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311. MRI of Acute Myocardial Infarction: Evaluation of Functional Recovery

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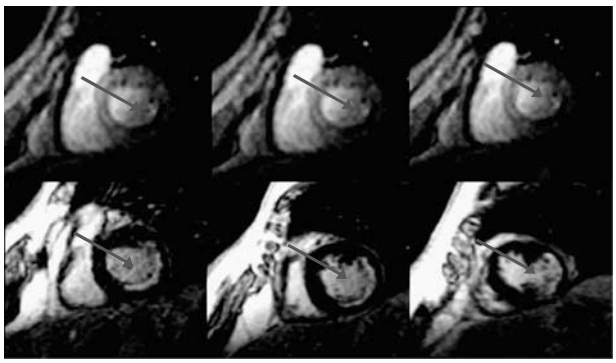
Introduction: Myocardial viability assessment is crucial in prognostic stratification and therapeutic decision making for patients after acute myocardial infarction. MRI has shown high capability in viability assessment, also compared to current clinical tools.

Purpose: To define contrast enhanced (CE) MRI role in functional recovery prediction after acute myocardial infarction.

Methods: 43 consecutive patients with first AMI (64 ± 9 yrs., 36 anterior, 7 inferior, 37 primary PTCA, 6 thrombolysis) underwent cine- and CE-MRI (GE Signa Horizon Echospeed; GE Signa LX Excite) within fifth day after onset. Cine-MRI was performed in short axis (6–8 slices, Fastcard and FIESTA sequences); first pass imaging (IR-prep FGRE and FGRE-ET with iv 0.1 mmol/kg Gd-DTPA, 3 mL/s) was obtained on three short axis slices (basal, mid-ventricular and apical); multi-slice short axis (6 slices) delayed T1 imaging (IR-prep FGRE) was obtained 20 min after Gd injection. A total amount of 731 segments were classified as: 1) normal first-pass, absent or delayed hyperenhancement; 2) hypoenhancement at first-pass, delayed hyperenhancement; 3) hypoenhancement both at first-pass and delayed imaging. Segments out of first-pass slices (total amount 774) were classified at delayed imaging as normal (= type 1), hyperenhanced (= type 2) and hypoenhanced (= type 3). Patterns 2 and 3 were considered non viable. At six months MRI assessed functional recovery.



Results: Pattern 1 was observed in 1262 segments, with functional recovery appreciated in 1195 (94.7%). Pattern 3 was present in only 31 segments, without recovery (100%). Pattern 2 was observed in 212 segments: out of them, 39 showed recovery (18%).

Conclusions: Patterns 1 and 3 respectively identify viable and non viable tissue. Pattern 2 is less specific early after AMI, as it may represent also viable myocardium and should be carefully interpreted.

312. Delayed Contrast-Enhanced MRI Using a 3D Inversion Recovery Sequence Combined with Parallel Imaging Techniques (SENSitivity Encoding-SENSE)

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Introduction: Delayed contrast enhanced MR imaging has been shown to allow assessment of myocardial viability in patients with ischemic heart disease.

Purpose: The purpose of this study was to evaluate a 3D IR sequence combined with parallel imaging techniques [SENSE] (data acquisition with coverage of entire LV within one breath hold) in comparison with a standard 2D approach (data acquisition with coverage of entire LV in multiple breath holds).

Methods: 29 patients with known or suspected infarction underwent MRI for viability assessment (Intera, 1.5 T, Philips). For the standard approach a segmented 2D IR gradient echo sequence (TR/TE 6.8/1.85, Flip angle 15°, in plane resolution 1.2 × 1.2, slice thickness 8 mm) with ten breath holds (duration 146 sec) to cover the LV in the short axis was used. The SENSE 3D approach (SENSE factor 2) consisted of a segmented 3D IR sequence (TR/TE 4.3/1.2, Flip angle 14°, in plane resolution 1.3 × 2.5, slice thickness 8 mm reconstructed to 1.3 × 1.3 × 4 mm) with coverage of the entire LV within one single breath hold (duration 14 sec). Images were obtained 10 to 30 min after the i.v. injection of 0.2 mmol/kg Gd DTPA. TI was adapted to the time of nulling the signal of the myocardium. With both techniques presence or absence of myocardial delayed

hyperenhancement was noted and the extent of myocardial hyperenhancement was calculated as percent of left ventricular mass.

