

# Transpulmonary speed of sound input into the supraclavicular space

R. PACIEJ, A. VYSHEDSKIY, J. SHANE, AND R. MURPHY  
*Faulkner/Brigham and Women's Hospitals, Boston, Massachusetts 02130*

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**Paciej, R., A. Vyshedskiy, J. Shane, and R. Murphy.** Transpulmonary speed of sound input into the supraclavicular space. *J Appl Physiol* 94: 604–611, 2003. First published October 18, 2002; 10.1152/jappphysiol.00568.2002.—The transpulmonary speed of sound input at the mouth has been shown to vary with lung volume. To avoid the disadvantages that exist in certain clinical situations in inputting sound at the mouth, we input sound in the supraclavicular space of 21 healthy volunteers to determine whether similar information on the relationship of sound speed to lung volume could be obtained. We measured the transit time at multiple microphones placed over the chest wall using a 16-channel lung sound analyzer (Stethographics). There was a tight distribution of transit times in this population of subjects. At functional residual capacity, it was  $9 \pm 1$  (SD) ms at the apical sites and  $13 \pm 1$  ms at the lung bases. The sound speed at total lung capacity was  $24 \pm 2$  m/s and was  $22 \pm 2$  m/s at residual volume ( $P < 0.001$ ). In all subjects, the speed of sound was faster at higher lung volume. This improved method of studying the mechanism of sound transmission in the lung may help in the development of noninvasive tools for diagnosis and monitoring of lung diseases.

sound speed; lung parenchyma; sound recording; lung sound; noninvasive

IN PREVIOUS WORK IN OUR laboratory (2), we noted that transpulmonary sound speed varied with lung volume, offering the promise of providing information on lung tissue properties noninvasively. This work was done by introducing sound through the mouth. If similar information on the relationships of sound speed to lung volume could be obtained by supraclavicular injection of sound, then a more practical method for examining lung tissue properties could be obtained, as it provides a number of benefits. It simplifies the procedure by avoiding the necessity of using a mouthpiece. The need for the subject's cooperation in keeping the glottis open is also avoided. In addition, it simplifies interpretation of results by eliminating the uncertainty associated with sound transmission through the bronchial tree. Finally, it allows injection of sound with wider frequency content, which in turn leads to a cross-correlation function with a sharp peak in the envelope. With these thoughts in mind, we studied the relationship of sound speed input at the supraclavicular space to lung

volume and compared the results with those found when sound was input at the mouth.

## MATERIALS AND METHODS

Twenty-one subjects were examined with a 16-channel lung sound analyzer (Stethographics model 1602, www.stethographics.com). The system (STG) was described in detail in Bergstresser et al. (2). In short, the STG uses electret microphones mounted in commercially available stethoscope chest pieces to record data on a personal computer. Fourteen microphones were incorporated into a soft foam microphone pad. The microphone pad was positioned on a stretcher or a plastic reclining chair positioned at a 45° angle. Subjects were instructed to lie on the microphone pad. The microphone positions are shown in Fig. 1. One microphone, referred to as the reference microphone, was used to record sound inside a speaker chamber.

The STG software played the prerecorded sound through the speaker (diameter = 57 mm, resistance = 8  $\Omega$ , 0.5 W; Panasonic, P10177-ND) mounted in a conical plastic chamber. A soft flexible connector (length = 40 mm, diameter = 30 mm; part 2211E, RC Medical, Tolland, CT) conducted sound from the speaker chamber to the supraclavicular space. The back side of the speaker chamber was open to the air. Tight closing of the back side diminished the speaker output. Subjects were instructed to take a deep breath and to breathe out slowly to residual volume (RV). Neither flow nor volume of air ( $\dot{V}_A$ ) were measured.

**Subjects.** The 21 normal subjects entered into this study were volunteers who had no history of lung disease. Before the recording, verbal consent was obtained from every subject. Twelve subjects were men, and nine were women. Average age was  $50 \pm 23$  (range 10–84) yr. The average height was  $163 \pm 10$  (range 140–180) cm. The average chest circumference was  $83 \pm 14$  (range 60–107) cm.

**Input sound.** Our laboratory (2) has previously described how polyphonic sounds can be used to yield a cross-correlation function with a sharp peak in the envelope. We have observed that, when injected through the mouth, sounds with broader frequency content exhibit stronger destructive interference, presumably because higher frequency components travel further along the bronchial tree (16). In this study, we avoided the problem posed by not knowing the point of transfer of the sound from airways to the parenchyma by injecting the sound into the supraclavicular space. We were looking for the broadest meaningful frequency content of the injection sound. To investigate sound transmission through the supraclavicular space, we injected monophonic sounds

Address for reprint requests and other correspondence: A. Vyshedskiy, Faulkner/Brigham and Women's Hospitals, 1153 Centre St., Ste. 4990, Boston, MA 02130 (E-mail: andrey@stethographics.com).

