

My Approach to Echocardiographic Assessment for Constrictive Pericarditis

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Abstract

Constrictive pericarditis (CP) is a condition in which scarring and loss of elasticity of the pericardium result in impaired ventricular filling, diastolic dysfunction, and right heart failure. The diagnosis of this pathology is challenging, with frequent need for multimodal imaging techniques, among which echocardiography represents the initial imaging modality for the diagnostic evaluation, in addition to allowing the differentiation of CP from restrictive cardiomyopathy (RCM) and other conditions that mimic constriction.

How I Perform Echocardiographic Assessment for CP

Introduction

Constrictive pericarditis (CP) is characterized by focal or global scarring and loss of elasticity of the pericardium with or without associated thickening. Abnormal pericardium prevents diastolic filling, causing elevation of systemic venous pressures, despite preserved myocardial function of the ventricles. Therefore, it results in right heart failure, with the classic manifestations of edema of the lower limbs, pulsatile hepatomegaly, ascites, pleural effusion, fatigue, and low tolerance for effort.^{1,2}

The symptomatology of CP is not specific, and its manifestations can be confused with myocardial, coronary, pulmonary, or even gastrointestinal conditions, such as liver cirrhosis, RCM, endomyocardial fibrosis, among other pathologies, making the diagnosis challenging.^{3,4}

Several conditions can lead to CP, including infectious etiologies, connective tissue diseases, trauma, metabolic disorders (uremia), iatrogenic etiologies (pericardiotomy, radiotherapy), neoplasms or even idiopathies.⁵ In Europe and the United States, the most frequent etiology is idiopathic,

followed by post-cardiac surgery, while in other parts of the world, such as Brazil, tuberculosis is more common.⁵⁻⁷

The pathophysiology of CP is related to the elevation and equalization of cardiac pressures, since the heart is located in a fixed space determined by the rigid pericardium, to exaggerated ventricular interdependence and to the dissociation of intrathoracic and intracardiac pressures.^{6,7}

During inspiration, the rigid pericardium promotes the dissociation of intrathoracic and intracardiac pressures by preventing the decrease in intrathoracic pressure from being fully transmitted to the cardiac chambers. Thus, as the pressure in the extrapericardial pulmonary veins decreases during inhalation, there is attenuation of the pulmonary venous gradient toward the left atrium, contributing to the reduction of left ventricle (LV) filling.^{5,6}

During inspiration, the decrease in LV filling allows for an increase in right ventricle (RV) filling with a deviation of the interventricular septum to the left, configuring ventricular interdependence. Consequently, fillings related to the left heart (mitral, aortic, and pulmonary venous flows) decrease, while fillings related to the right heart (tricuspid, hepatic, pulmonary artery, and inferior vena cava) increase. During expiration, all of the above mechanisms are reversed, except the superior vena cava (SVC) flow, which is not influenced by respiration in CP.⁸

The restriction to emptying of the left atrium during diastole results in an increase in its pressure and, subsequently, in pulmonary venous pressure.

In CP, there is no impairment of ventricular relaxation during the initial phase of diastole, with early filling being faster than normal due to increased driving force resulted from high atrial pressure. However, the myocardium cannot continue its relaxation after meeting the rigid pericardium during middle and late diastole, leading to increased end-diastolic pressure in the ventricles and atria.⁸

There are three subtypes of CP, namely transient, chronic, and effusive constrictive pericarditis. Transient CP is a temporary form of constriction due to underlying inflammation, which may resolve spontaneously or after drug treatment. Chronic CP results from chronic inflammation and permanent scarring, potentially necessitating surgical intervention. Effusive CP is characterized by the coexistence of constriction by the visceral pericardium and tense pericardial effusion, with elevated right atrial pressure persisting after pericardiocentesis.⁷

Pericardiectomy is the definitive treatment in refractory patients to clinical treatment, as it relieves pericardial containment and, in the absence of concomitant myocardial dysfunction, effectively restores diastolic filling.⁹

Keywords

Pericardium, Constrictive pericarditis, Heart failure.