Results: There were no significant differences in the incidence of delayed myocardial hyperenhancement (19/29 vs. 19/29) and the size of delayed myocardial hyperenhancement (14.6% ± 5.9 vs. 15.2% ± 5.4 of LV mass) between the standard 2D approach and the SENSE 3D approach.

Conclusions: Detection of delayed myocardial hyperenhancement indicative for myocardial infarction using a segmented 3D IR sequence with SENSE is feasible and accurate compared to standard 2D sequences. The 3D approach combined with SENSE can reduce scan duration allowing viability assessment of the entire left ventricle within one breath hold. This approach may improve work flow and patient compliance in comprehensive cardiac exams by the reduction of total investigation time and number of breath holds.

313. Cardiac Energetics are Related to Plasma Metabolites and Insulin Resistance in Patients with Heart Failure

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Introduction: Insulin resistance is commonly found in patients with heart failure (HF) and correlates with cardiac dysfunction.

Purpose: We tested whether insulin resistance is also related to abnormal cardiac muscle energetics in patients with HF.

Methods: Twelve patients with chronic HF due to non-ischaemic cardiomyopathy (without diabetes mellitus) and 20 healthy control subjects were studied. Circulating fasting plasma metabolites were measured, and resting cardiac function and energetics were assessed using magnetic resonance (MR) imaging and ^{31}Phosphorus MR spectroscopy (^{31}P MRS), respectively (Fig. 1).

Results: Left ventricular volumes were increased, and systolic function was reduced in HF (mean ejection fraction 38 ± 5% vs. 70 ± 2% in control subjects, $p < 0.001$). In HF, fasting plasma free fatty acids (FFA, 0.67 ± 0.1 in HF vs. 0.47 ± 0.1 in controls, $p < 0.01$), glucose, insulin and relative insulin resistance (5.2 ± 2.6 in HF vs. 1.3 ± 0.2 in controls, $p < 0.05$) were significantly higher than in controls. Left ventricular ejection fraction correlated negatively with plasma FFA ($r = -0.69$, $p < 0.001$) and insulin resistance ($r = -0.56$, $p < 0.01$). Cardiac energetics [phosphocreatine (PCr)/ATP ratios] were reduced from 1.7 ± 0.1 in controls to 1.1 ± 0.1 in HF ($p < 0.01$) and correlated negatively with FFA ($r = -0.46$, $p < 0.05$) and glucose concentrations ($r = -0.38$, $p < 0.05$).

Conclusions: Abnormal cardiac muscle function and energetics in HF correlate with plasma metabolite concentrations and insulin resistance, suggesting that a metabolic therapy may be successful for HF.

314. Rapid Cardiovascular MRI 3D Quantitation of Aortic Stenosis Velocities in Comparison with Echocardiography: Who is Correct?

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Introduction: Limitations of transvalvular assessments by echocardiography (Echo) exist. Yet, Echo remains the workhorse diagnostic modality. Phase velocity mapping (PVM) by CMR can rapidly and reliably acquire velocity and flow data in 3D, not possible by Echo.

Hypothesis: CMR can provide similar assessment of valvar velocities in symptomatic aortic stenosis (AS) patients as Echo and, if discordant, determine which modality approached the truth.

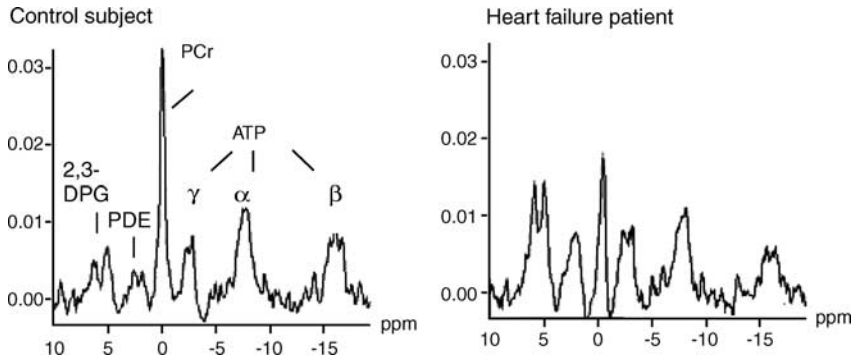

Figure 1.

