

CARDIOMYOPATHY

Differential diagnosis of restrictive cardiomyopathy and constrictive pericarditis

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The differentiation of restrictive cardiomyopathy and constrictive pericarditis has been a perennial problem in clinical cardiology. Constrictive pericarditis requires surgical treatment and is usually curable, while restrictive cardiomyopathy, short of cardiac transplantation, is treatable only by medical means and often responds unsatisfactorily. The opinion has often been expressed that there are difficult cases in which only an exploratory operation will allow the two conditions to be distinguished. However, such cases were relatively rare in the past and should be extremely so in the present era. Many differences exist between the two conditions, even though no one diagnostic method can be relied upon to make the distinction by itself.

Constrictive pericarditis

Constrictive pericarditis was recognised in the 19th century and its surgical treatment was developed early in the 20th century. Paul Wood noted in 1961 that only details had been added to the picture presented to the English speaking world by Paul Dudley White in his 1935 St Cyres lecture.^{1,2} White described a “chronic fibrous or callous thickening of the wall of the pericardial sac that is so contracted that the normal diastolic filling of the heart is prevented . . . There may or may not be calcification . . . Parietal pericardium or epicardium may be preponderantly involved . . . one area may be involved, other areas free . . . associated heart disease is extremely rare . . . insidious evolution makes diagnosis more difficult than that of active constrictive pericarditis”. A history of several years duration and a

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Table 1 Comparison of certain features in subacute (elastic) and chronic (rigid shell) constrictive pericarditis

Subacute (elastic)	Chronic (rigid shell)
Paradoxical pulse usually present, other signs of interdependence usually prominent	Paradoxical pulse usually minimal or absent, other signs of interdependence less prominent
Usually an XY waveform (“M” or “W” waveform)	Y is predominant, X sometimes minimal
Dip–plateau pattern less conspicuous, because early diastolic nadir may not approach zero	Dip–plateau usually conspicuous, because early diastolic nadir often reaches zero
Calcification usually absent	Calcification often present
Pericardial effusion sometimes present, generalised or loculated. Constriction is by the visceral pericardium	Pericardial effusion absent. The two layers of pericardium are fused, and jointly constrict the heart
P waves usually normal	P waves often wide, notched and low in amplitude
Atrial fibrillation or flutter rare	Atrial fibrillation or flutter common

Varieties of constrictive pericarditis

- Typical forms
 - chronic (calcific, rigid shell)
 - subacute (non-calcific, elastic)
- Effusive-constrictive
- Localised
- Occult

predominant clinical feature of ascites, simulating liver disease, were notable in White’s series.

Haemodynamic features delineated in the 1940s and ’50s included the narrow pulse pressure in the right ventricle with normal systolic pressure and greatly increased diastolic pressure, a prominent early diastolic dip and later diastolic plateau in right ventricular pressure waveforms, and an additional prominent systolic dip in the right atrial waveform, giving a “W” atrial waveform. Comments on the difficulty of distinguishing constrictive pericarditis from restrictive cardiomyopathy began to appear in the medical literature only after the pressure recordings from cardiac catheterisation began to be used in the diagnosis of constrictive pericarditis. One may suspect that cardiac catheterisation data in the two conditions were more similar than the clinical features.

Since 1960 the clinical profile of constrictive pericarditis has changed greatly. Tuberculous aetiology has become rare in developed countries, while new aetiologies have appeared. Two of them, previous cardiac surgery and previous radiotherapy, are now responsible for up to one third of cases in some centres.³ The term “chronic” is often no longer included in the title, because so many cases are now more appropriately considered to be acute or subacute. Subacute constrictive pericarditis differs in several respects from the chronic cases, as Paul Wood noted in his delineation of the differences between active and inactive tuberculous constrictive pericarditis. A distinction between elastic (subacute) and rigid shell (chronic) constriction has been proposed to help to rationalise these differences⁴ (table 1). Other additions to the clinical profile include the recognition of effusive–constrictive pericarditis,⁵ occult constriction,⁶ localised constriction,⁷ and reversible constriction.⁸ These variant forms of constrictive pericarditis each have some features that differ from the classic chronic constrictive pericarditis of the past (table 2).

