# Ventricular arrhythmia and tetralogy of Fallot repair with transannular patch

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

Dear Editor,

I have read the manuscript "Ventricular arrhythmia and tetralogy of Fallot repair with transannular patch - Original Investigation" published in the recent issue of the Anadolu Kardiyoloji Dergisi (1). I congratulate the authors for choosing such an important topic to study. However; I think there are some problems with the manuscript and I would like to criticize it:

1. In the abstract section the sentence "Thirty-nine patients with mean age of 12.1+3.1 years were studied prospectively for 7.1+2.1 years after operation" means that the patients have been studied prospectively for 7.1+2.1 years after operation. However, this statement is not true; in fact the patients were seen after 7.1+2.1 years of operation.

2. While comparing the groups, gender, body surface area, detailed echocardiographic findings (Pulmonary stenosis, right ventricular systolic pressure and right ventricular filling patterns) and the drugs that the patients taking (especially antiarrhythmics) are not given.

3. Ventricular arrhythmias occur with an incidence of 0.5-6% in patients who have undergone complete repair of tetralogy of Fallot (TOF) (2). In these patients delayed conduction, and increased inhomogeneity of repolarization provide the basis for reentry tachycardias, and sudden death. Re-entry circuits may include a ventricular septal defect patch, right ventricular outflow patches, and ventricular scars. Haemodynamic problems, such as pulmonary stenosis or regurgitation and decreased right ventricular ejection fraction, have also been associated with an increased risk of ventricular arrhythmias (3). Restrictive right ventricular physiology has been related to a reduced risk of ventricular arrhythmias in adult Fallot patients (4). Since right ventricular systolic and diastolic pressures are correlated with the development of arrhythmias in these patients. Therefore, these parameters should be given.

In the present study, isolated right ventricular dysfunctions with pulmonary regurgitations have been detected in Group 1. Therefore the prevalence of ventricular arrhythmias is expected to be higher in Group 1. However; the frequency of arrhythmia and the risk for arrhythmia is the same in the both groups in the study. Lower rate of ventricular arrhythmia could also be secondary to right ventricular diastolic dysfunction in the present study and RV filling patterns should have been studied.

4. QT dispersion (QTD) has been used to predict malignant ventricular arrhythmias after surgical repair of TOF. It has been reported that QTD was already abnormal before surgery, but increased markedly in the post-operative period. Abnormal dispersion of QT and of JT is secondary to natural history of TOF and related to the fibrosis in the right ventricle that develops over time. Patients who had operation in the early years of life had no abnormal QT before the surgery (3, 5). Abnormal dispersion of QT persists after surgery into adulthood (6).

In the present study, although the mean operation age was 5.2, which is a fairly late age for operation of TOF, and no patient were below the age of 1, the risk for and the frequency of arrhythmia is very low even after a mean of 7.1 years after operation. These results are quite interesting and may be secondary to the low sensitivity of the tests used and small study population. As indicated by the authors, if EP study has been performed, the results could have been changed (5). Are the QTD values given in 'ms' or 's'? Even if they are given in 's', these values are within normal limits. These patients have such a severe disease and had such a severe operation, I think these results may indicate the failure of this test.

5. Although QTD has been used as a marker for vulnerability to ventricular arrhythmias and risk for sudden cardiac death, however; clinical utility of QTD have been the subject of intense debate over the past several years (7). Rautaharju (8) has shown that the association between QTD and nondipolar voltage (NDPV) in T waves of the 12-lead ECG is weak, and QTD is unlikely to represent any meaningful myocardial repolarization event in the interval domain and the author recommended to use direct measurement of NDPV as a potential marker of localized dispersion and heterogeneity of ventricular repolarization for evaluation of the risk of adverse cardiac events.

It has been shown that, QT dispersion failed to estimate the global dispersion of ventricular repolarization measured using monophasic action potential mapping technique in swine and patients (9). In a recent review it is indicated that QT dispersion is unsuccessful in predicting the risks of drug-induced Torsades de Pointes (10). Two new parameters are introduced as potential surrogates of transmural dispersion of repolarization (TDR), Tpeak-Tend interval, and the nondipolar components of the T wave (11). There are methodological problems associated with QT dispersion measurements and I think QTD is an unreliable method to evaluate the risk of ventricular arrhythmia and SCD.

