Medical Journal of the
Islamic Republic of Iran

Volume 12
Number 1
Spring 1377
May 1998

Original Articles

STUDY OF LATE POTENTIALS IN PEDIATRIC PATIENTS AFTER OPEN HEART SURGERY

ALI AKBAR ZEINALOO, M.D*., J.G. SHAKIBI, M.D., F.A.C.C., AND A. SHAH-MOHAMMADI, M.D.

*From the Department of Pediatric Cardiology, Shahid Rajai Heart Hospital, Tehran, and the *Department of Pediatric Cardiology, Childen's Medical Center, 62 Gharib Ave., 14194-Tehran, Islamic Republic of Iran.*

ABSTRACT

This study was conducted to assess the incidence of abnormalities of ventricular depolarization (late potentials) in children with sinus rhythm after open heart surgery and their relation to spontaneous ventricular tachycardia. Open heart surgery, particularly operations involving ventriculotomy, may predispose patients to the development of ventricular tachycardia (VT) or ventricular fibrillation (VF). Previous studies on children with right bundle branch block (RBBB) have shown that late potentials may be a risk factor for developing VT or VF following open heart surgery. After corrective surgery for congenital heart defects, scars may create fractionation and delay of the electrical signals in the heart muscle, providing a substrate for arrhythmias and sudden cardiac death.

To find normal values of signal averaged EKG (SA-EKG) indices in children and their changes following open heart surgery, we studied 20 normal children and 20 children with congenital heart disease (CHD) following total correction of heart defects without ventriculotomy. All patients were in normal sinus rhythm and did not have RBBB. The mean age was 8.8±2.6 years for the control group and 8.1±2.1 years for the operated patients. SA-EKG was performed for the operated group on the day before and on the 2nd and 4th days after operation. Noise level was less than 1 microvolt. The SA-EKG parameter values were as follows: control group: filtered QRS-duration 40Hz (F. QRS-d), 84.2±9.5 ms; high frequency low amplitude signals (HFLA), 18.9±9.5 ms; root mean square 40 (RMS 40), 181.0±89.4 μv; patients: F.QRS-d, 97.2±19.3*, 116.4±21.2* and 122.2±220.4 before operation, 2nd day post-op and 4th day post-op, respectively; HFLA, 205±22.3, 8.9±7.0*, and 15.4±16.4ms, respectively; RMS 40, 146.4±110.9, 92.1±65.9, and 112.8±60.3, respectively. Values marked with an asterisk are statistically significant.

Except for a significant difference between the QRS duration of normal children and pre-op values of operated patients ($p<0.05$), there was no remarkable difference between the SA-EKG values pre- and postoperatively.

This study in which there was no RBBB, contrary to previous studies, shows that SA-EKG indices are not a predictive value for VT or VF postoperatively. Increased thickness of the ventricular myocardium may be a reason for the increased QRS duration before operation.

Keywords: Late potentials, Ventricular arrhythmias, Operated congenital heart defect in children, Signal-averaged electrocardiography.

MJIRI, Vol. 12, No. 1, 1-4, 1998.

INTRODUCTION

During the past decade, many investigators have discovered low amplitude, high frequency waveforms in the terminal QRS complex, both in laboratory models of myocardial infarction and in patients vulnerable to sustained ventricular tachycardia (VT). These microvolt level signals are called ventricular late potentials.[6] Late sudden death attributed to ventricular tachycardia (VT) and ventricular fibrillation is a rare but catastrophic event in children and adolescents after surgical correction of congenital heart defects using right ventriculotomy.[7]

Late potentials have been recognized as microvolt signals that are fractionated and delayed beyond the QRS duration in border zones surrounding the scar of a previous myocardial infarction or in small areas of diseased myocardium. These areas of delayed activation have been shown to act as the arrhythmogenic substrate for re-entrant ventricular tachycardia. The areas can be measured by direct endocardial catheter (invasive) or by surface signal-averaged microvolt signals (non-invasive).[7,13] In adults, late potentials after myocardial infarction correlate with both induced and spontaneous VT and are one of the important risk factors for sudden death. In children, data on late potentials are sparse.[7] The presence of late potentials is poorly defined in children who have undergone operative repair of congenital heart defects.[1] Late potentials have been shown to have a high predictive value for the later occurrence of serious ventricular arrhythmias, especially in patients with ischemic heart disease.[1] Ventricular arrhythmias and sudden death usually occur late after surgical correction (ventriculotomy) and are associated with older age at the time of surgery and with greater right ventricular (RV) systolic or end-diastolic pressure.[11]