Restrictive cardiomyopathy

As the haemodynamics of constrictive pericarditis became known in the 1940s and ’50s it quickly became apparent that amyloid and other forms of myocardial or endocardial disease could have similar haemodynamic features. At the same time, the concept of idiopathic cardiomyopathy was evolving, leading to Goodwin’s 1961 classification into three types.⁹ The hypertrophic and dilated forms

Major forms of restrictive cardiomyopathy

- Amyloid
- Other infiltrative diseases
- Endomyocardial fibrosis
- Idiopathic restrictive cardiomyopathy

quickly became well known, but the third type, “constrictive cardiomyopathy” (later renamed “restrictive cardiomyopathy”), defined as congestive heart failure with neither hypertrophy nor dilatation, was less common and received less attention. The definition of restrictive cardiomyopathy has varied considerably, and the term has usually been used in a broad sense, to include such entities as amyloidosis, tropical endomyocardial fibrosis, endocardial fibroelastosis, haemochromatosis, and eosinophilic endomyocardial disease, as well as the idiopathic cases. Idiopathic restrictive cardiomyopathy, strictly defined to include normal ventricular wall motion as well as normal wall thickness and ventricular chamber dimensions, proved to be relatively rare. Such a strict definition can be carried a step further by defining restrictive cardiomyopathy as those patients who cannot be differentiated from constrictive pericarditis by means of physical examination, chest radiograph, and cardiac catheterisation.¹⁰ No large series of patients with such a condition have been described.

On the other hand, the concept of diastolic heart failure, or diastolic dysfunction, has received a great deal of attention in the past 20

Table 2 Features of variant forms of constrictive pericarditis

Effusive–constrictive pericarditis	Pericardial effusion is present, sometimes loculated, with constriction by the visceral pericardium
Occult constrictive pericarditis	Haemodynamics are normal at rest, but assume the features of constriction after an acute volume load
Localised constrictive pericarditis	Constriction limited to the right or left ventricle. Ventricular interdependence reduced or absent
Transient constrictive pericarditis	During the resolution of acute pericarditis with effusion, constriction develops, but then resolves spontaneously over a few weeks

years.¹¹ When the definition is broadened to permit what is considered to be a lesser degree of systolic dysfunction associated with predominant diastolic dysfunction, diastolic heart failure becomes very common, and perhaps includes about one half of all cases of congestive heart failure. It is particularly common in elderly patients. Such patients may be referred to as having restrictive cardiomyopathy, even though it appears likely that hypertension and increased arterial stiffness are background factors in many of them.¹²

Whatever definition of restrictive cardiomyopathy is used, it is clear that patients who simulate constrictive pericarditis are relatively rare, and that cardiac amyloidosis is the most frequent diagnosis among them. The others have miscellaneous diagnoses, with a small number representing an idiopathic restrictive cardiomyopathy.

Differentiating features

Table 3 lists 17 features, obtained by eight different clinical methods, that provide useful clues in differentiating constrictive pericarditis

Table 3 Features useful in differentiating constrictive pericarditis from restrictive cardiomyopathy

Feature	Constrictive pericarditis	Restrictive cardiomyopathy
Past medical history	Previous pericarditis, cardiac surgery, trauma, radiotherapy, connective tissue disease	These items rare
Jugular venous waveform	X and Y dips brief and “flicking”, not conspicuous positive waves	X and Y dips less brief, may have conspicuous A wave or V wave
Extra sounds in diastole	Early S3, high pitched “pericardial knock”. No S4	Later S3, low pitched, “triple rhythm”. S4 in some cases
Mitral or tricuspid regurgitation	Usually absent	Often present
ECG	P waves reflect intra-atrial conduction delay. Atrioventricular or intraventricular conduction defects rare	P waves reflect right or left atrial hypertrophy or overload. Atrioventricular or intraventricular conduction defects not unusual
Plain chest radiograph	Pericardial calcification in 20–30%	Pericardial calcification rare
Ventricular septal movement in diastole	Abrupt septal movement (“notch”) in early diastole in most cases	Abrupt septal movement in early diastole seen only occasionally
Ventricular septal movement with respiration	Notable movement toward left ventricle in inspiration usually seen	Relatively little movement toward left ventricle in most cases
Atrial enlargement	Slight or moderate in most cases	Pronounced in most cases
Respiratory variation in mitral and tricuspid flow velocity	Greater than 25% in most cases	Less than 15% in most cases
Equilibration of diastolic pressures in all cardiac chambers	Within 5 mm Hg in nearly all cases, often essentially the same	Within 5 mm Hg in a small proportion of cases
Dip–plateau waveform in the right ventricular pressure waveform	End diastolic pressure more than one third of systolic pressure in many cases	End diastolic pressure often less than one third of systolic pressure
Peak right ventricular systolic pressure	Nearly always less than 60 mm Hg, often less than 40 mm Hg	Frequently more than 40 and occasionally more than 60 mm Hg
Discordant respiratory variation of ventricular peak systolic pressures	Right and left ventricular peak systolic pressure variations are out-of-phase	Right and left ventricular peak systolic pressure variations are in-phase
Paradoxical pulse	Often present to a moderate degree	Rarely present
MR/CT imaging	Shows thick pericardium in most cases	Shows thick pericardium only rarely
Endomyocardial biopsy	Normal, or non-specific abnormalities	Shows amyloid in some cases, rarely other specific infiltrative disease

