

Sound transmission in the lung as a function of lung volume

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Bergstresser, T., D. Ofengeim, A. Vyshedskiy, J. Shane, and R. Murphy. Sound transmission in the lung as a function of lung volume. *J Appl Physiol* 93: 667–674, 2002. First published April 26, 2002; 10.1152/jappphysiol.00050.2002.—We were interested in how the transmission of sound through the lung was affected by varying air content in intact humans as a method of monitoring tissue properties noninvasively. To study this, we developed a method of measuring transthoracic sound transit time accurately. We introduced a “coded” sound at the mouth and measured the transit time at multiple microphones placed over the chest wall by using a 16-channel lung sound analyzer (Stethographics). We used a microphone placed over the neck near the trachea as our reference and utilized cross-correlation analysis to calculate the transit times. The use of the coded sound, composed of a mix of frequencies from 130 to 150 Hz, greatly reduced the ambiguity of the cross-correlation function. The measured transit time varied from 1 ms at the central locations to 5 ms at the lung bases. Our results also indicated that transit time at all locations decreased with increasing lung volume. We found that these results can be described in terms of a model in which sound transmission through the lung is treated as a combination of free-space propagation through the trachea and a propagation through a two-phase system in the parenchyma.

sound recording; lung sound; speed of sound; sound speed

THERE ARE CERTAIN CONDITIONS that affect the lung, such as pulmonary edema, in which the fluid content increases as the disease progresses, and therefore the tissue properties are likely to change in proportion to the severity of the illness. The speed of sound in the lung is affected by tissue properties (14). Accordingly, measurement of this speed offers the potential of providing clinically useful information. It has been difficult to measure transpulmonary sound speed accurately because it has not been easy to separate the precise arrival of the signal from the background noise (5, 6). We developed a signal with a coded signature that allows accurate detection of the arrival time. We used this sound signature to test the hypothesis that sound speed changes with the volume of air in the lung.

The experimental results are interpreted in terms of a theoretical model in which the sound propagation through lung parenchyma is treated as a compres-

sional wave in a homogeneous two-phase system. That is, because the wavelength is expected to be large compared with the alveolar dimensions, we simulated the sound transmission with the assumption that the lung consists of a homogeneous mixture of gas (humid air) and soft tissue phases. This simple model yields an expression for the sound speed as a function of the volume fraction of air in the lungs. We compared that volume dependence with experimental results.

MATERIALS AND METHODS

Subjects were examined with a 16-channel lung sound analyzer (Stethographics model 1602, www.stethographics.com). The system (STG) uses miniature electret microphones (model 3301-0, Gentex) mounted in commercially available stethoscope chest pieces to amplify, filter, and multiplex lung sounds to an analog-to-digital converter (model DAS-1801ST, Keithley) and then stores the data on a computer. A custom-designed circuit board is used for the amplification and filtering of each channel by employing a one-pole high-pass and low-pass filter with cutoff frequencies of 80 and 2,000 Hz, respectively.

For ease of application, 14 microphones were incorporated into a soft foam microphone pad as shown in Fig. 1. The microphone pad was positioned on a stretcher or a plastic reclining chair positioned at a 45° angle. Subjects were instructed to lie in a recumbent position on the microphone pad. One microphone was used to record sound at the trachea.

The STG software played the prerecorded sound through a speaker (diameter = 57 mm, resistance = 8 Ω, 0.5 W; model P10177-ND, Panasonic) mounted in a conical plastic chamber that easily fit into a subject's palm. A disposable mouthpiece (length = 60 mm, diameter = 30 mm) conducted sound from the speaker chamber to the mouth. A plastic straw was attached to the side of a mouth piece. To avoid glottis closure, subjects were instructed to hold the speaker chamber next to the mouth, to take a deep breath, close their lips around the mouthpiece, and expire slowly through the plastic straw. The average expiration time was 14 ± 4 s.

Subjects

Twelve normal subjects entered into this study and were volunteers who had no history of lung disease. Before the recording was made, verbal consent was obtained from every subject. Ten subjects were men, and 2 were women. Average age was 45 ± 15 yr, and range was 19–69 yr. Average height

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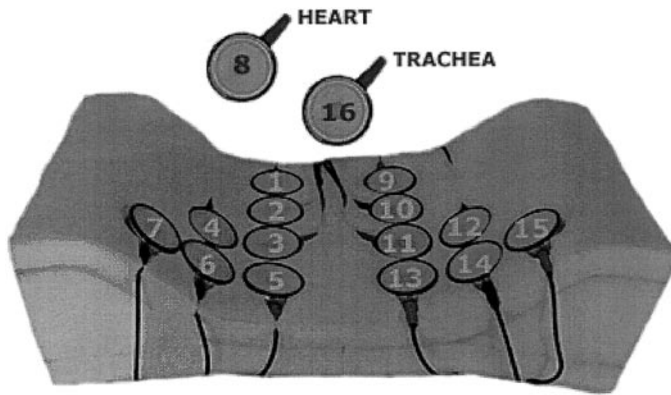


Fig. 1. This figure illustrates the 14 microphones incorporated into a soft foam microphone pad. One microphone was used to record sound at the trachea.

was 173 ± 10 cm with a range of 150–182 cm. Average chest circumference was 100 ± 14 cm with a range of 70–110 cm.

