Surgical substrates of postoperative junctional ectopic tachycardia in congenital heart defects

Ali Dodge-Khatami, MD
Owen I. Miller, MD
Robert H. Anderson, MD
Allan P. Goldman, MD
Juan Miguel Gil-Jaurena, MD
Martin J. Elliott, MD
Victor T. Tsang, MD
Marc R. de Leval, MD

See related editorial on page 615.

From the Cardiothoracic Unit, Great Ormond Street Hospital for Children National Health Service Trust, and the Institute of Child Health, London, United Kingdom.

Research at the Institute of Child Health and Great Ormond Street Hospital for Children National Health Service Trust benefits from research and development funding received from the National Health Service Executive.

Received for publication July 12, 2001; revisions requested Aug 31, 2001; revisions received Oct 9, 2001; accepted for publication Oct 12, 2001.

Address for reprints: Marc R. de Leval, MD, Cardiothoracic Unit, Great Ormond Street Hospital for Children NHS Trust, Great Ormond St, London WC1N 3JH, United Kingdom (E-mail: DelevM@gosh.nhs.uk).

J Thorac Cardiovasc Surg 2002;123:624-30

Copyright © 2002 by The American Association for Thoracic Surgery

0022-5223/2002 \$35.00+0 12/1/121046

doi:10.1067/mtc.2002.121046

Background: Junctional ectopic tachycardia is a major cause of postoperative morbidity after surgery for congenital cardiac disease. To elucidate the mechanism of junctional ectopic tachycardia, surgical correlations were studied in four types of congenital heart defects involving closure of a ventricular septal defect, relief of right ventricular outflow tract obstruction, or both.

Methods: Between 1997 and 1999, a total of 343 consecutive patients underwent repair of tetralogy of Fallot ($n = 114$), common truncus arteriosus ($n = 10$), ventricular septal defect ($n = 161$), and atrioventricular septal defect ($n = 58$). Variables studied included demographic and bypass data, surgical approaches toward ventricular septal defect closure and relief of right ventricular outflow tract obstruction, and resection as opposed to division of muscle bundles.

Results: Junctional ectopic tachycardia occurred most frequently after repair of tetralogy of Fallot ($n = 25$; 21.9%), with no cases occurring after repair of common trunk, 6 occurring after repair of ventricular septal defect (3.7%), and 6 occurring after repair of atrioventricular septal defect (10.3%). Stepwise logistic regression revealed that resection of muscle bundles ($P < .0001$), higher bypass temperatures ($P < .03$), and relief of right ventricular outflow tract obstruction through the right atrium ($P < .05$) significantly and independently predicted postoperative junctional ectopic tachycardia.

Conclusions: Relief of right ventricular outflow tract obstruction appears to be more important in the causation of junctional ectopic tachycardia than does ventricular septal defect closure, which may explain the higher incidence of this complication after tetralogy of Fallot repair. Muscular resection seems to be more arrhythmogenic than is simple division. Increased traction through the right atrium for relief of right ventricular outflow tract obstruction would fit the hypothesis that enhanced automaticity of the His bundle, the morphologic substrate for junctional ectopic tachycardia, may result from direct trauma or infiltrative hemorrhage of the conduction system. When feasible, techniques avoiding both extensive muscle resection and excessive traction should be applied during resection of right ventricular outflow tract obstruction.

Junctional ectopic tachycardia (JET) is a relatively malignant arrhythmia that is rarely reported in its spontaneous congenital form¹ and occurs more frequently after surgical correction of congenital heart defects.²⁻⁷ Aggressive management is mandatory because of the potential for considerable morbidity; the lack of atrioventricular synchrony and the high ventricular rate may lead to diminished cardiac output, which may in turn lead to increased adrenergic tone, further accelerating the heart rate.^{2,8} With careful medical and nursing support, JET is a self-limiting disorder that usually resolves in 2 to 8 days. Intensive care typically includes surface cooling, atrial pacing, and antiarrhythmic rate-control therapy.³⁻⁶ Although catecholamines may worsen the tachyarrhythmia, such inotropes are commonly used to support the circulation. JET has been reported after the repair of every type of cardiac anomaly, but it is most often observed after correction of tetralogy of Fallot and surgical intervention in proximity to the atrioventricular node and the bundle of His—in other words, operations that include closure of a ventricular septal defect (VSD).⁷

