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300. TIME COURSE OF Gd-DTPA ARRIVAL AND UPTAKE IN FIRST PASS DCE-MRI STUDIES OF MYOCARDIAL PERFUSION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

Aleksandra Radjenovic, PhD, Philip J. Antill, Sven Plein, PhD, John P. Ridgway, PhD, John P. Greenwood, PhD. ***University of Leeds, Leeds, United Kingdom.***

Background: Assessment of myocardial perfusion by dynamic contrast enhanced MRI (DCE-MRI) following acute myocardial infarction (AMI) could provide important diagnostic information about the possible presence of ischemic areas outside of the infarct zone. Quantitative analysis of first pass DCE-MRI can be performed by measuring various timing parameters which characterise contrast kinetics: the time of contrast arrival into the microvascular bed, the microvascular transit time and the extravasation time.

Purpose: To perform a quantitative analysis of the time course of Gd-DTPA enhancement in normal, ischaemic and infarcted myocardium in patients with AMI, at rest and under adenosine induced stress, and also to identify those timing parameters which exhibit the strongest degree of association with myocardial status.

Methods: Forty six patients (mean age = 56 ± 9.4 years, range 36-75 years, male = 41, female = 5) presenting with a confirmed first AMI were recruited. During the index admission (1-9 days after presentation), all patients underwent rest/stress DCE-MRI and delayed enhancement viability MRI, followed by x-ray angiography. Scanning was preformed on a whole body 1.5T MR scanner (Gyroscan Intera CV, Philips Medical Systems). First pass DCE-MRI myocardial perfusion imaging was performed at rest and during a 5-minute adenosine infusion (140 μg/kg/min) (1). The time course of enhancement was analyzed in 16 myocardial segments, defined according to AHA guidelines (2). The same 16-segment model was used to record locations of scar tissue (detected on delayed enhancement MRI) and segments affected by significant stenoses (≥70%) determined by x-ray coronary angiography. In DCE-MRI series, epicardial and endocardial contours were traced using MASS software (Medis, The Netherlands). Arterial input function was derived from a region of interest placed within the left ventricular cavity of the most superior (4th) slice. Two studies were excluded from analysis, due to poor SNR of the DCE-MRI curves.

The statistical analysis was performed on 44 studies (704 segments), divided into three groups: "Normal", "≥70% stenosis" and "Scar." Three timing parameters were extracted from each segment at rest and at stress (Table 1). Analysis was also performed on three cumulative timing parameters: t12 = t1 + t2, t23 = t2 + t3 and t123 = t1 + t2 + t3.

TABLE 1
Definition of timing parameters

t1	Delivery phase: the time delay between the arrival of the contrast into the basal slice of the left ventricle and the arrival of the contrast into the myocardial segment
t2	Initial microvascular phase: the time between the arrival of contrast into the microvascular bed and the time when 50% of the maximal signal intensity is reached
t3	Late microvascular phase: the time required for SI to increase from 50% to 100% of the maximal value

Results: Two-way analysis of variance was performed at $\alpha = 0.05$ significance level. Segment group (Normal, n = 389; ≥70% stenosis, n = 155; Scar, n = 160) and segment location (16 AHA segments) were considered as fixed factors. Whereas segment status was a significant factor in 3 out of 6 timing parameters measured at rest, all 6 showed a significant variation with segmental status under stress. Table 2 presents a summary of the results (the significance of the relationship of the six measured timing parameters with myocardial status, represented by the statistic F and the associated p value).

The time of arrival (t1) at stress (and associated cumulative parameters t12 and t123) displayed particularly strong relationship to the segmental status. Estimated marginal means for t1 at stress were (mean ± SE): 2.47 ± 0.08 s (Normal), 2.96 ± 0.13 s (≥70% stenosis) and 3.59 ± 0.13 s (Scar). All post-hoc pairwise comparisons (performed with Bonferroni correction) were statistically significant. Estimated mean values of t1 are presented in Fig. 1.

