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Factors Influencing Doppler Blood Flow and its Measurements

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1. Introduction

Doppler ultrasound has become one of the most important imaging modalities for measuring hemodynamic changes in various clinical settings. The accuracy of these measurements is the preconditioning to the appropriate interpreting of clinical observations and to the reasonable therapeutic strategy-makings. In this chapter, we studied two major factors that influence Doppler blood flow and its measurements. One is the intrathoracic pressure changes and the other is cardiac motion.

2. Research done by other researchers and our contribution

Respiration, including spontaneous and resistant respiration, can cause intrathoracic pressure changes, which would lead to Doppler flow alterations due to heart-lung interactions. Although spontaneous respiration on hemodynamics in humans has been studied and its regularity has been proved, quantitative evaluation of resistant respiration and forced maneuvers on hemodynamics and the underlying mechanisms has not been fully elucidated. We started to investigate comprehensively the factors that influence Doppler flow and its measurements fifteen years ago, and found that the intrathoracic pressure changes have great impact on Doppler blood flow and its measurements both in healthy subjects and patients with cardiopulmonary diseases. We comprehensively and quantitatively assessed the influence of spontaneous respiration and resistant respiration on cardiac transvalvular blood flow, superior vena caval and pulmonary venous blood flow in both healthy subjects and patients with pericardial effusion and chronic obstructive pulmonary diseases.

It is well known that blood flow velocity measured by Doppler ultrasound represents the net motion of the blood relative to the transducer. It is widely assumed that the measured velocity represents the actual flow. However, if the cardiac chamber or valvular annulus surrounding the blood has independent motion relative to the transducer, the flow we need to measure will be influenced by the motion of the heart and therefore will be the vector sum of the chamber or annulus velocity and blood flow velocity. For example, the pulsed Doppler flow velocity signal at the left ventricular outflow tract or through the aortic annulus is usually obtained from the apical five-chamber view. The blood moving away from the transducer during systole is ejected into the aortic root through the aortic annulus, while the annulus itself is moving towards the transducer at the same period of time. If we

need to measure the left ventricular stroke volume, we need the velocity of the blood relative to the aortic annulus, not to the transducer. While, the blood flow velocity “observed” by the transducer is only the velocity of the blood relative to the transducer. Therefore, cardiac output has been underestimated by the approach of Doppler echocardiography due to the aortic annulus motion opposite to the flow direction. On the other hand, Doppler echocardiography overestimates aortic flow routinely by measuring the outer edge of the spectrum. So, the routine Doppler method of stroke volume measurement seems to be accurate because the two errors are in the opposite direction and cancel each other. Similar situations will be encountered for right ventricular outflow or inflow tracts or the Doppler measurements of flow velocity of valvular stenosis or regurgitations. We think this problem of Doppler echocardiography should be stressed and the method of the correction of these errors should be explored as it has been either unrecognized or ignored in the clinical practice.

We designed an in vitro model that allowed us to observe and independently control the motion of a chamber containing a liquid and the motion of the liquid itself. This model enabled to observe the effects of the movement of the chamber on the Doppler signal derived from the motion of the liquid. We tested the hypothesis that cardiac motion can alter the velocity of the Doppler signal generated by blood flow in the in vitro model. We measured cardiac motion with M-mode echocardiography in human subjects (in vivo model) and compared the data with the Doppler signal generated by cardiac motion.

The in vitro and in vivo models were used in their study to demonstrate that cardiac motion can influence the measured Doppler signal generated by blood flow and to investigate the influence of the motion of the heart on measured velocity. In the in vitro model, the velocity of the cornstarch-water slurry and the motion of the apparatus were varied independently to simulate cardiac motion and blood flow. This procedure allowed us to separate the two components of the measured velocities. By stopping the motion of the piston, they could observe the pure flow profile, which could be kept constant. Similarly, the flow through the apparatus could be stopped, allowing us to measure the motion of the apparatus. They illustrated the potential alterations in peak velocities as well as changes in the slope of the velocity curve (acceleration) and were able to cause apparent changes in cycle length. Although some of the relations between the motion of tubing and the flow of the slurry do not appear naturally on a recurring basis, they illustrate the capacity of certain changes in timing to alter apparent flow patterns. Intermittent changes in cardiac motion relative to flow could be caused by atrial or ventricular ectopic activity or respirations, resulting in changes in cardiac motion. Because such changes might not be synchronized with flow alterations, changes in both the slope and the height of the Doppler signal might occur. The velocity of cardiac motion also varies throughout the cardiac cycle and therefore may not affect events at different parts of the cycle similarly. In measurements utilizing slopes, such as mitral valve half-time or pulmonary acceleration time, changes in cardiac motion might cause a significant change in a Doppler-derived measurement.

Their experiments in vitro and in vivo provided important information about the interaction of cardiac motion and blood flow in the Doppler spectrum. The measured Doppler spectrum of blood flow velocity is modified by cardiac motion and could be corrected for more accurate measurement.

In reviewing the previous study, we noticed that it is important to find that the measured Doppler spectrum of blood flow velocity is modified by cardiac motion in the in vitro model. However, in the experiment of the in vitro model, the simulated blood, the cornstarch-water slurry, was carried by the simulated vessel and, thus, the velocity of the simulated blood would naturally be the vector sum of the two motions, the motion of the simulated blood itself and the motion of the vessel, i. e., there should be no difficult to understand that the transducer would record a modified velocity. In the echocardiographic laboratory examination, however, if we need, for example, to measure the left ventricular stroke volume, the transducer should be located at the apex of the heart and directed to the base to display the 5-chamber view. During systole, the aortic valve is open and therefore, the aortic root does not carry the blood to move in any direction, which is not like the in vitro model's condition. Is the previous in vitro experiment still valid to provide the important information? To further confirm the previous conclusion and to find out if the conclusion is valid in the Doppler echocardiographic examination, we designed the following experiments.

3. Methods

3.1 Methods of studying the influence of intrathoracic pressure changes on Doppler flow

3.1.1 Doppler Echocardiography

Echocardiography was performed in all adult subjects with left decubitus position and spontaneous respiration by Siemens Sequoia 512 (Mountain View, CA, USA) with a 2.5–4.0 MHz transducer. Electrocardiogram and respiratory tracing by a nasal thermo-sensitive transducer (E99H450, fittings to Sequoia 512) were recorded simultaneously with echocardiograms. The protocol was approved by the Human Subjects Ethics Committee of the Fourth Military Medical University, and each subject provided an informed written consent.

The Doppler flow spectra across the four cardiac valves were recorded by pulsed Doppler echocardiography. The sample volume was put at the tips of mitral and tricuspid valves and 1 cm above the aortic and pulmonary valve. The superior vena cave (SVC) blood flow spectra were recorded by placing the transducer in the right supraclavicular region. The right-upper pulmonary venous flow was studied by pulsed Doppler from the apical four-chamber view, and the sample volume for Doppler examination was placed 0.5–1.0 cm into the right-upper pulmonary vein proximal to where it enters the left atrium and at an angle as parallel as possible to the direction of the blood flow.

