



Pulmonary Doppler signals: a potentially new diagnostic tool

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Aims

To overcome the limitations due to ultrasound attenuation by the air in the lungs, in order to study the pulmonary system using an advanced signal processing technology.

Methods and results

Pulsed spectral Doppler signals were obtained over the chest wall using a signal processing and algorithm package (transthoracic parametric Doppler, TPD, EchoSense Ltd, Haifa, Israel) in conjunction with a non-imaging Doppler device (Viasys Healthcare, Madison, WI, USA) coupled with an electrocardiogram. The signals picked up by a transducer positioned at various locations over the chest wall, were treated for noise, analysed parametrically and displayed in terms of both velocity and power originating from moving ultrasound reflectors. Clear reproducible lung Doppler signals (LDS) were recorded. Up to five bidirectional triangular waves with peak velocities of 20–40 cm/s, that survived the 40 dB/cm attenuation of the lung, were recorded during each cardiac cycle. The first signal coincides with early ventricular systole, the second with late systole, the third and fourth with diastole, and the last with atrial contraction.

Conclusion

LDS originate from different elements and phases of cardiac activity that generate mechanical waves which propagate throughout the lung and are thus expressed in pulsatile changes in ultrasound reflections. While such signals could originate either from pulsatile blood flow or reflections from movement of the blood vessel—alveolar air interface, the experimental evidence points towards the tissue—air interface movements due to vessel expansion as the origin. The LDS can potentially be an important tool for diagnosing and characterizing cardio-pulmonary physiological states and diseases.

Keywords

Lung • Cardio-vascular • Pulsations • Diagnosis • Ultrasound

Introduction

The ability to non-invasively collect information regarding the pulmonary aspect of the cardio-pulmonary system may be of prime importance. Ultrasound, the widely used powerful essentially harmless non-invasive diagnostic tool, cannot be used for studying both the lung and the pulmonary parenchyma vessels.¹ This unfortunate limitation is due to two main factors: ultrasound scattering by alveolar air makes image reconstruction virtually impossible and at the same time, the extremely high total ultrasound energy attenuation by the air in lungs² (in the range of 40 dB/cm, depending on level of inflation) wipes out the signals originating from lung

parenchyma and blood vessels. The above is true for both imaging and Doppler modes of ultrasound operation.

A Doppler ultrasound system incorporating a special signal processing package (transthoracic parametric Doppler, TPD-03, EchoSense Ltd., Haifa, Israel) by means of which clear Doppler signals of pulmonary origin can be recorded from the chest wall was used. These signals, obtained from the right lung, including areas remote from heart and main pulmonary arteries and veins, are in full synchrony with the ECG.

As the power levels of ultrasound reaching the chest wall, velocity and signal shape are determined by the properties of the lung parenchyma and the cardio-pulmonary vascular system,

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these signals may provide valuable information and new insights into the structural and functional characteristics of the lung parenchyma and vasculature in health and disease.

Methods

The study included 31 healthy volunteers (16 F, 15 M, mean age 57 ± 16 years) and one subject with atrial fibrillation. The trial was approved by the institutional review board of Elisha Medical Center, Haifa, Israel, and a written informed consent was signed by all subjects.

Lung Doppler signals (LDS) measurements were made using a commercially available, non-imaging, pulsed Doppler device (Sonara/tek, Viasys Healthcare, Madison, WI, USA) the output of which was fed to a PC. The data received were processed and displayed by a software package, based on adoptive filtering and pattern recognition algorithms designed specifically for the pulmonary system (TPD-03, EchoSense Ltd., Haifa, Israel). The output display of the TPD includes real-time tracings of both velocity and power (reflected). In the former the velocity is plotted on the Y-axis while the power is represented by colours, according to the colour scale at the right-hand side. In the latter the power is plotted on the Y-axis while the velocity is represented by colours, according to the colour scale.

Unless otherwise stated, measurements were made from the chest wall over the right lung of each sitting subject by means of a 2 MHz, 16-mm diameter ultrasound transducer (Viasys Healthcare, Madison, WI, USA). Pulse repetition frequency was 3 kHz. The sample volume of 3 mm was placed at selected depths in the range of 5–10 cm. Depth was corrected for the differences in wave propagation velocity in chest tissues (about 1450 m/s) and in the air-rich lung (700–1000 m/s, depending on degree of inflation). Measurements were obtained at the second to sixth intercostal spaces (ICS) at distances of 5 and 10 cm to the right of the mid-sternal line. A transducer with a focal length of 6 cm was positioned perpendicular to the chest wall and the impedance was matched by the standard ultrasound gel. The maximal transmitted power was 74 mW/cm^2 , i.e. only about 10% of $I_{\text{SPTA},3}$, which according the FDA guidelines³ for peripheral vessels is 720 mW/cm^2 . The subjects were also connected to a standard two

lead ECG (Norav Medical Ltd., Yokneam, Israel) the output of which was incorporated in the display.

