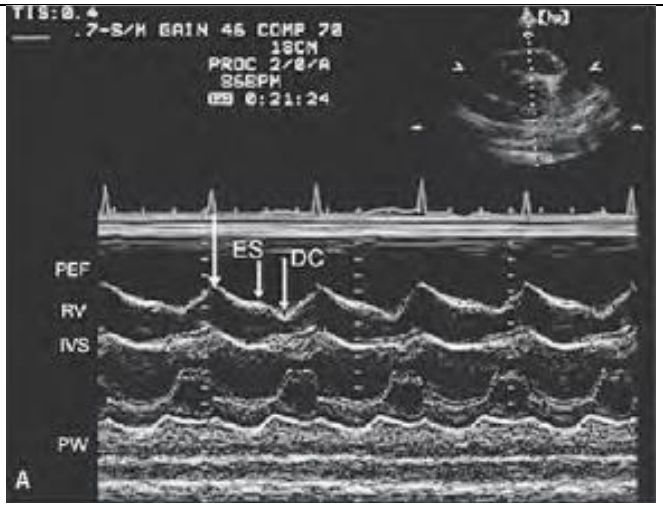
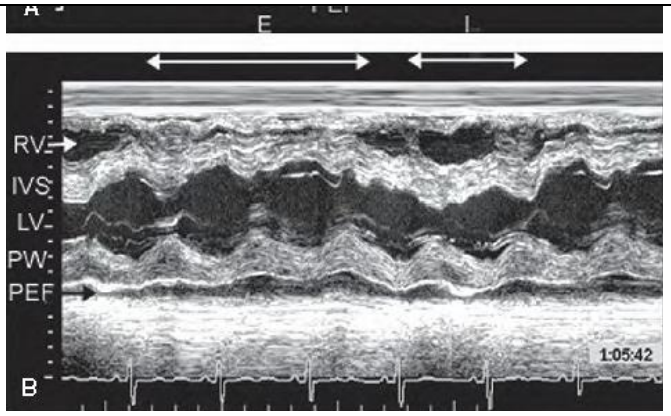
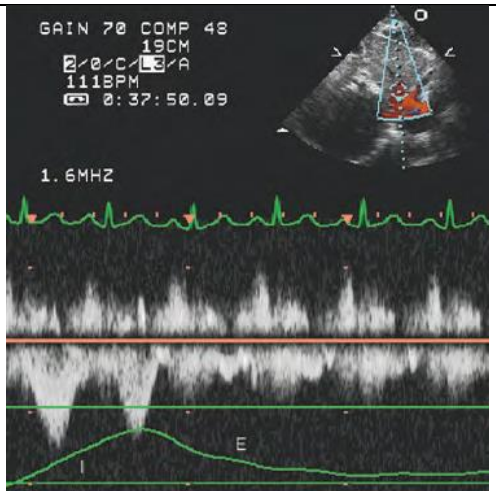
PERICARDIAL DISEASE

- The pericardial effusion ends anteriorly to the descending aorta and is best visualised in the PLAX.
- PSAX is actually very useful sometimes for looking at posterior effusions and also for guiding drainage from the anterior intercostal approach.
- Suprasternal and apical 2C are generally not useful for assessing pericardial effusions.
- Pericardial effusion will overlap the RA and does not usually overlap the LA.
- A pericardial effusion does not usually exceed 4 cm in depth.
- The heart is usually hypermobile within a pericardial collection
- Pericardial effusions are common in cardiac amyloidosis, along with valve thickening and LV thickening, with small ventricles and large atria.
- In pericardial tamponade, Patients usually have a tachycardia, elevated JVP, quiet heart sound and hypotension are often breathless although have clear lungs.
- TB is a recognised cause of calcification in various parts of the body, including the pericardium.
- Pericarditis, uraemia and connective tissue disorders are some of the causes of pericardial constriction.
- Pericardial constriction classically leads to equalisation of diastolic pressures in all cardiac chambers; this can be demonstrated at cardiac catheterisation.
- In congenital absence of pericardium, the entire cardiac structure is shifted towards the left, resulting in the appearance of RV volume overload (RV appears enlarged) in the standard parasternal views. Bronchogenic cysts, ASD and bicuspid AVs are all associated with this condition. Absence of the left-sided pericardium is more common.
- Pericardiocentesis can be performed using echo or fluoroscopy; indeed, it is sometimes performed blind in emergency situations. Agitated saline can be used to confirm the needle is in the pericardial space (and not the RV) before advancing the guidewire. Slowing of the tachycardia usually results when pericardial fluid is removed, and reduction in fluid cavity is immediately seen on echo. Loculated effusions may increase the risk of cardiac laceration.

