Right ventricular pacing causes RV dysfunction - a 2D/3D Echocardiographic study

**Introduction**: Right Ventricular (RV) apical pacing, though generally well-tolerated, may have detrimental cardiac effects, and the pacemaker (PM) lead can cause tricuspid valve regurgitation (TR). The effect of PM on Left ventricular (LV) contractility and synchrony are well demonstrated, but little is known about its influence on RV function. The complex geometry of the RV makes its evaluation more difficult than the LV. Traditionally the RV has been largely considered a conduit ventricle and thus has been the neglected ventricle. However, in recent times the effect of RV function affecting prognosis in heart failure patients has rejuvenated interest in RV dysfunction. Assessment of TR and evaluation of RV function after pacemaker implantation was study objective.

**Methods**: The study was performed in 40 patients who required permanent PM for AV block in structurally normal heart. The baseline right sided echocardiographic parameters including 3D RVEF (Ejection fraction) following RV pacing, within 24 hours and at follow up of 6 months.

**Result**: The age was 70+11 years. Immediately post PM implantation (within 24 hours), there was no change in the grade of TR. However, during follow up at 6 months, 9 subjects (22.5%) developed TR, though this was mild in 1 patient and trivial in 8 subjects. The pulmonary arterial systolic pressure (PASP) didn't change immediately post-PM (within 24 hours). However, at follow up after 6 months, the mean PASP increased 25.13 + 0.791 mm Hg vs 31.38 + 11.821 mm Hg, p = (0.002). This was likely related to left ventricular diastolic dysfunction which was unmasked or increased by right ventricular pacing. The RVEF was found to be significantly depressed both soon after PM insertion and at follow up (Table).

**Conclusion**: TR develops in a significant proportion of patients who undergo RV pacing, and its severity increases with time. There is mild RV dysfunction post-PM, which increases over the course of six months. RV ventricle dysfunction was not related to presence of TR and without significant dilatation of right side of heart. Absence of TR should not be taken as sole evidence of absence of RV dysfunction.