and restrictive cardiomyopathy. The length of the list indicates that no one or two of the features have sufficient sensitivity and specificity to be decisive in all cases. More items could be included, but the list would then become even more unwieldy. The items listed have been selected for having reasonable sensitivity and specificity, and for the most part of being readily and reliably ascertained.

Past medical history

Although many cases of constrictive pericarditis are idiopathic, some have a history of definite or possible acute pericarditis in the past. Factors such as previous cardiac surgery, radiotherapy, connective tissue disease, and thoracic trauma may be the background for the development of constrictive pericarditis, and none of these factors are expected in the past medical history of patients with restrictive cardiomyopathy.

Jugular venous waveform

The typical jugular venous pulse in constrictive pericarditis has two dips per cardiac cycle: the outward movements are less conspicuous. The two dips are the X and Y troughs, in systole and early diastole, respectively. Y is sometimes more prominent than X, especially in chronic (rigid shell) cases, many of whom have atrial fibrillation, a factor that minimises the systolic descent. These features are also common in restrictive cardiomyopathy. What distinguishes constrictive pericarditis is the brief duration of Y, giving a “flicking” appearance in an otherwise continuously distended vein. This is the essential feature of Friedreich’s sign, considered by Paul Wood to be the most characteristic physical sign of constrictive pericarditis. Subacute (elastic) cases are more likely to have comparable X and Y dips, giving a “W” or “M” contour, or even a dominant X descent, as in cardiac tamponade. Patients with restrictive cardiomyopathy sometimes have waveforms with conspicuous outward pulsations, caused by large A waves, or obvious tricuspid regurgitation, patterns not seen in constrictive pericarditis.

Examination of the jugular venous pulse is not completely superseded by recording the atrial pressure waveforms at cardiac catheterisation. The jugular veins exhibit dynamic volume changes, which can be more conspicuous than pressure changes in the relatively low pressure atrial-venous system.

Early diastolic sounds

Extra heart sounds in the early diastolic filling period occur in both constrictive pericarditis and in restrictive cardiomyopathy, but can be suspected of being one or the other of two types by the practised auscultator. In restrictive cardiomyopathy, especially when tricuspid regurgitation is prominent, an S3 frequently occurs, falling approximately 0.12–0.18 s after S2, and demonstrating a “thudding” low pitched character. Such sounds result in a “triple rhythm” effect, that needs deciphering as to which members of the trio are S1, S2, and S3. In constrictive pericarditis an earlier filling

sound occurs, usually 0.06–0.12 s after S2, and somewhat high pitched and “snapping” in character. The auscultatory impression tends to be that of a widely split S2 or a mitral opening snap. The term “pericardial knock,” although commonly used, is not particularly appropriate. The sound does not resemble a knock on a door, and is not caused by the calcified heart knocking against the chest wall, as students sometimes misconceive.

Late diastolic sounds

Some patients with restrictive cardiomyopathy have S4, resulting from a powerful atrial contraction in response to the increased resistance to ventricular filling. S4 does not occur in constrictive pericarditis, despite similar degrees of resistance to filling, probably because the fibrotic constricting process impairs the atrial contraction.

Mitral/tricuspid regurgitation

The physical examination is also helpful when it indicates pronounced mitral or tricuspid regurgitation. Echo Doppler and cardiac catheterisation are part of this assessment. Mitral and tricuspid regurgitation are rarely prominent in constrictive pericarditis, but are often prominent in restrictive cardiomyopathy.

