

Different habitus but similar electrocardiogram: Cardiac repolarization parameters in children – Comparison of elite athletes to obese children

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ABSTRACT

- Introduction** : The standard 12-lead electrocardiogram (ECG) remains a widely used tool in the basic cardiac evaluation of children and adolescents. With the emergence of inherited arrhythmia syndromes, the period of cardiac repolarization has been the focus of attention. So far, data on cardiac repolarization and its normal variants in healthy children are scarce. This may cause uncertainties in the differentiation between pathologies and normal variants. As abnormal autonomic regulation seems to be a major influencing factor on cardiac repolarization, this study aimed to evaluate the parameters of cardiac repolarization of children in extremely good physical shape to obese children to improve knowledge about cardiac repolarization in these subgroups of pediatric patients that are vastly affected by the alterations of autonomic regulation.
- Methods** : A total of 426 pediatric volunteers (84 lean, healthy controls; 130 obese healthy pediatric volunteers; and 212 elite athletes) were enrolled in the study, and the parameters of cardiac repolarization were determined in 12-lead ECG.
- Results** : Most importantly, there were no pathological findings, neither in the healthy controls nor in the obese or athletes. Athletes showed overall shorter corrected QT intervals than children from the other groups. This is also true if a correction of the QT interval is performed using the Hodges formula to avoid bias due to a tendency to lower heart rates in athletes. Athletes showed the shortest Tpeak-to-end ratios between the groups. The comparison of athletes from primarily strength and power sports versus those from endurance sports showed endurance-trained athletes to have significantly longer QT intervals.
- Conclusions** : This study suggests that neither obesity nor extensive sports seems to result in pathological cardiac repolarization parameters in healthy children. Therefore, pathology has to be assumed if abnormal repolarization parameters are seen and might not be simply attributed to the child's habitus or an excellent level of fitness.
- Keywords** : Athlete, cardiac repolarization, children, long QT, obesity

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INTRODUCTION

The standard 12-lead electrocardiogram (ECG) remains a widely used tool in the basic cardiac evaluation of children and adolescents. With the emerging focus of present medicine to the preventive aspect and risk stratification, the inherited arrhythmia syndromes and thereby the period of cardiac repolarization have been the focus of attention.^[1-5] While the interpretation of cardiac arrhythmias is well established, the period of cardiac repolarization provides more challenges to the physicians.

To date, there are various clearly pathological phenomena of cardiac repolarization reported. Still, the interpretation of repolarization disturbances remains complex, as cardiac repolarization underlies variable influencing factors such as autonomic nervous system regulation. Nevertheless, the clinical consequence of missing or falsely diagnosing a potentially lethal disease as an inherited arrhythmia syndrome such as long QT syndrome demands a distinct differentiation of pathological phenomena and normal variants. Especially in children, data on these phenomena are scarce. In addition, autonomic regulation is more variable in children than that in adults, and hormonal influences during puberty add other influencing factors. While recent studies added some data on normal values of cardiac repolarization in healthy children, the influence of conditions such as obesity or extensive physical fitness remains less defined.^[6-14]

The aim of this study is, therefore, to compare the parameters of cardiac repolarization in children at the extremes of physical fitness and to increase knowledge about cardiac repolarization in these subgroups of pediatric patients that are vastly affected by deviations of autonomic regulation.

METHODS

Study population

After analysis of the LIFE Child Study at the Leipzig Research Centre for Civilization Diseases (LIFE) from 2011 to 2014 database and the database from the Leipzig Institute for Applied Training Sciences, a total of 426 pediatric volunteers (84 lean, healthy controls; 130 obese healthy pediatric volunteers; and 212 elite athletes) were enrolled in the study.^[15] All participants underwent a thorough cardiac evaluation including medical history, physical examination, anthropometric evaluation, 12-lead ECG, echocardiography, and blood samples. Written consent of the parents was obtained with inclusion into the study.

Definitions

Childhood obesity was classified as adjusted body mass index (BMI) >1.28 standard deviation score (SDS), and

children with an adjusted BMI ≤ 1.28 were classified as lean.

Children and adolescents were considered as elite athletes, if being either part of a German youth national team or equivalent in their specific sports.