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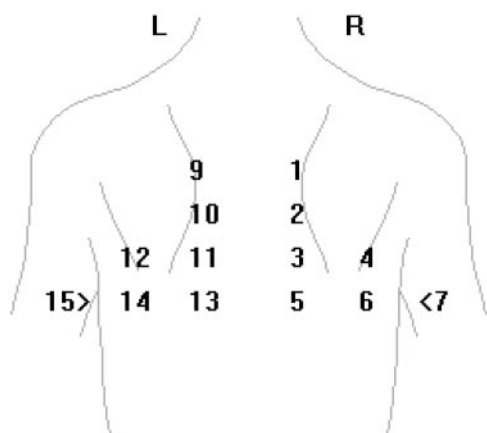


Fig. 1. Illustration of the position of the 14 microphones on the back. One microphone was positioned inside the speaker chamber to provide a reference. L, left; R, right.

from 50 to 800 Hz. Sounds with frequency  $<70$  Hz and  $>140$  Hz were transmitted poorly. Therefore, we constructed a polyphonic sound that contained frequencies from 70 to 140 Hz (Fig. 2). This sound was used in the remaining studies. The sound contained 14 cycles. The cycle frequencies were varied in the following pattern (*cycles 1–14*): 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120, 125, 130, and 140 Hz. The sound was played in a loop every 250 ms, yielding 80 transmission “images” over 20 s of the recording.

We tested for transmission of sound in the room. No detectable sound was transmitted through the room (Fig. 3A). The sound input while the subject slowly expired from vital capacity (VC) to residual volume (RV) resulted in a strong signal at microphones on the side of the sound injection (Fig. 3, *B* and *C*). On average, about half of input sound acoustic energy was transmitted to the contralateral side compared with the ipsilateral side ( $53 \pm 16\%$  left to right,  $60 \pm 16\%$  right to left). The reduction of the sound transmitted to the contralateral side is statistically significant ( $P < 0.005$  in both cases).

We addressed the question of the mechanism of contralateral sound transmission experimentally by injecting sound into a number of structures around the chest and the neck.

We found that there was no difference in contralateral sound transmission between the case when the microphone pad was made from a continuous sheet of foam and the case when two separate microphone pads were applied to the back. Application of a heavy metal object between the scapulae made no difference on sound transmission to both ipsilateral and contralateral sides.

We input sound into each lung base posterolaterally. The sound on the contralateral side was markedly attenuated in each case (right and left). Minimal sound was transmitted to the microphones when input into the 7th vertebral. Minimal sound was transmitted when input into the outside surface of the trachea. Minimal sound was transmitted when the input was into the trapezius muscle just posterior to the supraclavicular space. These observations are inconsistent with contralateral sound transmission through the chest surface.

Furthermore, we have input sound into the carotid artery on the neck (either left or right). Sound was transmitted equally well to both ipsilateral and contralateral upper body microphones. This should not be a surprise, because low-frequency heart murmurs (aortic murmur particularly) are known to be well transmitted through the carotid arteries. Recall that, in our experiments, the input sound is very similar to the sound of murmurs (both are 70–140 Hz). These observations, taken together, are consistent with the theory that the sound is transmitted contralaterally via the large blood vessels and other tissue located between the lungs. The wavelength of sound in lung parenchyma is  $24 \text{ m} \cdot \text{s}^{-1} \cdot 100 \text{ Hz}^{-1} = 0.24 \text{ m}$  or 24 cm. At that frequency, the wavelengths in those intervening structures would be much longer because the sound speeds in those structures are much higher than the speed in lung parenchyma. Because the wavelengths within the obstructions are much longer than the total dimension of the obstructions, it should be possible for the compressional waves to traverse them with little attenuation.

We concluded that, when input into supraclavicular space, most of the acoustic energy is transmitted through lung parenchyma, not through the chest surface.

Sound was injected consecutively through left and right supraclavicular spaces. Sound input on the right was used for the right lung transit time analyses, and that on the left was