Electrocardiogram

The P waves in constrictive pericarditis tend to be wide and notched, but low in amplitude, reflecting intra-atrial conduction delay. This is seen in chronic cases more often than in the subacute cases. In restrictive cardiomyopathy the P waves may be wide, but have a particular tendency to be increased in amplitude, reflecting left atrial overload or hypertrophy, as in hypertension or aortic valve disease. The difference probably reflects the invasion of atrial myocardium by fibrosis in constrictive pericarditis. Also, the raised atrial and ventricular diastolic pressure causes less stretch of atrial myofibrils in constrictive pericarditis than in restrictive cardiomyopathy, because of the external compression by the pericardium.

Conduction defects, both atrioventricular and intraventricular, are more often features of restrictive cardiomyopathy than they are of constrictive pericarditis. Bundle branch block occurs in perhaps 20–30% of patients with restrictive cardiomyopathy and is rare in constrictive pericarditis. Low voltage is not critically helpful; it is less common in constrictive pericarditis than it is in cardiac tamponade, and it does occur in some instances of restrictive cardiomyopathy, especially those with amyloid. Left ventricular hypertrophy, however, would be an important factor favouring restrictive cardiomyopathy. Amyloid is notable for showing Q waves simulating infarct in some cases, but this can also occur in constrictive pericarditis, probably on the basis of fibrosis invading the myocardium.

Plain chest radiograph

Pericardial calcification seen in the plain chest radiograph is highly specific for constrictive pericarditis, in the context of a differential

diagnosis between constrictive pericarditis and restrictive cardiomyopathy. Calcification is usually absent in subacute constrictive pericarditis, and is therefore less frequent overall than it was in the past. However, it still occurs in approximately a quarter of cases.¹³ It is unclear whether more sensitive methods, such as electron beam computed tomography (CT) or cine fluoroscopy will show pericardial calcification in a higher proportion of patients with constrictive pericarditis, while retaining a similarly high specificity.

Echocardiographic imaging

Echocardiography will have been carried out by the time that a differential diagnosis between constrictive pericarditis and restrictive cardiomyopathy is formulated, because the problem applies only to patients who show normal ventricular chamber dimensions and systolic wall motion. Three further clues from standard echocardiography are particularly important.

The septal notch

In constrictive pericarditis the rate of filling is rapid in early diastole, and the rate of change in ventricular pressure at this time in the cycle is particularly rapid. Slight asymmetry of right and left ventricular filling rate can result in rapid changes in the pressure differential between the two sides of the ventricular septum. The septum may therefore shift in position very abruptly, responding to such rapid changes in pressure. The abnormal septal motion may take several forms, and does not necessarily fit the definition of a “notch”.

Ventricular septal shift with respiration

Reciprocal changes in left and right ventricular volumes with respiration are one aspect of the increased degree of ventricular interdependence that is characteristic of constrictive pericarditis. Because the heart is enclosed within a relatively fixed volume, enlargement of one ventricle tends to be associated with a corresponding decrease in volume of the other ventricle. This contrasts with the non-constricted heart, in which enlargement of one ventricle can be associated with a corresponding increase in volume of the two ventricles combined. The volume of the right ventricle increases in inspiration, both normally and in constrictive pericarditis, as a result of lowered intrathoracic pressure drawing in a greater venous return. This aspect of ventricular interdependence is best seen in the two dimensional echocardiogram as a movement of the ventricular septum toward the left ventricle with inspiration and toward the right ventricle in expiration.

Atrial enlargement

Major enlargement of the right and left atrium is a hallmark of restrictive cardiomyopathy. This occurs in response to pronounced chronic elevation of atrial pressure, and is enhanced by mitral and tricuspid regurgitation. Some enlargement of the atria often occurs in constrictive pericarditis, in which the same sustained

elevation of atrial pressure is present, but it is rare to see the major enlargement that is characteristic of restrictive cardiomyopathy. Presence of the constricting process around the atria appears to account for this difference.

Thickening of the pericardium in the echocardiogram is a second line feature in differentiating constrictive pericarditis and restrictive cardiomyopathy. The limited resolution of echocardiography lowers the specificity of this finding, although it is sometimes notable in patients with constrictive pericarditis. Transoesophageal echocardiography gives better resolution than the conventional transthoracic study, but chest CT or magnetic resonance imaging (MRI) have better specificity.