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How to assess?

Suspicion of CP is based on clinical history and physical examination, and requires evaluation and confirmation by imaging and hemodynamic data.⁶

Echocardiography is the most commonly used imaging modality for the initial assessment of pericardial disease, as it is widely available, safe, fast, and inexpensive compared to other methods, aiding in the diagnosis of CP and excluding other causes of diastolic heart failure.¹⁰

Doppler echocardiographic findings in constriction:

- Increased pericardial thickness (≥ 3 mm), which may be absent in up to 12 to 18% of patients.⁷ Due to the exacerbated pericardial refraction, even in normal individuals, it may be difficult to detect pericardial thickening and calcification, mainly by transthoracic echocardiogram, with the transesophageal echocardiogram having much higher sensitivity and specificity;

- During systole, restricted movement of the posterior wall of the left ventricle is observed due to pericardial adhesion. There is also an abrupt relaxation of the posterior wall with subsequent flattening of the endocardial movement during the rest of the diastole, which can be well observed in the M-mode;⁶

- Mitral annulus reversus, present in 74% of patients with CP, which represents the reversal of the normal ratio of early diastolic myocardial velocities with lateral e' smaller than septal

e'.⁶ The septal e' / lateral e' ratio ≥ 0.91 has a sensitivity (S) of 75% and specificity (E) of 85%⁹ (Figures 1A, 1B, 1C and 1D);

- Normal or increased mitral inflow propagation velocity (> 100 cm/s) in Color M-Mode (S 74%; E 91%) (Figure 2);

- Abnormal and oscillatory movement of the interventricular septum beat to beat, related to ventricular interdependence due to subtle differences in the timing of mitral and tricuspid valve opening and left and right atrial contraction (AC). In M-mode, a notch is observed at the beginning of diastole, followed by paradoxical movement and then normal movement of the interventricular septum⁷ (Figure 3);

- Presence of a septal leap with respirophasic deviation of the interventricular septum toward the left ventricular cavity during inspiration (two-dimensional mode: S 62% and E 93%; M-mode: S 93% and E 69%)^{9,11} (Video 1);

- Ventricular interdependence with respiratory variation $\geq 25\%$ of peak mitral E wave velocity (S 88%; E 67%) or $\geq 40\%$ of peak tricuspid E wave velocity (S 90%; E 88%) determined by pulsed wave Doppler at the level of the cuspid tips in an apical four-chamber view. The consensus for calculating the percentage of respiratory variation is (expiration - inspiration)/expiration^{9,12} (Figure 4A and 4B);

- Preserved or increased velocity e' of the septal mitral annulus, ≥ 9 cm/s (S 83%; E 81%);^{7,9}

- Percent change in mitral flow E velocity between inspiration and expiration $\geq 14.6\%$ (S 84%; E 73%);¹¹

