We examined QRS changes in patients with diabetes mellitus (DM) and metabolic syndrome (MetS), the first degree offspring of patients with DM and MetS, and compared them with a control group of healthy subjects. We hypothesized that electrocardiographic changes indicating electrical remodeling of myocardium are present already in apparently healthy offspring (HO) of patients with DM and MetS.

Material and Methods: A total of 530 subjects were included in the study. The study population consisted of 5 groups:

(1) group MetS (n = 111): patients with MetS aged 20 to 78 years; mean, 54 years;

(2) group HO MetS (n = 121): apparently, HO of patients with MetS aged 15 to 51 years; mean, 29 years;

(3) group DM (n = 90): patients with MetS aged 40 to 79 years; mean, 63 years;

(4) group HO DM (n = 126): apparently, HO of patients with DM aged 15 to 61 years; mean, 34 years; and

(5) group 3 (n = 82): HO of apparently healthy subjects aged 19 to 58 years; mean, 27 years.

All subjects underwent blood pressure and anthropometric measurements. Fasting blood samples were obtained, and biochemical characteristics were measured. Standard 12-lead electrocardiograms were recorded in all subjects and were evaluated by 1 expert blinded to diagnoses. The QRS amplitude parameters were analyzed as well as the estimated QRS spatial vector magnitude (QRSmax), the Sokolow-Lyon index (SLI), and the Cornel voltage criterion.

Results: Both patients with DM and MetS showed significant changes with respect to the healthy subjects: left axis deviation, increase in R_I amplitude, decrease in R_{II} amplitude and deep S_{III}, high R_{aVL} amplitude, and a decrease in R_{aVF} amplitude. The QRSmax and SLI were significantly decreased, whereas the Cornel voltage increased significantly, following the increase in R_{aVL}.

The QRS changes in offspring of both patients with MetS and DM also differed significantly with respect to the HO and followed the same pattern as patients with DM and MetS with 1 exception: the QRSmax and SLI values increased significantly.

Conclusion: The offspring of both patients with DM and MetS displayed significant changes in QRS complex that are indicative of early changes in the sequence of depolarization and could be signs of early subclinical cardiovascular damage.

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The effect of reduced intercellular coupling on the QRS complex pattern

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Classical electrocardiographic (ECG) diagnosis of left ventricular hypertrophy (LVH) is based on voltage criteria, that is, increased amplitude of the QRS complex in particular leads. However, the spectrum of QRS complex patterns in patients with LVH is wider, and the increased QRS amplitude is only found in a minority of patients with increased left ventricular mass. In our previous study [1], we found decreased QRS complex amplitude associated with reduced connexin43 expression in the left ventricle in spontaneously hypertensive rats. Because this finding of decreased QRS complex amplitude was in contrast with the classical diagnostic concept, we studied the effect of reduced intercellular coupling on the resultant amplitude and morphology of the QRS complex of the surface ECG using a computer model.

Our computer model [2] reliably represents 3 interacting mechanisms by which reduced intercellular coupling affects the QRS complex: (1) reduction of the depolarization wavefront velocity, (2) attenuation of the current generator associated with the wavefront, and (3) modification of current flow through the thorax.

We showed that the gradual reduction of intercellular coupling, which is part of the hypertrophic remodeling of left ventricle in LVH, resulted in QRS complex changes that are consistent with the following ECG findings in patients with LVH:

prolongation of the QRS complex duration;

shift of the electrical axis of the heart to the left and the pattern of left anterior fascicular block; and

decrease in QRS complex amplitude in all leads of the 12-lead ECG except of the lead aVL, where the QRS complex amplitude increased.

Our results stress the importance of understanding the role of impaired electrical properties influencing the sequence of depolarization that are reflected in QRS complex changes in patients with LVH. These changes are not currently considered in the interpretation of QRS complex morphology. It is well documented that the connexin43 reduction creates a basis for triggering and maintaining arrhythmias; the early recognition of these changes is therefore of great clinical importance.

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Feasibility of high-frequency QRS analysis in patients with acute myocardial infarction

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Background: Risk stratification of patients with chest pain relies on identification of repolarization abnormalities. However, the initial electrocardiogram (ECG) of patients with suspected acute myocardial infarction is often normal or nondiagnostic. It has been shown that ischemia brings about depolarization changes that can be quantified by computerized analysis of high-frequency mid-QRS components (HFQRS). Morphological changes in HFQRS were shown to be a sensitive marker of ischemia in animal models and humans undergoing intracoronary balloon occlusions. We aimed to study the morphological patterns of HFQRS in patients with acute ST-elevation myocardial infarction (STEMI) before and after revascularization and to compare these patterns with healthy controls.

Methods: Eleven patients with STEMI diagnosis (age, 53 ± 12 years; 8 men) admitted to the intensive cardiac care unit (ICCU) were included in the study. No patient had prior history of CAD. High-resolution, 12-lead ECG (HyperQ System; BSP Ltd, Tel Aviv, Israel) was acquired on ICCU admission and before discharge. The HFQRS analysis was carried out using designated software performing (1) accurate alignment and averaging of QRS complexes, (2) band-pass filtering of the average complexes in the frequency range 150 to 250 Hz, and (3) extraction of HFQRS morphological index (HFMI) quantifying the extent of reduced amplitude zones in the HFQRS signal. The HFMI values at admission and discharge were compared among the study group and against a control set of 443 subjects (age, 54 ± 10 years; 278 men) without acute myocardial ischemia.

Results: The time from onset of symptoms to first ECG recording at the ICCU was 1.1 ± 0.6 hours (mean ± SD). The discharge ECG recording was performed 20.2 ± 6.4 hours after revascularization. At ICCU admission, average troponin T level was 1.2 ± 2.3 ng/mL; 8 patients had elevated troponin T, and 5 patients met the ECG criteria for acute myocardial ischemia. Peak troponin T was 2.1 ± 2.1 ng/mL. At discharge, 8 patients had repolarization abnormalities. The HFMI at admission was higher compared with discharge (6.5% ± 3.4% vs 3.3% ± 1.4%, $P < .01$) and with a healthy control group (2.6% ± 2.1% $P < .001$). The HFMI decreased from admission to discharge in 9 patients ($P < .05$) by average of 55% ± 18%.

Conclusions: Analysis of HFQRS morphology is feasible in patients with STEMI and was found to be in agreement with the clinical, ECG, and biochemical findings. The HFQRS-derived indexes may aid in risk stratification of patients with chest pain and in early detection of acute myocardial infarction.

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Increased repolarization heterogeneity and ventricular instability in patients with end-stage renal disease with an increased risk for death

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