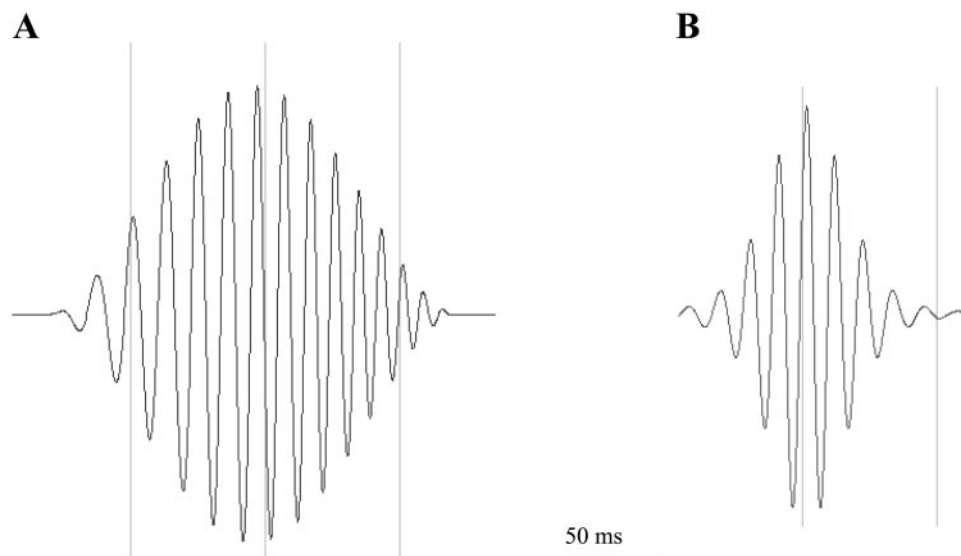


Fig. 2. *A*: the input sound contained 14 cycles. The cycle frequencies were varied in the following pattern (*cycles 1–14*): 70, 75, 80, 85, 90, 95, 100, 105, 110, 115, 120, 125, 130, and 140 Hz. *B*: the autocorrelation of the input sound exhibits a sharp peak in the envelope.

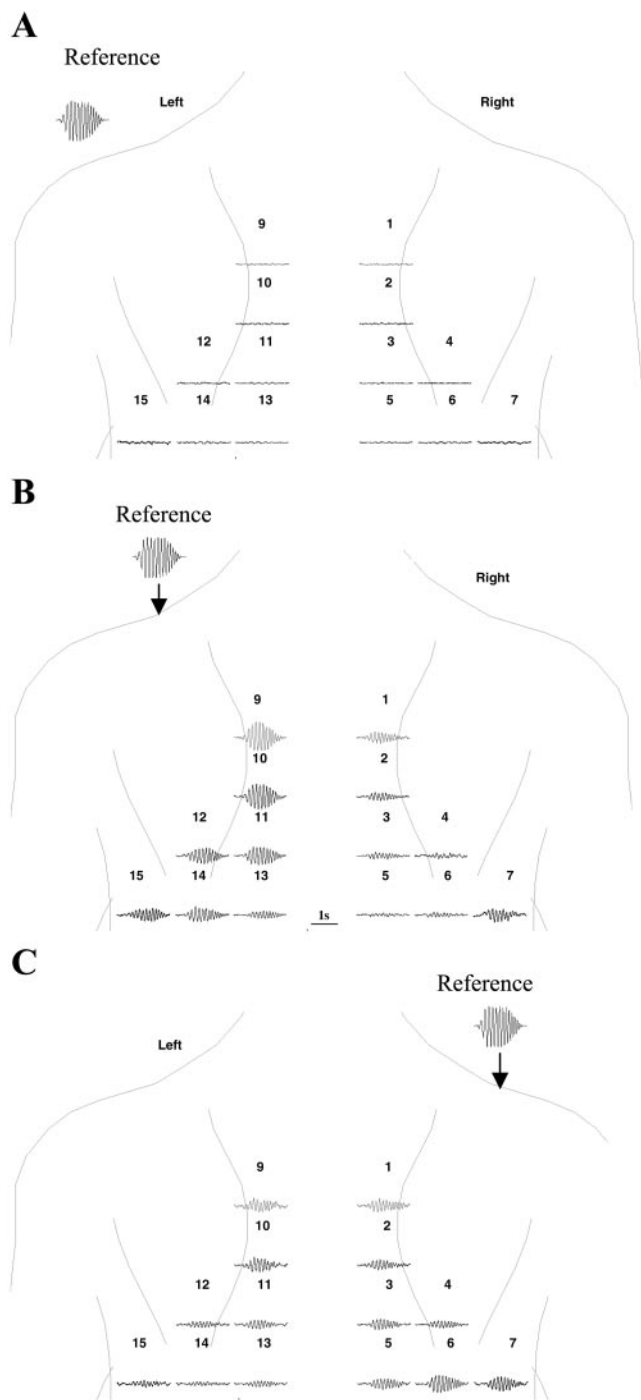


Fig. 3. Sound transmission through the lung. *A*: no detectable sound was transmitted when the speaker played sound into the room air near the subject. *B*: the sound input into the left supraclavicular space resulted in a strong signal on the side of injection. *C*: the sound input into the right supraclavicular space also resulted in a strong signal on the side of injection.

used for the left lung transit time analyses. We did not analyze transscapular sound transmission.

During the recordings, the subjects were asked to move the shoulder on the recording side slightly forward. This maneuver created a pit in the supraclavicular space. The flexible connector was lightly pressed against the skin in this pit. The pressure was adjusted to achieve the maximum amplitude of

transmitted sound. High pressure on the speaker chamber tended to reduce transmitted sound, presumably because of speaker damping. Care was used to hold the speaker chamber parallel to the spine.