3.1.2 Quantification of intrathoracic pressure

In order to quantify the influence of intrathoracic pressure on Doppler flow measurements, we developed an instrument that could quantify intrathoracic pressures. The device for quantification of intrathoracic pressure was refit by a watch sphygmomanometer, a mask and tubes. The examinees were told to press the mask tight against their nose and mouth, making the oral cavity and the thorax a closed system. Then, ask the examinees to inspire or expire through the mask of the device with their mouth open to generate negative or positive intrathoracic pressure changes. These pressure changes could be quantified and read directly from the attached watch sphygmomanometer.

3.2 Methods for studying the effect of cardiac motion on Doppler flow

We designed an in vitro model that allowed us to observe if the motion of the simulated blood vessel or heart chamber may modulate the Doppler spectrum of the motion of the simulated blood in it to further confirm the previous study. To test the hypothesis that cardiac motion can alter the velocity of the Doppler signal generated by blood flow and that the motion of the vessel would also influence the resultant flow velocity measurement, we used combined motion of the simulated blood vessel and the simulated blood flow. Using the measurement of left ventricular stroke volume as an example, we analyzed the error generated in the routine Doppler echocardiographic examination and proposed some approaches to correct it. We, then, discussed the theoretical meaning of the study.

3.2.1 Echocardiographic equipment

An Acuson Sequoia 256 ultrasound system with a 5- or 3-MHz transducer was used for the in vitro model experiments. Images were recorded on the hard disc in the equipment. Machine factors were adjusted as shows in the screen. It reads as follows: the pulsed Doppler transmit power was set at <100; the log compress was set at 35 dB; the length of the gate was 6.0mm; the width of the gate or the focus was 3mm. For the in vitro apparatus, the depth of the pulsed Doppler gate was 55mm, and the gate was placed within the middle of the straight portion of the tubing, away from the walls.

3.2.2 In vitro model

The in vitro model was designed to independently control the flow of simulated blood through a chamber and the motion of the chamber relative to the Doppler beam (Figure 1 and 2). A 220-cm length of silicon rubber tubing with an inner diameter of 6mm was connected to a variable pulse rate and flow velocity perfusion pump (Longer Pump YZ1515X). A 5% (5g Fiber/ 100ml tap water, equate Fiber Therapy, original texture.) fiber-water slurry was driven by the pulsatile pump through the tubing to simulate the blood flow within the vessel or heart chamber. Both the velocity of flow and the frequency could be independently controlled along a continuum. The flow rate for pulsatile flow through the pump was varied from 100 to 300ml/ min, corresponding to average linear velocities of 8 to 25cm/ s. A 6-cm section of the silicon tubing was fixed to a rigid plastic board (Figure 1b), and the board was cyclically moved by a specially designed machine (TD-4 cardiac motion simulator) to simulate the heart base motion. The tubing attached to the board was aligned with the echocardiographic transducer placed above it, and the motion of the board was parallel to the Doppler ultrasound beam as well. This eliminated possible artifacts from beam angulation. The pulsed Doppler gate was within the tubing, away from the wall and well above the curved portion of the tubing. The Doppler beam was parallel to the motion of the simulated blood and the tubing. The plastic board was coupled by means of gears, rubber bands and connectors to TD-4 cardiac motion simulator with a variable rate control. The board could be cyclically moved up and down to mimic the motion of cardiac base. Both the rate and the excursion of the board could be controlled and varied independently of the flow within the tubing. The motion of the board could be continuously varied over a range of velocities from 0.3 to 60cm/ s, and its maximal excursion for these series of experiments was 1 to 2cm. This is comparable to the motion of cardiac base in humans (Strunk et al., 1976). The tubing and board were placed within a

water bath, with the echocardiographic transducer above the vertical portion of the tubing.



Fig. 1a. Photo of the whole set of the in vitro model experiment. From the right to the left of the photo the whole set is displayed. Right: the perfusion pump which drives the simulated blood in the tubing; Middle (from top to the bottom): the top is the TD-4 cardiac motion simulator (specially designed and home-made) that moves the plastic board up and down; the middle is the gears and connectors that connects the plastic board below; the bottom is the water tank with the plastic board in it. The tubing with 5% fiber-water slurry, the simulated blood, is fixed on the plastic board that is connected with the connector above. The plastic board with the tubing may move up and down freely by the simulator. The echo transducer is separately fixed on the frame of the setup; Left: Ultrasound machine, Sequoia 256

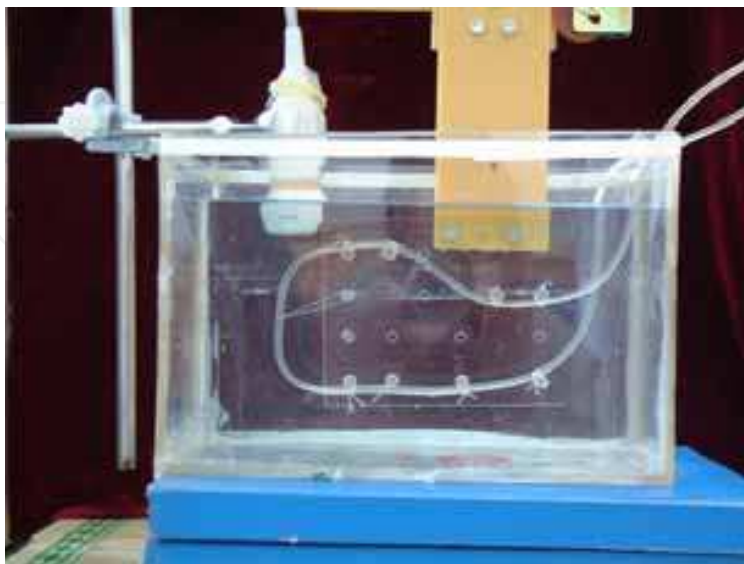


Fig. 1b. Photo of the plastic board. The board carries the tubing which should have a straight and vertical part outside the board

