

**Ecocardiografia 2016,
Milano 22-23 marzo**



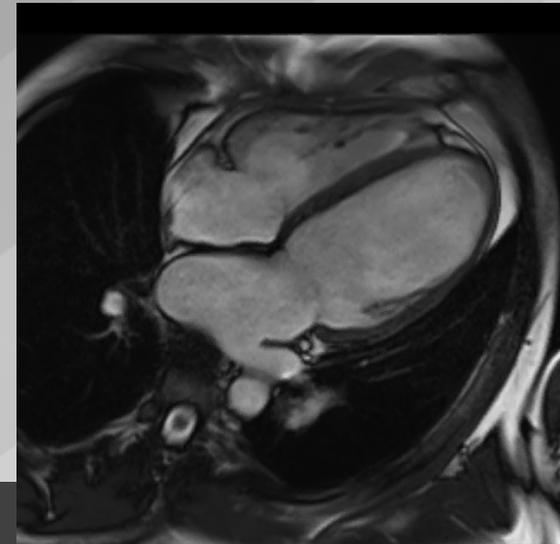
**Paziente con disfunzione ventricolare sinistra.
L'opportunità di integrare i dati clinici, ecocardiografici, coronarografici ed
Istologici con la caratterizzazione funzionale e tissutale della RM**

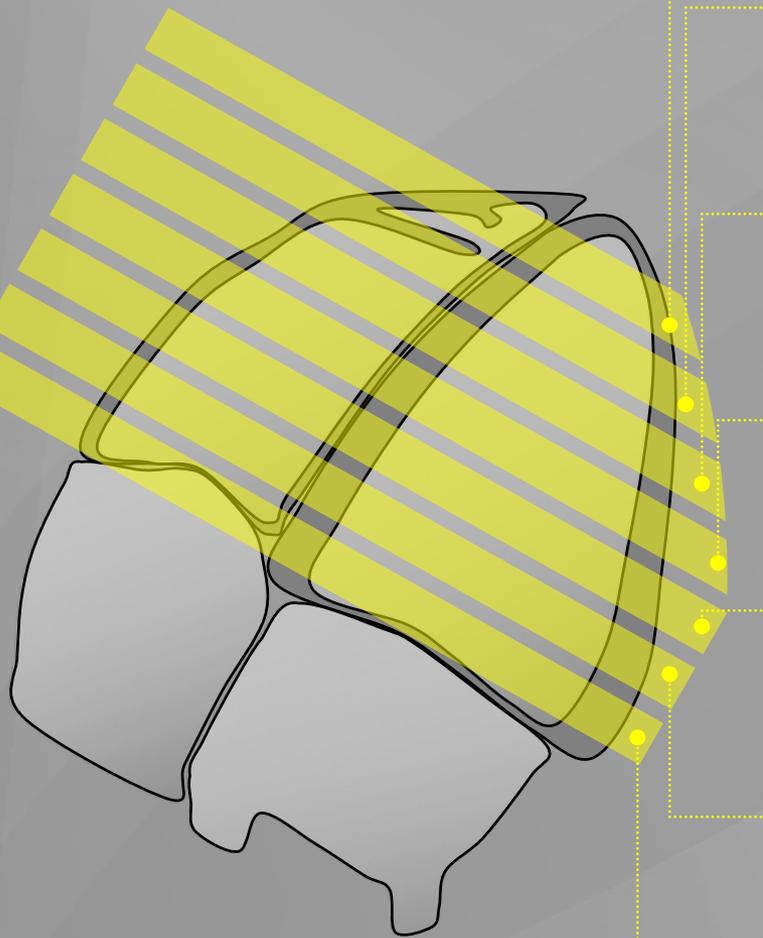
Alberto Roghi e Patrizia Pedrotti
Dipartimento Cardio-Toraco-Vascolare A. De Gasperis
Ospedale Niguarda Ca' Granda, Milano



PERCHE' FARE LA RM NELLO SCOMPENSO CARDIACO?

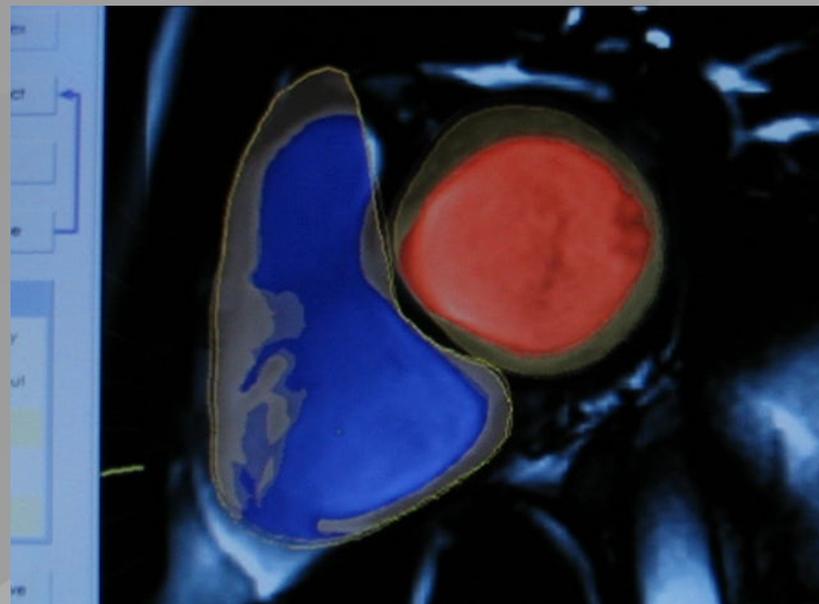
**TOP
FIVE**





Cortesia del Dr. S. Pedretti H. Niguarda

1 – FUNZIONE VENTRICOLARE



➤ La RMC è il gold standard per calcolare i volumi e la FE biventricolari e la massa (Lancet 1985, Radiology 1990, 2005; JCMR 2003)



