BODY SURFACE POTENTIAL MAPPING IN ANIMAL EXPERIMENTS - PILOT STUDY

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Abstract: The aim of this article is to present a pilot case study in the area of body surface potential mapping. This technology was used to record data from animal experiments with the goal to find out, what information could be derived from recorded ECGs in the protocol including induced myocardial ischemia. Data recorded before and after the ischemia were compared. Differences in ECG beat morphology were observed, also local changes in analysed intervals (QT, ST, QS, PT) were visible. It was hypothesiced that QT interval will be prolonged after myocardial ischemia. Nevertheless, according to out pilot study this hypothesis was declined.

Keywords: ECG, BSPM, body surface potential mapping, pilot study, animal experiment, myocardial infarction, ischemia

1 INTRODUCTION

The usage of classical 12-lead electrocardiogram (ECG) has become an absolute standard examination for patients with potential cardiac malfunction. Also, ECG signal processing discipline could profit from additional information source. 12-lead ECG could not be optimal for diagnostics of some kinds of syndromes and arrhythmias, such as acute coronary syndromes (acute cardiac ischemia, myocardial infarction), since the coverage of the standard pre-cordial leads over the thorax is limited [2]. Body Surface Potential Mapping (BSPM) is one of additional methods used for further understanding of heart function. The method itself is based on multi-lead (multi-channel) measurement of the electric heart action from much broader portion of the torso [1].

Basis of the BSPM method lays in the usage of the grid of multiple unipolar electrodes. Standard amount of electrodes in the set is 32 and more (up to more than 100). The setup of electrodes could be different with every application. The system generates simultaneous recordings [3].

Since the classical ECG measurement provides us with information mainly about the voltage variation in time at a given site, BSPM method adds spatial as well as the temporal and amplitude components of cardiac electrical activity. The added spatial information provides us with sensitive localization of the electrical events (local conduction disturbances, regional heterogeneities of ventricular recovery) and understanding of the cardiac electrical activation [4].

BSPM imaging method is used also to unveil various mechanism which produce arrhythmias. Identification of signs of susceptibility to arrhythmias and the site of their origin can be absolutely crucial for diagnostics. Localising the site of the origin of ectopic ventricular activation in patients with structurally normal heart and with myocardial infarction can be listed as an example. In this case, BSPM is used for enhancing the spatial resolution of ventricular activation abnormalities. Therefore, we can better understand the genesis of such arrhythmia.

BSPM method is used for evaluation of atrial fibrillation and guidance for it's ablation, treatment of heart failure or is useful in the assessment of biventricular pacing. It is also able to separate the activation of the right and left chambers.

Practical outcome of BSPM are maps of different character made up from measured signals transformed into colored scales. Created maps can be e.g. immediate potential maps, integral maps (cumulation amplitude), isochronic maps or differential maps. Isochronic maps from BSPM can be used to clarify the possible mechanisms responsible for the great number of patients non-responsive to cardiac resynchronization therapy and the prediction of response to such therapy.
BSPM and ECG imaging in general has a huge role in understanding of arrhythmias and conduction disturbances. It's main advantage lays in being non-invasive and it's potential future in emergency department.

2 EXPERIMENT AND IT'S BACKGROUND

Proposed pilot study case was added as an improvement to ongoing series of animal experiments held on domestic female pigs. In the original study 24 juvenile adult pigs weighting 25-30 kg were listed. Animals were sedated using IM 8 mg/ kg ketamine and 0.1 mg/kg medetodimine and anaesthetized using a 10 mg/kg/h continuous IV infusion of 2% propofol. Pigs were monitored by ECG and were mechanically ventilated with 50% oxygen. To prevent lifethreatening arrhythmias and thrombosis, the animals were pre-treated with intravenous amiodarone (150 mg), lidocaine (30 mg) and heparin (3000 IU). Myocardial infarction was induced by inflating an angioplasty balloon in the mid left anterior descending coronary artery in anaesthetized pigs. After 90 min, the balloon was deflated, and the restoration of normal coronary flow was documented by angiography.
A 7F sheath was inserted into the right femoral artery to monitor blood pressure and access the left anterior descending coronary artery (LAD), through which a 7F Amplatz Left 0.75 catheter was selectively positioned. A standard hydrophilic angioplasty wire was placed in the distal LAD and an over-the-wire 2.5 x 15 mm angioplasty balloon was inflated at 6 atm in the mid-LAD segment distal to the first diagonal branch in order to provoke acute myocardial infarction (MI). Total coronary artery occlusion was confirmed by ST-segment elevation on the ECG and angiography.
The experimental procedure was approved by the Animal Care and Use Ethics Committees of the University of Valencia and the INCLIVA Biomedical Research Institute, and complies with the Guide for the Care and Use of Laboratory Animals published by the United States National Institutes of Health (NIH Publication No. 8523, revised 1996) and with the ARRIVE guidelines.

