

# Ventricular arrhythmia and tetralogy of Fallot repair with transannular patch

## *Fallot tetralojisinde transannuler yama ile tamir ve ventriküler aritmi*

Süleyman Özkan, Tankut Akay, Bahadır Gültekin, Birgül Varan\*,  
Kurşat Tokel\*, Sait Aşlamacı

Departments of Cardiovascular Surgery and \*Pediatric Cardiology, Başkent University Ankara Hospital, , Ankara, Turkey

### ABSTRACT

**Objective:** Life-threatening arrhythmias and sudden death remain to be serious late complications after correction of tetralogy of Fallot. The aim of this study was to detect ventricular arrhythmia incidence and to find out the relationship between ventricular arrhythmia and the transannular and infundibular patch repair techniques to correct tetralogy of Fallot. That is still unpredictable exactly.

**Methods:** Thirty-nine patients with mean age of 12.1±3.1 years were studied prospectively for 7.1±2.1 years after operation. They were all investigated with electrocardiography, echocardiography, treadmill and Holter monitorization. Right ventricular functions, exercise capacity and arrhythmia patterns were assessed. Lown criteria were used for grading the arrhythmia patterns. QRS duration, QT dispersion and QT dispersion indexes were calculated.

**Results:** Follow up time was 5 to 13 years in both groups. All QT dispersion times and indexes were within normal ranges and there were no differences between two groups. Holter and treadmill studies did not reveal during any ventricular arrhythmia risk in the study and control groups.

**Conclusion:** Seven years of follow-up after correction of tetralogy of Fallot revealed that transannular patch reconstruction is not a cause of tendency for ventricular arrhythmia according to Lown criteria, QT dispersion, QT dispersion indexes and QRS duration do support the results of previous studies. (*Anadolu Kardiyol Derg 2005; 5: 297-301*)

**Key words:** Tetralogy of Fallot, transannular patch, ventricular arrhythmia

### ÖZET

**Amaç:** Hayatı tehdit edici ventriküler aritmiler ve ani ölüm halen Fallot tetralojisi tamirlerinin ciddi geç komplikasyonları olmaya devam etmektedir. Bu çalışmanın amacı ventriküler aritmi insidansını araştırmak ve halen kesin öngörülemeyen, ventriküler aritmi ile Fallot tetralojisi tamirlerinde transannuler ve infundibuler yama tekniklerinin ilişkisini belirlemektir.

**Yöntemler:** Ortalama yaşları 12.1±3.1 yıl olan 39 hasta prospektif olarak 7.1±2.1 yıl araştırıldı. Tüm hastalar elektrokardiyografi, ekokardiyografi, tredmil ve Holter monitorizasyonla takip edildiler. Sağ ventrikül fonksiyonları, egzersiz kapasiteleri ve aritmi paternleri değerlendirildi. Aritmi paternlerinin sınıflandırılması için Lown kriterleri kullanıldı. QRS süreleri, QT dispersiyonu ve QT dispersiyon indeksleri hesaplandı.

**Bulgular:** Her iki grupta takip süreleri 5 ve 13 yıl idi. Tüm QT dispersiyon zamanları ve indeksleri her iki grupta da normal sınırlardaydı ve farklılık göstermiyordu. Çalışma ve kontrol gruplarında, Holter ve tredmil çalışmalarında hiçbir aritmi riski ortaya çıkmadı.

**Sonuç:** Fallot tetralojisi tamirlerinin 7 yıllık takipleri, transannuler yama tekniğinin, önceki çalışmalarının tersine; Lown kriterlerine, QT dispersiyonu, QT dispersiyon indeksi ve QRS sürelerine göre, ventriküler aritmi eğilimi yaratmadığını göstermiştir. (*Anadolu Kardiyol Derg 2005; 5: 297-301*)

**Anahtar kelimeler:** Fallot tetralojisi, transannuler yama, ventriküler aritmi

### Introduction

Surgical repair of tetralogy of Fallot (TOF) was first created by Lillehei (1) in 1955. Mortality in the first year of life was 50% in those days but nowadays, survival for long-term is about 85% (2). Long-term survival after TOF repair resulted in to suspect the existence of an unexpected pathology causing sudden death.

