# How to? / *Nasıl yapalım*? (Echocardiography / Ekokardiyografi)

# How to evaluate hepatic vein flow using transthoracic echocardiography

Transtorasik ekokardiyografi ile hepatik ven akımlarını nasıl değerlendirelim?

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E valuation of hepatic vein (HV) flows is the basic component of hepatic ultrasound. In daily practice, evaluation of HV flows as a part of echocardiographic assessment may better clarify cardiac physiology. In this article, acquisition of HV flows in transthoracic echocardiography (TTE), and the interpretation of the changes in HV flows in the setting of different cardiac pathologies are discussed.

#### Acquisition of hepatic vein blood flow images

The 4-chamber view is acquired if a slightly upward section is obtained when the transducer is placed under the subxiphoid process with the notch facing toward the left shoulder (Fig. 1a). Then the transducer is oriented slightly rightward to center the RA-liver border in the middle of the screen, and the transducer is rotated so as to obtain an image of the vena cava inferior (VCI) and a longitudinal axis view of the HV of interest. Frequently, the left HV, where the blood flow courses parallel to the cursor (Fig. 1b), is preferred. The pulsed wave Doppler cursor is placed 2 to 4 cm proximal to the point where the HV opens into the VCI and in the middle of the vein lumen, and a small

sample volume (2-4 mm) is selected. If the HVs are not dilated or the degree of respiratory translation is above physiological limits, it is difficult to hold the sample volume within the yein lumen.

### Abbreviations:

ECG	Electrocardiography
HV	Hepatic vein
COPD	Chronic obstructive pulmonary
	disease
CP	Constrictive pericarditis
PHT	Pulmonary hypertension
RA	Right atrium
RCM	Restrictive cardiomyopathy
RV	Right ventricle
TI	Tricuspid insufficiency
TTE	Transthoracic echocardiography
VCI	Vena cava inferi

and HV tracings may not be obtained during all phases of respiration. In this case, it is appropriate to select a larger sample size or to record the HV flow tracing during post-expiratory apnea. In conditions where simultaneous timing of ECG and HV flow are of importance, respiratory tracings should be observed.

In spite of brief moments of retrograde HV flow, HV flow is mainly an antegrade flow that enables return of the blood from the liver to the heart. Therefore, on TTE, its tracing is observed below the baseline. In other words, it moves away from the transducer. RA pressure is the most important factor affecting the direction and velocity of the HV flow.

#### Waves

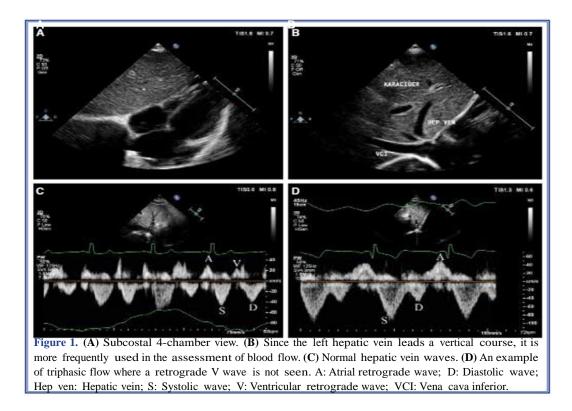
Basically, HV flows constitute 2 antegrade waves (S and D), 1 retrograde (A), and 1 transitional wave (V) (Fig. 1c).

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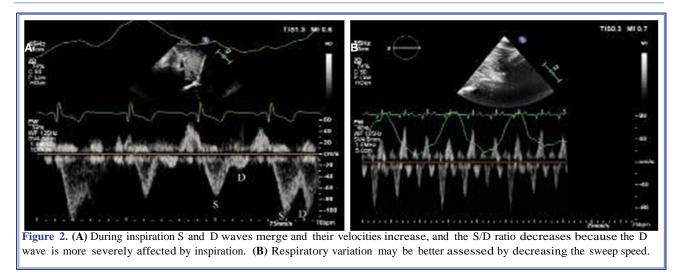
During ventricular contraction, the tricuspid annulus gets closer to the apex, and with relaxation of the RA, an S wave emerges below the baseline. Normally, most of the HV flow occurs during this phase. When RA pressure rises, the pressure gradient between the HV and the RA decreases, leading to a decrease in the volume and velocity of systolic flow.

At the end of systole, as a result of systemic venous flow against the closed tricuspid valve, a V wave occurs. The peak point of the V wave indicates the opening of the tricuspid valve and transition from systole to diastole. The peak point may be below, above, or on the baseline. If it does not pass above the baseline, it should not be referred to as retrograde flow. This is a transitional wave, and it is not included in the triphasic components (S, D, and A waves) of HV flow (Fig. 1d).

Following the opening of the tricuspid valve, a D wave is seen below the baseline during diastole. The descending arm of the D wave corresponds to decreased RA pressure during the early diastolic filling phase, while its ascending arm represents increased RA pressure due to increased right ventricle (RV) volume. During atrial contraction, the flow from the VCI to the RA decreases and reverses for a short while. This flow constitutes an A wave, which approaches to the transducer with its peak point above the baseline. This is a small wave, and cannot be detected in normal individuals. It is almost always larger and higher than the V wave.

