INDIAN HEART JOURNAL 67 (2015) 175–182

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/ihj

C3 – Core Curriculum in Cardiology

Invasive hemodynamics of constrictive pericarditis

Shrenik Doshi, Sivasubramanian Ramakrishnan, Saurabh Kumar Gupta *

All India Institute of Medical Sciences, New Delhi, India

ARTICLE INFO

Article history:
Received 10 April 2015
Accepted 13 April 2015
Available online 13 May 2015

Keywords:
Constrictive pericarditis
Restrictive physiology
Cardiac catheterization
Hemodynamics

ABSTRACT

Cardiac catheterization and hemodynamic study is the gold standard for the diagnosis of pericardial constriction. Careful interpretation of the hemodynamic data is essential to differentiate it from other diseases with restrictive physiology. In this hemodynamic review we shall briefly discuss the physiologic basis of various hemodynamic changes seen in a patient with constrictive pericarditis.

1. Introduction

With the advent of newer imaging tools, there has been a shift to noninvasive imaging particularly when chronic constrictive pericarditis (CCP) is suspected.[1] Cardiac catheterization nonetheless remains diagnostic of the constriction physiology especially when anatomic information from other imaging tools is inconclusive. In this hemodynamic review we shall briefly discuss hemodynamic alterations seen in patients with pericardial constriction and how they are different compared to other pericardial compression syndromes and restrictive cardiomyopathy (RCMP).

2. Normal and abnormal pericardium

The normal pericardium is a bi-layered, fibroblastic sac surrounding the heart.[2,3] Majority of the cardiac mass including proximal most great arteries and caval veins are intrapericardial, with the exception of the left atrium (LA). Pericardial cavity, a potential space between two layers of pericardium can have 15–35 ml of serous fluid. As a result of relatively inelastic physical properties, the pericardium limits acute dilatation and enhances mechanical interactions of the cardiac chambers.[4] Accumulation of large amount of fluid especially if rapid or thickening of pericardium results in pericardial compression syndrome characterized by increased pericardial pressure. When both fluid and pericardial constriction exist together it is referred to as effusive-constrictive pericarditis.[2,3]

3. Physiology of pericardial constriction

The compliance of the thickened, stiff pericardium of CCP is markedly reduced. This allows only limited distention of

* *Corresponding author*. Department of Cardiology, Room No. 23, 7th Floor, CT Centre, AIIMS, New Delhi, 110029, India. Tel.: +91 1126594681.
E-mail address: drsaurabhmd@gmail.com (S.K. Gupta).

http://dx.doi.org/10.1016/j.ihj.2015.04.011

cardiac chambers and results in fixed total cardiac volume.[2,5] In addition, thickened pericardium prevents normal transmission of thoracic pressure to the cardiac chambers (Fig. 1).

3.1. *Fixed pericardial volume*

The normal pericardium stretch to accommodate physiologic changes in cardiac volume and facilitate mechanical interaction between left (LV) and right ventricle (RV) during diastole. An abnormally thick, inelastic pericardium of CCP shows reduced ability to adapt and limits the total cardiac volume. Compression does not occur until the cardiac volume approximates that of the pericardium, which occurs in mid-to-late diastole. As a result ventricular filling is severely restricted and occurs only in early diastole. The end diastolic pressure in all cardiac chambers including caval and pulmonary veins during diastole is equal and reflects elevated pericardial pressure. The elevated pericardial pressure shows more detrimental effect on right sided chambers owing to their overall lower pressures.

3.2. *Dissociation of thoracic and cardiac chamber pressure*

Normally pressure in cardiac chambers, especially during diastole varies with respiration. Elastic pericardium transmits changes in thoracic pressure to the cardiac chambers while a thickened pericardium blocks this transmission. The changes in thoracic pressure however continue to be transmitted to the pulmonary circulation.[4] Consequently, pressure in the pulmonary veins declines during inspiration while LV diastolic pressure remains unaltered. LV filling is thus reduced in inspiration allowing preferential filling of RV due to shift of interventricular septum. The flow across tricuspid valve shows >40% increase during inspiration compared to expiration. Exactly opposite sequence happens during expiration when LV fills preferentially with enhanced trans-mitral flow. The RV and LV thus fills out of phase to each other and represent exaggeration of otherwise normal ventricular interdependence.

4. Hemodynamic study in patient with suspected CCP

The hemodynamic study for suspected pericardial pathology should be carefully planned. Attention must be paid to the fluid status to avoid evaluation during low volume, low output state. Patients can be mildly sedated but should remain awake during the study to simulate hemodynamics changes at rest. Oxygen supplementation should be limited to patients with hypoxia and should be started after obtaining samples for oximetry to calculate cardiac output. Irregular rhythm (atrial fibrillation, frequent ventricular ectopics) during the hemodynamic study is an important source of error. Temporary pacing if used in such circumstances can improve the diagnostic accuracy.[6]

The importance of accurate calibration and positioning of the pressures transducer system cannot be overemphasized. Although high-fidelity manometer-tip catheters are more accurate, fluid filled catheters are generally sufficient in clinical practice. It is also important to analyze appropriate beats for proper assessment of respiratory variation in the ventricular pressures. Peak inspiratory and expiratory beats are identified by lowest and highest diastolic nadir in LV and pulmonary capillary wedge pressure (PCWP) respectively.[7] Complete hemodynamic study must be recorded and reviewed retrospectively.