Elevated right ventricular pressure was underlined as a risk

factor for sudden death (3). Trifascicular block, premature ventricular contractions (4) and ventricular arrhythmias (5) were suggested as a relation with sudden death. Silka (6) found an incidence of sudden death in repaired TOF patients to be 1.5 / 1000 patient-years. This ratio obviously has increased 20 years after operation, which was confirmed by others (7).

Abnormal hemodynamic conditions underlie these arrhythmias; tricuspid insufficiency, pulmonary regurgitation were fo-

**Address for Correspondence:** Dr. Süleyman Özkan, Başkent Üniversitesi Ankara Hastanesi, Kalp ve Damar Cerrahisi, Mareşal Fevzi Çakmak Cad. 10.sok. No:45 06490 Bahçelievler, Ankara, Türkiye, Tel: 0312 2126868/1372,1373. Fax: 0312 2237333 email: sozkan11@hotmail.com

**Note:** This work was presented as oral presentation at the VII. National Congress of the Turkish Society of Cardiovascular Surgery in Antalya, Turkey, October 23-27, 2002.

und in the majority of patients with arrhythmias (6,8). Evidence of the electrophysiological mechanism of ventricular tachycardia after TOF repair is macro-reentry mostly located in the right ventricular outflow tract, the septum or in the area of the ventriculotomy; myocardial specimens of these areas show extensive fibrosis, degeneration and fatty replacement of myocardium supporting this mechanism (9).

Certain electrocardiographic markers namely QRS prolongation (10-12) and marked QT dispersion (13) correlate with right ventricular dilatation and are predictive of sustained monomorphic ventricular tachycardia and sudden death.

While the risk of surgery has fallen, use of transannular patch repair technique for right ventricular outflow tract reconstruction has increased up to 90% and over (14). One of the major concerns with patch reconstruction is the its deleterious effects on right ventricular physiology and arrhythmia. Long-term consequences of repair of TOF including sudden death are mostly influenced by the technique of correction. However, the relationship between ventricular arrhythmias and sudden death in patients repaired with transannular patch is not certain, the dilatation and dysfunction of right ventricle accompanying with arrhythmias have the value to investigate the clinical and etiological specifications.

The aim of this study is to define the tendency of ventricular arrhythmia pattern in long term after repair of TOF according to pulmonary outflow reconstruction technique.

## Methods

The database of the Cardiovascular Surgery at Başkent University Hospital was searched for all 123 TOF patients operated for total correction before 1996. Thirty-nine of them were randomly selected patients to study prospectively. Their ages were 12.1+3.1 years and mean operation age was 5.2+3.2 years. The selected patients were not different from all operated patients according to their demographic specifications. They were also first available and contacted ones. Mean follow up was 7.1+2.1 years (between 5-13 years) (Table 1). There were 26 patients in transan-

nular patch reconstruction group (group 1) and 13 patients in infundibular patch or primary reconstruction group (group 2).

Background clinical information (Table 1) was obtained from the patients' cardiac catheterization data and surgical discharge summaries and details on surgical procedures from surgeons' notes. Patients of both groups were investigated by electrocardiography (ECG), transthoracic echocardiography, Holter monitorization and treadmill. Echocardiography, ECG, and treadmill test were done on the first day of study. Holter monitorization was begun after treadmill test, with the evaluation of recordings on the next day. Right and left ventricular outflow tracts gradients, biventricular dimensions, pulmonary insufficiency and volumes were calculated with echocardiography by an independent cardiologist. Resting 12-lead electrocardiographies were recorded and compared with preoperative recordings. Existence of sinus rhythm, block, supraventricular and ventricular arrhythmias, QRS complex durations, QT dispersion and QT dispersion indexes were examined. QRS duration was defined as the distance between the first and last deflection from isoelectric line in QRS complex. Also, the longest QRS duration in any lead was recorded. QT dispersion was calculated as the ratio of the difference between the longest and the shortest QT time to mean QT time. QT dispersion index was calculated according to body area so as to eliminate the differences of patients' ages. Holter recordings were examined for ventricular arrhythmias' frequencies, complexities and degrees according to modified Lown criteria (15). Furthermore, heart rate, supraventricular extrasystoles, supraventricular and ventricular tachycardias, ventricular extrasystoles and frequencies were recorded. All patients were treated to treadmill according to modified Bruce (16) test for children (Quinton 5000R). Existence of arrhythmia pattern, maximal heart rate, blood pressure and METS values (70 kg, 40 years old, male person's respiratory oxygen uptake for a minute, 1 MET equals to 3.5 ml/min/kg) were recorded. Reasons for termination of tests were also recorded. All data were analysed and compared for both groups separately. Cardiac catheterization and angiocardiology were not performed. Statistical analyses were performed with SPSS v.9.0 and