Results

Typical tracings of LDS recorded over the right lung of healthy subjects are depicted in Figure 1. The tracings show two striking features: the individual signals are in sync with the cardiac cycle and consist mostly of simultaneously timed positive and negative components. This fact reflects the coexistence of simultaneous movements of opposite directions relative to the ultrasound beam direction. These bidirectional, triangular-shaped pulmonary Doppler signals are of very high power, often exceeding 100 dB (vs. about 20–30 dB for blood flow in systemic arteries and veins and about 60–70 dB for cardiac tissue Doppler signals), indicating movement of very efficient echo reflectors. The LDS waves typically have peak velocities of up to 30 cm/s and durations in the range of 70–200 ms.

As seen in Figure 1A, during each cardiac cycle the LDS typically consists of five discrete signals, usually bidirectional, numbered 1–5 and 1*–5* for the positive and negative signals, respectively. Note that the positive polarity reflects movements towards the chest wall.

Table 1 lists the average maximal velocities of the positive signals (\pm SD) recorded at different ICS in the 31 subjects.

The signals can be divided into two groups: the first group consisting of signals 1, 3, and 5 that appear in practically all normal subjects. The second group consisting of signals 2 and 4, each of which appears in 30% of the subjects while both appearing at the same time in about 10%. Signals 1 and 1*, which are sometimes double humped, appear about 20 ms after the R-wave of the ECG and typically have peak velocity of 20–30 cm/s. This timing implies that they are associated with ventricular systole. The 20–30 ms delay with respect to the R-wave is most likely due to signal propagation time from the heart to the recording location. Signals 2 and 2* that

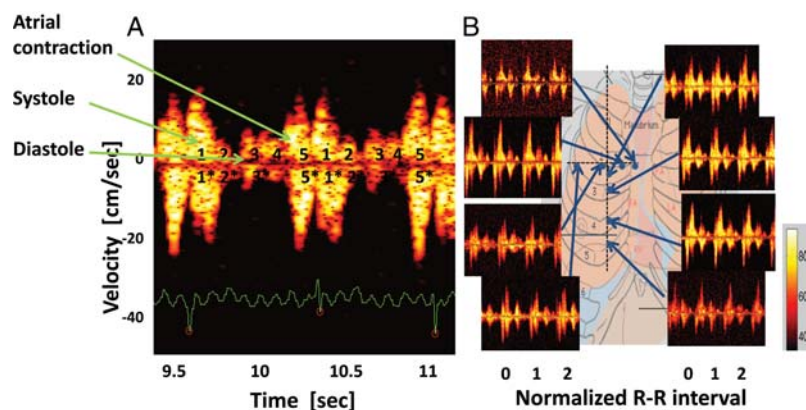
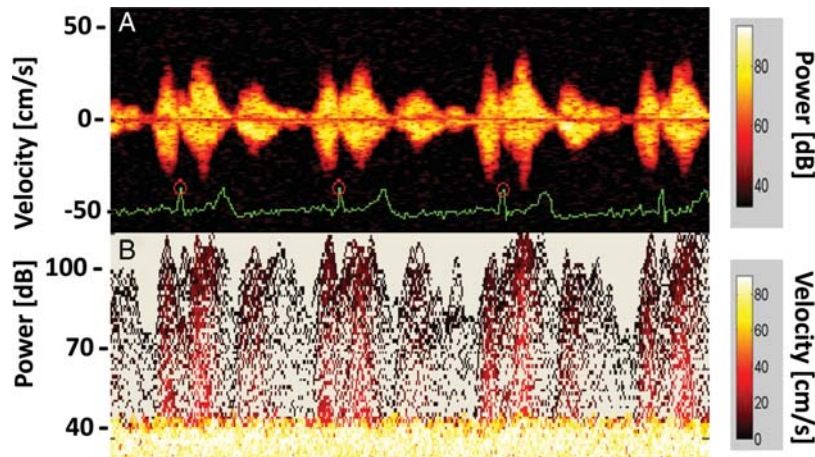


Figure 1 Lung Doppler signals measured over the right lung using the transthoracic parametric Doppler system. (A) Typical tracing obtained from a subject with normal lungs at the fourth intercostal space, 6 cm to the right of the mid-sternal line with the corresponding ECG tracing. (B) Lung Doppler signals obtained at different locations over the right chest at positions indicated by arrows.