Input Sound

Because sharp autocorrelation function allows unambiguous peak detection, we experimented with a number of sounds with the goal of finding one with a sharp autocorrelation and minimal attenuation. We noted that, at frequencies of <100 Hz, sound emitted from small speakers becomes distorted and that transit time decreases with increasing frequency, presumably because sound travels further through large airways before switching to lung parenchyma. In addition, we have observed strong destructive interference, at some frequencies more than at others, probably due to multiple sound transmission paths. Minimal interference was observed at frequencies of ~ 150 Hz.

Autocorrelation of the monophonic sounds we studied did not exhibit a sharp peak in an envelope. An example of this

is presented in Fig. 2A. We noted that both polyphonic sounds with broader frequency content exhibited sharper autocorrelation peaks (Fig. 2, B and C). We then examined the attenuation of mono- and polyphonic sounds through the lungs. We found that monophonic, 150-Hz sound was transmitted with little change in the appearance of its waveform. Polyphonic sounds were partially attenuated (Fig. 2, B and C). The best data from input sound in terms of the trade-off between yielding sharp autocorrelation function and unattenuated sound transmission are presented in Fig. 2B. This sound was used in the remaining studies. The sound contained 11 cycles. The cycle frequencies were varied in the following pattern (in Hz, *cycles 1 through 11*): 130, 130, 140, 140, 150, 150, 150, 140, 140, 130, 130. The sound was played in a loop every 250 ms, yielding 40 transmission “images” over 10 s of the expiratory maneuver. This input sound did not change over time, as indicated by the sound recorded at the trachea.

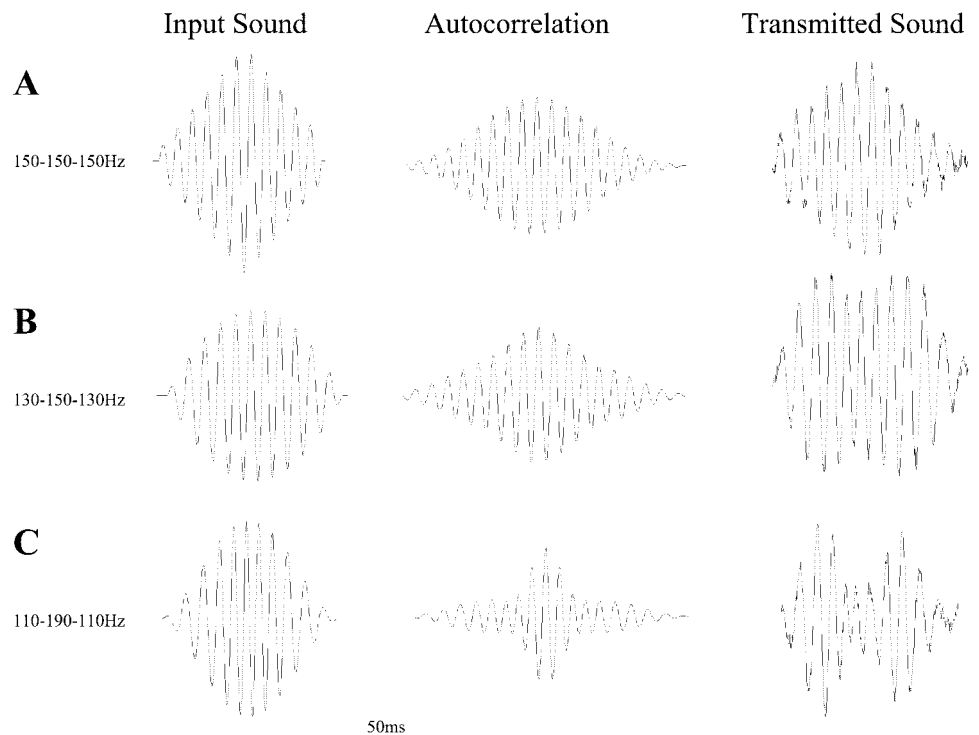
We tested for transmission of sound in the room and also examined the results with the glottis open and closed. No detectable sound was transmitted through the room (Fig. 3A). The sound input while the subject slowly expired from vital capacity to residual volume resulted in a strong signal at all microphones consistent with the hypothesis that this maneuver kept the glottis open (Fig. 3B). Closing the glottis by a Valsalva maneuver prevented most sound transmission to the bases (Fig. 3C).