**Table 1. Clinical, echocardiographic and treadmill test variables of study patients**

	All patients	Group 1	Group 2
Patient age (years)	12.4+3.1	12.9+3.4	13.2+4.7
Operation age (years)	5.2+3.2	5.2+2.7	5.3+3.1
Follow-up duration (years)	7.1+2.1	8.1+3.2	7.8+3.1
RV mass (g)	96+41	100+41	86+43
RV End Systolic Diameter (mm)	21+5.7	21+5	19+7
RV End Diastolic Diameter (mm)	32.0+6.2	33.0+5.3	28+7
LV mass (g)	129+54	127+49	133+65
LV End Systolic Diameter (mm)	25.0+7.3	23+5	28+10
LV End Diastolic Diameter (mm)	36+6	36+6	34+6
LV Ejection Fraction (%)	65+12	67+11	60+13
LV Fractional Shortening (%)	35+9	37+9	32+9
Residual Shunt, n	-	-	-
Pulmonary Insufficiency, n	4	4	0
METS	10.5+3.0	10.7+2.5	10.1+3.9

LV – left ventricle, METs- metabolic equivalent, RV – right ventricle

data are presented as mean (+ 2SD) or median with range. Comparisons were made using Mann-Whitney U and  $\chi^2$  tests. The null hypothesis was rejected when  $p < 0.05$ .

## Results

There were no differences between two groups according to age, operation age and follow up time. Ages of operation time in group 1 varied between 2 and 14 years and in group 2- between 2 and 10 years. Follow-up time ranged within 5 to 13 years in both groups. All patients were in NYHA class 1 before and after operation. According to ECG recordings all patients were in sinus rhythm preoperatively but 84% of them have right bundle branch block postoperatively in both groups. So, there was no any difference between two groups according to right bundle branch block ( $p=0.887$ ). QT dispersion times and indexes were in normal ranges in both groups and there were no differences in both groups ( $p=0.448$ ), also QT and QTc times are listed ( $p>0.05$ ), (Table 2).

There was only one patient in Group 1 who had QRS duration longer than 180 ms. This patient had also severe tricuspid insufficiency but had not severe right ventricular dysfunction yet. Furthermore dysrhythmia did exist Holter study for this patient. Only mild pulmonary insufficiency was detected. Biventricular deficiency and severe valvular insufficiency were not detected in echocardiographic studies (Table 1) though isolated right ventricular dysfunctions with pulmonary regurgitations were detected in Group 1. Third degree pulmonary insufficiency was revealed only in four patients and all were in Group 1. None of the patients showed arrhythmia even in patients with pulmonary regurgitations and isolated right ventricular diastolic dysfunctions.

Holter results were evaluated according to the existence of ventricular extrasystoles, tachycardias and couples of extras to determine the Lown grades. Lown grade calculations revealed no difference between two groups ( $p=0.09$ ). Lown grade 0 was in majority in Group 1 and there were no patients in grade 3 in Group 2 (Table 3). During treadmill no arrhythmia signs were detected in all patients. None of them stopped the test because of any complaint. Mean METS degrees were over 10 in both groups (Table 1).

**Table 2. QRS duration and QT dispersion in study patients**

	Group 1			Group 2			P
	min	max	mean	min	max	mean	
QRS duration (ms)	80	173	135+24	78	189	129+27	0.640
QT dispersion (ms)	0.04	0.20	0.11+0.04	0.04	0.28	0.09+0.06	0.448
QT dispersion index	0.11	0.47	0.27+0.09	0.10	0.55	0.22+0.10	0.448
QT (ms)	339	536	417+51	326	501	400+52	0.363
QTc (ms)	405	517	468+28	406	513	464+38	0.965

