



ISCHEMIC HEART DISEASE

Dr. Monica Andriani
UOA Cardiologia
(Direttore Prof. M. Rinaldi)
Città della Salute e della Scienza di Torino

ISCHEMIC HEART DISEASE

**CHRONIC ISCHEMIC
HEART DISEASE**

CHEST PAIN

**ACUTE ISCHEMIC
HEART DISEASE**

VOLUMES

**SEGMENTAL
FUNCTION**

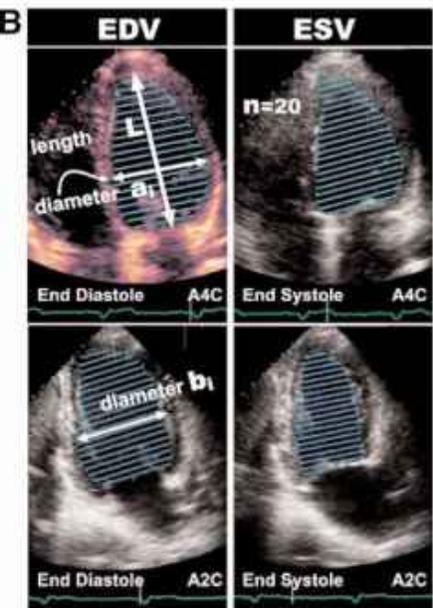
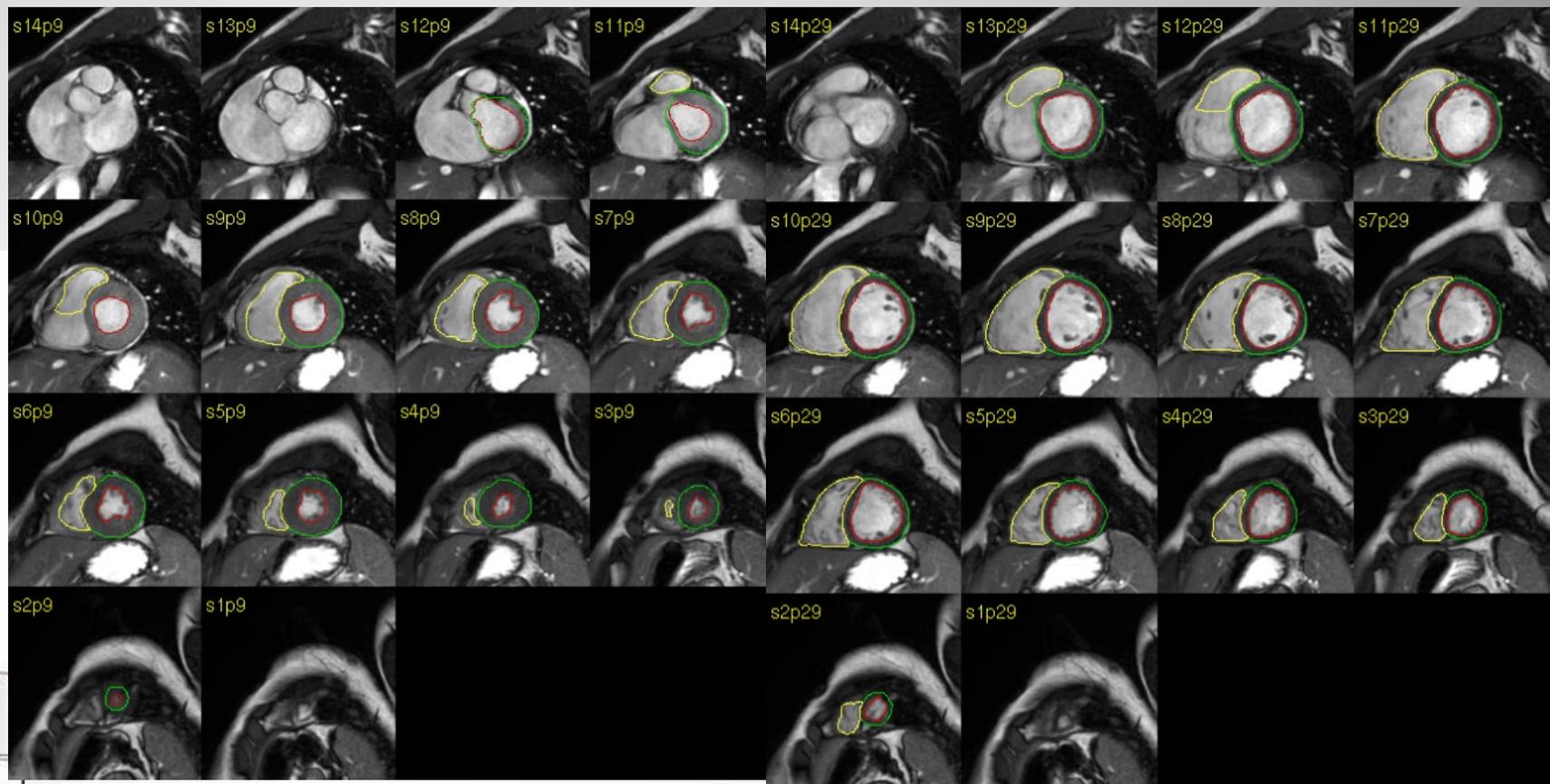
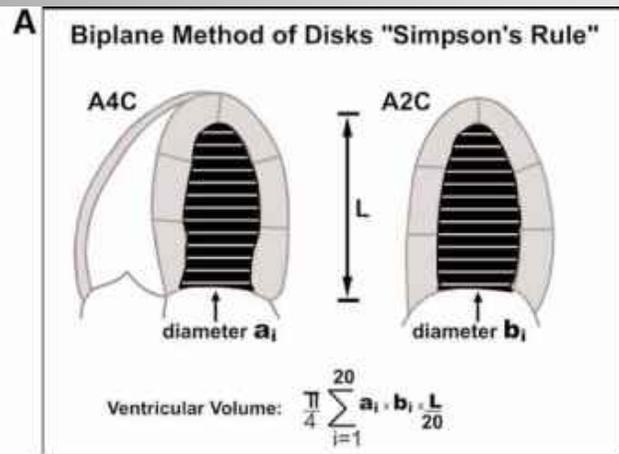
**GLOBAL
FUNCTION**



**GOLD
STANDARD**



Volume and Ejection Fraction



Method of Disks
Calculation of Ejection Fraction
using Biplane Apical Views

Volume of each disk:

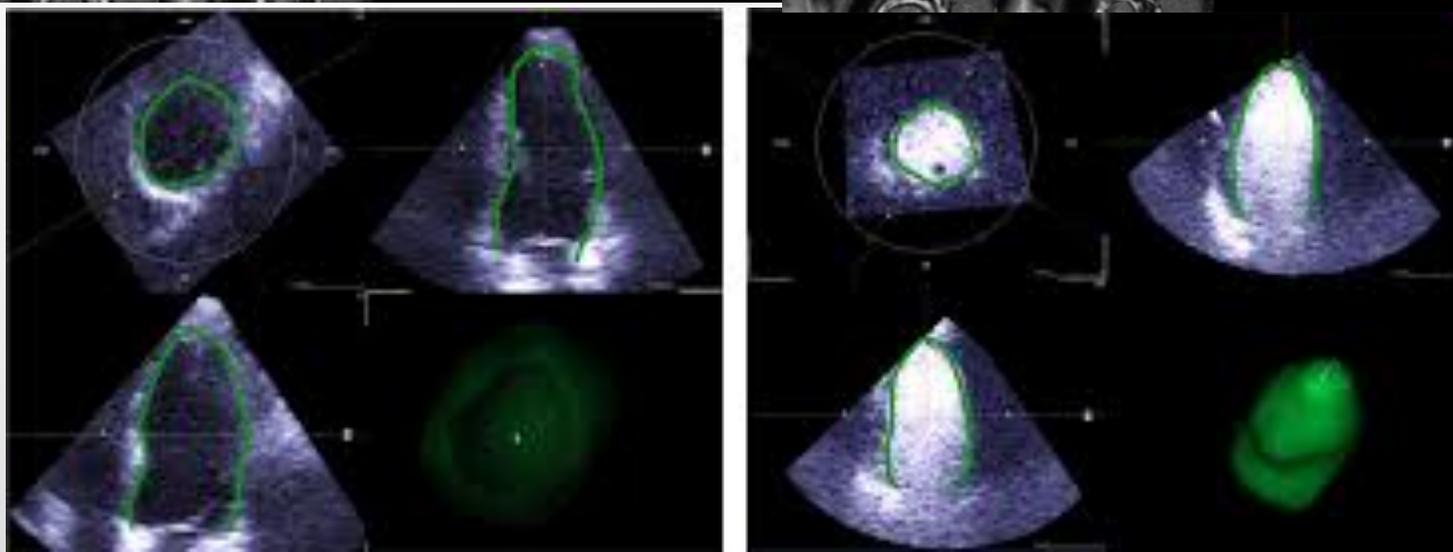
$$\frac{\pi (a_i \times b_i) L}{4n}$$

Total Ventricular Volume:

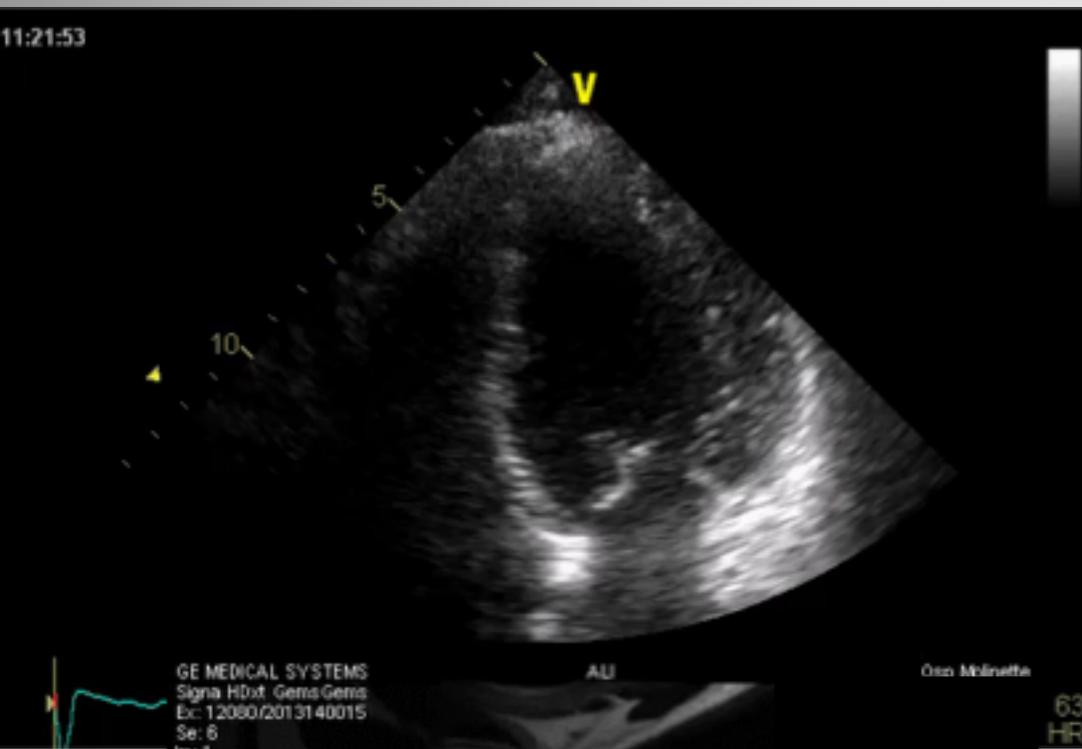
$$\frac{\pi}{4} \sum_{i=1}^{20} a_i \times b_i \times \frac{L}{20}$$

Ejection Fraction:

$$\frac{EDV - ESV}{EDV} \times 100\%$$



11:21:53

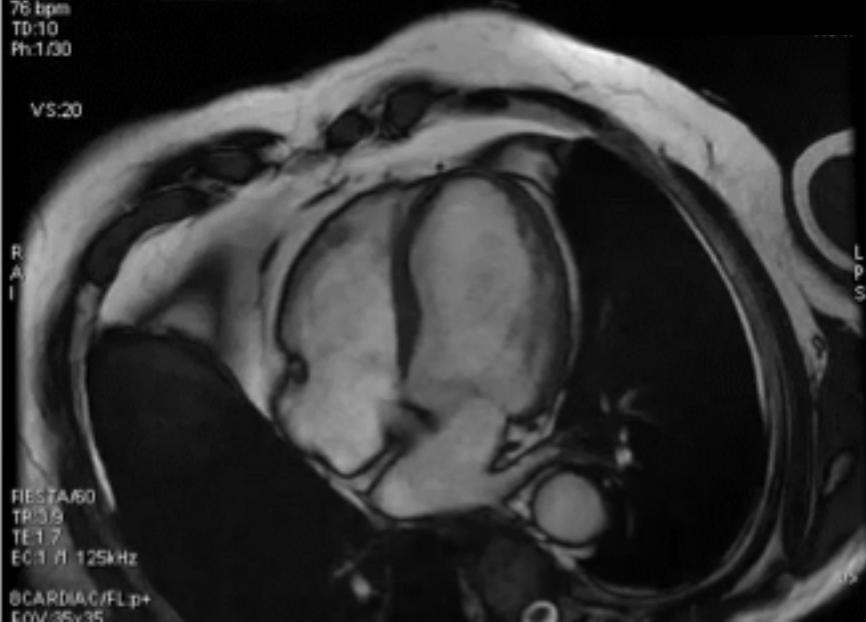


GE MEDICAL SYSTEMS
 Signa HDxt, Gems/Gems
 Ex: 12080/2013140015
 Se: 8
 Im: 1
 O Ax: 153.2
 DFOV: 35.0cm
 76 bpm
 TD:10
 Ph:1/30

AU

Osp Molinette

63 HR



VS:20

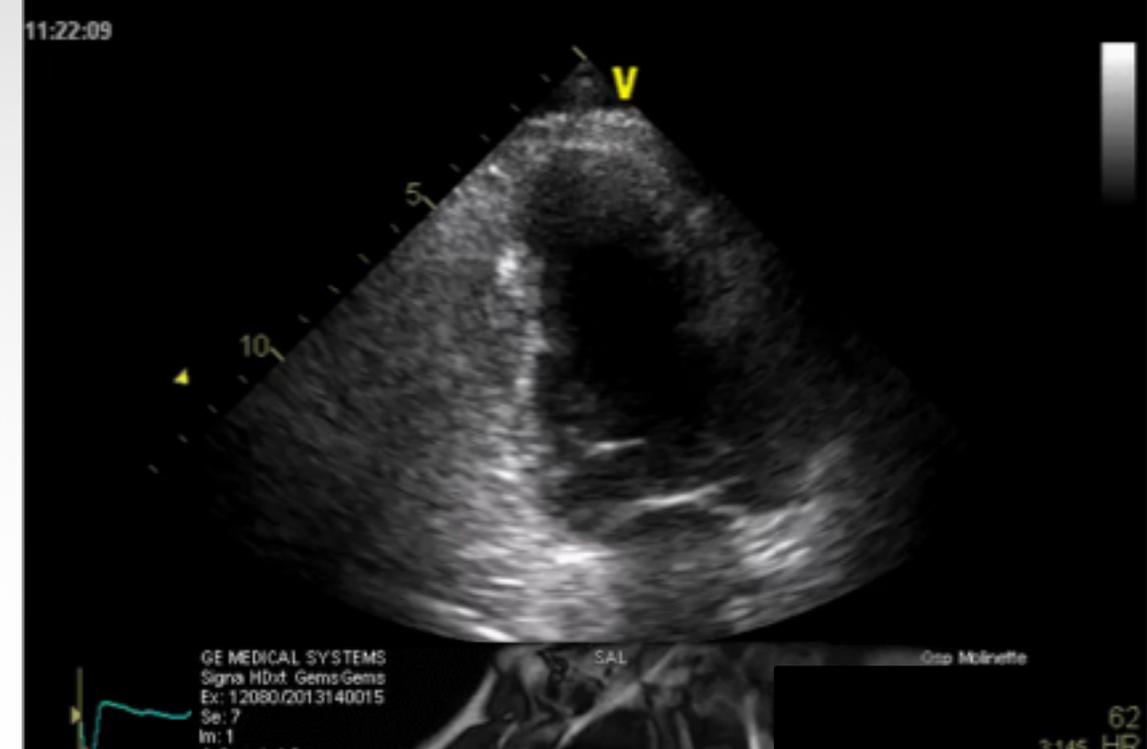
R
A
I

S
A
L

RESTA/50
 TR:3.9
 TE:1.7
 EC:1 // 125kHz

@CARDIAC/FL p+
 FOV:35x35

11:22:09

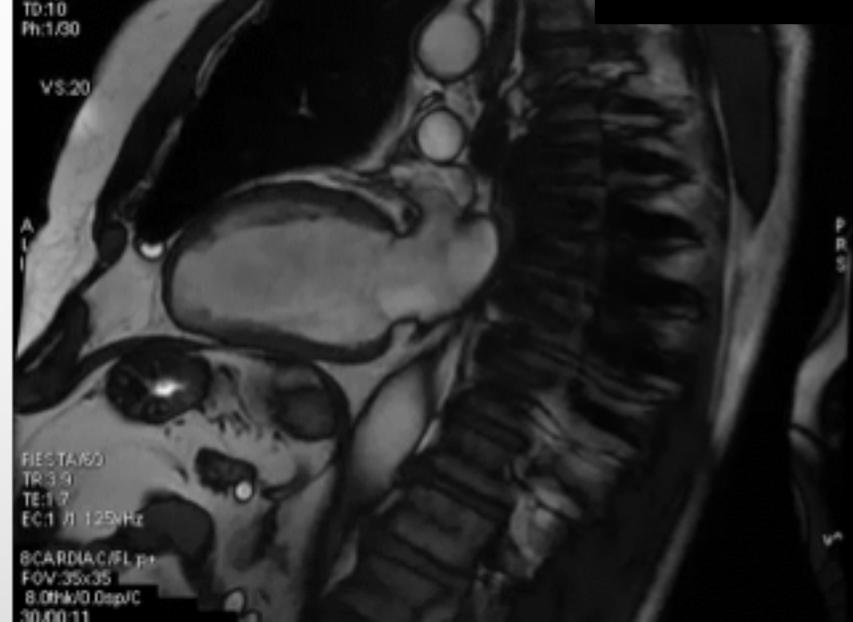


GE MEDICAL SYSTEMS
 Signa HDxt, Gems/Gems
 Ex: 12080/2013140015
 Se: 7
 Im: 1
 O Sag: 14.6
 DFOV: 35.0cm
 76 bpm
 TD:10
 Ph:1/30

SAL

Osp Molinette

62
3:145 HR



VS:20

A
U
I

S
A
L

RESTA/50
 TR:3.9
 TE:1.7
 EC:1 // 125kHz

@CARDIAC/FL p+
 FOV:35x35
 8.0Hz/0.0sp/C
 30.00:11

GE MEDICAL SYSTEMS
Signa HDxt GemsGemis
Ex: 7427118478.2012.3
Se: 8
Im: 1
O Cor A 39.6
DFOV 35.0cm
106 bpm
TD:10
Ph:1/30