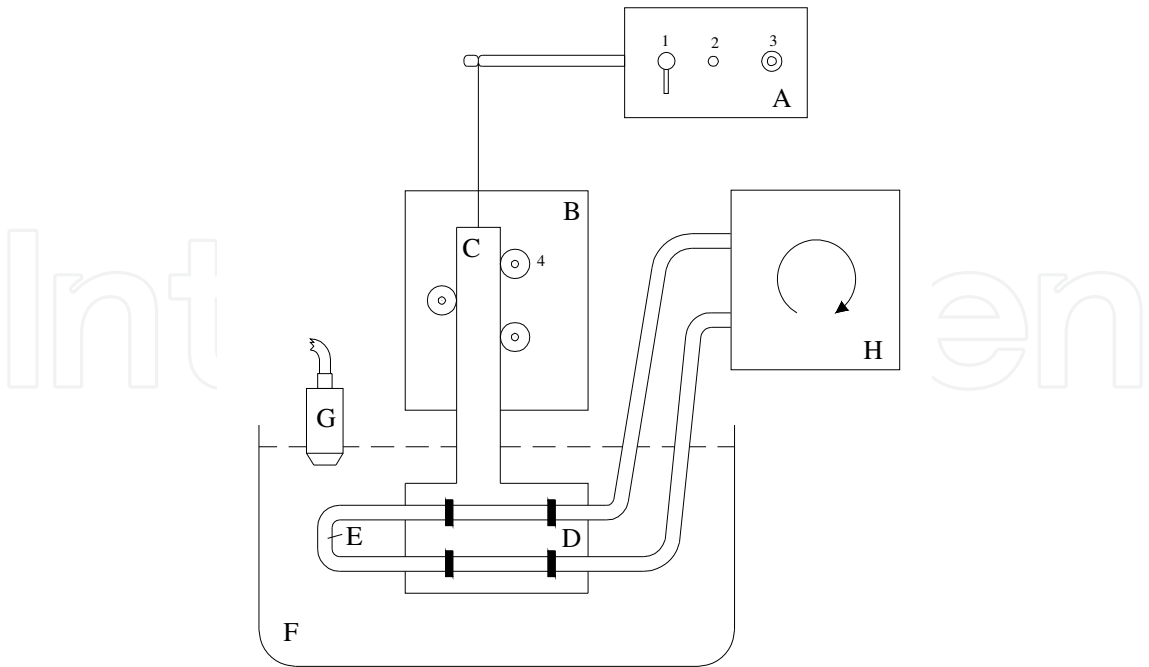


Fig. 2. Sketch of the in vitro model. A, the cardiac motion simulator; B, the connector with the gears; C, the connection of the connector with the plastic board; D, the plastic board that carries the tubing; E, the tubing with the fiber-water slurry in it; F, the water tank; G, the echocardiographic transducer

4. Results

4.1 Influence of the intrathoracic pressure changes on Doppler flow

4.1.1 Influence of spontaneous respiration on Doppler flow measurements in healthy subjects

4.1.1.1 Four cardiac valves

The transmitral and aortic Doppler flow velocities increased significantly on expiration compared on inspiration, while the tricuspid and pulmonary Doppler flow velocities increased significantly on inspiration compared on expiration (Figure 3-6, Table 1). The averaged respiratory variation index (RVI), which was calculated as the [(average velocity on expiration-the average velocity on inspiration)/ average velocity on expiration] for mitral valve, aortic valve and pulmonary vein, and [(average velocity on inspiration-the average velocity on expiration)/ average velocity on inspiration] for the four cardiac valves were shown in table 1.

	MV	AV	TV	PV
Inspiration	76.6±15.5	104.2±13.7	62.2±17.2	89.8±15.8
Expiration	83.8±17.7	109.4±14.1	52.8±14.1	84.8±15.0
RVI	8.4±3.7%	4.7±2.0%	16.4±5.7%	5.7±2.6%

Note: MV, mitral valve; AV, aortic valve; TV, tricuspid valve; PV, pulmonary valve.

Table 1. Doppler flow velocities across the cardiac valves during expiration and inspiration and the respiratory variation index

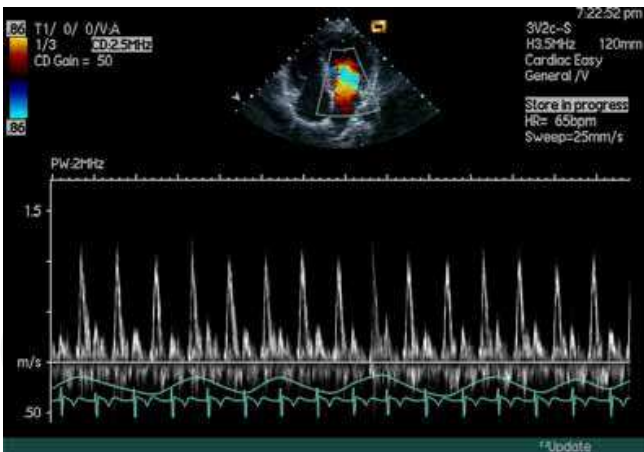


Fig. 3. The velocity across the mitral valve is decreased during inspiration and increased during expiration with RVI 8.4%. From top to the bottom are: color Doppler imaging, Doppler wave, respiratory wave (upward, inspiration; downward, expiration), and electrocardiogram

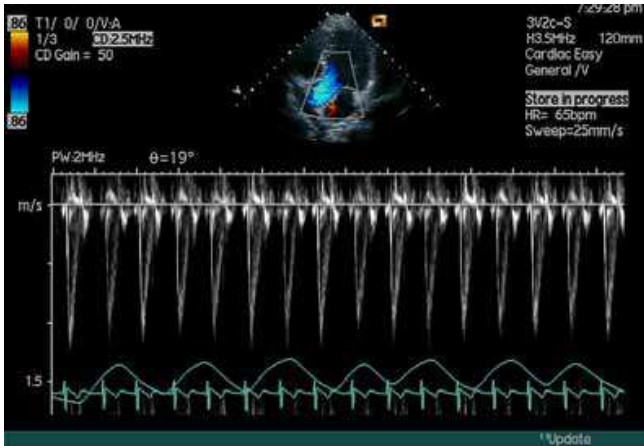


Fig. 4. The velocity across the aortic valve is decreased during inspiration and increased during expiration with RVI 4.7%

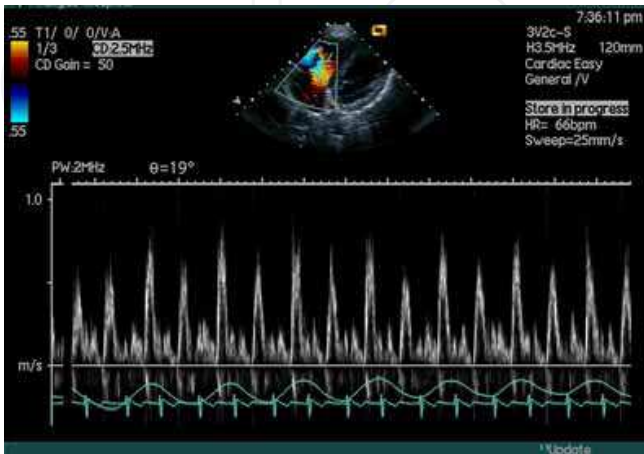


Fig. 5. The velocity across the tricuspid valve is increased during inspiration and decreased during expiration with RVI 16.4%



Fig. 6. The velocity across the pulmonary valve is increased during inspiration and decreased during expiration with RVI 5.7%

4.1.1.2 Pulmonary veins

The systolic peak flow velocity (PVs) and its integral (PV VTIs) of the pulmonary vein did not vary dramatically between inspiration and expiration; however, the diastolic peak flow velocity (PVd) and its integral (PV VTId) increased remarkably from inspiration to expiration, resulting in significantly decreased PVs/ PVd and PV VTIs/ PV VTId from inspiration to expiration.

4.1.1.3 Superior and inferior vena cava (SVC and IVC)

Both the systolic and diastolic peak flow velocities of SVC and IVC significantly increased from inspiration to expiration, while the reversed flow velocity during systole and diastole showed no significant difference between inspiration and expiration.

4.1.2 Influence of resistant respiration on cardiac valvular Doppler flow measurements in healthy subjects

There was no significant difference in RVIs between the spontaneous respiration and with the intrathoracic pressure change of -4mmHg, while significant difference in RVIs were found between the spontaneous respiration and with the intrathoracic pressure change of -8mmHg and -12mmHg, respectively (Figure 7-10, $p < 0.01$).

