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Review

Electrocardiological Features in Obesity: The Benefits of Body Surface Potential Mapping

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Key Words
Obesity · Metabolic syndrome · Electrocardiogram · Electrocardiology · Cardiovascular risk

Abstract
Background: Various ECG abnormalities are commonly observed in obesity and in metabolic syndrome. ***Summary:*** Some of these abnormalities are caused by the pushed-up position of the diaphragm due to obesity and others occur as a result of the complications of the condition. The position of the R wave may change, various arrhythmias may develop or the QT interval may be prolonged, which increases the tendency to malignant arrhythmias. In obesity, the ECG signs of ventricular hypertrophy are less informative due to the accumulation of epicardial and subcutaneous adipose tissue. In general, it can be concluded that a microcirculation disorder is present in metabolic syndrome that may primarily be associated with ST-T wave abnormalities. ***Key Messages:*** Body surface potential mapping is a more sensitive method than traditional ECG with potentially greater use for diagnosis mainly in the early phase of non-ST elevation myocardial infarctions.

Introduction

The epidemic of obesity has spread throughout the world. Obesity-induced insulin resistance and hyperinsulinaemia make type 2 diabetes mellitus a more and more frequent problem, and atherogenic dyslipidaemia occurs as well. The increased sympathetic activity in obesity triggers an elevation of blood pressure. The presence of three of the aforementioned factors at a time is defined as metabolic syndrome, where the cardiovascular risk associated with each factor is not simply added up but multiplied [1].

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Table 1. Common ECG abnormalities in obesity

Frequency ↑
PQ interval ↑
QRS width ↑
QRS amplitude ↑ or ↓
QTc interval ↑
QT dispersion ↑
'Late potentials' ↑
ST-T abnormalities
ST depression
Left shift of the R axis
Flatter T waves (in inferolateral leads)
Left atrial abnormalities
Inferior Q waves without necrosis

ECG Abnormalities in Obesity

A number of ECG abnormalities may be associated with obesity of various causes. A left shift of the P, QRS and T axes, morphological deviation of the P wave, low QRS amplitude, flattening of T waves (mainly in inferolateral leads) and potentially prolonged QT and QTc intervals are present at a significantly higher rate in obese than in non-obese individuals. The prolongation of the QT and QTc intervals is caused by the heightened sympathetic activity characteristic of obesity that increases the reduced heart rate variability; all these elements have the potential to cause arrhythmia [2]. Various arrhythmias occur more commonly in obese individuals, especially in those with co-existing sleep apnoea or left ventricular hypertrophy. Many ECG abnormalities are reversible, since they may change proportionately with reduced body weight, as was confirmed in a study demonstrating a reduction of the mild left shift of the P and QRS axes after weight loss [3]. In obesity, ECG may change in many ways (table 1) due to an elevation of the diaphragmatic level, left ventricular hypertrophy caused by increased cardiac output, the presence of epicardial and subcutaneous adipose tissue – functioning as electric insulation layers – as well as sleep apnoea/hypoventilation syndrome [4].

Complete bundle branch blocks caused by elevated pressure are easy to recognise, but the diagnosis of, for instance, an incomplete right bundle branch block is often wrong [5]. The assessment of right ventricular pressure elevation is even more difficult [6].

Left ventricular hypertrophy is a significant predictor of cardiovascular morbidity and mortality [7]. In one study, the sensitivity of the ECG criteria for left and right ventricular hypertrophy was investigated in abnormally obese patients and compared with echocardiography [8]. The results demonstrated that the sensitivity of the ECG criteria for three different left ventricular hypertrophies ranged between 0 and 20%, while their specificity was within the range of 73–100%. Similarly, the sensitivity of the ECG criteria for right ventricular hypertrophy was quite low (0–16%), while their specificity ranged between 95 and 100%. The combination of ECG criteria did not increase their sensitivity and actually caused a significant decrease in their specificity.

Another study showed that the use of the Cornell voltage criteria was much more useful than that of the Sokolow-Lyon voltage criteria [9]. The Cornell voltage criteria [10] showed sensitivity in obese and non-obese subjects, while the sensitivity of the Sokolow-Lyon voltage criteria was demonstrated to be quite low in obese individuals [11]. ECG as the first cardiological examination method is inexpensive, accessible and easy to perform; however, in obese individuals, it is not always the most preferred tool for the diagnosis of ventricular hypertro-

phies. Thus, it can be concluded that ECG has very limited use in the diagnosis of left and right ventricular hypertrophy in obese subjects (particularly in abnormally obese patients).

