

Changes in Body-Surface Electrocardiograms from Geometric Remodeling due to Obesity

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Abstract. Both diabetes and obesity are responsible for deleterious changes to the heart. To separate consequences of geometric changes from electrophysiologic ones, we investigated how changes in cardiac and torso geometry, associated with obesity, affected body-surface electrocardiograms. For this study, we modified the realistic heart and torso models of the simulation package ECGSIM. Body-surface potentials were calculated from transmembrane potentials on the heart surface and studied using isopotential maps and spectral and principal-component analysis. We found relative errors over the body-surface during the Q-T interval of 12, 14, and 68% for hypertrophy of the heart, extension of the abdomen, and heart displacement with obesity, respectively. The major change to the standard 12-lead set also occurred with heart displacement. The mean relative error over the T wave in the precordial leads V3 to V6 was 83% with heart displacement. These results demonstrate the limitations of using standard lead sets to characterize electrocardiographic changes in obese subjects and point to the need for more comprehensive measures, such as body-surface mapping and inverse electrocardiography, to describe electrical remodeling in obesity and diabetes.

Keywords: Bidomain Model, Obesity, 12-lead Electrocardiograms, Forward Electrocardiology, Transmembrane Potentials

1. Introduction

Myocardial disease is common in type 2 diabetics, including elevated risk for myocardial infarction, heart failure, and sudden cardiac death. Both diabetes and obesity, which is widespread among diabetics, are responsible for deleterious electrophysiologic changes to the heart and for geometric changes to the heart and torso [Devereux et al., 2000]. To separate consequences of geometric changes from electrophysiologic ones, we investigated variations in body-surface electrocardiograms (ECGs) during the Q-T interval due to changes in cardiac and torso geometry associated with obesity while maintaining normal electrophysiology of the heart.

In this study, we were interested in apple-shaped obesity (body fat stored around the abdomen, chest and surrounding internal organs), because this body shape is associated with a higher risk of diabetes and heart disease [Carey et. al, 1997; Y Wang et. al, 2005]. Several striking changes in heart and torso anatomic structure are associated with obesity, including increased thickness of the left ventricular walls, changes in the position and orientation of the heart, and enlargement of the abdominal volume. Based on these variations, three obesity-associated anatomic features were characterized: 1) left ventricular hypertrophy (LVH), 2) heart displacement, and 3) abdominal enlargement, measured via waist circumference.

2. Material and Methods

To quantify effects of geometric changes, we modified the

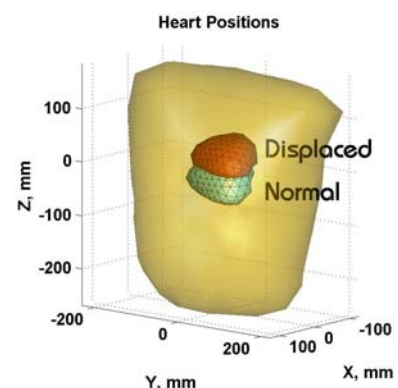


Figure 1. Normal torso with normal and obesity-displaced hearts.

realistic heart and torso models of the simulation package ECGSIM [van Oosterom and Oostendorp, 2004] as input to our bidomain forward-problem solution [S Wang, 2009] to quantify changes in body-surface potentials that occur with geometric alterations, such as heart displacement illustrated in Figure 1.

To simulate LVH effects, both the inter-ventricular septum (IVS) and the posterior wall (PW) of the normal heart model were increased by 20% (LVH20). Values were selected to match measured increases in the thickness of the IVS and PW of 11-22 and 13-25% seen in obese groups [Avelar et al., 2007]. To simulate the obese heart displacement, the heart was shifted (8% of the torso height) and rotated (6, 4.5 and 28 degrees in the coronal, frontal and sagittal planes). We also enlarged the waist circumference of the ECGSIM torso to match changes in obesity. Displacement and enlargement values were taken from experimental measurements on normal and obese subjects made by our group.

Our bidomain, forward-problem model was used to calculate body-surface ECGs, V , from cardiac transmembrane potentials, Φ_m , on the heart surface. From the bidomain, forward-problem solution A ,

$$V = A\Phi_m \quad (1)$$

Body-surface potentials V were calculated from A matrices found for 1) a normal heart and in a normal torso, 2) an hypertrophied heart in a normal torso, 3) a displaced heart in a normal torso, and 4) a normal heart in a torso with an enlarged abdomen. These body-surface potentials were compared using isopotential maps and spectral and principal component analysis. The standard 12-lead set from all four models was also extracted and compared.

3. Results

Differences in isopotential maps with hypertrophy, extended abdomen, and displaced heart compared to values with a normal heart in a normal torso are summarized in Table 1. Relative errors over the body-surface during the Q-T interval were 12, 14, and 68% with hypertrophy, extended abdomen, and a displaced heart, respectively.

	20% LVH	Extended Abdomen	Displaced Heart
Relative Error	0.1195	0.1358	0.6821
Correlation Coef.	0.9977	0.9950	0.7838

Table 1. Body-Surface ECGs during QRS and T compared to normal ECGs.