ECHOCARDIOGRAPHIC FEATURES OF PERICARDIAL TAMPONADE

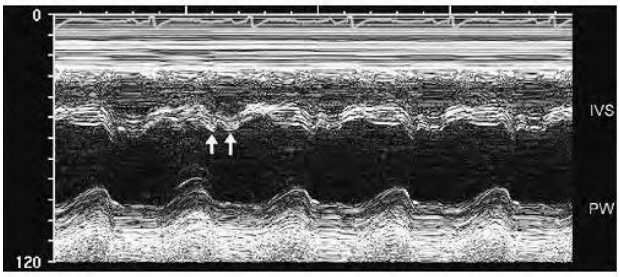
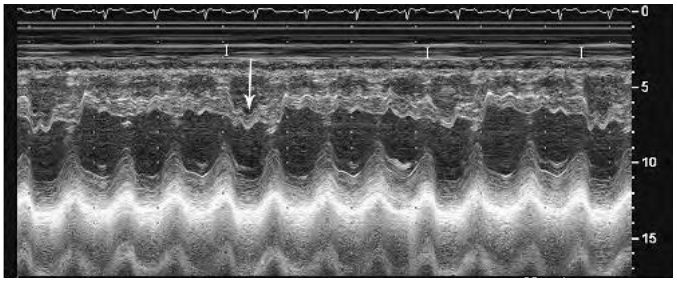
- **Diastolic chamber collapse**- as pressure within the pericardium rises, the right-sided chambers collapse (at least in part) during diastole. The first chamber to be affected is the right atrium (RA), which is seen to collapse during atrial systole, followed by the right ventricle (beginning with the right ventricular outflow tract, RVOT). Rarely, LA or even left ventricular (LV) collapse may be seen in severe cases.
- **Exaggerated respiratory variation of mitral and tricuspid inflow velocities**- in a normal individual, **i**nspiration increases the flow of blood returning to the r**i**ght heart and decreases the flow of blood into the left heart; while **e**xpiration increases the flow of blood to the l**e**ft heart and decreases the flow of blood into the right heart. The normal respiratory variation in E wave size is < 25% for the TV and < 15% for the MV (<10% for both LVOT and RVOT peak velocities and VTIs). Cardiac tamponade and constrictive pericarditis exaggerate this respiratory variation. In case of suspected tamponade or constrictive pericarditis, the PW Doppler recording of mitral and tricuspid inflow velocities should be performed at both a slow and a fast sweep speed. The slow speed is useful for evaluation of respiratory variation, whereas the fast speed is used to obtain measurements
- **Dilated IVC with reduced or absent inspiratory collapse-** the IVC is normally 1.5–2.5 cm in diameter and this falls by > 50% on inspiration. In the presence of tamponade, the IVC dilates and the inspiratory fall in diameter is reduced or absent.
- PW Doppler imaging of SVC and hepatic vein flows can also reflect the elevated intrapericardial pressure and altered filling patterns. Normally, vena caval flow occurs in both systole and diastole and is nearly continuous. In the presence of elevated intrapericardial pressure, flow during diastole is truncated and the majority of flow into the heart occurs during ventricular systole. The hepatic vein flow pattern may also reflect the exaggerated respiratory phase dependency of RV filling