Although the genesis, impact, and long-term consequences of postoperative arrhythmias in these groups of patients are well described,⁹⁻¹² relatively little has been reported about possible surgical contributions to their origin. In this study we analyzed various perioperative variables active at the time of surgical repair and their potential relation to postoperative JET. It is hoped that certain surgical variables may be modified in an attempt to prevent or lessen the incidence of this malignant arrhythmia.

Methods

Between January 1997 and December 1999, a total of 343 consecutive patients underwent surgical repair for selected congenital heart defects at Great Ormond Street Hospital for Children National Health Service Trust, London. The lesions selected included closure of isolated VSD ($n = 161$), repair of tetralogy of Fallot ($n = 114$), repair of atrioventricular septal defect (AVSD, $n = 58$), and repair of common truncus arteriosus ($n = 10$). In all cases a VSD was closed surgically.

Data were collected retrospectively from patient records, operative notes, cardiopulmonary bypass (CPB) data sheets, and postoperative 12-lead ECGs. Surgery was performed with similar techniques by the same three surgeons (M.R.d.L., M.J.E., V.T.T.) during the 3-year period under standard CPB techniques and cold antegrade crystalloid cardioplegia.

We recorded age at repair, weight, and previous palliative operations. Other variables recorded were the surgical techniques used to achieve VSD closure and to relieve right ventricular outflow tract obstruction (RVOTO). Specifically, we distinguished between resection and division of obstructing muscle bundles. *Resection* was defined as excision of muscle leaving denuded raw surfaces of myocardium devoid of endocardium, whereas *division* was defined as the result of simple sharp-blade splitting of obstructive muscle bundles.

Dichotomous intraoperative variables were pulmonary valvotomy or commissurotomy, pulmonary transannular patch, right ventricular patch, pulmonary arterioplasty patch, transatrial closure of VSD, resection of muscle bundles (vs division), transatrial relief of RVOTO, transventricular relief of RVOTO, and transpulmonary relief of RVOTO. CPB data included aortic crossclamp time, CPB time, deep hypothermic circulatory arrest time, and lowest temperature on CPB.

Diagnostic criteria for JET included the following: (1) narrow QRS tachycardia in the absence of surgically induced right bundle branch block, (2) a rate between 170 and 230 beats/min or greater than the 98th percentile for age, (3) atrioventricular dissociation with hemodynamic repercussion, and (4) a ventricular rate faster than the atrial rate. The primary outcome variable was the occurrence of JET, as manifested on the postoperative 12-lead ECG. When available, an atrial-lead ECG obtained with the temporary epicardial pacing wires was used as an adjunct to diagnosis.

Univariate analysis followed by stepwise logistic regression was applied with the SAS statistical program (SAS Institute, Inc, Cary, NC).

Results

The median age of the patients was 5.9 months, with a range from 0.1 to 369 months.

Mortality

Overall mortality was 2.9%, with 10 patients dying, but the rate was 8.1% ($n = 3$) among patients with JET ($P < .05$). According to diagnosis, deaths occurred in 4 patients after repair of tetralogy of Fallot, 3 of whom had JET. There were 4 deaths after repair of common truncus arteriosus and 2 after repair of AVSD, but none of these patients had JET. The total incidence of postoperative JET was 10.8% ($n = 37$). It was most frequently seen after repair of tetralogy of Fallot ($n = 25$, 21.9%), followed by AVSD ($n = 6$, 10.3%) and VSD ($n = 6$, 3.7%). No cases were seen after repair of common truncus arteriosus. The incidences of JET as a postoperative complication were statistically similar among the three surgeons, even when this was considered for the tetralogy subgroup (25.8% vs 15.8% vs 24.4%).