**Table 3. Ventricular ectopy Lown grades according to Holter monitoring in study patients ( $p=0.09$ )**

LOWN GRADE	Group 1		Group 2	
	Patients (n)	%	Patients (n)	%
0	7	26.9	8	61.5
1	17	65.3	4	30.7
2	2	7.7	1	7.8

## Discussion

Early results of TOF repair are nearly perfect (17,18). While the risk of operative mortality decreases, transannular patch reconstruction increased up to 90% in some series (14). Patch reconstruction has the major deleterious effects on right ventricular physiology and it can cause arrhythmia. Some patients died suddenly many years after successful repair. This is mostly referred as a consequences right ventricular dilatation and arrhythmia. A significant correlation between right ventricular size and QRS duration was detected (10). Conversely, QRS width was not particularly prolonged in cases of significant residual right ventricular outflow obstruction alone, suggesting that the QRS prolongation reflects right ventricular dilatation rather than an increase in right ventricular mass due to hypertrophy; dilated ventricle in turn creates the conditions required for reentry. In our study, QRS prolongation was not predominant reflecting the probability of low incidence of arrhythmia. Reconstruction with transannular patch in TOF patients especially with residual defects and regurgitation was shown as a reason of sustained ventricular tachyarrhythmias (19). In our patients residual defects and regurgitations were in expected normal ranges. This also can explain why arrhythmia was not seen in our patients. Right ventricular hypertrophy, right bundle branch block (RBBB), right ventricular overload patterns can be detected in surface electrocardiography. In our study, RBBB was seen in 84% in all groups of patients but this was resulting from the technique of ventricular septal defect closure and this is an acceptable event. Especially, QRS complex time prolongation and QT interval dispersion are mentioned as a risk factor for malignant ventricular dysrhythmias (13,20). QRS duration more than 180 ms is a sensitive predictor of sudden death (10). However, QRS duration may differ with surgical technique and resections, therefore it should be followed for long terms. Kremers et al. (21) showed that there is good evidence from electrophysiological studies that sustained ventricular tachycardia results from reentry, which requires areas of slow conduction. Furthermore, fragmented electrograms indicative of localized areas of slowed conduction have been recorded from both inflow, ventricular septal defect (22), outflow, outflow patch (23), throughout the

right ventricle (24) and also from ventriculotomy scar (25). These are all implicated as areas of potential reentry circuit areas. Rahman et al. (26) suggested that the low incidence of arrhythmia in patients with a QRS duration more than 180 ms may be a reflection of the shorter follow-up period which resembles to our result also. Therefore QT dispersion has a superior value to detect arrhythmia probability. Besides, there are some reports about sudden death and poor prognosis, which are not relevant

with ventricular arrhythmias that was detected in postoperative Holter recordings (27). This also reinforces the results of our study.

Treatment spectrum extends from only treatment of symptomatic patients to surgical treatment of asymptomatic patients who has ventricular premature complexes (28). Oechslin (29) found that long-term complications of right ventricular outflow tract are the most prevalent reasons for reoperation and were often associated with sustained ventricular tachycardia. None of the patients in our study was reoperated for arrhythmia. The differences between these studies are the populations of the patients and the follow up. Pulmonary regurgitation is reported to be well tolerated through childhood and adolescence in the absence of important additional lesions (30,31). Another study observed a significantly lower 25-year survival in patients with a transannular outflow patch compared with that of patients without a patch (32). The ability of a patient to tolerate pulmonary valve regurgitation varies and depends on the protective effect of right ventricle's compliance (33,34). Residual lesions accelerates the arrhythmia potential. In our study major residual defects were not detected predominantly and mean follow-up for all patients was 7 years that we can consider as mid- to long term. This may be reason of why we cannot claim that there is a potential risk or QRS and QT calculations are enough predictors for fatal arrhythmias after correction of TOF on the contrary of the literature. There were no any changes on ECGs that could be considered as precursors of fatal arrhythmias. There were no patients with Lown grade 3 arrhythmia. Therefore we did not plan to perform electrophysiologic study yet though it seems a lack of our study. Exceptionally there was one patient who had QRS duration longer than 180 ms and this is the only case we have to pay attention on it. QRS and QT calculations also show that right ventricular enlargement was not dominant resulting from any residual pathology. Pulmonary insufficiency was not predominant in our patients and this can explain low Lown grades, also patients with pulmonary insufficiency have not arrhythmogenic patterns yet. So, outflow tract reconstruction technique that might result in residual pathologies is not a major risk factor for tendency of fatal ventricular arrhythmias in our patients. However, limitation of this study was the limited population of the groups, follow-up time and lack of electrophysiological study. While increasing the follow-up time, new patients will be included in the study and it will reinforce the accuracy of the results. Consequently, low incidence of arrhythmia in our patients might be due to appropriate and balanced corrective surgery, low and acceptable residual pathologies, shortage of ventricular dysfunction and not long enough follow-up time.

