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ORIGINAL INVESTIGATION

Footprints of Cardiac Mechanical Activity as Expressed in Lung Doppler Signals

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Aims: To determine the diagnostic information contained in cardiac pulsatile pressure waves as expressed in the Doppler signals recorded over the right lung. Methods and Results: The pulsatile characteristics of the pulmonary vascular system were studied by means of the novel pulse Doppler technology in 38 control volunteers, 31 patients with atrial fibrillation (AF) and 7 patients with atrial flutter. The Doppler velocity waveforms recorded were interpreted in relation to the cardiac cycle mechanical events that generate them: Ventricular systole (S), diastole (D) and presystolic left atrial contraction (A). It was demonstrated that in all cases of AF, wave-A was absent. With longer diastole a high frequency velocity waves were visible. It is assumed that they represent the atrial mechanical fibrillation. In the patients with atrial flutter, the single A-wave was replaced by a waveform termed F, the frequency of which exactly matched that of the flutter wave on the ECG. The F-wave had both a positive and negative component. Conclusion: The lung Doppler signals contain distinct signatures typical of arrhythmias such as AF and atrial flutter that can be used for both diagnosis and to gain insight into the nature of the phenomena. (Echocardiography 2014;00:1–4)

Key words: lung, cardiovascular, fibrillation, flutter, diagnosis, ultrasound

It was generally accepted that ultrasound cannot be used for studying the normal lung. However, it was recently shown that clear reproducible signals in synchrony with the cardiac cycle can be recorded over the lungs by a new pulsed Doppler ultrasound system (Transthoracic Parametric Doppler [TPD], EchoSense Ltd., Haifa, Israel). These Doppler velocity and power signals, termed lung Doppler signals (LDS) when recorded from normal subjects typically consist of three main components: S, D, and A corresponding in time with ventricular systole, diastole and the presystolic/atrial contraction periods (see Fig. 1). The origin of the LDS is assumed to be the movements, due to pressure pulsation, of the interface between the alveolar air and the blood vessel walls which are very strong ultrasound reflectors.[1] In fact, the amplitude or power of the Doppler signals thus recorded may reach values of 30–40 dB higher than those recorded from the flow of blood in the heart and the main peripheral arteries. It therefore stands to reason that the characteristics of these signals may be reflected in patients with various cardiac pathophysiological conditions.

It is the purpose of this study to record LDS in patients with the new modality for monitoring cardiac activity via its footprint in the pulsations of the pulmonary arteries and veins. Special reference is made to atrial fibrillation (AF) and atrial flutter to determine whether their lung Doppler footprint may provide information regarding cardiac and pulmonary vascular function.

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Methods:

The study included 38 control volunteers 31 patients with AF and 7 patients with atrial flutter (AFL) (See Table I). LDS were successfully recorded from all 76 subjects. The LDS were recorded simultaneously with ECG (Lead I) by means of the TPD. 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. The LDS wave was obtained by a 2 MHz, single element pulse Doppler transducer (Viasys Healthcare, Madison, WI, USA), 16 mm in diameter with a focal length of 5 cm. The Doppler pulse repetition frequency was 3 kHz and length of the sample volume was 3 mm. Signal processing was made by the TPD

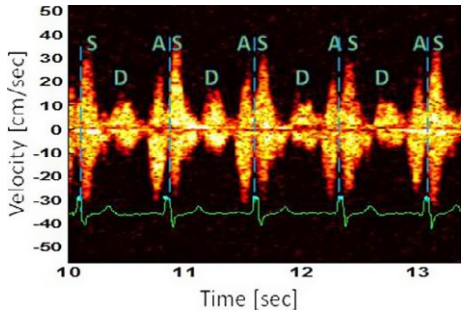

Figure 1. Typical LDS recorded from a subject with normal sinus rhythm using the TPD system. (transducer placed at the 4th intercostal space, 5 cm to the right of the mid-sternal line). LDS = lung Doppler signals; TPD = Transthoracic Parametric Doppler.

