Introduction

The ECG is a fast and efficient procedure for initial diagnosis of cardiac dysfunction, although it has seen little in the way of development for several decades and cannot provide detailed quantitative information about regional cardiac activity. The diagnostic capability of an ECG can be improved by increasing the number of leads used to sample the cardiac electric field since this increases the amount of information available about the electrical source.

Our aim was to develop a non-invasive quantitative measurement system that electrically images the heart with high spatio-temporal resolution. To achieve this we 1) concurrently mapped the electrical activity from the epicardium and body surface and 2) developed a visualisation system to process and display the recorded signals. To test the sensitivity and robustness of the visualisation system, regional ventricular ischaemia was induced in an anaesthetised pig.

Methods

A young 29 kg domestic pig was anaesthetised (using halothane in oxygen for induction and 100 mg.kg\(^{-1}\) i.v. for maintenance), artificially ventilated and thoracotomised. A suture snare was fitted to the equatorial region of the left anterior descending (LAD) coronary artery. An electrode sock containing 127 stainless steel contact electrodes with an inter-electrode spacing of ca. 7 mm was then placed over the epicardium with a known orientation. The chest was re-closed and an elasticated vest containing 256 Ag-AgCl electrodes with an inter-electrode spacing of ca. 15 mm was fitted. The suture snare was passed out of the chest cavity. Simultaneous body surface epicardial potentials were recorded at 20 s intervals during a four minute period of LAD occlusion followed by a period of reperfusion. Data were sampled at 2 kHz using a UnEmap data acquisition system and visualised using an anatomically accurate computational model of the epicardium obtained from 3D echocardiography. Body surface signals were visualised using a customisation of a porcine model obtained from computational tomography scans.

Heart electrode sock

(a) Heart signals were recorded using an elasticated sock with 127 electrodes connected to a UnEmap cardiac mapping system. A single heart cycle was identified and the epicardial activation time for each electrode is determined using the most negative electrocardiographical slope. An epicardial activation map was fitted to the electrode activation times and displayed using the 2D Hammer projection, where the left ventricle (LV) makes up the central portion of the projection, the right ventricle (RV) comprises the border regions and the apex was retained as a single point. The activation maps were further visualised on a 3D heart obtained from 3D echocardiography.

Epicardial Activation

(b) Torso signals were recorded using 256 electrodes sewn into an elasticated vest. A potential marker identifies the peak R wave at 0 s and 300 s. The body surface potential maps were displayed using a 2D representation of the body surface. The centre of the display reflects the left mid-axillary line and the left and right edges of the display reflect the right mid-axillary line. The body surface maps were visualised on a 3D anatomically realistic porcine torso model obtained from computational tomography.

Conclusion

During ischaemia propagation of electrical activation was progressively slowed across the ischaemic region. After 240 s of regional ventricular ischaemia, the time for total ventricular activation increased from 17 ms to 153 ms. The body surface potential maps showed a corresponding area of ST segment elevation, which was also present in Lead V\(_1\) ECG, whilst the Lead II ECG remained relatively unchanged. The electrical activation sequence had virtually recovered after 60 s reperfusion.