Table 1 Average peak lung Doppler signals velocities in normal subjects

| | Sig 1 positive | | Sig 3 positive | | Sig 5 positive | |
|------|-------------------------|------|-------------------------|------|-------------------------|------|
| | Av. peak velocity, cm/s | SD | Av. peak velocity, cm/s | SD | Av. peak velocity, cm/s | SD |
| ICS2 | 11.60 | 4.23 | 12.67 | 3.62 | 16.06 | 4.44 |
| ICS4 | 24.87 | 6.48 | 16.56 | 2.96 | 26.73 | 6.54 |
| ICS6 | 28.12 | 7.76 | 14.35 | 3.22 | 23.35 | 6.10 |

**Figure 2** Lung Doppler signals obtained at the right chest—the velocity (upper tracing) and the reflected power (lower tracing). The colour scale for the velocity and power is given at the right of the tracings.

appear at the tail end of signal 1 have a lower peak velocity, about 10 cm/s, and their timing corresponds to the terminal part of the systole. Signals 3 and 3*, which are sometimes double humped, appear during the diastolic rapid filling phase and have a peak velocity of up to 15 cm/s. Signals 4 and 4*, the peak velocity of which is usually under 10 cm/s, appear during the late diastole (diastasis). Note that at high heart rates and short diastasis they are usually masked by signal 5. Signals 5 and 5*, which typically have peak velocities of 30 cm/s, correspond in time with atrial contraction.

Figure 1B depicts a set of tracings recorded at different locations over the right lung. Very similar LDS signals are recorded over a wide area extending all the way to the axilla. LDS recordings of similar shape, amplitude, and power are also obtained from different depths inside the chest cavity (up to depths of about 10 cm).

The two tracings in Figure 2 depict the velocity and reflected power as recorded over the right chest. It is seen that the reflected power of all LDS exceeds 80 dB while that of signal 1 reaches levels above 100 dB.

Figure 3 provides evidence for the origin of signal 5. Figure 3A depicts signals typical of normal sinus rhythm, i.e. including signals 1, 2, 3, 4, and 5 while Figure 3B depicts LDS in the patient with atrial fibrillation. It is noted that signal 5 which represents atrial contraction is missing.

Figure 4 is designed to enable comparison between TPD tracings and standard Doppler flow velocity measurements. The figure depicts sequential measurements made from a normal subject by the TPD (middle tracing), at the fourth intercostal space about 10 cm to the right of the sternum, and by a IE 33 Philips (Andover, MA, USA) cardiac echo system used to measure blood flow velocity at the main pulmonary artery (upper tracing) and pulmonary venous flow (lower tracing). Note that the velocities of the pulmonary artery and the pulmonary vein were in the range of 70–80 cm/s while those of the lung signals are 10–20 cm/s.

The LDS are seen to be similar to the flow velocity tracings in some aspects and different in others. The systolic flow wave (S) is seen in all three tracings with peaks at similar times. However, the LDS recordings (middle trace) show an additional earlier peak marked by ‘*’ that begins with the onset of the QRS complex and corresponds in time with the isovolumetric contraction (there is no corresponding flow wave). The diastolic waves (D), which appear only in the LDS and venous flow, seem to coincide in time.

Discussion

It is generally accepted, that due to the 40 dB/cm ultrasound attenuation by the air in the lungs: ‘Air containing lung has no

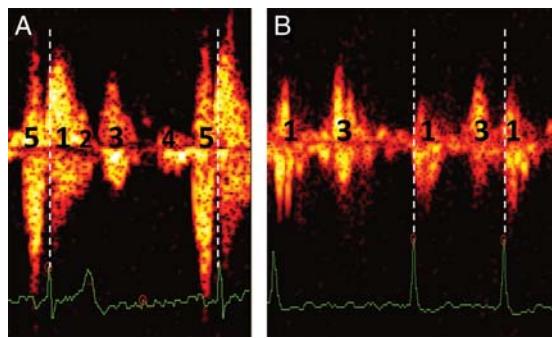


Figure 3 Recordings of lung Doppler signals over the right lung. (A) lung Doppler signals recorded during normal sinus rhythm. (B) lung Doppler signals recorded from a patient with atrial fibrillation.