TABLE I

Studied Population

	Control n = 38	AF n = 31	AFL n = 7
Average age (years)	72	72	75
Gender (female/male)	13/25	10/21	2/5
Average BMI	26	30	

AFL = atrial flutter; BMI = body mass index.

software package (EchoSense Ltd.) When necessary, Depth was corrected for the differences between the wave propagation velocity in chest tissues (muscle, fat, etc. i.e. about 1540 m/sec) and in the air-rich lung (700 m/sec, depending on degree of inflation).[2,3] Maximal transmitted power was 74 mW/cm^2, i.e. only about 10% of the allowed $I_{SPTA.3}$ (Derated Spatial-Peak Temporal-Average Intensity).[4]

The transducer was positioned over the chest wall, at an angle close to 90° with respect to the chest wall, about 5 cm to the right of the mid-sternal line. Recordings were repeated at the second to the sixth intercostal spaces. Appropriate gel was placed under the transducer for impedance matching.

Results:

The spectral tracing recorded from normal subjects had clear, mostly symmetric bidirectional triangular signals that were synchronous with the cardiac events. The first wave, just after the R-wave, was termed S (systolic), the next, usually started at the end of the T-wave, was termed D (diastolic), and the wave which coincided with atrial contraction was termed A, as seen in Figure 1.

In all the 38 control volunteers, the systolic signal, S, had a mean positive peak velocity of 22.1±8.5 cm/sec and somewhat higher simultaneous negative peak of −26.2 ± 8.4 cm/sec. The mean peak positive and negative velocities of the diastolic signal D were smaller: 15.3 ± 3.5 and −9.1 ± 2.7 cm/sec, respectively, while those of the atrial signal A were 26.3 ± 8.4 and −21.4 ± 6.0 cm/sec, i.e. close to the S signals, with the exception that the dominant directionality was in most cases negative for S and positive for A.

The duration of the waves at their base was difficult to determine with accuracy as their bases often overlapped. Typically the estimated duration of waves S and A was about 200 msec while that of wave-D was somewhat longer, 200–300 msec.

Atrial wave A was absent in all 31 patients with AF across all lung fields, while waves S and D were present (Fig. 2A). During the presystolic Period, in the AF recordings there was usually a series of rapid oscillations, as seen in Figure 2B

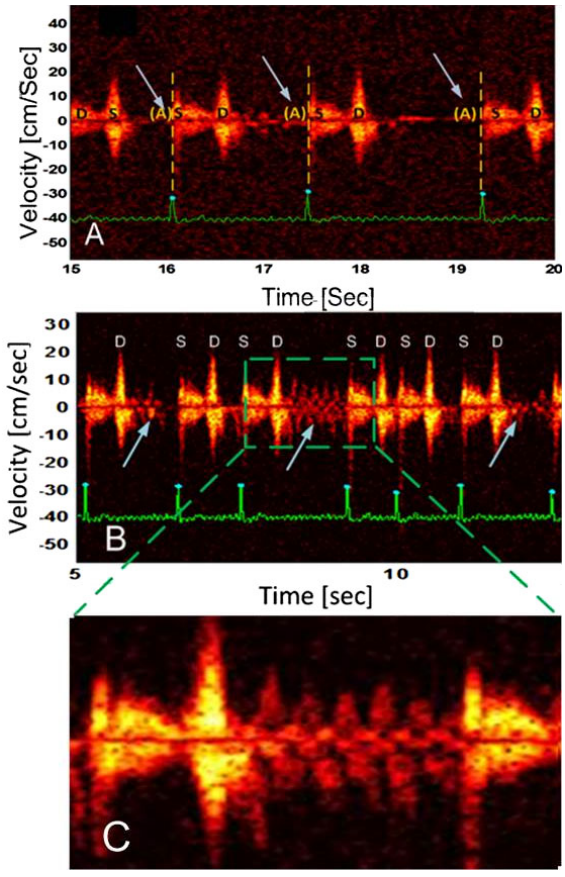

Figure 2. Lung Doppler Signals recorded from patients with atrial fibrillation. **A.** spectral record with typical systolic (S) and diastolic (D) waves without an atrial wave (A) in the presystolic period (diastasis) indicated by arrows. **B.** High frequency Doppler signals reflecting atrial contractions during diastasis. **C.** Expanded section of B illustrating the atrial waves.