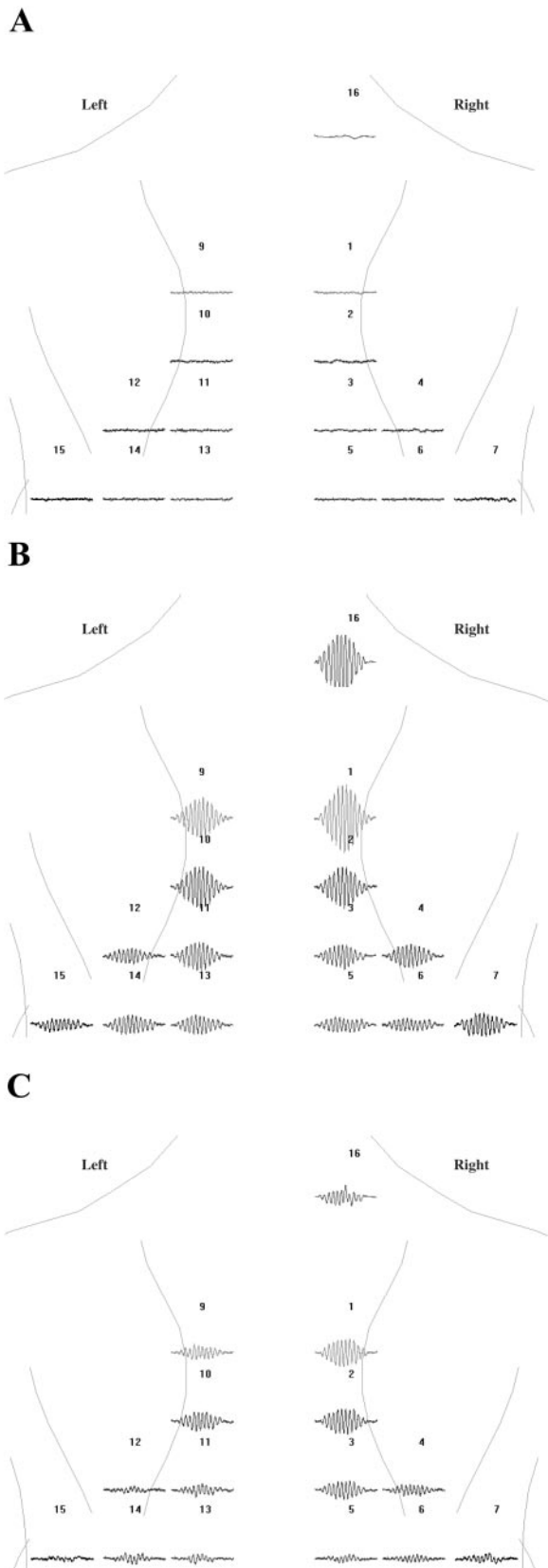
RESULTS

Transit Time Analysis

To study transit time through the lung, we injected polyphonic sound with frequencies of 130, 140, and 150 Hz, as described above, into 12 normal subjects through the mouth and measured arrival time difference between the tracheal microphone and 14 sites on the chest. Transit time analysis of all the data de-

Fig. 2. Autocorrelation and transmission of three different input sounds. A: monophonic 150-Hz sound. B: polyphonic sound made of 11 cycles in which cycle frequencies were varied in the following pattern (in Hz, *cycles 1 through 11*): 130, 130, 140, 140, 150, 150, 150, 140, 140, 130, and 130. C: polyphonic sound made of 11 cycles in which cycle frequencies were varied in the following pattern (in Hz, *cycles 1 through 11*): 110, 130, 150, 170, 190, 190, 190, 170, 150, 130, and 110. Transmitted sounds were recorded at *channel 5* (right base) at 80% of vital capacity and digitally band-pass filtered from 80 to 2,000Hz. The larger frequency spread sharpens the autocorrelation peak and attenuates the transmitted sound.





scribed here was performed with the use of a cross-correlation technique with further verification obtained by approximating the interval between the corresponding peaks. Figure 4A shows the results of the transit time analysis at the right upper posterior chest (*channel 2*) at functional residual capacity. The sound recorded at the trachea is shown on *top*, and the sound recorded at *channel 2* is shown on *bottom*. With the use of the time interval between corresponding peaks of two sounds to yield an approximation of transit time, the sound recorded from *channel 2* is delayed by 3 ms compared with the tracheal sound.

In Fig. 4B, the transit time analysis was refined by cross correlating the two sounds. The cross-correlation shows a clear peak (arrow) corresponding to an arrival time difference of 3 ms between the trachea and the *channel 2*. The correlation coefficient at the peak of the cross-correlation function was 0.98. We validated this method by testing the speed of sound in room air. The average of three measurements was 338 ± 8 m/s.