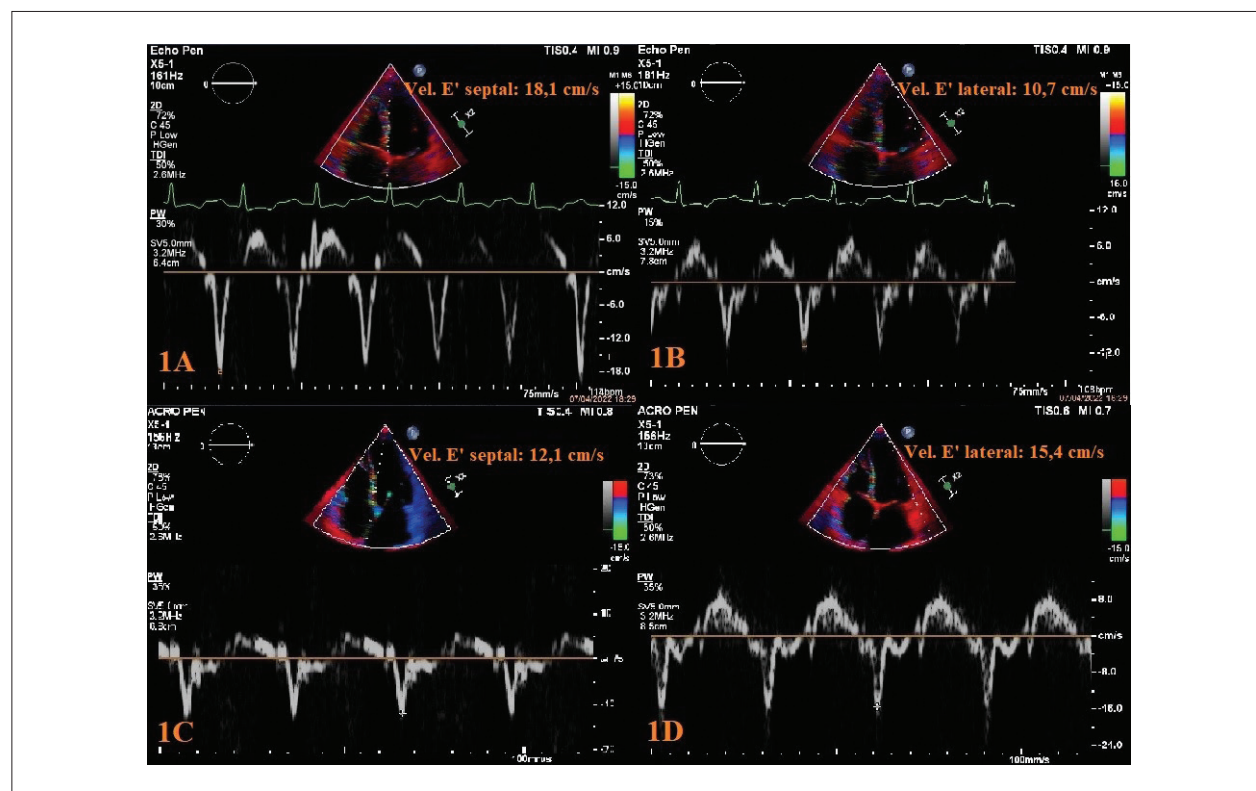


Figure 1 – Tissue Doppler demonstrating the phenomenon of annulus reversus in Figures 1A and 1B, in which the velocity of the septal mitral annulus (18.1 cm/s) is greater than the lateral velocity (10.7 cm/s). After pericardiectomy, there was normalization of this relationship, with the septal velocity (12.1 cm/s) lower than the lateral velocity (15.4 cm/s), demonstrated in Figures 1C and 1D.