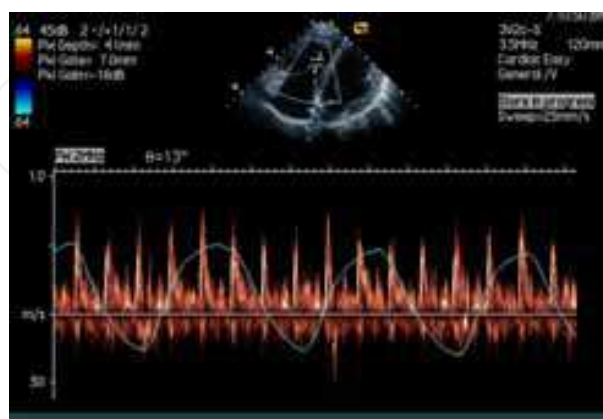


Fig. 7. Effects of spontaneous respiration on tricuspid flow. The velocity across the tricuspid valve is increased during inspiration and decreased during expiration with RVI 10.1%. From top to the bottom are: color Doppler imaging, Doppler wave, respiratory wave (upward, inspiration; downward, expiration), and electrocardiogram

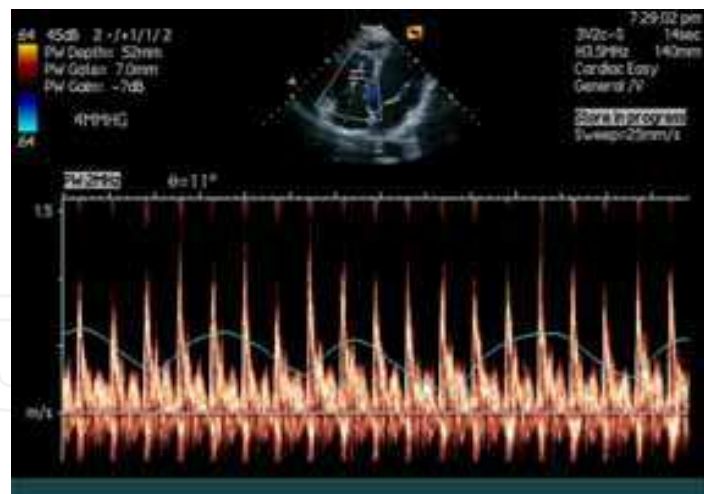


Fig. 8. Doppler spectra across the tricuspid valve with the intrathoracic pressure decrease of 4mmHg in the same subject as in figure 1. RVI is 11.35 %

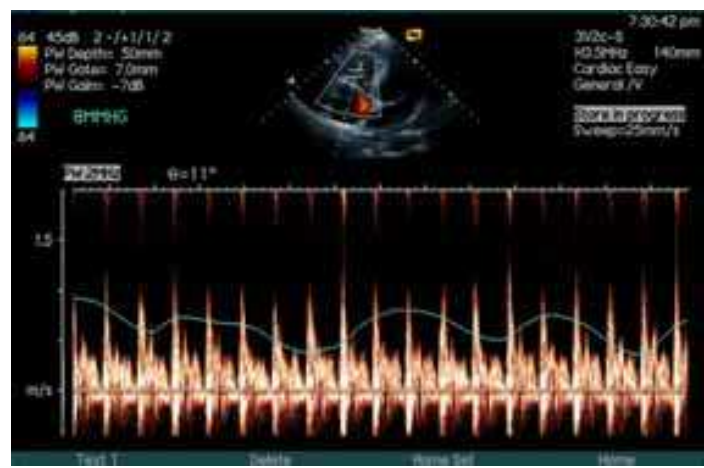


Fig. 9. Doppler spectra across the tricuspid valve with the intrathoracic pressure decrease of 8mmHg in the same subject as in figure 1. RVI is 19.0%

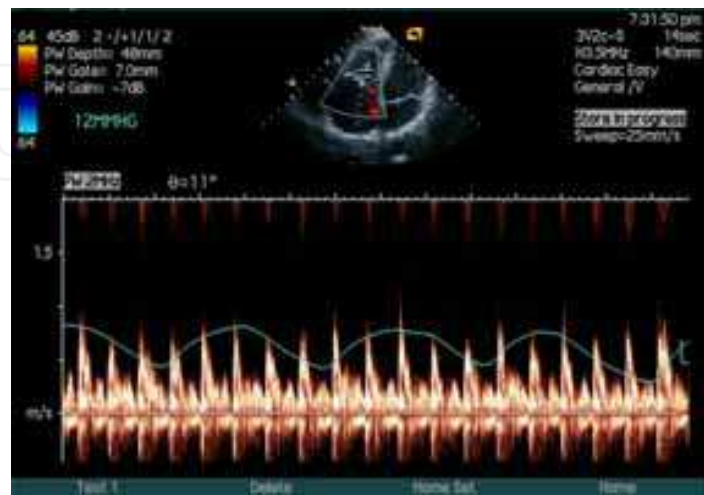


Fig. 10. Doppler spectra across the tricuspid valve with the intrathoracic pressure decrease of 12mmHg in the same subject as in figure 1. RVI is 21.3%

4.1.3 Influence of spontaneous respiration on Doppler flow measurements in patients with pericardial effusion

4.1.3.1 Four cardiac valves

In patients with pericardial effusion (PE), The RVI of the velocities across the mitral and the aortic valves increased significantly in patients with PE compared with normal subjects (Figure 11-14, Table 2).

Groups	MV	AV	TV	PV
Control	9.71±3.39	4.67±1.79	14.82±3.70	5.86±2.55
PE	14.57±7.89*	11.61±4.96**	24.97±6.19**	23.93±10.12**

Notes: PE, Pericardial effusion. *Compared to normal, p<0.05; **Compared to normal, p<0.001.

Table 2. Comparison of RVI between patients with PE and normal subjects

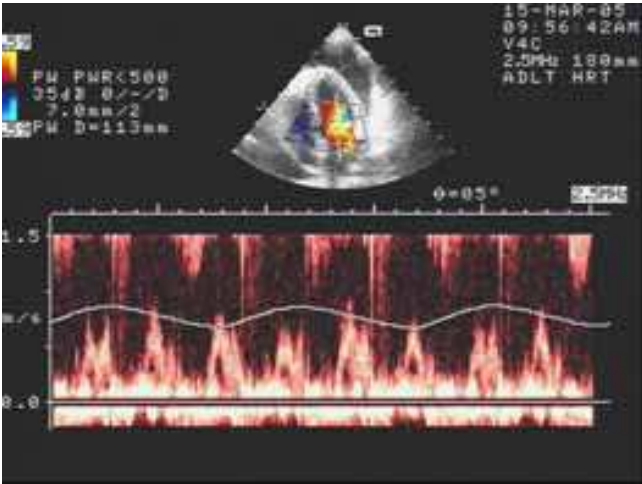


Fig. 11. RVI of the Doppler flow velocity across the mitral valve is significantly increased in a patient with large amount of pericardial effusion compared to normal subject shown in figure 1 (29.8% vs. 8.4%)