Figure 4C shows the arrival time difference between the trachea and all of the 14 chest sites. Circle size in the diagram is proportional to the transit time. Numbers indicate arrival time difference in milliseconds. There was a progressive increase of transit time from the microphones on the apical sites to the microphones over the basal sites. Transit time varied over the chest from 1 ms at the central sites to 5 ms at peripheral sites.

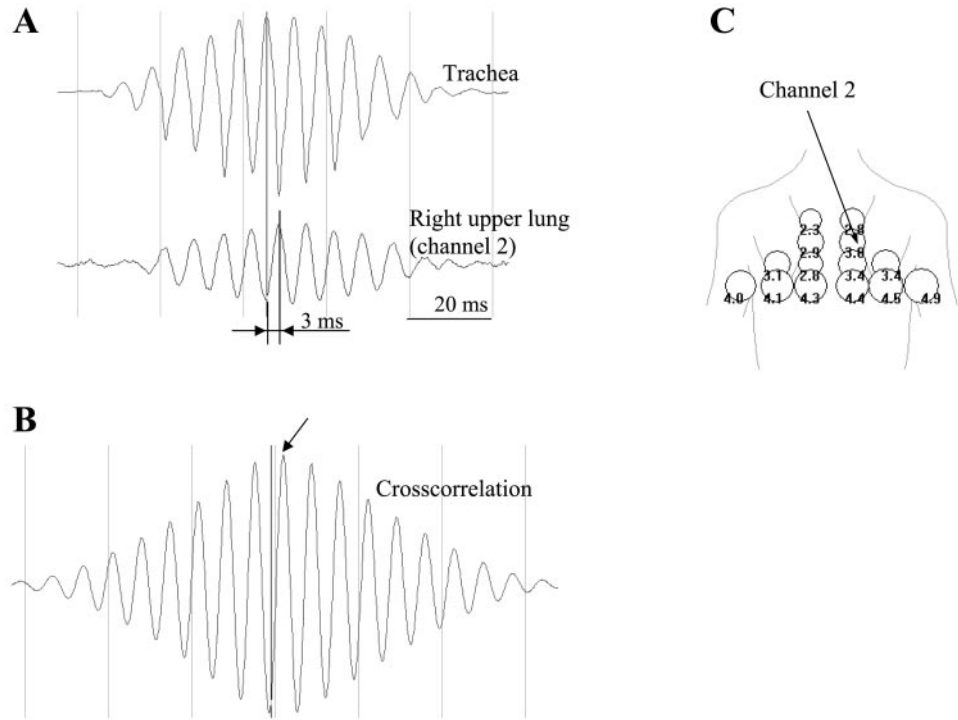
We used the increase of transit time from the central location to the periphery to approximate the speed of sound in the lung parenchyma. We assumed that sound travels in the trachea at a speed of 340 m/s to the carina and then jumps to the parenchyma. The distance between the trachea and carina is ~ 15 cm. Accordingly, we calculated that it takes 0.4 ms for the sound to travel from the tracheal microphone to the carina. Similarly, we calculated that the 4-ms transit time between the trachea and the right lower lateral chest (*channel 7*), a distance of ~ 15 cm, corresponds to a speed of sound of 37 m/s in the lung parenchyma.

Relationship of Transit Time to Lung Volume

Transit time was shorter the bigger the lung volume, as illustrated in a single patient (Fig. 5) and the entire group (Fig. 6). Figure 5 shows the transit time between the tracheal and chest microphones in one patient. In this patient, 75 sound signatures were analyzed during 17 s of constant flow during expiration from vital capacity to residual volume. For every recording microphone, the transit time in milliseconds is shown as a function of the approximate percentage of vital capacity. At all lung volumes, the transit time was minimal at the central channels (*channels 2, 3, 10, and 11*). The

Fig. 3. Sound transmission through the lung. A: no detectable sound was transmitted when the speaker played sound into the room air near the subject. B: sound input while the subject slowly expired from vital capacity to residual volume resulted in a strong signal at all microphones (glottis open). C: closing the glottis by a Valsalva maneuver prevented most sound transmission to the bases.

Fig. 4. Transit time analysis was performed with the use of a cross-correlation technique with further verification by approximating the interval between the corresponding peaks. A: transmitted sounds recorded at the trachea (*top*) and at the upper posterior chest (*channel 2, bottom*) at functional residual capacity. The time interval between corresponding peaks of two sounds is 3 ms. B: result of cross-correlation of the two sounds shown in A. The peak (arrow) corresponds to an arrival time difference (transit time) of 3 ms. C: transit time between the trachea and all of the 14 chest sites. Circle size in the diagram is proportional to the transit time. Numbers indicate arrival time difference (in ms).