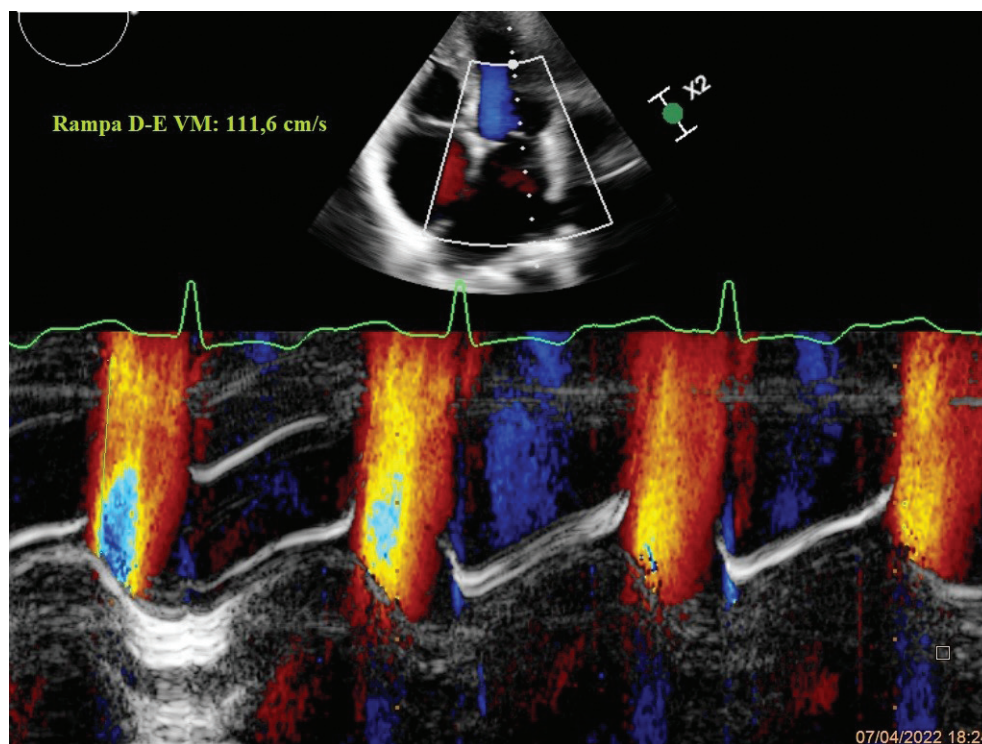


Figure 2 – Color Doppler M-mode recording in a patient with CP. Note the rather steep propagation velocity (V_p) (> 100 