

Spatio-temporal evolution of electrical activity recorded concurrently from the epicardium and body surface during cardiac ischaemia in the anaesthetised pig.

C. P. Bradley, M. P. Nash, and D. J. Paterson

University Laboratory of Physiology, University of Oxford, Parks Road, Oxford OX1 3PT, U.K.

We evaluated the spatio-temporal sequence of concurrent changes in body surface and epicardial potentials during regional ventricular ischaemia. A young 29 kg domestic pig was anaesthetised (using halothane in oxygen for induction and $100 \text{ mg} \cdot \text{kg}^{-1}$ α -chloralose *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. The chest was re-closed and an elasticated vest containing 256 ECG 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 and 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. The body surface signals were visualised using a customisation of a porcine model obtained from computational tomography scans.

During ischaemia propagation of electrical activation was progressively slowed across the ischaemic region. After 240 s of regional ischaemia, the time for total ventricular activation increased from 17 ms to 153 ms as shown in Figure 1 (see Nash *et al.* 2001 for details on hammer activation maps). 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 recovered after 60 s reperfusion. We conclude that high resolution spatio-temporal recordings can detect cardiac ischaemia that is not always identifiable using standard electrocardiographic limb leads.

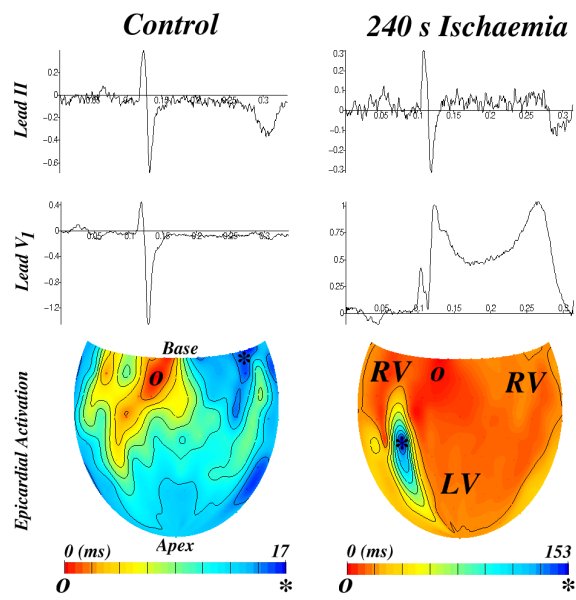
Figure 1: Control and 240 s ischaemia ECG leads II and V_1 and epicardial activation hammer maps. Circle (o) denotes regions of earliest epicardial activation and star (*) denotes regions of latest activation. See http://paterson.physiol.ox.ac.uk/CardiacMapping/PhysSoc_Oxford2001 for animations.

References:

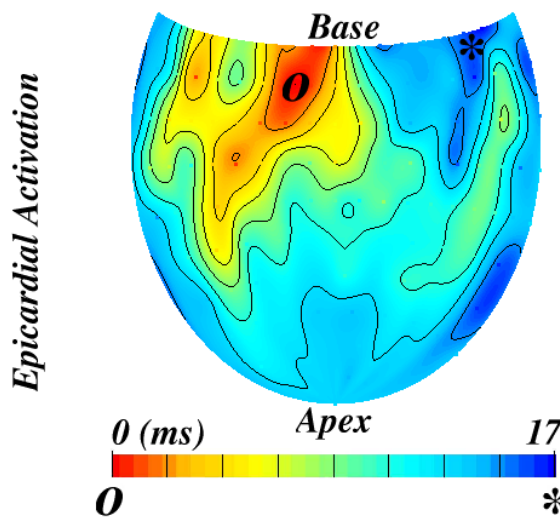
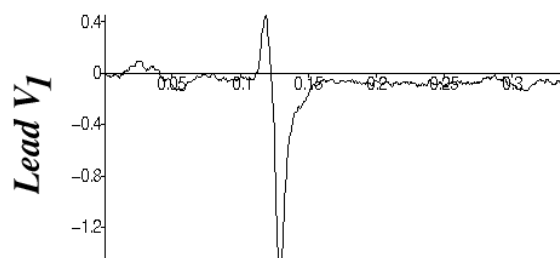
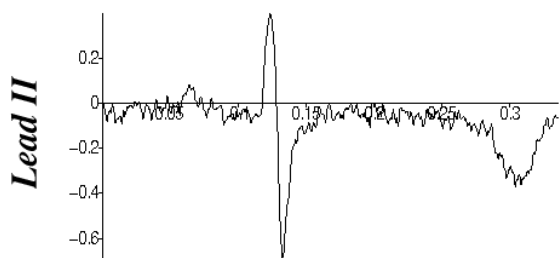
Nash, M.P., Thornton, J.M., Sears, C.E., Varghese, A., O'Neill, M. & Paterson, D.J. (2001). Ventricular activation during sympathetic imbalance and its computational reconstruction. *J Appl Physiol* **90**, 287-289.

Acknowledgements:

We are grateful to the Wellcome Trust and the British Heart Foundation for financial assistance.



Control



240 s Ischaemia