Volumetric Left Ventricular Ejection Fraction is Superior to 2-Dimensional Echocardiography for Risk Stratification of Patients for Primary Prevention Implantable Cardioverter-Defibrillator Implantation

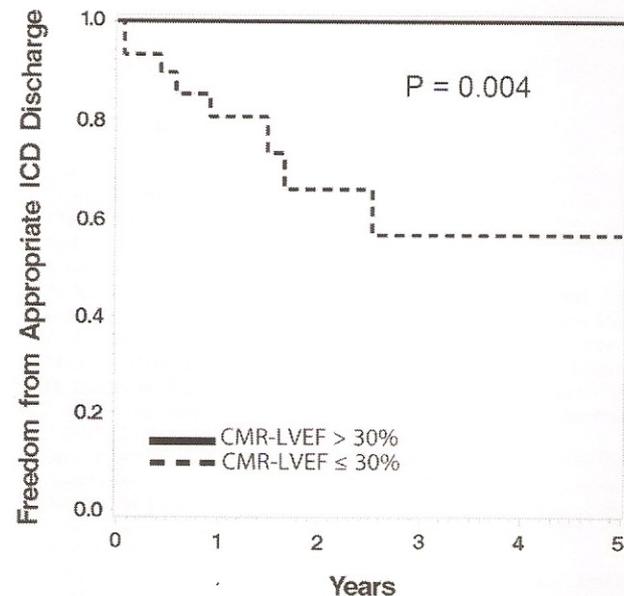
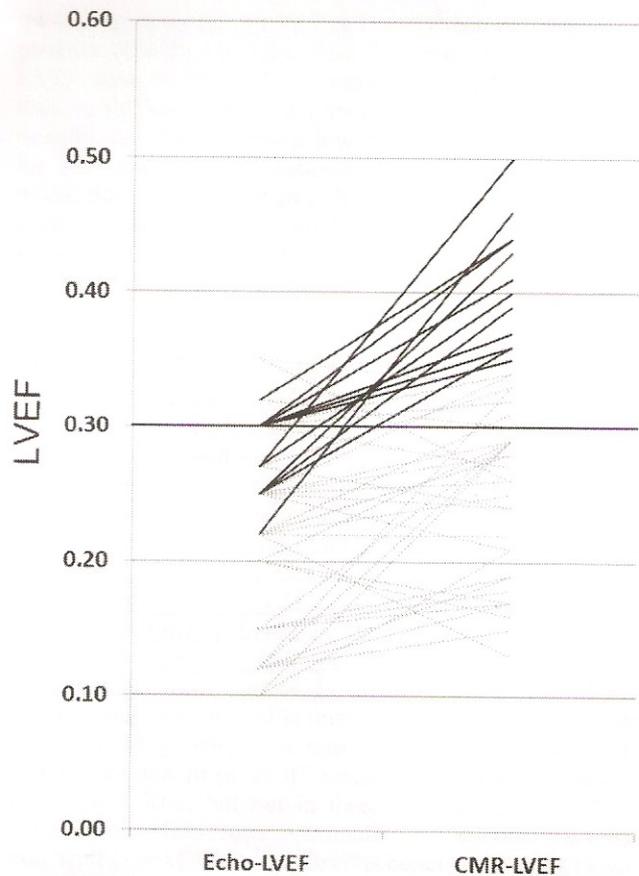
-48 pz con CMD (ischemica 52%)

-9 interventi corretti in 29 ± 25 mesi

-100% pz con criteri FE eco per ICD; il 25% non aveva criteri di FE alla RM

-0 interventi in pz senza criteri RM

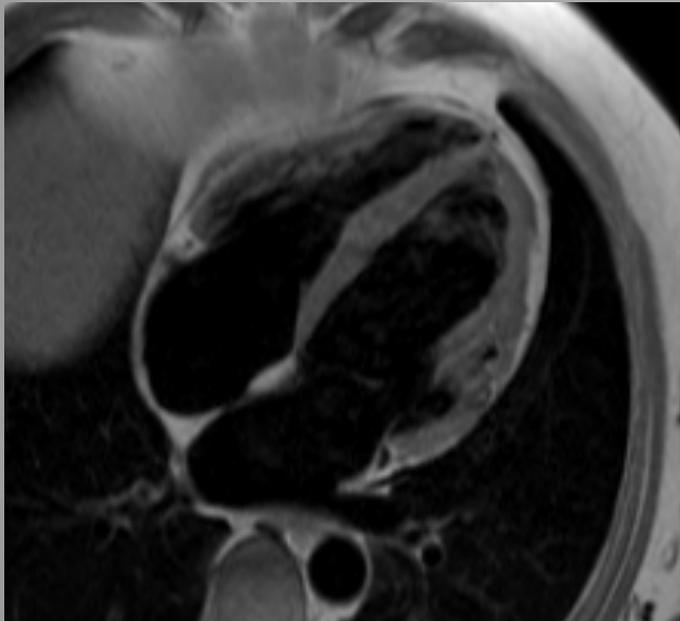
-Riclassificazione con criterio FE CMR $\leq 30\%$: no ICD in 40%, nessuno ha avuto intervento