## Conclusion

The major target must be to achieve a definite repair that will not effect the life comfort in long-term. Researches are condensed on right ventricular outflow tract reconstruction technique and its influences on residual pathologies and cardiac functions in long-term period rather than early-term. In our study, seven years of follow-up after correction of tetralogy of Fallot revealed that transannular patch reconstruction is not a cause of tendency for ventricular arrhythmia according to Lown criteria, QT dispersion, QT dispersion indexes and QRS duration on the contrary to the results of previous studies.

## References

1. Lillehei CW. Direct vision intracardial surgical correlation of the Tetralogy of Fallot, pentalogy of Fallot and pulmonary atresia defects: report of the first ten cases. *Ann Surg* 1955; 142: 418.
2. Nollert G, Fischlein T, Bouterwek S. Long-term survival in patients with repair of tetralogy of Fallot: 36-year follow-up of 490 survivors of the first year after surgical repair. *J Am Coll Cardiol* 1997; 30: 1374-83.
3. Garson A, Nihill MR, McNamara DG, Cooley DA. Status of the adult and adolescent after repair of tetralogy of Fallot. *Circulation* 1979; 59: 1232-40.
4. Quattlebaum TG, Varghese J, Neill CA, Donahoo JS. Sudden death among postoperative patients with tetralogy of Fallot. *Circulation* 1976; 54: 289-93.
5. James FW, Kaplan S, Chou TC. Unexpected cardiac arrest in patients after surgical correction of tetralogy of Fallot. *Circulation* 1975; 52: 691-5.
6. Silka MJ, Hardy BG, Menashe VD, Morris CD. A population-based prospective evaluation of risk of sudden death after operation for common congenital heart defects. *J Am Coll Cardiol* 1998; 32: 245-51.
7. Kavey RE, Thomas FD, Byrum CJ, et al. Ventricular arrhythmias and biventricular dysfunction after repair of tetralogy of Fallot. *J Am Coll Cardiol* 1984; 4: 126-31.
8. Roos-Hesselink J, Perloth MG, McGhie J, Spitaels S. Atrial arrhythmias in adults after repair of tetralogy of Fallot. *Circulation* 1995; 91: 2214-9.
9. Isaki T, Tsubota M, Watanebe Y, et al. Surgical treatment of ventricular tachycardia after surgical repair of tetralogy of Fallot. *Circulation* 1994; 90: 164-71.
10. Gatzoulis MA, Till JA, Somerville J, et al. 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.
11. Balaji S, Lau YR, Case CL, et al. QRS prolongation is associated with inducible ventricular tachycardia after repair of tetralogy of Fallot. *Am J Cardiol* 1997; 80: 160-3.
12. Berul CI, Hill SL, Geggel RL. Electrocardiographic markers of late sudden death risk in postoperative tetralogy of Fallot children. *J Cardiovasc Electrophysiol* 1997; 8: 1349-56.
13. Gatzoulis MA, Till JA, Redington AN. Depolarization-repolarization inhomogeneity after repair of tetralogy of Fallot: the substrate for malignant ventricular tachycardia? *Circulation* 1997; 95: 401-4.
14. Pacifico AD, Barger IM, Kirklín JW. Primary correction of tetralogy of Fallot in children less than four years of age. *Circulation* 1973; 48: 85-91.
15. Ryan M, Lown B, Horn H. Comparison of ventricular ectopic activity during the 24 hours monitoring and exercise testing in patients with coronary heart disease. *N Engl J Med*. 1975; 292: 224-9.
16. Bruce RA, Kasumi F, Hosmer D. Maximal oxygen uptake and non-metabolic assessment of functional aerobic impairment in cardiovascular disease. *Am Heart J* 1973; 85: 546-62.
17. Murphy JG, Gersh BJ, Muir DD, et al. Long-term outcome in patients undergoing surgical repair of tetralogy of Fallot. *N Eng J Med* 1993; 329: 593-9.
18. Karl TR, Sano S, Parnviliwan S, Mee RBB. Tetralogy of Fallot: favorable outcome of non-neonatal transatrial, transpulmonary repair. *Ann Thorac Surg* 1992; 54: 903-7.
19. Gatzoulis M, Seshadri B, Webber S. Risk factors for arrhythmia and sudden death late after repair of tetralogy of Fallot: a multicentre study. *The Lancet* 2000; 356: 975-81.
20. Kirklín JK, Kirklín JW, Blackstone EH, Milano A, Pacifico AD. Effect of transannular patching on outcome after repair of tetralogy of Fallot. *Ann Thorac Surg* 1989; 48: 783-91.
21. Kremers MS, Wells PJ, Black WH. Entrainment of ventricular tachycardia in postoperative tetralogy of Fallot. *Pacing Clin Electrophysiol* 1988; 11: 1310-4.