5. Hemodynamic alterations in pericardial constriction

CCP is characterized by restrictive ventricular filling that varies with respiration. Further, this respiratory variation has differential effect on LV and RV filling and forms the basis of hemodynamic changes unique to CCP.[2,6–9]

5.1. *Hemodynamics at rest*

5.1.1. *Right atrium*

The right atrial (RA) pressure is significantly elevated. Prominent 'y' descent in the jugular venous pulse and RA waveform,

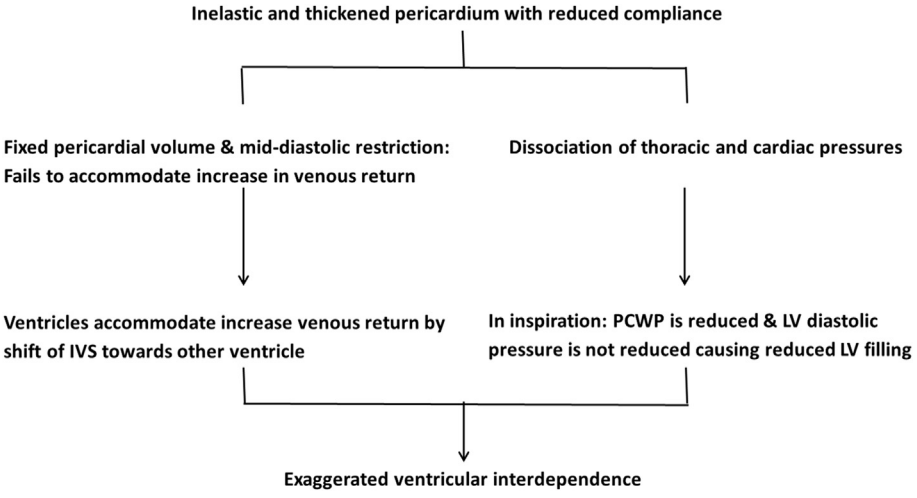

Fig. 1 – Physiologic basis of hemodynamic alterations in pericardial constriction.

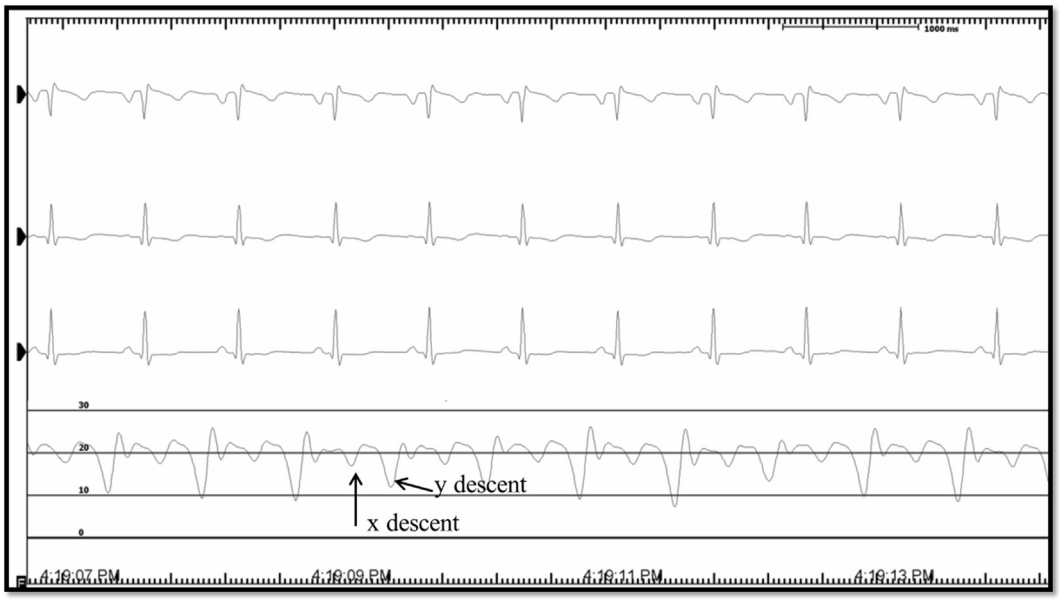

Fig. 2 – Right atrial (RA) pressure trace showing elevated mean atrial pressure (20 mm Hg) with prominent 'x' and 'y' descent. The 'y' descent is much more rapid and prominent than 'x' descent due to accentuated early diastolic filling of ventricles.

commonly referred to as 'Friedrich's sign' is hemodynamic correlate of rapid emptying of RA during early diastole (Fig. 2). The 'x' descent is also prominent but to a much lesser extent compared to 'y' descent. The combination of elevated mean pressure, inconspicuous positive waves and prominent descents result in famous 'M' or 'W' pattern.

