

VENTRICULAR INTERACTION AND SEPTAL DEFORMATION

- **Normally** septal motion is dominated by the LV, and the septum bulges towards the RV in the PSAX.
 - **RV overload** → septal bulge starts to flatten, and this gives the LV a '**D**' shape rather than its usual circular appearance in the PSAX:
 - **Isolated RV volume overload** (e.g. severe TR) → the RV is dilated and the septum is pushed towards the LV (D shape) in **diastole only** with normal septal movement (controlled by the LV) in systole.
 - **RV pressure overload** (e.g. severe PH) → the RV is hypertrophied and septal flattening is maintained throughout the cardiac cycle (**diastole and systole**). Therefore, D-shaped septum in both systole and diastole suggests RV volume overload causing the diastolic shape and an element of pressure overload causing the RV to take over septal dynamics in systole. A flat septum during systole and diastole implies LV and RV mass is equal such that neither ventricle controls septal motion any more than the other.
 - **LBBB** → the initial septal activation sequence is reversed and the RV and right septal activation precedes the LV activation → Early systolic downward motion (beaking) of the IVS, often followed by anterior (**paradoxical**) septal motion throughout the remainder of systole (sometimes called **septal bounce**). The magnitude of this abnormal motion can be subtle and is often only noted on M-mode. An additional consequence of LBBB is a fall in LV peak systolic pressure, which leads to impaired MV closure and exacerbates functional MR (which may occur not just in systole but also in late diastole, so-called '**pre-systolic MR**')
 - **RBBB** does not alter the initial sequence of activation of the LV and hence, the magnitude of cardiac dyssynchrony with RBBB is considerably **less** than with LBBB
 - **Paradoxical septal motion**- is defined as movement of the IVS away from the left ventricular free wall during systole which is the opposite of its normal movement which is inward toward the LV free wall during systole.
Causes:
 - **Conduction system based:** LBBB, RV pacing (particularly from RVOT), PVC, Ventricular preexcitation (WPW type B)
 - **Abnormal ventricular interaction:** RV volume overload, RV pressure overload, RVH, RV tumour, arrhythmogenic RV dysplasia, constrictive pericarditis, large pericardial effusion, following a pericardial incision or heart surgery, congenital absence of pericardium, MS, pulmonary embolism, pulmonary hypertension, ASD (dilatation of the entire right heart with paradoxical septal motion should raise the possibility of ASD, causing RV volume overload), repaired Tetralogy of Fallot
 - **MI or IHD** involving the septal myocardium, stunning or hibernation of the septal myocardium
 - **Miscellaneous:** Ascites, pregnancy, hiatus hernia
- Hint:** RV pacing leads are increasingly placed in the RVOT septum to produce more physiological pacing.

