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CHEST

Original Research

LUNG SOUNDS

Effect of Body Position on Lung Sounds in Healthy Young Men*

José Antonio Fiz, MD; January Gnitecki, BscEE; Steve S. Kraman, MD; George R. Wodicka, PhD; and Hans Pasterkamp, MD

Background: **The effect of body position on the generation of abnormal respiratory sounds (*eg*, snoring and stridor) is well recognized. Postural effects on normal lung sounds have been studied in less detail but need to be clarified if respiratory acoustic measurements are to be used effectively in clinical practice.**

Methods: **Lung sounds and airflow were recorded in six healthy male subjects. Two acoustic sensors were placed over corresponding sites of the right and left chest, first anteriorly and then on the back. Subjects were studied in sitting, supine, prone, and lateral decubitus positions. Lung sound intensity (LSI) was determined at flows of 0.4 to 0.6 L/s and 0.8 to 1.2 L/s within frequency bands of 150 to 300 Hz and 300 to 600 Hz.**

Results: **LSI was greater over the dependent lungs in the lateral decubitus positions. In the sitting position, LSI was greater on the left compared with the right posterior lung at the same airflow within the same frequency bands. Compared with sitting, neither the supine nor prone positions caused a significant change in LSI.**

Conclusions: **Our study confirms previously reported asymmetries of normal lung sounds. The insignificant change of flow-specific LSI between the upright and horizontal positions in healthy subjects is encouraging for the clinical use of respiratory acoustic measurements. Further studies should address postural effects on lung sounds in patients with acute lung injury and other lung pathologies.** ***(CHEST 2008; 133:729–736)***

Key words: posture; prone position; respiratory mechanics; respiratory sounds; supine position

Abbreviations: dB = decibel; FRC = functional residual capacity; LSI = lung sound intensity

Body position, or posture, has a significant effect on airway dimensions. The generation of respiratory sounds from airflow is affected by airway dimensions, and respiratory sound measurements could therefore become sensitive indicators of airway changes.

Snoring is the most extensively studied respiratory sound for which posture has a significant effect. Changes in airway configuration and neuromuscular activation during sleep are generally viewed as the key factors that lead to snoring.[1] The pharyngeal cross-sectional area decreases in the supine position compared with the upright position in healthy humans[2] and in patients with obstructive sleep apnea.[3] Even in the absence of snoring, this decrease can result in greater flow-turbulent noise over the trachea in the supine position, particularly in patients with obstructive sleep apnea.[4]

Other abnormal or adventitious respiratory sounds may also become apparent or enhanced in the recumbent positions. Stridor in children with laryngomalacia is typically more prominent when they are supine compared with prone.[5] Wheezing in patients with COPD may be triggered by changing from the sitting to the supine position.[6] A decrease in functional residual capacity (FRC) and airway diameters in the supine position is thought to be partially responsible for nocturnal symptoms of asthma.[7] Reduced FRC and increased closing volume in the supine position are also considered as mechanisms for posturally induced crackles in cardiac patients,[8] for whom they can be of prognostic significance.[9] In patients with pneumonia, inspiratory crackles may appear over the affected lung only in the lateral decubitus position.[10]

Effects of body position on normal respiratory sounds have attracted less attention and have been studied mostly in relation to changes in regional ventilation. In healthy adults, the rate of increase in lung sound intensity did not change significantly between dependent and nondependent lung zones in upright, head-down, and lateral decubitus positions, suggesting that airway closure did not affect sound generation.[11] In another study[12] of healthy adults, the functional relation between airflow and lung sound intensity did not change when subjects changed from sitting to the lateral decubitus position, although posture decreased the gain of the sound amplitude in the left lateral position. Comparing bilateral lung sound intensity in the lateral decubitus positions, greater intensity has been found over the dependent lung.[13]

Positioning patients to enhance airway clearance and to expand atelectatic areas of the lung is well established in chest physiotherapy. Placing patients in the prone position is often tried in patients with acute severe lung injury to improve ventilation and gas exchange in critical care.[14] Pronation may also improve oxygenation and lung mechanics in patients with COPD[15] and in premature and newborn infants receiving ventilation,[16] although healthy infants have reduced lung volume and increased upper airway resistance in the prone compared with the supine position.[17]