3 PILOT STUDY

The presented pilot case study was performed as an additional part to the previously described series of experiments. The aim of the study was to describe the potentially useful information from BSPM signal from recording of pigs torso while inducing acute myocardial infarction.
BSPM signal was recorded with the grid of 32 unipolar electrodes (4 strips by 8 electrodes, by BioSemi, Figure 1) with the sampling frequency of 2048 Hz. Besides 32 unipolar leads, 3 bipolar limb leads (aVR, aVL and aVF) were recorded. Data was recorded from 3 phases of the experiment - before, during and after the ischemia.

Data for the pilot study was recorded from 1 pig for which all 3 phases were recorded.
BSPM colorized maps were created for 4 chosen intervals: PT, QS, ST, and mainly QT. According to previous studies, QT interval prolongation is associated with heart ischemia [5]. Also, standard 12-lead ECG recordings differ in the QT prolongation positive confirmation. Therefore, BSPM recordings are used to add information for diagnosis [6].
The proposed signal outcomes were preprocessed by subtracting Wilson terminal lead and segmented into segments corresponding to proposed experiment protocol.

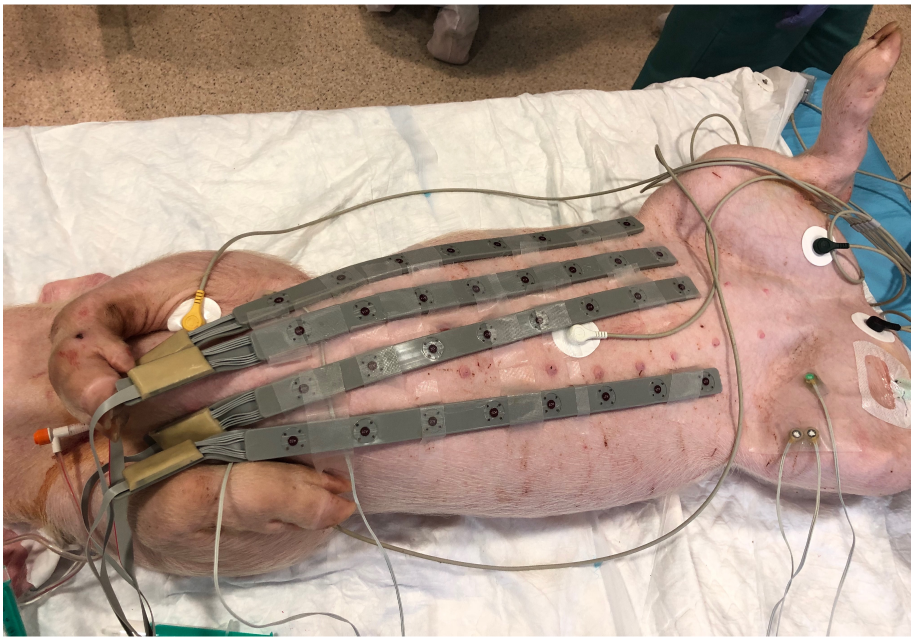

Figure 1: Setup of the electrodes while recording BSPM signals.

