Introduction: Transvenous right ventricular pacing poses a unique challenge in patients with surgical tricuspid intervention. Epicardial pacing has been used but lead failure rates are higher compared to transvenous pacing. The purpose of this study was to evaluate the changes in interventricular and intraventricular activation in patients with tricuspid valvular (TV) disease and/or surgery who underwent permanent coronary sinus (CS) lead implantation as the sole ventricular lead.

Methods: This study examined 10 patients with surgical TV or significant native TV dysfunction who underwent permanent pacemaker implantation with a left ventricular CS lead as the only ventricular pacing lead (to avoid crossing the TV). We assessed changes in interventricular and intraventricular activation. Interventricular activation was defined as time from the onset of the pacing spike (or QRS) to the peak flow through the outflow tracts as measured by pulsed wave doppler. Additional interventricular activation was defined as time from the onset of the pacing spike (or QRS) to the peak velocity of lateral wall activation as defined by the S’ with tissue doppler imaging (TDI). Intraventricular activation was defined as time from the onset of the pacing spike (or QRS) to the peak velocity of septal and lateral activation as defined by the S’ with TDI. All comparisons were made with intrinsic conduction prior to CS lead implant and with CS lead pacing post CS lead implantation.

Result: We examined 10 patients who had ECHOs prior to surgical intervention and ECHOs after CS lead placement to assess any change in ventricular activation timing. There was no statistically significant change in activation after CS pacing compared to prior CS pacing. The activation of the RV lateral wall as measured by the S’ on TDI showed a trend towards significance. Prior to implant it was 164 ms and after CS implant it was 195 ms (P =0.06).

Conclusion: We examined 10 patients who had ECHOs prior to surgical intervention and ECHOs after CS lead placement to assess any change in ventricular activation timing. There was no statistically significant change in activation after CS pacing compared to prior CS pacing. The activation of the RV lateral wall as measured by the S’ on TDI showed a trend towards significance. Prior to implant it was 164 ms and after CS implant it was 195 ms (P =0.06).