Evolution of Cardiac and Body Surface ECG Changes During Ventricular Pacing and Regional Ischaemia

Martyn Nash, Chris Bradley and David Paterson
University Laboratory of Physiology, Parks Road, Oxford OX1 3PT, U.K.

Introduction

The standard 12 lead ECG is a fast and efficient measure to diagnose abnormalities in cardiac electrical activity and function, however, the spatial and temporal resolution is rather limited. We have developed an integrated experimental and computational analysis system to facilitate the interpretation of electrocardiac activity during control and pathological conditions [1].

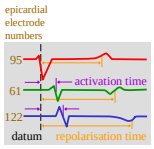
Objective: To simultaneously sample dense arrays of ventricular epicardial and body surface ECGs during (i) abnormal ventricular activation, and (ii) regional myocardial ischaemia; and to interpret the signals using an anatomical framework, in order to correlate the body surface recordings with the underlying electrocardiac activity.

Surgery and Mapping

- domestic pigs were anaesthetised with α-chloralose (100 mg/kg i.v.), ventilated, thoracotomised and pericardectomised. The torso surface was shaved and washed.
- core temperature, fluid balance (*ca.* 100 ml/hr saline) and arterial blood gases were all maintained, while arterial blood pressure and heart rate were monitored.
- unipolar torso and ventricular electropotentials were simultaneously recorded (sampling rate 2 kHz) using a 448 channel UnEmap cardiac mapping system.
- ventricular epicardial signals were recorded using an elasticated electrode sock with 127 stainless steel electrodes (inter-electrode spacing *ca.* 5 mm).
- body surface potential signals were recorded using an elasticated electrode vest containing 256 ECG electrodes (inter-electrode spacing *ca.* 15 mm). This was also performed prior to thoracotomy, as a non-invasive control.
- the ventricular signal analysis and epicardial activation mapping procedure are fully described in [2]. Torso mapping and anatomical model development techniques are detailed in [1].

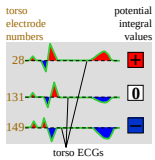
Epicardial Analysis

Epicardial activation times are computed using the minimum slope of the QRS complex. Repolarisation times are taken from the maximum slope of the T-wave. Red/blue denote earliest/latest epicardial activation or repolarisation, respectively.



Torso Integral Analysis

Torso electrode signals are integrated over a selected interval (*eg.* the QRS interval, or a full cardiac cycle) and an ECG integral field is fitted to a customised anatomical torso model. Red/blue denote positive/negative potential integral values, respectively.



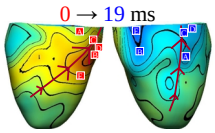
Ventricular Epicardial Pacing

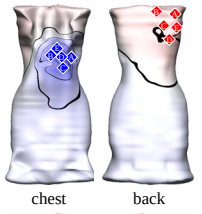
Aim: To examine the effects of abnormal cardiac activation on the body surface ECG recordings.

Protocol: Stimulus amplitude 10 V; pulse width 2 ms; rate approximately 25 pulses/min above baseline heart rate (156 ± 28 bpm). Data shown are mean ± SD (n=5).

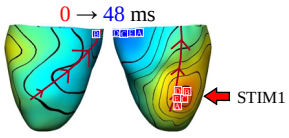
Results: Posterior epicardial pacing (STIM1) increased the dispersion of epicardial activation from 19 ± 2 ms (control) to 48 ± 7 ms ($p<0.01$) and decreased the arterial blood pressure by approximately 40/25 mmHg (control 120/80 mmHg). Activation dispersion for anterior epicardial pacing (STIM2) was 51 ± 4 ms ($p<0.01$ compared to control) and the drop in arterial blood pressure was approximately 45/20 mmHg. Epicardial activation and torso ECG integral maps show averaged data, with individual observations indicated for earliest (▲) and latest (▲) epicardial activation, and positive (◆) and negative (◆) extrema of the torso ECG integral.

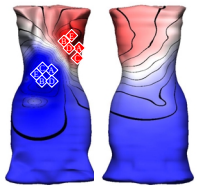
Control Activation Sequence



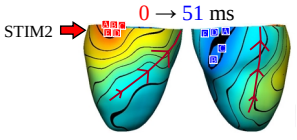


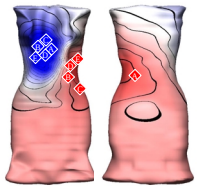
Posterior Apical Pacing





Anterior Basal Pacing





Ventricular Epicardial Activation
Earliest → Latest

Torso ECG Integral (QRS interval)
-0.01 → +0.01 mVs

Regional Ventricular Ischaemia

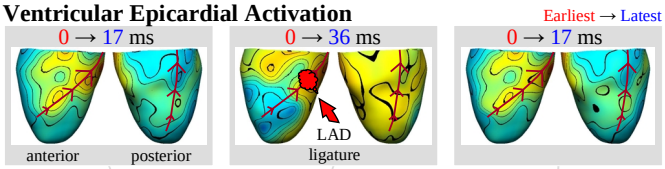
Aim: To investigate the effects of left anterior descending (LAD) coronary artery occlusion on the ventricular electropotential activity and concurrent body surface ECG patterns.