Morbidity

The mean duration of postoperative mechanical ventilation and mean stay on the intensive care unit were increased for patients in JET relative to those without: 187 versus 83 hours ($P < .0001$) and 210 versus 107 hours ($P < .0001$), respectively.

Surgical Technique

Resection of muscle bundles was performed in 79 patients, including 67 resections during tetralogy of Fallot repair, 1 resection in a patient with common truncus arteriosus, 10 resections during closure of VSD, and 1 resection during AVSD repair. Although one of the three surgeons more liberally resects muscle bundles (2-fold incidence relative to

TABLE 1. Stepwise logistic risk factors for JET in the complete study group (n = 343)

Factor	P value
Resection	<.0001
Higher CPB temperature	<.03
Transatrial RVOTO relief	<.05
Palliation	<.3
Pulmonary valvotomy	<.7
Transannular patch	<.4
Right ventricular patch	<.95
Pulmonary artery patch	<.7
Transatrial VSD closure	<.6
Trans-right ventricular RVOTO relief	<.1
Trans-pulmonary arterial RVOTO relief	<.3
Weight	<.4
Aortic crossclamp time	<.2
CPB time	<.8
Hypothermic circulatory arrest time	<.4
Age at operation	<.5

colleagues), this practice did not significantly affect the occurrence of JET among his patients. Pulmonary valvotomy or commissurotomy was necessary in 98 patients; 97 of these were patients with tetralogy of Fallot. Transannular patches were fashioned in 82 cases, right ventricular patches were fashioned in 106 cases, and augmentation pulmonary arterioplasty was performed in 112 cases.

Surgical Access

Closure of a VSD with a patch was performed with exposure through the right atrium in 314 cases (92%). By exclusion, other approaches for VSD closure were through the right ventricle or through the pulmonary trunk. Relief of RVOTO was achieved through the right atrium (n = 96), through the right ventricle (n = 65), or through the pulmonary trunk (n = 65). Multiple approaches to achieve RVOTO relief were possible and were recorded in a cumulative fashion. There was no significant difference in the choice of surgical approach among the three surgeons.

Stepwise logistic regression revealed that resection or excision of muscle bundles, higher CPB temperatures, and relief of RVOTO through the right atrium significantly and independently predicted the postoperative occurrence of JET ($P < .0001$, $P < .03$, and $P < .05$, respectively), as summarized in Table 1. Incisions and suture lines into the right ventricular outflow tract, as are seen with right ventricular or transannular patches, were not associated with a higher incidence of postoperative JET (Table 1).

Because of the higher incidence of JET after repair of tetralogy of Fallot, this subgroup of patients was analyzed individually. The same statistical model was used, with two additional variables taken into consideration, the preoperative oxygen saturation value on room air and the intraoperative ratio of right to left ventricular pressures measured

TABLE 2. Stepwise logistic risk factors for JET in tetralogy of Fallot (n = 114)

Factor	P value
Resection	<.05
Higher CPB temp	<.02
Transatrial RVOTO relief	<.6
Palliation	<.6
Pulmonary valvotomy	<.8
Transannular patch	<.5
Right ventricular patch	<.7
Pulmonary artery patch	<.2
Transatrial VSD closure	<.4
Trans-right ventricular RVOTO relief	<.4
Trans-pulmonary arterial RVOTO relief	<.3
Weight	<.99
Aortic crossclamp time	<.9
CPB time	<.7
Hypothermic circulatory arrest time	<.9
Age at operation	<.98
Preoperative oxygen saturation	<.4
Intraoperative right/left ventricular ratio	<.8

after CPB. Again, resection or excision of muscle bundles and higher CPB temperatures significantly and independently predicted postoperative JET, although to a less marked degree ($P < .05$ and $P < .02$, respectively). Relief of RVOTO through the right atrium did not retain a statistically significant effect (Table 2).