SAL

Osp Molinette

Mag = 1.00
FL:
ROT:

VS:20

R
A
I

S
L

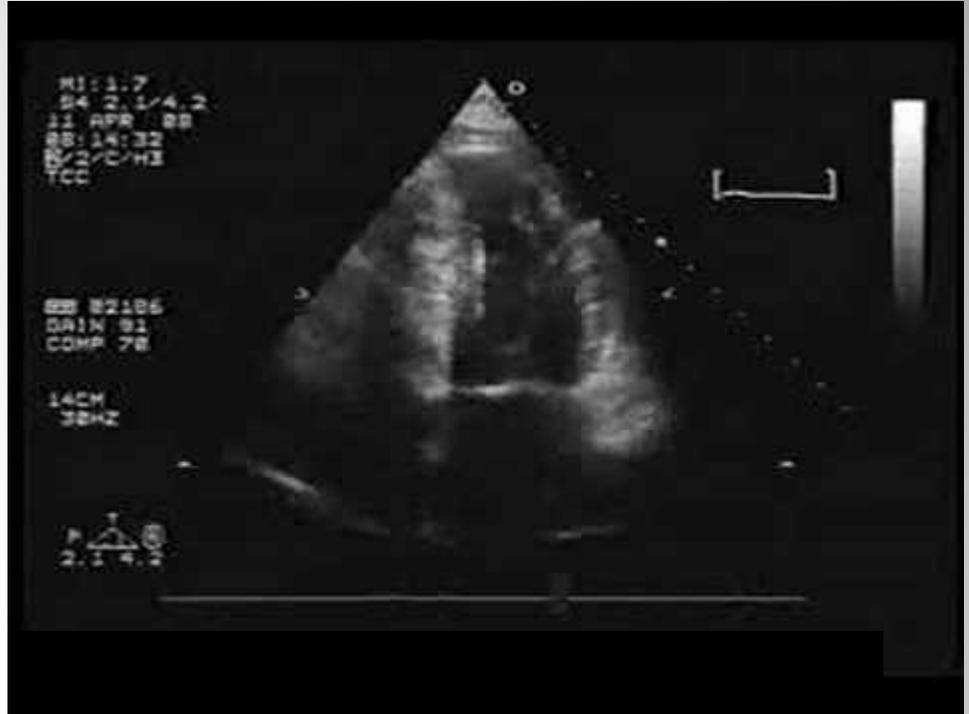
FIESTA:60
TR:3.9
TE:1.7
EC:1 /1 125kHz
8CARDIAC/FLp+
FOV:35x35
8.0thk/0.0sp/C
390/01.43 /0:08
224x224 /1.00 NEX
EG/SQ/Z512

IPR

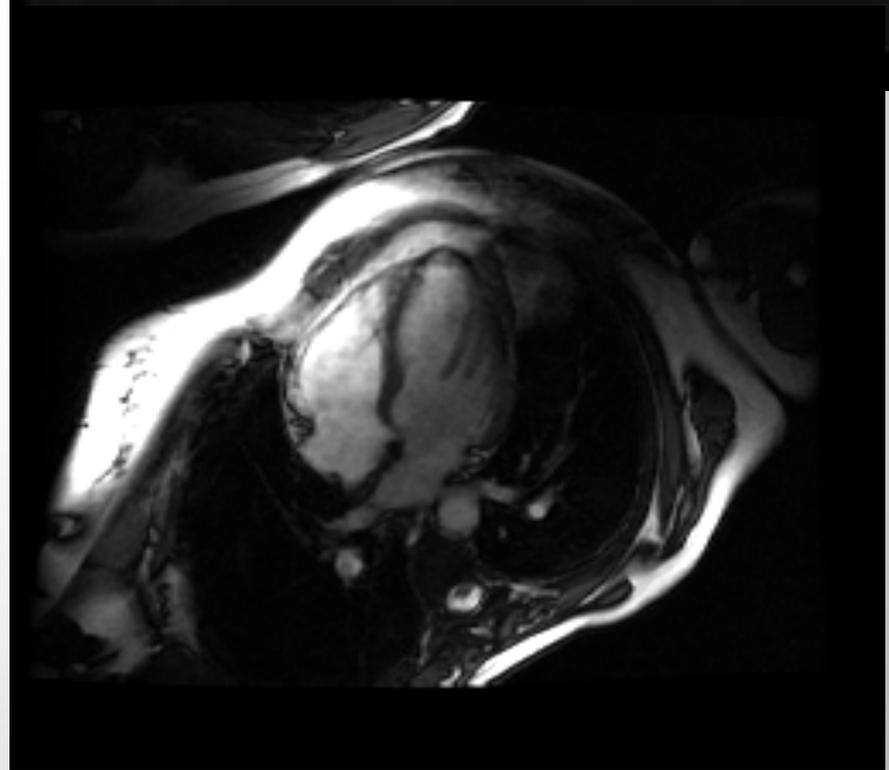
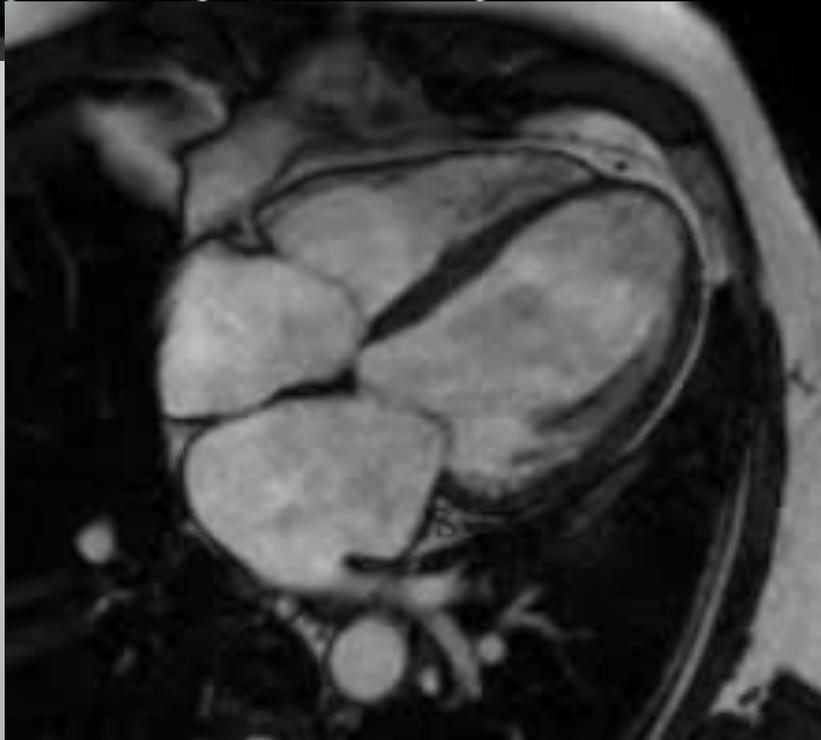
WW: 6290WL: 2567



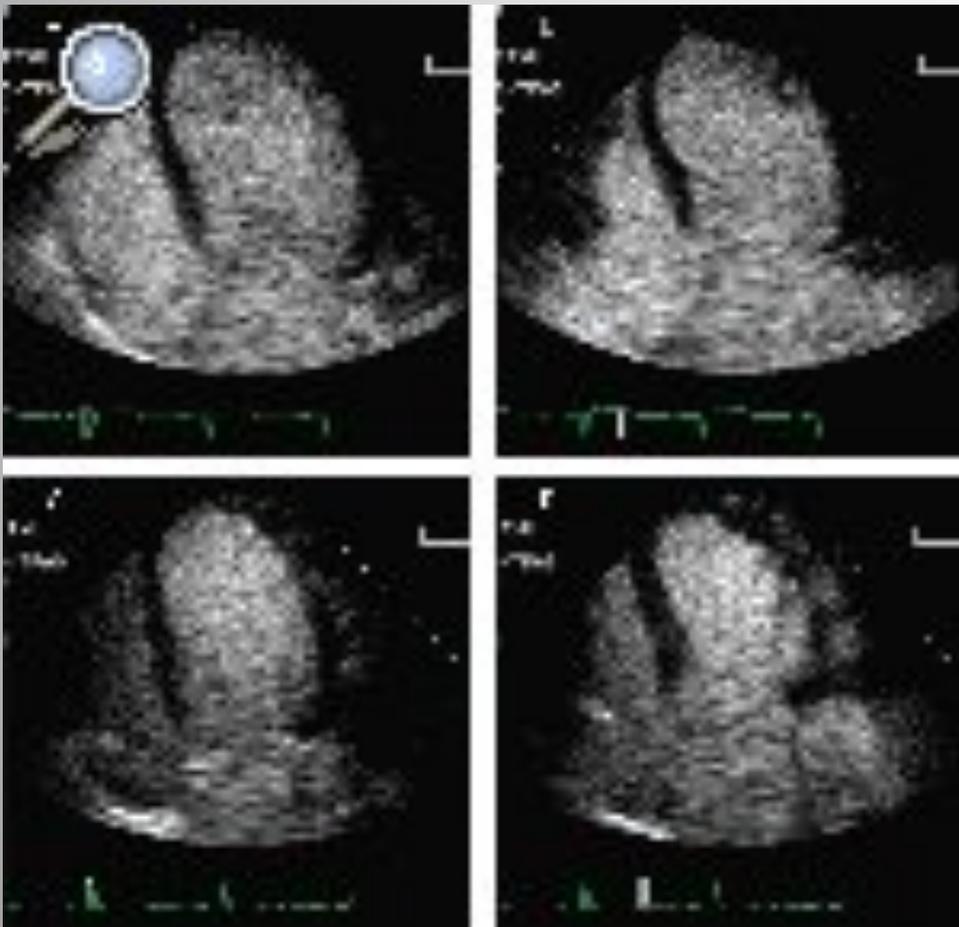
E
C
H
O



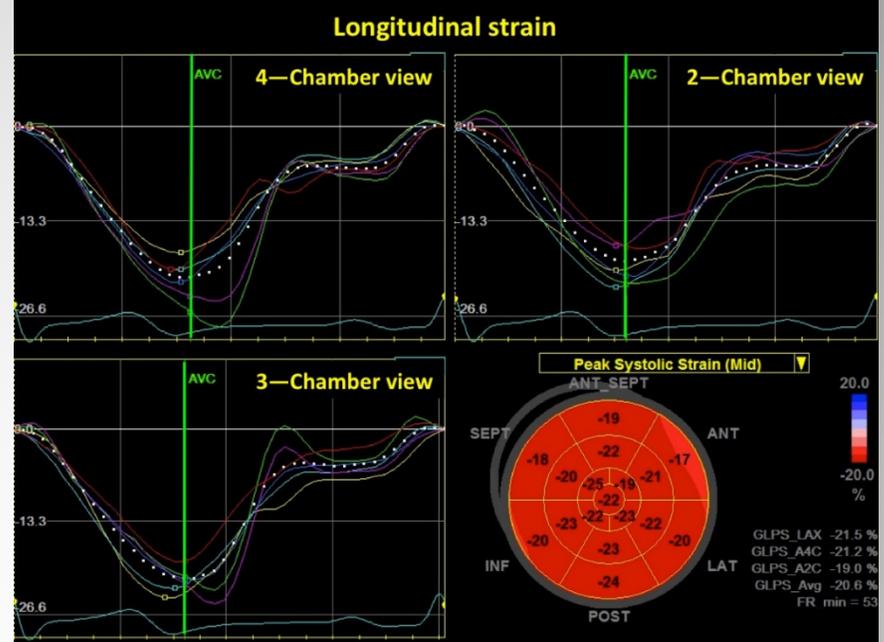
C
R
M



CONTRAST ECHOCARDIOGRAPHY



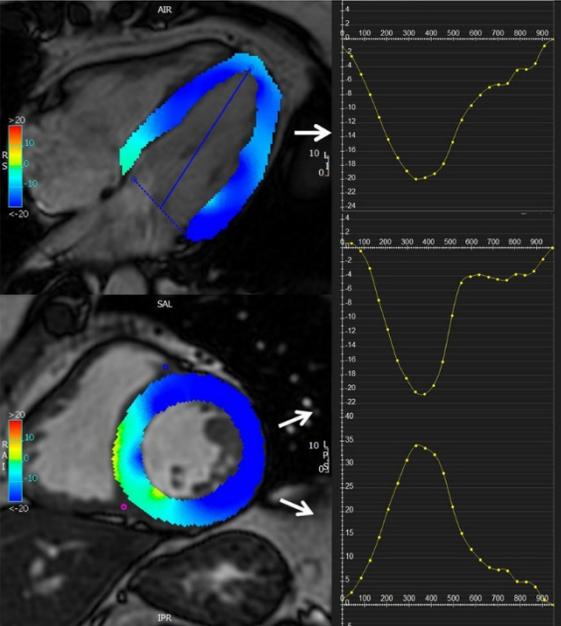
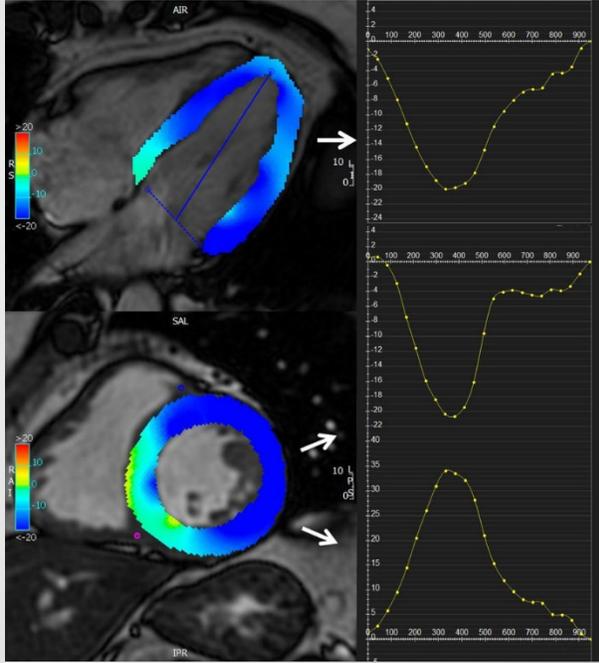
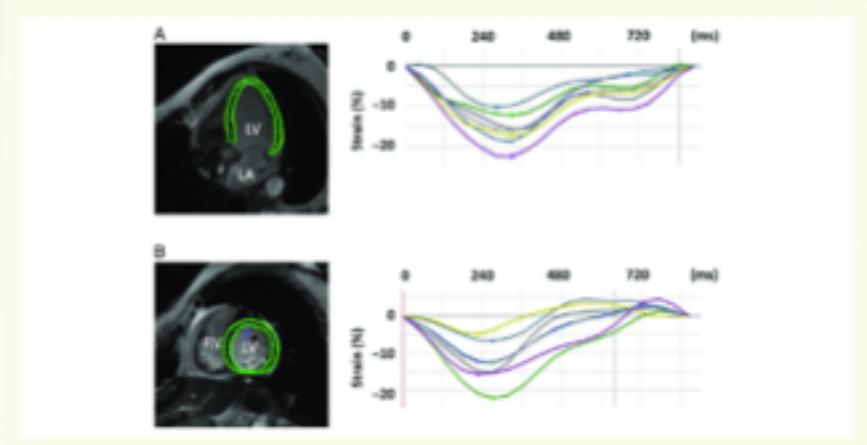
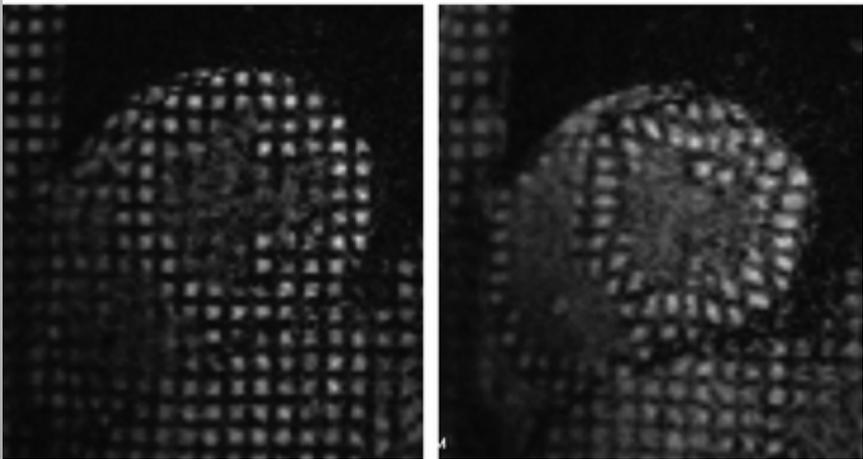
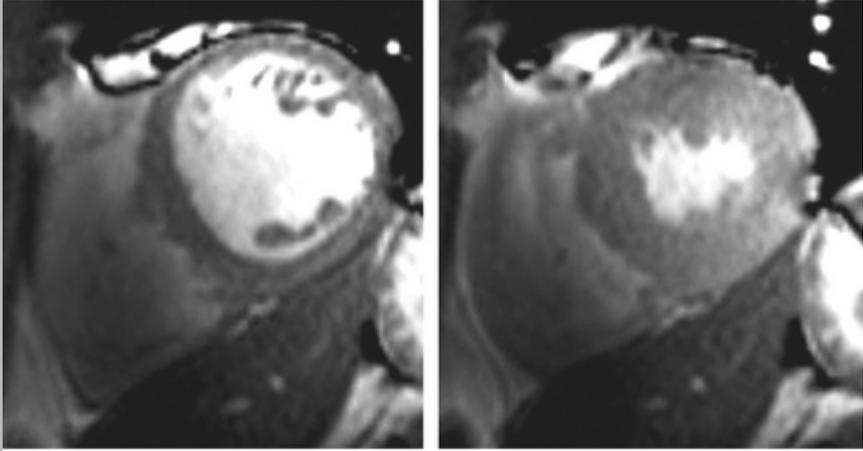
S T R A I N



GLPS_LAX	-14.5 %	GLPS_Avg	-13.2 %
GLPS_A4C	-11.0 %	AVC_AUTO	389 msec
GLPS_A2C	-14.1 %	HR_ApLAX	50.0 bpm