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## References

1. Özkan S, Akay T, Gültekin B, Varan B, Tokel K, Aşlamacı S. Ventricular arrhythmia and tetralogy of Fallot repair with transannular patch. Anadolu Kardiyol Derg 2005;5:297-301.
2. Kugler JD. Predicting sudden death in patients who have undergone tetralogy of Fallot repair: is it really as simple as measuring ECG intervals? J Cardiovasc Electrophysiol 1998;9:103-6.
3. Helbing WA, Roest AA W, Niezen RA, Vliegen HW, Hazekamp MG, et al. ECG predictors of ventricular arrhythmias and biventricular size and wall mass in tetralogy of Fallot with pulmonary regurgitation. Heart 2002; 88:515-9.
4. Gatzoulis MA, Till JA, Somerville J, Redington AN. Mechanoelectrical 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.
5. Balkhi RA, Beghetti M, Friedli B. Time course of appearance of markers of arrhythmia in patients with tetralogy of Fallot before and after surgery. Cardiol Young 2004;14:360-6.
6. Therrien J, Siu SC, Harris L, Dore A, Niwa K, Janousek J, et al. Impact of pulmonary valve replacement on arrhythmia propensity late after repair of tetralogy of Fallot. Circulation 2001;103:2489-94.
7. Chen A, Kusumoto FM. QT Dispersion: Much Ado About Something? Chest 2004; 125; 1974-7.
8. Rautaharju PM. Why did QT dispersion die? Card Electrophysiol Rev2002;6:295-301.
9. Liang Y, Kongstad O, Luo J, Liao O, Holm M, Olsson B, et al. QT dispersion failed to estimate the global dispersion of ventricular repolarization measured using monophasic action potential mapping technique in swine and patients. J Electrocardiol 2005; 38:19-27.
10. Shah, RR. Drug-induced QT dispersion: does it predict the risk of torsade de pointes? J Electrocardiol 2005; 38:10-8.
11. Rautaharju PM. A farewell to QT dispersion Are the alternatives any better? JElectrocardiol 2005; 38:7-9.

# Author`s reply

Dear Editor,

The data about control group were presented in Tables 1, 2 and 3 in our manuscript. Therefore it was not also mentioned in the abstract section. This study was planned prospectively. Since we do follow the patients in our clinic precisely, the last control investigations were done in planned manner accordingly with methodology of the study, and it was stated the mean follow-up was 7.1 years. For the drugs, which mentioned in the third question; they could reinforce the accuracy of the study indeed, also the patients could have been divided and studied separately according to the drugs that they have used. However, in that case, the study would be informing the effects of different drugs in different cases rather than different surgical techniques and their effects on arrhythmia, which we wanted to know. Moreover, such a study meaning drugs and their effects on surgical techniques and arrhythmia may be planned. We have declared the data about the right and left ventricular functional capacity on echocardiography rather than all data, which were within normal ranges. Besides, affirming all data that in normal ranges could affect a decrease in attention. On the other hand, I want to mention that the residual pulmonary stenosis was in acceptable ranges and not causing an unfavorable effect on hemodynamics.

It is obvious that the angiographic study must have been assessed to obtain the right ventricular pressures that were asked in fourth question. Also importance of electrophysiologic studies in such cases is well known. Nevertheless, one has to notice this study was based upon non-invasive tests. It is for sure to set up a study based on such invasive tests would be precious with stating the detailed ventricular pressures and calculations, arrhythmia patterns and arrhythmogenic focuses. We encourage such invasively planned studies but also want to remind the possible morbidity and high cost of such studies. Besides, other statements in question four are not in contradiction with our results.

In issue five, the accuracy and sensitivity of QT dispersion (QTD) was questioned. Naturally there are some issues confirming the QTD. We used as reference the following studies of Gatzoulis MA and associates' (1) and Kirklin JK and associates' (2). Some different non-invasive tests can be used in the new studies, however the objections enforced us to evaluate this way. We appreciate the correction of the ms to s of QTD by mistaken. Furthermore, it was evaluated in detail in discussion section why the results were in normal ranges. Patients were not evaluated only by QTD; Lown criteria according to Holter monitorization, effort capacities in treadmill, clinical, electrocardiographic and echocardiographic examinations were also carried out. According to the degree of gross residual pathology and follow-up time we stated the minority of arrhythmia tendency. That's why we claim the seven years follow-up time as mid- to long-term. It's unlucky that the population of the study was limited but it must be kept in mind that the patients of the study were the part of an operated 123 tetralogy of Fallot patient population that were operated before 1996 in our clinic. So, it is not always available to study with such a group of patients.

We would like to thank your attention for our study.

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## References

1. 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.
2. Kirklin JK, Kirklin 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.