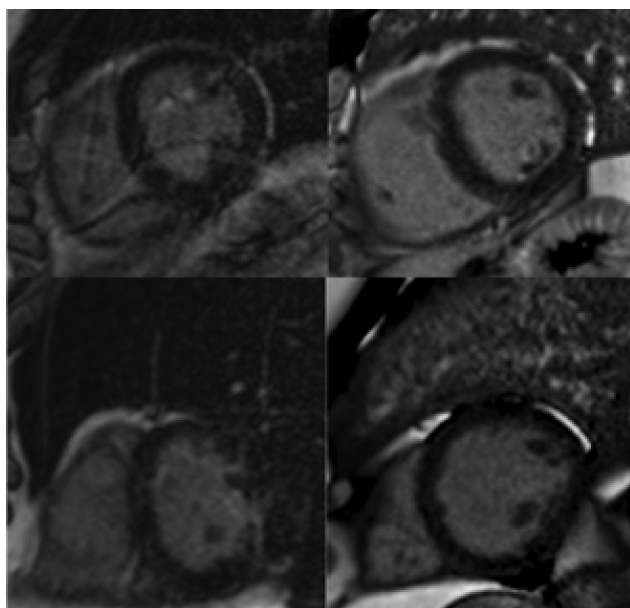


Late Gadolinium Enhancement	0	1	2	3	Maximum score
LV coverage	Full coverage	-	Apex not covered	Base or $\geq 1$ slice missing	5
Wrap around	No	1 slice	2 slices	$\geq 3$ slices	3
Respiratory Ghost	No	1 slice	2 slices	$\geq 3$ slices	3
Cardiac Ghost	No	1 slice	2 slices	$\geq 3$ slices	3
Image blurring/mis-trigger	No	1 slice	2 slices	$\geq 3$ slices	3
Metallic Artifacts	No	1 slice	2 slices	$\geq 3$ slices	3
Signal loss (coil inactive)	Activated	-	Not activated	-	2
Slice thickness	$\geq 10$ mm	11-15mm	-	$> 15$ mm	3
Inter-slice Gap	$< 3$ mm	3-4mm	-	$> 4$ mm	3
Correct LV long axis	$\geq 3$	2	1	None	3
LGE Score					31

**Abstract 013 Figure 1** Adapted Quality Scoring Method for LGE images in CMR: 10 criteria; range of scores 0 (optimal quality) to 31 (poorest quality).



**Abstract 013 Figure 2** Example images from a single patient showing bh-LGE image (left) with corresponding MOCO-LGE (right).

**Conclusion** MOCO-LGE is superior to bh-LGE in a clinical service, with better image quality, easier interpretation and faster scanning times.

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#### 014 WIDEBAND FREE BREATHING MOCO LGE CHANGES PATIENT CARE IN PATIENTS WITH IMPLANTABLE CARDIAC DEFIBRILLATORS

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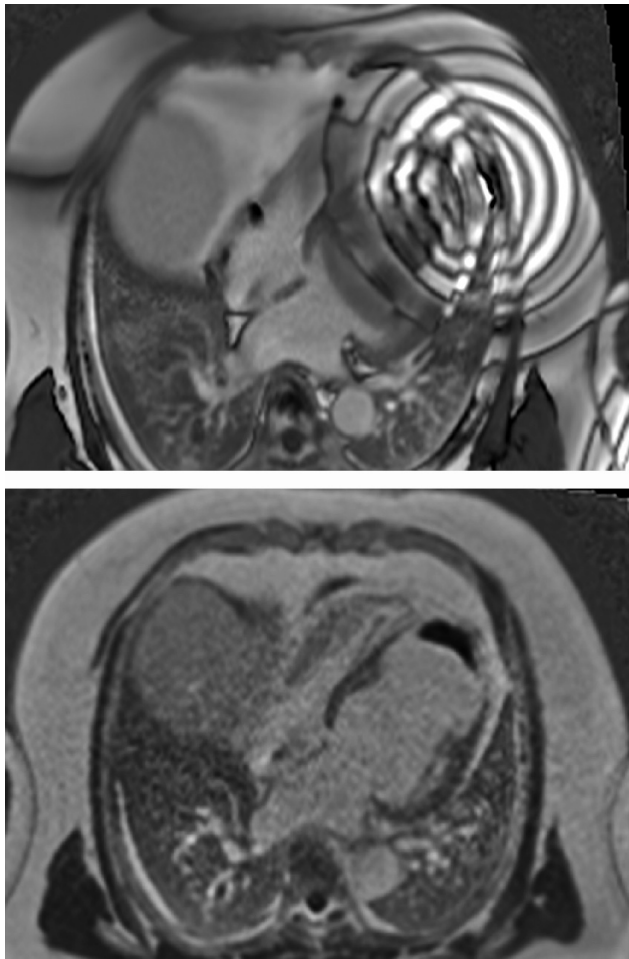
**Background** There is a growing need for CMR in patients with implanted devices, who represent a high-risk cohort. Recent reports suggest CMR may be safely performed but artefact remains a significant limitation to late gadolinium enhancement (LGE). It is still unknown whether novel sequences to reduce artefact can be used efficiently or provide clinically useful information from scar imaging.