Discussion

JET remains a troublesome postoperative arrhythmia of uncertain cause with a wide spectrum of severity. Treatment is largely supportive. Our protocol commenced with surface cooling to 34°C to 35°C,^{3,6} followed by atrial pacing to achieve atrioventricular synchrony⁴ and then an early introduction of amiodarone by continuous intravenous infusion,⁵ if cooling and pacing had not controlled the ventricular rate. To avoid stress-induced catecholamine release and to facilitate active cooling, all patients had maximal sedation and muscle relaxation. Concomitant postoperative electrolyte imbalance was aggressively managed with appropriate infusions of potassium, calcium, and magnesium.

Although the precise cause of JET is unknown, enhanced automaticity of the bundle of His in either its right atrial or right ventricular portion⁸ is thought to be the mechanism. In the selection of perioperative variables in our study, two assumptions were made regarding the mode of injury to the conduction system. The first is that JET is an iatrogenic consequence of repair that can potentially be avoided, rather than an intrinsic morphologic condition inherent in a specific congenital heart defect. Against this assumption, Bharti and colleagues¹³ reported JET in 3 unoperated patients with concordant connections without a VSD. They did find, however, distinct anatomic abnormalities of the atrioven-

Figure 1. High-power (hematoxylin-phloxine-saffarin stain) histologic specimen of a patient with JET revealing infiltrative hemorrhage in the conduction system.

tricular junction in all 3 cases, with displacement of the atrioventricular node and bundle downward on the atrial septum or toward the left side of the ventricular septal crest. In support of this assumption, autopsy studies of surgical specimens from patients with JET have disclosed streaks of hemorrhage penetrating the atrioventricular bundle and node on the left side of the ventricular crest, in addition to direct damage by sutures within the central fibrous body⁸ (Figure 1). Although it is unknown whether hemorrhage occurs near VSD sutures without causing JET, on the basis of these findings it is postulated that disruption of conduction tissue, either by direct trauma or by penetrating blood and interstitial inflammatory cells, may result in irritable foci leading to JET.⁸

The second assumption revolves around the mechanism of injury. Enhanced automaticity of the bundle of His may occur by direct trauma. With current understanding of the

Figure 2. Postmortem specimen in a patient with tetralogy of Fallot and postoperative JET. Note subendocardial hematoma on the left side of the ventricular crest, opposite to where suturing is performed to achieve VSD closure.

locations and pathways of the conduction system in each type of congenital heart defect,¹⁴ direct trauma by sutures is avoidable in most instances. Indirect mechanisms of trauma may include infiltrative hemorrhage, fibrosis, or stretching of the conduction system. Because multiple approaches are often necessary to achieve repair, an accumulation of potential trauma to the conduction system may occur. Intuition suggests that the accumulation of incisions and pathways for access to attempt repair may have an additive effect on the occurrence of JET. During repair of tetralogy of Fallot, approaching a VSD or RVOTO through the right atrium requires more traction on the heart than does approaching through a ventriculotomy. Likewise, during surgery for AVSD, considerable traction is often applied on the various cardiac structures to achieve transatrial exposure adequate for a satisfactory repair. On the basis of observation from gross pathologic and histologic specimens from patients with JET studied post mortem, we postulate that streaks of blood originating at a distance from the area of resection or excision of right ventricular muscle may penetrate and infiltrate the conduction system (Figures 2 and 3). Resection and excision of muscle, leaving raw surfaces of myocardium, could further increase infiltration of blood cells to

Figure 3. Postmortem specimen in the same patient with tetralogy of Fallot and postoperative JET. Subendocardial hemorrhage is evident at some distance from the area of right ventricular muscle resection.

ward the conduction system relative to that provoked by sharp-blade division of obstructing bundles. We found that resection or excision of muscle bundles within the right ventricular infundibulum was significantly and independently predictive of postoperative JET. We appreciate, nonetheless, that avoidance of resection is not always possible when achieving adequate RVOTO relief, and by no means do we systematically condemn it.