Doppler ultrasound studies

Doppler ultrasonic studies for differentiating constrictive pericarditis and restrictive cardiomyopathy were introduced in the late 1980s and have proven considerably valuable.^{14, 15} In constrictive pericarditis there is an exaggerated variation in the velocity of early diastolic filling of the two ventricles with respiration. The variation is reciprocal, the tricuspid velocity increasing in inspiration and the mitral velocity decreasing. The reciprocal ventricular variation reflects ventricular interdependence, and occurs to a much lesser degree in restrictive cardiomyopathy. It is usually prominent in subacute constriction and less prominent or absent in chronic (rigid shell) cases. It appears that in the chronic (rigid shell) cases the variations in intrathoracic pressure are not transmitted to the interior of the heart.

Further Doppler methods have been added, including the assessment of respiratory variation in pulmonary venous flow velocity, and the study of mitral annular movement (“tissue Doppler”).¹⁶ The place of the newer Doppler methods in differential diagnosis remains to be determined.

The sensitivity and specificity of the Doppler respiratory method may be as high as 85–90% in expert hands. However, the studies are difficult to carry out and to interpret. They should ideally incorporate a simultaneous graphic record of the phases of respiration. Irregular patterns of breathing, irregular cardiac rhythm, and short diastolic periods resulting from rapid heart rate cause difficulty in interpretation. Falsely positive results can be seen when intrathoracic pressure variations are exaggerated, as in asthma or chronic obstructive airway disease; in such conditions the flow velocity in the superior vena cava should be recorded, because the superior vena cava has much larger respiratory variation in flow velocity with respiration in pulmonary disease than it does in either constrictive pericarditis or restrictive cardiomyopathy.

Cardiac catheterisation

Cardiac catheterisation studies have perhaps received too much emphasis in the differential diagnosis between constrictive pericarditis and restrictive cardiomyopathy.¹⁷ Indeed, the diagnostic dilemma may almost be defined as existing when the cardiac catheterisation results do

not distinguish the two. However, a carefully conducted haemodynamic study is likely to yield important clues.

Equilibration of diastolic pressures

Nearly equal levels of diastolic pressure in all chambers of the heart are a hallmark of constrictive pericarditis, and reflect the usually symmetrical pathological process around the entire heart. Somewhat greater elevation of pressure on the left side than the right is more characteristic of restrictive cardiomyopathy. Comparison of instantaneous end diastolic pressure in the two ventricles is perhaps the most critical way to evaluate this, but in practice a comparison of the mean pressures in the right atrium and the left atrium (or the pulmonary artery wedge position) may be more reliable, because such recordings are less subject to troublesome artefacts. A discrepancy of more than 5 mm Hg is very unusual in constrictive pericarditis, but can be seen when the constriction is relatively or totally localised. The discrepancy is more than 5 mm Hg in many cases with restrictive cardiomyopathy that otherwise resemble constrictive pericarditis rather closely.

Dip-plateau waveform

The dip-plateau, or square root-like, waveform in the right ventricular pressure waveform is a classic hallmark of constrictive pericarditis, but is also the feature of restrictive cardiomyopathy that most commonly simulates constriction. The dip-plateau is most prominent in chronic (rigid shell) cases, where there is initially no limitation of filling and the ventricular diastolic pressure approaches zero before beginning its rapid rise to the elevated plateau level. In the subacute (elastic) constrictive cases, now more common, there is some limitation of filling even in beginning diastole, and the nadir does not approach zero.

Commonly used catheters that are soft, small in calibre, and connected to the transducers by long, fluid filled connectors produce distorted waveforms in right ventricular pressure recordings that obscure the differences in the dip-plateau waveform that occur in constrictive pericarditis and restrictive cardiomyopathy.

Discordant peak systolic pressure variation

Another aspect of ventricular interdependence, characteristic of constrictive pericarditis in contrast to restrictive cardiomyopathy, is the discordant variation of right and left ventricular peak systolic pressure levels with respiration.¹⁸ In restrictive cardiomyopathy the two pressures vary together, while in constrictive pericarditis they vary out-of-phase with one another. Right ventricular peak systolic pressure rises with the onset of inspiration, while the peak pressure falls in the left ventricle. This is a simple observation to make during cardiac catheterisation, but it may not be looked for, most frequently because there is an emphasis on assessing the similarity of diastolic pressures in the two ventricles and the pressure recorder is set at a sensitive calibration that leaves the

systolic peaks above the top of the scale. Such recordings must minimise artefacts to be accurately interpreted, and a graphic recording of respiration is also useful.