Conclusions: The time of arrival of contrast into myocardial tissue under adenosine-induced stress varies significantly

TABLE 2
Effect of myocardial status on timing parameters

	Rest		Stress	
Timing parameter	F	p value	F	p value
t1	0.9	n.s.	25.4	<0.0001
t2	3.9	<0.05	5.2	<0.01
t3	2.7	n.s.	9.3	<0.0001
t12	1.3	n.s.	26.6	<0.0001
t23	3.2	<0.05	9.3	<0.0001
t123	4.1	<0.05	23.3	<0.0001

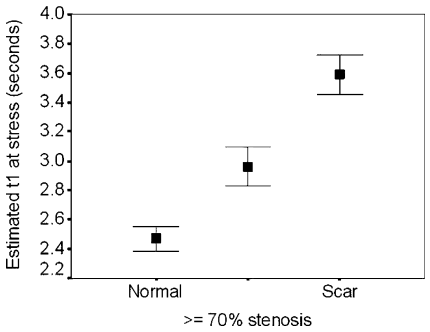

FIG. 1. Time of arrival of Gd-DTPA into myocardial segments under adenosine stress.

between segments supplied by stenosed coronary arteries, scarred segments and normal myocardium (Fig. 1). This parameter could therefore provide important quantitative diagnostic information in patients with AMI.

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301. LONG-TERM SAFETY AND EFFICACY OF AUTOLOGOUS BONE MARROW-DERIVED STEM CELL TRANSFER AFTER ACUTE MYOCARDIAL INFARCTION

Jan Bogaert, Maria Kalantzi, MD, Christophe Dubois, MD, Frank E. Rademakers, MD, PhD, Frans Van de Werf, MD, PhD, Steven Dymarkowski, MD, PhD, Stefan Janssens, MD, PhD. ***Gasthuisberg University Hospital, Leuven, Belgium.***

Background. Progenitor cell transfer may enhance myocardial functional recovery after AMI, but whether these effects are sustained remains unknown. We investigated long-term safety and efficacy of intracoronary autologous bone marrow-derived stem cell (BMSC) transfer 1 year after AMI in a randomized, double-blind, and placebo-controlled study.

Methods. We enrolled 69 patients who presented more than 2 h after AMI and randomly assigned them 24 h after successful recanalization to intracoronary injection of BMSC or placebo (CON). We measured changes in LV global and regional function and remodeling at 1 y follow-up using CMRI (n = 30 per group). Coronary flow velocity reserve (CFVR) was calculated from Doppler flow velocities at baseline and during adenosine-induced hyperemia in a subset of patients (n = 20).

Results. Global LV ejection fraction increased from 46.9 ± 8 to 49.9 ± 10% at 1 y in CON and from 48.5 ± 7 to 50.4 ± 7% in BMSC (p = NS for treatment effect). In contrast, regional contractility in segments with >75% transmural extent of hyperenhancement improved significantly more following BMSC (improved contractility in 31 of 83 segments versus in 14 of 87 segments in CON, p = 0.013). Wall motion of the infarct border zone increased more over time in BMSC (from 4.3 ± 1.9 to 5.9 ± 2.0 mm versus from 3.0 ± 2.1 to 3.7 ± 1.9 mm in CON, p = 0.039). The reduction in infarct size following BMSC (28% treatment effect at 4 months, p = 0.03) was less prominent at 1 y (23% treatment effect, p = 0.12) but hypertrophic remodeling was significantly reduced in BMSC (reduction in ED-wall thickness of adjacent and remote myocardium −0.6 ± 1.1 and −0.2 ± 1.0 mm in CON versus −1.1 ± 1.3 and −0.7 ± 1.0 mm in BMSC, p = 0.001 and 0.0001, respectively). CFVR in infarct-related arteries was normal (2.9 ± 0.4 in CON versus 2.8 ± 0.7 in BMSC).