5.1.2. *Right and left ventricle*

5.1.2.1. *Equalization of diastolic pressures:* In normal hearts the diastolic pressures in ventricles vary independently and remain unaffected by the other ventricle. Stiff pericardium limits the expansion of ventricles beyond a point after which it can only fill by compressing the other ventricle. In addition, fixed pericardium exerts an equal contact pressure on all the chambers causing elevation and equalization of diastolic pressure. Elevation and equalization of diastolic pressures (within 5 mmHg) in all cardiac chambers is the hallmark of pericardial constriction (Fig. 3). This diastolic equalization is not unique to CCP but is also seen in decompensated left sided heart failure, RCMP, severe tricuspid regurgitation, and acute mitral regurgitation and RV infarction. On the other hand it may be absent in cases with localized pericardial constriction or in patients with normal RA pressure.

5.1.2.2. *'Dip & plateau pattern' or 'square root sign':* Early diastolic filling of the ventricles is unimpeded and abnormally rapid, but late diastolic filling is abbreviated and halts abruptly when total cardiac volume expands to the volume limit set by the stiff pericardium. The pressure in the late diastole elevates and plateaus in accordance with the pericardial compression.

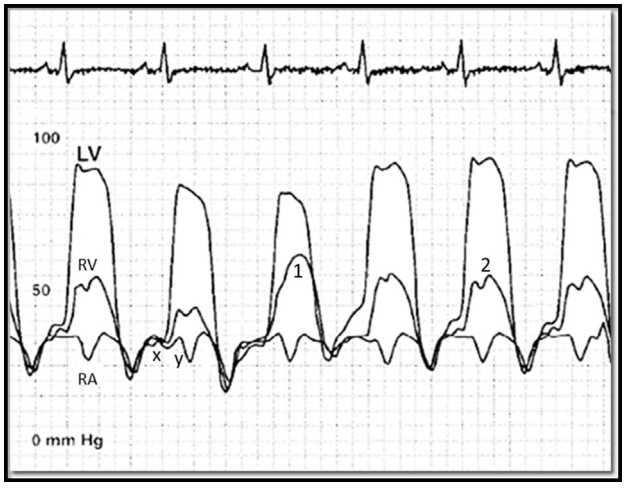

Fig. 3 – Elevated and equalized diastolic pressure in all the cardiac chambers.

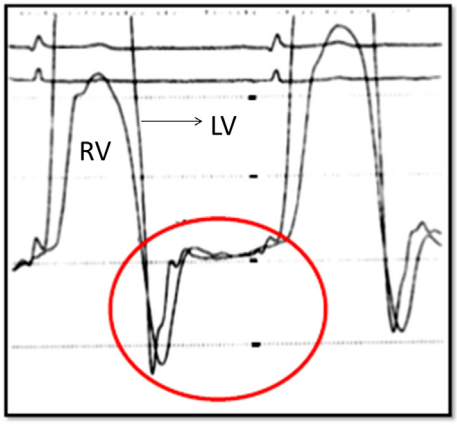

Fig. 4 – Simultaneous RV and LV trace from a patient with CCP showing square root sign. Also note that the diastolic pressures in both the ventricles are equal.

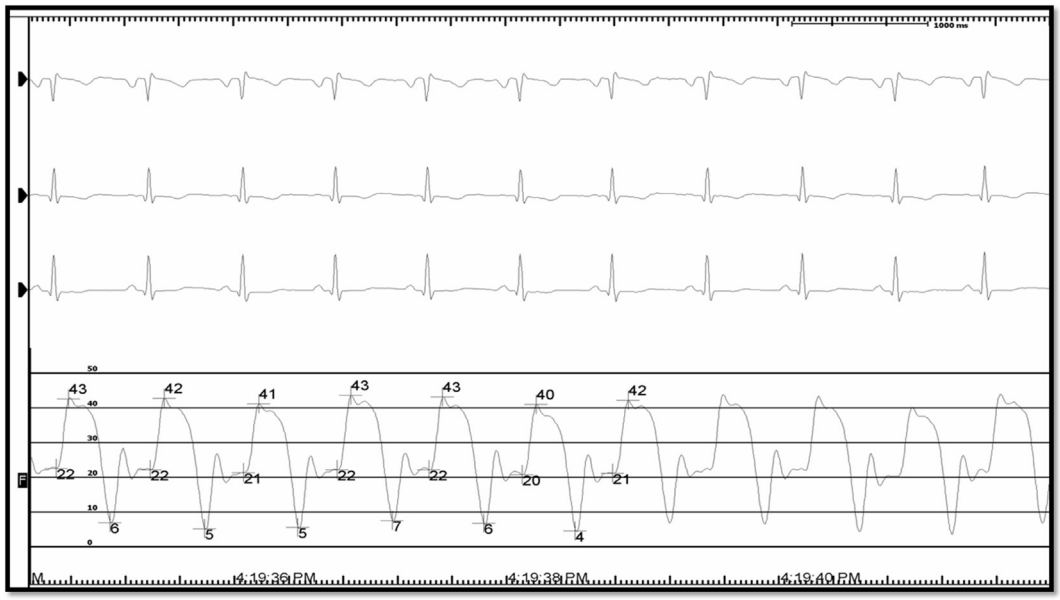

Fig. 5 – Right Ventricular (RV) pressure trace showing early dip followed by plateauing of pressure in mid-diastole ('dip & plateau' pattern or 'square root sign'), elevated RVSP and RVEDP. RVSP is > 1/3rd of RVSP that is < 50 mmHg.