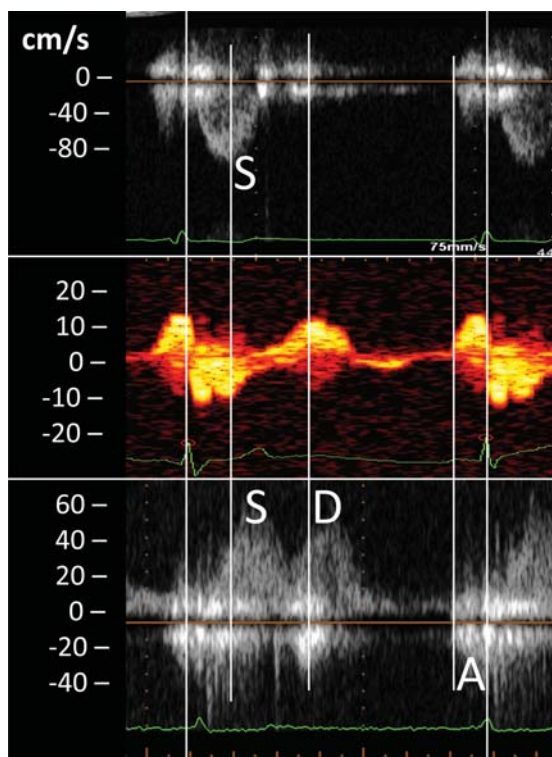


Figure 4 A comparison between transthoracic parametric Doppler tracings and standard Doppler blood flow measurements. Measurements made from a normal subject by the transthoracic parametric Doppler (middle trace), at the fourth intercostal space about 10 cm to the right of the sternum, and standard pulse Doppler imaging of the main PA blood flow (upper tracing) and pulmonary venous flow (lower tracing), see also text.

echo beyond the pleural interface at its surface'.^{1,2} The results presented in this study demonstrate, for the first time, that the above limitation can be overcome by means of the TPD signal processing

and analysis system acting on conventionally acquired ultrasound Doppler signals.

The TPD system consistently records very high power Doppler signals (LDS) that reach the chest surface despite the said attenuation. The synchronization of these signals with the ECG, i.e. the cardiac mechanical events, indicates that they may correspond to one of the following sources: tissue contraction, tissue mass dislocation, or blood flow. The notion that they are of tissue origin is inconsistent with a number of facts: Tissue Doppler signals² are generated by cardiac muscle contraction and cardiac muscle movements while the LDS are recorded over the non-contractile lung parenchyma and they persist when there are no respiration movements. The power of the LDS signals is 100-fold greater than that of typical Doppler tissue signals (90–110 vs. 60–70 dB) despite the large lung attenuation. The alternative possibility that the LDS originate from erythrocytes flowing in the large blood vessels or the heart itself can be ruled out in view of fact that the latter are characterized, even at their source, by low power (20–40 dB) and relatively high velocities (reaching 100 cm/s). Moreover, they have similar characteristics when recorded over wide areas of the left and right chest. Therefore, we postulate that these strong signals must originate from movement of some other powerful ultrasound reflectors possibly with the aid of a conducting network acting as a 'wave guide' that conveys them towards the chest wall. Thus, the main issues to be resolved here are: who are these moving reflectors, and is there a network that conveys the ultrasound energy within the chest cavity such that it can be recorded over wide areas of the chest wall? As the possibility that the reflectors are flowing erythrocytes or moving tissues was ruled out, we postulate that the generators of the LDS are Vascular (arteries and veins) wall pulsations resulting from the pressure waves propagating along the network of pulmonary blood vessels.^{4–6}

The proposed mechanism of the LDS generation is illustrated schematically in Figure 5A. Within this framework, the LDS originate from ultrasound energy reflected by the blood vessel–alveolar air interfaces. These circular interfaces undergo pulsatile expansion by the blood pressure waves that travel along the pulmonary vasculature.^{6–8} The expansion movements result in ultrasound wave reflections that reach power levels in excess of 100 dB, i.e. they are much higher than those produced by flowing blood. This high air–tissue junction reflective power is well known and serves as the basis for the use of encapsulated air bubbles as ultrasound contrast materials.⁹ Owing to the large power at the origin, the LDS can be recorded at the chest surface, having power levels of 80–100 dB, in spite of the attenuation inflicted by the lung parenchyma. The recorded signals are likely to represent the summation of ensembles of signals generated by numerous relatively small pulsating vascular elements of different size and orientation. Owing to the diversity of orientation, the specific ultrasound beam angle vs. the chest wall surface does not have a significant effect on the recorded velocity.