TAGGING

FEATURE TRACKING CMR



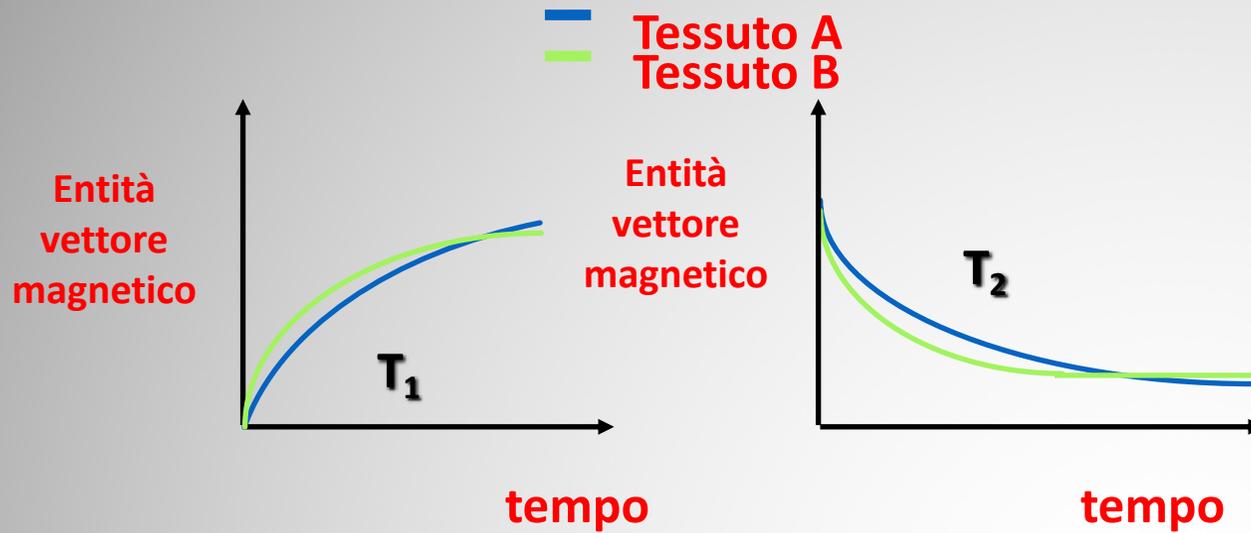


MVO

TISSUE CHARACTERIZATION

**NECROSIS
EXTENTION**

OEDEMA



Tissue Characterization

Table 2. Signal intensities of different tissues on T1- and T2-weighted images

Tissue	T1-weighted image	T2-weighted image
Fat	Bright	Bright
Aqueous liquid	Dark	Bright
Tumor	Dark	Bright
Inflammatory tissue	Dark	Bright
Muscle	Dark	Dark
Connective tissue	Dark	Dark
Hematoma, acute	Dark	Dark
Hematoma, subacute	Bright	Bright
Flowing blood	No signal due to black blood effect (► Chapter 7.2)	
Fibrous cartilage	Dark	Dark
Hyaline cartilage	Bright	Bright
Compact bone	Dark	Dark
Air	No signal	No signal

	T1	T2
Miocardio	880	75
Sangue	1200	360
Grasso	260	110
Muscolo	880	45
Polmone	820	140

GE MEDICAL SYSTEMS
Signa HDxt GemsGems
Ex: 11372/2013078037
Se: 12
Im: 8+C
O Cor A 83.9
DFOV 35.0cm
57 bpm
TD:651
Ph:1/1

SAL

Osp Molinette

ET:32

OEDEMA



R
A
I

L
P
S

FSE-XL/90
TR:2105
TE:83.9/EF
EC:1 /1 62.5kHz
TI:150.0
8CARDIAC/FL:p+
FOV:35x31.5
8.0thk/2.0sp
9/03:09 /0:21
256X224/1.00 NEX
EG/ED/SQ/Z512/BSP

WW: 1614WL: 613

IPR

GE MEDICAL SYSTEMS
Signa HDxt GemsGems
Ex: 11372/2013078037
Se: 12
Im: 5+C
O Cor A 108.2
DFOV 35.0cm
57 bpm
TD:651
Ph:1/1

SAL

Osp Molinette

ET:32

OEDEMA



R
A
I

L
P
S

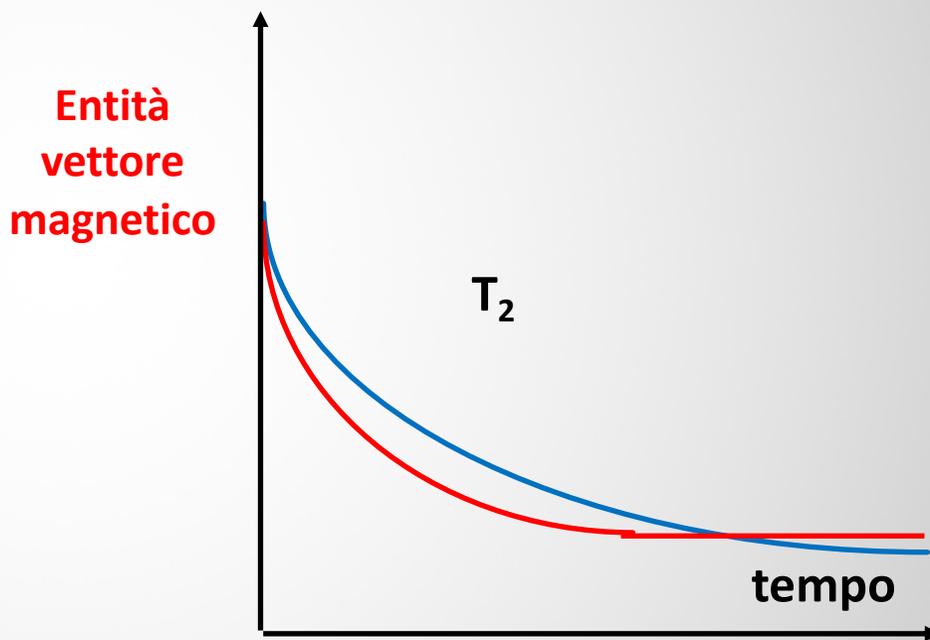
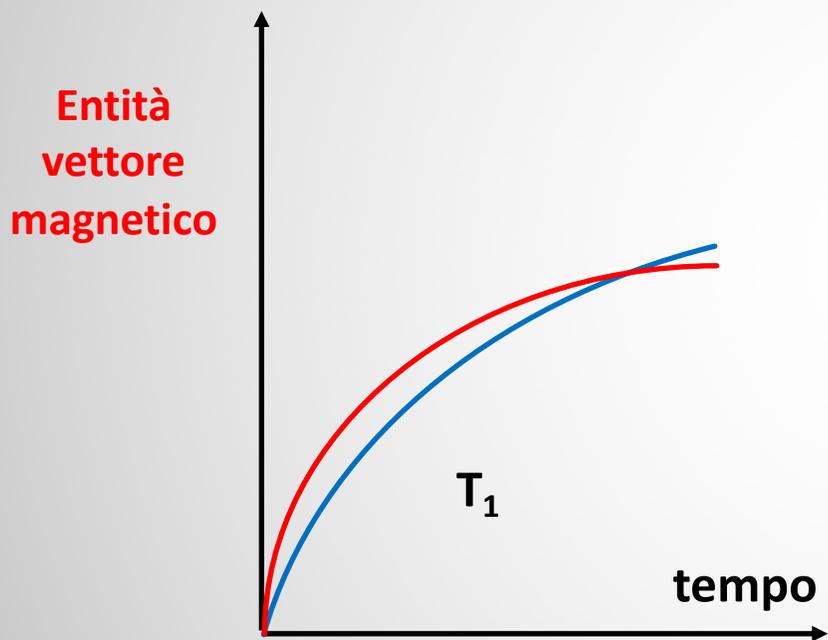
FSE-XL/90
TR:2105
TE:83.9/EF
EC:1 /1 62.5kHz
TI:150.0
8CARDIAC/FL:p+
FOV:35x31.5
8.0thk/2.0sp
9/03:09 /0:21
256X224/1.00 NEX
EG/ED/SQ/Z512/BSP

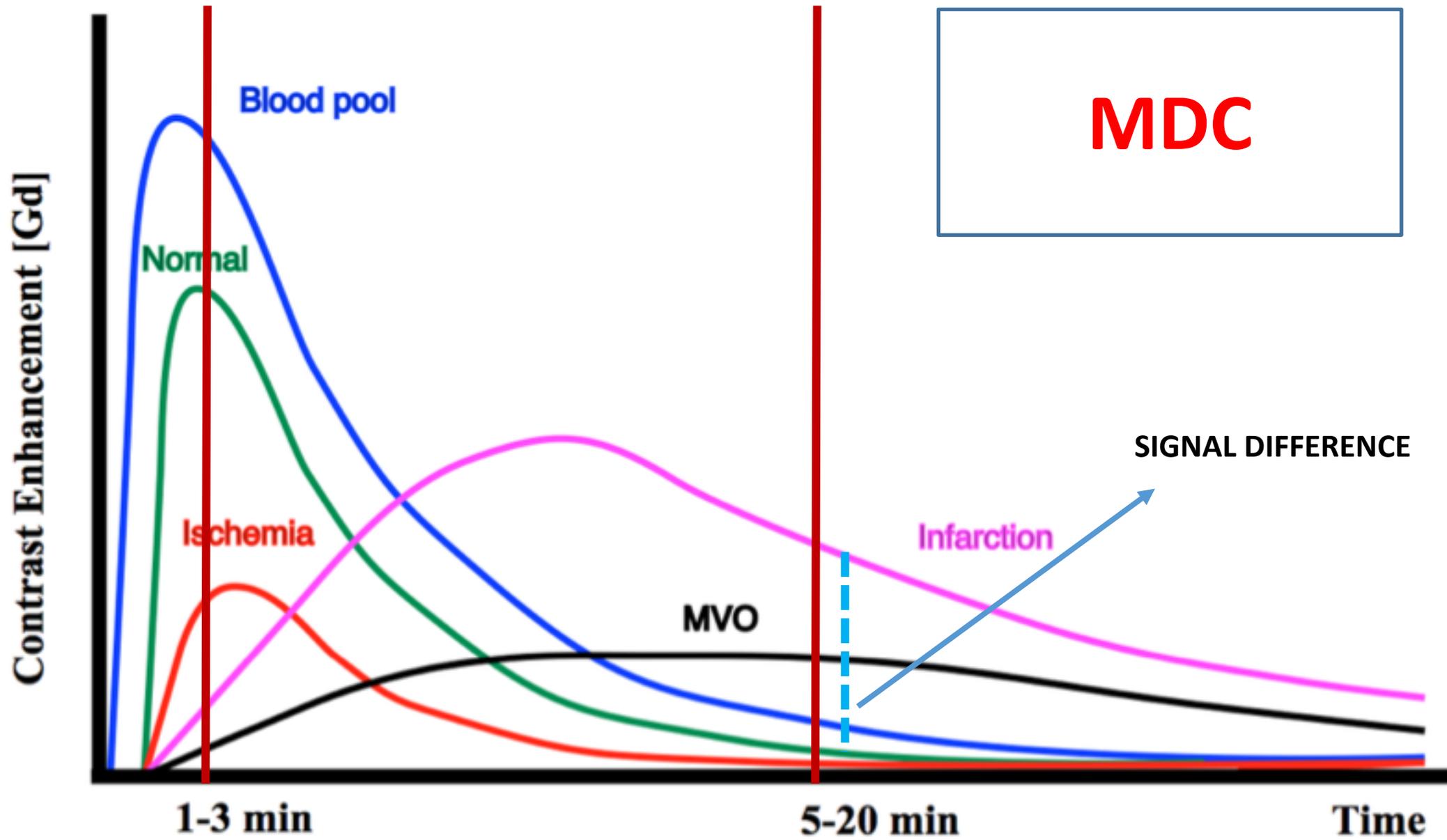
WW: 1614WL: 613

IPR

— Con mdc
— Senza mdc

MDC





Blood pool

MDC

Normal

Ischemia

MVO

Infarction

SIGNAL DIFFERENCE

1-3 min

5-20 min

Time

NECROSIS



GE MEDICAL SYSTEMS
Signal HDxt GemsGe
Ex: 2391 /2010348015

Osp Molinette

Se: 9
Im: 1 +C
O Ax: S 22.6
DFOV: 40.0cm
62 bpm
TD:325
Ph:1/1

**TRANSMURAL
HYPERENHANCEMENT**

R
A VS:24
I

FGR/20
TR:6.4
TE:1.6/Fr
EC:1 /1 20.8kHz
TI:220.0

8CARDIAC
FOV:40x32
8.0thk/8.0sp
1 /0:27

256X160/2.00 NEX
EG/SPF

WW: 380 WL: 190

PSR

ROT:

L
S
P
L

V^

GE MEDICAL SYSTEMS
Signal HDxt GemsGems
Ex: 2391 /2010348015
Se: 11
Im: 6+C
O Ax: 18.0
DFOV: 35.0cm
62 bpm
TD:325
Ph:1/1

SAL

Osp Molinette

VS:24

R
A
I

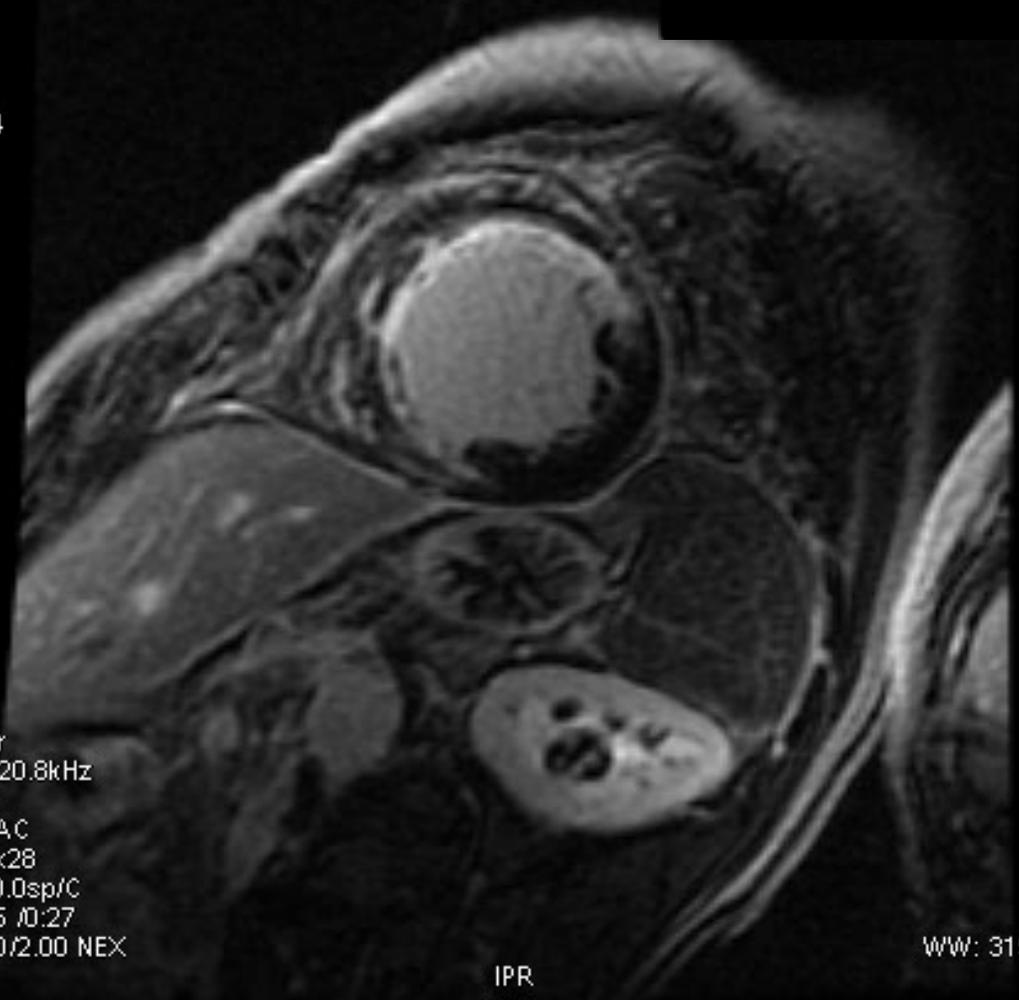
FGR/20
TR:6.4
TE:1.6/Fr
EC:1 /1 20.8kHz
TI:240.0
8CARDIAC
FOV:35x28
8.0thk/0.0sp/C
12/05:25 /0:27
256X160/2.00 NEX
EG/SPF