Conclusions. Intracoronary BMSC transfer after AMI is safe, has a sustained beneficial effect on regional systolic function recovery and reduces hypertrophic remodeling in adjacent and remote myocardium.

302. COMPARISON OF THE COMBINATION OF MR STRESS PERFUSION AND LATE ENHANCEMENT IN PATIENTS WITH AND WITHOUT BYPASS SURGERY

Christoph Klein,[1] Robert Manka,[1] Thomas Kokoschinski,[1] Bernhard Schnackenburg,[2] Eckart Fleck,[1] Eike Nagel[1]
[1]German Heart Institute Berlin, Berlin, Germany, [2]Philips Medical Systems, Hamburg, Germany

Introduction: MR stress perfusion (stressPERF) in combination with late enhancement (DE) has developed to a useful clinical tool to detect myocardial ischemia. Most studies have been conducted in patients without bypass grafting (CABG). As myocardial blood flow is more complex and as the first pass kinetic of a contrast bolus may be different in patients after CABG diagnostic performance of stressPERF may decrease.

Purpose: Comparison of the diagnostic accuracy of the same stressPERF/DE protocol in patients without and with CABG.

Methods: Forty-nine patients without and 26 patients with CABG underwent MR imaging including LV-function, stressPERF (adenosine 140 μg/min/kg body weight) (SSFP, TE/TR 2.7/1.4, FA 50°, 1 saturation prepulse per slice, 3 slices per heart beat) using a 0.05 mmol/kg contrast bolus of Gd-DTPA (Magnevist, Schering, Germany) and DE (3D inversion recovery technique ,TE/TR 2.8/6.6, FA 15°) 10 minutes after an additional 0.15 mmol/kg Gd-DTPA one day before invasive coronary angiography. Images were analysed visually using the standard 16 segment model. Ischemia was defined as regional hypoenhancement in stressPERF without enhancement in DE. Ischemia in invasive angiography was defined as a stenosis >75% in a vessel ≥ 2mm diameter.

Results: Prevalence of angiographically significant stenosis was 47% and 69% in non-CABG and CABG, respectively. Surgery was performed 5 ± 4 years before the examination. LIMA-graft was used in 86% of the patients. Enhancement was present in 24% and 70% of patients without and with CABG, respectively. Sensitivity, specificity and diagnostic accuracy are shown in Tables 1 and 2.

TABLE 1

	Per patient analysis		
	Sensitivity	Specificity	Diagnostic accuracy
No CABG	87%	85%	86%
CABG	67%	75%	69%

	Per vessel territoy analysis								
	LAD			LCX			RCA		
	Sensitivity	Specificity	Diag. accuracy	Sensitivity	Specificity	Diag. accuracy	Sensitivity	Specificity	Diag. accuracy
No CABG	87%	97%	92%	69%	92%	86%	75%	87%	84%
CABG	50%	77%	73%	40%	94%	73%	71%	92%	81%

Conclusions: StressPERF is feasible in patients post CABG. However, diagnostic accuracy is reduced compared to patients without CABG if similar visual criterias are applied. This is possibly due to the diffenrent kinetic of the first pass contrast bolus through the bypasses and more and larger areas of enhancement. Additionally, the standard 16 segment model may be inadequate for patients post CABG to define the vascular bed as long standing collateral flow has been established. Probably ajusted visual criterias or semiquantification needs to be applied for better diagnostic performance.

303. CLINICAL EVALUATION OF WOMEN WITH SUSPECTED CORONARY ARTERY DISEASE USING STRESS-PERFUSION MRI

Simon Greulich, MD,[1] Igor Klem, MD,[2] John F. Heitner, MD,[3] Han W. Kim, MD,[4] Jonathan W. Weinsaft, MD,[4] Srivani Ambati, MD,[4] Michele A. Parker, MSc,[4] Robert M. Judd, PhD,[4] Raymond J. Kim, MD,[4] Udo P. Sechtem, Prof.[1] [1]***Robert-Bosch-Krankenhaus, Stuttgart, Germany,*** [2]***Universitätsklinikum Münster, Münster, Germany,*** [3]***New-York Methodist Hospital, New York City, New York, USA,*** [4]***Duke University Medical Center, Durham, North Carolina, USA.***

Introduction: Noninvasive tests have limited accuracy for detection of CAD in women. A combined perfusion and infarction CMR stress test with a visual interpretation algorithm is a novel diagnostic tool showing high accuracy in non-selected patient populations; however, its clinical value in women with suspected CAD is unknown.