The impact of various risk factors in metabolic syndrome primarily manifests in various forms of myocardial ischaemia which may be caused by coronary artery disease facilitated by those risk factors and the microcirculation disorder of the myocardium [12]. The different risk factors may trigger various abnormal myocardial changes including, for example, myocyte hypertrophy, the accumulation of interstitial connective tissue (together called 'stiffness' development) and narrowing of the capillary lumen. Myocardial ischaemia induced by reduced microcirculation and its consequences may be detected with several electrocardiological methods. The worsening of myocardial function and compliance may lead to elevated end-diastolic pressure, atrial load, elevated right ventricular pressure and arrhythmias. The question is which electrocardiological method is suitable for the detection of the different abnormal changes – the cardiac impact of risk factors – associated with the early and late phases of myocardial damage.

Arrhythmias in Obesity

The risk of arrhythmias and sudden death is higher in obese individuals even without cardiac dysfunction. The Framingham Study has shown that the mortality rate is increased 6- to 12-fold in seriously obese men [13]. QT prolongation was observed in 30% of the patients with impaired glucose tolerance in the NHANES III (Third National Health and Nutrition Examination Survey) [14].

The high blood glucose level increases vasomotor tone and ventricular instability, since it reduces the accessibility of nitrogen monoxide [15]. In extremely obese patients, fatal arrhythmia and sudden cardiac death caused by the common dilated cardiomyopathy are not rare [16]. Serious hypoglycaemic episodes requiring intervention occur with a frequency of 62–170 episodes/100 patient-years with the treatment of type 1 diabetes mellitus [17, 18], while in type 2 diabetes mellitus, the frequency of serious hypoglycaemic episodes may be nearly 73/100 patient-years. Enhanced sympathoadrenergic activity induced by hypoglycaemia results in tachycardia, feeling of stress and vasoconstriction. As a sign of this, the systolic blood pressure increases while the diastolic value tends to decrease [19]. Previous reports have shown a relationship between hypoglycaemia and atrial fibrillation. However, the changes in repolarisation (prolongation of the QT interval) during hypoglycaemia have greater clinical significance. The significance of the prolongation of the QT interval during hypoglycaemia lies in the fact that it is an independent risk factor of sudden death and Torsade de pointes [20]. The 'late potential' test is less frequently used, although a positive result indicates 'late' depolarisation induced by ischaemia or myocardial disorder [21] and thereby susceptibility to arrhythmia (e.g. it yields positive results in 100% of extreme obesity cases).

Hypoglycaemia and the QT Interval

Hyperinsulinaemia-induced hypoglycaemia prolongs the QTc interval and decreases the T wave amplitude (even in healthy individuals). In the EURODIAB (Europe and Diabetes Study), a higher HbA_{1c} value, female sex and an elevated systolic blood pressure have been shown to be related to QTc prolongation in subjects with normal baseline QTc intervals, but physical activity and normal body weight had a protective effect against this prolongation. Sawicki et al. [22] have found the degree of QT distribution to be the most important independent risk factor for all-cause, cardiac and cerebrovascular death. Many studies in either

healthy subjects or (type 1 and 2) diabetic patients have investigated the effect of hypoglycaemia on the QT interval. Marques et al. [23] and Lee et al. [24] have demonstrated a significant QTc prolongation during hypoglycaemia episodes in patients with type 1 diabetes mellitus, and other investigators have found consistent results [25, 26].

Mechanism of QTc Changes

There are two basic mechanisms in the background of the prolongation of the QT interval. One is the hypoglycaemia-induced activation of the sympathoadrenergic system where epinephrine and norepinephrine are released into the bloodstream. The other includes the reduced potassium level caused by elevated insulin and epinephrine levels. Studies in healthy subjects and patients with type 1 diabetes mellitus have shown that QTc prolongation caused by hypoglycaemia-induced activation of the sympathoadrenergic system could be successfully treated with beta-blockade [27]. In a study by Robinson et al. [28], the QTc prolongation was slightly decreased by the administration of potassium infusions.