Methods: Twenty-seven subjects (20 symptomatic AS; 7 controls) underwent blinded CMR (GE 1.5TCV/i) PVM analysis of AS, followed by blinded Echo (Philips 5500, Andover, MA). CMR PVM parameters: TR 19 ± 4 ms, X, Y *and* Z encoding, and offline analysis of aortic transvalvar velocities (A_{vel}).

Results: There was a high degree of correlation between CMR and Echo transvalvar metrics. Overall, 19/20 (95%) and 14/20 (70%) pts by CMR and Echo had peak A_{vel} > 4.0 m/s. Concordance for A_{vel} > 4 m/s was 88%. Importantly, discordance was partially explained by 2/20 Echo's having cosine theta errors > 35°. Nevertheless, the correlations were r = 0.89, 0.88 and 0.74 for peak, mean velocity and peak gradient, respectively, $p < 0.0005$ for all. The peak and mean CMR vs. Echo A_{vel} were: 426 ± 153 vs. 352 ± 213, 278.5 ± 136.8 vs. 251.3 ± 114.6 cm/s, respectively. There was no difference in control gradients (5.3 vs. 4.2 mmHg). Bland-Altman CI's were reasonable for bias and precision. Despite this, the Echo A_{vel} were consistently and proportionately lower then CMR. Importantly, since either technique can under but not overestimate velocity and CMR has undergone phantom correlations, this suggests that CMR represents velocities closer to the truth. The difference in A_{vel} acquisition time was 1 ± 2 min fewer for CMR.

Conclusion: CMR has theoretic, practical, and now precision and time advantages in AS. Transvalvular velocities are similar and both techniques identify patients legitimate for AS surgery. While CMR demonstrates high correlation with Echo, it may have higher inherent accuracy as correlated to flow phantoms. When added to its capacity as the 'gold standard' for LV structure, function, mass, and thoracic aortic imaging, CMR should strongly be considered for assessment of AS.

315. The Histological Basis of Late Gadolinium Enhancement in Anderson-Fabry Disease

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Introduction: Anderson-Fabry Disease (AFD), an X-linked disorder of sphingolipid metabolism is a cause of LVH. We have previously reported that late gadolinium enhancement (LGE) CMR demonstrates focal myocardial enhancement in up to 50% of patients affecting the basal infero-lateral wall of the left ventricle. It has been suggested but not proved that this is due localized fibrosis.

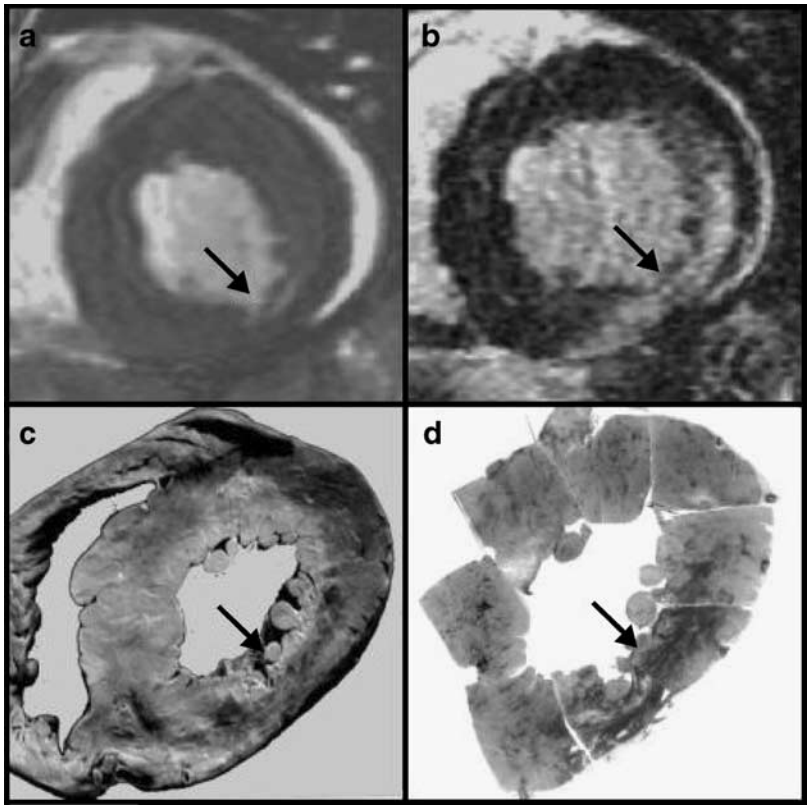

Figure 1.