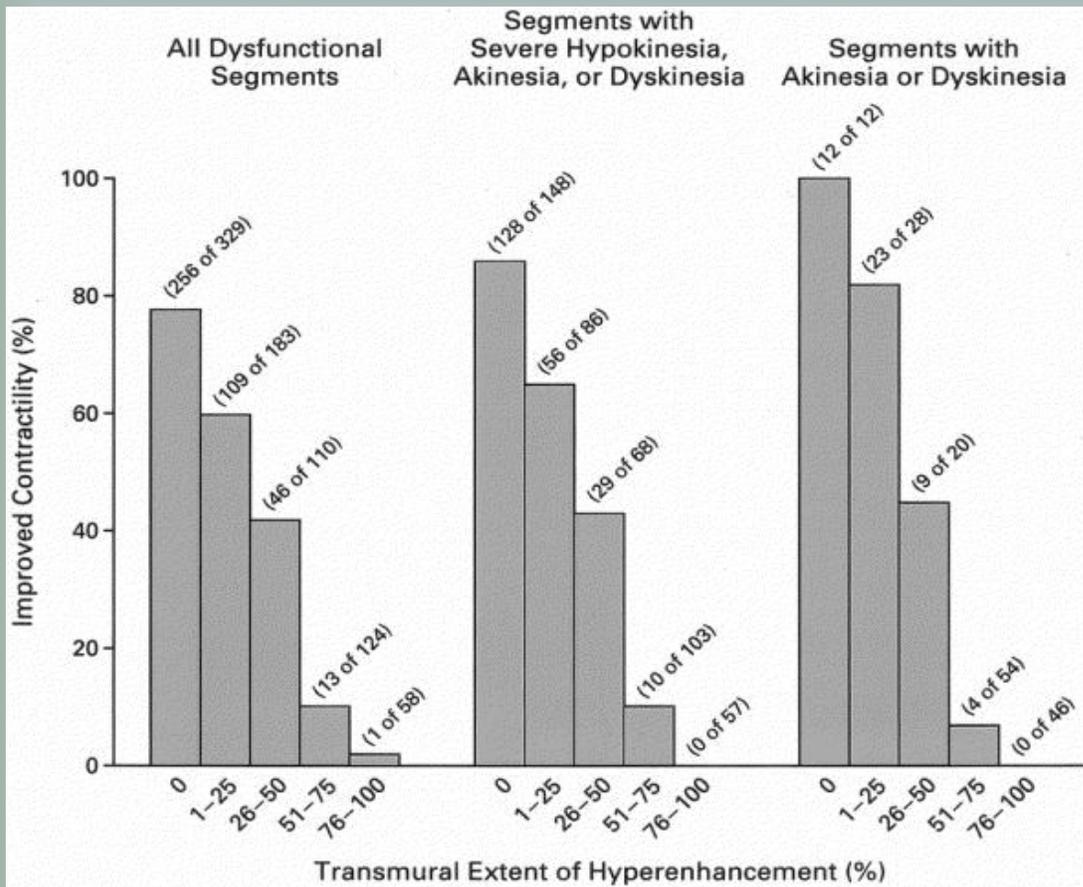
L
S
P
S

V^

WW: 319 WL: 159

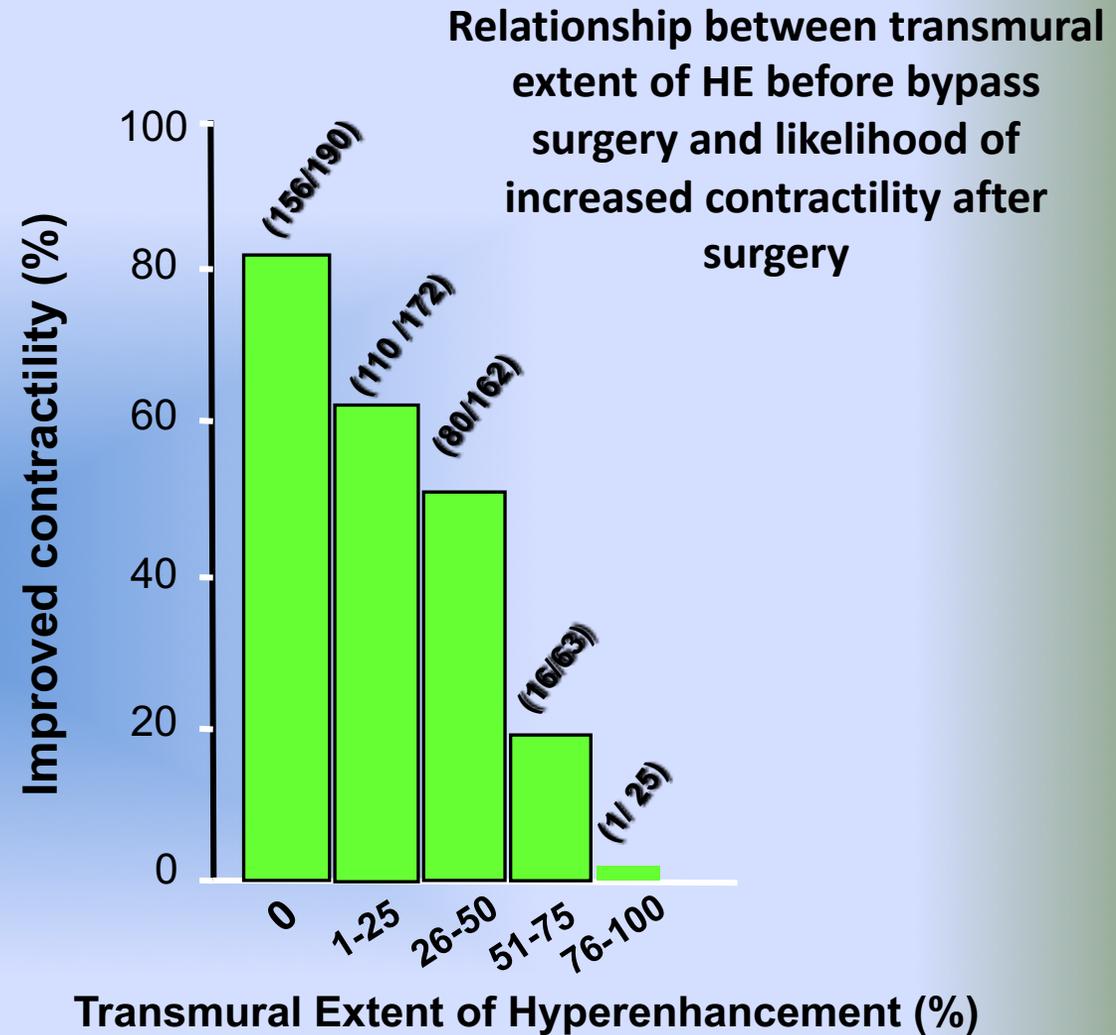
IPR



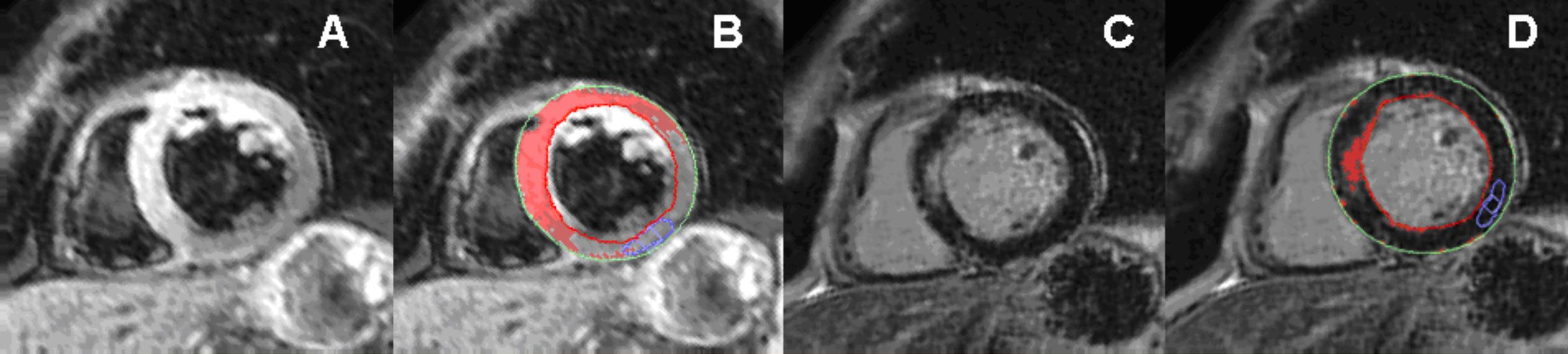


- No hyperenhancement = 78% segments improved
- Only 1 out of 58 segments improved if hyperenhancement > 75%
- Less certain outcome for segments between 25-50%
- Same relationship in segments with most dysfunction
- Recent studies have also shown increased areas of DE indicates worse prognosis

Kim, R et al, NEJM 2000

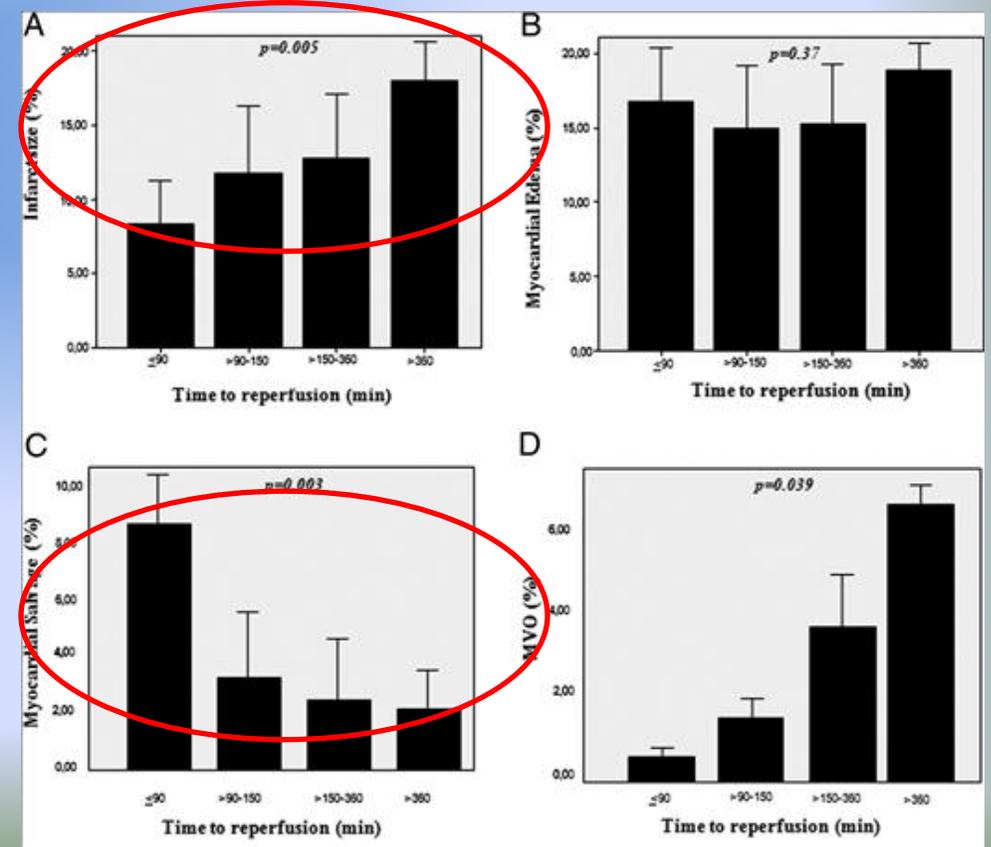


Selvanayagam J et al Circulation 2004



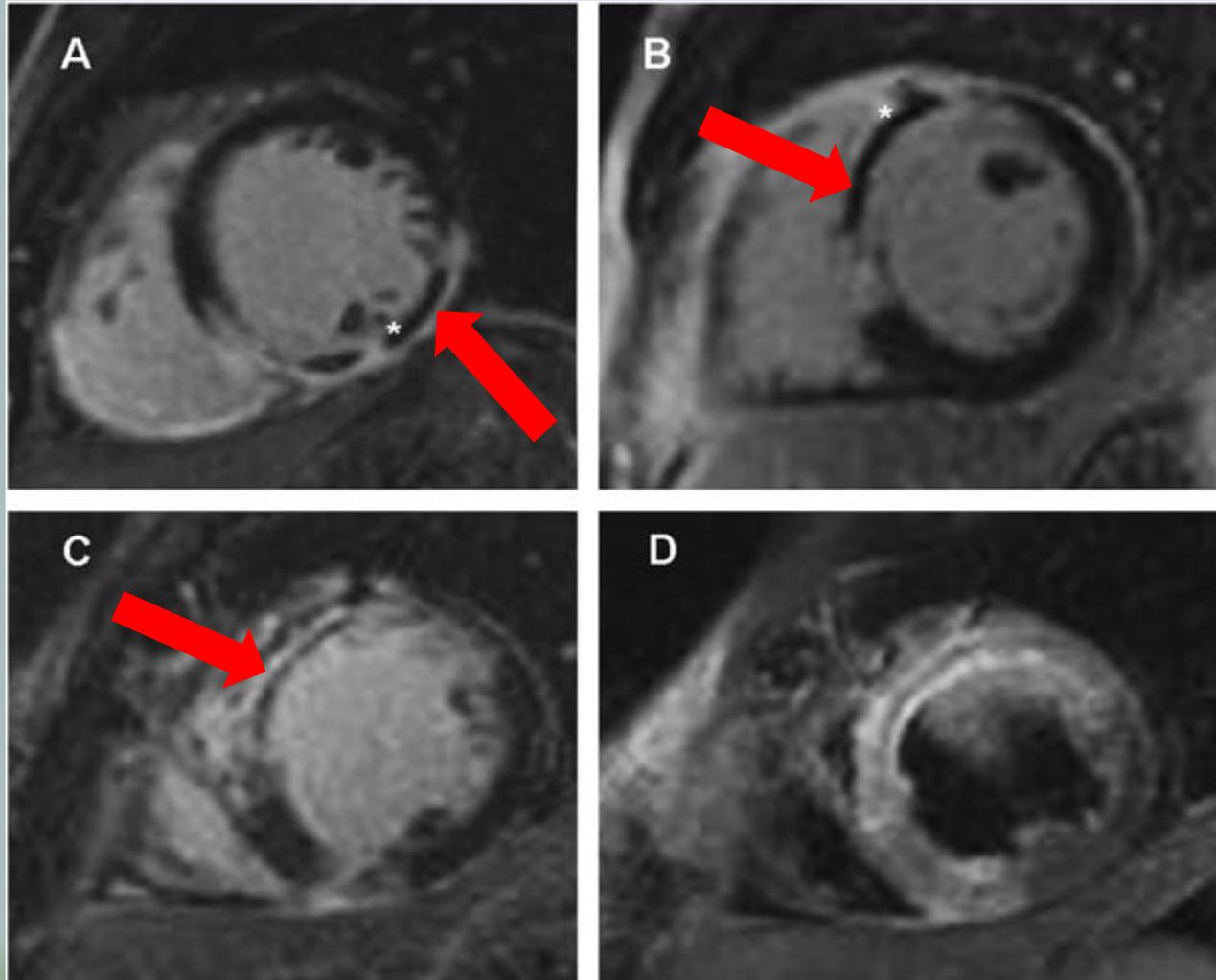
At first, the application of T2-weighted imaging in the clinical setting is used to differentiate acute from chronic myocardial infarction. However, the most important application of MRI ischaemia-related oedema regards the evaluation of 'salvaged myocardium'

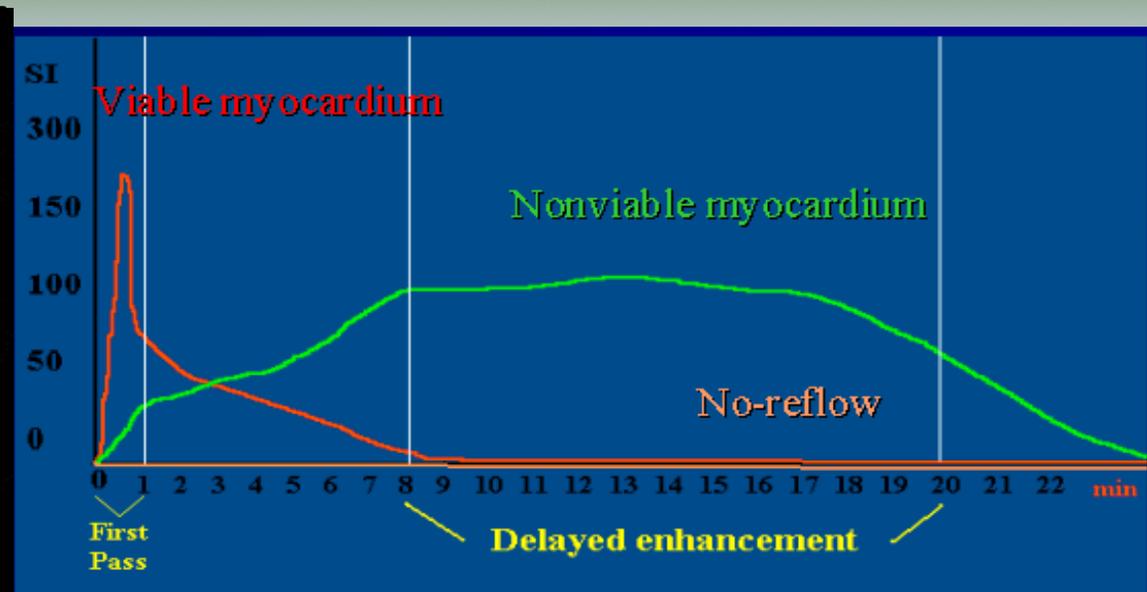
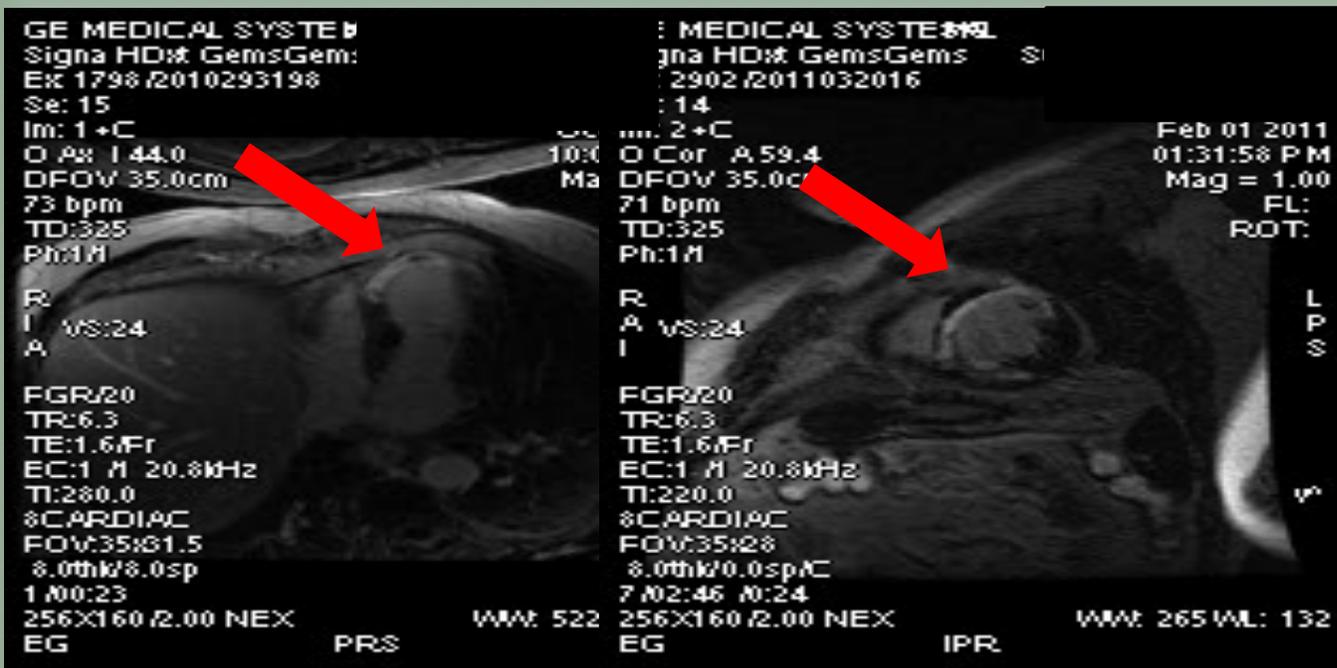
J Am Coll Cardiol. 2009;54(23):2145-2153



MVO

After a prolonged ischaemia the necrosis becomes transmural and as final consequences a microvascular damage may appear inside infarction