Lung sounds in the prone position have not been studied in comparison with other body positions that are commonly used clinically. A shift in lung parenchyma and a change in the vertical distribution of lung tissue between supine and prone positions may be the major events in healthy humans, with changes in ventilation and perfusion much less significant.[18] While pulmonary perfusion is more homogeneous in the prone position,[19] the distribution of ventilation in the prone position appears to be similar to the supine position in healthy humans.[20] However, there is a substantial decrease in FRC from sitting to supine, while that decrease is significantly less when changing from sitting to the prone or lateral decubitus positions.[21] Based on this information, we did not predict a major change in flow-specific lung sound intensities between upright and horizontal positions. The objective of our study was to record lung sounds under standardized conditions of body position and airflow in healthy subjects and to compare lung sound intensities in different frequency bands.

Materials and Methods

Six healthy male subjects were recruited from coworkers and friends (Table 1). All were lifetime nonsmokers, and none had significant past or present respiratory disease. The subjects had not had a respiratory infection within the preceding 6 weeks and were not receiving any medications at the time of study. Findings of chest auscultation before the recordings were normal. All subjects gave informed consent to participate. The study was approved by the Health Research Ethics Board of the University of Manitoba.

At baseline, lung function was measured by spirometry (Spirovit SP-10; Schiller America; Miami, FL). Measurements were obtained according to established guidelines[22] and were referenced to normative data.[23] Respiratory sounds were recorded with two contact sensors (EMT 25C; Siemens Canada; Mississauga, ON, Canada) that were attached with double-sided adhesive discs (3M Health Care; St. Paul, MN). Respiratory airflow was recorded with a pneumotachograph (Fleisch No.3; Phipps and Bird; Richmond, VA) and pressure transducer (Validyne Engineering Corporation; Northridge, CA). Flow and sound signals were amplified and filtered (DA100C; Biopac Systems, Harvard Apparatus Canada; Saint Laurent, QC, Canada) before analog-to-digital conversion (DAQ card 1200; National Instruments; Austin, TX) and acquisition by computer (Lifebook Notebook; Fujitsu Computer Systems; Sunnyvale, CA). The band-pass filter for respiratory sounds was set with cut-off frequencies of 0.05 Hz and 5,000 Hz, and the amplification factor was 200. The digitization rate was 10,240 samples per second with 12-bit resolution. The flow signal was subsequently decimated to 320 samples per second.

A total of 10 data sets were obtained from each subject. The sequence of recordings is shown in Table 2. Each recording was at least 60 s in duration. The subjects wore a nose clip and breathed through the mouthpiece of the pneumotachograph. Each patient was coached to increase airflow from shallow breathing to a maximum reaching 1.5 to 2.0 L/s, then decreasing to finish with a breath hold of at least 5 s. The sensors were removed and reapplied only for the change from anterior to posterior recording sites. For the last two recordings, only one sensor was moved (Table 2).

We used custom-developed software for data acquisition, analysis, and display (R.A.L.E.; PixSoft; Winnipeg, MB, Canada). After each recording, good signal quality was verified by visual and auditory examination before storing the data. Segments with artifacts were marked for exclusion from the calculation of power spectra. The sound signals were parsed into segments of 2,048 data samples with 50% overlap of samples between successive segments. Each segment was windowed with a Hanning function before power spectral estimates were obtained by fast Fourier transformation. Average sound power in two frequency bands (150 to 300 Hz and 300 to 600 Hz) was estimated for two ranges of airflow (0.4 to 0.6 L/s and 0.8 to 1.2 L/s). Statistical analysis of differences in lung sound intensity (LSI) between body positions, airflow ranges, and sensor locations was performed with paired Student t test, and $p < 0.05$ was considered significant.