This pattern of accentuated early dip in diastolic pressure followed by plateauing in mid-late diastole is referred to as 'Dip & plateau' or 'square root sign' (Fig. 4). Similar to diastolic equalization, 'square root sign' is also not exclusive to CCP and is also present in patients with RCMP and severe bradycardia.

5.1.2.3. *Elevated RV systolic pressure:* Restricted ventricular filling and consequent increase in filling pressures mandates RV systolic pressure (RVSP) to rise to maintain forward flow. This elevation in RVSP however is generally limited to less than 50 mm Hg. The increase in RV end diastolic pressure (RVEDP) is much more pronounced with levels more than 1/3rd RVSP (Fig. 5).

5.1.2.4. *Left ventricular rapid filling wave (LVRFW):* Ventricular filling during diastole is complex interplay of various active and passive processes. Accentuated filling of ventricles during early diastole is represented by deep RFW (Fig. 6). Similar to mitral annular E′ wave on tissue Doppler imaging, LVRFW measuring >7 mm Hg has a sensitivity of 93% and specificity of 57% in diagnosing CCP (Fig. 6). In patients with RCMP, LV RFW is reduced due to abnormal myocardial relaxation.

5.2. *Respiratory hemodynamics*

Many conventional hemodynamic changes are also present in a patient with RCMP and therefore has limited ability in confirming the diagnosis of CCP.[9–11] Fixed cardiac volume in CCP necessitates enhanced interventricular dependence to maintain cardiac output. The use of Doppler demonstration of respiratory variation in flow velocity across atrio-ventricular valve to indicate CCP is well established.[12,13] Use of similar

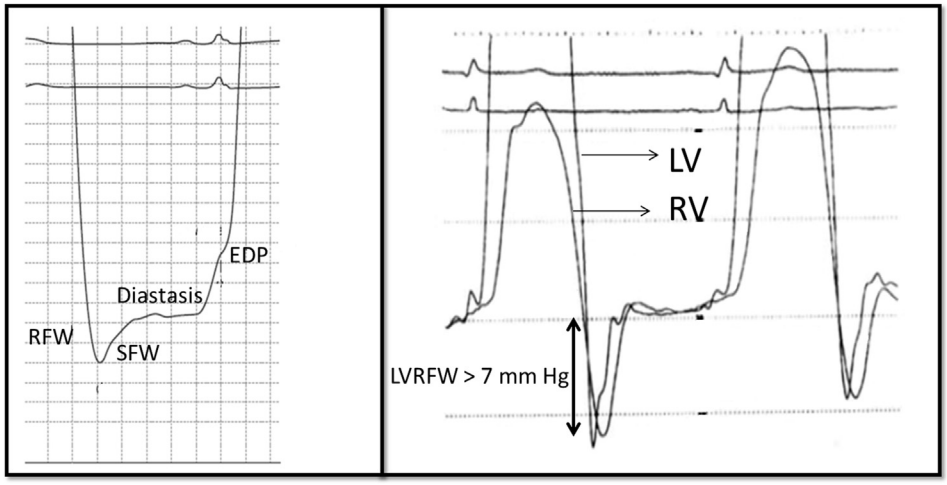

Fig. 6 – Left ventricular rapid filling wave (LVRFW) in a normal heart (panel A) and in a patient with CCP (panel B). A height of LVRFW > 7 mm Hg strongly suggests the diagnosis of CCP.

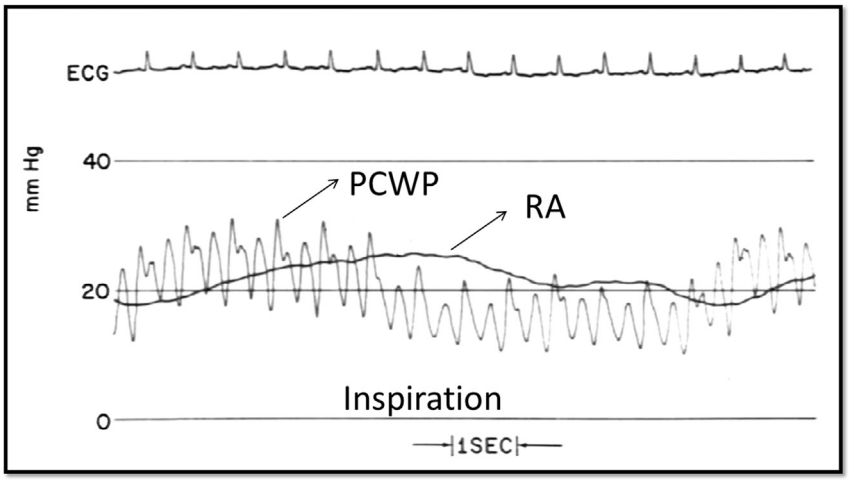

Fig. 7 – Right atrial (RA) pressure trace showing decline <5 mm Hg in inspiration.