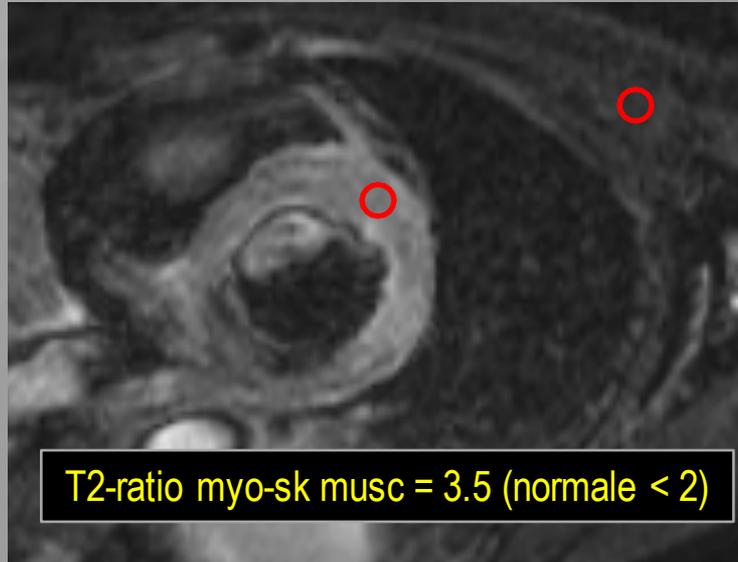
- Studio piccolo
- Retrospettivo
- Singolo centro
- ...ma real world



2 – CARATTERIZZAZIONE TISSUTALE - PRE-CONTRASTO



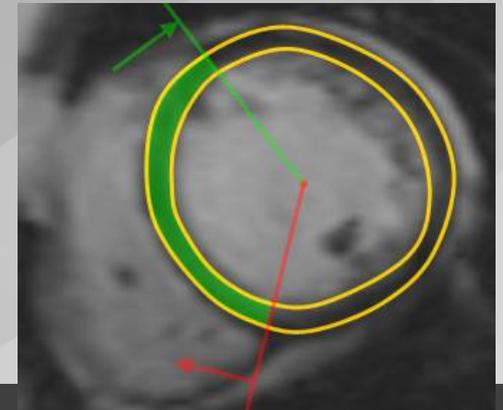
Immagini T1-pesate – Infiltrazione
adiposa
Cardiomiopatia aritmogena



T2-ratio myo-sk musc = 3.5 (normale < 2)

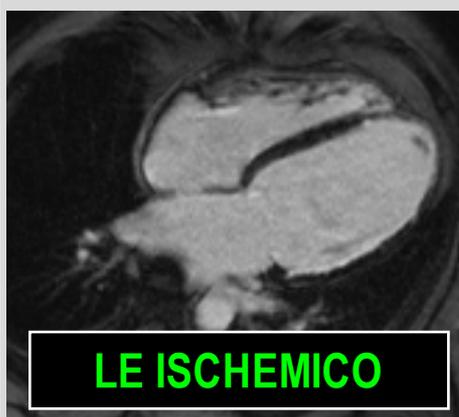
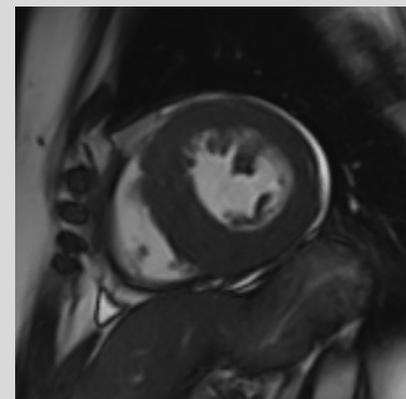
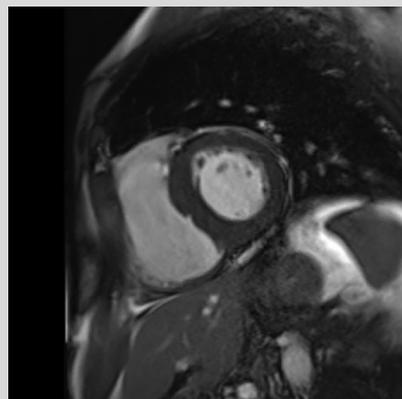
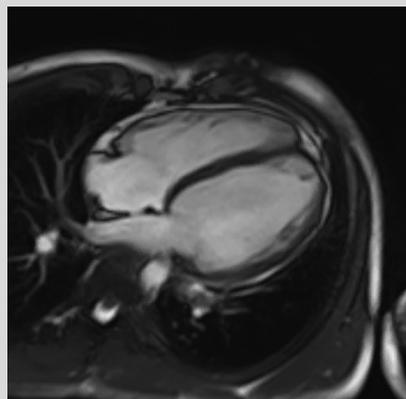
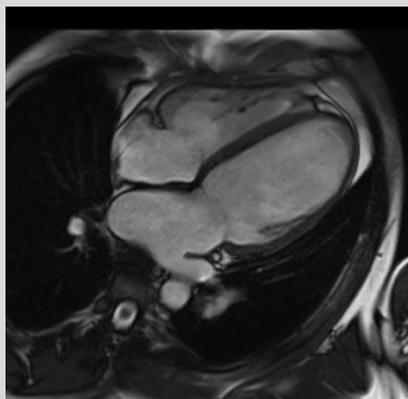
Immagini T2-pesate
STIR - Edema
Miocardite fulminante