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Table 1—*Subject Characteristics**

Subject No.	Race	Age, yr	Height, cm	Weight, kg	Body Mass Index, kg/m^2	FVC, L	FVC, % of Predicted	FEV_1, L	FEV_1, % of Predicted
1	White	20.2	174	84	27.7	5.62	104	4.77	108
2	Asian	31.4	170	68	23.5	4.70	113	3.93	116
3	White	19.4	184	77	22.7	6.45	108	4.97	104
4	White	16.0	195	76	20.0	5.33	78	4.36	76
5	White	19.0	175	79	25.8	5.14	94	3.95	88
6	White	21.0	171	70	23.9	6.17	119	5.11	120
Mean		21.2	178	76	24.0	5.57	103	4.52	102
SD		4.8	8.8	5.4	2.7	0.6	13.5	0.5	15.5

*Percentage of predicted normal values are from Morris et al.[23] FEV_1/FVC ratio was normal (82%) in tall and slim subject 4, who was healthy but whose FVC and FEV_1 were slightly below predicted.

Results

The results of baseline spirometry are presented in Table 1. Figure 1 shows LSI (in decibels [dB]) at the posterior recording sites in the sitting position. LSI during expiration was close to the background noise level (*ie*, the sound intensity during breath hold). Consequently, only lung sounds during inspiration were used in the further analyses.

LSI over the posterior recording sites in the prone and sitting positions is shown in Figure 2. Differences on either side were not significant between prone and sitting. However, LSI in the prone position was greater on the left than on the right side in both frequency bands ($p < 0.05$) and at both airflows ($p < 0.05$). In the sitting position, LSI was significantly greater on the left side only at lower airflow (Fig 2). At the anterior recording sites, LSI was significantly greater on the left side only at higher airflow and lower frequencies (Fig 3). This difference disappeared in the supine position, a postural effect that did not quite reach statistical significance ($p = 0.06$). Overall, the differences on either side between sitting and supine positions were not significant. Also, the LSI at anterior and posterior recording sites on the same sides was not significantly different within the same range of airflow and frequency band.

LSI in the left and right lateral decubitus positions is shown in Figure 4. At the posterior recording sites and in the left lateral decubitus position, LSI was significantly greater over the dependent lung (*ie*, on the left side) in both frequency bands and both airflow ranges. In the right lateral decubitus position, LSI over the dependent lung was significantly greater only at the higher flows and higher frequencies. At the anterior recording sites and in the left lateral decubitus position, LSI was significantly greater on the left only in the 150- to 300-Hz band at high flows (32.4 ± 3.1 dB vs 28.1 ± 2.3 dB on the right; $p < 0.05$) [mean ± SD]. There was no significant change in the flow-sound relation of tracheal sounds between sitting and prone positioning (Fig 5).

Table 2—*Recording Sequence**

Body Positions	Recording	Sensor 1	Sensor 2
Sitting	Anterior	RUL	LUL
Supine	Anterior	RUL	LUL
Left lateral decubitus	Anterior	RUL	LUL
Right lateral decubitus	Anterior	RUL	LUL
Sitting	Posterior	RLL	LLL
Prone	Posterior	RLL	LLL
Left lateral decubitus	Posterior	RLL	LLL
Right lateral decubitus	Posterior	RLL	LLL
Sitting	Posterior	RLL	TRA
Prone	Posterior	RLL	TRA

*RUL = right upper lobe; LUL = left upper lobe; RLL = right lower lobe; LLL = left lower lobe; TRA = trachea.

Discussion

Significant postural effects on lung sounds were found only in the lateral decubitus positions, in which greater sound intensity was observed over the dependent lung. Similar observations of lung sounds in the lateral decubitus position were reported by Jones et al,[13] who found greater sound intensity over the left lung in the dependent position. In their healthy young adult subjects, LSI over the right lung in the dependent position was greater than on the left side only during expiration. We analyzed sounds in the typical tidal airflow range at approximately 0.5 L/s and at modestly increased flows around 1 L/s, while their analysis focused on sounds at higher flows at approximately 2 L/s. Expiratory sounds are well known to have significantly lower intensity than inspiratory sounds at isoflows.[24] At the relatively low flows in our study, expiratory sounds had insufficient signal-to-noise ratio to be considered in the further analyses. However, a lesser effect of the lateral