Figure 5B compares the time course of the pressure waves in the pulmonary artery and vein with the corresponding LDS waves by superposing the pressure tracings^{6–8} on the LDS tracing. We see that neither one of the arterial nor venous pressure waves

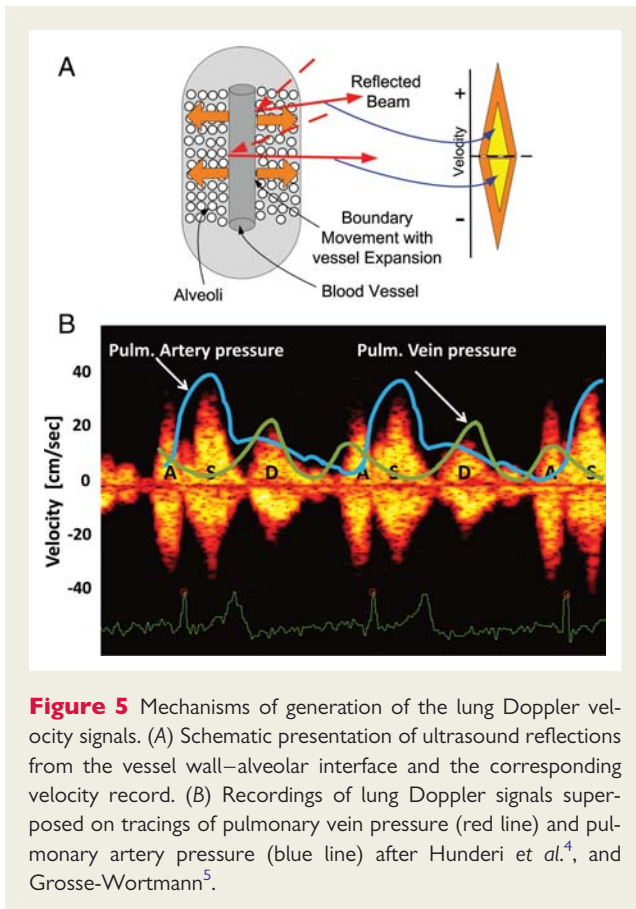


Figure 5 Mechanisms of generation of the lung Doppler velocity signals. (A) Schematic presentation of ultrasound reflections from the vessel wall–alveolar interface and the corresponding velocity record. (B) Recordings of lung Doppler signals superposed on tracings of pulmonary vein pressure (red line) and pulmonary artery pressure (blue line) after Hunderi *et al.*⁴, and Grosse-Wortmann⁵.

alone corresponds to the whole shape of the LDS wave, rather, both are required to reconstitute the LDS shape.

From morphological studies¹⁰ of the distribution of pulmonary vessels of different diameters, together with the fact that very similar signals are recorded over large chest surface areas, one may conclude that we are dealing with vessels with diameters of 1 mm or smaller. The recorded velocities of these expanding walls are in the order of 10–30 cm/s, values similar to those reported for peripheral arteries of similar dimensions.^{11,12} Note, that in spite of the fact that the pulmonary blood pressure is much lower than peripheral blood pressure, the expansion velocities in the two are comparable in view of the higher compliance of the pulmonary vessels.¹³ It should be noted that in cases where the signal source is wall–air interface movement, optimal signal strength would be obtained when the ultrasound beam is perpendicular to the vessel axis.

On the basis of the above one may conclude that the movements of the tissue–alveolar air interface, generated by the

blood pressure waves propagating through the pulmonary vasculature, are the source of the recorded LDS. Furthermore, it can be concluded that the LDS may contain information of significant diagnostic and physiological value regarding the pulmonary parenchyma and vasculature, as well as the cardio-vascular system. The reason these potentially important signals were not recorded to date may be due to the fact that in view of the erroneous assumption that all signals originating within the lung are totally wiped out by the attenuation caused by the air in the lung parenchyma, nobody looked for them.

Conflict of interest: Y.P. is a minority shareholder in Echosense, A.K., E.S. & R.S. are employees of Echosense. I.K. has no affiliation with echosense.

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