Purpose: To test the hypothesis that this CMR stress test can accurately diagnose CAD in women.

Methods: Women without prior history of cardiac disease who were referred for coronary angiography (CA) were prospectively enrolled. Cine, adenosine and rest perfusion, and delayed enhancement CMR was performed in all pts. within 24 hrs before CA. All CMR studies were analyzed visually, first stress/rest perfusion alone, and than in combination with delayed enhancement CMR using a prespecified algorithm for integrative analysis. CAD was defined as coronary stenosis > or = to 70% on CA.

Results: MRI was performed in 127 women, mean age was 62.2 ± 11.2 yrs, 28 pts (22%) had diabetes, BMI was 28.7 ± 5.1, number of CAD risk factors was 2.3 ± 1.2. Prevalence of CAD was 28% (35 pts); in 19 (54%) women single vessel disease (SVD) was found on CA, and 16 (46%) pts had multivessel disease (MVD). The overall sensitvity, specificity, and accuracy of stress/rest perfusion alone for the detection of CAD was 80%, 60%, and 65%. The combination with DE-CMR using the algorithm had similar sensitivity (86%, $p = 0.16$), but higher specificity and accuracy (89% and 88%, respectively, $p < 0.0001$ for both). The sensitivity for detection of pts with SVD (74%) was lower than for MVD (100%) ($p < 0.05$).

Conclusions: A combined perfusion and infarction CMR stress test with a visual interpretation algorithm is effective for the detection of coronary artery disease in women. There are limitations in the detection of women with single vessel disease.

304. EFFECT OF DISTAL EMBOLIZATION ON MYOCARDIAL PERFUSION RESERVE FOLLOWING PERCUTANEOUS CORONARY INTERVENTION: AN QUANTITATIVE MR PERFUSION STUDY

Joseph B. Selvanayagam, MBBS FRACP, DPhil,[1] Adrian S. H. Cheng, MRCP,[1] Michael Jerosch-Herold, PhD,[2] Kazem Rahimi, MRCP,[1] Stefan Neubauer, MD, FRCP,[1] Adrian Banning, MD, FRCP,[3] [1]***University of Oxford, Oxford, United Kingdom,*** [2]***Oregon Health & Science University, Portland, Oregon, USA,*** [3]***John Radcliffe Hospital, Oxford, United Kingdom.***

Introduction & Purpose: Percutaneous Coronary Intervention (PCI) results in enlargedluminal cross-sectional area and improved myocardial blood flow. Previous studies using intra-coronary Doppler have shown that there is a subset of patients who demonstrate persistent impairment in microcirculatory function after PCI even after substantial conduit area enlargement. Distal embolization of plaque contents has been

postulated as the main mechanism for this. We sought to investigate this by we evaluating PCI induced changes in myocardial perfusion reserve index (MPRI) and procedure related myonecrosis using high resolution quantitative CMR. We hypothesized that MPRI would be impaired in myocardial segments with new 'distal' PCI induced injury and that myocardial segments 'upstream' to the injury in territory of the culprit vessel would also demonstrate persistent microvascular dysfunction following the procedure.