Means of the spectral magnitudes of the Fourier transforms of the Q-T potentials over the body surface in the three altered geometries were compared to spectra on the normal torso with a normal heart, as shown in Figure 2. At each frequency the

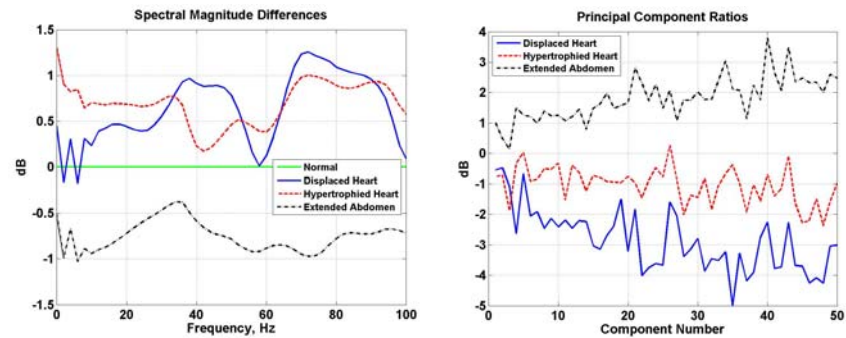


Figure 2. Spectral and principal component changes with geometric alterations in obesity. Both plots are relative to the value in the normal torso with a normal heart.

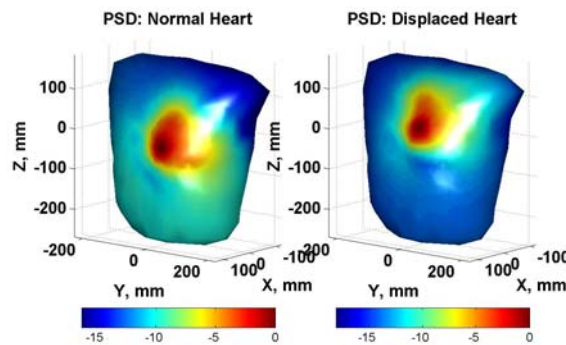


Figure 3. Total energy at each torso site over the Q-T interval for the normal and displaced hearts. The color scale is in dB.

magnitudes were adjusted so that normal was always at 0 dB. The changes relative to normal are similar for the hypertrophied and displaced hearts. On the other hand, the spectral magnitudes for the extended abdomen were less than those in the normal heart and torso. In all cases, however, spectral changes were relatively small, only about ± 1 dB. The change in the ratios of principal components was similar, only about ± 1 -2 dB. A 1 dB change is about 12%, which is roughly the relative error in Table 1 for the hypertrophied-heart and extended-abdomen results. These results suggest that, although there are indeed changes in body-surface

waveforms with the geometric alterations described here (which might be accentuated by considering QRS and T wave potentials separately), the major change is a shift of the body-surface patterns with the displaced heart.

This pattern shift is illustrated in Figure 3 which shows the relative power in dB at each torso site over the Q-T interval for the normal and displaced hearts. Note that the dB scale for each surface plot is similar, but the patterns of the power deposition are quite different.

The consequence of the waveform changes and pattern shift of electrocardiographic potentials with obesity is clearly seen in the standard 12-lead set as shown in Figure 4. It is especially important to identify effects of obese geometry on the T wave because they may confound T wave changes seen in diabetics [Devereux et al., 2000]. Relative errors with heart displacement over the T wave in the precordial leads V3 to V6 were 117, 98, 74, and 44 %, respectively. The mean relative error was 83%.

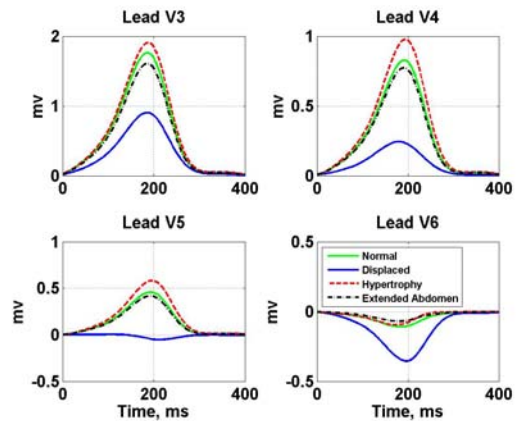


Figure 4. Changes in the T wave due to geometric changes with obesity.

4. Discussion

Our results suggest that geometric changes accompanying obesity may have a significant effect on electrocardiograms that may be confused with electrophysiologic changes due to diabetes. An understanding of the geometric effects is essential to separating changes that occur due to obesity from those due to diabetes. These results also demonstrate the limitations of using the standard 12-lead signals in the presence of hypertrophy, an enlarged abdomen, and a displaced heart to characterize electrical remodeling with diabetes. ECG changes associated with obesity, especially the large error with heart displacement, suggest the use of body-surface maps and inverse solutions on the heart-surface to get a comprehensive, stable measure of cardiac sources in obese and diabetic subjects.

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