hemodynamic parameters based on respiratory variation improves the diagnostic accuracy. Hurrell and colleagues[8] were the first to demonstrate the utility of dynamic respiratory changes in LV and RV pressures and mechanisms were described by Higano et al.[14] In normal individuals, an inspiratory decrease in thoracic pressure is transmitted to the cardiac chambers causing PCWP- LV diastolic gradient to persist irrespective of the phase of respiration and as a result transmitral flow and LV filling remains unchanged. In pericardial constriction physiology, encasing pericardium does not transmit the inspiratory decrease in pulmonary venous and thoracic pressure to cardiac chambers resulting in dissociation. During inspiration, pressure in pulmonary vein falls but LV diastolic pressure remains unchanged causing reduced LV filling. The opposite happens during expiration. The constricting pericardium decreases LV volume out of phase with the corresponding increase in RV volume.[8]

1) Respiratory variation in RA pressure

Central venous pressure (CVP) and RA pressure fails to decline or sometime may increase (Kussmaul's sign) during inspiration due to dissociation of thoracic and cardiac chamber pressures. <5 mm Hg and <3 mm Hg decrease in RA pressure has sensitivity of >70% and 90% respectively, in diagnosing CCP (Fig. 7).

2) Alteration in pulmonary capillary wedge pressure (PCWP) to Left Ventricular (LV) gradient

The flow of blood from pulmonary veins to LV is determined by the pressure gradient during diastole. Normally during inspiration with declining thoracic pressure there is reduction in PCWP and is accompanied by decline in LVEDP. The PCWP- LV gradient thus exists in both phases of respiration allowing uninterrupted LV filling. In a patient of CCP there is inspiratory fall in thoracic pressure as usual. However, owing to the dissociation of thoracic and cardiac pressures LV diastolic pressure remains unchanged. Thus early diastolic gradient between PCWP and minimum LV diastolic pressure is decreased or at times abolished during inspiration (Fig. 8). Consequently the LV filling is reduced in inspiration. During expiration rise in PCWP overcomes the elevation in LV diastolic pressure causing enhanced gradient to improve LV

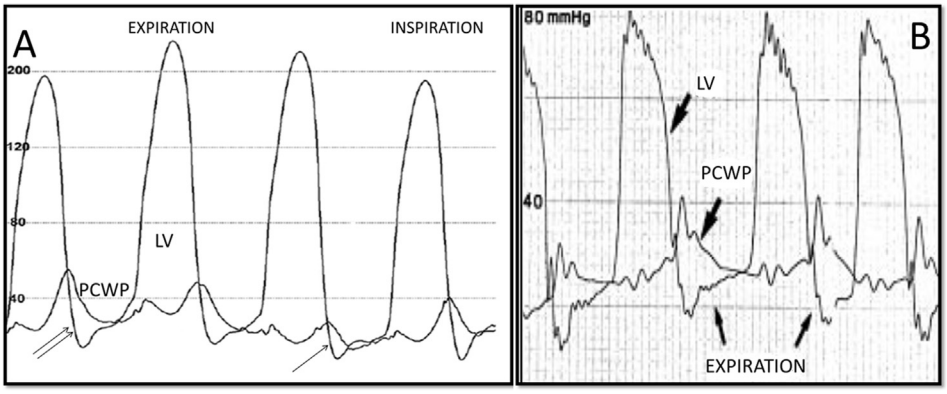

Fig. 8 – Simultaneous LV and PCWP pressure trace. Panel A from a patient of CCP shows marked decrease in PCWP-LV diastolic pressure gradient during inspiration (single arrow) compared to expiration (double arrow). Panel B shows absence of respiratory variation in PCWP-LV diastolic pressure gradient in a patient with RCMP.

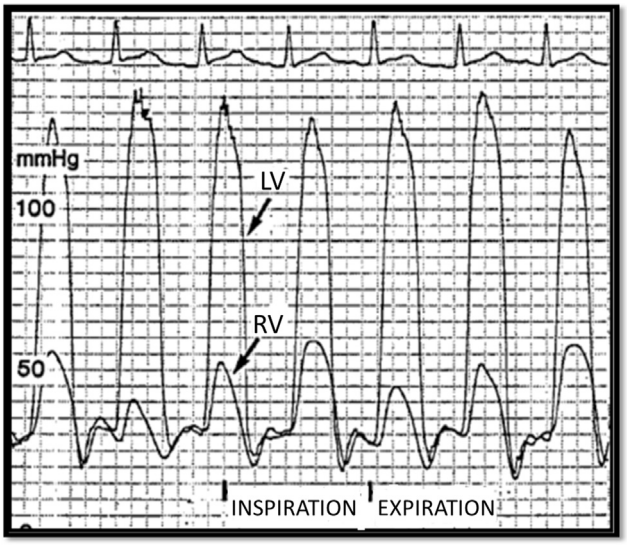

Fig. 9 – Simultaneous RV and LV pressure trace shows ventricular discordance. During inspiration peak systolic pressure in LV is reduced with corresponding increase in RV pressure i.e. they move in opposite direction. Exactly reverse happens during expiration.