Paradoxical pulse

Paradoxical pulse is not often mentioned as a distinguishing feature between constrictive pericarditis and restrictive cardiomyopathy. Indeed, some authors state that paradoxical pulse is not a feature of uncomplicated constrictive pericarditis, and some state that it does occur in restrictive cardiomyopathy. Both statements are doubtful. Paradoxical pulse is indeed minimal or absent in the classic chronic (rigid shell) constrictive pericarditis that typified the condition in years past. In the subacute (elastic) cases that are more often seen currently, a moderate paradoxical pulse is often present. This may occur with or without the presence of some pericardial effusion. The respiratory variation is readily seen in direct arterial pressure recordings during cardiac catheterisation, although it is not as pronounced as that typically seen in cardiac tamponade and is often not readily detected by bedside examination. The respiratory variation in restrictive cardiomyopathy is rarely enough to raise a suspicion of constrictive pericarditis, particularly if cases of cor pulmonale are correctly recognised. Since paradoxical pulse is an exaggerated degree of a normal phenomenon, its definition is arbitrary, and it should be treated as a continuous variable, not a categorical one.

Magnetic resonance and computed tomographic imaging

CT and MRI of the thorax have been used since the early 1980s as an improved method of evaluating abnormal thickening of the pericardium. Most cases of constrictive pericarditis do indeed show an apparent pericardial thickness of 3 mm or more, at least in some areas.¹⁹ CT and MRI often appear to show only focal areas of pericardial thickening in cases where the constriction is present around the entire heart. Surgeons often note variable degrees of pericardial thickness in different areas, that do not necessarily correspond to differences in the degree of constriction. It is perhaps insufficiently realised, however, that some patients have constriction with relatively small degrees of thickening. The normal pericardium is less than 1.0 mm thick; a considerable increase may not exceed the threshold of abnormality in a CT or MRI. Indeed, the constricting pericardium can be visually unimpressive, or even appear normal at first glance to the surgeon at the time of operation. Some cases of occult constriction appear to have anatomically normal visceral and parietal pericardium. Thus, the principal limitation of CT and MRI is the occurrence of falsely negative studies. In addition, the finding of thickened pericardium does not necessarily indicate that constriction is present.

CT and MRI have approximately equal value in demonstrating thickening of the pericardium. CT is therefore preferable in most cases, with MRI usually reserved for

Points frequently helpful in favouring constrictive pericarditis over restrictive cardiomyopathy

- History—active pericarditis
- Examination—Freidreich's sign, paradoxical pulse
- ECG—absence of intraventricular conduction defect
- Chest radiograph—pericardial calcification
- CT/MRI—thickened pericardium
- Echocardiogram—septal notch
- Doppler—ventricular interdependence
- Cardiac catheterisation—close equilibration of diastolic pressures
- Biopsy—absence of amyloid or other infiltrative disease

patients with an intolerance of iodinated contrast agent. CT is analogous to a single “snapshot”, while MRI represents the average of many heart beats, gated to the cardiac cycle; for this reason, neither method is well adapted to assessing the variations in chamber volumes with respiration.

Endomyocardial biopsy

Endomyocardial biopsy is a nearly certain method of diagnosing cardiac amyloidosis. Therefore, the form of restrictive cardiomyopathy that is the most frequent simulator of constrictive pericarditis should be diagnosable without exploratory thoracotomy, if the diagnosis is considered as a possibility preoperatively. Some other entities such as haemochromatosis and eosinophilic cardiomyopathy can also be diagnosed at biopsy. Cases of idiopathic restrictive cardiomyopathy, however, have only non-specific abnormalities in the endomyocardial biopsy, and such abnormalities may also be found in some cases of essentially uncomplicated constrictive pericarditis. The biopsy is therefore helpful chiefly if it shows a specific infiltrative disease.²⁰

Conclusions

Many new diagnostic methods have been introduced since the earliest proposals that only exploratory thoracotomy will differentiate constrictive pericarditis and restrictive cardiomyopathy in all cases. It seems that exploratory thoracotomy should be needed very rarely if ever in the current era. However, a major pitfall is to expect that only one or two diagnostic methods can be regularly decisive in making the differential diagnosis. The apparent urge to rely on one or two methods, to the relative neglect of other methods, may be a disadvantage resulting from the current tendency to divide cardiology into subspecialties based on

the use of specific technologies. A broad clinical viewpoint, in which the results of many diagnostic methods are synthesised, is necessary to achieve the optimal differential diagnosis.

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