Methods: Forty patients with 1 or 2 vessel coronary disease undergoing PCI were studied with pre-PCI and 24 hour post PCI DE-MRI and first pass perfusion MR imaging at rest and stress. For perfusion imaging, 3 short axis images were acquired during every heart-beat using a T1-weighted turboFLASH sequence, with low-dosage Gd-DTPA bolus injection (0.04 mmol/Kg for rest and stress). DE-MRI was performed after a further administration of 0.045 mmol/Kg Gd-DTPA. In each slice, MBF was determined for 8 myocardial sectors in mL/min/g by deconvolution of signal intensity curves with an arterial input function measured in the LV blood pool. MPRI (Stress MBF/Rest MBF) values were calculated and subdivided according to presence and location of new delayed hyperenhancement. As per previous studies, new hyperenhancement occurring in the myocardium distal to the stent was deemed 'distal' injury. *Results:* When all patients were considered, mean MPRI was 2.49 ± 0.91 in segments without and 2.08 ± 0.72 in segments with significant coronary stenosis pre PCI (mixed model $z = -7.87$, $p < 0.001$). Post procedure, there was a significantly greater increase in the mean MPRI in revascularized myocardial segments (0.33 ± 0.88) than in nonrevascularized segments (0.07 ± 0.79, $z = 3.99$, $p < 0.001$). When pre- and post-DE-MRI were compared, 21 (84%) of these patients had new 'distal' type HE. Eighty-two of 407 (20%) intervened myocardial segments demonstrated new irreversible injury post revascularisation. Mean MPRI in such segments did not show statistically significant changes, with MPRI $= 2.16 \pm 0.95$ prePCI, and 2.00 ± 0.85 postPCI. In contrast, across all patients, mean MPRI in revascularized myocardial segments not demonstrating new irreversible injury was significantly increased postprocedure (2.06 ± 0.65 prePCI and 2.50 ± 0.90 postPCI; $p < 0.001$). When the two groups (HE vs. no HE) were compared, the absolute change in MPRI pre- and postprocedure was significantly different in the non-HE segments (0.46 ± 0.89) from the HE segments (-0.16 ± 0.60; $z + -5.26$, $p < 0.001$). MPRI in myocardial segments 'upstream' to procedural injury (HE) compared to MPRI in remote segments is illustrated in Fig. 1. Changes in mean MPRI postPCI in segments upstream to new injury was not significantly different when compared with perfusion changes in remote myocardium ($z = -1.14$; $p = 0.25$) indicating that there was no significant reduction in perfusion reserve in these segments.

Conclusion: MPRI is reduced in myocardial segments demonstrating new 'distal' irreversible injury at 24 hours post PCI. These reductions seem to be confined to the segments with injury and do not affect the entire supply territory of the culprit vessel.

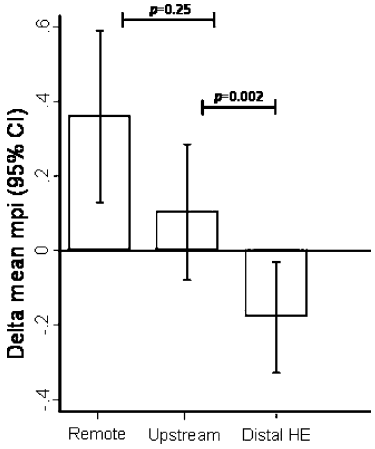


FIG. 1.

305. ACCURACY OF FULLY QUANTITATIVE CMR MYOCARDIAL PERFUSION IN DETECTION OF CORONARY DISEASE AS MEASURED BY QUANTITATIVE CORONARY ANGIOGRAPHY

Federico E. Mordini, MD, Tariq M. Haddad, MD, Li-Yueh Hsu, DSc, W. Patricia Bandettini, MD, Peter Kellman, PhD, Tracy Lowrey, RN, Anthony H. Aletras, PhD, Andrew E. Arai, MD. ***National Institutes of Health, Bethesda, Maryland, USA.***

Background: The detection of coronary disease by contrast enhanced MRI is traditionally performed by qualitative assessment. Qualitative interpretation introduces subjective factors that could be minimized by objective or quantitative measures of perfusion. A dual bolus method for quantifying first pass perfusion studies has been validated in an animal model and in normal humans.