Possible Benefits of Body Surface Potential Mapping versus ECG in Obesity

ECG actually records the electric activity (potential) of the heart. The principle of this method is that the electric activity of the heart detectable on the body surface (ECG leads) is used to draw conclusions on various cardiac events and the underlying anatomical and pathological changes of the heart. In his first tests, Waller [29] essentially performed body surface potential mapping (BSPM) and assumed the presence of an electric dipole in the background of the potential distribution, and this is why the measurement of the summation vector had been the objective of electrocardiological examination for a long time. In many instances, specific calculated parameters of vectorcardiography (change in spatial size of the summation vector, change in azimuth angle, spatial velocity and spatial angular velocity) indicate the change in the order of electric depolarisation more reliably. In such cases, the underlying cause may either be a bundle branch block or a change in the myocardium [30].

Later, numerous attempts have made to find which leads may be the most informative ones. Wilson et al. [31] introduced the 12-lead ECG, consisting of 3 dipolar, 1 frontal (modified extremity unipolar) and 6 chest unipolar leads. In the extremity leads used by Wilson et al. (VR, VL and VF), only small surface amplitudes could be detected. Therefore, Goldberger [32] amplified them (aVR, aVL and aVF), and these are the leads that are in use today.

Due to numerous electrodes (63 in the Montreal system) placed on the body surface (even on the back), minor electrocardiological events can also be detected that are less detectable or non-detectable with 12-lead ECG [33, 34]. In general, qualitative and quantitative evaluations of isopotential maps provide the most information, but they are also the most time-consuming procedures. The assessment of isoareal maps is faster [35]. Purely quantitative parameters may also provide useful information [36, 37]. By using BSPM, the ratio of dipolar to non-dipolar forces during depolarisation can be determined, and with an increased non-dipolar activity, this may indicate myocardial inhomogeneity and major malignant arrhythmias [38–41]. There are some specific clinical alternatives for the use of BSPM that may be useful for the examination of outcomes of metabolic syndrome: minor electric potential losses [42–45], the presence and location of coronary artery stenoses [46], the electric viability of the myocardium [47, 48], electric changes following revascularisation [49, 50], various intervals [51] and the electric activity of the left atrium [52] can be accurately determined, or clinical indices such as overload [53] or even haemodynamic information can be obtained [54].

It is a well-known fact that micro- and macrovascular complications are often present at the time of the first diagnosis of diabetes. Ždarská et al. [55] studied patients with type 1 diabetes without cardiovascular symptoms by using BSPM. Elevated heart rate, QRS and QT changes as well as depolarisation and significant changes in repolarisation were found compared with the non-diabetic controls.

Approximately 15–20% of patients hospitalised with chest symptoms suffer from acute coronary syndrome, while in diabetic patients, the evaluation of clinical symptoms is much more difficult due to autonomic neuropathy [56]. The 1-year mortality after acute coronary syndrome is significantly higher in diabetic patients than in non-diabetic subjects [57] despite up-to-date treatment.

An evaluation of non-ST elevation myocardial infarction in diabetic patients may be even more difficult, due to the often atypical symptoms and non-specific ECG changes and since cardiac necroenzyme kinetics may only surely indicate the problem within 14–16 h. McClelland et al. [58] studied patients examined due to chest pain by using BSPM evaluated with an automated program (in addition to the standard diagnostic procedures of ECG and assessment of cardiac necroenzymes). Acute myocardial infarction confirmed by biochemical markers was found in 53 of the 103 patients. It was diagnosed with a significantly better specificity by BSPM than by standard 12-lead ECG (45 vs. 64%, $p < 0.001$), while no significant difference in sensitivity was shown (98 vs. 94%) [58].

Conclusions

There are various ECG abnormalities observed in obesity and in metabolic syndrome. In these conditions, we have several difficulties to evaluate the ECG features. One of the most important factors includes the amount of thoracic and epicardial adipose tissue (functioning as electric insulation layers), which lowers the electric potentials from unipolar leads. Therefore, the ECG criteria for left and right ventricular hypertrophy have a poor sensitivity but strong specificity. The elevated diaphragmatic level results in changes in the position of the R axis. The enhanced sympathetic activity characteristic of obesity increases the risk of malignant arrhythmias via a prolongation of the QTc interval. The importance of BSPM is high because the method has a greater sensitivity than traditional ECG, and since BSPM is evaluated by computer, it is now cheap and cost effective; however, specific knowledge is required, and its availability is very limited in general practice.

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