The noninvasive techniques used for the assessment of patients at risk for sudden death due to malignant ventricular arrhythmias include the standard ECG, the treadmill exercise test, and 24 to 48 hour ambulatory Holter monitoring. These tests have poor predictive value, with low sensitivity and specificity.[13] Signal averaging EKG (SA-EKG) is a new, noninvasive, computerized method of analyzing ECG signals that identifies patients at risk for ventricular tachycardia. This technique averages multiple QRS complexes and with high-pass filtered methods minimizes noise and exposes signals of the microvolt level that are normally hidden in the noise.[13] It has been demonstrated that among all the methods of investigation used clinically, an abnormal SA-EKG is the most accurate noninvasive means of predicting the induction of VT in a population at risk.[3] The use of a signal averaging EKG has been limited in the pediatric population. These potentials were successfully recorded in 75% of patients with surgically corrected heart defects. The most interesting application of signal averaging EKG in the pediatric population has been for the detection of ventricular late potentials (LP) in a population at risk of developing ventricular arrhythmias such as corrected tetralogy of Fallot.[3] It have been shown that in postoperative patients, 88% with VT have had LP, versus 7% LP in those without VT.[3] The reported prevalence of abonormal signals has ranged from 60% to 90%, depending on the method of signal processing, the definition of late potentials, and the patient group studied. Conversely, the incidence of abnormal signals in normal subjects is quite low and has been reported from 0% to 7% when essentially the same recording techniques were used.[10] The purpose of this study was to determine the incidence of LP before and after open heart surgery without ventriculotomy and the role of late potentials in the occurrence of VT in children with congenital heart disease.

MATERIALS AND METHODS

The study population consisted of two groups: 20 patients and 20 healthy children. Standard 12-lead ECGs, chest roentgenograms, physical examination and echocardiography were studied with SA-EKG in both groups.

Twenty unselected consecutive patients were studied. Eleven were male and nine female, mean age 8.1 years (range 4 to 11 years). Eleven patients had tetralogy of Fallot, five ventricular septal defect (VSD), two VSD with aortic insufficiency and two atrial septal defect. SA-EKG was performed for patients on the day before surgical correction, and the 2nd and 4th days postoperation.

All patients had normal sinus rhythm and no bundle branch block (BBB) and they had no extracardiac abnormality or disease. Total correction was performed for the patients

Table I. SA-EKG values in patients and healthy controls.

Subjects	No.	Mean age (Yr)	F.QRS duration (ms)	RMS 40 Hz (μv)	HFLA LAS (ms)	Noise level (μv)
Normal children	20	8.8±2.6	84.2±9.5	181.0±89	18.9±9	0.3-0.9
Patients	20	8.1±2.1				
Pre-op			97.2±19.3	146.4±110.9	20.5±22.3	0.2-1.2
2nd day post-op			116.4±21.2	92.1±56.9	8.9±7.0	
4th day post-op			122.2±22.4	112.8±60	15.4±16.4	
P value			<0.05	NS	NS	

via the right atrium.

The control group was selected after precise history taking, physical examination and echocardiography. Mean age was 8.8 years (range 3 to 12 years). Thirteen of them were male and 7 female. Signal-averaged EKG (high-resolution EKG) recordings were obtained by commercial SA-EKG equipment (cardiogram PPG Hellige EK 56 EA LR). After skin prepping and cleaning we used adhesive silver chloride electrodes. SA-EKGs were recorded with 12 standard leads with analysis on corrected orthogonal leads X,Y and Z from which the device computes the 12 standard leads.

The computer algorithm calculates the three conventional time domain indices: the duration of filtered QRS (F. QRS-d), the root mean square (RMS 40) voltage or vector magnitude ($v=x^2+ y^2+z^2$) in the terminal 40 ms of the QRS complex that was calculated by combining the filtered signals from standard bipolar orthogonal leads, and the duration of the low amplitude signals (40 μv) at the terminal QRS complex (LAS 40). A bandpass filter of 40-250 Hz was used. The mean number of averaged beats was 100. The RMS noise level range in patients was 0.3-1.2 μv (mean =0.7 μv) and in controls 0.3-0.9 μv (mean = 0.6 μv). The duration of the highly amplified QRS complex was measured from the beginning of the Q wave to the end of the ventricular electrical activity, defined as the transition to a constant level of baseline noise. All measurements and computations were made automatically without manual intervention.

Statistical analysis

The data are expressed as the mean and standard deviation and the analysis was performed with the two-tailed t-test. The categorical data were compared using chi-square analysis. A probability (P) value of <0.05 was considered statistically significant.