T2* = 5.34 msec. –
Ferro cardiaco
Cardiomiopatia da
sovraccarico di ferro





3 – CARATTERIZZAZIONE TISSUTALE - POST-CONTRASTO



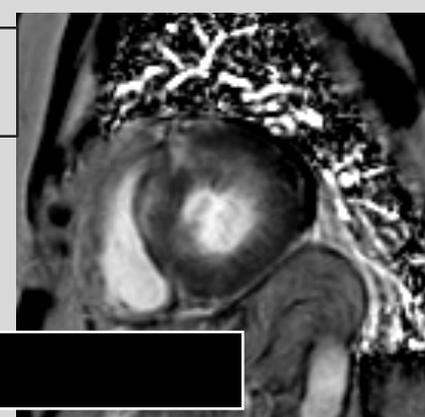
LE ISCHEMICO



SCAR - INFILTRAZIONE



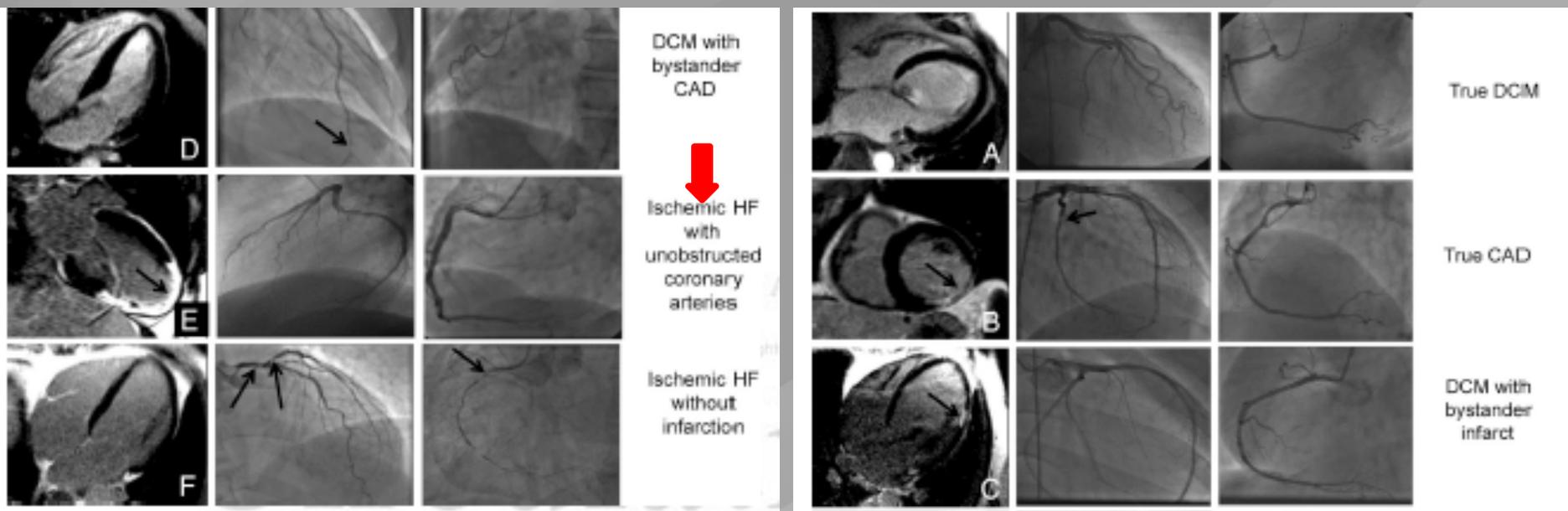
LE NON ISCHEMICO





LE ISCHEMICO

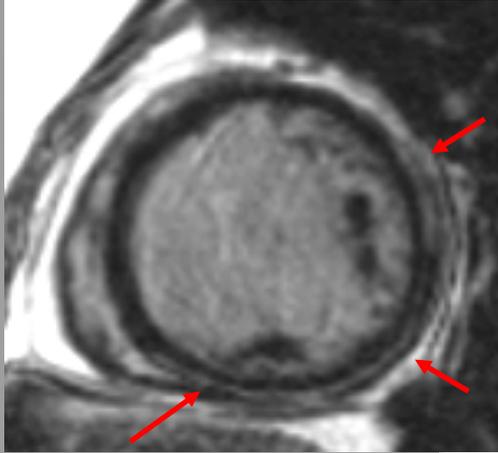
Role of Cardiovascular Magnetic Resonance as a Gatekeeper to Invasive Coronary Angiography in Patients Presenting With Heart Failure of Unknown Etiology