Purpose: We hypothesized that LGE in AFD was caused by myocardial fibrosis.

Methods: A 57 year old male patient with AFD was found to have extensive LGE in the basal infero-lateral wall. After 22 months of clinical stability, he sustained a witnessed sudden cardiac death. The heart was examined histologically for collagen scar and the results compared to the previous in-vivo CMR scan.

Results: Cine and LGE CMR are shown next to the ex-vivo macroscopic section and reconstructed stained histological samples, Figure 1. There is concordance between the region of thinning, LGE and collagen. There was extensive myocyte vacuolation everywhere but no significant epicardial coronary artery disease. Overall, 18% of the examined heart (2 basal slices) was collagen, ranging from 0% to 100% per segment. Collage percentage per segment was from 4%, 42% and 62% respectively in segments with no, intermediate and complete LGE ($p < 0.0004$). The pattern of collagen was dissimilar to that found in myocardial infarction.

Conclusions: Focal myocardial LGE in the basal infero-lateral segment of AFD patients represents myocardial fibrosis. How an intracellular storage disease causes focal myocardial fibrosis remains unknown.

316. Faster Flow Quantification Using Sensitivity Encoded Phase Velocity Magnetic Resonance Imaging: In Vitro and In Vivo Validation

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Background: Phase velocity cine (PVC) MRI is a noninvasive method for measuring blood flow velocity and volume flow rate in the cardiovascular system. Sensitivity-encoding (SENSE) is a MRI technique to reduce imaging time by parallel data acquisition but is potentially vulnerable to inaccuracies. This study tested the agreement between conventional and SENSE PVC MRI in a flow phantom and in subjects with congenital and acquired heart disease.

Methods: Measurements were performed in a 1.5 T scanner using a segmented k-space PVC MRI sequence and then repeated with a SENSE (ASSET) factor of 2. The flow phantom used a computer controlled piston pump to generate physiologic arterial waveforms at 10 different flow rates (0.5–4.9 L/min). In the subjects (median age 24, range 7–69 years), measurements were performed in the ascending aorta (AO) ($n = 33$) and/or the main pulmonary artery (MPA) ($n = 24$) without breath-holding. Data were analyzed offline with semi-automated vessel wall border detection. Agreement was assessed using the method of Bland and Altman and a paired t-test.

Results: Utilization of SENSE reduced the scan time by 50% resulting in a mean scan time for the subjects of 59 ± 19 seconds. In the phantom, measurements without and with SENSE agreed closely with a mean difference (bias) of 0.01 ± 0.08 L/min or 0.12 ± 3.8% ($p = 0.68$). Compared to timed flow measurements, SENSE data had a mean difference (bias) of − 0.14 ± 0.1 L/min or 7.4 ± 6.4% ($p = 0.001$). In the subjects, measurements without and with SENSE also agreed closely with a mean difference (bias) of 0.08 ± 0.36 L/min or 1.3 ± 7.2% ($p = 0.08$). For in vivo data ($n = 20$), interobserver variability was 3.3% for acquisitions without SENSE and 2.4% with SENSE. Compared to standard imaging, use of SENSE reduced the signal to noise ratio by 28% in the phantom ($n = 10$) and 27% in vivo ($n = 22$), and reduced the velocity to noise ratio by 27% in the phantom ($n = 10$) and 24% in vivo ($n = 22$).

Conclusion: PVC MRI flow measurements performed with a SENSE factor of 2 were twice as fast and agreed closely with the conventional technique in vitro and in vivo. PVC MRI combined with SENSE can be used for rapid quantification of blood flow in patients with congenital heart disease.