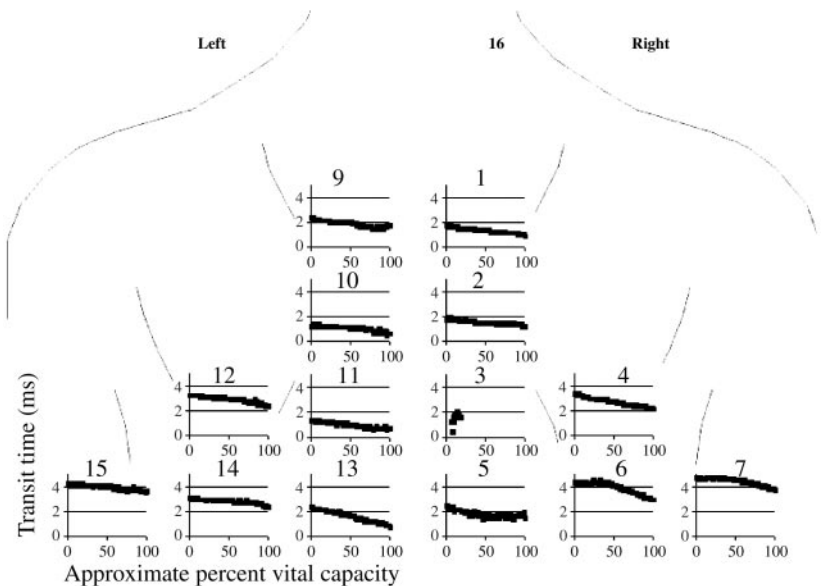


transit time increased gradually to the lung bases (*channels 5, 6, 7, 13, 14, and 15*). This observation is consistent in all subjects (Fig. 6).

In all chest locations, the transit time varied inversely with lung volume (Fig. 5). In the patient presented in Fig. 5, filling the lungs with air from residual volume to vital capacity reduced sound travel time by up to 2 ms. The reduction of transmission time at increased lung volume was also a consistent finding in all subjects examined (Fig. 6). The paired two-sample *t*-test indicated that the reduction of transit time is statistically significant ($P < 0.05$) at all channels ex-

cept *channels 3, 5, and 10*. In addition, at all lung volumes the transit time on the left is shorter than that on the right. This was statistically significant ($P < 0.05$) at all channels except *channels 1 and 9*. The reason for the shorter transit time on the left is not clear. A possible explanation is that sound is transmitted through the heart at a higher speed than through the lung. In this scenario, the sound jumps from the trachea to the heart and propagates through the heart before jumping to the parenchyma. The wavelength of the sound in the trachea, over 2 m at 150 Hz, is large compared with the dimensions of the heart. The trans-

Fig. 5. Transit time between the trachea and all of the 14 chest sites (in ms) shown as a function of percent vital capacity in one normal patient. In this patient, *channel 3* produced unreliable data at large lung volumes.



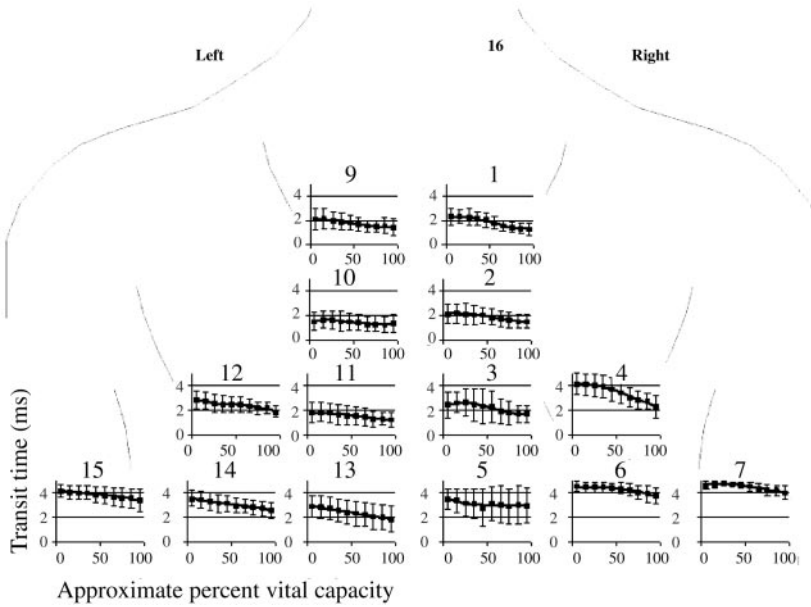


Fig. 6. Transit time between the trachea and all of the 14 chest sites (in ms) shown as a function of percent vital capacity. Data are averaged among 12 normal adult patients. Error bars indicate standard deviations.

mitted wave would be expected to directly traverse the obstruction, which is consistent with this hypothesis. The observation that transit time is not significantly different between *microphones 1* (right) and *9* (left) that are above the heart is also consistent with this hypothesis.