INTEGRAZIONE TRA ANATOMIA CORONARICA E CARATTERIZZAZIONE TISSUTALE MIOCARDICA

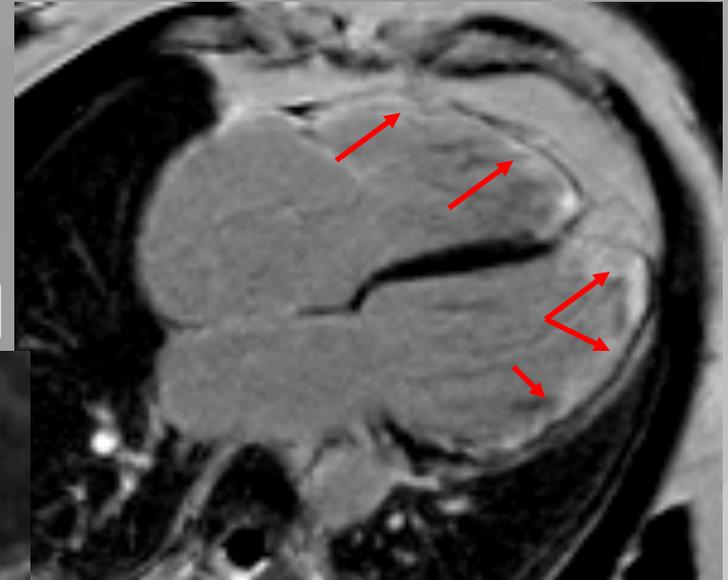
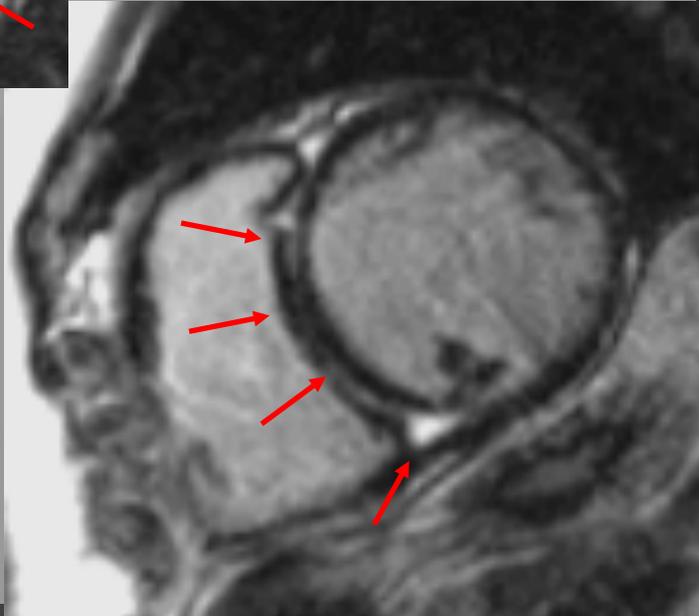


LE NON ISCHEMICO



LE subepicardico
(es. miocardite,
sarcoidosi)

LE intramiocardico (aspecifico)



LE esteso, focalmente
transmurale, biventricolare (?)



3 – LATE ENHANCEMENT E PROGNOSE

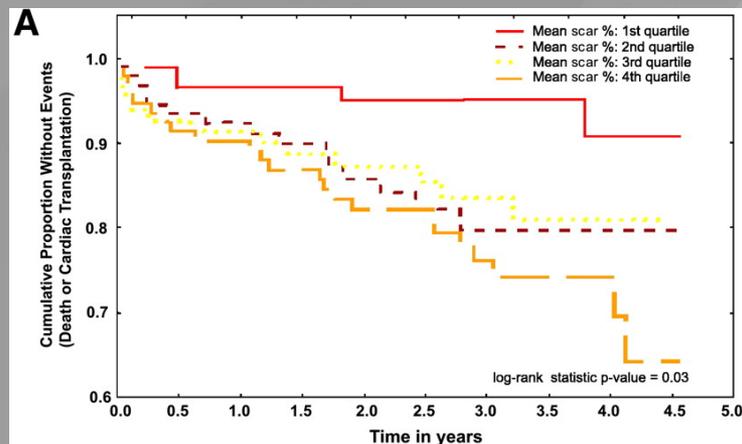
CARDIOMIOPATIA ISCHEMICA

- 349 pz cmio ischemica
- FE 24%
- Quantificazione cicatrice con LE (% di miocardio; punteggio transmuralità scar)
- End point composito (mortalità da tutte le cause e HTX)

Table 2. DHE-CMR Analysis of the Study Population, on the Basis of Composite End Points of Survival or Cardiac Transplantation

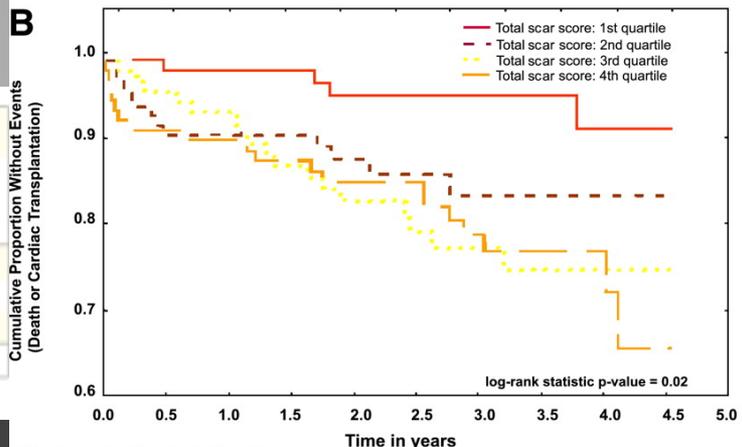
	Group 1 No Events (n = 293)	Group 2 Composite Events (n = 56)	p Value
Mean scar % on DHE-CMR	30 ± 20	39 ± 22	0.003
Transmurality score on DHE-CMR	7.8 ± 5	9.7 ± 5	0.004
Total scar score on DHE-CMR	2.0 ± 1.1	2.5 ± 1.1	0.004

DHE-CMR = delayed hyperenhancement cardiac magnetic resonance.