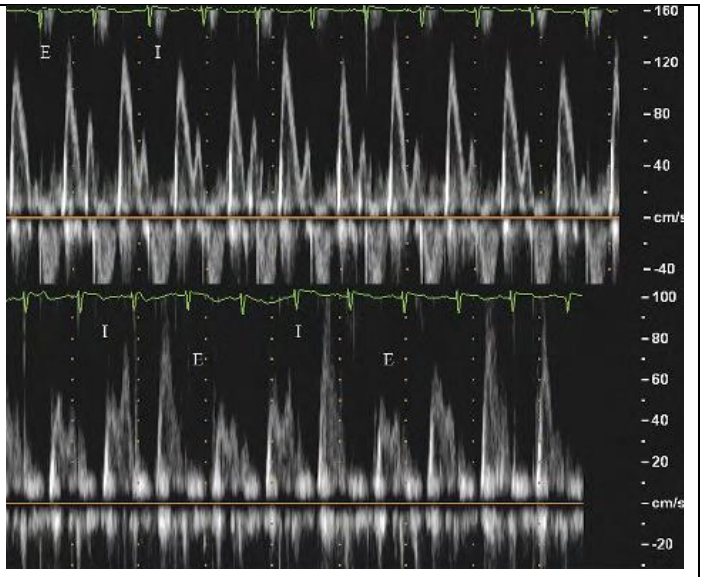
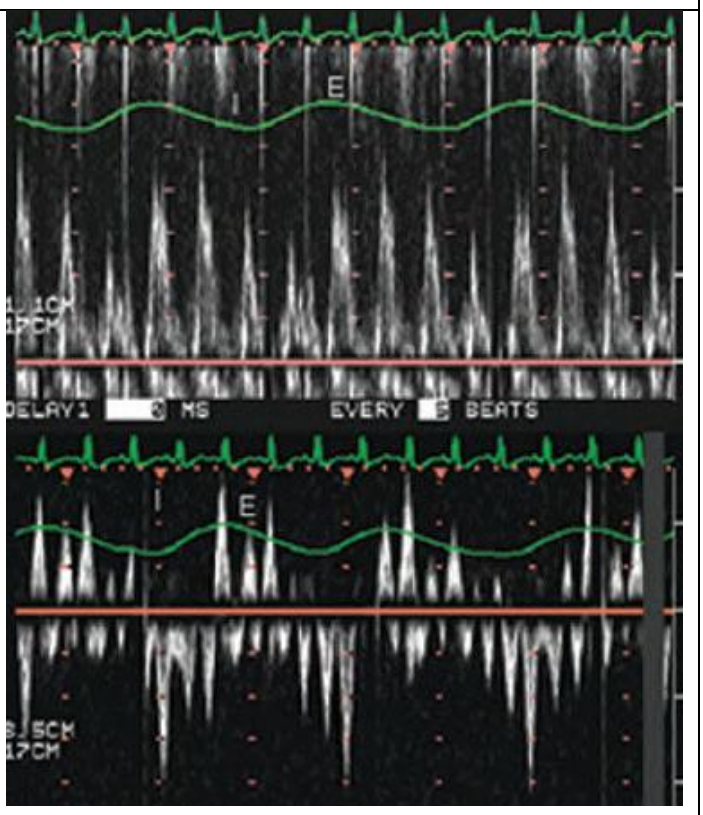
Early diastolic collapse of the right ventricular free wall in patient with pericardial tamponade. the unlabelled arrow denotes the beginning of systole. The position of the right ventricular free wall at end-systole is also noted. Immediately after end-systole, the right ventricular free wall moves posteriorly, indicative of diastolic collapse. DC, diastolic collapse; ES, end-systole, PE, pericardial effusion.	
Respiratory variation in right ventricular size and septal position in patient with pericardial tamponade.	
PW Doppler imaging of the hepatic vein recorded in a patient with a hemodynamically significant pericardial effusion. Note the loss of forward flow in the hepatic veins during the expiratory (E) phase of the respiratory cycle. Flow out of the hepatic veins is confined exclusively to the early inspiratory (I) phase.	

Hint: High right-sided pressures may mask features such as collapse of the RA and RV, as well as Doppler VTIs.