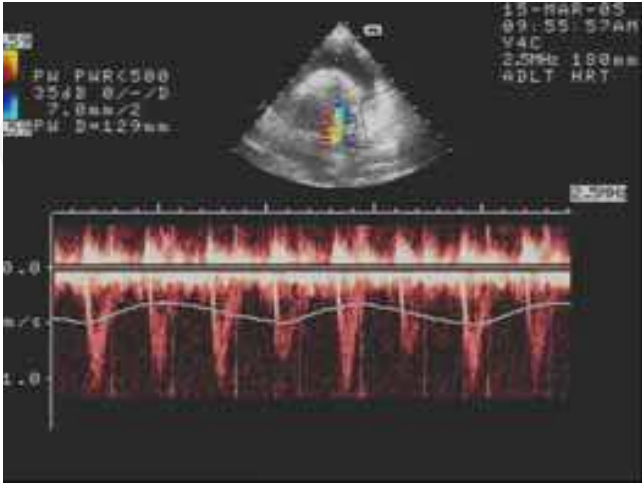


Fig. 12. RVI of the Doppler flow velocity across the aortic valve is significantly increased in a patient with large amount of pericardial effusion compared to normal subject shown in figure 2 (33.3% vs. 4.7%)

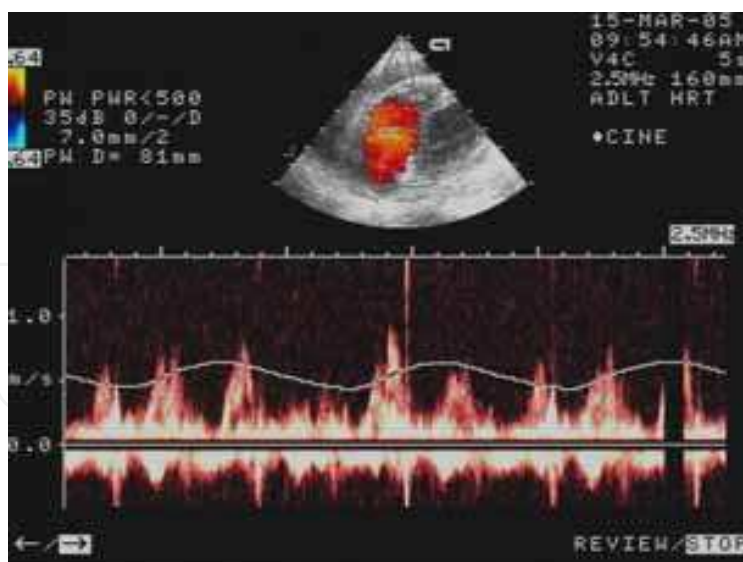


Fig. 13. RVI of the Doppler flow velocity across the tricuspid valve is significantly increased in a patient with large amount of pericardial effusion compared to normal subject shown in figure 3 (48.9% vs.16.4%)

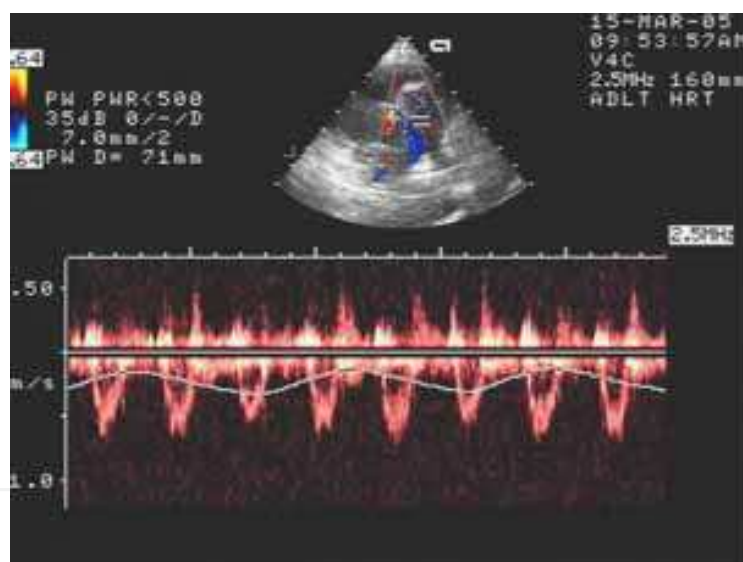


Fig. 14. RVI of the Doppler flow velocity across the pulmonary valve is significantly increased in a patient with large amount of pericardial effusion compared to normal subject shown in figure 4 (27.7% vs. 5.7%)

4.1.3.2 Inferior vena cava (IVC)

In patients with pericardial effusion, RVI of the systolic and diastolic peak flow velocities of IVC significantly increased compared to normal subjects ($p < 0.001$).

4.1.4 Influence of spontaneous respiration on Doppler flow measurements in patients with chronic obstructive pulmonary disease (COPD)

RVI of the four cardiac valves in COPD patients were significantly higher than that in normal subjects (Figure 15-18; Table 3)

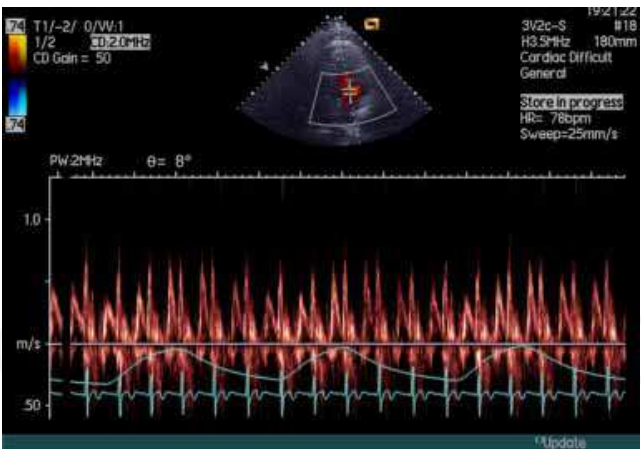


Fig. 15. RVI of the Doppler flow velocity across the mitral valve is significantly increased in a patient with obstructive pulmonary disease compared to normal subject shown in figure 1 (14.27% vs. 8.4%)

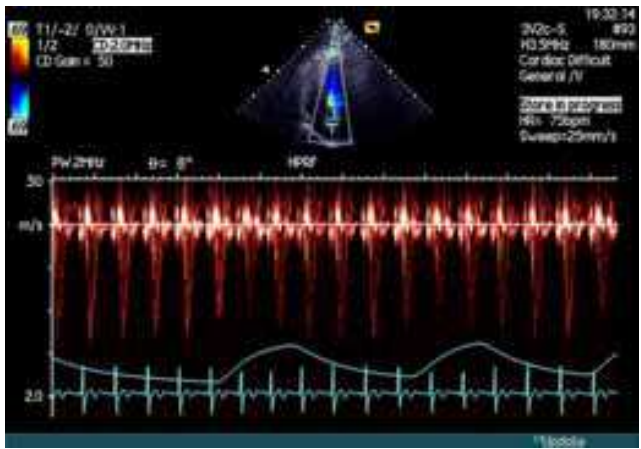


Fig. 16. RVI of the Doppler flow velocity across the aortic valve is significantly increased in a patient with obstructive pulmonary disease compared to normal subject shown in figure 1 (11.6% vs. 4.7%)