When stretch trauma is evoked, multiple approaches to achieve either VSD closure or RVOTO relief may enhance the probability of injury to the bundle of His and thus postoperative JET. Although relief of RVOTO through the right atrium was a significant predictor of JET when the complete study group was considered, this trend did not attain statistical significance in the specific analysis of patients after repair of tetralogy of Fallot. This mechanism may thus be somewhat overstated, as is supported by the findings of Karl and coworkers,¹⁵ who favor a two-incision transatrial plus transpulmonary approach for repair of tetralogy of Fallot. In a study of 366 nonneonatal patients with

tetralogy of Fallot, transatrial and transpulmonary repair was feasible in all cases, with a low mortality rate and actuarial freedom from reoperation of 95% at both 5 and 10 years. Unfortunately, these authors did not mention the incidence of JET or any other postoperative arrhythmia. With a minimal right ventriculotomy in 36 patients, Kawashima and associates¹⁶ reported improved right ventricular function and a statistically nonsignificant decrease in the occurrence of right bundle branch block; again, however, they did not refer to their incidence of JET. Malm and colleagues¹⁷ reported successful repair of AVSD with RVOTO in 20 patients with a transatrial-transpulmonary technique and resection of muscle bundles mainly through the right atrioventricular valve, without operative deaths or postoperative JET.

Interestingly, younger age did not correlate with increased JET, as has been reported by other groups.^{7,18} It is conceivable that smaller patient size is associated with more delicate cardiac structures and with enhanced difficulty in surgical access, and thus a greater need for traction, notably to achieve exposure through a transatrial approach.⁷ Although transatrial RVOTO relief was a risk factor for JET in our study, no association with younger age was found.

When considering our cohort of patients with tetralogy of Fallot, a relatively high percentage of repairs were performed with a transannular or right ventricular patch (71.9% and 82.5%, respectively). The fact that neither of these variables was statistically associated with postoperative JET may support and underscore the finding that trauma to the bundle of His, rather than to the right ventricular muscular mass, is the causal factor leading to JET. Although large ventriculotomies and transannular patches lead to diminished cardiac output and restrictive right ventricular physiology in the immediate postoperative setting, these did not necessarily coincide with JET. The occurrence of right bundle branch block, late ventricular arrhythmias, and sudden death, nonetheless, is well documented after repair of tetralogy of Fallot producing extensive ventricular scars⁹⁻¹²; however, that is beyond the scope of this study.

Curiously, despite minor variations in technique, approach, and cooling temperatures, no difference in the incidence of JET as a postoperative complication was noted among the three surgeons.

We recorded the lowest mean temperature during CPB and found a higher temperature to be an independent and significant risk factor for postoperative JET. The wide variability of intervals spent at each temperature, along with the differences in rate of cooling and rewarming, make it difficult to interpret this result. Furthermore, because hypothermia is known to reduce the automaticity of cardiac pacemaker cells,^{3,6} it is conceivable that lower CPB temperatures, incomplete rewarming, or prophylactic controlled hypothermia in the intensive care unit may reduce the

potential for JET. To test this hypothesis would require a controlled model with specific temperature targets, warming and cooling rates, and exposure times at each temperature, and this again is beyond the limits of this retrospective analysis.

Limitations of the Study

The conduction system is intimately related to the borders of inlet and perimembranous VSDs, but this is not always the case for subarterial and muscular VSDs. Our groups of patients included all types of VSD. Although the bundle may be susceptible to direct suture trauma during repair of the former two types of VSD, infiltrative hemorrhage or stretching of the conduction system may also occur during closure of the latter two types of defect. Although JET has been reported after the repair of virtually every congenital defect, even extracardiac ones,⁷ we did not include other simpler intracardiac congenital lesions or extracardiac lesions in our study. In fact, Walsh and coworkers⁷ reported a negative association of JET after operations to close defects within the oval fossa, coarctation or aortic arch repair, pacemaker insertion, and systemic-to-pulmonary arterial shunts. It may be argued that many complex lesions include a VSD among a long list of other anomalies, but these were not analyzed in this study in an attempt to minimize the potential effects of a wide morphologic spectrum.

