

Effects of Respiratory Cycle on Pulmonary Venous Flow and Cardiac Cycle on Pulmonary Venous Diameter of Dogs: A Transesophageal Echocardiography Study

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ABSTRACT. We investigated 12 anesthetized normal dogs using transesophageal echocardiography to understand the effects of respiration on the pulmonary venous flow. Additionally, we observed whether the diameter of the pulmonary vein changes with the heart beat. The pulsed Doppler wave form of pulmonary venous flow predominantly demonstrated two backward flows, with one peak occurring during ventricular systole and another during ventricular diastole. Sometimes a small forward flow occurred during left atrial contraction. In comparison with expiration, the peak velocity and velocity-time integral of the flow wave under inspiration occurred during both systole and diastole were significantly smaller. The diameter of the pulmonary vein decreased during left atrial contraction and increased during left ventricular systole and diastole.—**KEY WORDS:** canine, pulmonary venous diameter, pulmonary venous flow.

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Transesophageal echocardiography has rapidly become an accepted clinical procedure in human cardiology in the past decade. It is safe and non-invasive, except for the required local anesthesia of the oropharynx. It also provides high-quality images for monitors and has few impediments to surgeons during cardiac operations. Recently, using transesophageal pulsed Doppler echocardiography to assess the pulmonary venous flow (PVF) has become a widely accepted new method to help evaluate left ventricular diastolic function of various cardiac diseases in human medicine [5, 14]. It has been reported that there are several influential factors, such as heart rate and respiration, in the analysis of the PVF [7, 12]. In human medicine, the variable of the PVF recorded during the expiratory period is assessed routinely, since it is reported that the change in intrathoracic pressure during respiration may affect the value of the PVF. However, the effect on the PVF is still controversial. As a clinical utility, simply multiplying the velocity obtained from Doppler echocardiography by the cross-sectional area of a vessel or orifice gives an instantaneous flow rate. This product is then integrated over the cardiac cycle to yield the time average flow rate [1]. The volume of flow passing through a vessel can be estimated by this method. It is of interest to know whether the volume of the PVF can be estimated from the Doppler echocardiography. Therefore, as a preliminary study, this study was conducted to recognize the effect of the respiratory cycle on the PVF and to observe how the pulmonary venous diameter (PVD) changes during the cardiac cycle using transesophageal echocardiography.

In the present study, we examined 12 mongrel dogs, each weighing from 5.5 to 10.5 kg (mean weight 7.71 ± 1.62 kg, 3 males and 9 females), obtained from an animal shelter. These dogs were identified as healthy by the results of

physical examination and hematology, and the ages were estimated among 1 to 3 years old according to previous report [4]. There was no evidence of cardiac or pulmonary abnormality based on either auscultation or two-dimensional echocardiography. No *Dirofilaria immitis* infection was identified by microfilarial detection and immunodiagnostic test (VetRED®, AGEN Biomedical).

These dogs were anesthetized for insertion of the transesophageal probe. The anesthetic protocol included premedication with atropine (0.025 mg/kg, IM) and droperidol (0.5 mg/kg, IV), induction with pentobarbital sodium (20 mg/kg, IV), and maintenance with halothane (1–1.5%) under spontaneous respiration. The dog was initially examined at left lateral recumbency. A 5 MHz biplane transesophageal probe (EUP-ES322, Hitachi Medical Corporation) connected to an ultrasound instrument (EUB-565A, Hitachi Medical Corporation) was navigated according to the method reported by Loyer and Thomas [6]. With the transesophageal probe in an unflexed position, the imaging plane was switched to transverse plane, and the probe was advanced into the stomach until the liver image was visible on the screen. The denominated middle position was achieved by slowly withdrawing and slightly flexing the probe from the stomach to a position just caudal to the point where interference by the trachea was encountered. In this position the probe was located caudal to the tracheal bifurcation and dorsal to the left atrium, between the cranial and caudal pulmonary veins.