**Methods** We used a novel free-breathing wideband MOCO sequence with PSIR (WB-MOCO) designed for a clinical environment. Patients with implantable cardiac devices (including MR non-conditional) referred clinically for CMR were scanned according to local standard operating procedure (based on national guidelines and Magnasafe Registry). WB-MOCO LGE approach was used primarily, with paired comparator conventional PSIR FLASH LGE or free breathing MOCO SSFP. Conventional and WB-MOCO LGE were assessed for artefact on a scale of 0 to 4 (0=no artefact, 4=completely obscured). A panel of three CMR cardiologists judged impact on patient care.

**Results** Of the 67 patients (age  $54 \pm 19$  years, 47 male), 17 had ICDs, 9 cardiac resynchronisation devices, 19 pacemakers and 22 implantable loop recorders (ILR).

20 (44%) pulse generators were non-conditional; 11 (16%) inpatients; 10 (15%) with AF; 7 (10%) pacing dependent. Every patient referred was scanned successfully. 10 leads had parameter changes by Magnasafe criteria with no clinical significance, and 80% normalising at follow up ( $66 \pm 40$  days).

With conventional LGE imaging, 22 (33%) scans were non-diagnostic. WB-MOCO LGE completely removed artefact in 19 (87%), and achieved diagnosis in the remaining 3. 10 (15%) patients had significant artefact on conventional LGE, which WB completely or almost completely removed.



**Abstract 014 Figure 1** Conventional LGE (le4) with device artifact and WB MOCO LGE revealing a large apical thrombus secondary to a large LAD infarct (right).

Overall, CMR with WB-MOCO LGE changed management in an additional 26 (39%) patients compared to CMR with conventional LGE (33 patients, 49%). This benefit was in 77% of defibrillators, 25% of pacemakers and 5% of the ILR groups.

**Conclusion** The WB-MOCO sequence permits high quality LGE imaging in device patients, and is robust enough for a clinical setting. This adds clinical utility particularly in

defibrillator patients, also pacemaker patients but with limited utility in ILRs. Our electrophysiology department has now incorporated this into their protocol prior to ventricular tachycardia ablation.