Simulation

Model: sound speed in the two-phase system. To investigate the mechanism of sound transmission through the lung parenchyma, we have developed a model of sound speed in the two-phase system as follows. We start with the general expression for the sound speed (*C*) in a compressional wave

$$C = \sqrt{\frac{B}{\rho}} \tag{1}$$

where *B* is the adiabatic bulk modulus of the homogeneous medium and ρ is the density.

The bulk modulus is just the reciprocal of the compressibility, which can be written in terms of the compressibilities of the air and tissue (*K_A* and *K_T*, respectively)

$$\frac{1}{B} = \nu K_A + (1 - \nu) K_T$$

where $\nu = V_A / (V_A + V_T)$ (*V_A* is volume of air in the lungs, and *V_T* is volume of tissue). We ignore the second term because the air in the alveoli is much more easily compressed than the surrounding tissue. With the use of the standard thermodynamic result for the adiabatic bulk modulus of a gas (*B_A*)

$$B_A = \frac{1}{K_A} = \gamma P$$

where γ is the ratio of heat capacities at constant pressure and constant volume, and *P* is the ambient

pressure. Accordingly, we replace the bulk modulus in *Eq. 1* with

$$B = \frac{1}{\nu K_A} = \frac{1}{\nu} B_A = \frac{1}{\nu} \gamma P \tag{2}$$

We also express the density in terms of the densities of the air (ρ_A) and tissue (ρ_T)

$$\rho = \nu \rho_A + (1 - \nu) \rho_T$$

Here, we ignore the first term because the density of air is much less than the density of the tissue. Inserting this result and *Eq. 2* into *Eq. 1*, we arrive at a result that has been derived in various forms by others (8, 9, 11)

$$C = \frac{1}{\sqrt{\nu(1 - \nu)}} \times \sqrt{\frac{B_A}{\rho_T}} \tag{3}$$

or

$$C = \frac{1}{\sqrt{\nu(1 - \nu)}} \times \sqrt{\frac{\rho_A}{\rho_T}} \times C_A \tag{4}$$

where *C_A* is the sound speed in air. These expressions were used to compare the volume dependence of the experimental results with the model.

Comparison of Model Prediction and Experimental Results

In our model, it was assumed that the sound travels in the trachea as a free-space propagation as far as the carina and then jumps to the parenchyma where it is treated as propagation in a two-phase system. The simulation used the assumptions found in Table 1.

Note that the value 344 m/s is the speed of sound in dry air at room temperature, (20°C or 293 K). It would seem that we should have used instead a value appropriate for the conditions of temperature and humidity present in the lungs (37°C or 310 K) and the partial

Table 1. *Simulation model assumptions*

	Model Assumption	Ref.
M_T	800 ± 200 g	12
ρ_A	1.293 kg/m ³	3
ρ_T	0.998×10^3 kg/m ³	3
C_A	344 m/s	3

M_T , lung tissue mass; ρ_A , air density; ρ_T , tissue density; C_A , speed of sound in air.

pressure of water vapor (47 Torr), which is the saturated vapor pressure of water at 310 K. There are three effects. The square of the wave speed is proportional to temperature (1), and the composition affects the density (2) as well as the value of the ratio of heat capacities (γ) (3).

For dry air, the ratio of heat capacities is nearly the ideal value for diatomic molecules nitrogen and oxygen (i.e., 7:5 or 1.40). Water vapor is polyatomic, so its ratio is temperature dependent (~ 1.3 in the relevant temperature range). We used the first and second laws of thermodynamics applied to a mixture of ideal gases to find the correction to γ for air containing water vapor at a partial pressure of 47 Torr. We find that the value of γ is lowered by $\sim 0.58\%$. That calculation is straightforward.

The temperature ratio, 310:293, increases the speed by $\sim 2.8\%$ since it enters as a factor under the square root.

In the denominator, the density of dry air must be multiplied by the factor $[(B - 0.3783e)/760]$, where B is the barometric pressure (in Torr), and e is the vapor pressure of the moisture in the air (in Torr) (in this case, 47 Torr at 37°C) (3). That factor is 0.977.

The result of applying these three corrections raises the value of the sound speed by $\sim 3.8\%$ from 344 to 357 m/s.

We note that C_A appears in *Eq. 4* only as a convenient way of expressing B_A in terms of C_A and the density. The only way the humidity affects C in *Eq. 3* is through the dependence of B_A on γ , which would lower the value of C by only 0.29% because the correction of 0.58% occurs under the square root. This correction is negligible for the two-phase system.

For the speed of sound in large airways, it is clearly appropriate to use the value 357 m/s; however, because the transit time from the tracheal microphone to the carina is an order of magnitude smaller than the total transit time, that correction should be insignificant as well. We therefore ignored these effects and used the values in Table 1 throughout.

According to our estimations, lung air volume varied from 2.5 to 7 liters. For a given value of lung mass, tissue density, and fractional volume of air in the lungs, the overall density of the lung was determined. For the purpose of simplicity tissue, density was assumed to be spatially constant and independent of orientation with the gravitational field.