filling. This respiratory variation in the early diastolic gradient between PCWP and LV diastolic pressure has a sensitivity of 93% and a specificity of 81% for diagnosing CCP.[8]

3) Exaggerated ventricular Interdependence (ventricular discordance)

When the cardiac volume causes the pericardium to reach the steep portion of its pressure-volume curve, a phenomenon of ventricular interdependence is observed. Put simply, both the ventricles, with their common inter-ventricular septum, are forced to exist in a finite-volume cavity. Thus the total volume of both the ventricles during diastole is fixed and therefore, one ventricle fills preferentially at the expense of the other ventricle. For example e.g., during inspiration with reduced LV filling during inspiration allows shift of septum increasing filling and output from RV. The filling of ventricles is thus out of phase to the other ventricle and is commonly known as 'ventricular discordance' (Fig. 9). Ventricular filling and pressure changes in healthy individuals and RCMP in contrast occur in phase and referred to as ventricular concordance. Ventricular discordance if assessed properly is shown to have a sensitivity of 100% and specificity of 95% for diagnosing constrictive pericarditis.[2,6–15]

Conventional assessment of enhanced ventricular interdependence by comparing peak ventricular pressures is not sensitive. A change in systolic area calculated by multiplying LVSP and systolic ejection period is better determinant of beat to beat change in stroke volumes.[6] The systolic area index (SAI) is then calculated as the ratio of RV area (mm Hg × s) to the LV area (mm Hg × s) in inspiration versus expiration (Fig. 10). The index is significantly higher in patients with CCP compared with RCMP. A ratio > 1.4 has a sensitivity of 97% &predicted accuracy of 100% for identification of CCP.[6]

An intact ventricular septum is mandatory for ventricular discordance. In the presence of a large ventricular septal defect (VSD) effect in concordant changes in ventricular pressures despite coexisting CCP.[15,16] In an interesting case of large VSD and CCP reported by present authors, ventricular discordance was conspicuous by its absence although other respiratory changes consistent with pericardial constriction were unequivocally present.[16]

Assessment of respiratory variation in ventricular filling in patients with suspected CCP has become standard in cardiac catheterization laboratory around the world. These dynamic changes have much improved sensitivity and negative predictive value for the diagnosis of CCP compared to traditional diagnostic criteria (Table 1).

6. Comparison with cardiac tamponade

Tamponade and CCP, while having several features in common, differ in their effect on how they alter ventricular filling.

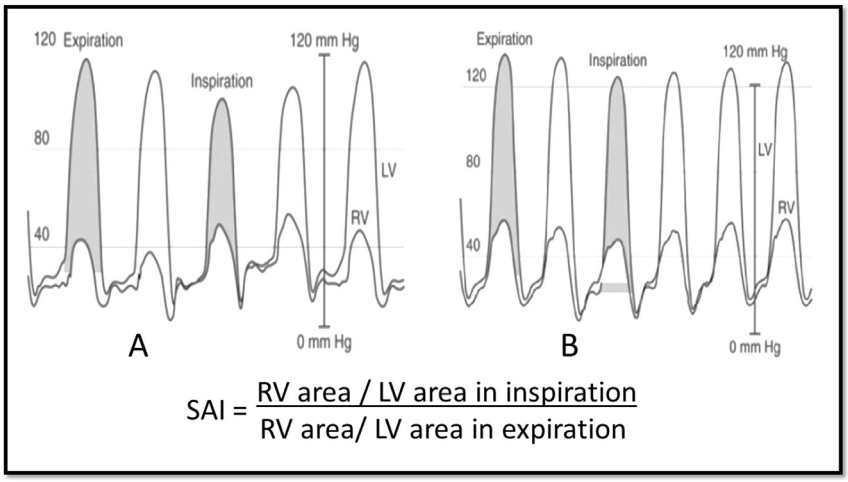

Fig. 10 – Systolic area index (SAI) from a patient with CCP (A) and restrictive cardiomyopathy (RCMP) (B). Ventricular discordance in CCP result in lower SAI compared to RCMP. Adapted from Talreja et al[7] with permission.

Table 1 – Diagnostic accuracy of various hemodynamic parameters for the diagnosis of CCP.

Criteria	Sensitivity	Specificity	PPV	NPV
Hemodynamics at rest				
LVEDP-RVEDP≤5 mmHg	60	38	4	57
RVEDP/RVSP >1/3	93	38	53	89
PASP < 55 mmHg	93	24	47	25
LV RFW ≥ 7 mmHg	93	57	61	92
Respiratory variation in hemodynamics				
Respiratory change in RAP <3 mmHg	93	48	58	92
PCWP/LV respiratory gradient ≥5 mmHg	93	81	78	94
LV/RV interdependence	100	95	94	100
Systolic area index >1.1	97	100	100	95

LV – left ventricle; LVEDP – left ventricular end diastolic pressure; LVRFW – left ventricular height of rapid filling wave; PASP – pulmonary artery systolic pressure; PCWP – pulmonary capillary wedge pressure; RAP – right atrial pressure; RV – right ventricle; RVEDP – right ventricular end diastolic pressure; RVSP – right ventricular systolic pressure.
Adapted from Talreja et al[7] and Higano et al[14] with permission.