317. A Non-invasive Method of Calculating Pulmonary Vascular Resistance Using MR Flow Data

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Introduction: Pulmonary hypertension is assessed at cardiac catheterization by measurement of pulmonary vascular resistance (PVR) and the response of PVR to vasodilators. However, there are risks attached to cardiac catheterization, therefore a non-invasive method of PVR quantification is desirable. Doppler echocardiography has been used to accurately assess systolic and diastolic pressure. Unfortunately, it cannot reliably measure mean pulmonary artery pressure, required to calculate PVR. Parameter optimization of a 2 element Windkessel model can be used to calculate PVR without knowledge of the mean pulmonary artery pressure. We use MR flow data as an input to a Windkessel model, subsequent parameter optimization allows, the non-invasive quantification of PVR and assessment of vasoreactivity.

Purpose: To demonstrate the feasibility of this potentially non-invasive method of PVR quantification.

Methods: 15 patients underwent cardiac catheterization, in an MR interventional suite (1.5 T Intera I/T MRI scanner, Philips, The Netherlands) with x-ray back-up (BV Pulsera cardiac x-ray unit, Philips, Best, The Netherlands). Invasive pressure and MR flow was acquired at baseline (condition 1) and at 20 ppm nitric oxide (condition 2) allowing calculation of PVR by the traditional method.

MR flow data was inputted into a 2 element Windkessel model, parameter optimization of vascular resistance and

compliance against systolic and diastolic pressure allowed calculation of PVR. PVR calculation using this method was done at condition 1 and 2.

All data is expressed as median (inter-quartile range) unless otherwise specified. Correlation coefficients and Bland Altman analysis were used to compare the actual PVR and the modelled PVR at condition 1 and 2 and the percentage change in PVR.

Results: All patients successfully underwent MR guided cardiac catheterisation. At condition 1 the mean invasively measured PVR was 14.0 ± 9.8 WU and the mean non-invasive PVR was 14.5 ± 10.5 WU (percentage difference = 4%). At 20 ppm NO the mean invasively measured PVR was 12.8 ± 9.3 WU and the mean non-invasive PVR was 13.0 ± 9.7 WU (percentage difference = 2%).

The correlation coefficient between the invasive PVR and the non-invasive PVR was 0.99 ($p < 0.05$) and Bland Altman analysis revealed a bias of 0.5 WU, an upper limit of agreement of 2.5 WU and a lower level of agreement of −1.5 WU at baseline. At 20 ppm the correlation coefficient between the invasive PVR and the non-invasive PVR was also 0.99 ($p < 0.05$), the bias of 0.2 WU, the upper limit of agreement was 2.4 WU and the lower level of agreement was −2.2 WU.

The mean percentage change using invasive PVR was 6.0% and 9.1% using non-invasive PVR. The correlation coefficient was 0.93 ($p < 0.05$) and the Bland Altman analysis revealed a bias of 3.1%, an upper level of agreement of 10.6% and a lower level of agreement of −16.8%.

Conclusion: We have demonstrated the feasibility of using a 2 element Windkessel model and MR flow data to quantify PVR. Currently PVR is quantified at cardiac catheterisation using invasive pressure and flow measurements. We have previously demonstrated the feasibility of combining invasive pressure measurements of MR flow data to accurately invasively calculate PVR. Invasive catheterisation, however, is associated with significant morbidity and mortality, due to vascular damage, x-ray radiation exposure, and general anaesthetic. Therefore an accurate non invasive method would be useful, particularly in paediatric practice. Using simple modelling techniques and MR flow data we have been able to accurately quantify PVR and assess vasoreactivity. This method may form the basis of fully non-invasive physiological assessment of pulmonary hypertensive disease and would represent a significant step forward in management of this disease.

318. Assessment of Differential Branch Pulmonary Blood Flow: A Comparative Study of Phase-Contrast Magnetic Resonance Imaging and Radionuclide Lung Perfusion Imaging

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Introduction: The assessment of patients with branch pulmonary artery stenosis is a frequent clinical scenario in paediatric cardiology. The functional significance of these stenoses is important, and may be defined using radionuclide lung perfusion scanning. Serial investigations can be used to assess the affect of surgical or catheter-based interventions. Radionuclide lung perfusion imaging is associated with a radiation dose of approximately 1 mSv; equivalent to approximately 50 PA chest radiographs or 6 months background radiation exposure in the UK. However, magnetic resonance (MR) imaging can be used to assess both branch pulmonary artery anatomy and pulmonary artery blood flow, in a minimally invasive fashion without exposure to x-ray radiation.