cm/s), helping to differentiate between constrictive and restrictive physiology.

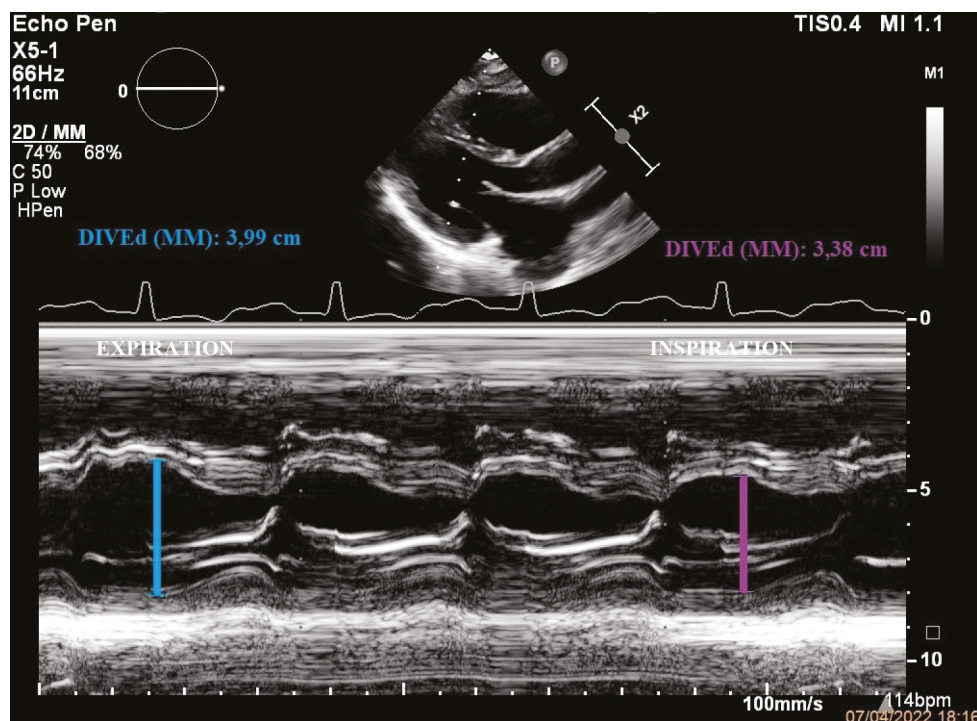
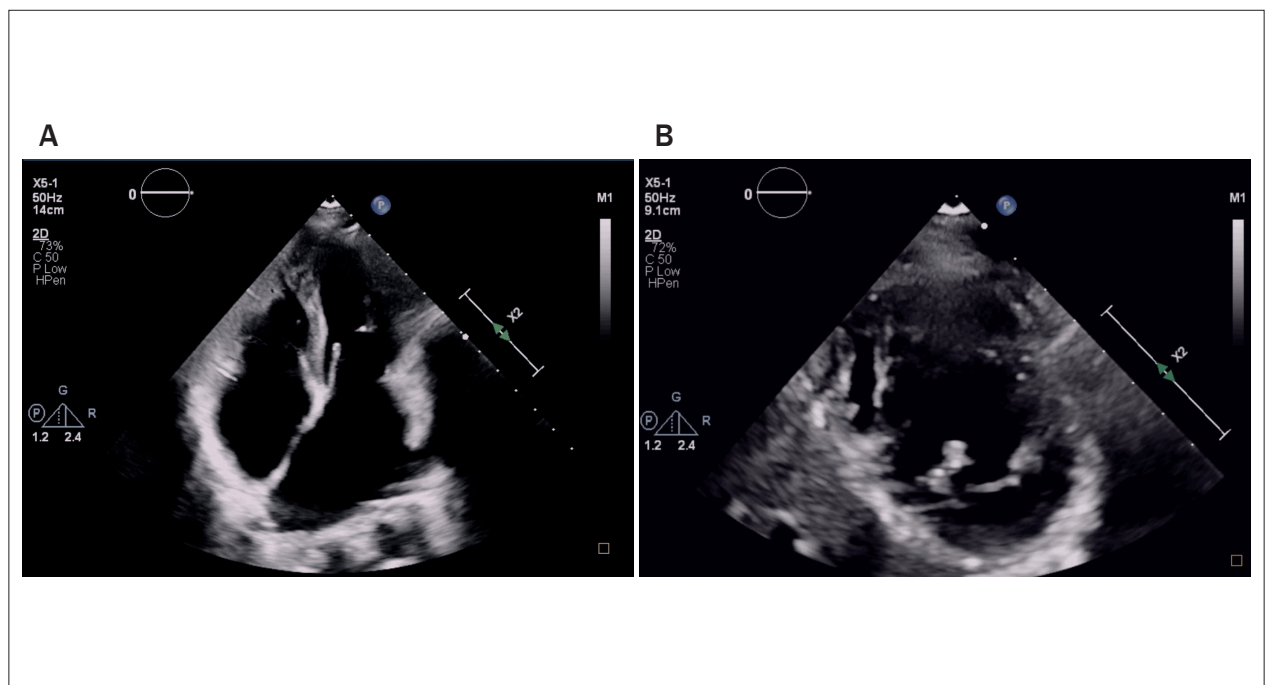