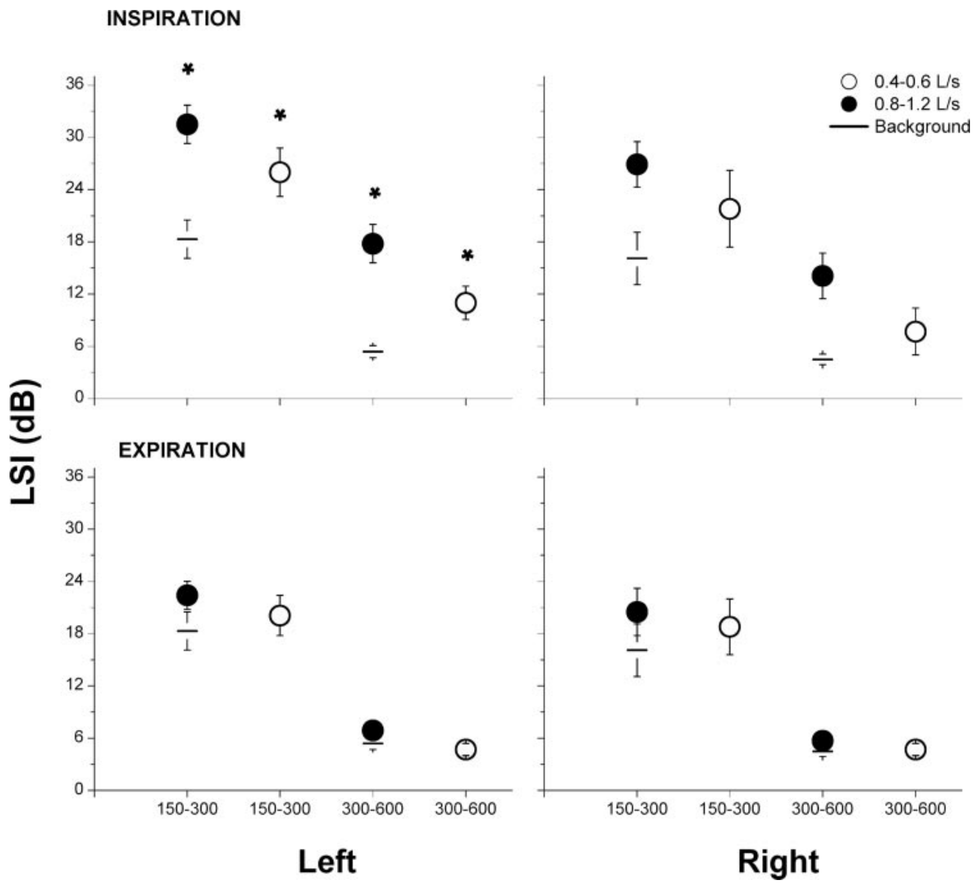

FIGURE 1. LSI during inspiration (*top panels*) and expiration (*bottom panels*) over the right and left posterior lower lung within the tidal airflow range (0.4 to 0.6 L/s) and at moderately increased flows (0.8 to 1.2 L/s). Mean values (± SD) are shown for two frequency bands. The average background noise level during breath hold is plotted as horizontal lines. Asterisks indicate statistically significant differences ($p < 0.05$, paired *t* test) between left and right sides at the same flow in the same frequency band.