*Simulation.* The theoretical prediction of the speed of sound in a two-phase system is discussed in detail in the book *One-dimensional Two-phase Flow* by Wallis (14). This two-phase model yields the following expression for the sound speed as a function of the volume fraction of air in the lungs

$$C = \left[ (\nu\rho_A + (1 - \nu)\rho_T) \times \left( \frac{\nu}{\rho_A C_A^2} + \frac{1 - \nu}{\rho_T C_T^2} \right) \right]^{-0.5} \quad (1)$$

where  $C$  is the speed of sound in the two-phase system;  $\nu$  is the fraction of air by volume, where  $\nu = V_A/(V_A + V_T)$ , where  $V_T$  is the tissue volume (7);  $\rho_A = 1.293 \text{ kg/m}^3$  is air density (3);  $\rho_T = 0.998 \times 10^3 \text{ kg/m}^3$  is tissue density (3);  $C_A = 344 \text{ m/s}$  is speed of sound in the air (3), also see discussion of the effects of temperature and humidity in Bergstresser et al. (2); and  $C_T = 1,460 \text{ m/s}$  is speed of sound in the tissue (3). For the purpose of simplicity, tissue density was assumed to be spatially constant and independent of orientation with the gravitational field.

The applicability of a two-phase system description to sound propagation through lung parenchyma is discussed by Bergstresser et al. (2). In short, sound propagation through lung parenchyma can be treated as a compressional wave in a homogeneous two-phase system. In other words, because the wavelength is expected to be large compared with the alveolar dimensions, the sound transmission can be simulated by using the assumption that the lung consists of a homogeneous mixture of gas (humid air) and soft tissue phases.

In our laboratory's previous work (2), we were using an approximation of Eq. 1 that can be used as long as  $\nu$  is  $>0.01$  and  $<0.99$

$$C = \frac{1}{\sqrt{\nu(1 - \nu)}} \sqrt{\frac{\rho_A}{\rho_T}} C_A \quad (2)$$

## RESULTS

*Transit time analysis.* An example of the transit time analysis through the right chest of the polyphonic sound with frequencies of 70–140 Hz is shown in Fig. 4A. The sound recorded at the speaker (reference) is shown on the *top*, and the sound recorded at *channel 6* is shown on the *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 6* is delayed by 14.9 ms compared with the reference.

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 14.9 ms between the reference and *channel 6*. The correlation coefficient at the peak of the cross-correlation function was 0.9.

Figure 5A shows the transit time between the reference and all of the 14 chest sites in a different subject at functional residual capacity. Circle size in the diagram is proportional to the transit time. Numbers indicate transit time in milliseconds. There was a progressive increase in this transit time from the microphones on the apical sites to the microphones over the

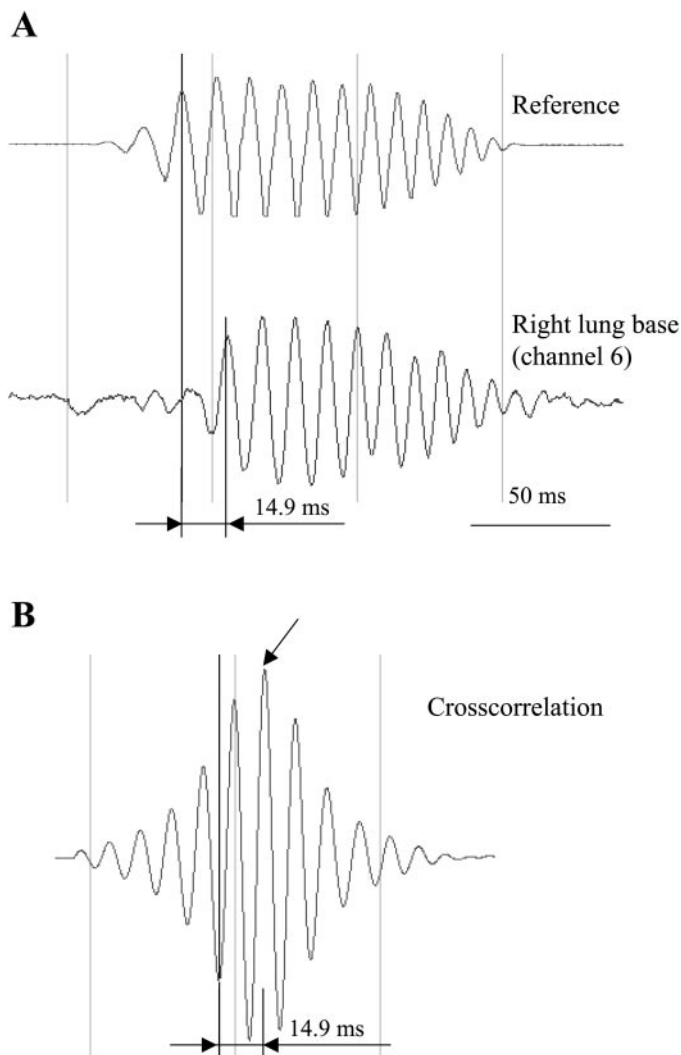


Fig. 4. Transit time analysis was performed with the use of a cross-correlation technique with further verification by approximating the interval between the corresponding peaks. A: the reference sound is displayed on the top. The transmitted sound recorded at the right lung base (channel 6) at functional residual capacity is shown at the bottom. The time interval between corresponding peaks of 2 sounds is 14.9 ms. B: the result of cross-correlation of the 2 sounds shown in A. The peak (arrow) corresponds to an arrival time difference (transit time) of 14.9 ms.

basal sites. Transit time varied over the chest from 6.6 ms at the apical sites to 13.9 ms at basal sites.