With respect to the hypothesis implicating infiltrative hemorrhage, postmortem specimens were not available from any of the 10 patients who died during the study. Thus, histologic confirmation of either direct suture damage or infiltrative hemorrhage in the bundle is lacking among the 3 patients with postoperative JET who died after repair of tetralogy of Fallot. Although none of the 7 other patients who died (1 after tetralogy of Fallot repair, 4 after repair of common truncus arteriosus, and 2 after AVSD closure) had JET, histologic data are similarly unavailable to confirm the absence of such lesions in patients without JET.

Circulatory support is often required in the postoperative setting of prolonged or complex repairs of congenital heart defects, and many patients return from the operating room routinely receiving low dose inotropes. Treatment with low doses of such inotropes as dopamine or dobutamine is not generally incriminated in the genesis of JET, although these drugs may aggravate or sustain the vicious cycle of adrenergic stimulation. Initial collection of data in our study included a variable entitled "inotropes." This variable was arbitrarily defined as any dose of enoximone, adrenaline and noradrenaline, or a dose of dopamine or dobutamine in excess of 15 $\mu\text{g}/(\text{kg} \cdot \text{min})$.

This degree of support was necessary in 48 patients after repair of tetralogy of Fallot (42.1%), in 5 patients after repair of common truncus arteriosus (50%), in 12 patients after closure of a VSD (7.5%), and in 24 patients after repair

of an AVSD (41.4%). In an initial analysis including this variable among all the others previously mentioned, stepwise logistic regression revealed that "high-dose" inotropic support ($P < .0001$) and resection of muscle bundles ($P < .0001$) significantly and independently predicted postoperative JET. Because of the retrospective nature of the study, however, determination of the time course for inotropic support was imprecise. In some instances it was unclear whether inotropic treatment preceded JET, thereby potentially incriminating it in the genesis of the arrhythmia, or whether exogenous catecholamine therapy was required to improve an unstable hemodynamic situation after JET developed. Accordingly, it would have been incorrect to state that high-dose inotropic treatment predicted the occurrence of postoperative JET, and this variable was excluded from the model. A prospective study analyzing all treatment and support variables may better help to elucidate the role of inotropic support in perioperative JET.

Metabolic acidosis, hypovolemia, and electrolyte imbalance are commonly encountered postoperatively in patients with JET and constitute an insult added to injury. Accordingly, these conditions were aggressively corrected during the perioperative period. Although it would have been desirable to include them as variables in the statistical model, the duration and degree of imbalance before correction were imprecisely known, reason for their omission in this retrospective study.

Conclusion

Our findings suggest a causal relationship between surgical trauma to the bundle of His and postoperative JET. This trauma may result from direct suture damage, but is much more likely to arise indirectly from stretch traction or infiltrative hemorrhagic streaks in the conduction system. Some morphologic variables leading to postoperative JET are perhaps inherent in the various disease types and cannot be altered. In contrast, surgical approaches or incisions may be influenced through modification of operative technique or CPB strategy, thus allowing prevention or at least lessening of the incidence of this morbid arrhythmia. When feasible, more gentle surgical technique and judicious selection of approaches to achieve repair may reduce the incidence of iatrogenic factors. Sharp division of muscle bundles may be preferable to extensive muscle resection and could diminish the potential for infiltration of hemorrhage into the conduction system.

We thank Mr Thierry Murier of the University of Neuchatel, Switzerland, for his statistical analysis.