To observe the caudal pulmonary veins, we slightly advanced the probe again to a position between the right and left caudal pulmonary veins (Fig. 1-A). This position was located between the middle and the caudal position that was described by Loyer and Thomas. The imaging plane was maintained in transverse plane to produce a long-axis four chamber view. To reduce the angle between the PVF and the Doppler sampling beam, we rotated the probe clockwise enough for adequate right caudal lobe PVF recording with pulsed Doppler mode (Fig. 1-B). In addition,

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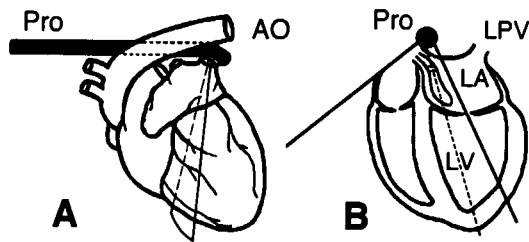


Fig. 1. Schemes of the location of pulmonary veins relative to transesophageal probe (A), and the echocardiographic imaging plane for recording right caudal pulmonary venous flow (B). AO=aorta; LA=left atrium; LPV=left caudal pulmonary vein; LV=left ventricle; Pro=transesophageal probe.

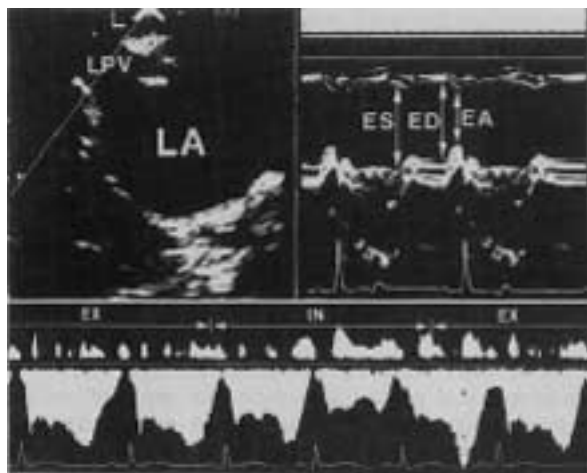


Fig. 2. The imaging plane on the top demonstrates M-mode measurement of the left caudal pulmonary venous diameter (left) and the changes of pulmonary venous diameter during cardiac cycle (right). At the bottom, the pulsed Doppler wave form of pulmonary venous flow changes with respiration. Both the S wave and the D wave decreased during inspiration in comparison with expiration. EA=diameter during the end of left atrial contraction; ED=diameter during end-diastole; ES=diameter during end-systole; EX=expiration; IN=inspiration; LA=left atrium; LPV=left caudal pulmonary vein.

the two-dimensional color Doppler image was used for alignment of the blood flow with the sampling line. The size of sample volume was set at 2 mm and the screen sweep speed of the ultrasound instrument was set at the maximum (1.25 sec/screen). The pulsed Doppler waveform of the PVF was recorded along with marking out the inspiration or expiration period. After the right caudal lobe PVF was recorded, the dog was turned over to right lateral recumbency to measure the PVD of left caudal pulmonary vein. The imaging plane was switched to longitudinal plane and the probe was advanced slightly further. In order to observe the left caudal lobe pulmonary vein, the tip of the probe was flexed slightly toward the right, that is, the left side of the dog. In this imaging view, the direction of the left caudal lobe pulmonary vein was almost perpendicular

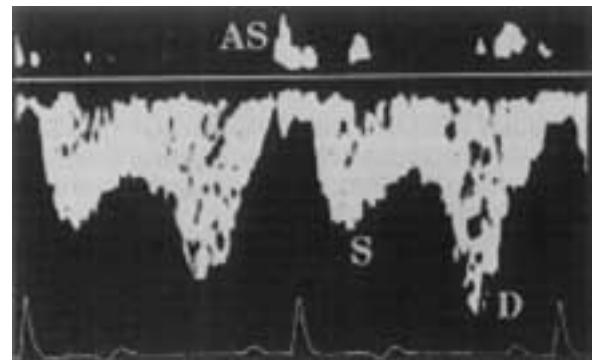


Fig. 3. The normal pulsed Doppler velocity pattern of the dog recorded by transesophageal echocardiography. AS=the small forward flow during left atrial contraction; D=the backward flow during left ventricular diastole; S=the backward flow during left ventricular systole.

to the M-mode beam line (Fig. 2). Then the diameter change of the pulmonary vein during cardiac cycle was simultaneously recorded by M-mode with electrocardiogram. The graphic information was recorded on a S-VHS videocassette recorder for retrospective analysis.