As mentioned above, we assumed that the transition to tissue transmission occurred at the carina. It is

readily admitted this assumption may represent a serious oversimplification. However, it is an approximation that was made in the interest of mathematical convenience so that a comparison between observed and calculated transit times could be made for the purpose of examining the volume dependence. The actual treatment of the transition from propagation in large airways to propagation in parenchyma should probably be examined as a separate problem. Such an analysis would certainly require much more sophisticated mathematical methods than were presented in our simplified model.

In this approximation, all paths through the parenchyma are calculated as the three-dimensional straight-line distance between each microphone and the position of the carina as estimated from anatomical landmarks. Sound transmission time through the trachea, constant at 0.4 ms, was added to the transmission time through parenchyma, calculated from *Eq. 4*, to calculate the overall transmission time.

Figure 7 shows the results of this model superimposed on experimental data. The two solid curves represent the model's prediction for a lung mass of 600 g (*bottom curve*) and 1,000 g (*top curve*). The simulation correctly describes the relationship between transit time and lung volume, although it tends to overestimate the transit time.

DISCUSSION

The acoustic properties of the lung parenchyma are a function of the air content of the lung (5, 9, 14). We were interested in how transthoracic sound speed in particular was affected by varying air content in intact humans as a method of monitoring tissue properties noninvasively. To study this, we first had to develop a method of measuring sound speed accurately.

Using a "coded" input signal, we were able to measure the transit times of sound through the lung with precision. The frequencies of the input sound we used (130–150 Hz) are in the range of frequencies shown by others to exhibit optimal transfer of acoustic energy through the lung (1, 8). Many previous studies have inserted sound over a range of frequencies. Rice made his measurements from sound inserted from a spark source generating frequencies between 5 and 30,000 Hz, and Kraman used filtered white noise between 100 and 500 Hz. Because the lungs behave as a band-pass filter, the detected frequencies were between 100 and 1,000 Hz and peaked at ~ 250 –350 Hz. Wodicka et al. (14) introduced sound at the mouth of healthy subjects at resting lung volume and showed a strong trend of decreasing acceleration of the chest wall with increasing frequency. The largest transmission of the input sound they observed occurred in the range of 150 to 200 Hz. The largest sound transfer observed by Donnerberg et al. (2) in canine lung was ~ 180 –230 Hz.

The sound speed we calculated of 37 m/s is consistent with speeds that have been reported by others (1, 7, 9, 10, 14). Rice (9) found values of sound speed in excised

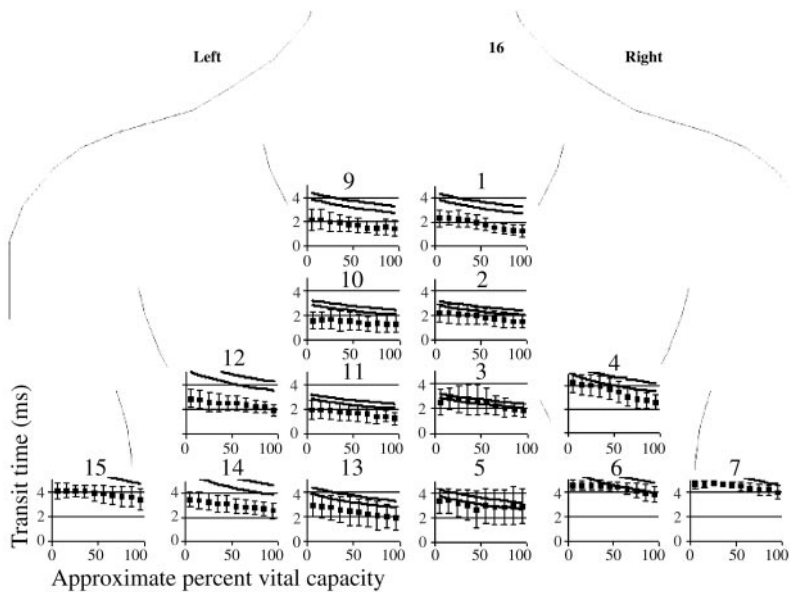


Fig. 7. Results of the simulation are superimposed on the experimental data. The two solid curves represent the model's prediction for a lung mass of 600 g (bottom curve) and of 1,000 g (top curve). Sound transmission time through the trachea, constant at 0.4 ms, was added to the transmission time through parenchyma calculated from Eq. 4 to calculate the overall transmission time. The following three-dimensional straight-line distances between each microphone and the position of the carina were used: channels 1 and 9: 10.7 cm; channels 2 and 10: 7.6 cm; channels 3 and 11: 7.5 cm; channels 4 and 12: 14.2 cm; channels 5 and 13: 10.6 cm; channels 6 and 14: 16.0 cm; channels 7 and 15: 19.4 cm.

horse lungs to be between 25 and 70 m/s, and Kraman (6) found values in human lungs between 23 and 33 m/s. Our results show credible relations with anatomic distances, i.e., the delay times are greater at sites farther away from the input sound and shorter at more proximal sites. They are consistent with the findings of Mahagnah and Gavriely (8), who observed transit times at two sites over the chest and found that they were 1.5 ± 0.5 ms to the anterior upper lobe and 5.2 ± 0.5 ms to the right lower base (1). Kraman (6) found transit times of 2–5 ms. Wodicka et al. (13) found propagation times of 0.9–4.1 ms by using a phase-estimation technique.

