

Surgical Cryoablation for Life-Threatening Postoperative Junctional Tachycardia Alexander J. Tsoutsinos, John Papagiannis, Andrew C. Chatzis and George E. Sarris Ann Thorac Surg 2007;84:286-288 DOI: 10.1016/j.athoracsur.2007.02.052

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Table 1. Structural Features and Clinical Data

	Patient 1	Patient 2	Patient 3
Age at repair	5 days	8 days	10 days
Gender	Female	Female	Male
Body weight	2,620 g	2,881 g	2,290 g
Aortic obstruction	IAA (type B)	Coarctation	IAA (type A)
Isthmus of aortic arch		2.6 mm	
Ascending aorta	5.2 mm	6.2 mm	5.3 mm
Ventricular septal defect	Perimembranous outlet	Doubly committed	Doubly committed
Subaortic diameter	4.1 mm	Outlet septum lacking	Outlet septum lacking
Aortic annular diameter (% of normal)	3.8 mm (69%)	4.2 mm (66%)	5.1 mm (86%)
Leaflets of aortic valve	Tricuspid, fused	Dysplastic, thickened	Bicuspid, fused
Preoperative LVEDV (% of normal)	109%	93%	89%
Cardiopulmonary bypass time (minutes)	281	199	220
Cardiac arrest time (mins)	134	79	83
LAP (mm Hg) within first 24 hours	10	8	8
CVP (mm Hg) within first 24 hours	11	8	10
Initial mixed venous saturation in ICU (%)	40	46	44
Duration of inotropic support (days)	36	14	25
ICU/HDU stay (days)	18	38	42
Postoperative catheter			
LV ejection fraction	0.72	0.71	NA
LVEDV (% of normal)	71%	118%	96%
LV pressure (systolic/end diastolic)	104/~10	94/~7	98/~10
Aortic pressure (systolic/diastolic/mean)	102/38 (63)	95/44 (68)	92/36 (55)
RV ejection fraction	0.74	0.56	NA
RVEDV (% of normal)	88%	150%	91%
RV pressure (systolic/end diastolic)	33/~7	26/~6	33/~6
PA pressure (systolic/diastolic/mean)	22/8 (14)	23/2 (11)	32/6 (10)
Cardiac index	3.3	3.0	4.1
Follow-up	60 mos	54 mos	20 mos

CVP = central venous pressure; HDU = high dependency unit;IAA = interruption of aortic arch; ICU = intensive care unit; LAP = left atrial LVEDV = left ventricular end-diastolic volume; LV = left ventricle; NA = not available;RV = rightPA = pulmonary artery;pressure; ventricle; RVEDV = right ventricular end-diastolic volume.

We thank Dr Masaki Hamamoto for his contribution in operative schemas.

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Surgical Cryoablation for Life-Threatening Postoperative Junctional Tachycardia

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Junctional ectopic tachycardia is usually a limited but potentially life-threatening postoperative arrhythmia.

FEATURE ARTICLES

Accepted for publication Feb 20, 2007.

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We describe a case of malignant postoperative junctional ectopic tachycardia in a 13-month-old child who had undergone transatrial and transpulmonary repair of tetralogy of Fallot resistant to all conventional therapeutic measures and complicated by recurrent ventricular tachycardia. The arrhythmia was finally treated by open surgical cryoablation of the atrioventricular node and the implantation of a permanent pacemaker. The patient made an uneventful recovery followed by good longterm outcome.

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unctional ectopic tachycardia often occurs in the early postoperative period after open heart surgery for congenital heart disease [1, 2]. It is particularly common after repair of tetralogy of Fallot and other procedures that require surgical maneuvers near the atrioventricular (AV) node and bundle of His, yet its pathogenesis remains speculative [2-4]. Significant hemodynamic compromise can occur during junctional ectopic tachycardia (JET) due to the rapid heart rates and loss of AV synchrony. Reported therapeutic measures include combinations of fluid replacement, correction of electrolyte and metabolic imbalance, avoidance of inotropic agents as tolerated, systemic hypothermia, antiarrhythmic drugs, and overdrive pacing [4]. The arrhythmia typically abates and completely resolves in a few days. We present an unusual case of severe and medically refractory early postoperative JET leading to recurrent episodes of ventricular tachycardia, treated effectively by intraoperative cryoablation of the AV node and the implantation of a permanent pacemaker.

A 13-month-old boy (8.2 kg) underwent complete transatrial and transpulmonary correction of tetralogy of Fallot. This consisted of the closure of the ventricular septal defect with a Dacron patch (Boston Scientific, Natick, MA), subpulmonary myectomy, pulmonary valvotomy, enlargement of the main pulmonary artery, pulmonary valve annulus, and distal right ventricular outflow tract with an autologous pericardial patch and tricuspid valvuloplasty. Despite an immediate post-cardiopulmonary bypass rhythm of JET at 185 bpm, initial hemodynamic measurements were excellent. Intraoperative transesophageal echocardiogram confirmed complete closure of the ventricular septal defect, absence of significant residual right ventricular outflow tract, and good ventricular filling and function.