References

1. Villain E, Vetter V, Garcia JM, Herre J, Cifarelli A, Garson A Jr. Evolving concepts in the management of congenital junctional ectopic tachycardia. *Circulation*. 1990;81:1544-9.

2. Gillette PC. Diagnosis and management of postoperative junctional ectopic tachycardia. *Am Heart J*. 1989;118:192-4.
3. Balaji S, Sullivan I, Deanfield J, James I. Moderate hypothermia in the management of resistant automatic tachycardias in children. *Br Heart J*. 1991;66:221-4.
4. Till JA, Rowland E. Atrial pacing as an adjunct to the management of post-surgical His bundle tachycardia. *Br Heart J*. 1991;66:225-9.
5. Raja P, Hawker RE, Chaikitpinyo A, Cooper SG, Lau KC, Nunn GR, et al. Amiodarone management of junctional ectopic tachycardia after cardiac surgery in children. *Br Heart J*. 1994;72:261-5.
6. Pfammatter JP, Paul T, Ziemer G, Kallfelz HC. Successful management of junctional tachycardia by hypothermia after cardiac operations in infants. *Ann Thorac Surg*. 1995;60:556-60.
7. Walsh EP, Saul P, Sholler GF, Friedman JK, Jonas RA, Mayer JE. Evaluation of a staged treatment protocol for rapid automatic junctional ectopic tachycardia after operation for congenital heart disease. *J Am Coll Cardiol*. 1997;29:1046-53.
8. Till JA, Ho SY, Rowland E. Histopathological findings in three children with His bundle tachycardia occurring subsequent to cardiac surgery. *Eur Heart J*. 1992;13:709-12.
9. Gatzoulis MA, Till JA, Somerville J, Redington AN. Mechano-electrical interaction in tetralogy of Fallot: QRS prolongation relates to right ventricular size and predicts malignant ventricular arrhythmias and sudden death. *Circulation*. 1995;92:231-7.
10. Meijboom F, Szatmari A, Deckers JW, Utens EM, Roelandt JR, Bos E, et al. Cardiac status and health-related quality of life in the long term after surgical repair of tetralogy of Fallot in infancy and childhood. *J Thorac Cardiovasc Surg*. 1995;110:883-91.
11. Friedli B. Electrophysiological follow-up of tetralogy of Fallot. *Pediatr Cardiol*. 1999;20:326-30.
12. Oechslin EW, Harrison DA, Harris L, Downar E, Webb GD, Siu SS. Reoperation in adults with repair of tetralogy of Fallot: indications and outcomes. *J Thorac Cardiovasc Surg*. 1999;118:245-51.
13. Bharati S, Moskowitz WB, Scheinman M, Estes NA 3rd, Lev M. Junctional tachycardias: anatomic substrate and its significance in ablative procedures. *J Am Coll Cardiol*. 1991;18:179-86.
14. Anderson RH, Ho SY. The morphologic substrate for pediatric arrhythmias. *Cardiol Young*. 1991;1:159-76.
15. Karl TR, Sano S, Pornviliwan S, Mee RB. Tetralogy of Fallot: favorable outcome of non-neonatal transatrial, transpulmonary repair. *Ann Thorac Surg*. 1992;54:903-7.
16. Kawashima Y, Kitamura S, Nakano S, Yagihara T. Corrective surgery for tetralogy of Fallot without or with minimal right ventriculotomy and with repair of the pulmonary valve. *Circulation*. 1981;64 Suppl 2:147-53.
17. Malm T, Karl TR, Mee RB. Transatrial-transpulmonary repair of atrioventricular septal defect with right ventricular outflow tract obstruction. *J Card Surg*. 1993;8:622-7.
18. Parry AJ, McElhinney DB, Kung GC, Reddy VM, Brook MM, Hanley FL. Elective primary repair of acyanotic tetralogy of Fallot repair in early infancy: overall outcome and impact on the pulmonary valve. *J Am Coll Cardiol*. 2000;36:2279-83.