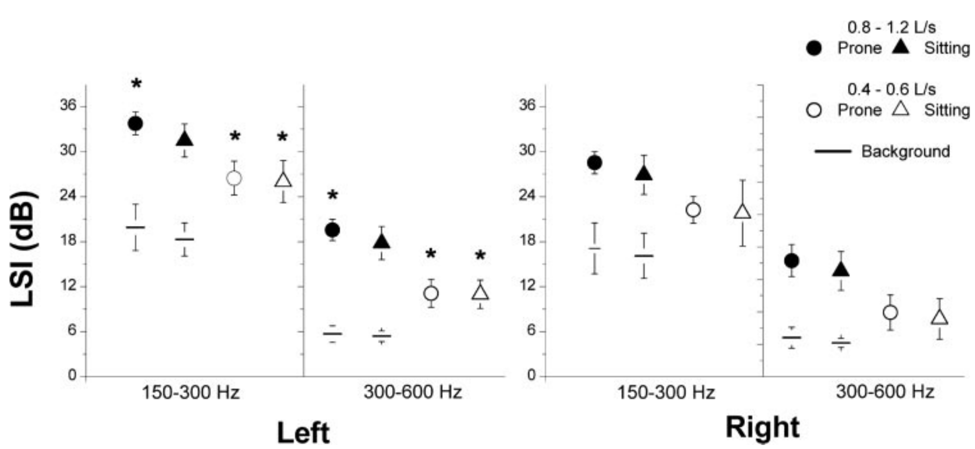

FIGURE 2. LSI in the sitting and prone position over the posterior right lower lung (*right panel*) and left lower lung (*left panel*). Differences between sitting and prone were not significant at given airflows and within given frequency ranges. Asterisks indicate significant differences between left and right sides in the same position, same flow, and same frequency band.

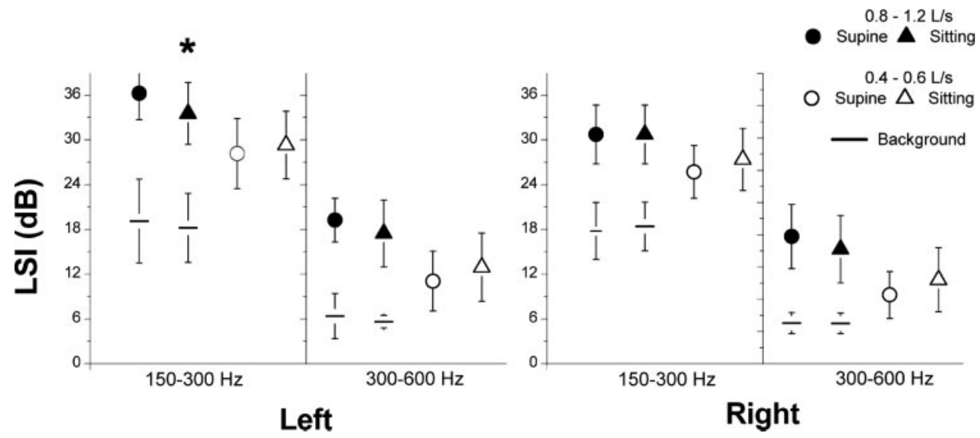

FIGURE 3. LSI in the sitting and supine positions over the anterior right upper lung (*right panel*) and left upper lung (*left panel*). Differences between sitting and supine were not significant at given airflows and within given frequency ranges. Compared to the right chest, sounds on the left had greater intensity at lower frequencies, likely due to heart sounds.

decubitus position on inspiratory LSI over the right dependent lung was also seen in our study. The right hemidiaphragm was likely elevated due to the liver in this position, and greater volume changes than those reached by our subjects may have been needed to increase the difference of LSI compared with the left side. Furthermore, a lesser excursion of the right hemidiaphragm in the left compared with the right lateral decubitus positions has been found on dynamic MRI of normal subjects.[25] If this was the case in our subjects, it could have contributed to the difference of LSI in the left lateral decubitus position.

Consistent findings of asymmetries in normal lung sounds are now well recognized even if "equal breath sounds bilaterally" remains a standard clinical description of normality. In the sitting position, LSI over the left posterior lower chest was greater than on the right, confirming earlier reports.[13,24,26] This