Figure 5B shows the relationship between the transit time measured at functional residual capacity and the three-dimensional straight-line distances between the reference microphone and 14 chest microphones. Linear regression yielded a speed of sound of 2.4 cm/ms = 24 m/s.

**Relationship of transit time to lung volume.** Transit time was shorter the bigger the lung volume, as illustrated in Fig. 6. For every recording microphone, the transit time in milliseconds is shown as a function of the approximate percentage of VC. At all lung volumes, the transit time was minimal at the apical sites (channels 1 and 9). There was a strong tendency for the

transit time to increase gradually the further away the microphones were from the apices. This observation was consistent in all subjects.

In all chest locations, the transit time varied inversely with lung volume (Fig. 6). Filling the lungs with air from RV to VC reduced transit time by up to 3.6 ms in some subjects. The paired two-sample *t*-test indicated that the reduction of transit time is statistically significant ( $P < 0.001$ ) at all channels, except channels 7 and 15. The largest transit time reduction occurred at central channels: channel 3 on the right ( $1.4 \pm 1.0$  ms) and channel 11 on the left ( $1.2 \pm 0.8$  ms).

**Comparison to sound input at the mouth.** There was a tight distribution of transit times among the diverse population of subjects (Fig. 6). The standard deviation of the average transit time data was ~10% compared with 30% when sound was injected through the mouth (2).

The transit time dependence on the lung volume was strongest at the central sites and weakest at the pe-

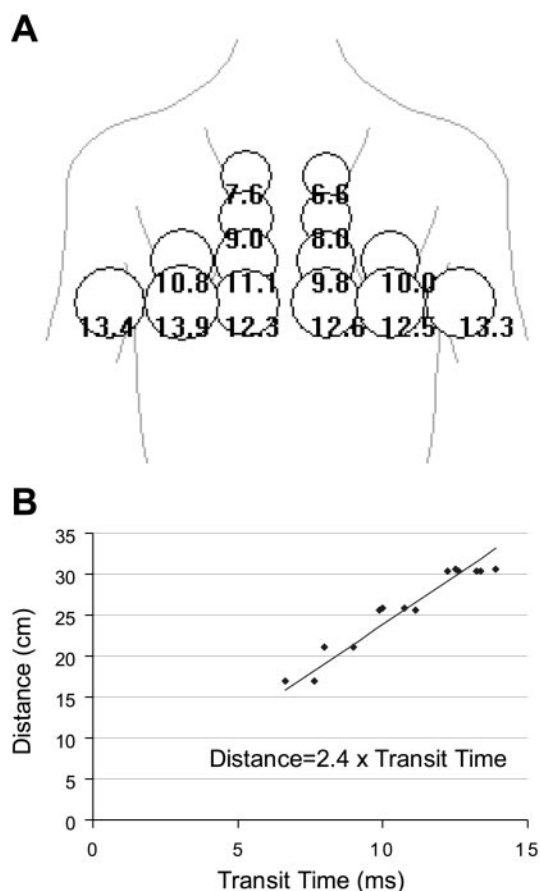


Fig. 5. A: transit time between the reference microphone and all of the 14 chest sites in 1 subject. Circle size in the diagram is proportional to the transit time. Nos. indicate transit time in milliseconds. B: transit time measured at functional residual capacity as a function of the three-dimensional straight-line distances between the reference microphone and 14 chest microphones. The following distances between each microphone and the supraclavicular space were used: channels 1 and 9, 17.0 cm; 2 and 10, 21.1 cm; 3 and 11, 25.6 cm; 4 and 12, 25.9 cm; 5 and 13, 30.4 cm; 6 and 14, 30.6 cm; 7 and 15, 30.4 cm. The linear regression yielded a speed of sound of 2.4 cm/ms = 24 m/s.

