Post cardiac surgery junctional ectopic tachycardia: A 'Hit and Run' tachyarrhythmia as yet unchecked

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Junctional ectopic tachycardia (JET) presents in two scenarios: The rarer idiopathic and incessant form seen early in infancy with a structurally normal heart and the more common self-limiting variety limited to the early postoperative period after surgical repair of congenital heart disease.

Postoperative arrhythmias after open-heart surgery for congenital heart disease contribute significantly to both morbidity and mortality. Arrhythmias often well tolerated in a normal heart can be a major cause of morbidity and mortality after cardiac surgery for congenital heart disease.[1] JET is the most frequent hemodynamically significant tachycardia in the postoperative setting and usually occurs within the first 24 hours after operation.[2-4] Hemodynamic instability results from loss of atrioventricular synchrony and extreme tachycardia.[5]

Diagnosis of JET generally requires a heart rate greater than 170 beats per minute with QRS morphology similar to the sinus rhythm QRS complex, atrioventricular dissociation, and ventricular rate faster than the atrial rate.[6] JET can occur at a lower rate in adult population with increased vagal tone and slower atrioventricular (AV) conduction as compared to children. It can also present with 1:1 retrograde ventriculo-atrial conduction. In patients with postoperative bundle branch block, JET can manifest as wide QRS tachycardia with atrioventricular dissociation or 1:1 retrograde ventriculo-atrial conduction. In this scenario, the P waves may fall within the QRS or ST-T segment. At fast rates, sinus tachycardia and supraventricular arrhythmias can pose a diagnostic challenge. P waves can be the clue in such situations that can be identified with oesophageal leads or Lewis leads or the atrial pacing wires.

The precise mechanism of JET is not known, but it is believed to result from mechanical trauma to the proximal conduction tissue related to suture placement or indirect stretch injury with resultant edema which can occur during resection of muscle bundles, correction of right ventricular outflow tract and correction of ventricular septal defects.[7] Paradoxically, JET also occurs in patients in whom surgery does not involve areas around the atrioventricular node. Furthermore cardiopulmonary bypass (CPB) with ischemia–reperfusion and the related cellular biochemical effects as well as medical interventions such as electrolyte shifts and catecholamine administration may affect the stability of the cellular membrane and result in an increased myocardial irritability and automaticity.[8]

Risk factors associated with JET include younger patient age, duration of CPB and aortic cross-clamp time (ACC), electrolyte disturbances, use of inotropes and type of surgery.[6,9] Several studies have elucidated the risk factors associated with JET. [Table 1] Although higher dose of inotropic agents have been accepted in all major studies as a significant risk factor, the role of surgery involving peri-nodal areas is still debated. Moak et al.,[11] in their recent study had categorically refuted mechanical injury to the AV node area as a strong risk factor for postoperative JET.

The prospective study on postoperative JET by Abelaziz et al.,[12] published in this issue of the Annals, has analyzed 194 patients after their cardiac surgery under CPB. They have divided their cohort into 3 groups – one with postoperative JET, one with other postoperative arrhythmias and finally those with no arrhythmias. This division further helps to highlight the risk factors that may be more specific for JET with respect to other postoperative arrhythmias. They have used the conventional measures to diagnose JET, but have still highlighted the importance of atrial leads, Lewis leads and esophageal leads in confirming their diagnosis.

The staged protocol for management of JET has some scientific basis, even though the use of bolus dose of magnesium sulphate is questioned by several studies.[2,4,9]

The number of patients who developed JET in the postoperative period (27%) in this study sample is significantly high as compared to previous studies. The reasons that the authors have extrapolated for this high number include the higher number of surgeries being done in their centre, which have an inherently high risk of developing postoperative JET like Tetralogy of Fallot

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Table 1: Incidence and risk factors for postoperative JET

Study	Type of study	Study population	No. of patients with JET	Risk factors of postoperative JET
Hoffman et al., 2002[9]	Prospective	594	33 (5.6%)	Younger age (<6 months) Longer CPB times (>75 min) Dopamine use postoperatively at dose-3 to 7.5 µg/kg/min (OR 6.2 $P \leq 0.01$)
Batra et al.(2006)[2]	Prospective	336	27 (8%)	Younger age (2.75 +/− 2.44 vs. 5.38 +/− 7.25 years; $P < 0.01$), Longer CPB times (126 +/− 50 vs. 85 +/− 73; $P < 0.01$), Higher inotrope score (6.26 +/− 7.55 *vs.* 2.41 +/− 8.11; $P < 0.01$)
Delaney et al. (2005)[3]	Prospective	189	16 (8.5%)	Younger age (22 *vs.* 45 months) Longer CPB time (189 *vs.* 109 min) Longer ACC time (105 *vs.* 44 min)
Grosse-Wortmann et al. 2010[10]	Prospective	494 (96 neonates, 398 infants & children)	Neonates- 9% Infants & children – 5.4%	Neonates- VSD repair (OR-18.82, $p = 0.001$) Infants & children- VSD repair (OR-3.69, $P = 0.01$) Subaortic stenosis repair (OR-5.93, $P = 0.04$)
Mildh et al.2011[4]	Retrospective	1001	51 (5%)	Longer CPB time (138 *vs.* 119 min; $P = 0.002$) High body temp (38.0 *vs.* 37.4°C; $P = 0.013$) High troponin (3.7 *vs.* 2.1 µg/l; $P < 0.001$) Norepinephrine use (23/51 *vs.* 35/130, $P = 0.019$) Surgery involving closure of VSD (65%)
Moak et al.2013[11]	Prospective	750	115 (15.3%)	Total surgical time > 120 minutes (HR-1.272, $P = 0.005$), ACC time > 30 minutes (HR-1.473, $P = 0.03$), CPB times > 60 minutes use of cardioplegic solution, administration of inotropic agents