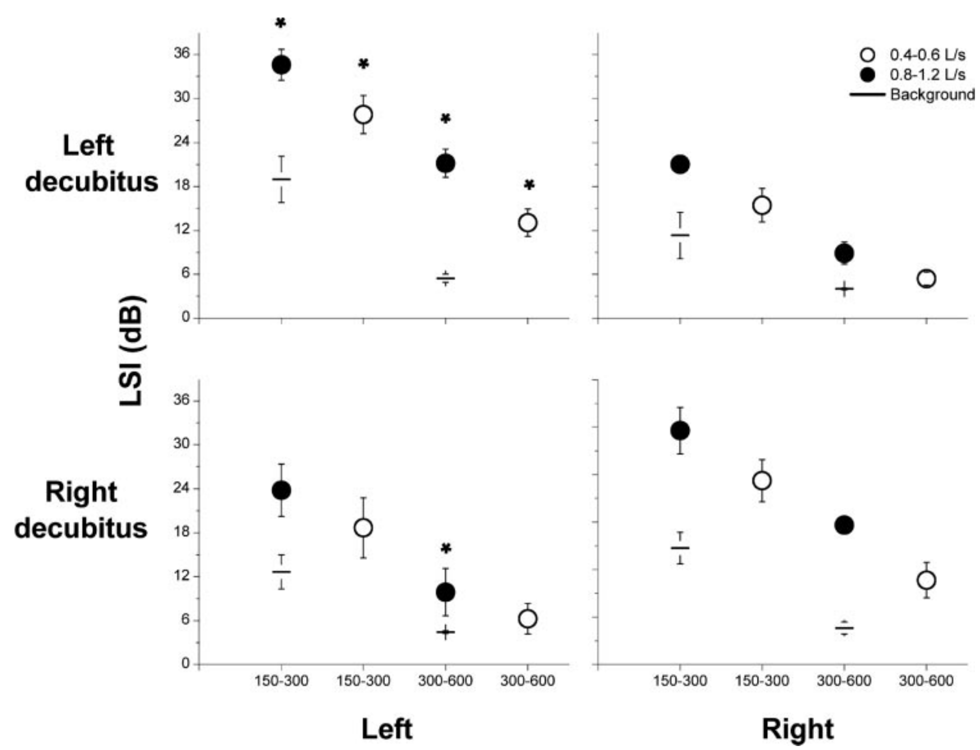

FIGURE 4. LSI in the lateral decubitus position over the posterior right and left lower lung. *Top panel*: Left lying position. *Bottom panel*: Right side dependent.

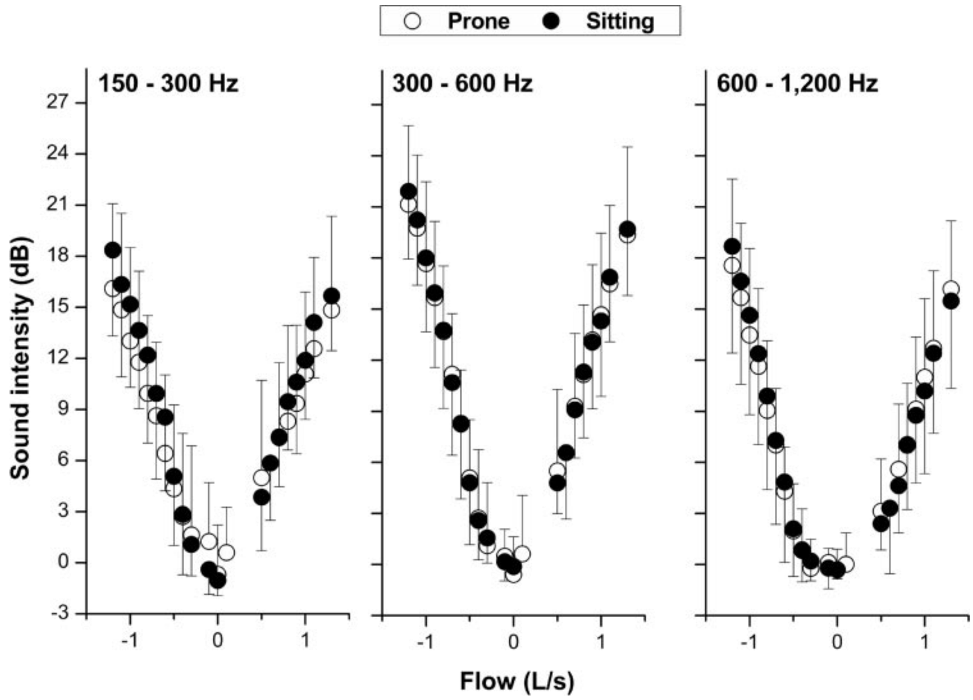

FIGURE 5. Intensity (power) of respiratory sounds at the trachea above the suprasternal notch at 150 to 300 Hz (*left panel*), 300 to 600 Hz (*center panel*), and also for higher frequencies (600 to 1,200 Hz, *right panel*). Inspiratory flows are plotted with positive and expiratory flows with negative values.