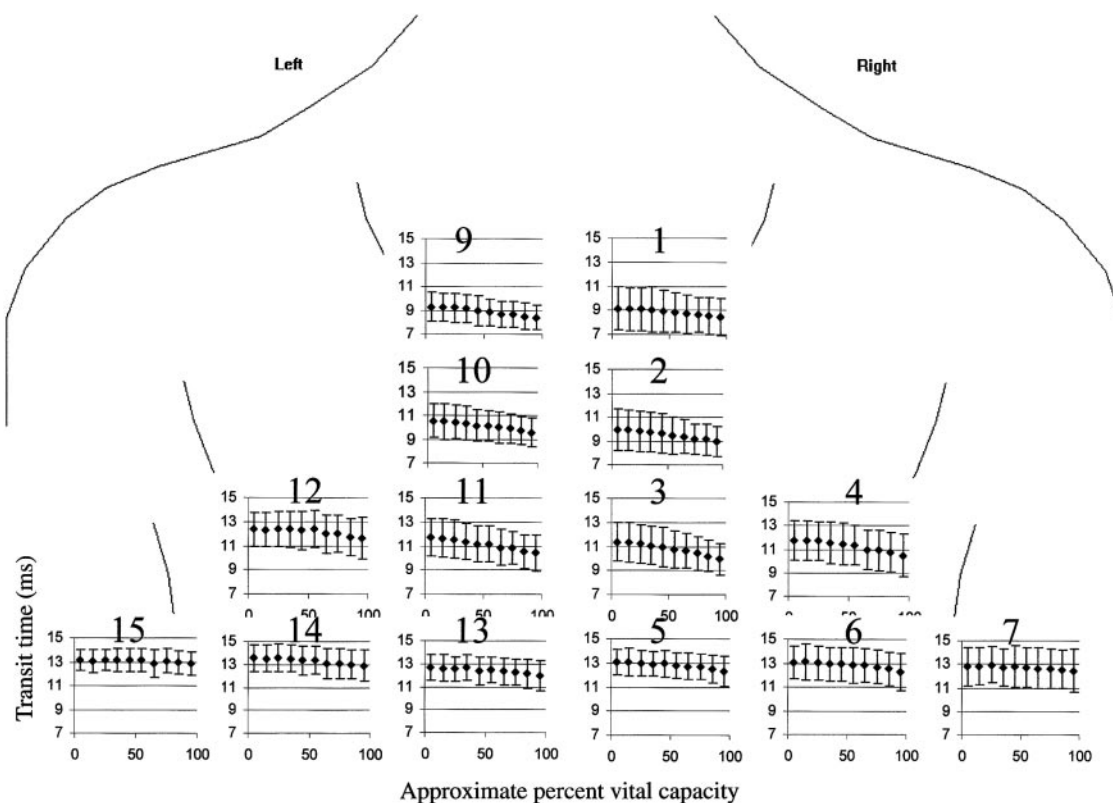


Fig. 6. Transit time between the reference microphone and all of the 14 chest sites in milliseconds shown as a function of approximate percentage of the vital capacity (VC). Values are means  $\pm$  SD, averaged among 21 normal patients.

ripheral sites, similar to the results obtained with sound injection through the mouth. The average decrease in the transit time from VC to RV was 0.8 ms with both methods of sound injection.

Transit time was shorter on the left than on the right when sound was injected through the mouth (2). Transit times did not vary significantly between left and right channels when the sound was injected into supraclavicular space.

*Relationship of speed of sound in lung parenchyma to biometric data.* The transit time was related to the three-dimensional straight-line distance from the microphones to the injection point. Linear regression was used to calculate the speed of sound for each patient, as noted in the caption for Fig. 5B. The calculated speed of sound was  $24 \pm 2$  (range: 28–20) m/s at VC,  $23 \pm 2$  (range: 27–19) m/s at functional residual capacity, and  $22 \pm 2$  (range: 28–19) m/s at RV.

Speed of sound showed little correlation with subject height (Fig. 7A; correlation coefficient =  $-0.4$ ), chest circumference (Fig. 7B; correlation coefficient =  $0.0$ ), and age (Fig. 7C; correlation coefficient =  $0.2$ ).

*Comparison between experimental and theoretically predicted speed of sound.* The theoretically predicted speed of sound in a homogeneous two-phase system is shown in Fig. 8 as a function of the  $\nu$  in the lungs. The minimum speed of sound ( $\sim 24$  m/s) occurs when air occupies one-half of the lung volume. In the theoretical case when only air is present,  $\nu = 1$ , the speed of sound

increases to 344 m/s. When no air is present,  $\nu = 0$ , the speed of sound increases to 1,460 m/s.

To show the limiting values at  $\nu = 0$  and  $\nu = 1$ , Eq. 1 was used to calculate the theoretical curve in Fig. 8. The simplified expression, Eq. 2, diverges at both limits, i.e., at  $\nu < 0.01$  and  $\nu > 0.99$ . It should be pointed out, however, that, except for values of  $\nu$  very close to those limiting values of 0 and 1, the numerical results of the two expressions are nearly indistinguishable. In particular, the portion of the curve with volume fractions of air that are experimentally accessible, the theoretical values calculated from the two expressions are virtually identical. Also, for volume fractions of air,  $\nu > 0.01$ , the calculated sound speeds are essentially independent of the acoustical properties of the tissue. The reason for this is clear: for the propagation of a compressional wave through lung parenchyma, it is the air that is compressed, not the tissue (2).

The experimental data from the normal subjects are superimposed on the theoretical curve. The *leftmost* data point corresponds to a lung RV that was assumed to be 1 liter. The *rightmost* data point corresponds to a VC that was assumed to be 5.5 liters.  $V_A$  in conducting airways was assumed constant at 0.15 liters.  $V_A$  in the lung parenchyma was calculated by subtracting  $V_A$  in conducting airways from lung volume. The  $V_T$  was assumed to be 0.8 liters at RV (15), linearly increasing to 1.7 liters at VC (13). These assumptions yielded  $\nu$  in the lung parenchyma of 0.52 at RV and 0.76 at VC.