Initial pharmacologic support consisted of milrinone (0.5 mcg/kg/min), dobutamine (5 mcg/kg/min), and sodium nitroprusside (0.5 mcg/kg/min). During the first postoperative day, increasing tachycardia (up to 200 beats/min) (Fig 1), accompanied by mild hypotension prompted discontinuation of dobutamine, loading with digoxin, initiation of active cooling (cooling blanket) and attempts at overdrive pacing. This was unsuccessful, as a trial therapy with procainamide was also unsuccessful. After stabilization of the hemodynamic measurements, a

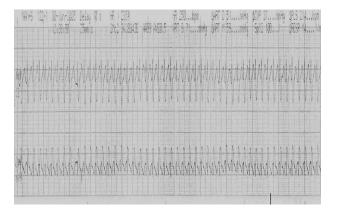


Fig 1. A 12-lead electrocardiogram showing junctional ectopic tachycardia.

continuous infusion of amiodarone was started after an initial loading dose, while body temperature was further reduced down to 33°C. Due to continuation of JET on the postoperative day 2, an esmolol infusion was added. Recurrent episodes of severe tachycardia followed by ventricular tachycardia and severe hypotension required direct current cardioversion. Because of hemodynamic instability, an epinephrine infusion (0.05 mcg/kg/min) became necessary. The patient remained under deep anesthesia and muscle relaxation. On postoperative day 3, despite normal acid base balance and electrolyte levels, hemodynamic instability became more pronounced and two episodes of ventricular tachycardia required external cardiac compressions and electric cardioversion.

An attempt to control the arrhythmia in the electrophysiology laboratory with or without extracorporeal membrane oxygenation was considered, yet severe hemodynamic instability and recurrent episodes of ventricular tachycardia, cardiac arrest, and the risk of air aspiration and embolization under extracorporeal membrane oxygenation deemed emergency surgical cryoablation safer. Under normothermic cardiopulmonary bypass and with the heart in persistent rapid JET, the previous right atriotomy was reopened and a brief test of cryolesion at -30°C was applied at the apex of the triangle of Koch leading to immediate cessation of the arrhythmia. Four permanent lesions (at -80°C for 2 to 3 mins each) were created in the triangle of Koch. The procedure was performed using a system for transvenous cryoablation because of the unavailability of a surgical system. To circumvent the technical problem of the catheter tip temperature being low, precluding initiation of therapy because the electronics interpreted the catheter as being outside the body, the catheter tip was placed at the target site and the atrium was filled with warm saline. This raised the temperature at the tip of the catheter allowing the system to start refrigerant flow.

The cardiac rhythm was altered to AV dissociation with a ventricular rate of less than 100 beats/min. A test of isoproterenol infusion failed to induce JET or other tachycardia. Dual-chamber pacing was started through temporary electrodes. Permanent pacemaker epicardial electrodes (ie, atrial and ventricular steroid-eluting bipolar electrodes) were placed, and after routine chest closure the patient was transferred to the intensive care unit in good hemodynamic condition. He was extubated on postoperative day 7. On postoperative day 20 the patient remained in complete heart block and a permanent pacemaker was placed. He was discharged home and remains well and asymptomatic in DDD-paced rhythm at 1 year of follow-up.

Comment

We described an unusually severe case of postoperative JET requiring emergency ablation of the AV node. The pathogenesis of JET after repair of congenital heart defects remains elusive, although surgical maneuvers near the conduction system, catecholamines, and inflammatory mediators are considered potential factors [1]. This arrhythmia constitutes the most common postoperative complication after tetralogy of Fallot repair in our institutional experience [3]. A variety of treatment schemes have been proposed [1, 4–7]. However, a small number (8.1%) of patients with JET have persistent arrhythmias with potentially lethal outcomes [2, 8].

We used all conventional therapeutic measures including normalization of electrolytes and acid-base balance, use of the lowest possible inotropic doses, moderate hypothermia, and pharmacologic therapy at doses that were tolerated. Extracorporeal membrane oxygenation as a method of temporary support that could allow recovery from the arrhythmia with maintenance of AV conduction was considered; nonetheless, the potential for other complications was significant. Extreme hemodynamic instability in the absence of mechanical circulatory support precluded safe transport to the catheterization laboratory for transcatheter ablation of the AV node. Therefore we chose to proceed with open surgical cryoablation of the AV node as has been reported by Braunstein and colleagues [8] in a patient with intractable JET and cardiogenic shock after a Senning operation for transposition of the great arteries using a 3-mm cryoprobe at -70° C.

In our case, despite the unavailability of a surgical cryoablation system, we were able to successfully apply cryoenergy intraoperatively using a system designed for transvenous therapy. An additional point that this case proves is that the site of origin of JET is in fact the compact AV node and not the bundle of His as has been suggested in some reports [2, 4]. This was obvious as the tachycardia ceased immediately after application of cryoenergy on the compact AV node, at the apical part of the triangle of Koch.

In conclusion, this case demonstrates that in rare cases of life-threatening postoperative JET that are resistant to all conventional medical therapies, open surgical cryoablation can provide a safe and effective solution.

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Unusual Pathogenesis of Spontaneous Pneumothorax Secondary to Wegener's Granulomatosis

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Spontaneous pneumothorax represents a rare and potentially severe complication of Wegener's granulomatosis. A 31-year-old man with Wegener's granulomatosis on immunosuppressive therapy was admitted for a right massive spontaneous pneumothorax. After chest drainage he presented with a prolonged air leak that required a surgical treatment. Histologic findings did not reveal any necrotizing granulomatous vasculitis, but only subpleural fibrous tissue. We hypothesize that pneumothorax could be related to the subpleural fibrous retraction induced by immunosuppressive therapy.

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Wegener's granulomatosis (WG) is an idiopathic systemic inflammatory disease that primarily affects the upper and lower respiratory tract and the kidneys. The pleuro-pulmonary lesions of WG are gen-

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Accepted for publication Jan 22, 2007.

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