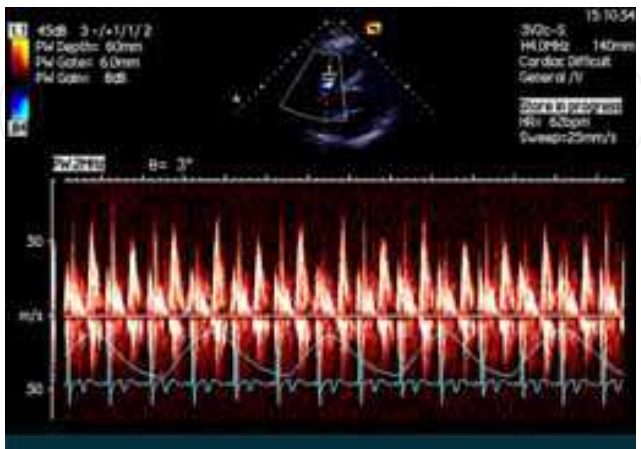


Fig. 17. RVI of the Doppler flow velocity across the tricuspid valve is significantly increased in a patient with obstructive pulmonary disease compared to normal subject shown in figure 1 (27.7% vs.16.4%)

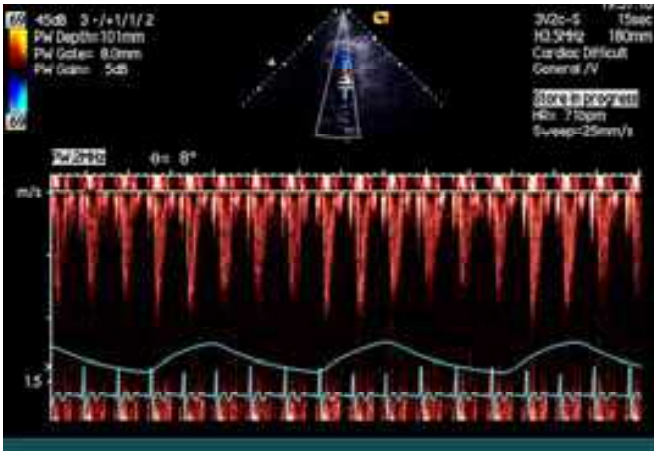


Fig. 18. RVI of the Doppler flow velocity across the pulmonary valve is significantly increased in a patient with obstructive pulmonary disease compared to normal subject shown in figure 1 (16.8% vs.5.7%)

Groups	MV	AV	TV	PV
Control	10.5±8.7	4. 7±1.8	14.9±6.3	8.6±2.3
COPD	16.3±8.7*	11.6±5.0**	27.7±8.4**	13.5±5.0**

Notes: COPD, Chronic obstructive pulmonary disease. *Compared to normal, p<0.05; **Compared to normal, p<0.001.

Table 3. Comparison of RVI between patients with COPD and normal subjects

With the whole set of the in vitro model devices, the Doppler spectra were recorded in series. Measurements of the velocity and amplitude of the tubing in the in vitro apparatus was either by the M-mode echocardiography or directly read from the Doppler blood flow velocity waveforms. The distance the tubing moved was determined by measuring the amplitude of the M-mode tracing of the tubing. It was compared with direct measurements of the distance that the apparatus moved. The sloped of the M-mode measurements represents the velocity of the tubing. We compared the M-mode measurements with the Doppler velocities obtained with the apparatus maintained at a constant speed.

4.2 Effect of cardiac motion on Doppler flow

Figures 19 to 21 demonstrate the Doppler signals derived from the motion of the silicon tubing, the motion of the fiber-water slurry within the tubing during peristaltic pumping and their combined motions. The motion of the tubing was parallel to the flow of the slurry within the tube. Figure 19 shows Doppler spectrum of the motion of the tubing being moved by TD-4 cardiac motion simulator while the fluid is not being pumped. The sample volume of the Doppler ultrasound is in the center of the tubing in figure 19. However, we noticed that we may record similar spectrum with the sample volume located anywhere outside the tubing, but close to it or to the plastic board. The apparent Doppler signal is a sinusoidal pattern with a maximal velocity of 15 cm/ s and occurs regularly with a frequency of about 2 Hz. Figure 20 demonstrates a typical pulsed Doppler recording generated by the pulsatile motion of the fluid alone while the tubing is stationary. The maximal velocity is approximately 45 cm/ s and occurs regularly with a frequency of about 4 Hz.

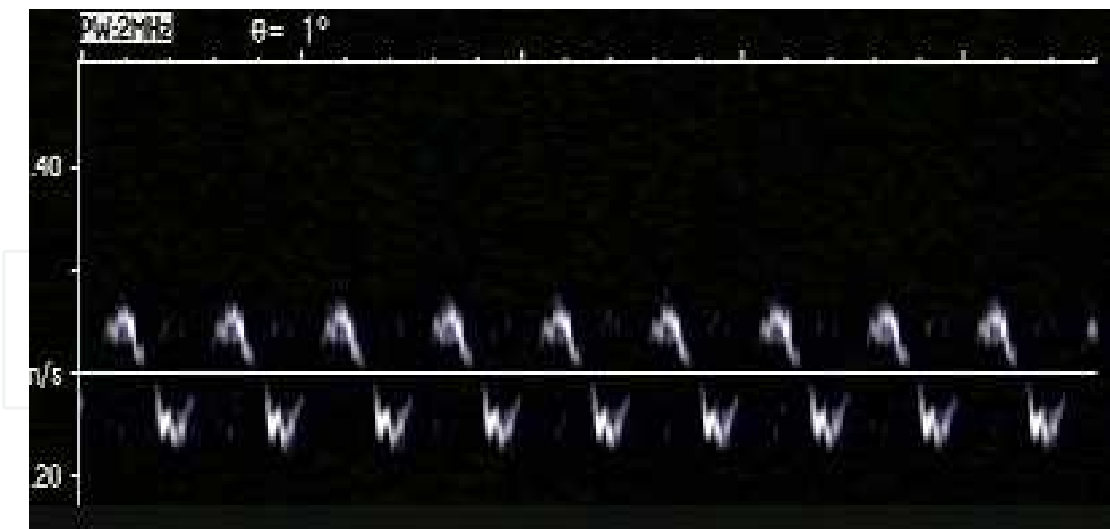


Fig. 19. Doppler signal derived from only the motion of the tubing. The fiber-water slurry is within the tubing but is not being pumped. The Doppler signal is generated entirely by the motion of the tubing, reflected by the fiber-water slurry

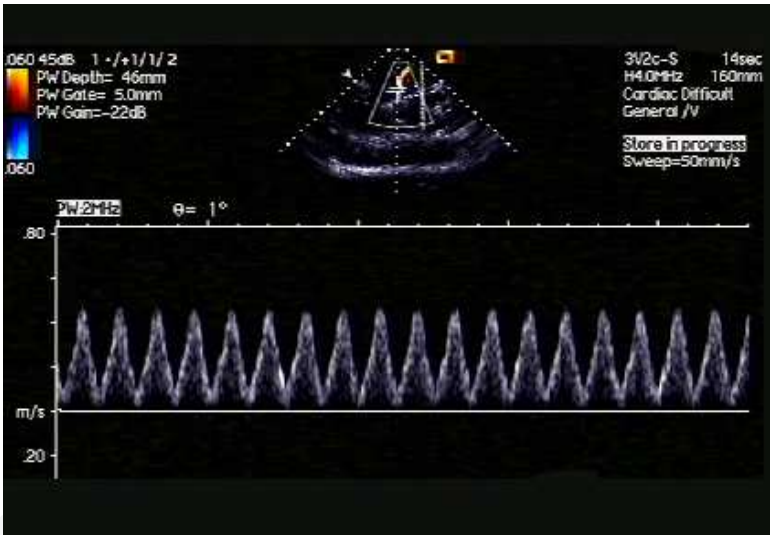


Fig. 20. Doppler signal derived from the pulsatile pumping of the simulated blood, the fiber-water slurry, while the tubing remains stationary