RESULTS

Standard criteria for late potentials: the result of the time domain SA-EKG was considered abnormal when two or three conventional variables were beyond the normal range: total QRS.d>120 ms; duration of LAS (40 μv)>40 ms; and RMS voltage of the last 40 ms of the QRS complex <20 μv at a 40 Hz filter setting.[5,9] Table I shows the results of our study. The QRS duration on SA-EKG in the control and operated group shows a significant difference between the pre-op and 2nd day post-op QRS duration of children with CHD and normal children ($p<0.05$), and a comparison of RMS and HFLA (LAS) in the two groups showed no statistically significant difference between the control group and the group with CHD. None of the 20 patients had complete RBBB on EKG postoperatively, and maximum FQRS duration was 140 ms in only 5 patients.

DISCUSSION

It has been demonstrated that late potentials have prognostic value with respect to the risk of subsequent ventricular tachyarrhythmias following myocardial infarction or in patients with cardiomyopathy (CMP), arrhythmogenic right ventricular dysplasia, or in children with CMP, congenital heart disease and surgically corrected congenital heart disease.[2,9,10] Indices measured on SA-EKG have been shown to be independent predictors of VT, VF and sudden death in MI, CMP, syncope and primary electrical disturbances of the heart. Previous studies showed abnormal SA-EKG values and thus a high risk for VT.[1,3,7]

This study shows no difference in low amplitude potentials between the two groups (there were no abnormal low amplitude after potentials in our patients) and only an

increased QRS duration on SA-EKG in children with CHD. We can speculate on the reason for increased QRS duration in our patients and the further increase on the 2nd post-op day. It has been shown that heart rate does not have a significant effect on SA-EKG indices. Increased thickness of the ventricular myocardium may be a reason for the increased original QRS duration.[9] Further significant increases might be due to increased conduction time. Decreased conduction velocity may be due to edema, hemorrhage or surgical manipulation. Age-related differences in the thickness of the ventricular muscle and the propagation of electrical excitation in cardiac muscle (and in the hypertrophied ventricle) might also influence the SA-EKG.

In conclusion, there was no significant difference in HFLA and LAS and RMS 40 values pre- and postoperatively, the QRS-40 Hz duration was significantly longer in children with CHD, and it increased further significantly on the 2nd post-op day. In contrast to previous reports, our patients did not have CRBB which invalidated the SA-EKG study. The increased F. QRS-d in children with CHD postoperatively as compared with normal is:

a) Not related to heart rate,

b) Related to myocardial mass (ventricular hypertrophy in children with CHD), and

c) Related to conduction time and velocity. Edema, hemorrhage, and surgery may increase QRS duration.

REFERENCES

1. Rovamo L, et al: Late potentials on signal-averaged EKG in children after right ventriculotomy. Pediatric Cardiology 16 (3): 114-119, 1995.
2. Simson MB: Use of signals in the terminal QRS complex to identify patients with ventricular tachycardia after MI. Circulation 64 (2): 235-242, 1981.
3. Fourier A: Basic science aspects of the electrocardiogram. In: Garson A Jr, McNamara DG, (eds.), Pediatric Cardiology. Philadelphia: Lea & Febiger, pp. 518-521, 1990.
4. Hosoya Y, et al: Spectoral analysis of body surface SA-EKG in patients with previous anterior myocardial infarction as a marker of VT. Circulation 85: 2060-2062, 1992.
5. Kulakowski P, et al: Frequency versus time domain analysis of SA-EKG. JACC 20 (1): 135-43, 1992.
6. Walter PF: Technique of SA-EKG. In: Hurst JW, Schlant RC, (eds.), The Heart. New York: McGraw-Hill Company, pp. 893-896, 1994.
7. Janousek J, et al: Role of late potentials in identifying patients at risk for VT after surgical correction of CHD. Am J Cardiol 75: 146-150, 1995.
8. Lander P, et al: Critical analysis of the SA-EKG. Circulation 83: 105-107, 1993.
9. Hayabuchi Y, et al: Age related criteria for SA-EKG late potentials in children. Pediatric Cardiology 15 (3): 107-111, 1994.
10. Breithardt G, et al: Standards for analysis of ventricular LP using SA-EKG. Circulation 83 (4): 1050-1053, 1991.
11. Zimmermans M, et al: Frequency of ventricular LP and fractionated RV EKG after operative repair of TOF. Am J Cardiol 59: 448-453, 1987.
12. Fuller MS, et al: SA-EKG, brief review. Acc Current Journal Review Sept. /Oct: 12-15, 1994.
13. Gaur GT: Standard Electrocardiography and Vectorcardiography. In: Brandenburg RO, Giuliani ER (eds.), Fundamental and Practical Cardiology. Chicago: Year Book Medical Publishers, pp. 311-314, 1991.