and C. As the fibrillating atrium does not generate an effective blood pressure pulsation, the 6–10 Hz oscillation most likely reflects vascular wall vibration. The characteristics of these mechanical oscillations, which resemble the mitral valve oscillation sometimes observed by standard echocardiography in AF, may provide insight into the nature and pathophysiology of these conditions.

A distinct characteristic of the LDS in AF patients is the peak velocity D/S ratio. Table II lists the average D/S ratios for 34 subjects in four matched groups subjects; patients with AF without meaningful comorbidities, corresponding Controls, patients having AF and both CHF and PH and corresponding Controls. The average values of the D/S ratio of all AF patients are seen to be significantly higher than their corresponding Controls. The relative larger value of the D-wave in AF patients may be due to lack of back pressure generated by the left atrial contraction.

The 7 patients with atrial flutter showed a very distinct spectral pattern (Fig. 3): a high frequency Doppler velocity waveform, F, in sync with the ECG flutter waves (see vertical interrupted lines in Fig. 3). Here the single presystolic A-wave was replaced by the continuous train of F-waves (average positive velocities of 17.1 ± 4.2 cm/sec and somewhat smaller negative velocities) that superposed the S & D waves. The F-waves had both a positive (F+) and a negative component (F−) generally of the same basic frequency, however, often there was a phase shift between the positive and negative waveforms (Fig. 3).

Note that the characteristics of both AF and AFL were present in the recordings from all locations over the chest.

TABLE II

Comparison of AF and Controls

	AF	Control	AF + PH + CHF	Control + PH + CHF
Age	68.7	69.8	67.9	64.1
Sex	4/5 M/F	2/7 M/F	1/7 M/F	3/5 M/F
BMI	33.19	28.9	26.9	27.30
D/S	1.55	0.88	1.51	0.72
P value	0.0065		0.000035	

AF = atrial fibrillation.

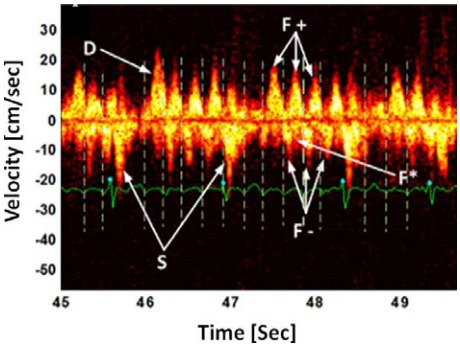

Figure 3. Biphasic high Doppler velocity signals recorded from a patient with AFL. The Doppler signals are in synchrony with the waves seen in the ECG. AFL = atrial flutter.

Discussion:

