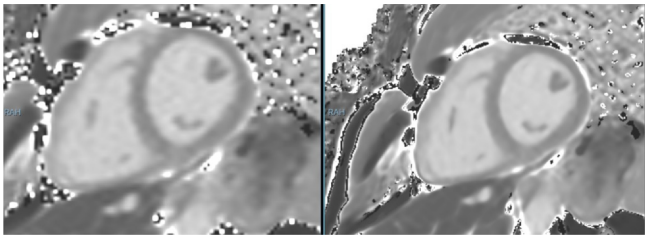
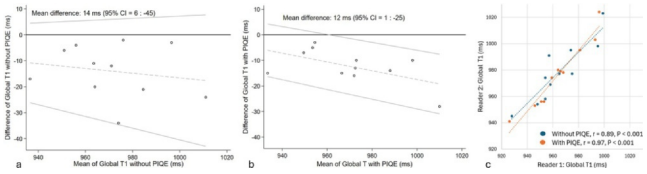
test

		No PIQE		With PIQE		P*	
		Mean	SD	Mean	SD	Mean	SD
Global T1 (ms)	1st scan	964±21	38±7	964±21	37±8	0.62	0.33
	2nd scan	973±24	41±9	970±25	36±6	0.23	0.055
	P**	**0.013**	0.4	**0.02**	0.79		
Septal T1 (ms)	1st scan	969±31	31±8	977±26	26±8	0.05	0.05
	2nd scan	981±33	34±6	982±30	30±10	0.66	0.08
	P**	0.1	0.08	0.8	0.13		
SNR^ (Myocardium)	1st scan	46±23		52±39		0.86	
	2nd scan	41±21		48±32		**0.02**	
	P**	0.25		0.37			
CNR^ (Myocardium to LV blood)	1st scan	9.7±5.1		10.5±7.3		0.53	
	2nd scan	8.3±5.3		8.7±6.7		0.86	
	P**	0.29		0.29			

* P value from Wilcoxon signed-rank test comparing images with and without PIQE
** P value from Wilcoxon signed-rank test comparing images of the 1st and 2nd scan
^ SNR and CNR were assessed on the last original MOLLI images





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Differentiating Ischemic From Healthy Myocardium Using Cardiovascular Magnetic Resonance Dipyridamole Rest and Stress T1 Mapping

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Background: Cardiovascular magnetic resonance (CMR) stress T1 mapping using adenosine and regadenoson can differentiate ischemic, infarcted, and healthy myocardium. However, the effect of dipyridamole remains unclear. This study evaluates whether dipyridamole-induced stress T1-mapping can distinguish myocardial tissue types.

Methods: Twenty-five healthy controls and 20 patients with coronary artery disease (CAD) underwent rest and dipyridamole stress T1-mapping (ShMOLLI), followed by gadolinium-based quantitative perfusion imaging at 1.5T. Native T1 values and stress T1 reactivity (dT1) were assessed across different myocardial tissues. Correlations between dT1 and myocardial blood flow (MBF) were examined.

Results: In healthy controls, global rest T1 was 934 ± 26 ms, with a stress-induced increase of 6.5 ± 0.6% ($p < 0.0001$). Infarcted myocardium showed elevated resting T1 (1146 ± 71 ms) with an absent stress response (dT1 = -0.9% ± 1.8%). Ischemic myocardium had mildly elevated rest T1 (965 ± 26 ms; $p < 0.001$) and blunted reactivity (dT1 = 1.3 ± 0.6%) compared to healthy myocardium. Remote myocardium had rest T1 similar to normal tissue (936 ± 19 ms; $p > 0.05$) but reduced stress reactivity (dT1 = 4.5 ± 1.1%; $p < 0.0001$) compared to norm. dT1 strongly correlated with stress MBF ($r = 0.80$) and myocardial perfusion reserve ($r = 0.70$) (both $p < 0.0001$).

Conclusion: Dipyridamole-induced stress T1-mapping can differentiate infarcted, ischemic, and healthy myocardium, supporting its use in non-contrast CMR for myocardial tissue characterization.

	Healthy subjects (n = 25)	CAD patients (n = 20)	*p* value
Age (yrs)	44.5 ± 16.3	62.5 ± 10.3	<.0001
BMI (kg/m^2)	22.4 ± 4.6	26.5 ± 4.4	0.831
Male (%)	11 (44)	13 (65)	0.161
Risk factors (%)			
Hypertension	0	10 (50)	
Diabetes mellitus	0	9 (45)	
Hypercholesterolemia	0	2 (10)	
Family history of CAD	0	3 (15)	
History of CAD	0	11 (55)	
smoking	0	1 (5)	
Medication (%)			
Aspirin	0	13 (65)	
Beta-blocker	0	8 (40)	
Statin	0	14 (70)	
CMR clinical indices			
LVEF (%)	59.2 ± 4.4	62.1 ± 7.6	0.147
LVEDVI (ml/m2)	61.0 ± 13.4	65.5 ± 17.0	0.266
Number of remote myocardial segments[&]		57	
Number of ischemic myocardial segments[#]		55	
Number of infarcted myocardial segments[#]		14	
Invasive coronary angiography			
1-vessel CAD[$]		7	
2-vessel CAD		4	
3-vessel CAD		9	