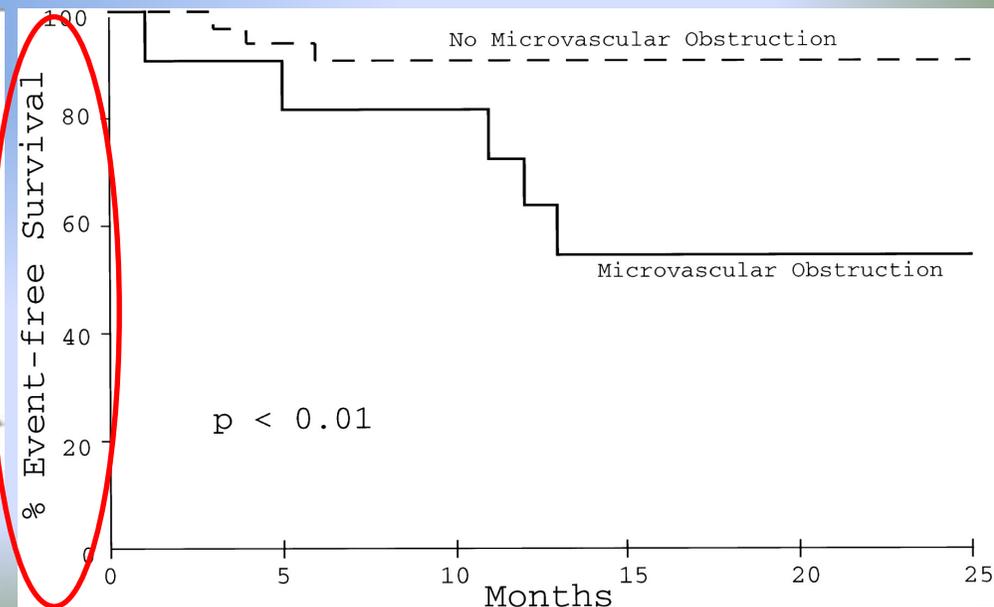
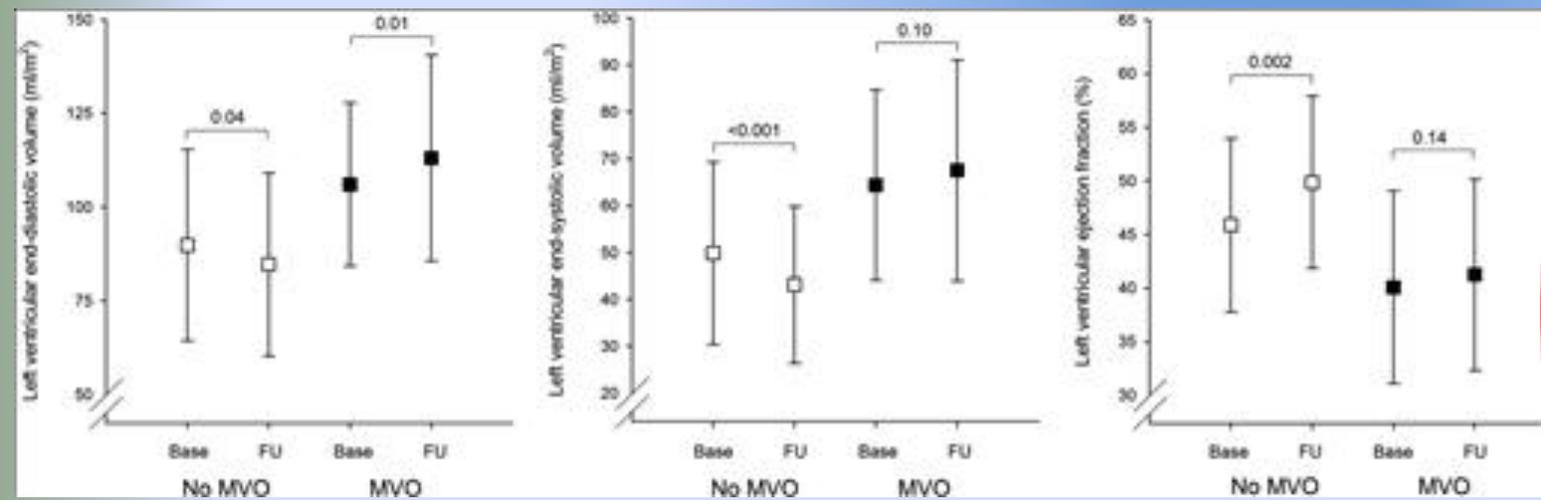


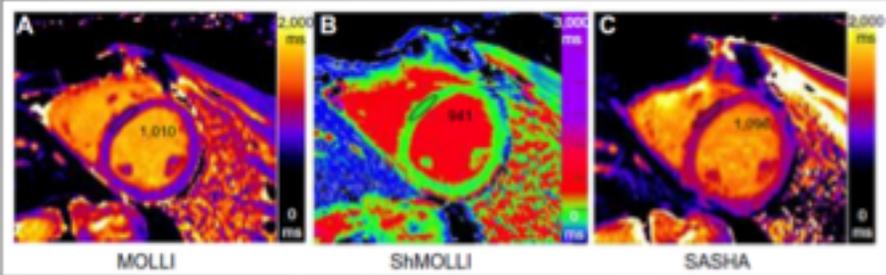
EDV

ESV

EF

Wu KC, Circulation 1998





NATIVE T1 e T2 MAPPING

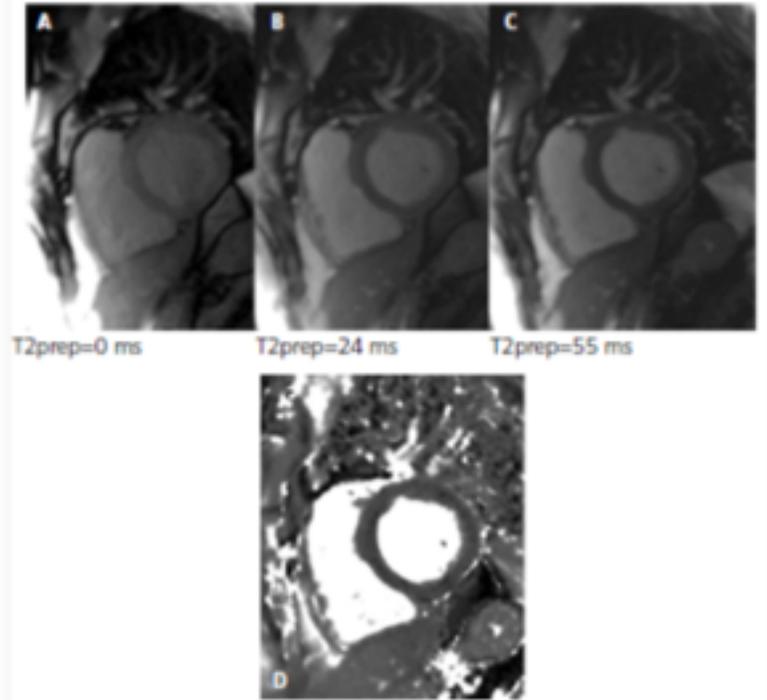
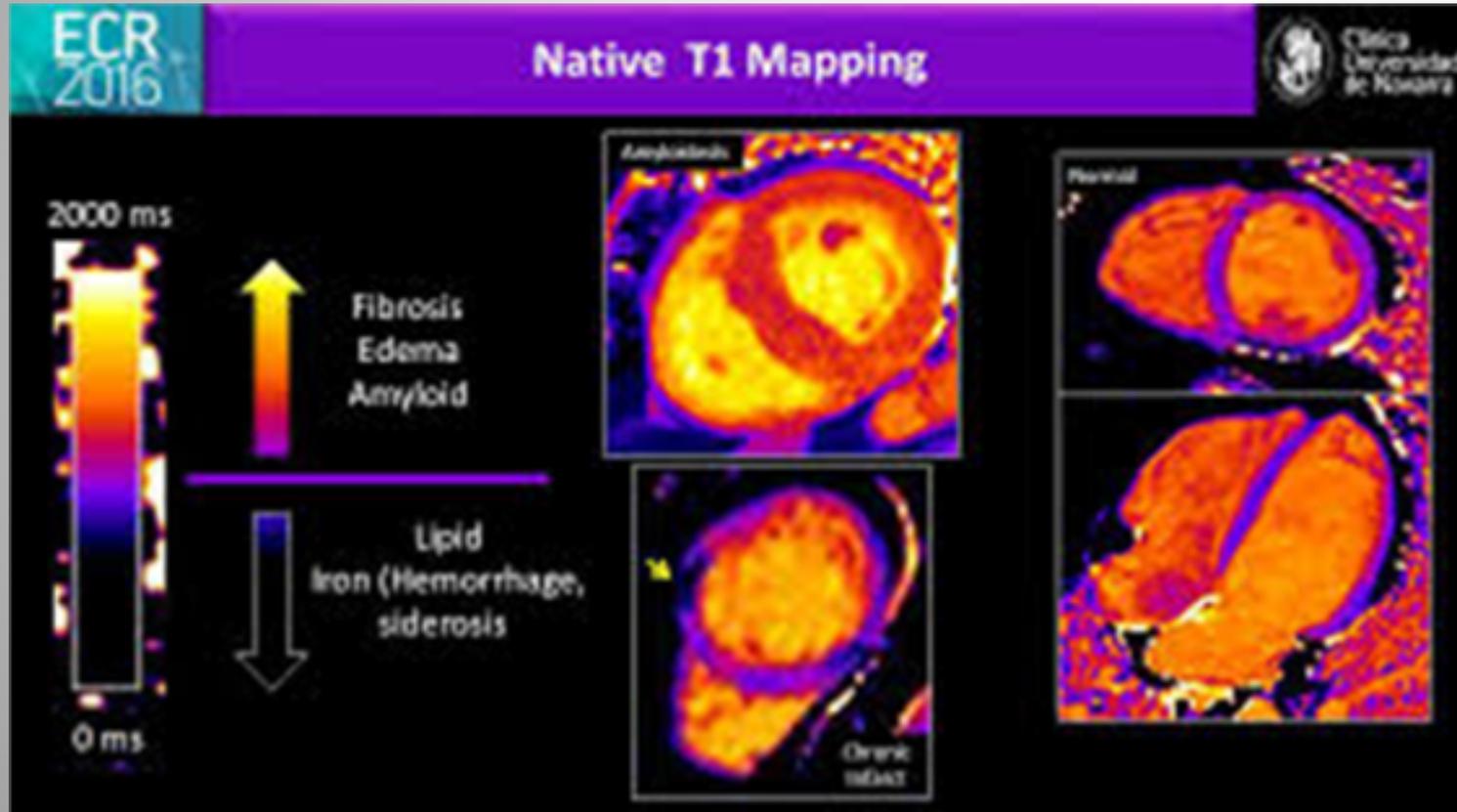


Figura 2. Mappa del T2. Vengono acquisite 3 immagini con differenti tempi di preparazione T2 (A-C), ottenute nella stessa fase diastolica e con un gap di 2 intervalli RR per consentire un sufficiente recupero della magnetizzazione longitudinale (T1). Le immagini acquisite vengono quindi processate per generare una mappa del T2 (D). I colori più chiari nella mappa a colori corrispondono a valori più elevati del T2.

T1 e T2 mapping: nuove prospettive in risonanza magnetica cardiaca
 Carlo Tessa et al.

T1 MAPPING

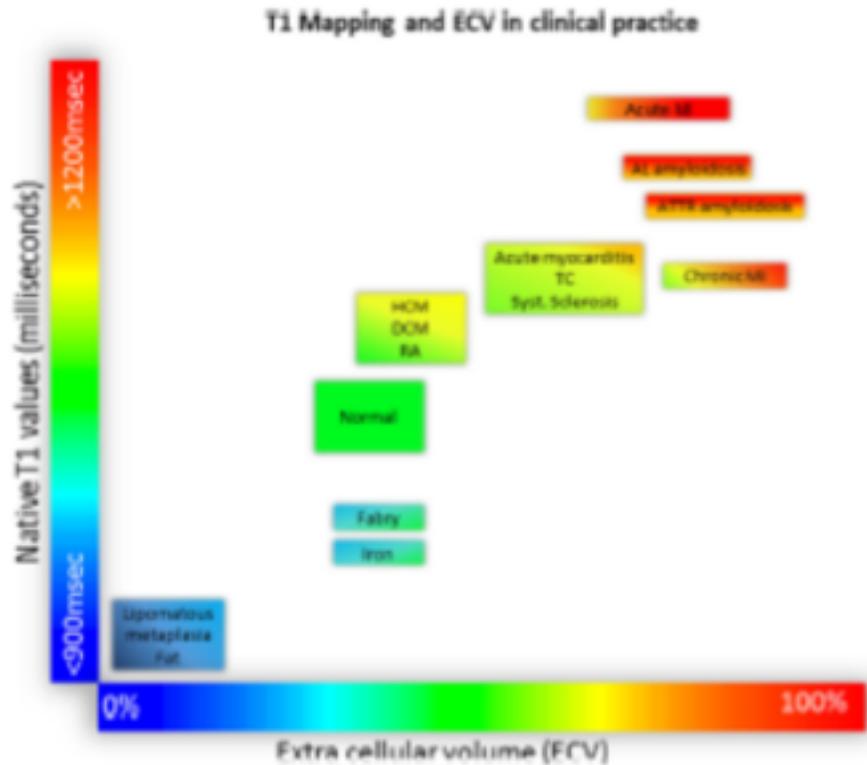
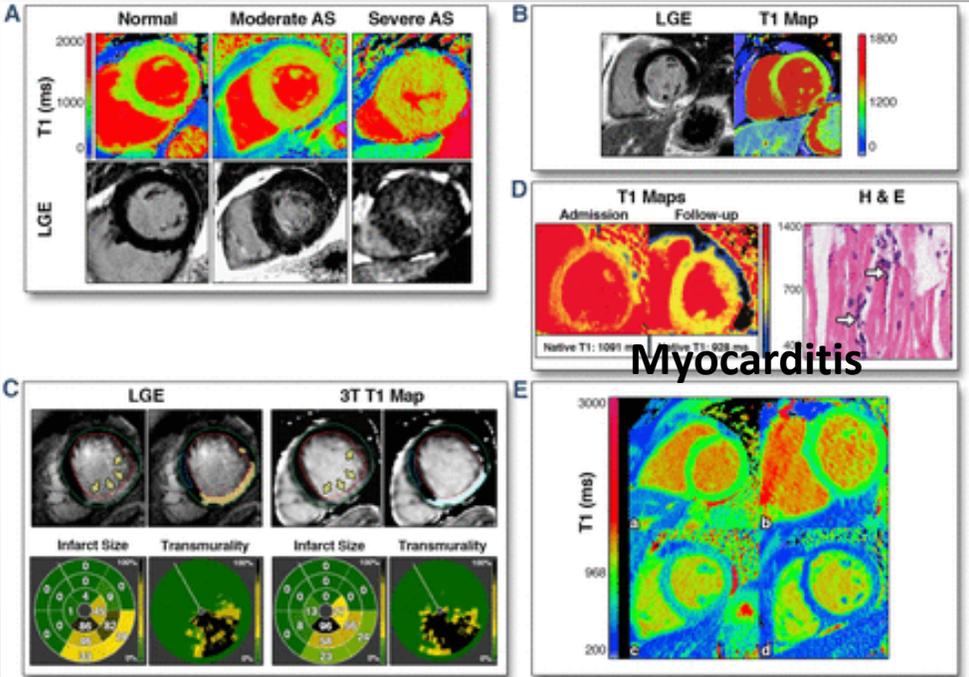


Fig. 2 Tissue characterisation using native T1 and extracellular volume fraction (ECV). Absolute values for native T1 depend greatly on field strength (1.5 T or 3 T), pulse sequence (MOLLI or ShMOLLI), scanner manufacturer and rules of measurements. For the purpose of comparability, only studies using 1.5 T scanners were considered in this figure. Figure adapted from Martin Ugander (SCMR 2014)

Aortic stenosis

Acute myocardial infarction



Myocardial infarction

Iron overload

Cardiac T1 Mapping and Extracellular Volume (ECV) in clinical practice: a comprehensive review
 Haaf et al. Journal of Cardiovascular Magnetic Resonance (2016) 18:89



VITALITY

**INDUCIBLE
ISCHEMIA**



ECHO STRESS PROTOCOLS

Test	Equipment	Protocols
Exercise	Semi-supine bicycle ergometer	25 W x 2' with incremental loading
Dobutamine	Infusion Pump	5 mcg/Kg/min 10-20-30-40 + atropine (0.25 x 4) up to 1 mg
Dipyridamole	Syringe	0.84 mg/Kg in 6' or 0.84 mg/Kg in 10' + atropine (0.25 x 4) up to 1 mg
Adenosine	Syringe	140 mcg/Kg/min in 6'
Pacing	External Pacing	From 100 bpm with increments of 10 beats/min up to target heart rate



ECHO STRESS

Drugs Infusion

DIP 0.84 mg/kg in 6 min

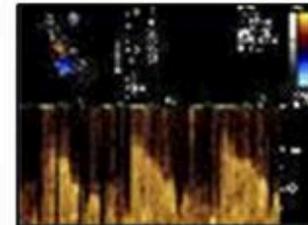
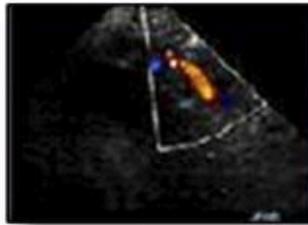
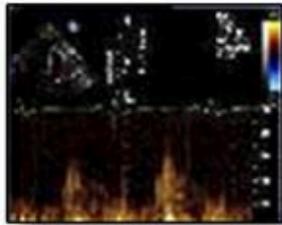
AMINO (up to 240 mg)

SonoVue
bolus (ml)

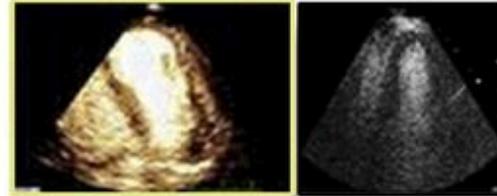
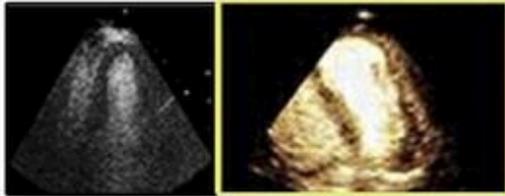


Timeline (min)

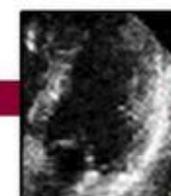
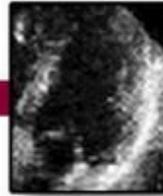
Contrast-LAD



