sound amplitudes were found at all recording sites within the frequency band of 150–300 Hz whereas low-frequency (75–150 Hz) and high-frequency sound (300–600 Hz) amplitudes were of acceptable signal strength only at the RL and RU recording sites, respectively. Passively transmitted sound had acceptable amplitudes within 150–600 Hz at all recording sites (Table 2). The observed variations in ΔA and Δt correlated with changes in lung volume (Figs. 3 and 4). Observations with narrow spacing of the sensors were considered significant only if they were reproducible over 2 consecutive days.

For lung sounds in the 150- to 300-Hz band, the most consistent findings of volume-dependent variations of ΔA were obtained at the RL and LL sites, whereas respective Δt variations were most consistent at the RL at low lung volumes (Tables 3 and 4). No breath-synchronous variations of lung sound ΔA or Δt were observed at the RU. However, within the 300- to 600-Hz band, there was volume-dependent variation of Δt in one subject (subject C) and of ΔA in two subjects (subjects B and C) at the RU site.

Lung sounds in the 150- to 300-Hz band recorded with narrow spacing of the sensors showed a positive correlation of ΔA with lung volume in 10 of 17 valid recordings. In those recordings, the variation of ΔA preceded the changing lung volume by 200 ± 140 ms. The strength of the cross correlation between ΔA and lung volume was 0.80 ± 0.10. There were 10 positive correlations of Δt in 21 valid recordings. In those recordings, the variation preceded the changing lung volume on average by 40 ± 120 ms. The strength of the cross correlation between Δt and lung volume was 0.67 ± 0.10.

With narrow distance of the sensors, the average ΔA changed from –6.3 ± 1.8 dB at 20% of vital capacity to +1.2 ± 0.8 dB at 50%. Correspondingly, the average Δt changed from –0.15 ± 0.06 ms at 20% to +0.50 ± 0.14 ms at 50% (Fig. 5). With the wider distance of the sensors, the average ΔA changed from –7.6 ± 1.1 to +1.6 ± 0.5 dB and the average Δt from –0.20 ± 0.08 ms at 20% to +0.73 ± 0.10 ms. This increase in the magnitude of volume-dependent variations of ΔA and Δt with increased sensor distance was statistically significant: average ΔA changed from 7.5 ± 1.1 to 9.2 ± 0.8 dB (P < 0.01, one-sided Student t-test) whereas average Δt changed from 0.65 ± 0.17 to 0.93 ± 0.15 ms (P < 0.05). The observed variations in ΔA and Δt were reproducible over 2 consecutive days and at both target volumes (Fig. 6).

Four of five subjects were available for further recordings that were added after the study results had been compiled and analyzed. This was done as an effort to elucidate whether movements of the diaphragm or changes in the spatial orientation of airways were the most likely mechanism behind the observed lung volume-dependent changes of phase and amplitude. If the former were the case, one would not expect to find lung volume-dependent variations of ΔA and Δt between horizontally rather than vertically aligned sensors.

With the use of the same setting and equipment, recordings were obtained at RL and LL, first with the original vertical alignment of the sensors, then moving the upper sensor lateral and horizontal to the lower sensor and keeping the narrow distance. Of 128 record-

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**Table 2. Sound signal strength**

<table>
<thead>
<tr>
<th>Freq, Hz</th>
<th>Lung Sounds</th>
<th>Transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Insp</td>
<td>Exp</td>
</tr>
<tr>
<td>75–150</td>
<td>RU</td>
<td>10.0 ± 2.9</td>
</tr>
<tr>
<td></td>
<td>RL</td>
<td>13.7 ± 2.7*</td>
</tr>
<tr>
<td></td>
<td>LL</td>
<td>12.7 ± 3.9</td>
</tr>
<tr>
<td>150–300</td>
<td>RU</td>
<td>19.8 ± 5.0*</td>
</tr>
<tr>
<td></td>
<td>RL</td>
<td>20.9 ± 3.0*</td>
</tr>
<tr>
<td></td>
<td>LL</td>
<td>20.1 ± 3.7*</td>
</tr>
<tr>
<td>300–600</td>
<td>RU</td>
<td>25.2 ± 5.2*</td>
</tr>
<tr>
<td></td>
<td>RL</td>
<td>20.7 ± 3.9</td>
</tr>
<tr>
<td></td>
<td>LL</td>
<td>23.2 ± 4.3</td>
</tr>
</tbody>
</table>

Values are means ± SE of signal-to-noise ratio (in decibels). Freq, frequency; Insp, inspiration; Exp, expiration; RU, right upper; RL, right lower; LL, left lower. *Data sets that met inclusion criteria for further analysis (see text for details).
ings in total, 108 were valid data sets. Amplitude variations of the same pattern that was originally observed were present again in 57 and 50% of recordings at low and high frequencies, respectively. Time variations were observed in 15 and 31%, respectively.