The complexity of the interaction between the motion of the tubing and the simulated blood is demonstrated in Figures 21, where the frequencies of the two motions are varied relative to each other. Flow of the simulated blood and motion of the tubing were controlled independently to create synchronized and desynchronized motion combination. We have recorded different combinations of the synchronized motions. But to make our description brief, we only use the desynchronized motion. The desynchronized motion actually covered the waveforms of all the motion combinations. In Figure 21, the frequency of the pulse rate of the peristaltic flow was slower than the motion of the tubing and is therefore slightly dissociated. This resulted in a continually varying alternant pattern of the peak amplitudes of the Doppler signals. The resultant waves change not only in amplitude (velocity), but also in apparent slope (acceleration and deceleration).



Fig. 21. Doppler signals generated by fluid and tubing motions. The frequency of the motion of the tubing and the frequency of the peristaltic pump were varied. The frequencies of two motions were very close, but desynchronized. The relative magnitude of the measured signals during the cycle varies even though the actual frequencies and velocities of motion and flow were constant. The two motions may have various phase combinations, yielding different modified Doppler flow velocity spectra

The Doppler signals of Figure 21 demonstrate the combined effect of motion of the tubing and flow through it. Although the actual velocity of the fluid through the tubing has not changed, its measured velocity has been altered by the motion of the tubing.

When the peak velocities are in phase (the ninth wave of the flow waveforms counted from the left side), the maximal velocities of both signals are additive, resulting in an increase in the measured velocity of the simulated blood. The maximal velocity of the wave is about 60 cm/ s that equals to the algebraic sum of the two maximal velocities, 45 cm/ s + 15 cm/ s.

When the peak velocities are out of phase (the eighth wave of the flow waveforms counted from the left side in figure 21), they negate each other. The measured velocity is thus the vector sum of the two velocities. The maximal velocities of both signals are subtractive, resulting in a decrease in the measured velocity of the simulated blood. The maximal velocity of the wave is about 30 cm/ s that equals to the subtraction of the two maximal velocities, 45 cm/ s - 15 cm/ s. Although flow through the tubing remains constant throughout, the desynchronized motion of the tubing (relative to the frequency of the pulsatile pump) results in an apparent twofold difference in velocity (30 to 60 cm/ s) between adjacent peaks.

The waves of 16th and 17th in figure 21 (counted from left side of the figure) demonstrates the Doppler flow pattern when the frequency of the motion of the tubing and the pulsatile flow are out of phase but are integral multiples of each other. When the flow and movement of the tubing are at similar frequency but out of phase, the observed relationship may have this pattern. Although the flow of the simulated blood is unchanged, the velocity patterns are quite different. These patterns were achieved merely by changing the degree of phase synchronization of the motion of the tubing relative to the simulated blood. The resultant Doppler spectrum of the combined motion is the algebraic sum of their Doppler signals, resulting in apparent changes in maximal velocity as well as slope of the velocity curve. There are different combinations of the two motions in figure 21.

5. Discussion

The present study shows that both spontaneous and resistant respiration could have great impact on the Doppler flow velocity measurements, and this impact may be augmented in some diseased states, including pericardial effusion and chronic obstructive pulmonary diseases. The results of these experiments revealed the influence of intrathoracic pressure changes on Doppler blood flow and furthered the understanding of the mechanism of respiration-driven hemodynamics or heart-lung mechanical interactions that have remained controversial for over a century.

Similar findings of spontaneous respiration effects on Doppler flow velocities across the mitral and tricuspid valves in adults were reported previously (Dabestani et al., 1988). Some other studies investigated the influence of respiration on Doppler flow velocities across the mitral and tricuspid valves in children aged from 3 to 12.5 and from 1.5 to 11, respectively, and found that the early peak flow velocity across the mitral valve decreased 7% and 8% (Alehan, et al., 1996; Riggs, et al., 1989), which are similar to those in the adults shown in the present study.

The present study showed that the diastolic peak flow velocity and its integral increased remarkably from inspiration to expiration, resulting in significantly decreased PVs/ PVd and PV VTIs/ PV VTId from inspiration to expiration. The similar respiratory effects on the pulmonary venous flow were also demonstrated in one study using transesophageal pulsed Doppler echocardiography. In addition, this study showed that compared to the spontaneous respiration, Valsalva maneuver has greater impacts on the pulmonary venous flow (Meijburg et al., 1992). These effects should be considered when the pulmonary venous Doppler variables are applied for clinical purposes, and it would be of importance if considering the respiratory variation of the pulmonary venous flow when adopting PVs/ PVd to identify pseudonormal left ventricular function.

In the condition of resistant respiration in normal subjects or in patients with chronic obstructive pulmonary diseases, in which the intrathoracic pressure changes are increased as in the resistant respiration, the impacts of respiration on Doppler flow measurements are much greater than that in normal subjects. Thus, the respiratory variation index may be an alternative parameter for assessing the severity and the therapeutic effect of COPD in clinic. This should be also applied in patients with pericardial effusion and the respiratory variation index of these Doppler flow velocities may better reflect the severity of pericardial effusion combined with the quantity of the fluid collection.

It has long been known that the interventricular septal shift that periodically occurs in the short-axis of the right ventricle and left ventricle is the key point of respiration-driven hemodynamics (Masuyama et al., 1986; Miller et al., 2006; Mitchell et al., 2005; Neumann et al., 2005). However, the mechanism of the septal shift has remained controversial.

In spite of its complexity, the circulatory system can be regarded hydromechanically as two enclosed fluid systems which are connected sequentially. One is the fully-intrathoracic fluid system, which includes the left ventricle, left atrium and pulmonary veins, and the other one is the partially-intrathoracic fluid system, which includes the right ventricle, right atrium and the vena cavae. Based on Pascal's law, the intrathoracic pressure changes during respiration may be transmitted without loss to all areas within the chest, including the fully-intrathoracic system. On the contrary, the intrathoracic pressure changes affect only the intrathoracic part, i.e., the right ventricle and atrium of the partially-intrathoracic system. With inspiration, the right ventricular pressure decrease will be mostly compensated by

venous return from the peripheral veins. Thus, the pressure difference occurs between the two sides of the interventricular and atrial septa would push the septa to move toward the left ventricle and left atrium on inspiration, and vice versa. So the filling of the left ventricle and left atrium is impeded, and consequently the blood flow velocities across the mitral and aortic valves decrease on inspiration compared to those on expiration. In the condition of chronic obstructive pulmonary disease, the intrathoracic pressure changes is greater compared to normal subjects, thus, the respiratory variation of the cardiac blood flow velocities is increased. For the pericardial effusion where the pericardial pressure usually increases, the pressure difference between the two sides of the interventricular and atrial septa is exaggerated, and thus causes greater respiratory variation of these Doppler parameters.