observation has been considered in relation to the different size and angulation of major bronchi on the left which could affect the generation of lung sounds. Passive transmission of noise introduced at the mouth also reveals asymmetries, with a trend toward left over right dominance at the posterior lower chest,[26] while the reverse is observed over the anterior upper lungs.[26,27] LSI at the anterior upper chest in our subjects was not greater on the right during inspiration. In fact, it was increased on the left at low frequencies, possibly because of heart sounds extending into this frequency range (we did not apply techniques to reduce the influence of heart sounds in this study[28]). The right-sided dominance of LSI over the anterior upper lungs is observed mostly during expiration anyway, and expiratory sounds were excluded in our study as mentioned above.

Subject 1 in our study was slightly overweight but otherwise healthy (Table 1). Body weight affects airway caliber, and increased wheezing in obesity may be due to reduced lung volumes and airway narrowing.[29] In normal subjects, however, the thickness of the subcutaneous fat layer appears to have little effect on the intensity of normal lung sounds.[30] Weight reduction in obese subjects has been shown to increase FRC and decrease airway resistance in the upright position.[31] It would be interesting to document flow-specific LSI during weight loss and in relation to pulmonary mechanics and body position.

Neither the supine nor the prone positions caused significant changes in LSI compared with the subjects sitting, even though one would have expected decreased compliance and increased flow resistance in the supine position.[32] While adventitious lung sounds (*eg*, crackles and wheezing) have been studied in recumbent subjects, normal lung sounds have not been compared under standardized conditions of airflow. Measurements of flow-specific LSI may be of value to detect bronchial constriction and dilation. Tracheal sound intensity has been found to increase with bronchial constriction by some[33] but not others,[34] while LSI at the chest has been more consistently shown to decrease during bronchial narrowing.[34,35] In young children, respiratory sound recordings will usually be made at the chest while they are held in upright or recumbent positions. Our observation of insignificant changes in the flow-sound relation between sitting and supine positions is therefore relevant and reassuring. Recent studies show that ventilation and perfusion per alveolus change relatively little with changes in posture[18] while lung density increases in the dependent parts of the lung in the supine position, analogous to the “Slinky” effect.[36] There is increased small airway closure in recumbent subjects,[32] but this would not be expected to have an effect on the generation of lung sounds.[11] Tracheal sound intensity may change in the horizontal position, especially in recumbent patients with obstructive sleep apnea,[4] but we did not find a significant change of tracheal sound intensity between sitting and prone positions in our healthy subjects.

Lung sounds in the prone position have not been previously reported even though "proning" has attracted much interest as a simple and safe method to increase lung volume and alveolar inflation to homogenize the distribution of ventilation and to improve oxygenation in patients with acute lung injury.[37] It would have been interesting but technically very challenging to compare simultaneously recorded lung sounds over anterior-posterior sites in the horizontal positions. Aside from the discomfort of lying on the sensors, a greatly increased mechanical load on the accelerometers would have changed their response in a way that would have been problematic to standardize. Using a larger array of sensors[38] may have uncovered regional changes in LSI that were not apparent in our recordings. However, single- or dual-sensor recordings are still more feasible in the clinical setting, although computerized multichannel sound analysis is gaining attention.[39,40]

Acoustic imaging of the human chest is emerging from the research domain to applications in clinical medicine. Measuring the intensity of normal lung sounds in their relation to airflow offers information beyond the limits of clinical auscultation. Whether infants are monitored for their response to treatment of bronchiolitis[41] or changes in pneumonia are detected with arrays of acoustic sensors,[42] patients are often not in an upright position during the data acquisition. Postural changes in lung mechanics tend to be small in healthy subjects,[43] and the findings in our group of healthy young men suggest that body position is also not a major variable in the measurement of LSI. Further studies need to extend these observations to patients with lung diseases.

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