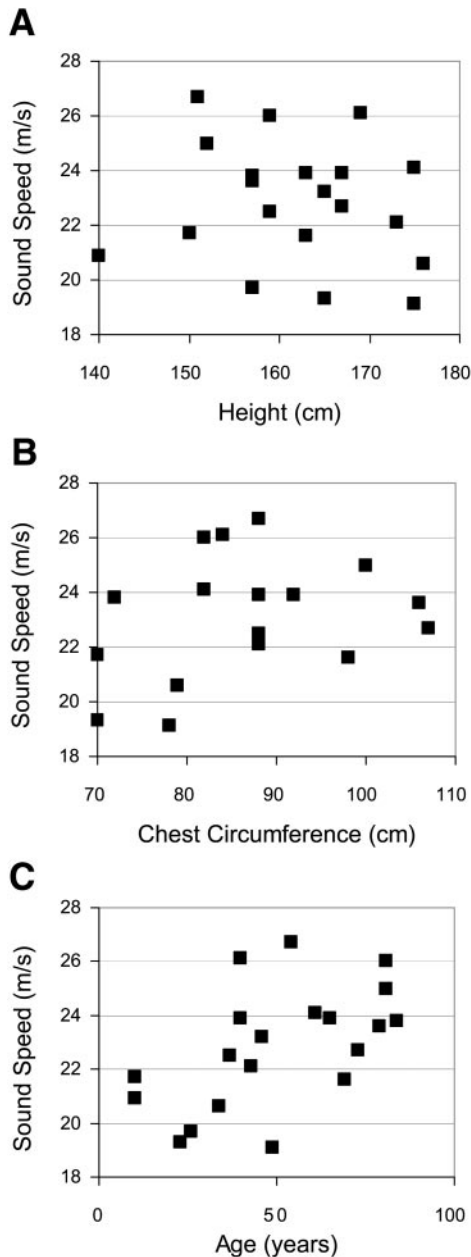


Fig. 7. Relationship of the speed of sound in lung parenchyma to biometric data. Speeds of sound as a function of height (correlation coefficient = -0.4; A), chest circumference (correlation coefficient = 0.0; B), and age (correlation coefficient = 0.2; C) are shown.

This range of  $\nu$  is close to that measured in dogs, 0.51–0.93 (1, 4).

**DISCUSSION**

Clinicians use alterations in the conduction of sound through the chest to obtain information on lung tissue properties. Whispered pectiloquy, the technique of examining the transmission of speech through the chest, is helpful in detection of conditions such as pneumonia and pleural effusions. We have been interested in obtaining more quantifiable information on

sound conduction. As noted, we recently reported a method for studying transpulmonary sound speed and showed that it is varied as a function of lung volume. In this present study, we show that the sound speed varies with lung volume in the same way as it does when the sound is input in the mouth. Furthermore, we show that sound input in the supraclavicular space travels rather uniformly in normal subjects. As sound input in the supraclavicular space requires little patient cooperation and, as mentioned, avoids the problem of knowing precisely where sound leaves the airways to enter the parenchyma, the technique provides a promise of yielding information on tissue properties noninvasively. This could have application as an aid in diagnosis and in monitoring conditions that alter lung properties such as pneumonia and congestive heart failure.

There were some differences in the results between the two input modalities. Calculated sound speeds were approximately one-half as fast when the sound was input through the supraclavicular space compared with sound input through the mouth. In the results previously reported (2) for sound speed through the mouth, it was assumed that the sound was transferred from the trachea to the parenchyma at the carina. If, in fact, the sound traveled further along large airways before switching to transmission through parenchyma, the shorter path length to the microphone would yield a slower speed and more closely agree with the results presented here. An advantage of the method of sound input through the supraclavicular space for the calculation of sound speed is that it avoids any assumption

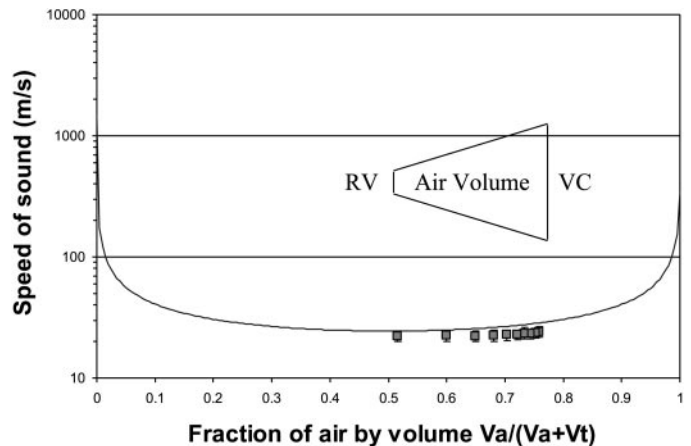


Fig. 8. The speed of sound in a two-phase system as predicted by Eq. 1 is shown as a function of the volume fraction of air in the lungs. The experimental data are superimposed on the theoretical curve. The leftmost data point corresponds to a lung residual volume (RV) that was assumed to be 1 liter. The rightmost data point corresponds to a VC that was assumed to be 5.5 liters. Volume of air in conducting airways was assumed constant at 0.15 liters. Volume of air in the lung parenchyma ( $V_A$ ) was calculated by subtracting volume of air in conducting airways from lung volume. The tissue volume ( $V_T$ ) was assumed to be 0.8 liters at RV (15), linearly increasing to 1.7 liters at VC (13). These assumptions yielded fraction of air by volume in the lung parenchyma of 0.52 at RV and 0.76 at VC. Values are means  $\pm$  SD.