Contrast-WM
and MP

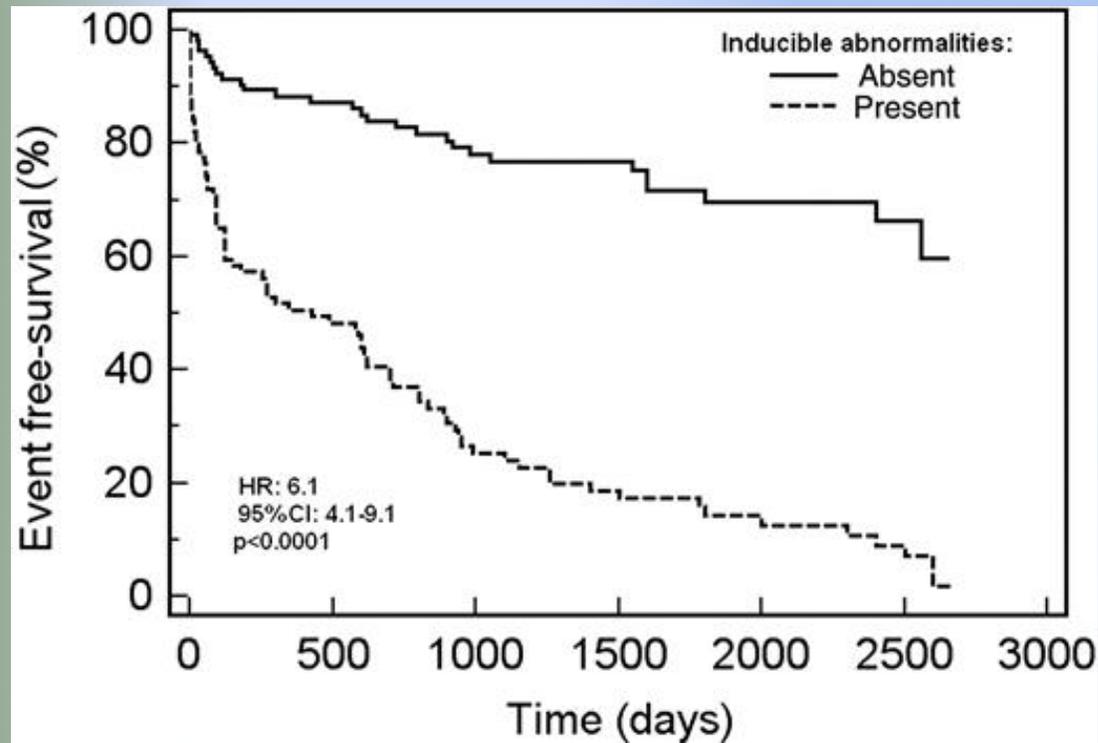


Standard
Echo

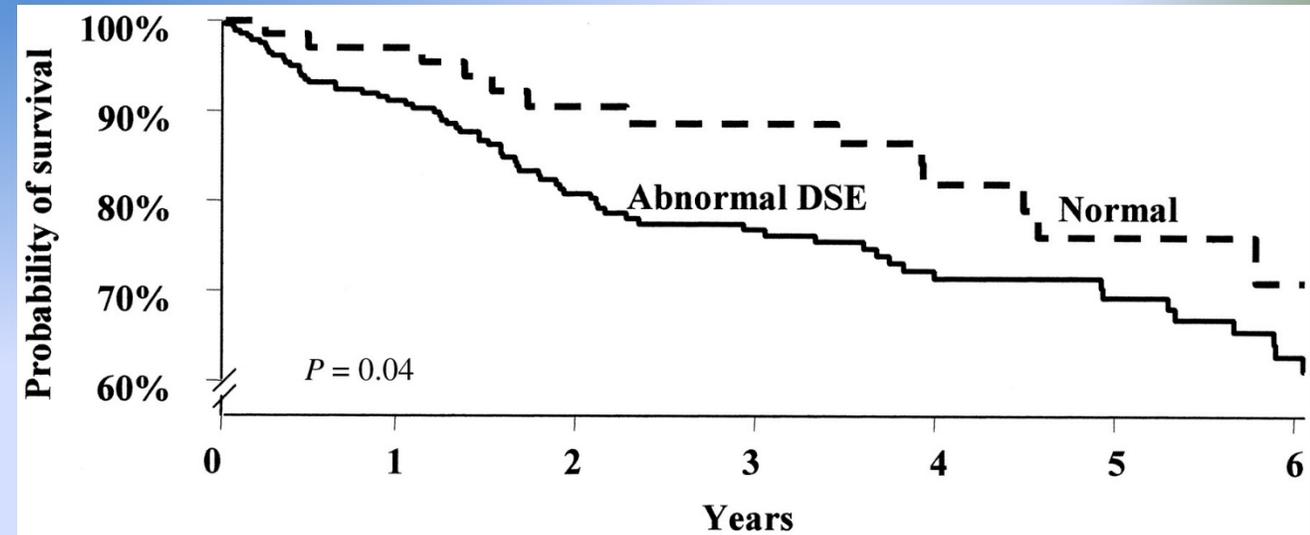
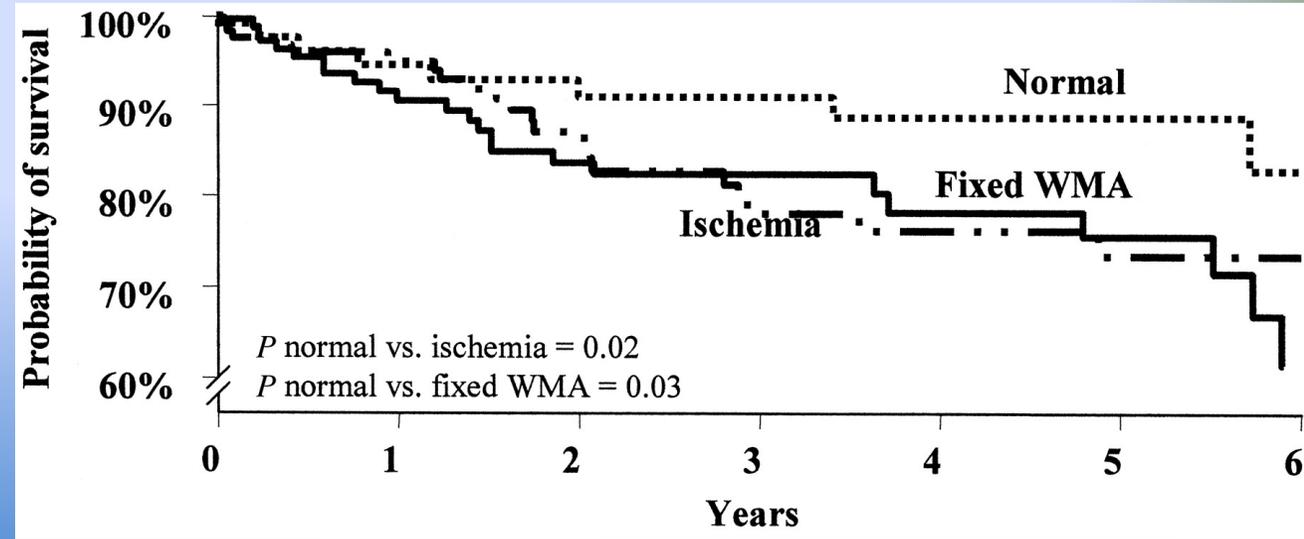


Continuous
Monitoring

ECHO STRESS PROGNOSTIC VALUE

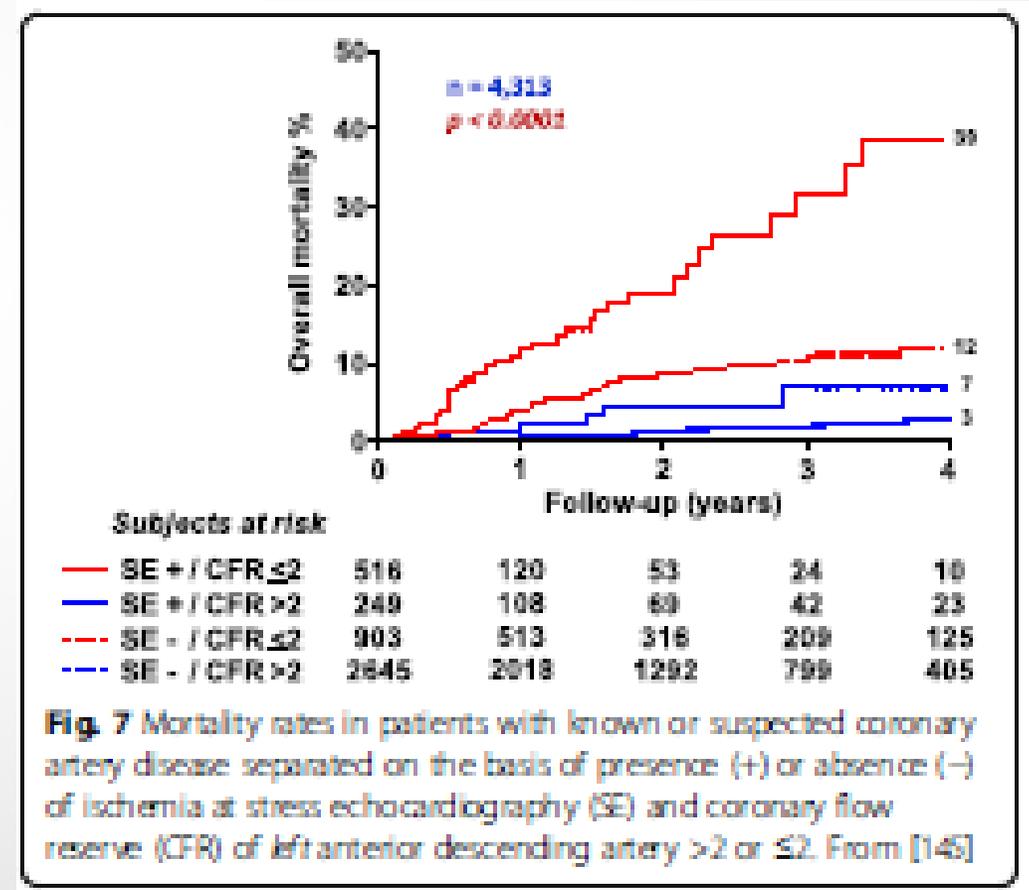
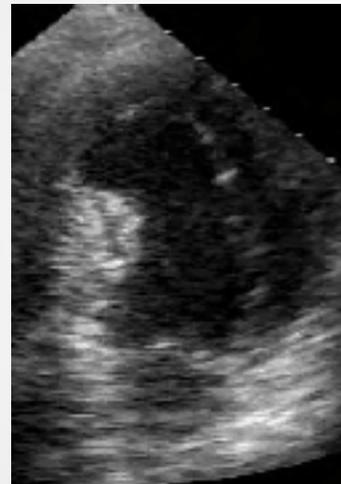
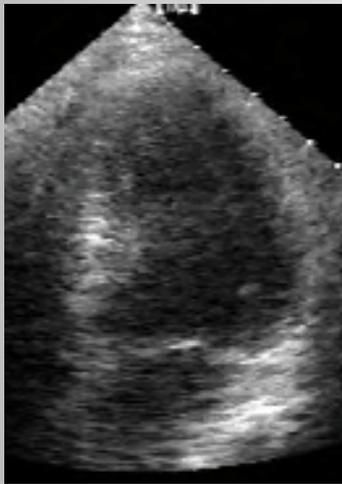
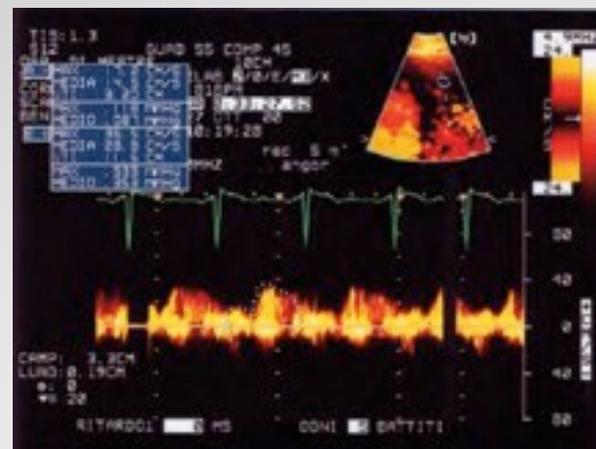


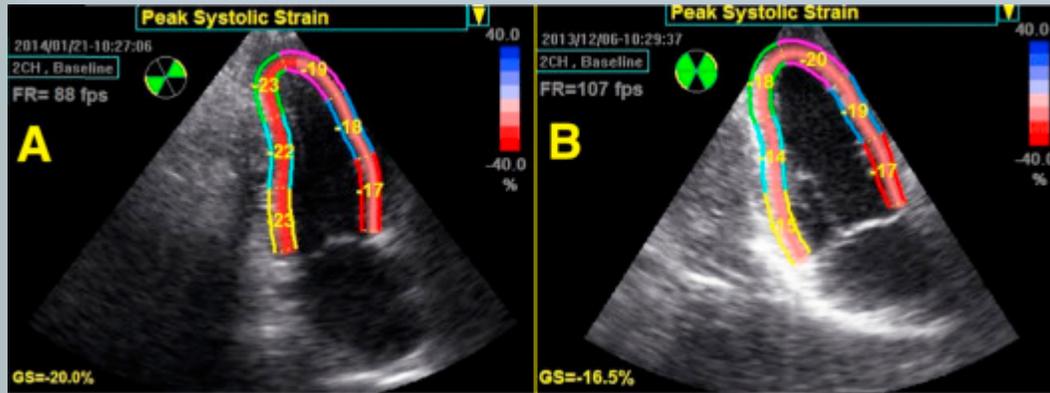
Long-term prognostic value of dipyridamole stress myocardial contrast echocardiography
 Paulina Wejner-Mik Piotr Lipiec Jarosław D. Kasprzak
European Journal of Echocardiography, Volume 12, Issue 10, 1 October 2011,



Prognostic Value of Dobutamine Stress Echocardiography in Patients With Diabetes Fabiola B. Sozzi

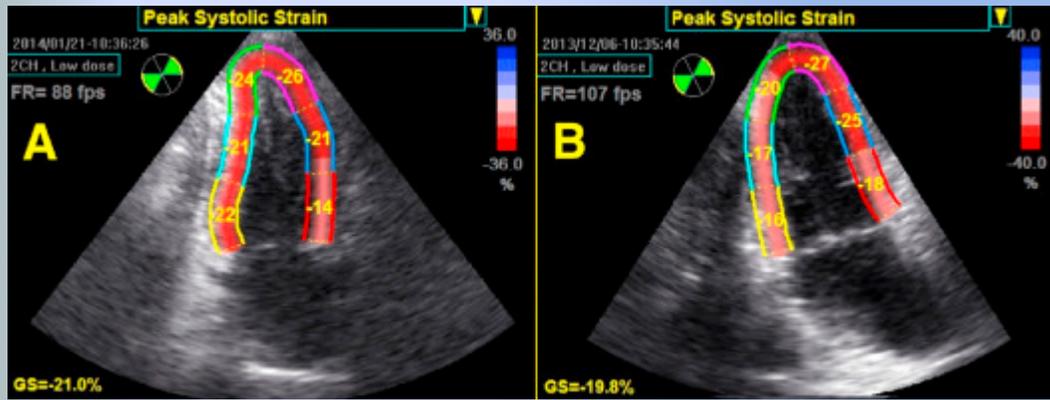
CORONARY RESERVE



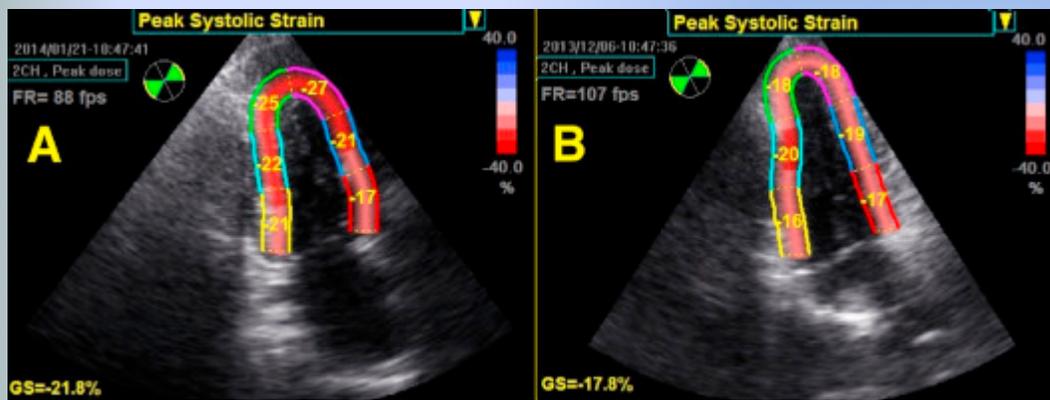


REST

SPECKLE TRACKING E STRESS



LOW DOSE



HIGH DOSE

According to ROC analysis these myocardial deformation parameters had the greatest predictive value of significant coronary artery stenoses: longitudinal strain at high dose (AUC 0.811, sensitivity 89.4%, specificity 64.7%), longitudinal strain rate at high dose (AUC 0.855, sensitivity 88.1%, specificity 71.0% at high doses). The sensitivity and specificity of inducible wall motion abnormalities were 74.0% and 85.0% (AUC 0.798) and was lower compared with the diagnostic value of longitudinal myocardial deformation parameters.

Dobutamine-stress echocardiography speckle-tracking imaging in the assessment of hemodynamic significance of coronary artery stenosis in patients with moderate and high probability of coronary artery disease

STRESS MR

•**CE-MARC Study** (752 patients): Stress CMR could safely be performed in all participants and had a better sensitivity and specificity (86.5/83.4%) than SPECT (66.5/82.6%) for detecting significant coronary artery stenosis
Lancet 2012

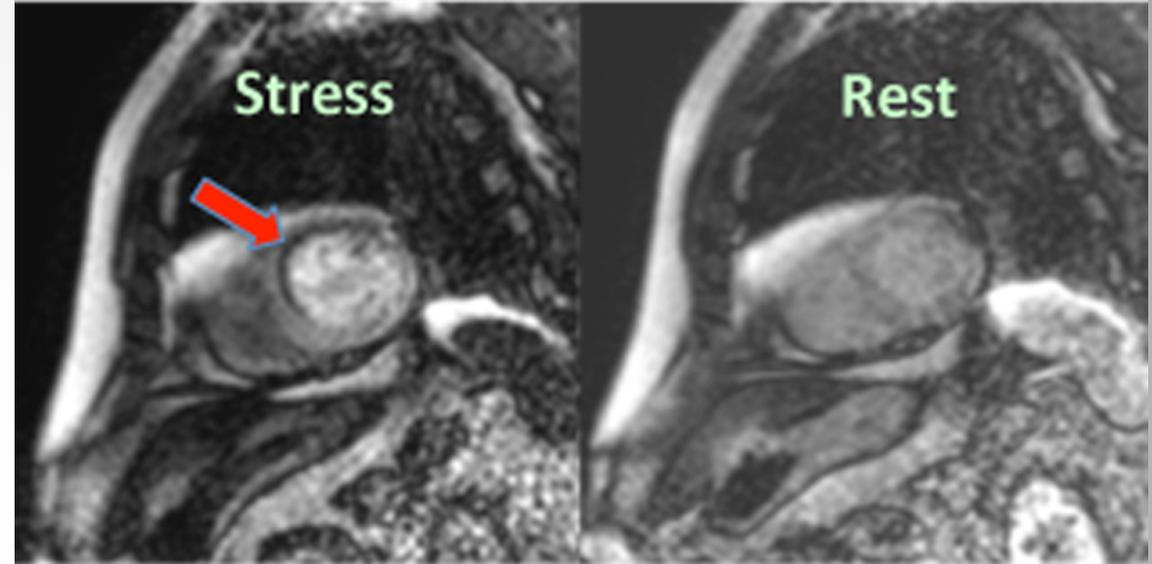
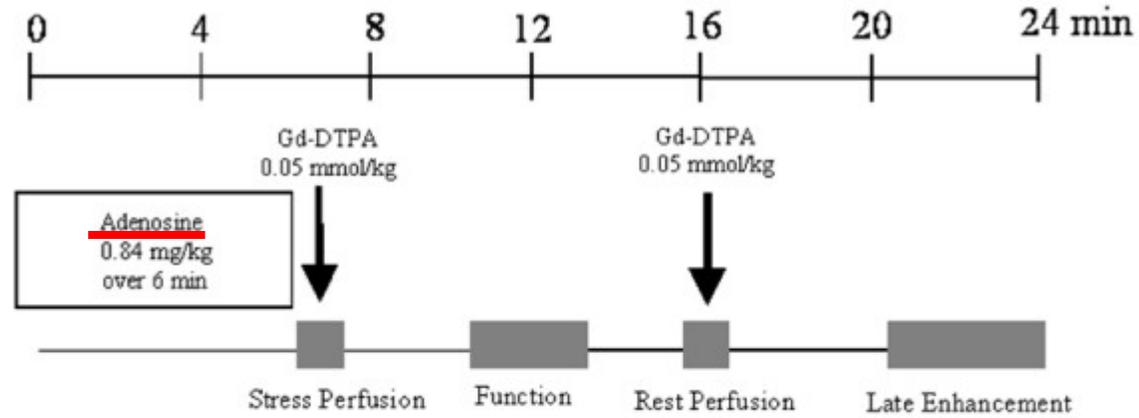
•**MR-INFORM Study** (918 patients): Stress CMR had a similar outcome as invasive FFR for guiding the indication for revascularization, but was associated with a significant reduction of invasive revascularization procedures
ACC March 2017