The motion of blood cells within the heart or great vessels consists of the propulsion of blood cells driven by the contraction of the heart and the motion of blood cells as a component of the chamber, which is moving cyclically. In the past, the “flow” signal generated by cardiac motion was neglected.

The motion of the base of the heart has been observed by many investigators (Isaaz et al., 1993). Atrioventricular and semilunar valvular annuli and the fibrous framework in the adjacent tissues should have similar patterns of motion because they move together as part of the base of the heart (Ormiston et al., 1993). The blood flowing through the chambers or the great vessels will have independent motion relative to the transducer. What the transducer observes, for example, in the apical five-chamber view, is motion of the annuli toward the transducer during systole while blood is flowing in the opposite direction from the aorta. We mimic this with our apparatus. By positioning the tubing and motion of the board along the ultrasound beam of the transducer, we were able to eliminate problems of angle correction from our calculations. The impact of the twisting motion that occurs in normal hearts was not included in our in vitro analysis. Although the motion of the heart is complex, the ultrasound transducer observes only one-dimensional signals in both Doppler and M-mode studies.

Even though the Doppler sample volume had a focus of 3 mm within the central portion or bore of the tubing, we believe that the signals generated by the motion of the tubing are caused primarily by reflection of the ultrasound beam off the walls of the tubing and are a result of beam width. Some of this signal may also be generated by the fiber particles close to the wall, which move with the tubing as it accelerates. We placed the Doppler sample volume outside the tubing, in the water bath itself, and outside the cardiac chamber, within muscle (data not shown), and we were still able to record the Doppler signals, although they were much weaker. Whether the source of the signal is reflectors adjacent to the wall of the chamber, the chamber itself or beam width artifact, these signals alter the measured signal generated by blood flow, and the direction and timing of the alteration correlate with the motion of the chamber.

The measured Doppler spectrum of blood flow velocity in clinical echocardiography always has the built-in error caused by cardiac motion. The resultant spectrum is different from the pure spectrum, not only in the amplitude of the peak velocity but also in the slope of the measured velocity. Thus, cardiac motion can alter the velocity signals used to measure hemodynamic variables. In mitral inflow, for example, the maximal velocity, the half-time and the relation between the E and A waves could be altered by movement of the annulus. Because the direction and the velocity of the motion of the annulus vary throughout the

cardiac cycle, the slopes of the E and A waves and their relative heights might not be affected equally. This observation may in part explain the difficulty in utilizing mitral inflow to analyze diastolic events (Shapiro et al., 1991). The changes seen in diastolic filling that are attributed to changes in compliance and relaxation might be overshadowed by the changes due to cardiac motion. This might further obscure the already difficult to understand effects of diastolic relaxation on the Doppler signal. Measurements of mitral valve area might also be inaccurate because of changes in slope caused by cardiac motion. Another example of problems that can be created by not considering the effect of cardiac motion on Doppler measurements is the use of the pulmonary artery flow acceleration time to diagnose pulmonary hypertension. Cardiac motion could alter the slope and therefore the acceleration time.

In this and our previous study, the in vitro models demonstrated an important phenomenon: the measured Doppler spectrum of blood flow velocity in clinical echocardiography has been modulated by the cardiac motion. The resultant spectrum is different from the pure spectrum. This may be easily understood as both the in vitro models used the tubing that carried the simulated blood. When the tubing is moving with a moving simulated blood in it, the velocity of the tubing motion is naturally added to the velocity of the blood. However, the clinical situation is different. In the Doppler echocardiographic settings, the heart chamber or the great vessels do not carry the blood moving. For example, the pulsed Doppler flow velocity signal at the left ventricular outflow tract or through the aortic annulus is usually obtained from the apical five-chamber view. The blood moving away from the transducer during systole is ejected into the aortic root through the aortic annulus, while the aortic valve is open and, thus, it does not carry the blood moving which is different from the situation of the in vitro models. So, it is hard to explain and compare the two situations. However, based on the theoretical analysis and our this study, we understood that when the blood is ejected into the aortic root in systole, the aortic annulus itself is moving towards the transducer at the same period of time. Now we may explain that the two motions in the opposite direction should be added. That means If we need to measure the left ventricular stroke volume, we need the velocity of the blood relative to the aortic annulus and that is the vector sum of the two velocities, or we may explain that the algebraic sum of the velocity of the aortic blood flow away from the transducer and the velocity of the aortic annulus towards the transducer is the net flow velocity of the blood relative to the annulus.

The measured Doppler spectrum of blood flow velocity in clinical echocardiography always has the built-in error caused by cardiac motion. We believe, however, that one could partially correct for the error generated by cardiac motion by using the M-mode recording to measure cardiac motion or by measuring the Doppler signal derived from the cardiac motion and using one of these to correct the measured Doppler velocity signal or we can use Doppler tissue imaging modality attached to some ultrasound equipments to measure the velocity of the adjacent annulus or wall and correct the error. These methods of correction may be useful, but they are time-consuming and not convenient in the clinical echocardiographic practice. New, simple and convenient methods need to be explored.

With the development of Doppler tissue imaging (DTI), the direct measurement of the tissue velocity becomes available (Isaaz et al., 2000). Using DTI, the velocity measurements of the cardiac chamber walls or valvular annuli become convenient and simple. We are now on the work of this research and hope get the results soon. As mentioned above, the routine

Doppler method of hemodynamic data analysis, for example, the stroke volume measurement seems to be accurate because the two errors are in the opposite direction and cancel each other. In addition to correct the errors of the Doppler spectra caused by cardiac motion, we need to explore new modality of tissue velocity measurement. The use of the outer edge of Doppler spectrum as the velocity will overestimate the value and to obtain accurate blood flow velocity measurement, we need to explore a method to obtain the average special velocity of the blood cells in the measured cross-section of the vessel at any moment and then we may have an accurate flow velocity curve. To correct these errors, there is much to be done.

6. Conclusion

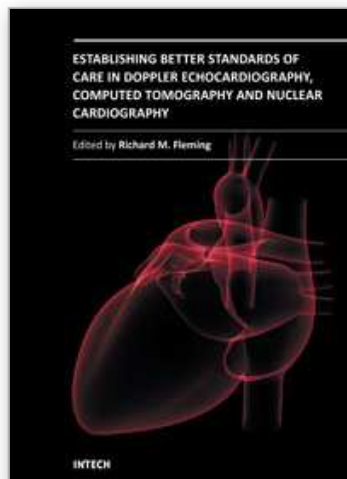
Intrathoracic pressure change is one of the factors that influence Doppler blood flow and its measurements. These effects should be considered when applying these parameters for clinical purposes. In addition, the respiratory variation index may be an alternative parameter for assessing the respiratory-related hemodynamic changes both in normal condition and in patients. The measured Doppler spectrum of blood flow velocity in clinical echocardiography always has the built-in error caused by cardiac motion. The resultant spectrum is different from the pure spectrum, not only in the amplitude of the peak velocity but also in the slope of the measured velocity. In addition to correct the errors of the Doppler spectra caused by cardiac motion, we need to explore new modality of tissue velocity measurement.

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