Number of patients at risk of events

1st Quartile	82	80	73	60	53	41	27	17	5	0
2nd Quartile	85	78	71	55	39	30	23	15	2	0
3rd Quartile	75	70	63	57	46	37	26	16	0	0
4th Quartile	84	82	75	67	60	42	24	16	2	0



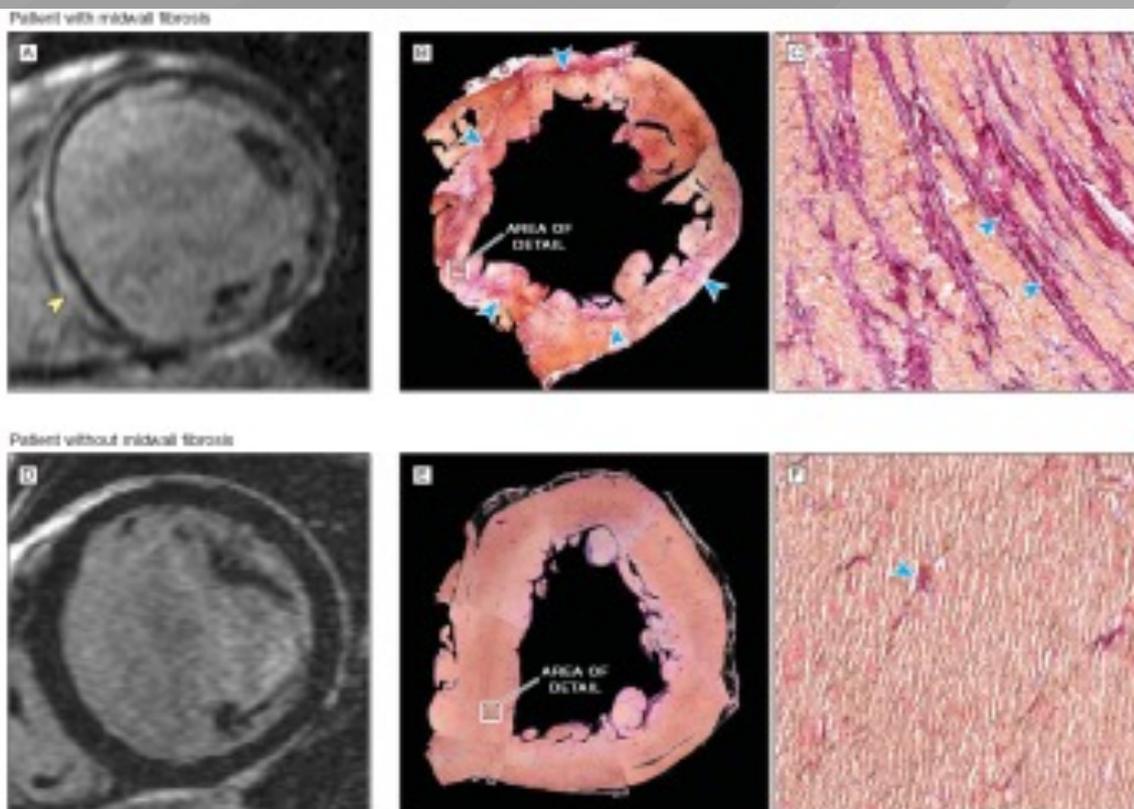


3 – LATE ENHANCEMENT E PROGNOSI

CARDIOMIOPATIA DILATATIVA NON ISCHEMICA

- 472 pz, CMD non ischemica
- FE media 37.2%
- Presenza e quantificazione LE
- End point primario: mortalità da tutte le cause
- End point-secondario: mortalità cardiaca o HTX; SCD o SCD abortita; morte per SCC o ospedalizzazione per SCC, o HTX
- LE indipendentemente correlato alla prognosi

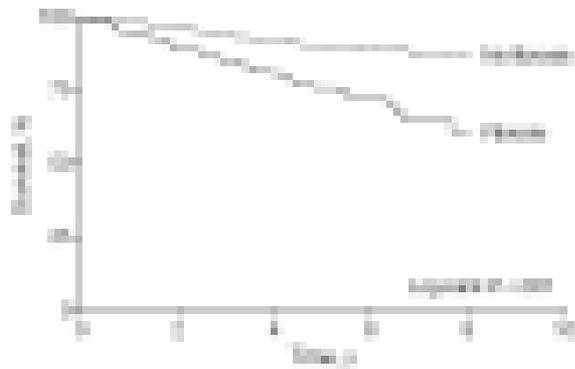
Association of Fibrosis With Mortality and Sudden Cardiac Death in Patients With Nonischemic Dilated Cardiomyopathy





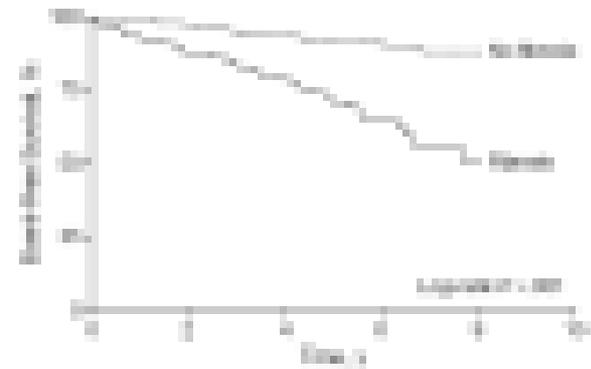
3 – LATE ENHANCEMENT E PROGNOSI

(A) All-cause mortality



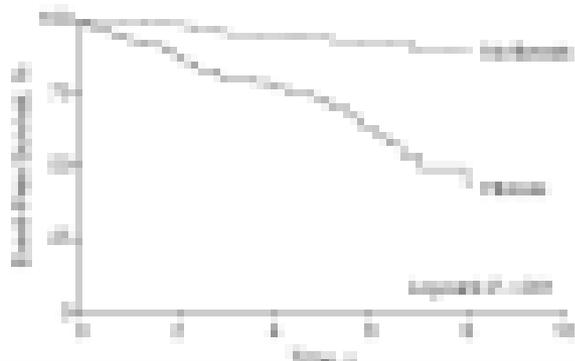
HR (95% CI)
LGE vs No LGE
1.25 (1.05-1.48)
Log-rank $P < .001$