4 RESULTS AND DISCUSION

Because of this study being a pilot, presented results are meant to be judged more as tentative. For further statistical confirmation of suggested hypothesis the number of subjects, that went through all 3 phases of the experiment, needs to be significantly higher.
Because of the amount of used recording channels, only examples of graphical outcomes will be presented.
In Figure 2, we can see the development of the beat morphology during the infarct induction by inflated angioplasty balloon. Every signal represents 1 beat from 1 minute long intervals. Inbetween those intervals were periodic 5 minutes breaks. Therefore, this figure represents circa 40 minutes of induced ischemia.

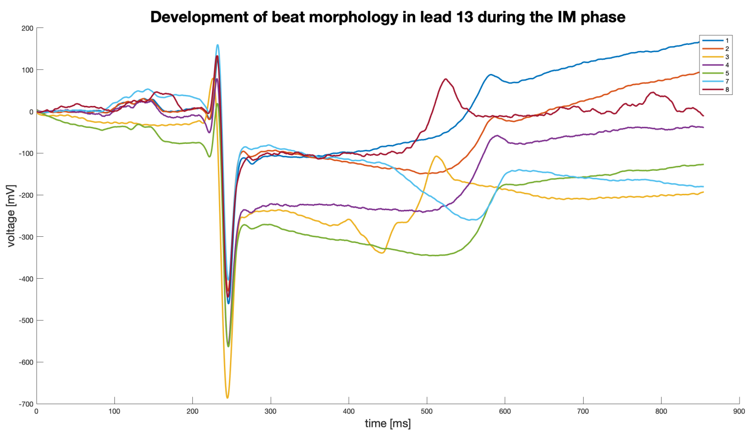

Figure 2: Development of ECG beat morphology during ischemia phase in time. Numbered signals have always 5 minutes interval in between them.

In Figure 3, we can observe an obvious difference in between ECG signal morphology. Displayed lead recorded activity at the border of medial and lateral left superior part of torso, where upper left part of heart was situated (atrium + upper part of ventriculus). It can be very clearly seen that after induced ischemia there is present a blockade in the electrical activity of the heart, which is represented with the flat state between P wave (connected tightly to the previous T wave) and Q wave.

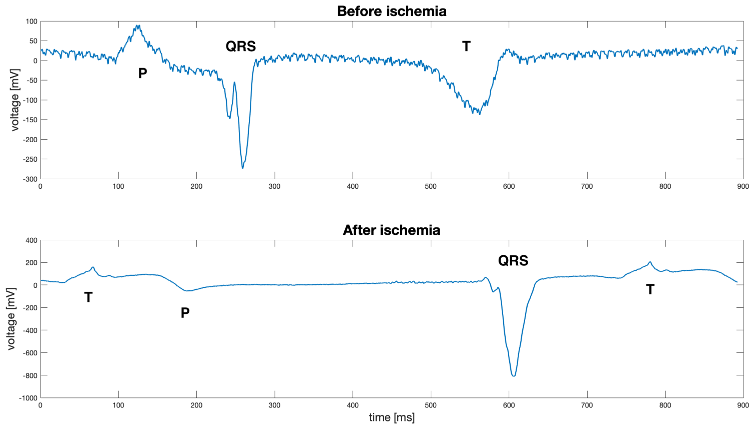

Figure 3: Comparison of ECG morphology between the phase before and after ischemia.

In Figure 4 4 colorized maps from all BSPM unipolar leads (front view on the recorded torso) are shown. On the left side, phase before the infarction is represented, on the right then phase after the infarction. From the color scales, it can be seen, that QT interval was shortened after the ischemia. This founding does not correlates with the hypothesis, that after ischemia, the QT interval is prolonged. Both prolonged and shortened QT intervals are associated with malignant ventriculus arrhythmias causing sudden death [7]. With the help of BSPM recordings implemented e.q. to sport clothing, sudden death could be avoided in active athletes.
In the cases of other intervals the results were following: QS and PT intervals were prolonged, ST interval shortened. These are associated with slower ventricular conduction, which could cause firts-degree heart block. Because of practical reasons, only QT interval comparison is visually represented in this article. Nevertheless, it must be reminded, that this study is only pilot which will not provide any generally applicable outcome.
When two-sample t-test was applied to QT interval data, the null hypothesis, that the data from before and after phase of the ischemia come from independent random samples with normal distribution, was confirmed at the significance level of 0,05 and with p-value lower than 0,001 (with the same outcome for the rest of the intervals QS, ST and PT).