Figure 3 – Beat-to-beat oscillatory movement of the interventricular septum and respirophasic movement recorded in windowed M mode parasternal long axis.



Video 1 – Representation of the phenomenon of ventricular interdependence in a patient with CP. Note the deviation of the septum to the left during inspiration and shift to the right during expiration.

Link video 1A: <http://abcimaging.org/supplementary-material/2023/3601/ABC-366-video-1A.mp4>

Link video 1B: <http://abcimaging.org/supplementary-material/2023/3601/ABC-366-video-1B.mp4>

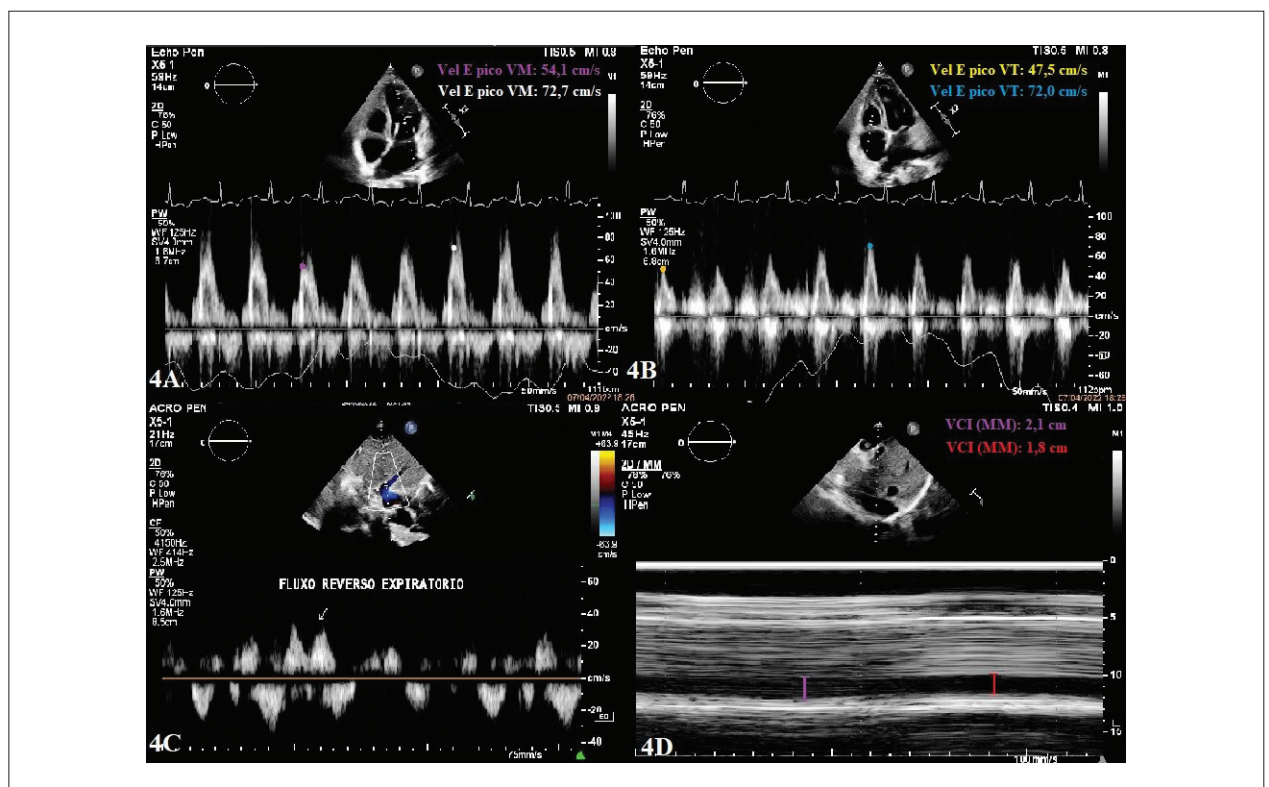


Figure 4 – Demonstration of transmitral (4A) and transtricuspid (4B) flow variation during breathing in a patient with CP. In the same patient, note the presence of reverse flow in the hepatic vein during expiration (4C), as well as dilation of the inferior vena cava with reduced collapsibility (4D).

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- Marked increase in early diastolic filling velocity (E velocity) of mitral flow with a rapid deceleration and a decreased late diastolic filling velocity after AC (A velocity), resulting in increased E/A ratio ($E/A > 2$);⁶

- It is not uncommon to observe less typical patterns, in which there is a normal or inverted E/A ratio with exaggerated respiratory variation or in which only the tricuspid inflow reveals classic alterations;

- Exaggerated respiratory variation in the isovolumetric relaxation time of the left ventricle;

- In CP, the average of early diastolic myocardial velocities (lateral e' and septal e') is preserved, despite having higher filling pressures. Septal e' velocity increases progressively with the worsening of CP, causing a drop in the E/e' ratio despite the increase in pulmonary capillary pressure (PCP). This inverse relationship between PCP and E/e' is called *annulus paradoxus*;⁷

- During expiration, there is reduced forward diastolic flow in the hepatic vein with prominent telediastolic reverse flow and increased systolic forward flow on inspiration. High specificity mainly if reverse flow velocity is $\geq 0.8 \text{ m/s}$ ^{7,11} (Figure 4C);

- Hepatic vein diastolic flow reversal ratio (expiratory diastolic reversal velocity divided by forward diastolic hepatic vein flow velocity) ≥ 0.79 is one of the most specific findings (S 76%; E 88%; PPV 96%; NPV 49%);^{7,12}