With horizontal alignment, sound at the lateral sensor became relatively louder during inspiration in 29 and 43% of recordings at low and high frequencies, respectively, whereas time variations were observed in 15 and 31%, respectively.

Fig. 4. Example of passively transmitted sound recorded at RL from subject A breathing at high lung volume (sensor spacing and frequency range same as in Fig. 3). Again, there is a positive correlation of ΔA (r = 0.87) and Δt (r = 0.88) with lung volume.

### DISCUSSION

Noninvasive assessment of regional ventilation is one of the most promising aspects of respiratory acoustic technology. However, there are highly complex relations of the breath synchronous vibrations at the chest wall that we measure as lung sounds (24) and the underlying changes in lung volume (16, 29, 30), intrapulmonary pressure and tissue density (31), spatial configuration and diameter of airways (18, 25), and gas density (26) and flow velocity (9). Furthermore, the transmission of sound from the airways through parenchyma and chest wall to a sensor at the surface is dependent on the sound frequency (35).

The comparison of lung sounds at two or more sensors on the chest surface has shown that regional ventilation, at least between different lobes, may account some of the observed differences in timing and amplitude. Using two microphones over the left anterior upper and posterior lower chest in human volunteers, Leblanc et al. (19) measured normal breath

<table>
<thead>
<tr>
<th>Freq. Hz</th>
<th>Site</th>
<th>Volume</th>
<th>Lung Sounds</th>
<th>Transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td>75–150</td>
<td>RU High Low</td>
<td>+2, -0(4) +4, -0(5)</td>
<td>Narrow, 2 days Wide, 1 day Narrow, 1 day</td>
<td></td>
</tr>
<tr>
<td>150–300</td>
<td>RU High Low</td>
<td>+0, -0(2) +0, -0(1)</td>
<td>Narrow, 1 day</td>
<td></td>
</tr>
<tr>
<td>300–600</td>
<td>RU High Low</td>
<td>+1, -0(1) +1, -0(2)</td>
<td>Narrow, 1 day</td>
<td></td>
</tr>
</tbody>
</table>

Positive and negative prefixes identify the number of subjects with positive and negative breath synchronous variations, respectively. Total numbers of subjects with valid data sets are shown in parentheses.

### Table 4. Volume-dependent variations of time difference

<table>
<thead>
<tr>
<th>Freq. Hz</th>
<th>Site</th>
<th>Volume</th>
<th>Lung Sounds</th>
<th>Transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td>75–150</td>
<td>RU High Low</td>
<td>+0, -0(3) +0, -0(2)</td>
<td>Narrow, 2 days Wide, 1 day Narrow, 1 day</td>
<td></td>
</tr>
<tr>
<td>150–300</td>
<td>RU High Low</td>
<td>+0, -0(1) +1, -0(3)</td>
<td>Narrow, 1 day</td>
<td></td>
</tr>
<tr>
<td>300–600</td>
<td>RU High Low</td>
<td>+1, -0(1) +1, -0(2)</td>
<td>Narrow, 1 day</td>
<td></td>
</tr>
</tbody>
</table>

Positive and negative prefixes identify the number of subjects with positive and negative breath synchronous variations, respectively. Total numbers of subjects with valid data sets are shown in parentheses.
sound intensity at controlled volumes, flows, and body position. They found a relative increase of sound intensity at the caudal sensor during inspiration and concluded that lung sound intensity correlated with regional ventilation. Ploy-Song-Sang et al. (27) used two microphones lateral to the anterior axillary line on the right chest with a 10-cm distance between the cranial and caudal sensors. They found that, below closing volume, sound was louder at the cranial compared with the caudal sensor and that the opposite was true when their healthy adult subjects breathed above closing volume. They explained the observed variations by the changes in regional ventilation that were based on the simultaneously recorded swings in esophageal pressure. Although changes of sound amplitude between regions of the lung have been observed during breathing, the relative timing of sound measured at two separate sensors had until now only been studied with passive sound transmission. In these transmission studies, one microphone would typically be placed at the neck and another one on the chest, and cross-correlation analysis would be used to determine the timing of sound relative to the tracheal site (15, 34).