Age adjustment

All anthropometric parameters were adjusted for age and sex, based on the national reference values of German children from Kromeyer-Hauschild *et al.* (2001) and accordingly were presented in SDS. Participants were stratified into overweight/obese and lean children applying an adjusted BMI 1.28/1.88 SDS as a cutoff according to the current German guidelines.^[16]

Electrocardiogram analysis

For each child, a 12-lead ECG was recorded using an electrocardiograph (General Electrics, MAC 5000). The frequency response of this recorder is flat to 150 Hz. A paper speed of 50 mm/s and amplitude of 10 mm/mV was used. The same technician recorded the ECGs throughout the study.

All ECGs were anonymized by pseudonymization and analyzed by two physicians with extensive experience in the analysis of pediatric ECGs. Both physicians had no access to demographic or clinical data.

Parameters of cardiac repolarization were measured in leads II and V5 of a standard 12-lead ECG. All parameters were determined from the measurements of at least six consecutive beats in lead II and V5. Minimum, maximum, and average values were acquired. Tpeak to end (TPE) was measured in milliseconds from the peak of the T wave to the end of the T wave. The end of the T wave was defined as the return of the descending limb to the TP baseline when not followed by a U wave or if distinct from following the U wave. If there was a terminal low-amplitude signal interrupting the terminal portion of the T wave, the downslope of the T wave was extended by drawing a tangent to the steepest proportion of the downslope until it crosses the TP segment to determine the end of the T wave. The QT interval was then corrected for heart rate using the Bazett's formula (QT/\sqrt{RR}) (QTc) and in addition the Hodges's formula in patients with a resting heart rate below 50 bpm as suggested in literature.^[17-19] The RR interval was measured in seconds and taken as the immediate RR interval preceding the TPE interval from the same lead, in which the TPE interval was measured. QT dispersion was calculated as the difference between maximum and minimum QT interval within the measurement of six consecutive beats.

Statistical analysis

Statistical analysis was carried out with SPSS 21.0 software (SPSS Statistics, IBM, Ehningen, Germany). Continuous data were assessed for normality, and the Student's *t*-test

was used for normally distributed data. To characterize the influence between continuous variables, bivariate correlation was used, and the Pearson’s correlation coefficient (*r*) was reported. For this study, α was set at 0.05; thus, $P < 0.05$ (two-sided) was considered statistically significant.

RESULTS

Patients’ characteristics

Table 1 shows the patients’ basic characteristics of the stratified groups: lean, overweight/obese, and elite athletes including anthropometric data. All patients had structurally normal hearts in echocardiography.

Parameters of cardiac repolarization

Table 2 shows the parameters of cardiac repolarization of the three groups: lean, overweight/obese, and athletes. Although there were significant differences between the groups, no pathological findings were recorded. First of all, there is a trend toward shorter corrected QT intervals in athletes despite insignificant differences in average absolute QT intervals. This is also true if a correction of the QT interval is performed using the Hodges’s formula to avoid bias due to a tendency to lower heart rates in

athletes. Second, athletes showed statistically significant shorter TPE ratios compared to the other groups, albeit clinically probably not leading to a significant difference.

Comparison of athletes from strength and power sports versus athletes from endurance sports

Table 3 shows the comparison of sportsmen from strength and power sports versus endurance sports. As expected, the different physiognomy can be seen in the anthropometric data. Comparing the absolute QT intervals of both groups, endurance-trained athletes tend to have significantly longer QT intervals. When the QT intervals are corrected for heart rate, this effect persisted only in the corrections using the Hodges’s formula and was not found to be statistically significant when using the common Bazett’s formula. There were no statistically significant differences in the parameters of cardiac inhomogeneity (TPE) between the groups.