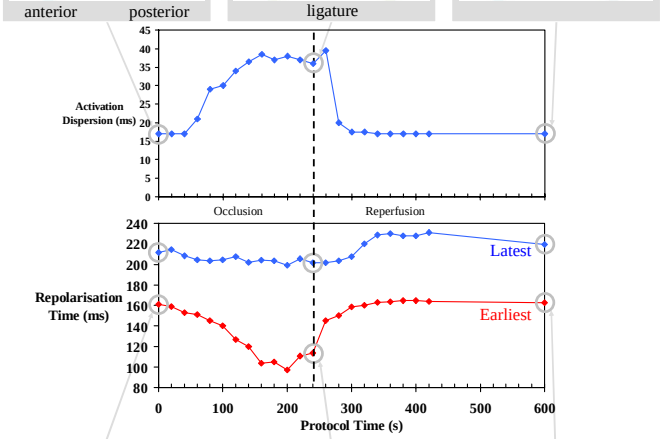
Protocol: The LAD was ligated proximally and occluded for 4 minutes. Epicardial and torso electropotentials were sampled simultaneously at 20 s intervals.

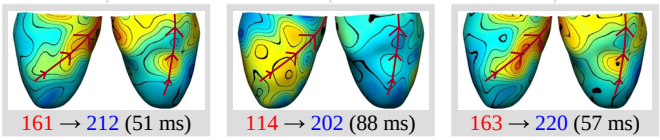
Ventricular Mapping Results: ECG activity was largely unchanged during the first 40 s of LAD occlusion. Subsequently, dispersions of epicardial activation and repolarisation progressively increased during the occlusion, corresponding to the slowing of excitation propagation and shortening of action potential duration throughout the ischaemic tissue.

Ventricular Epicardial Activation

Earliest → Latest





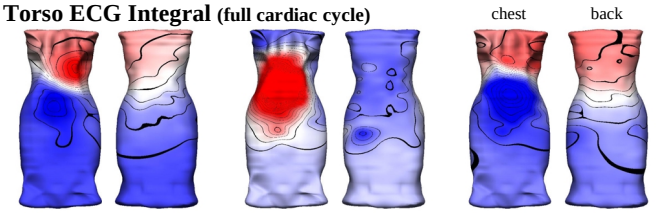


Ventricular Epicardial Repolarisation

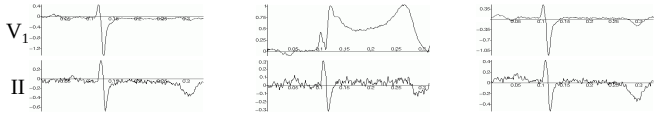
Earliest → Latest (Dispersion)

Body Surface Mapping Results: In relation to the changes in epicardial ECG activity, the concurrent torso integral maps highlighted a localised region of positivity on the chest that increased in area during the LAD occlusion, which correlated with an elevating ST segment of the ECG Lead V_1. Interestingly, the ECG Lead II remained comparatively unchanged during the entire protocol. Following release of the occlusion, the epicardial activation sequence rapidly recovered and all ECG activity was fully restored to the control state after six minutes of reperfusion.

Torso ECG Integral (full cardiac cycle)



Standard ECG Leads



Summary

- Abnormal ventricular electrocardiac activity was readily detected using non-invasive body surface integral mapping. This approach may prove useful for the identification of sites of abnormal automaticity or ectopic ventricular activity.
- Interpretation of high spatio-temporal resolution body surface recordings using a anatomical-computational framework can identify cardiac ischaemia that is not always detectable using standard ECG limb leads.

References

[1] MP Nash, CP Bradley, A Kardos, AJ Pullan & DJ Paterson. An experimental model to correlate simultaneous body surface and epicardial electropotential recordings in-vivo. *Chaos, Solitons and Fractals*, **12** (in press), 2001.

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

Further information and links to references are available at: http://paterson.physiol.ox.ac.uk/ECGmapping/

Acknowledgements

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