the emergence and growth of collateral arteries in the thigh (upstream of the ligation point).

Conclusion A pre-existing collateral circulation provides the residual blood supply after femoral ligation. A rapid increase in the diameter of a small number of collateral arteries appeared to be the major mechanism for acute restoration of blood supply to the ischaemic lower leg and foot pad. Future work will use histology and immunohistochemistry to investigate the role of angiogenesis in reperfusion following femoral ligation.

18 TOWARDS NON-INVASIVE CHARACTERISATION OF RE-ENDOTHELIALISATION AND RESTENOSIS FOLLOWING CORONARY STENTING: AN IN VITRO INVESTIGATION USING IMPEDANCE SPECTROSCOPY

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Following the permanent implantation of a coronary stent, optimal arterial wall healing is characterised by re-endothelialisation, the regrowth of a functional Endothelial Cell (EC) monolayer over the exposed stent surface, which reduces the risk of thrombosis. However restenosis, arising from the proliferation and migration of medial Smooth Muscle Cells (SMCs) can cause luminal narrowing to reoccur. Previous research has suggested that the stent itself could be used as an electrode and, when combined with non-invasive impedance spectroscopy techniques, monitor post stenting recovery. This could then inform clinicians on cell regrowth without the need for invasive imaging techniques. In this study we investigated the feasibility of this concept using two *in-vitro* models representing the cellular regrowth scenarios: re-endothelialisation and restenosis.

Primary porcine ECs and SMCs were seeded onto platinum electrodes and electrical impedance spectroscopy measurements were made for up to 10 days in the frequency range 1 KHz to 100 KHz. Endothelium function was assessed through the measurement of the impedance response of confluent EC monolayers to the addition of a gap junction enhancer, dipyridamole, or an inhibitor (heptanol or carbenoxolone).

Our results show that confluent, stent surface comparable populations of SMCs and ECs give rise to distinct impedance signatures, providing a novel method of non-invasively characterising these cell types. Gap junction inhibition of EC monolayers dose dependently reduced total impedance. Conversely dipyridamole's enhancing effect on gap junction formation caused an increase in total impedance. These novel findings show the importance of intercellular gap junction communication in maintaining EC barrier function. Our current work is focused on the translation of this technology towards *in-vivo* monitoring of in-stent restenosis and recovery of a functional endothelium.

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19 ADVERSE CARDIAC REMODELLING UNDER PRESSURE OVERLOAD. A MAGNETIC RESONANCE IMAGING STUDY IN MICE

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Pressure overload, a hallmark of valvular heart disease and hypertension, is the leading cause of heart failure. With the progressive nature of this condition a better understanding of the process underlying the transition to heart failure is vital. Recent studies suggest that interstitial myocardial fibrosis occurs early in this transition and has a profound effect on cardiac function. The recently developed T1-mapping Cardiovascular Magnetic Resonance Imaging (CMR) technique has the potential to quantify the extracellular volume fraction (ECV) and therefore evaluate the expansion of the extracellular matrix (primarily diffuse fibrosis) over time.

We aimed to assess the feasibility of CMR (including functional and ECV imaging) to monitor cardiac remodelling using an animal model of pressure overload heart disease.

Fifteen mice were subjected to a 6 week Angiotensin-II infusion (AngII). CMR (cine and T1 mapping) was performed before and following Angiotensin II infusion at 2, 4 and 6 weeks. ECV was calculated from the T1 relaxation times pre and post-contrast infusion).

Mean blood pressure increased from 65±12 (baseline) to 84±14 mmHg ($p<0.001$) and ECV increased from 24.28% ±3.35% (baseline) to 30.03%±5.34% after 2 weeks of AngII ($p=0.011$). ECV plateaued at 4 and 6 weeks and stayed significantly higher compared to baseline ($p=0.001$). Cine imaging revealed left ventricular (LV) hypertrophy during infusion which remained stable at 4 and 6 weeks. Interestingly, systolic function was maintained after 2 and 4 weeks of AngII but was impaired at six weeks (EF 56.3% compared to 64.4% at baseline and 59.8%; 60.7%, at 2 and 4 weeks ($p=0.014$). This drop in cardiac performance was accompanied by a trend towards LV dilatation at 6 weeks compared to baseline (LV end diastolic volume 68 µl vs 63 µl, $p=0.056$).

Prolonged pressure overload results in ECV expansion, LV hypertrophy and subsequent systolic dysfunction. T1 mapping CMR shows promise in monitoring this transition.

20 THE POLYPHENOL DELPHINIDIN INDUCES ANTIOXIDANT EFFECTS IN HUMAN UMBILICAL VEIN ENDOTHELIAL CELLS THROUGH ACTIVATION OF ENDOGENOUS GLUTATHIONE: IMPORTANCE OF USING RELEVANT CONCENTRATION IN IN VITRO SYSTEMS

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