22. Kugler JD, Pinsky WW, Cheatham JP. Sustained ventricular tachycardia after repair of tetralogy of Fallot: new electrophysiologic findings. *Am J Cardiol* 1983; 51: 1137-43.
23. Donwar E, Harris L, Kimber S, Mickleborough L. Ventricular tachycardia after surgical repair of tetralogy of Fallot: results of intraoperative mapping studies. *J Am Coll Cardiol* 1992; 20: 648-55.
24. Deanfield JE, McKenna W, Rowland E. Local abnormalities of right ventricular depolarization after repair of tetralogy of Fallot: a basis for ventricular arrhythmia. *Am J Cardiol* 1985; 55: 522-5.
25. Horowitz LN, Vetter VL, Harken AH, Josephson ME. Electrophysiologic characteristics of sustained ventricular tachycardia occurring after repair of tetralogy of Fallot. *Am J Cardiol* 1980; 46: 446-52.
26. Abd El Rahman MY, Abdul Khalig H, Vogel M, Alexi M, Gutberlet M. Relation between right ventricular enlargement, QRS duration and right ventricular function in patients with tetralogy of Fallot and pulmonary regurgitation after surgical repair. *Heart* 2000; 84: 416-20.
27. Cullen S, Celermajer DS, Franklin RC. Prognostic significance of ventricular arrhythmia after repair of tetralogy of Fallot: a 12-year prospective study. *J Am Coll Cardiol* 1994; 23: 1151-5.
28. Garson A, Randall DG, Gillette PC, et al. Prevention of sudden death after repair of tetralogy of Fallot: treatment of ventricular arrhythmias. *J Am Coll Cardiol* 1985; 6: 221-7.
29. Erwin NO, David AH, Louise H. Reoperation in adults with repair of tetralogy of Fallot: Indications and outcomes. *J Thorac Cardiovasc Surg* 1999; 118: 245-51.
30. Lillehei CW, Varco RL, Cohen M, Warden HE. The first open heart corrections of tetralogy of Fallot: a 26-31-year follow up of 106 patients. *Ann Surg* 1986; 204: 490-502.
31. Ilbawi MN, Idriss FS, DeLeon SY, Muster AJ. Factors that exaggerate the deleterious effects of pulmonary insufficiency on the right ventricle after tetralogy repair: surgical implications. *J Thorac Cardiovasc Surg* 1987; 93: 36-44.
32. Klinner W, Reichart B, Pfaller M, Hatz R. Late results after correction of tetralogy of Fallot necessitating outflow reconstruction: comparison with results after correction without outflow tract patch. *Thorac Cardiovasc Surg* 1984; 32: 244-7.
33. Gatzoulis MA, Clark AL, Cullen S, Newman CG. Right ventricular diastolic function 15 to 35 years after repair of tetralogy of Fallot. *Circulation* 1995; 91: 1775-81.
34. Norgard G, Gatzoulis MA, Morales F, Lincoln C, Shore DF. Relationship between type of outflow tract repair and postoperative right ventricular diastolic physiology in tetralogy of Fallot: implications for long-term outcome. *Circulation* 1996; 94: 3276-80.