Our present study is the first attempt to quantify and characterize the variations in amplitude and timing of respiratory sounds over a close range on the chest surface. We found reproducible volume-dependent variations in the amplitude and timing of lung

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**Fig. 6.** Volume-dependent variations of $\Delta t$ and $\Delta A$ were reproducible on consecutive days (sensor distance = 33 mm; frequency range = 150–300 Hz). $\Delta A$ and $\Delta t$ in subject C (LL, high volume) are not shown because of insufficient valid segments (62 and 60%, respectively).
sounds over relatively short distances. These observations were prominent at the lower lobes during low-volume breathing, whereas there were fewer valid recordings from the RU recording site, and positive findings were more sporadic (Tables 3 and 4). The observed magnitude of amplitude variation, i.e., ≥6 dB on average when breathing at the lower lung volumes, is relevant since this should be readily apparent on subjective auscultation with a dual-head stethoscope.

The direction of change was always toward a relative increase in sound amplitude at the caudal sensor as lung volume increased. Similar findings were obtained from both sides of the chest. In contrast, no breath synchronous amplitude changes of lung sounds were observed at the upper chest. Over the relatively narrow range that was compared, the separation distance between the sensors did not change the pattern of volume-dependent amplitude variation but did change its magnitude. The variations of amplitude at the lower lobes showed a similar pattern in the transmission experiments. All this points to a potential in synchrony-dependent amplitude variation but did change its magnitude. The variations of amplitude at the lower lobe recording sites and is consistent with earlier observations by Ploy-Song-Sang et al. (27).

An increase in distending pressure and an increase in volume tends to decrease the amplitude of sound transmitted through the lungs. This has been observed in excised pig lungs (20) and also in healthy human subjects (30). In both settings, a frequency dependence of the volume effect on transmitted lung sounds has been observed. In the excised pig lungs, the finding of greater attenuation with greater distending pressure was most prominent at lower frequencies of 100–300 Hz. In contrast, the in vivo observations in humans showed the greatest effect at frequencies >300 Hz. In none of these investigations were breath volume-dependent amplitude changes compared over distances as narrow as in our study. The focus of our study was on breath synchronous changes of lung sounds over narrow distances, and we did not employ a tracheal reference sensor. Thus we cannot comment on the absolute degree of attenuation of transmitted sounds but only on the relative change between neighboring sensors.

A relatively increased sound transmission to the caudal sensor with increasing lung volume was observed in all octave bands above 150 Hz. At low lung volumes, this was consistently observed at the RL chest but only in two subjects at the LL chest and in one subject at the RU chest. It is, therefore, difficult to see how lung volume-related changes in tissue density could have explained the variation in transmitted sound amplitude. The volume signal in our study was derived from flow measured at the mouth, and airway resistance was not known. Furthermore, because sound generation is related to airflow and not to lung volume, there were systematic discontinuities in the ΔA curve when the flow signal became critically low. These factors may explain the observed delay between ΔA and lung volume changes.

More variable findings of volume-dependent amplitude changes were observed at the RU recording site, the only location that had a sufficiently high signal-to-noise ratio in all subjects to allow measurements at frequencies above 300 Hz. It is conceivable that changes in the spatial orientation of larger airways during breathing (11) that are related to individual anatomy could explain the opposite direction of amplitude variation in some subjects. One would have to consider that the position of the surface microphones relative to sound-conducting larger airways would be critical in determining whether volume-dependent variations in amplitude would become apparent and what direction that relationship would show.

The attenuation of sound transmitted through the lungs has been studied by several investigators (4, 17, 19, 20, 28, 30), although fewer studies have focused on the propagation speed of sound through airways and lung tissue. Propagation speed is typically measured by the phase delay of sound from a reference site at the trachea to various locations on the chest surface. Lung volume and thus lung tissue density have an effect on sound propagation speed (32), i.e., sound travels slower through lungs with greater tissue density. For sound from 125 to 500 Hz, Kraman (15) found a propagation time in healthy adults of 2 ms between trachea and upper chest and of 5 ms to the lower chest. Rice and Rice (32) and Wodicka et al. (34) found somewhat shorter propagation times over similar distances. Some of the observed differences may be explained by the dependence of propagation pathways on the sound frequency, i.e., sound at higher frequencies arrives faster at the chest surface because of a longer travel distance within the airways rather than parenchyma (21).