In normal individuals, occasionally a momentary retrograde flow called a C wave can be observed following the A wave. The C wave is a reflection of an instantaneous flow that occurs during systole due to increased RV pressure that causes the tricuspid valve to bulge to the right atrium immediately before the opening of the pulmonary valve.

In the literature, various terminology is used to refer to HV waves. A V wave may be called ventricular reversal or systolic reversal, and an A wave atrial reversal. Basically, persistent forward movement of one component of the D wave, and the presence of a retrograde A wave with greater flow velocity than that of the V wave have been used in the differentiation of waves. However, the most reliable method requires simultaneous ECG recording.



### Factors affecting hepatic vein flow

Under normal conditions, during inspiration, S and D waves increase while the A wave diminishes (Fig. 2). Valsalva maneuver and post-expiratory apnea cause a loss of the triphasic char- acteristics of the HV wave, and it assumes almost a monophasic pattern. Therefore, when the patient is told to hold their breath, a Valsalva maneuver may inadvertently be induced, with a resultant decrease in HV flow. Triphasic waves of HV flow may be better evaluated during comfortable respiration in a quiet environment at the end of expiration.

Since hepatic blood flow increases following a meal, examination during a fasting period is preferred. However, some studies have demonstrated that it is not affected. Exercise does not affect the flow patterns, but it increases flow rates. During pregnancy, the amplitude of the waves decreases, and in sedation, tracings assume irregular configurations. HV flows may change in primary liver diseases. In Budd-Chiari syndrome, a retrograde monophasic HV flow is observed, while in cirrhosis, a notched, monophasic, antegrade flow with a small amplitude may be seen.

## Relationship between right atrial pressure and hepatic vein flow

An increase in RA pressure induces enlargement of HVs, and decreases the collapsibility of a HV following inspiration. The dominancy of the S wave seen in normal HV flow is lost with increased in RA pressure, and Vs/Vd drops below 1. Indeed, the pressure gradient between the HV and the RA decreases in parallel with an increase in RA pressure. The ratio between the velocity-time integral of the S wave and those of S and D waves is called the systolic filling fraction. A systolic filling fraction below 55% has been found to be more than 85% sensitive and specific for the presence of RA pressure >8mmHg.

In the prediction of RA pressure, the width and collapsibility of the VCI is frequently used. However, occasionally, the VCI may enlarge, and its collapsibility may decrease during inspiration in healthy individuals with normal systemic venous pressure. In these patients, HV flow measurements may be used to predict RA pressure.

### **Tricuspid regurgitation**

Under normal conditions, during the systolic phase, blood is ejected towards the RV outflow tract. In TR, it is at the same time ejected backwards over the RA toward the VCI and the liver. This condition decreases the amplitude of the S wave or induces formation of reverse S waves. A reverse S wave merges with other retrograde waves to form an ASV complex. In this case, the D wave is the only antegrade wave, and the flow assumes a biphasic pattern (Fig. 3).

Mild degrees of TR do not induce variations in HV flows, while in moderate TR the amplitude of the S wave is smaller than that of the D wave. In severe TR, retrograde S waves with a late systolic peak are seen. However, a decrease in the amplitude of the S wave or a reverse S wave is not specific to moderate or severe TR, respectively. RA pressure and compliance, and RV systolic function and compliance affect the

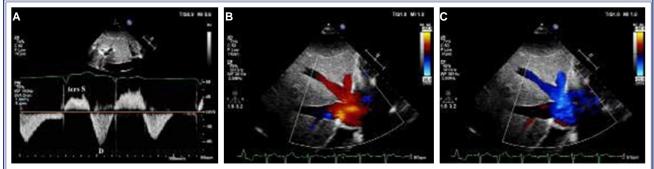
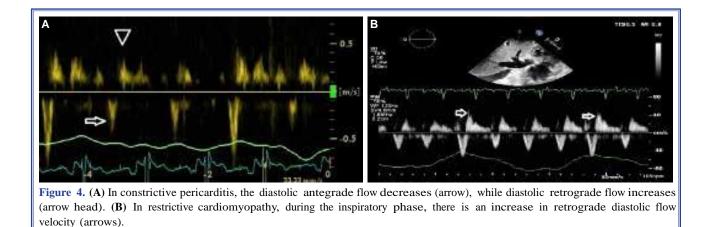


Figure 3. (A) In a case with atrial fibrillation and severe tricuspid regurgitation, there is an inverted S wave and the A wave is not visible. (B) Color Doppler image of an inverted S wave. (C) Color Doppler image of a D wave.