The mechanical activity of the heart is usually assessed by noninvasive cardiac imaging techniques such as echocardiography, or by invasive intracardiac pressure and blood flow measurements. The study presented here introduces a new modality for monitoring cardiac activity via its footprint in the pulsations of the pulmonary arteries and veins. Within this framework, one can regard the pulmonary blood vessels as "waveguides" that originate in the right ventricle (arteries) and left atrium (veins) from which they conduct, as mechanical waves, the pressure pulsations generated by the various cardiac events. As pulsations in the pulmonary arteries appear primarily during systole while the venous pulsations consist of both a diastolic and a presystolic component (generated by back flow from the left atrium[5]), the LDS can be assumed to represent mechanical activity in both the right ventricle and left atrium. It was previously suggested[1] that the LDS reflect the movements of the blood vessel wall–alveolar air interface, which is an almost perfect ultrasound wave reflector. Within this framework, it was suggested that the simultaneous bidirectional movement of the LDS is due to the fact that with each pressure pulse the highly compliant vessels expand such that the reflecting vessel wall closer to the transducer moves toward it while the opposite wall moves away from it. This mechanism is illustrated schematically in Figure 4. Furthermore, as there are pulsations in both pulmonary arteries and veins, we can assume that the LDS that originate from a whole vascular bed reflect both arterial and venous vessel elasticity characteristics. Note that one can diagnose pathologies, such as pulmonary fibrosis and edema in which fluid replaces alveolar-air, the lung becomes transparent such that Doppler blood flow signals appear in addition to the LDS.

The main finding of the study regarding AF is that when the main venous backflow wave (A) is missing,[1] small oscillations in the now silent presystolic trace can be clearly observed and studied (Fig. 2B). In the illustrated case, the fibrillation frequency is about 450/min and peak velocity is about 10 cm/sec. Note that this small and relatively weak wave can also be seen in the

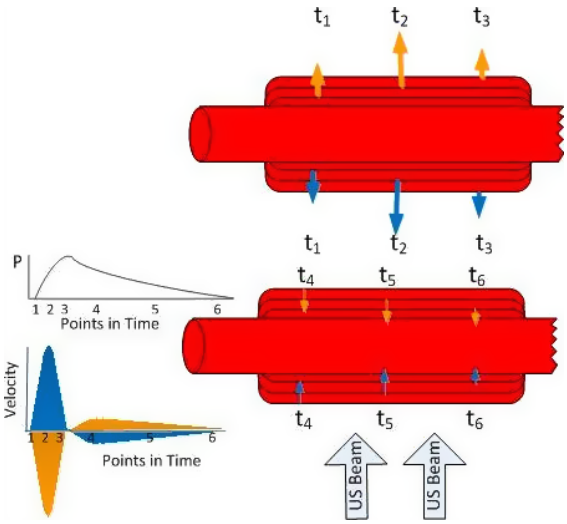

Figure 4. Schematic presentation of the suggested mechanisms of generation of lung Doppler signal generation. The LDS are generated by vascular wall/alveolar air border movement due to the vessel expansion with the blood pressure pulse (inset). Note that the return of pressure to nominal diameter is much slower than the upraise. LDS = lung Doppler signals.

previously published figures.[1,6] In view of the small size of the AF-waves in the ECG, it is not possible to compare the timing of the electric activity with the mechanical waveforms.

The second main finding presented here is the appearance of the F-waves typical of atrial flutter. As these high power signals that can be assumed to originate from well-structured synchronized contraction. The basic frequency of the F-waves is the same as that of the corresponding ECG waves indicating their common origin (Fig. 3). The surprising finding is that in different patients there is a variable time relationship between the positive and negative F-waves. The explanation for this finding is not known. It may be associated with atrial contraction and relaxation associated with each flutter wave.

As previously reported, the main advantages of the TPD as an ultrasound diagnostic tool are: as a nonimaging device it is relatively small and simple, easy to use, i.e. nonprofessionals can learn to operate it within a couple of days, the procedure is of very short duration (5–10 min), relatively easy to interpret, the procedure is relatively insensitive to mechanical artifacts such that it can used for monitoring moving subjects (a coin-like patch probe can be used), and finally, the velocity measurements are far easier to implement and more versatile than the corresponding Doppler capabilities of conventional cardiac ultrasound systems, which are dependent on imaging. Furthermore, it addresses both the cardiac and pulmonary vascular systems.

In summary, we believe that the data presented in this work indicate that the described novel Doppler recording modality may serve as a useful tool for studying specific aspects of the cardiac and pulmonary systems and provide valuable information of both pathophysiological and diagnostic nature.

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