Rice (31) made measurements of sound propagation over distances as short as in our study. However, those observations were obtained from spark sound input at the surface of excised horse lungs with measurement of the propagation speed on the surface at various distances from the input site. It is difficult to compare those results with the in vivo measurements from our study subjects. The phase delay variations in our study were generally <1 ms with a distance of 33 mm between the sensors. Rice also observed propagation speeds of <1 ms over similar distances, but a direct comparison with his findings is not possible since the source of lung sounds in our subjects was not known.

The most consistent findings of volume-dependent variation in the phase difference of lung sounds were observed at the lower lobes. All subjects who met the inclusion criterion of reproducible findings on 2 consecutive days showed an increasingly later arrival of lung sounds at the caudal sensor with increasing lung volume. If the average propagation speed of lung sounds is the same on the path from the source to the surface sensors, the finding of volume-dependent variations in phase would suggest a changing location of the source(s) during the respiratory cycle. The spatial orientation of the bronchial tree during the respiratory cycle has been modeled and shows greater changes at the lower lobes (11). A pair of sensors would have to be in
a certain position relative to large airways from where lung sound originates to register volume-dependent variations of phase.

It is also possible that the sound propagation speed differs on the paths to both sensors, i.e., with increasing volume during inspiration the tissue density may decrease faster on the path to the cranial compared to the caudal sensor when airway closure affects the more dependent portions of the lung. This would fit with the more common finding of phase changes at the lower lobes during breathing at low lung volume. However, the magnitude of phase changes would exceed the expected variations, given that the volume change was only \( \sim 25\% \) of the total lung capacity (31). Furthermore, the reversal of phase difference that was observed during the respiratory cycle in four of five subjects would not be explained by relative changes of tissue density on the propagation paths to the surface sensors.

To explain the observed reversal of phase difference, i.e., sound reaching the caudal sensor slightly earlier than the cranial sensor at the lowest lung volume and the cranial sensor before the caudal sensor at the highest volume, one has to assume a change in the position of the predominant sound source(s) relative to the recording sites. The origin of normal lung sounds is still not entirely clear, but several studies have indicated that lung sounds are generated in large- to medium-size airways, proximal to the sites of physiological airway closure (2), and that the origin of expiratory sounds is more central than that of inspiratory sounds (14). If the sound propagation speed remains essentially constant at the level of the two sensors during the relatively narrow range of target volume, i.e., between 36 and 60% of total lung capacity, our findings would indicate that the sound source(s) were relatively closer to the cranial sensor during inspiration than during expiration. This would agree with acoustical images of the human chest from multisensor recordings where healthy adults showed more peripheral sources of inspiratory sound (13).

The prominence of findings at the lower chest and the absence of volume-dependent phase differences at the RU chest may indicate an effect of the diaphragm on propagation pathways. The presence of the liver below the right diaphragm and the different size and direction of the right and left main bronchi are anatomical factors that may lead to more positive findings at the RL lobe. Also, the spatial orientation of airways relative to the surface sensors could have an effect since passive sound transmission showed some phase difference in synchrony with volume changes, albeit in several subjects. The finding that volume-dependent phase variations between horizontally positioned sensors can occur, although they are much less common, also supports the concept of airway spatial orientation as a factor contributing to our observations.

In summary, our findings confirm spatial differences in lung sound phase and amplitude relative to lung volume. Similar variations of phase and amplitude within narrow areas were also present on passive sound transmission. Varying lung sound generation is, therefore, less likely an explanation of our findings than effects of sound transmission. The consistency of positive and negative observations within study subjects and the predominance of findings at the lower lobes suggest that individual anatomy may have had a significant influence.

The mechanisms behind volume-dependent phase and amplitude differences of lung sounds over short distances are complex and need further exploration with physical models. Visualizing the changes in airway geometry and regional ventilation during sound recording at multiple sites on the chest surface should follow, but this still poses a significant technical challenge. However, lung volume-related variations of respiratory sounds illustrate a diagnostic potential of chest surface acoustic mapping that will become more apparent as our understanding of respiratory acoustics advances.

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REFERENCES