- Dilation of the inferior vena cava ($\geq 21 \text{ mm}$) and the suprahepatic vein with reduced respiratory variation ($< 50\%$) (Figure 4D);

- The left atrium, which is only partially covered by the pericardium, may be enlarged. Enlargement of the right atrium may also occur (present in 61% of patients with CP);

- Premature opening of the pulmonary valve;

- Slight increase in pulmonary artery systolic pressure (generally not exceeding 50 mmHg);

- “Warm septum” sign on dot-tracking echocardiography: due to adhesion of the free walls by the diseased pericardium, mainly the lateral wall, the modular value of the longitudinal tension in these areas is reduced in relation to the septal wall, where it is increased (*strain reversus*).^{6,7,13}

The classic findings of constriction are more prominent in euvoletic patients. If absent in patients with suspected CP, it is advised to reassess them after fluid replacement, if there is volume depletion, or to examine them in the sitting position, if there is volume overload.

Definitive diagnosis should be based on the combination of clinical and echocardiographic data, in addition to other complementary methods (e.g., computed tomography, magnetic resonance or invasive hemodynamic evaluation), when necessary.

Table 1 – Echocardiographic aspects in the differentiation between CP and RCM

Echocardiographic findings	CP	RCM
E/A Ratio	Increased ($E/A > 2$)	Increased ($E/A > 2$)
E deceleration time	Short ($\leq 160 \text{ ms}$)	Short ($\leq 160 \text{ ms}$)
Respiratory variation in mitral and tricuspid E-wave velocity	$\Delta \text{VMi} \geq 25\%$ $\Delta \text{VTri} \geq 40\%$	Not variable
Velocity of annular e'	Normal	Reduced
Breathing movement of the interventricular septum	Present	Absent
Pulmonary hypertension	Rare	Frequent
Mitral inflow propagation velocity (color M-mode)	Increased ($> 55 \text{ cm/s}$)	Reduced
LV isovolumetric relaxation time	Variable with breathing	Stable with breathing
Flow in the pulmonary veins	$S > D$, with small increment of D on expiration	$S < D$ throughout the respiratory cycle
Diastolic reverse flow in the hepatic vein	During expiration	During inspiration
Flow in the SVC	$S < D$, with a slight reduction in D during expiration and no variation in AC with the respiratory cycle	$S \ll D$, with a decrease in D on expiration and an increase in AC on inspiration
Strain analysis	Reduced circumferential strain, torsion, and distortion velocity	Normal circumference strain
	Overall normal longitudinal strain, although there may be <i>strain reversus</i>	Reduced longitudinal strain
Appearance of the pericardium	Glossy/thick	Normal
Atrial size	Normal or slightly dilated	Very dilated

S: velocity of the wave formed by the flow of the pulmonary veins towards the left atrium during ventricular systole; D: flow wave velocity from the pulmonary veins towards the left ventricle, using the left atrium as a conduit during ventricular diastole; ΔVMi : respiratory variation of E-wave velocity in the mitral valve; ΔVTri : respiratory variation of E-wave velocity in the tricuspid valve; E/A: ratio between the early diastolic velocity of the mitral flow (E) and the velocity of atrial contraction of the mitral flow (A).

How to differentiate CP and RCM

Both CP and RCM manifest themselves as a chronic clinical condition of volume overload, making the evaluation of multiple echocardiographic parameters important to establish the differential diagnosis, which has major implications for therapy and prognosis (Table 1).

In both conditions, increased E/A ratio with a shortened deceleration time can be noticed, however the respiratory variation in the velocity of the E wave is increased in constriction (> 25%), while in RCM it is normal or slightly variable (< 15%). In addition, this parameter tends to normalize in CP after pericardiectomy.¹

Since RCM is a myocardial disease, it tends to have lower tissue (septal and lateral) Doppler velocities in contrast to CP.¹

In CP, there is respirophasic movement of the interventricular septum, which is not usually present in RCM.