[&] no ischemia or infarction
[#] Based on results of first-pass perfusion and late gadolinium enhancement imaging
[$] ≥70% stenosis in a major coronary vessel
Abbreviations: CAD: coronary artery disease; CMR: cardiovascular magnetic resonance; LVEF: left ventricular ejection fraction; LVEDVI: indexed left ventricular end-diastolic volume

	Healthy Subjects (n = 25)	CAD patients (n = 20)			
	Normal	Remote	Ischemic	Infarct	LV blood pool
Rest T1 (ms)	934 ± 26	936 ± 19	965 ± 26	1146 ± 71	1486 ± 79
Stress T1 (ms)	994 ± 28	979 ± 22	978 ± 27	1135 ± 71	1489 ± 83
dT1 (%)	6.5 ± 0.6	4.5 ± 1.1	1.3 ± 0.6	-0.9 ± 1.8	0.2 ± 2.8
Resting MBF	0.9 ± 0.2			0.6 ± 0.2	

		0.9 ± 0.3	0.9 ± 0.4	
Stress MBF	3.1 ± 0.7	2.1 ± 0.7	1.5 ± 0.5	0.7 ± 0.4
MPR	3.8 ± 0.8	2.6 ± 1.0	2.0 ± 0.8	1.1 ± 0.3

Abbreviations: CAD: coronary artery disease; dT1:delta T1; MBF: myocardial blood flow; MPR: myocardial perfusion reserve

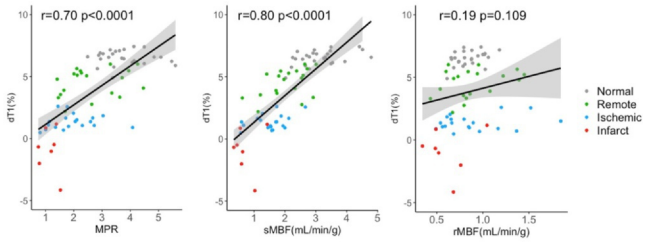

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Image-based navigated CMRA late after gadobutrol reliably delineates the coronaries in children and adults

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Background: 3D whole-heart cardiac magnetic resonance angiography (MRA), particularly coronary evaluation, remains challenging using conventional respiratory navigation. Motion correction strategies have shown promise in improving efficiency and quality. We previously reported excellent results with a non-contrast 3D whole-heart sequence with bright-blood (BB) and dark-blood (DB) phase sensitive inversion recovery (IR) using an interleaved T2prep-IR and no prep sequence with image-based navigation (iNAV) and a variable-density Cartesian spiral-like profile order trajectory (VD-CASPR). However, resolution and contrast limitations still make resolving very small structures like anomalous coronaries structures challenging. We reported using the above BB-BOOST strategy after ferumoxytol infusion with improved results. However, this precludes scar analysis or prolongs scan time for dual contrast. To this end we evaluated a previously introduced single-interleave CMRA iNAV sequence for BB imaging after gadolinium infusion using stead state free precession (SSFP) with T2-preparation with 8 refocusing pulses (8-MLEV). We hypothesized that iNAV-CMRA with 8-MLEV acquisitions improved reliability of the MRA in comparison to BB-BOOST, particularly in delineating coronary anomalies.

Methods: Patients undergoing clinically indicated CMRs from April to August 2025 were prospectively enrolled and scanned at 1.5T (AVANTO FIT, Siemens) using an iNAV T2-prepared SSFP research CMRA sequence employing 8-MLEV. A subset of patients had an iNAV or conventional diaphragmatic respiratory navigated IR-FLASH either just after gadolinium infusion or after ferumoxytol. Both imaging methods were rated on a 1-4 scale: 1 = non-diagnostic, 2 = significant image degradation, 3 = mild image degradation, and 4 = sharp delineation of the coronaries delineating coronary dominance. Group comparisons used a paired t-test (two-tailed, $p < 0.05$).

Results: 75 pts, mean age 9.8 years (range = 1 month- 69 years) underwent T2-prep iNAV imaging late after gadavist infusion. 22 had an iNav BB-BOOST during slow gadavist infusion for comparison and 10 after ferumoxytol infusion. 12 pts had coronary anomalies (5 with right coronary either high or from the left sinus, 2 with left coronaries from the right sinus). Coronary anomalies were delineated on all late CMRA iNAV exams with a mean score of 3.6, compared to a score of 2.8 for BB-BOOST during gadavist infusion ($p < 0.01$) and 3.8 after ferumoxytol (p = NS)

Conclusion: In our series spanning a large age range from infants to adults, T2-prep SSFP iNAV imaging consistently delineated the coronary anatomy including in a range of coronary anomalies and was equivalent to ferumoxytol BB-BOOST iNAV in delineating the coronaries, with the latter requiring a longer scan duration.