Objective: Our objective was to determine the sensitivity, specificity, and accuracy of fully quantitative analysis of dual bolus stress perfusion studies in patients with known or suspected coronary disease versus a reference standard of coronary stenosis as measured by conventional quantitative coronary angiography (QCA).

Methods: Sixty-seven patients were referred for MRI stress testing utilizing dipyridamole (0.56 mg/kg over 4 minutes) and dual bolus first pass gadolinium enhanced imaging (0.005 mmol/kg followed by 0.1 mmol/kg). CMR was performed on either a GE or Siemens 1.5T scanner with either a 4 element or a 12 element phased array surface coil. A saturation recovery, hybrid echoplanar perfusion sequence obtained 3 images every heart beat. The 3 imaging slices per patient were divided into 12 radial sectors and analyzed as endocardial, epicardial, and transmural regions. Myocardial perfusion (mL/min/g) was quantified using

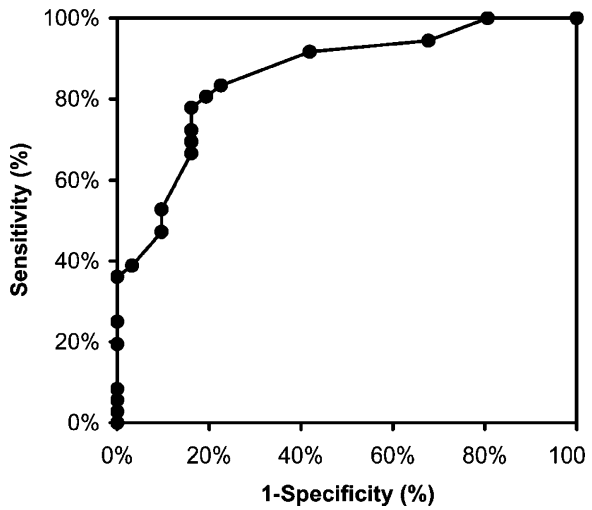
TABLE 1
Sensitivity and specificity of dipyridamole stress perfusion compared with QCA

Interpretation Method	Sensitivity (%)	Specificity (%)	Accuracy (%)
Clinical Interpretation	81%	84%	82%
Duke Qualitative	89%	71%	81%
Dual Bolus Fermi Function	81%	81%	81%

Fermi function constrained deconvolution methods. Qualitative assessment of the perfusion exam was performed by our standard protocol and also with a published algorithm from Duke University. All perfusion results were correlated to cardiac catheterization studies obtained within 90 days of the MRI. A cardiologist blinded to the MRI results performed QCA by standard means. Statistics are presented as mean ± SD.

Results: Patients averaged 60 ± 11 years, and 45 were men (67%). Thirty-six patients (54%) had coronary stenoses >60% in diameter, 5 had 3vessel disease (VD), 6 had 2 VD, and 25 had 1 VD. Clinical qualitative assessment of dipyridamole stress perfusion images yielded a sensitivity of 81% and specificity of 84%. The Duke qualitative interpretation method, which starts with late gadolinium images, had a sensitivity of 89% but a specificity of only 71%. Quantitative analysis of dual bolus stress perfusion yielded a sensitivity and specificity of 81% and 81%. The overall accuracy of all 3 methods ranged from 81–82%.

The optimal threshold for quantitative stress perfusion in all subjects was a 20% or greater endocardial flow reduction relative to a user selected normal segment. This agreed well with the optimal threshold predicted by the coefficient of variation (standard deviation/mean) which averaged 0.10 ± 0.2 for the 3 slices in subjects with no significant stenosis. Based on these results, a 2 standard deviation threshold should correspond to a 20% reduction in myocardial perfusion and define the normal limits. The optimal threshold for QCA was a 60% diameter stenosis as determined by ROC analysis.



In segments identified as normal, myocardial blood flow averaged 2.70 ± 0.76 mL/min/g while true positive perfusion defects averaged 1.51 ± 0.65 mL/min/g ($p < 0.001$). Owing to intersubject variablilty, intrasubject flow was best distinguished by the ratio of ischemic to normal (remote) flow which averages 0.57 ± 0.17.