ECHOCARDIOGRAPHIC FEATURES OF M-MODE IN CONSTRICTIVE PERICARDITIS

- **Thickened pericardium:** this can be difficult to assess on echo and can be more accurately assessed with CCT or CMR
- **Abnormal multiphasic diastolic motion (2nd reverberation) of the ventricular septum** related to an increase in ventricular interdependence (*abrupt posterior motion early in diastole, caused by rapid RV diastolic filling, followed by little motion in mid-diastole, caused by equalization of RV and LV pressures, followed by abrupt anterior motion at the end of diastole due to further RV filling after atrial contraction*).
- **IVS 'bounce' during inspiration**: a shift in the ventricular septum towards the LV with inspiration and towards the RV with expiration due to ventricular interdependence. During inspiration the RV filling increases, and because of the constraints imposed by the pericardium this leads to shifting of the septum towards the LV
- **Exaggerated respiratory variation of mitral and tricuspid inflow velocities** as seen in pericardial tamponade
- **Dilated IVC with reduced or absent inspiratory collapse** as seen in pericardial tamponade
- **Expiratory increase in diastolic flow reversal of hepatic vein flow recorded by PW Doppler.** Diastolic flow reversal of hepatic vein flow is seen in PH and constrictive pericarditis. Respiratory variation helps differentiate between them. It is augmented with expiration in constriction, whilst in PH it remains constant.
- **Increased SVC flow reversal during expiration**.
- **Annulus paradoxus**: paradoxical to the positive correlation between E/E′ and LA pressure in patients with myocardial disease, an inverse relationship was found in patients with constrictive pericarditis. This is due to the fact that E′ is reduced in patients with diastolic dysfunction and increased filling pressures, but is not reduced, despite increased filling pressures, in patients with constrictive pericarditis.

• Thickened posterior pericardial echoes. • Multiphasic diastolic motion (second reverberation) of the septum (*abrupt posterior motion early in diastole, caused by rapid RV diastolic filling, followed by little motion in mid-diastole, caused by equalization of RV and LV pressures, followed by abrupt anterior motion at the end of diastole due to further RV filling after atrial contraction*)	
• In elastic constriction (as opposed to classic calcific pericardial constriction), there is respiratory-dependent interaction of the right and left ventricular filling that manifests as exaggerated respiratory variation in septal position (inspiratory septal shift resembling the type of septal motion abnormality seen in cardiac tamponade). As the total intracardiac volume is limited by the constrictive pericardium, any inspiratory increase in right-sided filling must be accompanied by a reciprocal decrease in left-sided filling. Note that with inspiration (I), there is expansion of the right ventricular cavity with abrupt posterior motion of the ventricular septum (*arrow*).	

• Pulsed Doppler recording of the mitral (upper panel) and tricuspid (lower panel) valve inflows in a patient with documented calcific constriction. Notice the relatively mild degree of variation in mitral inflow from expiration (E) to inspiration (I) but the dramatic respiratory variation in tricuspid flow inflow in this patient.	
• PW Doppler recording of mitral inflow and hepatic vein flow recorded in a patient with constrictive pericarditis. **A:** Note the marked respiratory variation in mitral E-wave velocity. **B:** This is associated with exaggerated early expiratory (E) hepatic vein flow reversal.	

Separation of Constrictive Pericarditis from Restrictive Cardiomyopathy

Parameter	**Constriction**	**Restriction**
Atrial size	Normal	Dilated
Pericardial appearance	Thick/bright/calcified	Normal
Septal motion	Abnormal (2nd reverberation)	Normal
Septal position	Varies with respiration (inspiratory bounce)	Normal
Mitral E/A	Increased (≥2.0)	Increased (≥2.0)
Deceleration time	Short (≤160 ms)	Short (≤160 ms)
Annular e′ (LV diastolic function)	Normal	Reduced (≤10 cm/sec)
Pulmonary hypertension	Rare	Frequent
Left ventricular function	Normal	Normal
LV dimensions	Normal	LVH
Mitral/tricuspid regurgitation	Infrequent	Frequent (TR > MR)
Isovolumic relaxation time	Varies with respiration	Stable with respiration
Respiratory variation of mitral E velocity	Exaggerated (≥25%)	Normal
Hepatic vein flow	Expiratory **diastolic** reversal	Inspiratory **diastolic** reversal
Colour M-mode mitral valve Vp	Increased (≥55 cm/sec)	Reduced