The flow velocity tracings were measured by the moving of a track ball to determine the peak velocity and the velocity-time integral (VTI) of the wave forms occurring at ventricular systole (S wave) and diastole (D wave). All measurements were calculated by the software operating function in the ultrasound instrument. The VTI was defined as the area under the outer border of pulsed Doppler velocity tracing. The systolic fraction of PVF was expressed as dividing the sum of the VTI of the S and D waves into the VTI of the S wave. The diameter of pulmonary vein was calibrated by linear measurement on the M-mode tracing (Fig. 2). The peak percent change in the diameter of pulmonary vein was expressed as dividing the difference between the largest and the smallest diameter by the largest diameter, and the average of these peak percent changes was described as the mean peak percent change. Each variable was obtained by averaged two or three consecutive heart beats, and all values were expressed as mean \pm SD. Test of significance was analyzed by the Student *t*-test and the $p < 0.05$ was considered significant.

In this study, adequate recordings of the PVF and the PVD were obtained in ten of 12 dogs. Two dogs were excluded because the pulsed Doppler trace disappeared or artifact emerged when the sampling volume moved out of the pulmonary vein during inspiration, which made the evaluation unsuitable. Therefore, the variables were analyzed in the remainders. The wave form of the PVF recorded by transesophageal echocardiography demonstrated two backward waves in all ten dogs, with one peak occurring during ventricular systole and another during ventricular diastole (Fig. 3). Also, a small forward flow was sometimes observed during the left atrial contraction. Results of data analyses showed that the mean peak velocity and the VTI

Table 1. Effects of respiration on pulmonary venous flow velocities and velocity-time integrals as obtained in 10 normal anesthetized dogs

| | Expiration | Inspiration |
|--|----------------------------|-------------------------------|
| Peak velocity of systolic flow (S) (cm/s) | 34.93 ± 9.03 ^{d)} | 26.58 ± 2.96 ^{a),e)} |
| Peak velocity of diastolic flow (D) (cm/s) | 55.80 ± 13.97 | 40.02 ± 9.21 ^{c)} |
| S/D velocity ratio | 0.63 ± 0.1 | 0.66 ± 0.1 |
| VTI of systolic flow (VTIs) (cm) | 7.20 ± 1.91 ^{d)} | 4.39 ± 1.80 ^{b)} |
| VTI of diastolic flow (VTId) (cm) | 8.81 ± 2.50 | 4.81 ± 2.00 ^{c)} |
| Systolic fraction (VTIs/VTIs+VTId) | 0.45 ± 0.03 | 0.48 ± 0.06 |

VTIs, d=the velocity-time integral of systolic flow or diastolic flow.

a) p<0.005 vs. Expiration. d) p<0.01 vs. VTId of Expiration.

b) p<0.001 vs. Expiration. e) p<0.001 vs. D of Inspiration.

c) p<0.0001 vs. Expiration. f) p<0.0001 vs. D of Expiration.

of the D wave were significantly greater than those of the S wave during expiratory duration (55.80 ± 13.97 cm/sec vs. 34.93 ± 9.03 cm/sec, $p<0.0001$ and 8.81 ± 2.50 cm vs. 7.20 ± 1.91 cm, $p<0.01$, respectively). The wave form of the PVF during inspiratory duration was similar to that obtained during expiration, showing a diastolic predominance with the velocity and the VTI of the D wave greater than those of the S wave. At inspiratory duration, the mean peak velocities of the D and S waves were 40.02 ± 9.21 cm/sec and 26.58 ± 2.96 cm/sec, respectively, and the VTI of the D and S waves were 4.81 ± 2.00 cm and 4.39 ± 1.80 cm, respectively (Table 1). When comparing the values of the PVF of inspiration with those of expiration, both the S and D wave, the mean peak velocity and the VTI were all significantly smaller at inspiratory duration (Fig. 2). However, the systolic fractions of the PVF were 48% during inspiration and 45% during expiration, which showed no significant difference between each other.

In the present study, the PVD varied dynamically during the cardiac cycle. The diameter was smallest during the end of left atrial contraction (4.85 ± 1.20 mm) as matched with the timing on the electrocardiogram. It then extended before the next brief contraction, which preceded left ventricular systole. When the left ventricle contracted, the PVD extended, becoming largest at the end of left ventricular systole (7.43 ± 1.72 mm). During the end of left ventricular diastole, the PVD was smaller (6.14 ± 1.78 mm) than that during left ventricular systole, but larger than that during left atrial contraction. The peak percent change in the diameter of pulmonary vein in the study group distributed from 26% to 49%, and the mean peak percent change in diameter was 35%.