Conclusion: Quantitative analysis of stress perfusion images reproduced the accuracy of clinical interpretation methods of 2experienced centers. The advantage of quantitative perfusion analysis revolves around the objective determination of normal and abnormal. Overall results for any of these 3 interpretation methods are very encouraging in light of the high proportion of single vessel disease in this study.

306. EFFECTS OF SPATIAL AN TEMPORAL RESOLUTION ON THE DARK RIM ARTIFACT IN FIRST PASS MYOCARDIAL PERFUSION

Nidal Al-Saadi, MD,[1] Geir Torheim, PhD,[2] Glenn Reynolds, PhD,[3] Daniele De Marchi,[4] Silvia Burchielli,[4] Massimo Lombardi, MD.[4] [1]***Institute of Cardiac MRT, Berlin, Berlin, Germany,*** [2]***GE-Healthcare, Oslo, Norway,*** [3]***GE-Healthcare, Boston, Massachusetts, USA,*** [4]***C.N.R., Clinical Physiology Institute, Pisa, Italy***

Many different factors seem to contribute to the appearance of the subendocardial dark rim artifact in myocardial first pass perfusion studies. This artifact affects diagnostic accuracy of visual assessment. We aimed to evaluate the impact of spatial resolution and cardiac motion as determined by heart rate, on the severity of the dark rim artifact in an optimized multi-slice, and myocardial first pass perfusion sequence.

Methods: Six pigs were evaluated in general anesthesia. Three short axis locations were acquired every heart beat during the first pass of a contrast agent bolus (Omniscan GE Healthcare AS, Oslo, Norway) using an EPI/FGRE sequence (90° nonselective saturation prep, chemical shift fat suppression, TE 1.0, TR 6.0, 25°, Echo Train length: 4, ASSET factor = 2.0, acquisition time per image 70 ms) using a 1.5 Tesla, Excite HD, MRI scanner (GE Healthcare). A power injector was used to inject 0.075 mmol/kg of Omniscan with duration of 3 seconds, followed by a 5 mLbolus of saline at the same injection speed. First pass perfusion scans were repeated every 60 minutes, which allowed for adequate wash out of the contrast agent between scans. Perfusion scans were performed with two different FOVs (spatial resolution 2.6 mm = A, and 3.9 mm = B) and at two heart rates (HR 70 BPM = 1, and 110 BPM = 2), resulting in a set of 4 perfusion scans (A1, A2, B1, and B2). Heart rates were adjusted by pharmaceutical intervention, which yielded a cycle length of 0.857 s (70 BPM) and 0.545 s (110 BPM). A total of 9 sets (36 perfusion scans) were acquired in 6 pigs. Images were evaluated visually and scored for the severity of the dark rim (0–3) and transmural extent (0–100%). A paired t-test was performed to detect significant differences.

Results: More severe and wider dark rim artifacts resulted in the group with low spatial resolution (severity: A = 1.02 ± 0.52, B = 2.13 ± 2.13, $p < 0.0001$; transmurality: A= 47 ± 30, B = 77 ± 25%, $p < 0.0001$; heart rate A vs. B n.s.) and in the group of high rate (severity: 1 = 1.2 ± 0.65, 2 = 1.9 ± 0.8, $p < 0.0001$; transmurality: 1= 52 ± 33, 2 = 75 ± 24, $p < 0.001$; HR 1 vs. 2 68 ± 6 and 107 ± 10, $p < 0.0001$). Most severe artifacts were present in the group of combined low spatial resolution and high heart rate.(Severity 2.37 ± 067 vs 083 ± 0,37; transmurality 87 ± 17% vs. 42 ± 37).

Conclusion: The dark rim artifact is independently determined by spatial and temporal resolution. To minimize artifacts and increase reliability, a stress perfusion sequence should provide high spatial and temporal resolution especially at high heart rates.