Purpose: To compare differential total right and left lung blood flow acquired with phase-contrast MR imaging with radionuclide lung perfusion measurements in children and adolescents with congenital heart disease and suspected branch pulmonary artery stenosis.

Methods: Radionuclide lung perfusion and MR imaging were performed in 10 children and adolescents with suspected unilateral branch pulmonary artery stenosis (mean age 12.1 ± 5.9 years, range 3.1–17.2 years). Radionuclide lung perfusion scanning was performed using Technetium-99m macroaggregated albumin as part of routine clinical assessment. Percent counts to each lung were measured to give total right and left lung perfusion. MR imaging was performed using a 1.5 T scanner (Siemens, Erlangan, Germany) as part of routine MR assessment of congenital heart disease. The subjects gave informed consent for the MR study. A non-breath-hold, FLASH gradient echo phase-contrast MR sequence was used to measure flow in the pulmonary trunk and either the right or left branch pulmonary artery (TR = 23 ms, TE = 4.8 ms, flip angle = 15°, slice thickness = 5 mm, matrix = 256 × 256, field of view = 340 mm, averages = 1, velocity encode gradient = 1–4 m/s). The unstenosed branch pulmonary artery was chosen for phase-contrast MR. The percent total blood flow to the measured lung was calculated as:

$$\frac{\text{Net forward branch pulmonary artery blood flow (mL/s)} \times 100}{\text{Net forward pulmonary trunk blood flow (mL/s)}}$$

The percent total blood flow to the non-measured lung was 100% minus the measured lung blood flow calculated above.

Results: Phase contrast MR imaging was performed in all subjects. There was excellent correlation between the radionuclide and phase-contrast MR calculated total lung blood flow ($r = 0.98$, $p < 0.0001$) (Figure 1A). Good limits of agreement were demonstrated between the two imaging methods using Bland-Altman analysis (Figure 1B).

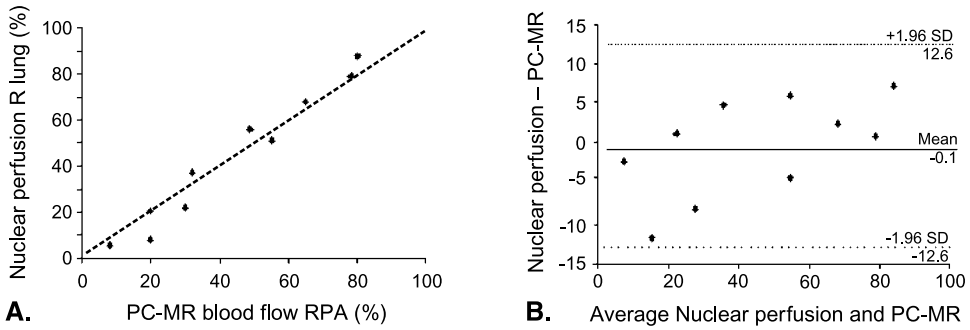

Figure 1. A. Plot of radionuclide vs. phase-contrast MR (PC-MR) measurement of blood flow to the right lung, showing good correlation. **B.** Bland-Altman plot for the same data.

Conclusions: Phase-contrast MR is an accurate method for measuring differential total right and left lung blood flow. This information can be used to assess the functional significance of branch pulmonary artery stenoses, and guide subsequent interventional management. If MR imaging is performed to assess the branch pulmonary arteries, additional radionuclide lung perfusion scanning can be avoided, reducing the overall radiation burden to this group of subjects.

319. Cardiac 1H-MR Spectroscopy in Normal and Guanidinoacetate N-Methyltransferase (GAMT) Deficient Mice In Vivo

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Introduction: MRI has become a routine tool to assess cardiac function in normal genetically modified mice. However, methods are lacking to demonstrate the metabolic consequences of gene alterations in the in vivo mouse heart. Guanidinoacetate N-methyltransferase (GAMT) catalyses an essential step in creatine synthesis. Thus, mice lacking GAMT show severely depleted creatine levels in various tissue types.