Symptomatic	Prior testing/abnormal results	Prior testing/uncertain	Follow-up new symptoms	Post PCI/CABG symptomatic
Intermediate pre-test probability of CAD-ECG uninterpretable OR unable to exercise	Abnormal rest ECG findings (potentially ischemic in nature such as LBBB, T-wave inversions) Intermediate to high global CAD risk	Prior exercise ECG test	Normal exercise ECG test	Evaluation of ischemic equivalent
High pre-test probability of CAD-ECG interpretable AND able to exercise	Abnormal prior exercise ECG test	Prior CCTA	Nonobstructive CAD on coronary angiography (invasive or noninvasive) OR normal prior stress imaging study	
High pre-test probability of CAD-ECG uninterpretable OR unable to exercise	Obstructive CAD on prior CCTA study	Coronary stenosis or anatomic abnormality of unclear significance found on cardiac CCTA	Abnormal exercise ECG test	
	Obstructive CAD on prior invasive coronary angiography	Coronary stenosis or anatomic abnormality of unclear significance on previous coronary angiography	Obstructive CAD on CCTA study	
			Abnormal CCTA calcium (Agatston Score >100)	

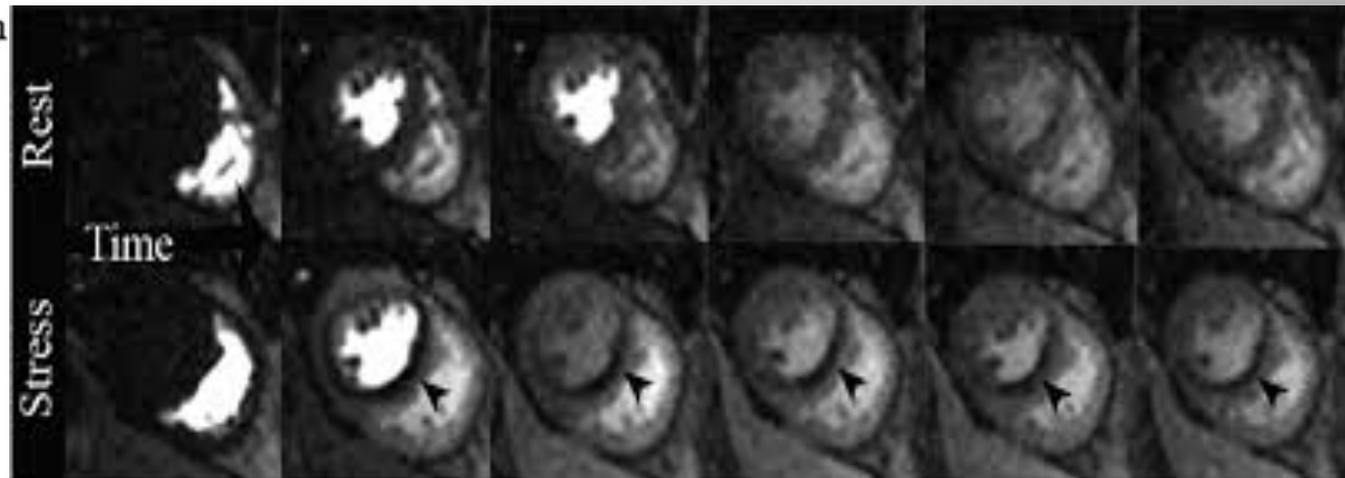
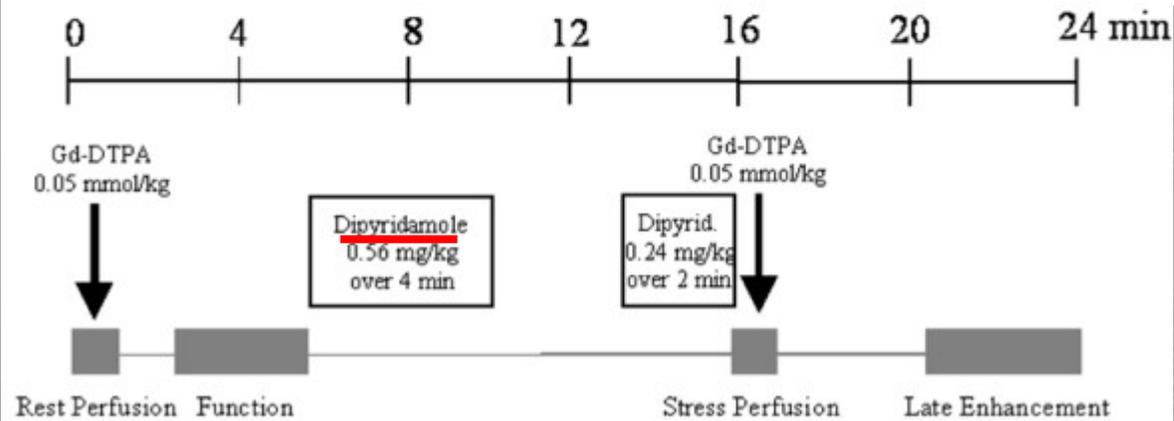


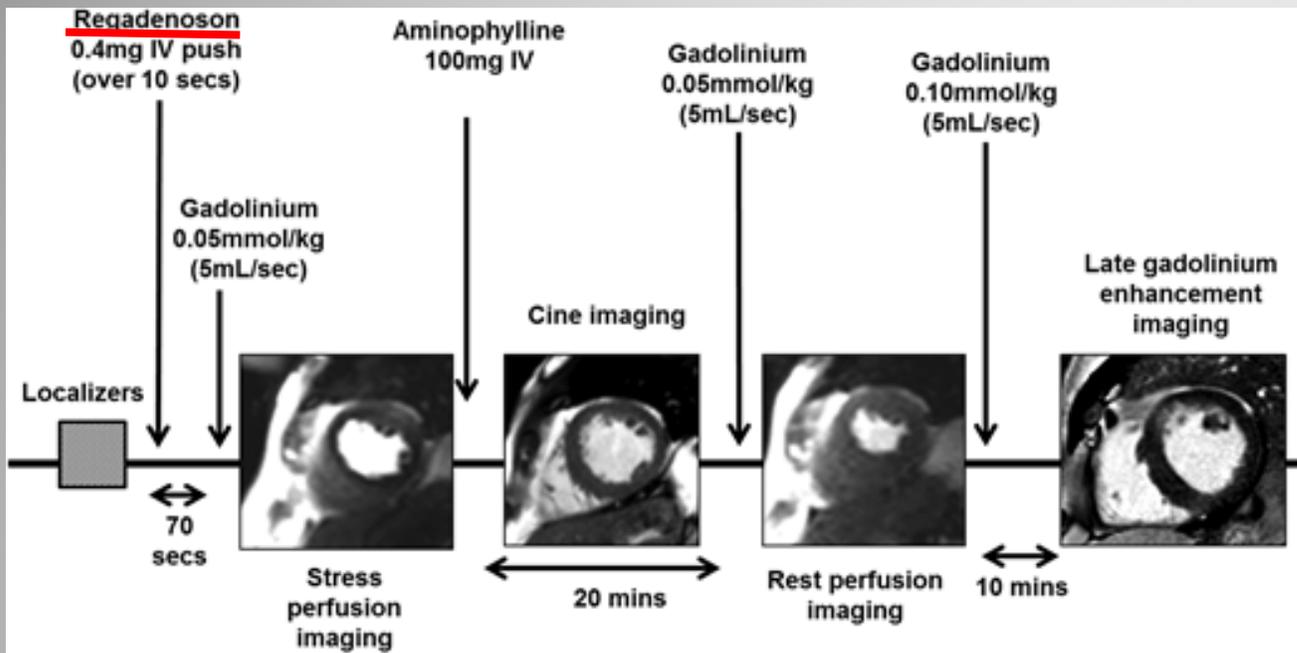
VASODILATORS

(a)

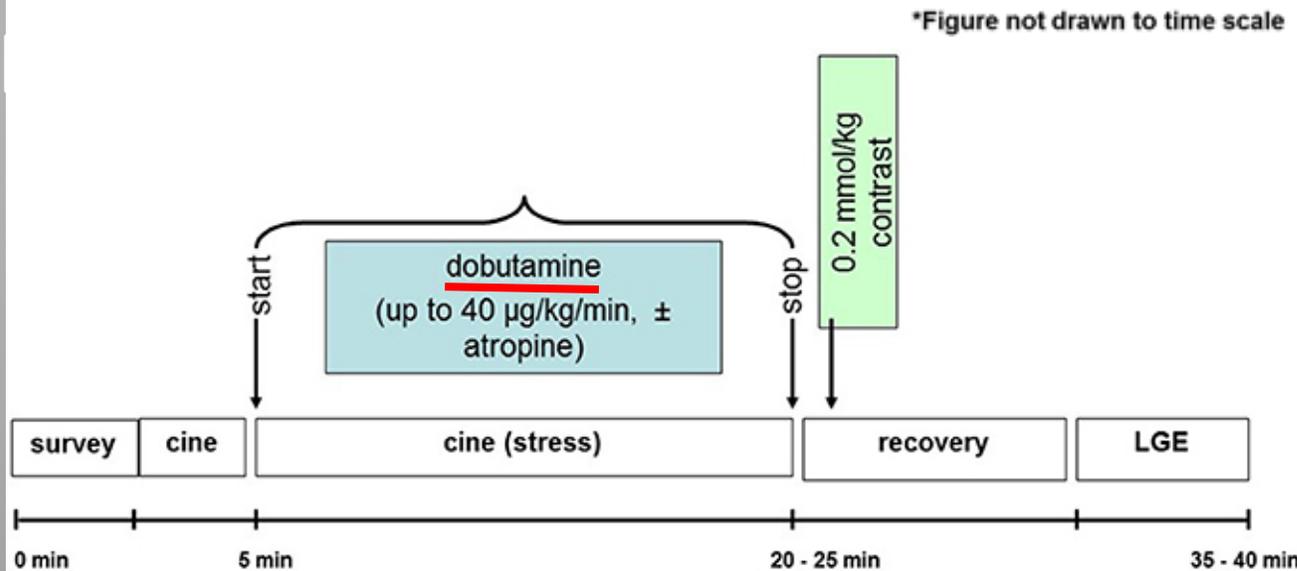


(b)

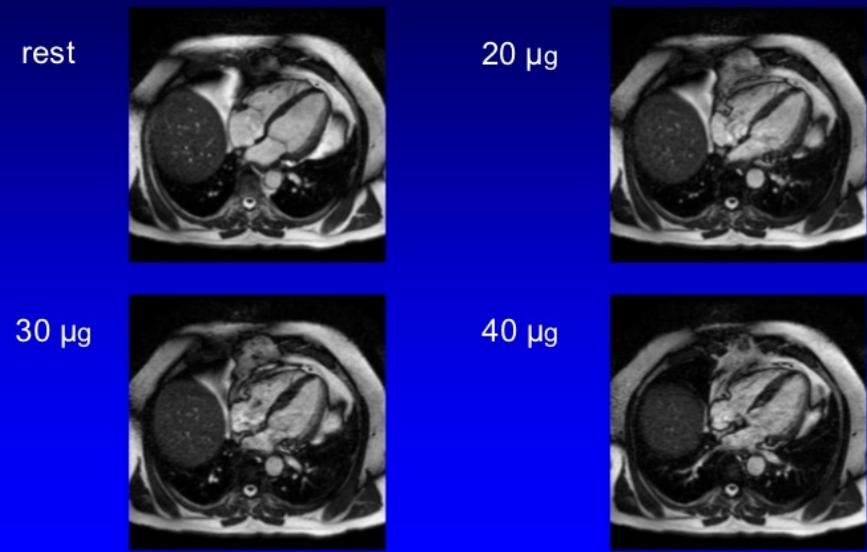




**INOTROPIC
AGENT**



Dobutamine-Stress MR: 4-Chamber



Study	Stressor(s)	Number of patients	MR-scanner	Definition of relevant stenosis (%)	Sensitivity (%) with 95%CI	Specificity (%) with 95%CI
Hundley et al., 1999	Dobutamine/Atropin	41	GE 1.5T	>50	83 (86–93)	83 (36–100)
Jahnke et al., 2006	Dobutamine	40	Philips 1.5T	≥50	83 (51–97)	89 (71–97)
Nagel et al., 1999	Dobutamine	172	Philips 1.5T	≥50	86 (78–92)	86 (75–93)
Paetsch et al., 2004	Dobutamine/Atropin	79	Philips 1.5T	>50	89 (77–96)	81 (61–93)
Paetsch et al., 2006	Dobutamine	150	Philips 1.5T	≥50	78 (67–87)	88 (78–94)
Pennell et al., 1992	Dobutamine	25	Picker 0.5T	≥50	91 (71–99)	100 (29–100)
Rerkpattanapipat et al., 2003	Exercise	27	GE 1.5T	>70	79 (49–95)	85 (55–98)
Schalla et al., 2002	Dobutamine	22	Philips 1.5T	>75	81 (54–96)	83 (36–100)
van Rugge et al., 1993	Dobutamine	45	Philips 1.5T	>50	81 (65–92)	100 (63–100)
van Rugge et al., 1994	Dobutamine	39	Philips 1.5T	≥50	91 (76–98)	83 (36–100)
Pooled data	Dobutamine ± Atropin	680		≥50–75	85 (82–90)	86 (81–91)

78-91% 83-100%

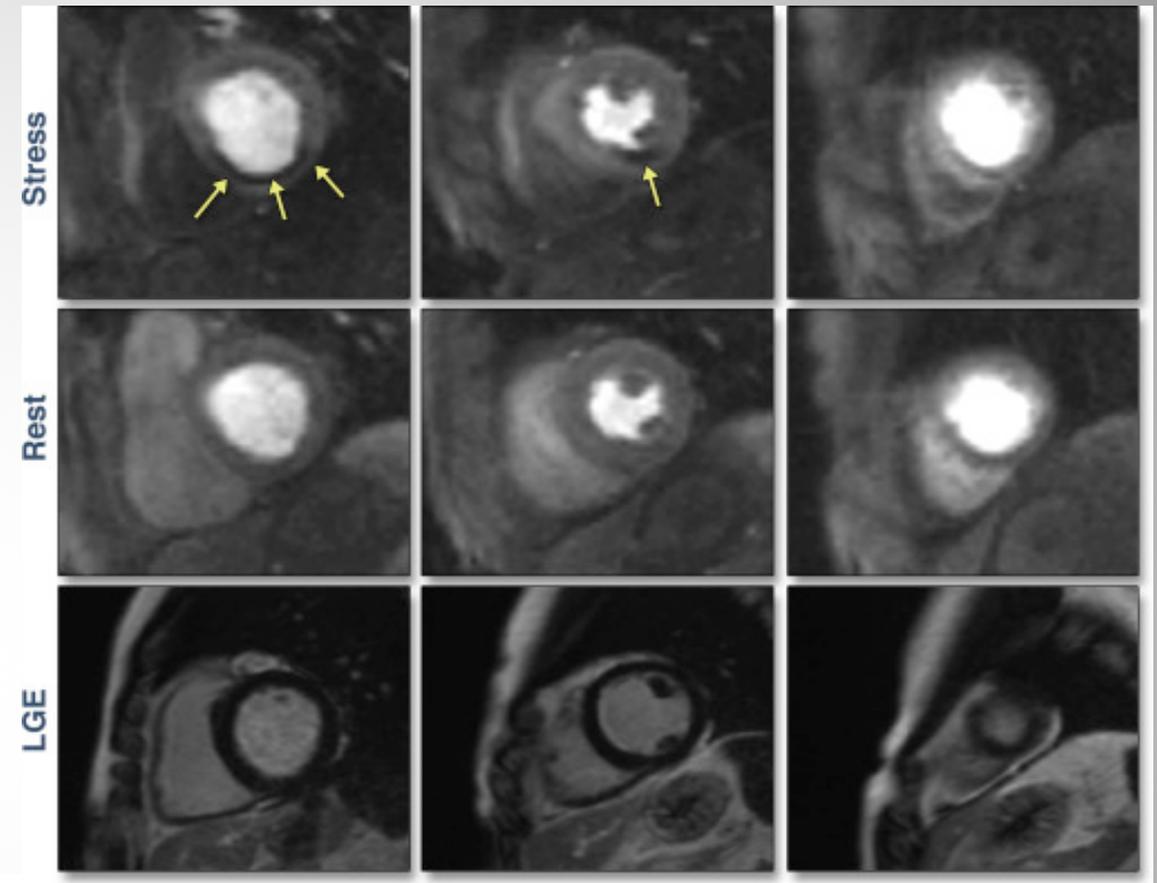
If contractile function improves after inotropic stimulation, it is safe to assume that there is a significant amount of **viability; the converse, however, is not necessarily true**

Study	Stressor(s)	Number of patients	MR-scanner	Definition of relevant stenosis (%)	Sensitivity (%) with 95%CI	Specificity (%) with 95%CI
Cury et al., 2006	Dipyridamole	47	GE 1.5T	≥70	87 (74–94)	89 (80–95)
Doyle et al., 2003	Dipyridamole	199	Philips 1.5T	≥70	58 (37–77)	78 (71–84)
Giang et al., 2004	Adenosine	44	GE 1.5T	≥50	93 (77–99)	75 (48–92)
Pennell et al., 1990	Dipyridamole	40	Picker 0.5T	Not specified	62 (45–77)	100 (3–100)
Ishida et al., 2003	Dipyridamole	104	GE 1.5T	≥70	90 (81–95)	85 (67–94)
Kawase et al., 2004	Nicorandil	50	Philips 1.5T	>70	94 (80–99)	94 (71–100)
Klem et al., 2006	Adenosine	95	Siemens 1.5T	≥70	89 (75–97)	87 (76–95)
Nagel et al., 2003	Adenosine	90	Philips 1.5T	≥75	88 (75–96)	90 (77–97)
Pilz et al., 2006	Adenosine	176	GE 1.5T	>70	96 (91–99)	83 (71–91)
Plein et al., 2004	Adenosine	71	Philips 1.5T	≥70	96 (88–100)	83 (52–98)
Plein et al., 2005	Adenosine	92	Philips 1.5T	>70	88 (77–95)	82 (52–90)
Sakuma et al., 2005	Dipyridamole	40	Siemens 1.5T	>70	81 (58–95)	68 (43–87)
Schwitzer et al., 2001	Dipyridamole	48	GE 1.5T	≥50	87 (71–95)	85 (35–93)
Takase et al., 2004	Dipyridamole	102	GE 1.5T	>50	93 (85–98)	85 (65–96)
Paetsch et al., 2004	Adenosine	79	Philips 1.5T	>50	91 (79–97)	62 (41–80)
Pooled data	Vasodilator stress	1237			91 (88–94)	81 (77–85)

58-96% 75-100%

Vitality/Ischemia vs Necrosis

	Ischaemic but viable myocardium	Non-viable myocardium
Rest perfusion	Normal signal	Signal loss
Stress perfusion	Signal loss	Signal loss
Myocardial delay enhancement	None	Presence