In human medicine, the influence of respiration on pulmonary venous flow as assessed by transthoracic or transesophageal echocardiography has been reported as trivial [5, 7]. In our study, the peak velocity and the VTI of the PVF in anesthetized dogs showed a significant decrease during inspiration in comparison with the value obtained at expiratory duration. On the other hand, the wave form during expiration was stable enough for consecutive recordings of the PVF during several cardiac cycles. Summer *et al.* [13] have postulated that the negative pleural

pressure impedes left ventricular ejection. During inspiration, the pleural pressure and thoracic aortic pressure decrease, and in effect, the afterload of the left ventricle increases. Additionally, some investigators have proposed that a shift of the ventricular septum toward the left ventricle during inspiration would increase left ventricular diastolic pressure [11], and others have suggested that inspiration increases venous capacitance of the pulmonary vessels and reduces venous return to the left heart [10]. It has been thought that both mechanisms, that is, increased afterload and diastolic pressure would result in a reduction of pulmonary venous return to the left heart. However, in our previous experiment there was no striking influence of respiration on the wave form of the PVF in conscious dogs with quiet breath [2]. The PVF in human also showed only minimal changes during quiet respiration in the studies reported by Smallhorn *et al.* [12] and Klein and Tajik [5]. Therefore, it is considered that the deeper breath under anesthesia might cause the amplitude of intrathoracic pressure to oscillate largely. Unfortunately, the transesophageal echocardiography can not be used in dogs without anesthesia.

It has been described that the diameter of pulmonary vein changes during the cardiac cycle [8, 9]. However, in our experience, it is difficult to measure the PVD in dogs with transthoracic echocardiography, since the vessel is far from the acoustic window and the vessel wall is always parallel to the direction of the ultrasound beam. In contrast, the pulmonary venous wall is near to and perpendicular to the sampling beam line of the probe in the esophagus with transesophageal echocardiography, which make the measurement of PVD possible with M-mode evaluation. Morgan *et al.* [8] and Rajagopalan *et al.* [9] used ultrasonic crystals to measure the PVD of the extraparenchymal pulmonary vein. They reported that the reverse flow during left atrial contraction produced an increase in the PVD, and the filling flow during left ventricular systole or diastole produced a decrease in PVD due to collapse of the pulmonary vein. However, in our experiment we sampled the orifice part of the pulmonary vein and obtained the opposite results, that is, the orifice part of the pulmonary vein simultaneously contracted with the left atrial

contraction and extended with the left ventricular systole and diastole. Eliakim *et al.* [3] have described the sphincter-like structures of the atrial-pulmonary venous junctions in dogs. The myocardial fibers in this area surrounded the pulmonary veins. Therefore, it is believed that the myocardial fiber functions as a sphincter which contracts to reduce the blood flow back to the pulmonary vasculature during left atrial contraction and extends to allow the blood flow from pulmonary vasculature filling the left heart during left ventricular systole and diastole. When semiquantitatively estimating the flow volume with Doppler ultrasound, it is important to assume that the opening of a conduit is static, and to measure the radius carefully, since the variable must be squared when determining the cross-section area of the opening. In this study, the mean peak percent change in the diameter of pulmonary vein was 35% during cardiac cycle. Therefore, it is considered that using the Doppler method to estimate the volume of the PVF is controversial since the atrial-pulmonary venous junction, that is, the orifice of pulmonary vein varies dynamically during cardiac cycle.

In conclusion, in anesthetized dogs, respiration significantly affects the variables of the PVF with the decrease of the mean peak velocity and the velocity-time integral during inspiratory duration. On the other hand, the diameter of the pulmonary vein is variable during cardiac cycle, which is smallest during left atrial contraction and largest during the end of left ventricular systole. Therefore, it is important to consider the effect of respiration on the pulmonary venous flow and the cardiac phasic change on pulmonary venous diameter when interpreting the information from the pulmonary venous flow recorded by

transesophageal pulsed Doppler echocardiography.

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