There is marked biatrial enlargement in RCM, but relatively normal or slightly increased sizes in CP.

Pulmonary hypertension is more common in RCM than in CP.

The left ventricular isovolumetric relaxation time is stable in RCM, while in CP there is exaggerated respiratory variation.¹

Mitral valve propagation velocity with Color M-Mode is increased in CP (>55 cm/s), although reduced in RCM.¹

In CP, during inspiration, the flow velocity in the pulmonary veins is predominantly systolic over diastolic, with a small increase in the latter in expiration, while in RCM, the flow in the pulmonary veins is consistently greater in diastole than in systole and persists throughout the respiratory cycle.¹⁴

In CP, there is an increase in diastolic reverse flow in the hepatic vein during expiration, while in RCM there may be reversal of diastolic flow in the hepatic vein during inspiration.⁶

In RCM, the flow in the SVC presents a much larger D wave than S wave, with a reduction in the D wave during expiration and an increase in reverse flow during AC during inspiration. In CP, the D wave is slightly larger than the S wave, with a slight reduction in the D wave during expiration and no change in the AC wave during the respiratory.¹⁴

CP is characterized by reduced circumferential strain, torsion, and shear rate, but with normal overall longitudinal strain despite regional differences. In RCM, there is reduced longitudinal strain but normal circumferential strain. This is due to the fact that subendocardial fibers, which are mainly responsible for longitudinal shortening, are more affected in RCM, while in CP there is greater involvement of subepicardial fibers, predominantly responsible for circumferential shortening.^{6,7,9,13,15}

According to the study carried out at the Mayo Clinic between January 2008 and December 2010, whose population consisted of patients with surgically confirmed CP, it was concluded that echocardiography makes it possible to differentiate CP from RCM and severe tricuspid insufficiency. The three independent criteria most associated with the diagnosis of CP were: ventricular septal deviation related to

breathing, preserved or increased e' velocity of the medial mitral annulus, and prominent reversal of the expiratory diastolic flow of the hepatic vein.^{2,11} Each of these criteria was significantly associated with CP in the subgroup of patients with atrial fibrillation.¹¹

According to the American Society of Echocardiography (2016), when the medial e' velocity of the mitral annulus is > 8 cm/s, mitral annulus reversus and hepatic vein expiratory flow reversal are present, RCM can be excluded and CP diagnosis can be established with confidence.¹²

Limitations of echocardiographic assessment in CP

The e' annular velocity through pulsed tissue Doppler may be reduced in cases of mitral valve replacement, severe annular calcification, and basal hypokinesia.

Diastolic dysfunction commonly associated with ischemia or advanced age can also make it difficult to interpret the mitral inflow pattern.

In aged and hypertensive patients, the LV cavity may be small, causing an increase in the propagation velocity of the mitral inflow despite the absence of CP.

A mitral flow pattern similar to that of CP can be seen in patients with chronic obstructive pulmonary disease, pulmonary thromboembolism, right ventricular infarction, shock, and large bilateral pleural effusion.

Diastolic posterior wall flattening during tachycardia may be difficult to detect.

In adolescents and young adults, the E/A ratio > 2 may be normal and they usually have mitral inflow propagation velocity > 100 cm/s and septal e' > 12 cm/s.

Conclusion

CP is an underdiagnosed cause of right and/or left ventricular failure caused by a reduction in the elasticity of the pericardium, resulting in impaired cardiac diastolic filling.¹⁵ It has characteristic pathophysiological mechanisms, which can be identified with Doppler echocardiography, and this test is useful both for diagnostic confirmation and for follow-up of treated patients. The decision to perform additional tests, such as magnetic resonance imaging (MRI), computed tomography (CT) or invasive hemodynamic study, must be individualized.¹¹

Author Contributions

Conception and design of the research: Oliveira ACR; writing of the manuscript: Rocha DFR, Oliveira ACR; critical revision of the manuscript for intellectual content: Silva JBM e Silva VN.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

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