Purpose: The development and application of cardiac ^{1}H-MRS in normal and guanidinoacetate N-methyltransferase (GAMT) deficient mouse hearts in vivo.

Methods: Six GAMT$^{-/-}$ and GAMT$^{+/+}$ mice (n = 3 each, 22.7 ± 0.6 g and 31.7 ± 1.5 g, respectively) were investigated in this study. After inducing anesthesia in an anesthetic chamber using 4% isoflurane in 100% oxygen, animals were positioned supine in a purpose-built animal holder for positioning mice vertically, and maintained at 1.5–2% isoflurane in 1 l/min oxygen flow throughout the MR experiments. Spectroscopic experiments were carried out on an 11.7 T (500 MHz) MR system comprising a vertical magnet (bore size 123 mm—Magnex Scientific, Oxon, UK), a Bruker Avance console (Bruker Medical, Ettlingen, Germany) and a shielded gradient system (548 mT/m, 160 µs rise time) (Magnex Scientific, Oxon, UK). Quadrature driven birdcage coils with inner diameters of 28 mm and 40 mm (Rapid Biomedical, Würzburg, Germany) were used to transmit and receive the NMR-signals. Cardiac triggered and respiratory gated (with steady state maintenance), water suppressed and unsuppressed cardiac spectra from a 2 µl voxel positioned in the intra-ventricular septum were acquired in diastole using a PRESS sequence (TE = 9 ms, TR ≈ 2 s, NAE = 512). All spectra were quantitatively analyzed using the time domain fitting software jMRUI.

Results: Figure 1 shows water-suppressed spectra of (A) a wild type and (B) a GAMT$^{-/-}$ mouse. The spectrum of the wild type animal clearly shows the resonance of creatine at 3.0 ppm as indicated by the white arrow in Fig. 1A. The mean signal-to-noise ratio for the methyl-group of creatine at 3 ppm was 8 ± 2 (n = 3, mean ± S.D.). No creatine resonance could be identified above the noise level in any hearts of the three

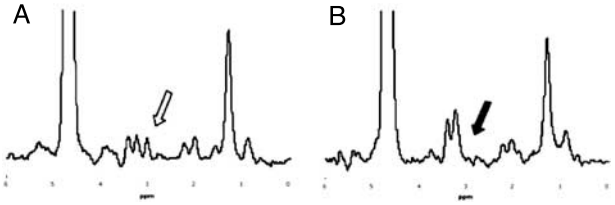

Figure 1.

mice lacking the enzyme guanidinoacetate methyltransferase (black arrow in Fig. 1B). The standard deviation of the normalized water peak amplitudes was 2.3% (n = 18). For metabolite-to-water ratios in normal mice, we measured values of: taurine—0.14 ± 0.03; carnitine—0.134 ± 0.004, and creatine—0.10 ± 0.02 and in $GAMT^{-/-}$ mice: taurine—0.17 ± 0.06; carnitine—0.2 ± 0.1.

Discussion: Various cardiac metabolites, such as creatine, taurine, carnitine or intra-myocardial lipids, were successfully detected and quantified relative to the total water content in a 2 µl voxel, using a single-voxel technique. In a murine model of GAMT deficiency, we confirmed the creatine deficiency non-invasively in the heart muscle of anaesthetized $GAMT^{-/-}$ mice. This is the first study to report on cardiac ^{1}H-MRS in mouse hearts in vivo.

320. Prevalence of Malignant Ventricular Arrhythmias in the Fibrotic Form of Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy as Assed by Contrast-Enhanced Cardiovascular Magnetic Resonance

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Introduction: Arrhythmogenic right ventricular (RV) dysplasia/cardiomyopathy (ARVD/C) is a heart muscle disease characterized by the replacement of the right ventricle (RV) myocardium by fatty and fibrous tissue, leading to malignant ventricular arrhythmias (MVA) and risk of sudden death. Contrast-enhanced cardiovascular magnetic resonance (CE-CMR) can well characterize areas of myocardial fibrosis.