### 015 CLINICAL UTILITY OF T1 MAPPING IN CARDIAC ATTR AMYLOIDOSIS – DIAGNOSTIC PERFORMANCE AND PROGNOSTIC CAPABILITY

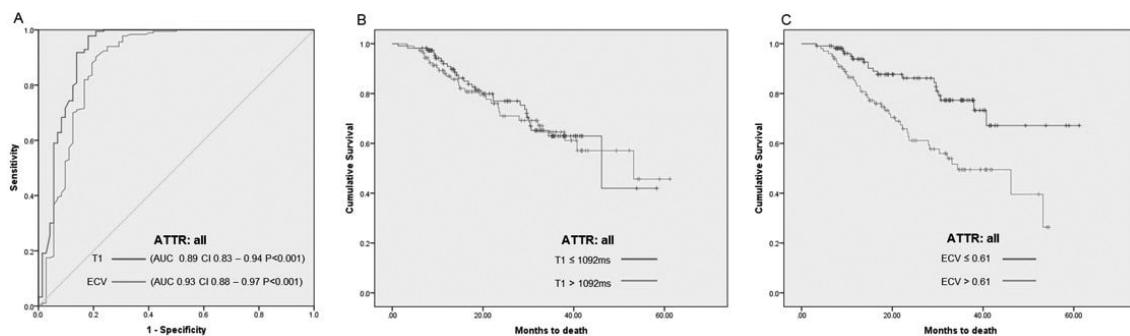
<sup>1,2</sup>K Norrington, <sup>1</sup>A Martinez-Naharro, <sup>1</sup>T Kotecha, <sup>1</sup>R Francis, <sup>1</sup>DF Hutt, <sup>1</sup>T Rezk, <sup>1</sup>C Quarta, <sup>3</sup>TA Treibel, <sup>1</sup>CJ Whelan, <sup>1</sup>D Knight, <sup>4</sup>P Kellman, <sup>5</sup>FL Ruberg, <sup>1</sup>JD Gillmore, <sup>2,3</sup>JC Moon, <sup>1</sup>PN Hawkins, <sup>1</sup>M Fontana. <sup>1</sup>National Amyloidosis Centre, University College London, Royal Free Hospital, London, UK; <sup>2</sup>Institute of Cardiovascular Science, University College London, London, UK; <sup>3</sup>Barts Heart Centre, West Smithfield, London, UK; <sup>4</sup>National Heart, Lung and Blood Institute, National Institutes of Health, Bethesda, Maryland, USA; <sup>5</sup>Amyloidosis Centre and Section of Cardiovascular Medicine, Department of Medicine, Boston University, School of Medicine, Boston Medical Centre, UK

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**Objectives** Cardiac failure caused by transthyretin amyloidosis (ATTR) is an underdiagnosed clinical entity which has an important overlapping clinical phenotype with hypertrophic cardiomyopathy (HCM). Native myocardial T1 mapping by CMR is useful for diagnosis in cardiac amyloidosis. Here, we investigate the diagnostic and prognostic value of T1 mapping in the largest ATTR population studied so far as well as patients with HCM. We aimed to: 1) assess the ability of native T1 to diagnose cardiac amyloidosis; 2) compare native T1 to extracellular volume (ECV), and; 3) stratify prognosis.

**Methods** 134 wild-type ATTR (ATTRwt) (122 males, age 76 ± 7 years), 95 mutant-type (ATTRm) (64 males, age 66 ± 12 years) and 12 mutation carriers (4 males, age 46 ± 8 years) were compared to 44 HCM patients. All subjects underwent CMR with standard SSFP-cine imaging, T1 mapping and ECV measurement. ATTR patients underwent <sup>99m</sup>Tc-DPD scintigraphy, the current diagnostic imaging reference standard for ATTR, with uptake determined by semi-quantitative score.

**Results** Native T1 and ECV were elevated in ATTR compared to HCM ( $p < 0.001$ ) (mean T1: in ATTRwt 1091 ± 52 ms, in ATTRm 1084 ± 68 ms, in HCM 1026 ± 64 ms; mean ECV: in ATTRwt 0.6 ± 0.1, in ATTRm 0.58 ± 0.2 ms, in HCM 0.38 ± 0.1 ms). No significant difference between native T1 and ECV was found between ATTRwt and ATTRm. Native T1 and ECV diagnostic performance was similar for ATTRwt and ATTRm (vs HCM: T1 AUC 0.89; ECV AUC 0.93;  $p = 0.11$ )



**Abstract 015 Figure 1** Diagnostic performance and prognostic capability. (A) Receiver operator characteristics curve (ROC) for the discrimination of possible or definite transthyretin (ATTR) cardiac amyloidosis by native T1 and ECV from HCM. Kaplan-Meier survival curves for (B) pre-contrast myocardial T1 and (C) extracellular volume at bolus. The median native myocardial T1 (1092ms) (B) and median extracellular volume (ECV) (0.61) (C) were used as the respective cut points for survival.