(B) Cardiovascular mortality or transplantation



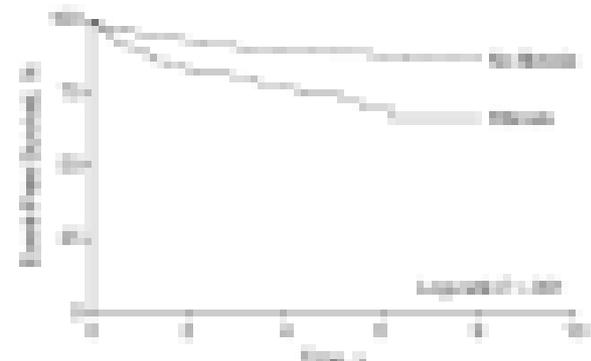
HR (95% CI)
LGE vs No LGE
1.55 (1.28-1.87)
Log-rank $P < .001$

(C) Median survival duration after transplantation



HR (95% CI)
LGE vs No LGE
1.55 (1.28-1.87)
Log-rank $P < .001$

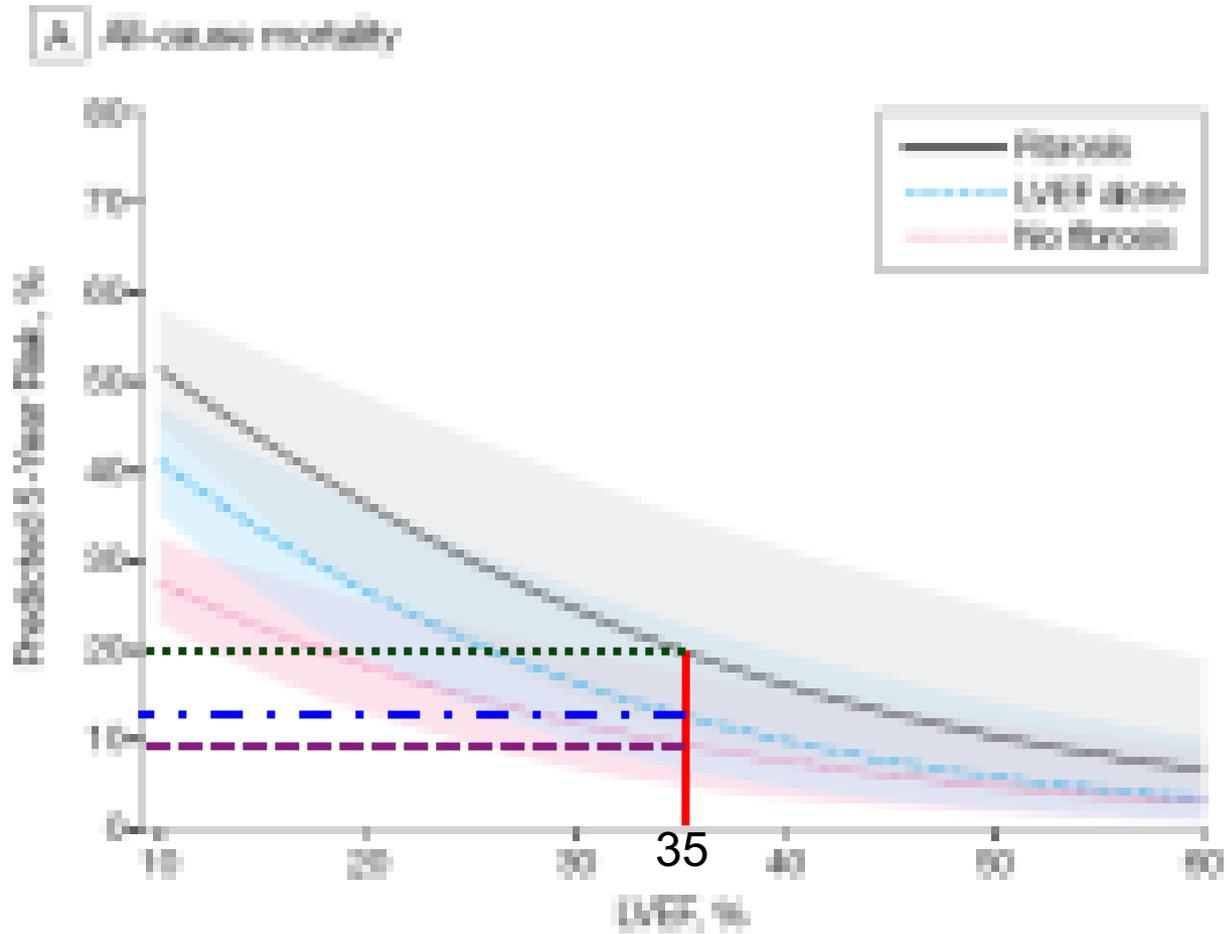
(D) Heart failure death, transplantation, or retransplantation