Features that are common include: diastolic dysfunction and preserved ventricular ejection fraction; heightened ventricular interaction; increased respiratory variation of ventricular inflow and outflow manifested by pulsus paradoxus (less frequent in CCP, seen in only 30%); diastolic equalization of pressure in all cardiac chambers; and pulmonary hypertension.[17]

The differences stems from differences in the pattern of restriction to ventricular filling. In CCP restriction is limited to mid-late diastole while in cardiac tamponade it is holodiastolic. This is reflected in rapid 'y' descent, 'dip & plateau' pattern and equalization of pressures only during late diastole in cases with CCP. On the contrary equalization of pressures occurs throughout the diastole in cases with cardiac tamponade. In addition, unimpeded transmission of thoracic pressure in cardiac tamponade results in preserved inspiratory increase in systemic venous return (absent Kussmaul sign) and respiratory variation in RA pressure. The preferential filling of RV in inspiration thus is due to increased filling unlike in CCP where it is secondary to reduced LV filling during inspiration.[17]

7. Hemodynamics of effusive-constrictive pericarditis

The hemodynamic alteration in effusive-constrictive pericarditis is the result of combined effect of fluid accumulation and pericardial constriction and therefore the hemodynamic changes are also intermediate.[18] In these patients hemodynamics mimics cardiac tamponade prior to pericardiocentesis and is characterized by - prominent 'x' descent; elevated, equalized diastolic pressures and prominent paradoxical pulse but with preserved 'y' and paradoxical rise in CVP during inspiration (Kussmaul sign) unlike classic cardiac tamponade. Further lack of hemodynamic normalization after pericardiocentesis would indicate underlying pericardial constriction. Pericardiocentesis results in relief of pulsus paradoxus with prominent 'y' descent in RA pressure trace and ventricular "dip and plateau" pattern characteristic of CCP[5] (Fig. 11).

8. Hemodynamics in a patient with constrictive pericarditis with atrial fibrillation

Atrial fibrillation is present in nearly one-third patients with CCP. Pressure measurements in the catheterization laboratory fluctuate with varying RR intervals and respiratory hemodynamics may not be assessed properly. Varying RR intervals of AF can be regularized with temporary ventricular pacing at rates just above the ventricular response.

9. Occult constrictive pericarditis

The presence of dehydration as well as gross congestive heart failure (CHF) interferes with the hemodynamic assessment. Diastolic equalization may not be present in a patient with CCP with low to normal right atrial pressures. Rapid infusion of 500–1000 ml saline over 6–8 min in such a scenario helps in unmasking the hemodynamic changes of CCP.[19] Gross CHF also interferes with the demonstration of hemodynamic abnormalities and can be unmasked by administering diuretics to allow some reduction in filling pressures.

10. Comparison with restrictive cardiomyopathy

The restrictive physiology is common to both CCP and RCMP. Although similar in presentation these two clinical entities differs significantly.[2,9] In a patient with RCMP, abnormal

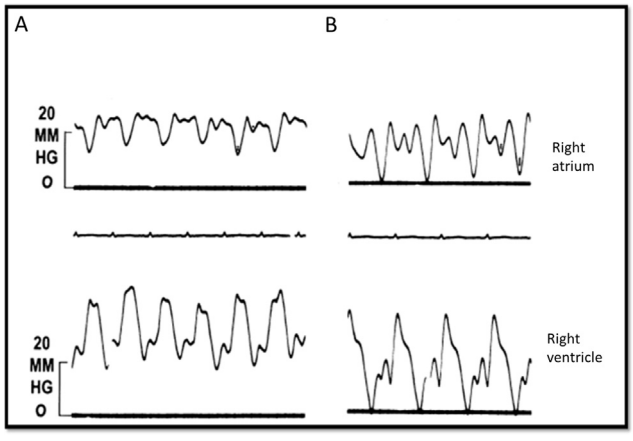

Fig. 11 – Demonstration of hemodynamics in effusive-constrictive pericarditis before (A) and after (B) pericardiocentesis. Prior to pericardiocentesis, 'x' descent is prominent in RA pressure trace while 'y' descent gains prominence after tapping. Pressure waveform in right ventricle (RV) shows elevated end-diastolic pressures and failure of rapid filling wave (RFW) to reach zero; post tapping the square root sign and prominent RFW is evident.

Table 2 – : Hemodynamic difference in CCP and RCMP.