Purpose: The aim of this study was to evaluate in ARVD/C patients (pts) the prevalence of MVA and the association with the finding of myocardial fibrosis as assed by CE-CMR.

Methods: We perform CE-CMR in 30 consecutive pts whom were under investigation of ARVD/C. The diagnosis of ARVD/C was defined by the international task force criteria (Br. Heart J. 1994; 71(3): 215–8). Electrophysiological study (EPS) was performed in selective cases with suspect or fulfilled diagnosis of ARVD/C.

All patients underwent MR examination in a 1.5 T magnet. Cine MR and CE-CMR images were performed to investigate respectively ventricular function and myocardial fibrosis. Patients received 0.2 mmol/kg of gadolinium-based contrast 10–20 minutes prior to image acquisition. CE-CMR pulse sequence was a fast gradient-echo with an inversion-recovery preparation pulse (delayed enhancement technique) using the following parameters: TR 3.6 ms, TE 1.8 ms, TI 230–280 ms, Flip Angle 25°, FOV 380–420, matrix 224 × 256, Slice Thickness/Gap 8.0/2.0, WFS 0.46 pixels.

Results: The mean age of study population was 39 ± 12 and 16 were male. We detected the presence of myocardial fibrosis by CE-CRM in 3 pts (10%), with predominant involvement of RV, but concomitant presence of areas of delayed enhanced in septum, anterior wall and inferior wall. All of these pts (100%) fulfilled the diagnosis of ARVD/C

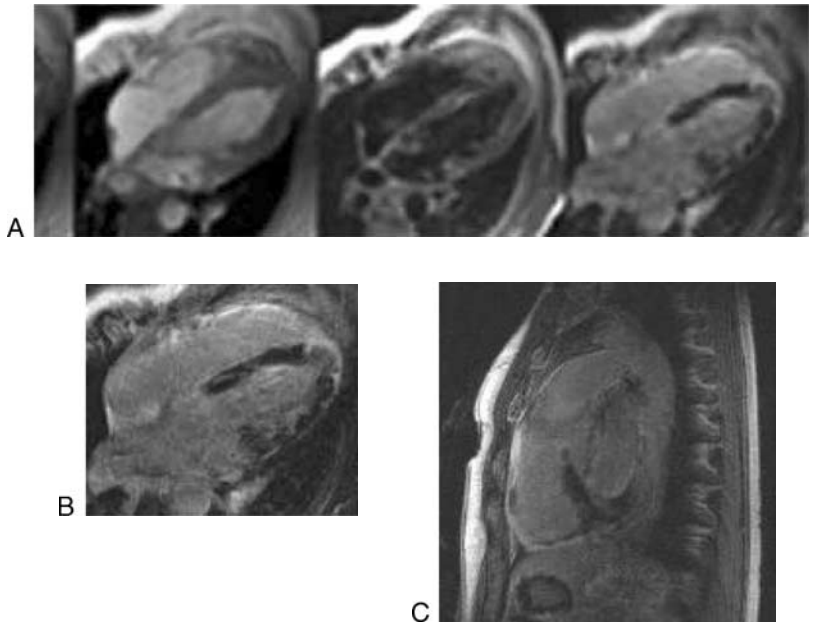

Figure 1.

and it was documented MVA in clinical setting or EPS. We did not detected myocardial fibrosis in 27 pts (90%). None of these pts (0%) fulfilled the diagnosis of ARVD/C ($p < 0.001$) and MVA were not documented.

Conclusion: CE-CMR could detect the presence of myocardial fibrosis with a major involvement of RV in patients with MVA and the diagnosis of ARVD/C by the international task force criteria. These findings may have implication to the potential usefulness of CE-CMR for evaluation and risk stratification of ARVC/D.

Figure 1A shows the cardiac four chamber view by different MR scanning techniques in a patient with myocardial fibrosis and MVA.

From left to right: Gradient Eco, Fast Spin eco and Delayed Enhancement techniques.

Figure 1B: Cardiac four chamber view by CE-CRM. Detection of myocardial fibrosis in the right and left ventricles.

Figure 1C shows RV flow track view by CE-CMR and the finding of miocardial fibrosis (high intensity signal) in the RV free wall.