5 CONCLUSION

The aim of this article was to present the result of a pilot case study performed on pigs. Recorded data were gained while induced infarction experiments in anaesthetized pigs. Infarction was by inflating an angioplasty balloon in the mid left anterior descending coronary artery. From recorded data, several outcomes were derived, most of them of comparative nature. ECG beat morphology difference between phase before ischemia and phase after ischemia was observed. Also visualisation of time development of ECG morphology from 1 of the 32 unipolar leads were presented. The hypothesis about the QT interval prolongation after heart ischemia was declined for one presented subject.
For further and more accurate information derived from BSPM measurement more subjects (animals) need to be involved in the study and compared.

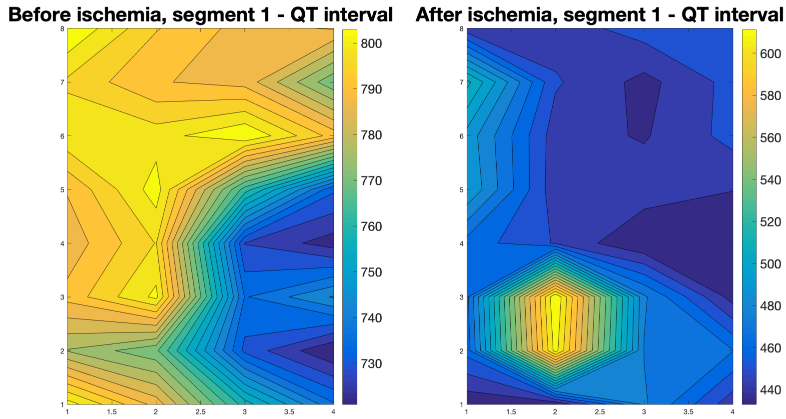

Figure 4: Comparison of QT interval between the phase immediately before and after ischemia.

Nevertheless, the usage of BSPM in clinical area when setting diagnosis is promising. Also, the potential in using such a technology in wearable technologies is possible. The example of such usage could be a prediction of sudden death in active athletes.

ACKNOWLEDGEMENT

I would like to express my appreciation to prof. Jose Millet from Polytechnic University of Valencia and the members of medical team from University Hospital of Valencia for the opportunity to personally witness the experiments, which were the subject of this pilot case study.

REFERENCES

[1] M. J. Daly et al., Epicardial potentials computed from the body surface potential map using inverse electrocardiography and an individualised torso model improve sensitivity for acute myocardial infarction diagnosis, European Heart Journal: Acute Cardiovascular Care, vol. 6, no. 8, pp. 728-735, Dec. 2017.

[2] V. Garcia-Bustos et al., Changes in the spatial distribution of the Purkinje network after acute myocardial infarction in the pig, PLOS ONE, vol. 14, no. 2, p. e0212096, Feb. 2019.

[3] L. D. Ambroggi and A. D. Corlan, Clinical use of body surface potential mapping in cardiac arrhythmias, p. 3.

[4] H. J. Bruns et al., Does body surface potential mapping (bspm) predict functional recovery in chronic ischemic myocardium after revascularization?, in Computers in Cardiology, 2005, Lyon, France, 2005, pp. 207-210.

[5] J. Jimd'ż˝nez-Candil and C. M. Luengo, QT Interval and Acute Myocardial Ischemia: Past Promises, New Evidences, p. 3.

[6] G. Tomassoni, E. Pisand'ż˝, L. Gardner, M. W. Krucoff, and A. Natale, QT prolongation and dispersion in myocardial ischemia and infarction, Journal of Electrocardiology, vol. 30, pp. 187-190, Jan. 1998.

[7] G. Tse, Y. W. F. Chan, W. Keung, and B. P. Yan, Electrophysiological mechanisms of long and short QT syndromes, IJC Heart & Vasculature, vol. 14, pp. 8?13, Mar. 2017.