Features	CCP	RCMP
Prominent 'y' descent	Present	Variable
Equal right & left-sided filling pressures	Present	Left 3–5 mm Hg > right
"Square root" sign	Present	Variable
Filling pressures >25 mm Hg	Rare	Common
PASP >50 mm Hg	No	Common
RVEDP/RVSP	> 1/3	<1/3
PCWP/LV diastolic gradient in expiration	Present	Absent
LV/RV interdependence	Discordance	Concordance
LVRFW	> 7 mmHg	≤7 mmHg

LV – left ventricle; LVRFW – left ventricular height of rapid filling wave; PASP – pulmonary artery systolic pressure; PCWP – pulmonary capillary wedge pressure; RV – right ventricle; RVEDP – right ventricular end diastolic pressure RVSP – right ventricular systolic pressure.

myocardium fails to relax and cause restriction to diastolic filling that is more pronounced but not limited to late diastole. The diastolic pressures although are nearly equal but with a difference of >5 mm Hg in LV and RV end diastolic pressures. LV myocardial involvement results in pulmonary arterial hypertension > 50 mm Hg unlike CCP. A comparison of hemodynamic abnormalities in CCP and RCMP is summarized in Table 2.

11. Conclusion

Hemodynamic assessment in a patient with suspected CCP is useful in the diagnosis, and more importantly in differentiating from other pericardial and myocardial disease. Demonstration of enhanced ventricular interaction by dynamic respiratory changes has better predictive value compared to conventional hemodynamic evaluation.

Conflicts of interest

All authors have none to declare.

REFERENCES

1. Yared K, Baggish AL, Picard MH, Hoffman U, Hung J. Multimodality imaging of pericardial diseases. *J Am Coll Cardiol Img*. 2010;3:650–660.
2. LeWinter MM, Hopkins WE. Pericardial diseases. In: Mann DL, Zipes DP, Libby P, Bonow RO, Braunwald E, eds. *Braunwald's Heart Disease*. 10th ed. Philadelphia: Elsevier Saunders; 2015:1636–1657 (Indian reprint).
3. Spodick DH. Macrophysiology, microphysiology, and anatomy of the pericardium: a synopsis. *Am Heart J*. 1992;124:1046–1051.
4. Applegate RJ, Johnston WE, Vinten-Johansen J, Klopfenstein HS, Little WC. Restraining effect of intact pericardium during acute volume loading. *Am J Physiol*. 1992;262:H1725–H1733.
5. Little WC, Freeman G. Pericardial disease. *Circulation*. 2006;113:1622–1632.
6. Sorajja P. Invasive hemodynamics of constrictive pericarditis, restrictive cardiomyopathy, and cardiac tamponade. *Cardiol Clin*. 2011;29:191–199.
7. Talreja DR, Nishimura RA, Oh JK, Holmes DR. Constrictive pericarditis in modern era. *J Am Coll Cardiol*. 2008;51:315–319.
8. Hurrell DG, Nishimura RA, Higano ST, et al. Value of dynamic respiratory changes in left and right ventricular pressures for the diagnosis of constrictive pericarditis. *Circulation*. 1996;93:2007–2013.
9. Baim Donald S, ed. *Grossman's Cardiac Catheterization, Angiography, and Intervention*. 7th ed. Philadelphia, USA: Lippincott Williams & Wilkins; 2006.
10. Shabetai R. Pathophysiology and differential diagnosis of restrictive cardiomyopathy. *Cardiovasc Clin*. 1988;19:123–132.
11. Appleton CP, Hatle LK, Popp RL. Demonstration of restrictive ventricular physiology by Doppler echocardiography. *J Am Coll Cardiol*. 1988;11:757–768.
12. Oh JK, Hatle LK, Seward JB, et al. Diagnostic role of Doppler echocardiography in constrictive pericarditis. *J Am Coll Cardiol*. 1994;23:154–162.
13. Fowler NO. Constrictive pericarditis: new aspects. *Am J Cardiol*. 1982;50:1014–1017.
14. Higano ST, Azrak E, Tahirkheli NK, Kern MJ. Hemodynamic rounds, series II: hemodynamics of constrictive physiology: influence of respiratory dynamics in ventricular pressures. *Cathet Cardiovasc Interv*. 1999;46:473–486.
15. Gupta SK, Saxena A, Talwar S. Chronic constrictive pericarditis: unique cause of heart failure in a child with tetralogy of Fallot. *Pediatr Cardiol*. 2012;33:165–167.
16. Gupta SK, Ramakrishnan S, Kothari SS, Saxena A, Airan B. Hemodynamics of large ventricular septal defect and copexisting chronic constrictive pericarditis masquerading as Eisenmenger's syndrome. *Cath Cardiovasc Interv*. 2014;83:263–269.
17. Spodick DH. Acute cardiac tamponade. *N Engl J Med*. 2003;349:684–690.
18. Sagrista-Sauleda J, Angel J, Sanches A, Permanyer-Miralda G, Soler-Soler J. Effusive-constrictive pericarditis. *N Engl J Med*. 2004;350:469–475.
19. Bush CA, Stang JM, Wooley CF, Kilman JW. Occult constrictive pericardial disease. Diagnosis by rapid volume expansion and correction by pericardiectomy. *Circulation*. 1977;56:924–930.