DISCUSSION

The current study evaluated the influence of obesity and extreme physical fitness on parameters of cardiac repolarization in children. The results of the current study demonstrate one major finding. In this pool of otherwise healthy children, there were no pathological phenomena of

Table 1: Patient’s characteristics

	Lean control	Obese	Athlete	P
Age (years)	10.6 (6, 4; 17, 3)	12.3 (5, 5; 19, 1)	15 (9, 6; 17, 9)	<0.001
Gender				
Male	40	70	125	0.197
Female	44	60	87	
Height (cm)	146 (116; 180)	157 (120; 188)	167 (129; 203)	<0.001
Height ADJ	0.05 (-2.17; 1.88)	0.72 (-2.03; 3.84)	0.17 (-2.13; 3.45)	<0.001
Weight (kg)	36.2 (19; 71.7)	70.33 (31.7; 136)	56.85 (27.1; 121.8)	<0.001
Weight ADJ	-0.049 (-2.59; 1.46)	2.31 (0.83; 4.53)	0.18 (-2.55; 3.31)	<0.001
BMI	16.91 (10.6; 22.6)	27.81 (20.1; 43.7)	20.05 (15.1; 38.4)	<0.001
BMI ADJ	-0.23 (-5.86; 1.26)	2.28 (1.41; 3.85)	0.1 (-2.43; 3.06)	<0.001

All numbers are depicted as median and range. BMI: Body mass index, ADJ: Adjusted to age

Table 2: Parameters of cardiac repolarization

	Lean control	Obese	Athlete	P
HR (bpm)	73 (56; 103)	78 (48; 118)	60 (42; 94)	<0.001
PR interval	128 (87; 196)	132 (57; 213)	140 (80; 190)	0.001
QRS width II	80 (58; 107)	82 (57; 108)	100 (80; 130)	<0.001
QT max II	370 (310; 410)	360 (300; 460)	370 (285; 455)	<0.001
QT avrg II	360 (305; 405)	355 (295; 455)	370 (285; 455)	0.476
QT max V5	360 (290; 400)	360 (300; 450)	380 (300; 470)	<0.001
QT avrg V5	355 (285; 395)	352 (295; 440)	375 (295; 455)	<0.001
TPE II	70 (50; 100)	70 (50; 100)	60 (40; 80)	<0.001
TPE V5	60 (50; 80)	70 (50; 100)	60 (40; 90)	<0.001
QTc max II (Bazett)	410 (350; 425)	410 (340; 455)	380 (290; 430)	<0.001
QTc avrg II (Bazett)	400 (350; 410)	400 (340; 440)	370 (290; 420)	<0.001
QTc max II (Hodges)	//	//	380 (330; 440)	//
QTc avrg II (Hodges)	//	//	370 (325; 430)	//
QTc max V5 (Bazett)	395 (355; 425)	405 (340; 460)	385 (300; 450)	<0.001
QTc avrg V5 (Bazett)	390 (350; 410)	400 (330; 450)	370 (300; 435)	<0.001
QTc max V5 (Hodges)	//	//	380 (325; 445)	//
QTc avrg V5 (Hodges)	//	//	375 (320; 435)	//

All numbers are depicted as median and range. The unit of the HR is depicted in beats per minute, all other parameters in milliseconds. HR: Heart rate, TPE: Tpeak to end, //: No value available

Table 3: Parameters of cardiac repolarization in athletes - endurance versus strength and power sports

	Endurance	Strength	P
Age (years)	15.01 (9.6; 17.2)	15.15 (9.9; 17.9)	0.461
Gender			
Male	64	61	0.93
Female	44	43	
Height (cm)	171 (145; 203)	165 (129; 199)	0.001
Height ADJ	0.68 (-1.64; 3.45)	-0.18 (-2.13; 2.74)	<0.001
Weight (kg)	58.8 (34.5; 103.3)	55.2 (27.1; 121.8)	0.714
Weight ADJ	0.29 (-1.78; 2.52)	0.01 (-2.55; 3.31)	0.088
Weekly training h	15 (3; 32)	10 (0; 30)	<0.001
HR (bpm)	59 (42; 76)	65 (43; 94)	<0.001
PR interval II	140 (90; 180)	140 (80; 190)	0.017
QRS width II	375 (310; 440)	100 (80; 120)	0.196
QT max II	390 (320; 460)	360 (290; 460)	<0.001
QT avrg II	380 (315; 450)	355 (285; 455)	<0.001
QT max V5	390 (320; 470)	370 (300; 460)	<0.001
QT avrg V5	380 (315; 455)	360 (295; 455)	<0.001
TPE II	60 (40; 80)	60 (40; 80)	0.044
TPE V5	60 (40; 90)	60 (40; 80)	0.330
QTc max II (Bazett)	380 (315; 430)	380 (290; 420)	0.397
QTc avrg II (Bazett)	370 (310; 420)	370 (290; 410)	0.214
QTc max II (Hodges)	385 (335; 440)	370 (330; 430)	<0.001
QTc avrg II (Hodges)	380 (335; 430)	365 (325; 425)	<0.001
QTc max V5 (Bazett)	385 (325; 450)	390 (300; 435)	0.578
QTc avrg V5 (Bazett)	375 (320; 435)	370 (300; 425)	0.361
QTc max V5 (Hodges)	385 (340; 445)	375 (325; 435)	<0.001
QTc avrg V5 (Hodges)	380 (340; 435)	370 (320; 430)	<0.001