ACC: Aortic cross clamp, CPB: Cardiopulmonary bypass, HR: Hazard ratio, OR: Odds ratio

(TOF), atrioventricular canal defect, Senning operation and also the later age of surgery for congenital heart disease (CHD) in their hospital. These explanations may be acceptable but even then the number of postoperative JET incidence for each respective surgery is still higher as compared to previous data.[9,13] It has also been shown previously that incidence of JET decreases with higher age and weight of the baby and so the latter explanation also cannot hold good.[9,14]

It is interesting to note that risk factors when compared between the 3 groups mentioned above have shown that CPB, aortic cross clamp time and postoperative use of inotropes were significantly higher in postoperative JET patients but there was no significant difference in these parameters between non-JET arrhythmia group and no arrhythmia group. This clearly highlights the importance of perioperative factors being a major determinant of JET and anatomical factors being a minor determinant only. The structural abnormalities and their corresponding electrophysiological changes may be stronger determinants of non-JET arrhythmias.

In this study, patients experienced JET in the immediate postoperative period (range, 1 to 24 hours post bypass; median, 6.8 hours). Previous studies have documented JET to occur in early postoperative period but not confined to first 24 hours.[11]

In the present study, JET successfully resolved in 86.5% (n = 47) of patients, with 57% (n = 30) responding

to conventional measures (cooling, magnesium sulphate, sedation and minimizing catecholamines) and the remaining 43% (n = 23) were controlled by IV amiodarone infusion in addition to conventional measures. In Hoffman's study, 39.4% required other measures in addition to conventional treatment for management.[9]

The statistical difference between the role of milrinone and other inotropes in causing JET is not readily explained. Although other inotropes act directly on the beta-receptors, milrinone acts only at the second messenger level but the final mechanism of action at the cellular level is common to both. In Hoffman's study, milrinone had an equally significant role in JET as other inotropes.[9]

Post-surgical JET has a mortality as high as 14%.[4] So preventive strategies should be given adequate importance in the early postoperative period. Prevention of hyperthermia and minimal inotropic usage are important. The administration of magnesium either as a bolus at the completion of the rewarming phase of CPB or as a continuous intravenous infusion after discontinuance from CPB has been demonstrated to lower the incidence of postoperative JET and other cardiac arrhythmias.[15] Mahmoud et al., demonstrated a lower incidence of PO JET (38% *vs.* 21%) when propranolol was initiated preoperatively for TOF patients.[16]

The management of JET patients requires a staged therapeutic approach beginning with conventional

measures that include active avoidance of hyperthermia, optimal sedation and pain control and minimizing exogenous catecholamines. In one of the earlier publications evaluating the effectiveness of a staged protocol, Walsh et al.,[6] found that conventional measures could control JET in 24% of patients. Based on available data and institutional experience, they refined their original protocol by avoiding ineffective drugs like digoxin, verapamil, propranolol and phenytoin. These measures significantly reduced the time to JET control. In the case of non-responders or patients with hemodynamic instability, they have followed a sequential order of atrial pacing, induction of hypothermia with posterior cooling blankets and finally a combination of hypothermia and antiarrhythmic drugs like amiodarone and procainamide. In 70 of their 71 patients, JET was managed successfully with 63 patients responding within 2 hours.

This order has undergone considerable changes overtime with intravenous amiodarone being preferred by several centres now as 1st or 2nd line option.[4,17] Active surface cooling using cooling blankets and cold saline infusions is effective in terminating resistant JET.[17,18] In the early postoperative period, surface cooling may be preferred prior to amiodarone infusion. In patients with JET presenting in the late postoperative period, the use of surface cooling prior to amiodarone infusion is still debated. Atrial pacing at fast rates for AV sequential pacing is another option for resistant JET.[9] Dexmedetomidine may be useful in prevention and treatment of perioperative JET.[19,20] Other antiarrhythmics like intravenous flecainide,[21] procainamide[22] and nifekalant[23] have been reported to be useful for controlling JET resistant to other measures. Ablation as a final resort after failed medical management in hemodynamically unstable patients have also been reported.[24,25]

The incidence of JET has gradually declined over the years with improved surgical techniques and preventive measures like minimizing inotropic usage and avoiding hyperthermia in the postoperative period. But JET continues to contribute significantly to postoperative morbidity. Management of JET with general supportive measures, active surface cooling and antiarrhythmics like amiodarone has reined this postoperative hazard to a great extent.

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