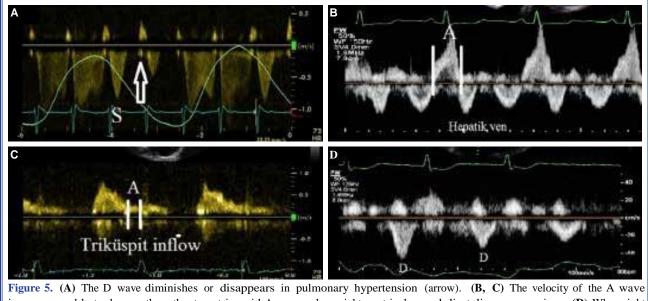


velocity and direction of the S wave independent of pulmonary artery pressure and grade of TR. If the RA is enlarged but compliant in a patient with severe TR, then a retrograde S wave may not be seen. In RV systolic dysfunction, following coronary artery bypass surgery or in atrial fibrillation, reverse S waves may be seen without the presence of serious TR. The severity of TR should absolutely be evaluated together with other variables.

### **Constrictive pericarditis**

In CP, dilatation of the VCI and increased diastolic reverse flow during expiration are observed on HV flow tracings. The basic pathophysiology in CP is dissociative intrathoracic and intracardiac pressure, which make an interventricular dependency of a relatively stable volume of the noncompliant heart surrounded by a thick, fibrotic, occasionally calcific pericardium more apparent. During inspiration, decreased intrathoracic pressure cannot be transferred adequately into the intracardiac space because of the thickened pericardium. This condition decreases the pressure gradient between intrathoracic pulmonary veins and the left heart cavities, leading to a decrease in left heart filling. A decrease in the left heart cavity during inspiration induces increases in right heart filling, and a leftward displacement of the interventricular septum. During expiration, the reverse occurs. In other words, left ventricular filling increases, the septum is displaced to the right, and RV filling decreases. In conclusion, a decrease in RV diastolic filling velocity and an increase in diastolic reverse flow are observed during expiration on HV flow tracing (Fig. 4a). During expiration, the ratio between diastolic retrograde and antegrade flow velocities becomes  $\geq 0.79$ , which is very specific to CP.

An increase in diastolic reverse wave velocity during expiration is also seen in chronic obstructive pulmonary disease (COPD), respiratory distress, postcoronary bypass surgery, RV infarction, and acute pulmonary embolism, in addition to CP. Because of clinical similarities, a differential diagnosis from COPD may be difficult. In CP, an increase in diastolic reverse flow happens frequently during expiration at the first beat, while in COPD it takes place at later heart beats. In addition, antegrade superior vena cava



increases and lasts longer than the transtricuspid A wave when right ventricular end-diastolic pressure rises. (D) When right ventricular systolic dysfunction develops, diminished and inverted S waves are observed and the D wave reappears.

flow rates in COPD demonstrate greater variabilities, while in CP, significant respiratory changes are not seen and antegrade flow velocities decrease.

## **Restrictive cardiomyopathy**

Respiratory changes in HV flow patterns seen in patients with CP are not seen in patients with restrictive cardiomyopathy (RCM). In RCM, a predominantly diastolic antegrade flow is seen on HV tracings, and retrograde flow increases during inspiration (Fig. 4b). An increase in venous return during inspiration cannot be met adequately by a noncompliant RV, so enddiastolic pressure and the velocity of diastolic retrograde flow increase.

CP and RCM are the main forms of diastolic dysfunction and increases in retrograde diastolic flow in a HV during inspiration or inspiration should bring them to mind, respectively. In fact, the most important difference between constriction and restriction is not physiological, but anatomical (pericardial wall thickness). Spirometry should be used during an examination of HV flows in the differential diagnosis between CP and RCM.

### **Pulmonary hypertension**

In pulmonary hypertension (PHT), the relaxation time capacity of the RV myocardium is impaired, isovolumetric relaxation time is prolonged, and the opening of the tricuspid valve is delayed. In severe cases, especially when accompanied by tachycardia, since the velocity of the diastolic antegrade flow decreases, D waves diminish or disappear on HV flow tracings (Fig. 5a). In PHT where RV enddiastolic pressure increases, RA end-diastolic pressure rapidly increases with RA contractions, and the tricuspid valve closes prematurely. This earlier closure induces an increased retrograde flow velocity of the A wave, prolongs its duration relative to a transtricuspid A wave (Fig. 5b, c). A diminished / absent D wave on an HV tracing together with increased duration and velocity of a retrograde A wave should prompt consideration of the presence of PHT. When RV systolic dysfunction develops, systolic antegrade flow decreases and an S/D ratio of <1 is observed (Fig. 5d).

## **Atrial fibrillation**

Since atrial contractions do not occur during atrial fibrillation (AF), a retrograde A wave is not seen on HV flow tracings. Since atrial relaxation cannot occur, the velocity of systolic antegrade flow decreases, and the S wave becomes smaller than the D wave.

# Conclusion

Knowledge of normal HV flow patterns seen in TTE and an awareness of the potential alterations in heart diseases are important in the diagnosis and differential diagnosis of many cardiac pathologies. It is a complementary approach to the assessment of echocardiographic examination results.