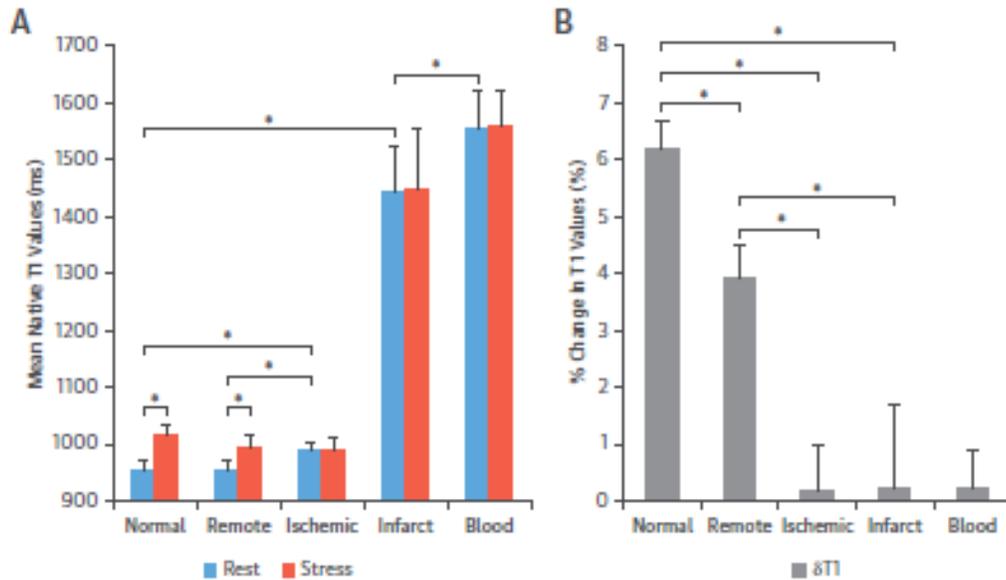


Rest	+	Stress	=	Diagnosis
Normokinesis	+	Normo-Hyperkinesis	=	Normal
Normokinesis	+	Hypo, A, Dyskinesis	=	Ischaemia
Akinesis	+	Hypo, Normokinesis	=	Viable
A-, Dyskinesis	+	A-, Dyskinesis	=	Necrosis

CMR Guidance for Recanalization of Coronary Chronic Total Occlusion
 JACC Cardiovascular Imaging 2016

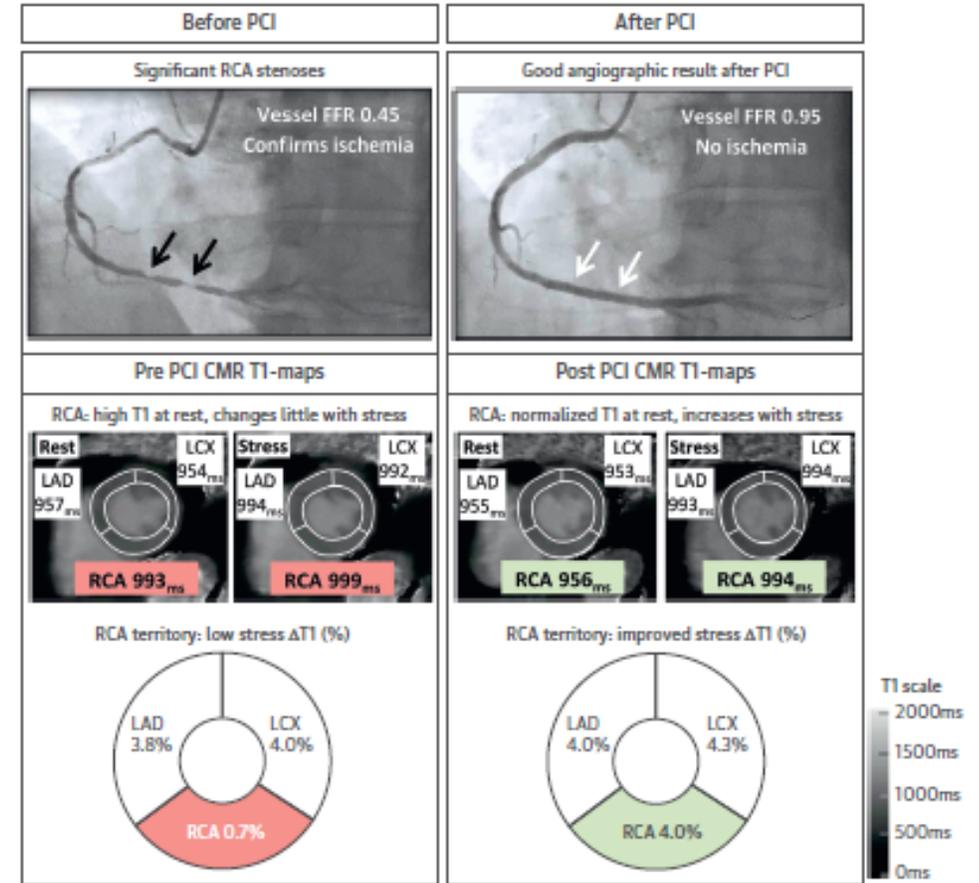
STRESS T1 MAPPING

FIGURE 1 Myocardial T1 at Rest and During Adenosine Stress at 1.5-T



(A) T1 values at rest in normal and remote tissue were similar and significantly lower than in ischemic regions. Infarct T1 was the highest of all myocardial tissue but lower than the reference left ventricular bloodpool of patients. During adenosine stress, normal and remote myocardial T1 increased significantly from baseline, whereas T1 in ischemic and infarcted regions remained relatively unchanged. (B) Relative T1 reactivity ($\Delta T1$) in the patient's remote myocardium was significantly blunted compared with normal and was completely abolished in ischemic and infarcted regions. All data indicate mean \pm 1 SD. * $p < 0.05$. Reprinted with permission from Liu et al. (4).

FIGURE 2 Noninvasive Assessment of Myocardial Ischemia Using Gadolinium-Free CMR Stress T1 Mapping



A 69-year-old male patient presented with angina for 3 months. On angiography, he had 2 significant right coronary artery (RCA) stenoses (black arrows), with a combined vessel fractional flow reserve (FFR) of 0.45, indicating coronary ischemia. The 1.5-T cardiac magnetic resonance (CMR) before coronary angiography showed an elevated resting T1 and reduced stress T1 response in the RCA territory ($T1_{rest}$ 993 ms to $T1_{stress}$ 999 ms; $\Delta T1 = 0.7\%$). Percutaneous coronary intervention (PCI) relieved the stenoses with good angiographic result (white arrows) and normalization of vessel FFR to 0.95. This finding was accompanied by significant improvements in the rest and stress T1 responses ($T1_{rest}$ 956 ms to $T1_{stress}$ 994 ms; $\Delta T1 = 4.0\%$).

IMAGING VIGNETTES

Diagnostic Value
 Biomarker-Positive
 Unobstructed Co

Adam N. Mather, MBBS, Tim
 John P. Greenwood, PhD, Sver

THE UNIVERSAL DEFINITION
 AN ELEVATED TROPONIN

limit (URL) together with at least
 changes of new ischemia; develop
 new loss of viable myocardium
 sensitive and specific for myoca
 Frequently, patients with ischemic
 invasive coronary angiography. I
 angiography may be normal or de
 correct diagnosis is important to e
 management (2). There may also

Cardiac magnetic resonance (CMR)
 pathophysiological effects of acu
 which demonstrate the diagnos
 diagnosis of ischemic symptom

Biochemical analysis of troponin I (TnI) (Accu TnI assay, Beckman Coulter, Brea, California) demonstrated interassay coefficient of variance of 10% at 0.06 µg/l and the 99th percentile value of 0.04 µg/l.

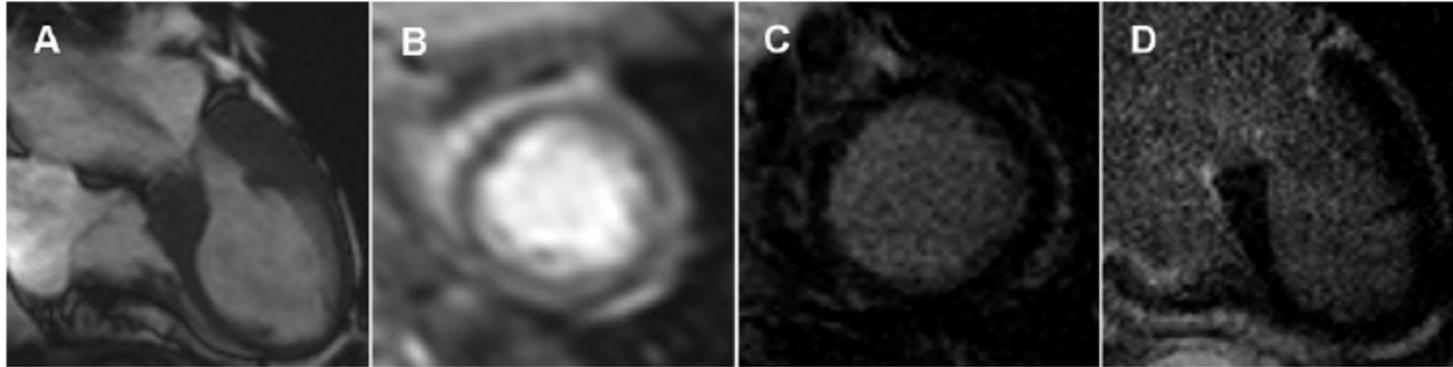
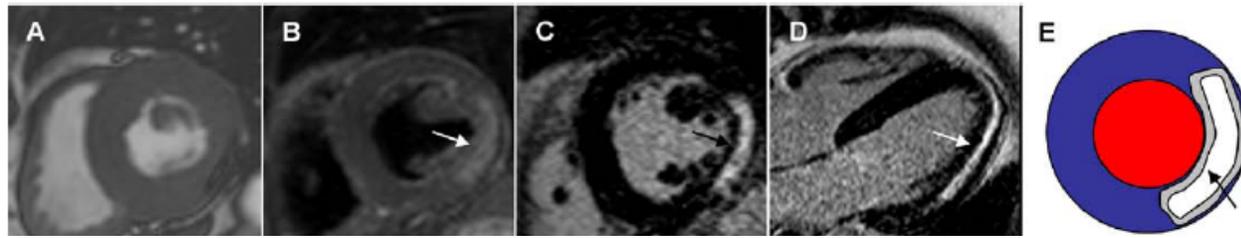
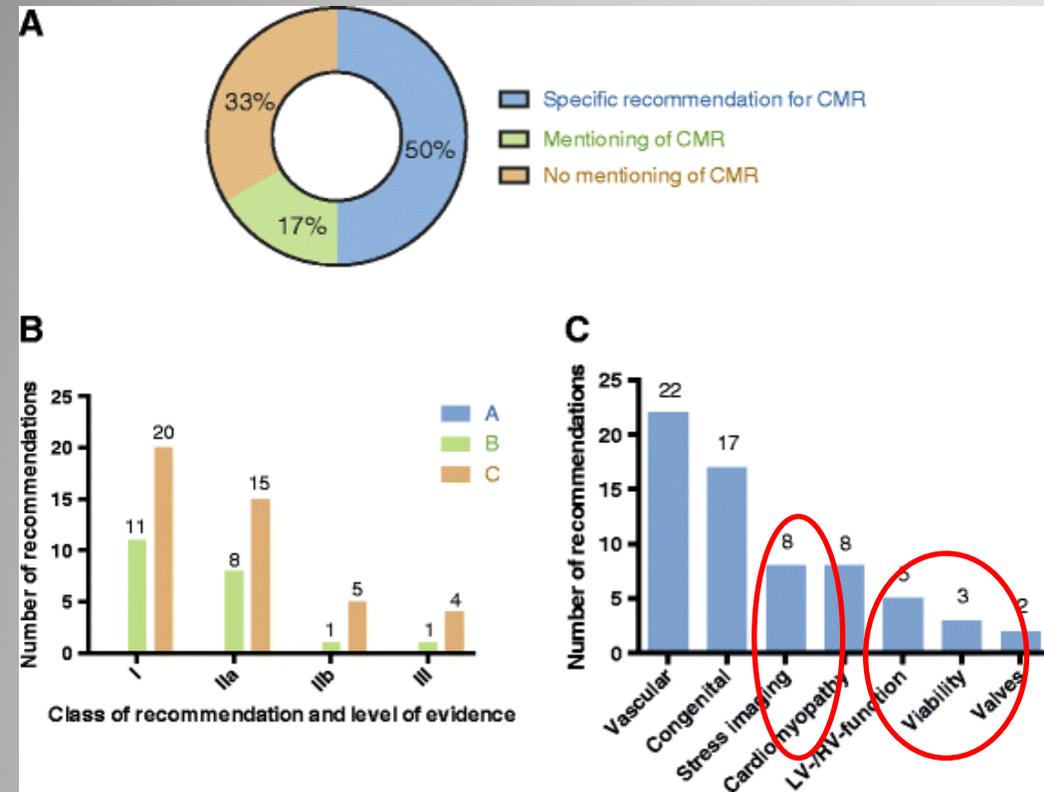


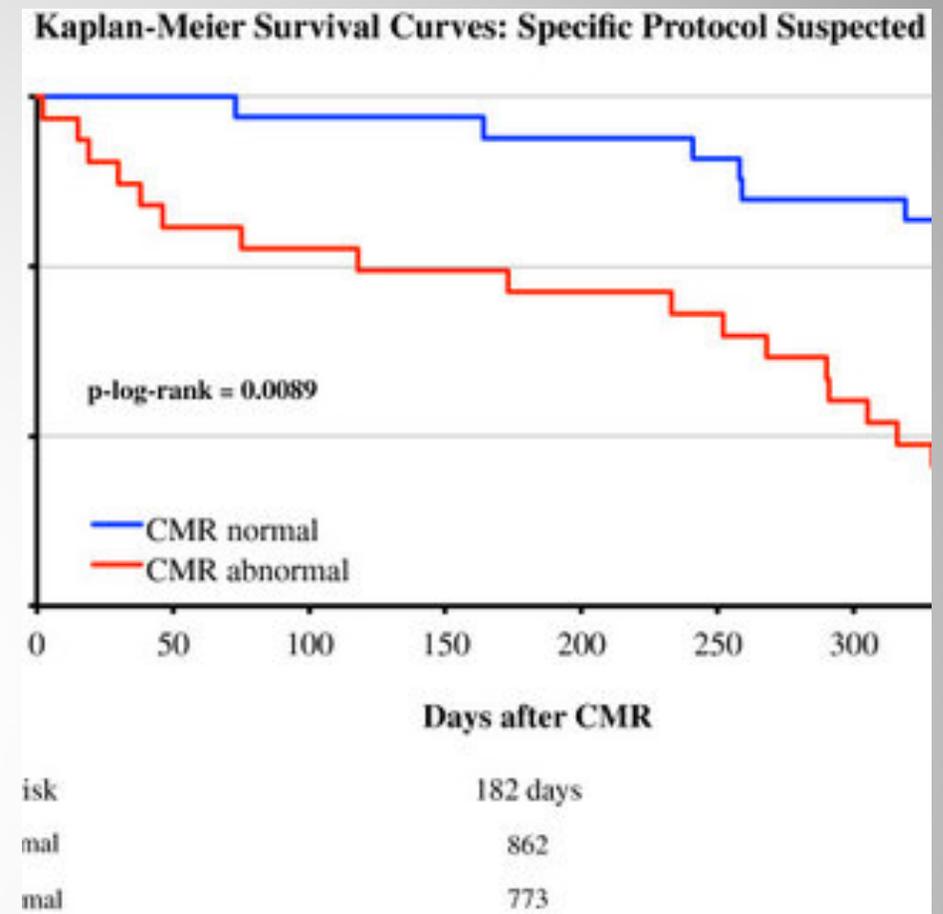
Figure 3. CMR Images of Takotsubo Cardiomyopathy

Case 3. A 67-year-old woman presented with central chest pain. There was anterior ST-segment elevation on her electrocardiogram. Emergency coronary angiography demonstrated only minor, nonobstructive atheroma but widespread wall motion abnormalities. CMR was subsequently requested to establish the diagnosis. (A) Cine imaging demonstrated apical ballooning and apical thinning of the LV. (B) Resting first-pass perfusion showed an apical subendocardial defect (this was normal in the basal segments), suggesting possible apical microvascular dysfunction associated with transient myocardial stunning. (C and D) Late gadolinium enhancement did not demonstrate any evidence of infarction or fibrosis, including in the apical region. These findings are typical of Takotsubo cardiomyopathy. A follow-up CMR scan at 6 months confirmed the diagnosis by showing complete resolution of ventricular dysfunction. Abbreviations as in Figure 1.

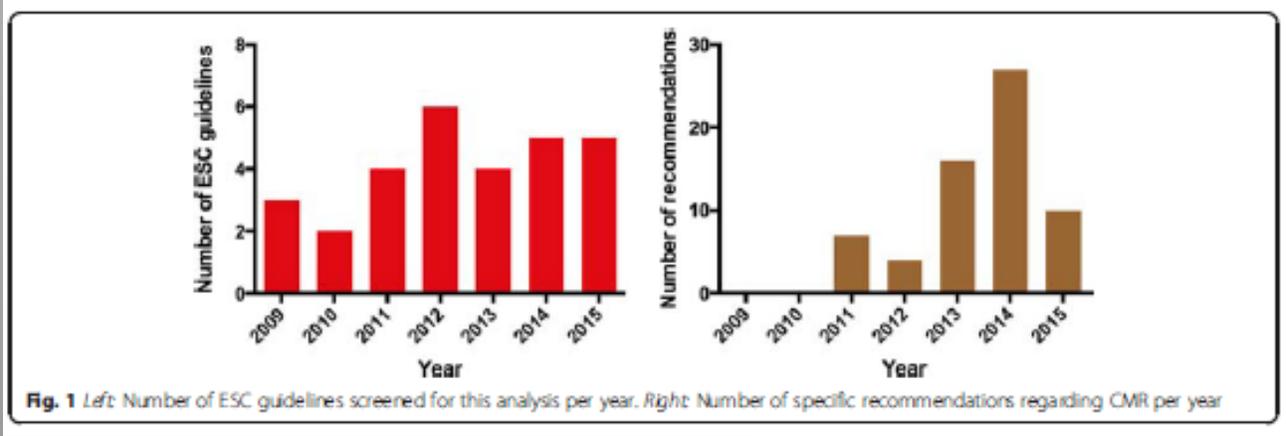
T1-weighted imaging (B). The pericardium is clearly seen between the layers of visceral and extracardiac fat (arrow). (C) T2-weighted imaging demonstrated hyperintense signal in the pericardium suggestive of acute inflammation (arrows). (D) Late gadolinium enhancement showed uptake of contrast within the entire pericardium (arrows), indicative of acute pericarditis. Despite no obvious myocardial inflammation, the evidence supported a unifying diagnosis of acute myopericarditis. Identifying the pericardium with echocardiography is often difficult, particularly in the absence of an associated pericardial effusion, as in this case. Therefore, CMR has added value over echocardiography in this setting as it can clearly delineate the pericardium between the layers of surrounding fat. Abbreviation as in Figure 1.



AHA/ACC



Representation of cardiovascular magnetic resonance in the AHA / ACC guidelines



European cardiovascular magnetic resonance (EuroCMR) registry – multi national results from 57 centers in 15 countries JCMR

ESC

GUIDELINES

Role of cardiovascular magnetic resonance in the guidelines of the European Society of Cardiology

PRO

CONTRO



e





FRIENDS or ENEMY?

