

The Effect of Coronary Occlusion on the Electrophysiological Function of the Heart

Liam Kampshof Supervised by : Prof Martyn Nash¹, Dr Jichao Zhao¹, A.Prof Peter Larsen²

¹ The University of Auckland ² The University of Otago (Wellington)

Background

Understanding how acute ischaemia develops in the heart and how it alters ventricular electrophysiological function is important in helping us understand disorders that occur during a heart attack. The aim of this research is to improve this understanding by analysing the progression of several measures of electrophysiological activity during localised cardiac ischaemia.

Data was collected using the Ensite NavX system (St. Jude Medical, Minnesota) to obtain high resolution, unipolar signals from the endocardial wall of eight normal sheep. A non-contact multi-electrode balloon was inserted into the left ventricle (LV). The left anterior descending artery (LAD) was occluded and electrograms were recorded as localised myocardial ischaemia developed

Activation Times

Method

The negative peak of the first temporal derivative is an accepted indicator of the activation time of a region of myocardium^{1,2} (See Fig. 1). Activation times (AT) were expressed relative to the earliest AT.

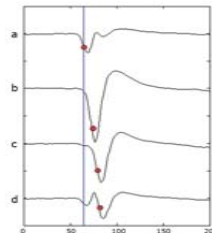


Fig. 1 AT detection. Red dot is AT. Blue datum is the earliest AT.

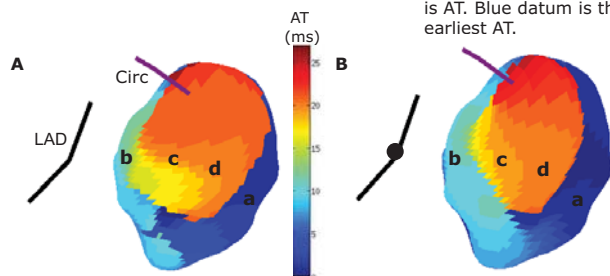


Fig. 2 Activation sequence of the left ventricle, (A), during sinus rhythm and, (B), 4 minutes after occlusion of the left anterior descending artery (LAD). Circ: Circumflex artery. Black circle represents LAD occlusion point.

Results

- There was negligible difference in the activation sequences before and after occlusion (Fig. 2)
- This may be due to the presence of the fast conducting Purkinje fibre network and the localised perfusion of the subendocardial tissue by blood in the LV cavity.

Activation – Recovery Interval (ARI)

Ischaemia is known to decrease myocardial action potential duration. ARIs can be estimated from unipolar cardiac electrical recordings by analysing the interval between excitation (activation) and repolarisation (recovery) in the tissue.³

Method

Repolarisation was detected as the most negative gradient of the T wave. Other methods proved to be ineffective with these data.

Results

A localised region of reduced ARIs (compared to pre-occlusion map) developed follow LAD occlusion (Fig. 3).

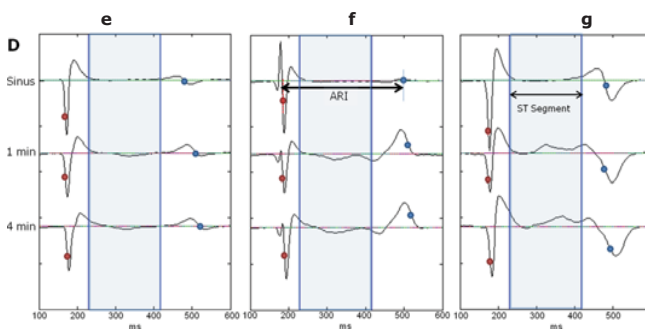
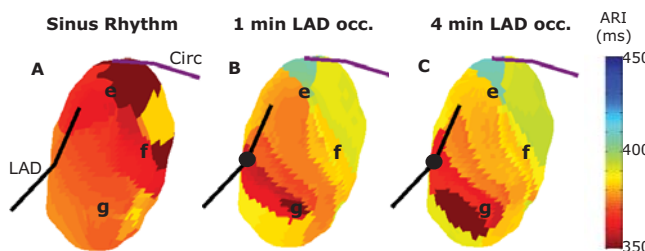


Fig. 3 ARIs, (A), sinus rhythm, (B), 1 minute post occlusion, (C), 4 minutes post occlusion, (D), Progression of signal at electrodes e-g as ischaemia develops. Blue dot indicates repolarisation. Vertical blue lines indicate the ST segment. Black circle represents LAD occlusion point.

ST Segment Elevation

Elevation of the ST segment of the electrogram is a known characteristic of ischemic cardiac tissue³. It can be characterised by the integral of the ST segment (Fig. 3D),

Method

The ST segment was detected as time between the latest S point and 50ms before the earliest T wave. The electrogram signal data was integrated over this time segment.

Results

- No ST segment elevation during sinus rhythm (Fig. 4A)
- After occlusion, there was a region of ST elevation consistent with occlusion of the LAD (Fig. 4B-C)

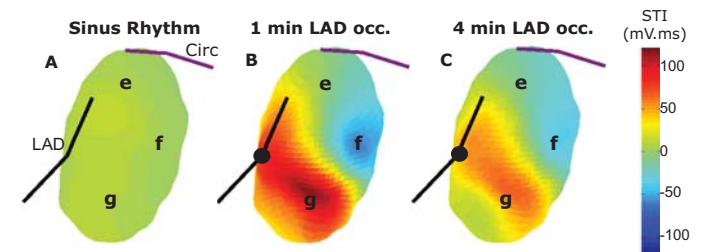


Fig. 4 ST segment integrals (shaded in Fig. 3). (A) Sinus rhythm, (B) 1 minute post occlusion, (C) 4 minutes post occlusion. Black circle represents LAD occlusion point.

Conclusion and Future Work

While AT remained relatively unchanged during LAD occlusion, the ST segment integral and ARI maps highlighted a localised region of ischaemia downstream from the occlusion site. Future work will examine sites of arrhythmia onset to see how they relate to this region.

References

1. Punske B. et al., "Spatial Methods of Epicardial Activation Time Determination in Normal Hearts". *Annals of Biomedical Engineering*, Vol. 31, pp. 781–792, 2003.
2. Lambiase P. et al., "Non-contact left ventricular endocardial mapping in cardiac resynchronisation therapy", *Heart*;90:44–51, 2004.
3. Nash M. et al., "Imaging Electrocardiographic Dispersion of Depolarization and Repolarization During Ischemia", *Circulation*;107:2257–2263, 2003.

Acknowledgments

This studentship was funded by the Cardiac Society of Australia and New Zealand. We would like to acknowledge the sheep experimental work done by Dr. Nigel Lever, A. Prof. Ian LeGrice, Prof. Bruce Small, Dr. Greg Sands and Linley Nisbet.