EVOLUTION OF CARDIAC ACTIVATION AND BODY SURFACE ELECTROPOTENTIAL CHANGES DURING VENTRICULAR ISCHAEMIA

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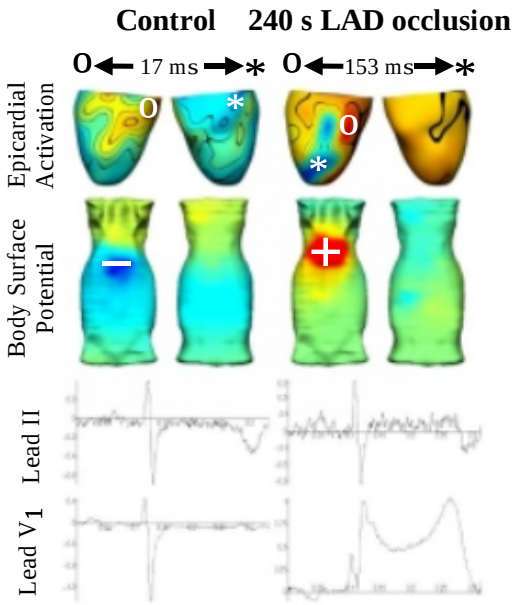
We aimed to characterise the changes in body surface and epicardial electropotentials during regional ventricular ischaemia. A young 29 kg domestic pig was anaesthetised, artificially ventilated and thoracotamised. A suture snare was used to ligate the left anterior descending (LAD) coronary artery mid-anteriorly. An elasticated sock containing 127 unipolar stainless steel contact electrodes (inter-electrode spacing approximately 7 mm) was then placed over the epicardium. The chest was re-closed and the electrode wires and ligature were passed out of the chest cavity. A vest containing 256 ECG electrodes (inter-electrode spacing approximately 15 mm) was then fitted to the torso. Simultaneous epicardial activation and body surface potential maps (BSPMs) 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 aquisition system and visualised using anatomically accurate computational models of the ventricular epicardium (obtained using 3D echocardiography) and the porcine thorax (obtained by customising a generic thorax model, which was derived from computed tomography imaging). Evolution of the changes in electrical activity brought about by ventricular ischaemia was followed using animated maps (see (1) for electrical mapping methods).



LAD occlusion caused the propagation of electrical excitation to progressively slow across the ischaemic region. Significant differences in the ventricular activation sequence, BSPMs and Lead V_1 ECG were observed after one minute of LAD occlusion, with the ischaemic zone being the last area activated. After 240 s of regional ischaemia, the time for total ventricular epicardial activation increased from 17 ms (control) to 153 ms, as illustrated in Figure 1. The BSPMs highlighted a corresponding area of ST segment elevation on the chest that was also evident in the Lead V_1 ECG, whilst Lead II remained relatively unchanged. The electrical activation sequence had recovered after 60 s reperfusion, but the repolarisation sequence was not restored until after six minutes of reperfusion. We conclude that the interpretation of high spatio-temporal resolution body surface recordings using a computational framework can detect cardiac ischaemia that is not always identifiable using standard ECG limb leads.

Figure 1: Epicardial activation, BSPM (at peak T) and ECG (Leads II and V_1) for control and 240 s LAD occlusion. Circle (o) and star (*) represent regions of earliest (red) and latest (blue) ventricular epicardial activation, respectively. Plus (+) and minus (-) highlight regions of positive (red) and negative (blue) torso electropotential, respectively. See `http://paterson.physiol.ox.ac.uk/CardiacMapping/IschaemiaStudy/` for temporal animations.

1. Nash MP, Thornton JM, Sears CE, Varghese A, O'Neill M, and Paterson DJ. Ventricular activation during sympathetic imbalance and its computational reconstruction. J Appl Physiol 90(1): 287-298, 2001.

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