Imaging Electrocardiac Dispersion During Ischaemia.

Simultaneous *in-vivo* body surface and epicardial measures.

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1. Introduction

The standard 12 lead ECG is a fast and efficient measure to diagnose abnormalities in cardiac electrical activity and function, however, the spatial 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].

Objectives:
(1) To simultaneously sample dense arrays of ventricular epicardial and body surface ECGs during regional myocardial ischaemia and to interpret the signals using an anatomical framework.
(2) To test whether body surface measures could accurately detect heterogeneity of ventricular repolarisation.

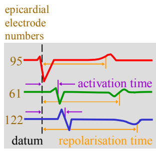
[Results for one case study are illustrated, but trends were similar for all studies.]

2. Methods

Surgery and Mapping:

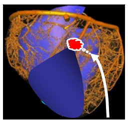
- domestic pigs were anaesthetised with α-chloralose (100 mg/kg i.v.), ventilated, thoracotomised and pericardectomised. The torso surface was shaved and washed.
- core temperature, fluid balance (*ca.* 100 ml/hr saline) and arterial blood gases were all maintained, while arterial blood pressure and 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].

Signal 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.



Regional Ventricular Ischaemia:

The left anterior descending (LAD) coronary artery was ligated with a suture snare, which was passed out of the re-closed chest. Controlled periods of LAD occlusion and reperfusion were induced using the snare.


controllable suture snare

3. Ventricular Activation and Repolarisation

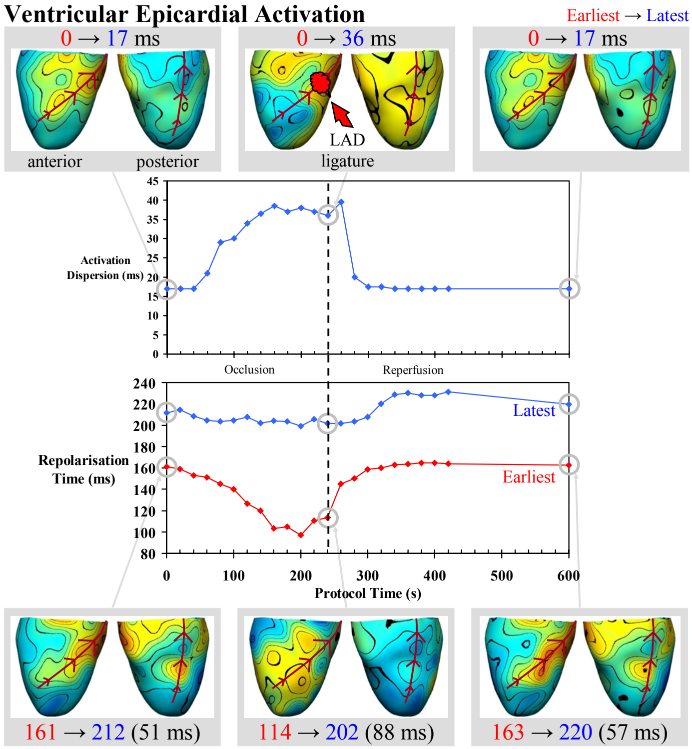
Aim: To investigate the effects of left anterior descending (LAD) coronary artery occlusion on the ventricular electropotential activity and concurrent body surface ECG patterns.

Protocol: The LAD was ligated proximally and occluded for 2-4 minutes. Epicardial and torso electropotentials were sampled simultaneously at 20 s intervals.

Case Study - 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)

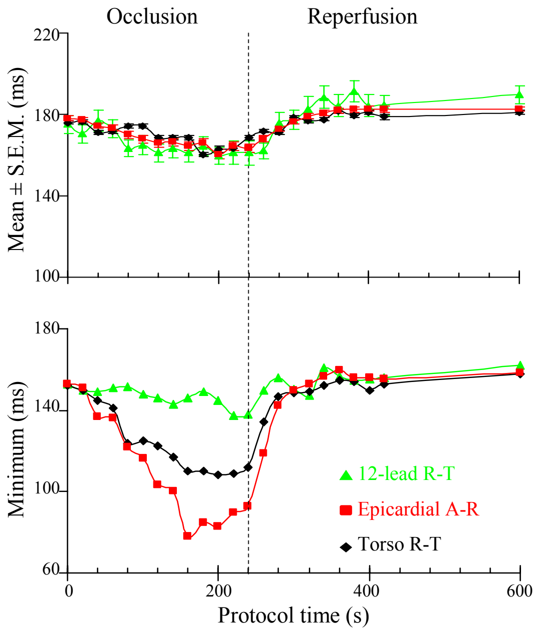
4. Activation-Recovery and R-T Intervals

Aim: To determine whether high density torso mapping could identify changes in ventricular dispersion of repolarisation and to compare the sensitivity with respect to indices derived from the 12-lead ECG.

Analysis: Epicardial activation-recovery (A-R) intervals were calculated from the activation and repolarisation data. Torso and 12-lead R-T intervals were compared to the ventricular data.

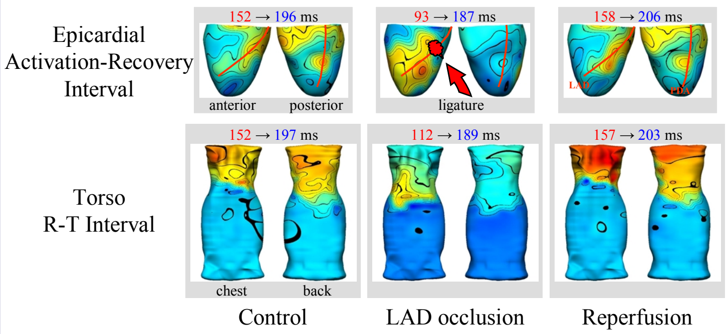
Case Study Results:

1) During LAD occlusion and reperfusion, the mean epicardial A-R intervals (n=113) showed no significant differences ($p<0.05$, paired t-test) compared to control values. Mean R-T intervals derived from the 12-lead ECG (n=12), or high density torso mapping (n=225) showed similar trends to the epicardial data.
2) The minimum epicardial A-R interval dramatically decreased during LAD occlusion with a graded response and the minimum torso R-T interval showed a similar trend. However, the minimum R-T interval calculated from the 12-lead ECG was not sensitive to the myocardial changes.



5. Dispersion Mapping

Case Study Results: Four minutes of LAD occlusion caused ventricular epicardial A-R intervals to decrease in a localised (red) region distal to the suture snare, consistent with shortened action potential duration during ischaemia. This was associated with a localised area of decreased torso R-T intervals on the chest, proximal to the ischaemia myocardium. Dispersion indices returned to their control state following reperfusion.



6. Conclusion

- Interpretation of high spatio-temporal resolution body surface recordings using a anatomical-computational framework can identify changes in the dispersion of ventricular repolarisation due to localised myocardial ischaemia that are not detectable using indices derived from the standard 12-lead ECG.

7. 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,* **13**:1735-1742, 2002.

[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/

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