All numbers are depicted as median and range. The units of the heart rate, age, weight, and height are presented in brackets, all other parameters in milliseconds. HR: Heart rate, TPE: Tpeak to end, ADJ: Adjusted to age

cardiac repolarization, neither in the group of elite athletes nor in the group of obese patients or healthy controls.

Yet, data analysis showed significant differences from the literature in adults.

First of all, the data showed no lengthening of the QT or corrected QT interval in elite athletes compared to lean probands. This finding stands in contrast to the findings in literature concerning adults.^[20-24] To rule out the Bazett's formula for the correction of QT interval in the group of elite athletes with generally lower heart rates as a possible bias, we compared the results to a correction with the Hodges's formula that might be less biased by lower heart rates. The results were independent from the formula used for the correction of QT interval to the heart rate. As reported by D'Ascenzi *et al.*, the reason for this incoherence of pediatric data with data from adult patient collectives may be because puberty seems to be a strong influencing factor regarding QT interval prolongation. This might explain why these changes are probably not yet reported in adolescents, but may develop in adult athletes. In addition, it may be speculated that physical adaptation to extensive sports is not fully developed in adolescence, and the full effects of physical adaptation will only be present in adulthood.

Second, the overall data demonstrated the longest corrected QT intervals in obese patients, as has been demonstrated in studies before.^[13,14] Those studies reported an increased parasympathetic tone in obese patients and the hormonal activity of body fat deposits as causative mechanisms. Particularly, the hormonal activity

of body fat leads to elevated estrogen levels which are assumed to promote QT interval prolongation. As this finding might not be unexpected, it gives some hints to the etiology of QT interval prolongation in children. Both athletes and obese children are supposed to have a rather increased parasympathetic tone, often referred to as a possible influencing factor for QT interval prolongation. Nevertheless, the presented cohort showed relatively long QT intervals in the obese and relatively short QT intervals in the athletes, making the basic parasympathetic tone a rather minor influencing factor in children.

When looking at the parameters of cardiac electrical inhomogeneity, there are only minor differences between the groups. In particular, there were no statistically significant differences between athletes and lean probands. Taking a closer look at the group of obese patients, statistically significant changes in the parameters of cardiac electrical inhomogeneity (TPE interval) could be demonstrated. Primarily implying a possibly elevated risk for cardiac arrhythmia, the actual data, although statistically significant, demonstrate only differences of a few milliseconds that are rather unlikely to represent a parameter of clinical significance or to conclude on an elevated arrhythmogenic risk. Yet, some more data are needed for an appropriate evaluation of this topic.

CONCLUSION

This study suggests that neither obesity nor extensive sports seems to result in pathological cardiac

repolarization parameters in healthy children. Therefore, pathology has to be assumed if abnormal repolarization parameters are seen and might not be simply attributed to the child's habitus or an excellent level of fitness.

Limitations

The main limitation of this study is the difference in median ages between the groups. As the parameters of cardiac repolarization might be influenced by puberty, this might have an effect on the reported data. Yet, with patients of the lean control group being the youngest, a possible age-related effect on parameters of cardiac repolarization should only be important if pathological parameters would have been found in the older participants of the two other groups.

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Conflicts of interest

There are no conflicts of interest.

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