Table 1. *Speed of sound in lung parenchyma obtained with a number of different sound injection methods*

Place of Sound Injection	Frequency Content of Input Sound	Subjects	Sound Speed	Reference
Supraclavicular space	Air-coupled speaker, 70–140 Hz	Human lung	22–24 m/s, depending on lung volume	This study
Lung surface	Sound from a spark gap, 5–30,000 Hz	Excised horse lungs	25–70 m/s, depending on lung volume	Rice (11)
Lung surface	Stress waves injected by a water jet	Goat and rabbit excised lungs	31–65 m/s, depending on lung volume in the goat; 16–37 m/s, depending on lung volume in the rabbit	Yen et al. (18)
Lung surface	Impulse surface distortion, 20–70 Hz	Excised sheep lungs	30–58 m/s, depending on lung volume in the sheep	Jahed et al. (6)
Esophagus	Stress waves generated by rapid deflation of esophageal balloon, 30–300 Hz	Anesthetized pigs	29–57 m/s, depending on lung volume in the pig	Jahed and Lai-Fook (5)
Mouth	130–150 Hz	Human lung	40–52 m/s, depending on lung volume	Bergstresser et al. (2)
Mouth	Wideband noise, 75–2,000 Hz	Human lung	62–82 m/s	Mahagnah and Gavriely (9)
Mouth	Band-passed white noise, 125–500 Hz	Human lung	23–33 m/s at functional residual capacity	Kraman (7)

about the location of the transition of the sound from large airways to propagation in parenchyma.

As noted, transit time was shorter on the left than on the right when sound was injected through the mouth (2). Transit times did not vary significantly between left and right channels when the sound was injected into supraclavicular space. A possible explanation is that, when injected through the mouth, sound is transmitted through the heart at a higher speed than through the lung. This scenario implies that the sound jumps from the trachea to the heart and propagates through the heart before jumping to the parenchyma. An appropriate theoretical treatment, utilizing the fact that the sound wavelength in the heart tissue is much longer than the heart's dimensions, predicts a reduction in the transit time of  $L/v$ , where  $L$  is the size of the heart and  $v$  is the speed of sound in the lung parenchyma. An analogous theoretical approach is described and some applications are discussed in Ref. 12. Substitution of 0.1 m for the size of the heart and 23 m/s for the speed of sound yields the reduction of transit time as much as 4.3 ms. When injected into the supraclavicular space, sound probably travels through the lung parenchyma around the heart; therefore, no difference between left and right transit times is observed.

The alternative explanation of the reduced transit time on the left base when sound is injected through the mouth can be that the sound travels longer within the airways. There is some evidence that sound injected through the mouth enters the parenchyma directly through the right tracheal wall that is in direct contact with the mediastinal aspect of the right lung. Several massive vascular structures isolate the trachea from the left lung so that the sound would pre-

sumably have to travel further down the airways before entering the parenchyma of the left lung (8, 17, 10).

Table 1 compares the speed of sound in lung parenchyma obtained with a number of different sound injection methods. Note that the results obtained by different groups of scientists are similar. Injection through the mouth tends to yield a faster sound speed than direct injection into lung parenchyma. In our case, sound injection through the mouth yielded a speed of sound that was twice as high as obtained when the sound was injected into the supraclavicular space. This effect could be an artifact of overestimation of the distance that the sound travels in the parenchyma when the sound is injected through the mouth. In other words, it is likely that sound propagates at high speed in the large airways over longer distances than assumed by the models before it switches to propagation through the parenchyma.

We have found that speed of sound of  $22 \pm 2$  to  $24 \pm 2$  m/s is very similar to that predicted by *Eqs. 1 and 2* at fraction of air of 0.5–0.8. The close correlation between the experimental and theoretical data and the tight distribution of speed of sound in healthy lung may provide a noninvasive means to deduce fraction of air in the lung from experimentally measured speed of sound.

**Conclusions.** Sound injection into supraclavicular space provides a noninvasive method of examining sound speed in lung parenchyma. This method yields consistent and reliable results while reducing the need for patients' cooperation. This improved method of studying the mechanism of sound transmission in the lung may help in the development of noninvasive tools for diagnosing and monitoring lung diseases.

Statement of financial interests: R. Murphy holds the rights to several lung sound-related patents. R. Murphy and A. Vyshedskiy have financial interests in Stethographics, Inc.

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