Accurate measurement of the sound speed enabled us to more closely examine the relationship of sound speed in the lung-to-lung volume. As noted above, we developed a simulation that correctly describes this relationship. Rice (9) developed a simulation that is virtually identical to ours and yielded similar results in excised horse lung. Our results confirm the observation of Rice. They are closer to clinical application in that they were done on intact humans with the use of a well-characterized input signal. Rice performed his experiments on excised horse lung, by incising the pleura at multiple sites and using a spark gap as his sound source.

The simulation presented here closely approximates experimental data. The simulation, however, tends to overestimate the transit time at the peripheral channels. One possible explanation for overestimation of transit time is that the model overestimates the distance between the sound source and the microphones, suggesting that sound could travel further along large airways before switching to transmission through parenchyma.

The fact that the value of the sound speed in the two-phase system is much lower than its value in either air (340 m/s) or soft tissue (1,500 m/s) is easily

understood from the model used here. We note from Eq. 2 that the numerator of Eq. 1 is dominated by the phase that has the smaller value for the bulk modulus (air), whereas the denominator is determined by the density of the more dense phase (tissue). This produces a smaller sound speed than the value for either one and is the expected behavior in a two-phase system (6, 11). Some confusion exists in the literature about this point. For example, Leung et al. (7) assert that "As tissue stiffness is $\sim 1,000$ -fold less than gas stiffness, the overall parenchymal stiffness will be dominated by tissue stiffness." In fact, the tissue stiffness, which we call bulk modulus, is much greater than the stiffness (bulk modulus) for air, not less. It is the compressibility or compliance of the tissue that is much less. As we note in our model above, it is precisely because the compressibilities of the two phases add directly that their stiffness (bulk modulus) constants add reciprocally. It is for this reason that the bulk modulus of air dominates parenchymal stiffness. Physically, when a compressional wave moves through the lung parenchyma, it is the air that gets compressed, not the tissue, simply because the compressibility of the air is $\sim 1,000$ times greater than the compressibility of the tissue. In our discussion of the model, we prefer to use the term bulk modulus rather than stiffness, because bulk modulus refers to a specific combination of elements of the more general stiffness matrix (4). Similarly, the volume compressibility is a specific combination of the elements of the more general compliance matrix (4).

Further evidence that the parameters contained in the expressions for the model (Eqs. 2, 3, and 4) occur as expected is also evident in the work of others. The sound speed was measured for the subjects in Kraman's study (6) after they had been breathing a mixture of 80% He-20% O₂. Mean speed increased by only 10%, whereas the predicted increase in speed through

the gas mixture alone was $>100\%$. The large expected increase for the gas is caused by the decreased density, which occurs because of the lower molecular weight of He. However, gas density is not a parameter in the two-phase system. The only effect on the model parameters is through the effect on γ . This effect was also suggested by Rice (9) and Mahagnah and Gavriely (8). For diatomic gasses O_2 and N_2 , the value of γ is 7:5 or 1.40; for monatomic He, it is 5:3 or 1.67. Replacing air with He would require multiplying Eq. 3 by $\sqrt{1.67/1.40} = 1.09$ or an $\sim 10\%$ increase as observed.

The speed of sound propagation in a mixture of gases is independent of pressure. This happens because both the bulk modulus of the gas and its density are proportional to pressure, and so it cancels pressure in the expression for the speed. However, the expression for the speed in the model is independent of the gas density, and so from Eqs. 2 and 3, we expect the speed to be proportional to the square root of the pressure. Rice measured the sound speed in excised horse lung as a function of pressure from 0.1 to 7 atm (9). His results clearly demonstrate the expected pressure dependence.

In conclusion, utilizing an improved method for studying transpulmonary sound speed, we have investigated the speed of sound in the lung as a function of lung volume. We find that the results can be described in terms of a model in which the lung is treated as a two-phase system. We find that the expression for the sound speed predicted by the model is also consistent with the work of others. The sound speed varies as expected with the ratio of heat capacities of the gas and gas pressure, and is independent of the gas density. It is our hope that this improved understanding of the mechanism of sound transmission in the lung will help

in the development of noninvasive tools for diagnosis and monitoring lung diseases.

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