HR (95% CI)
LGE vs No LGE
1.55 (1.28-1.87)
Log-rank $P < .001$

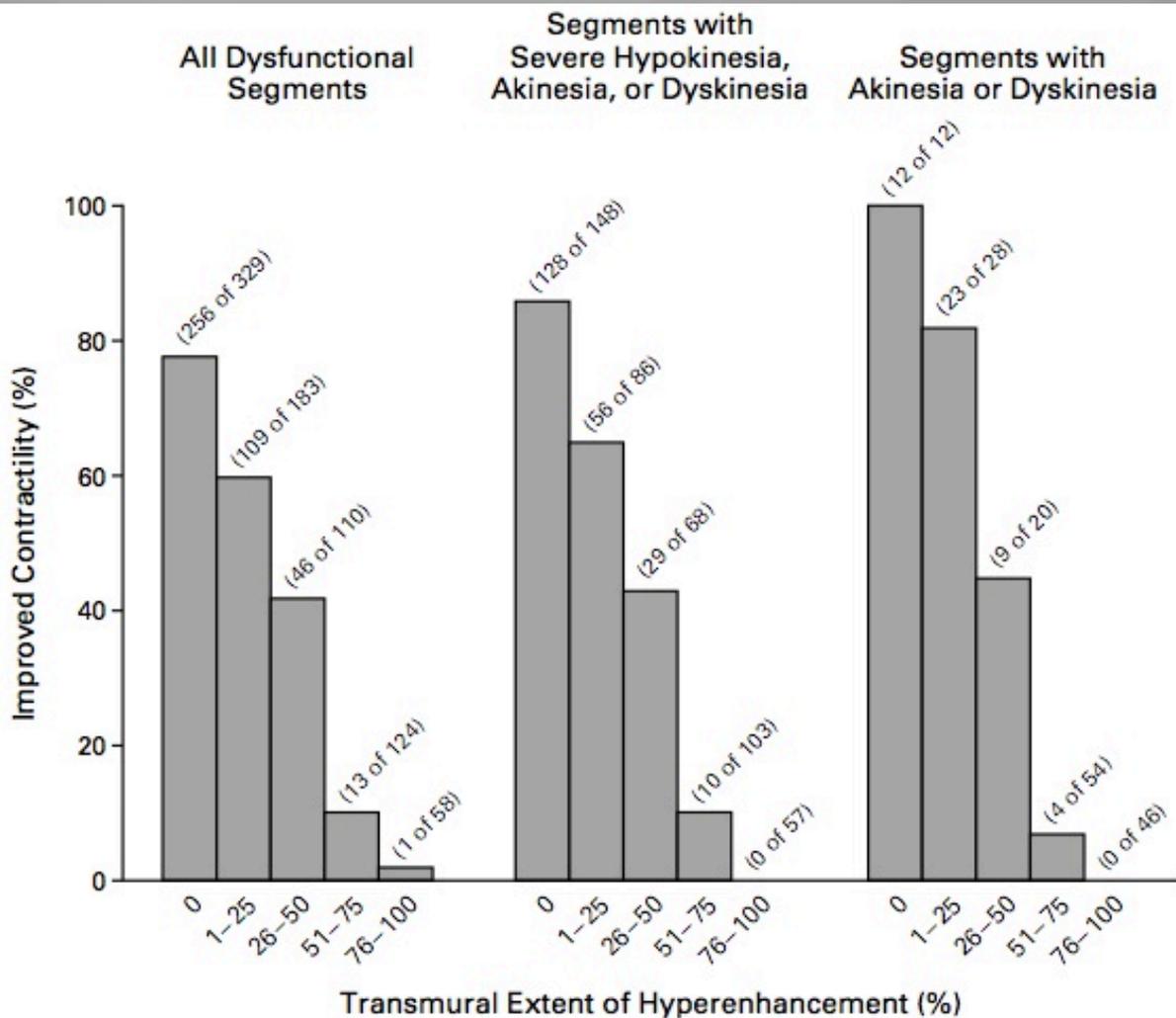


3 – LATE ENHANCEMENT E PROGNOSI





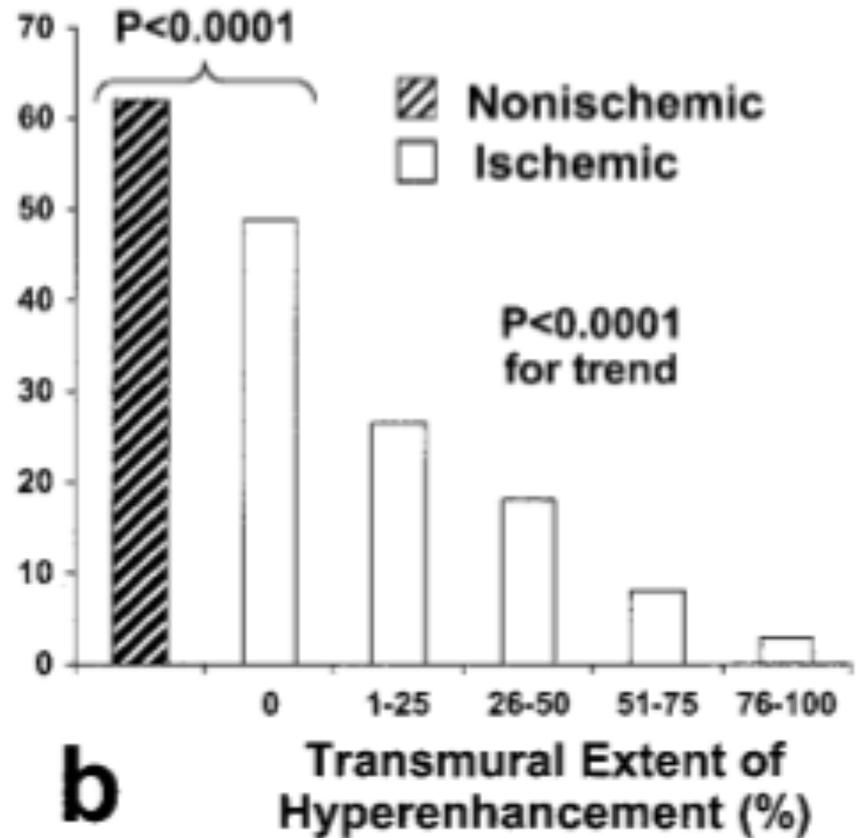
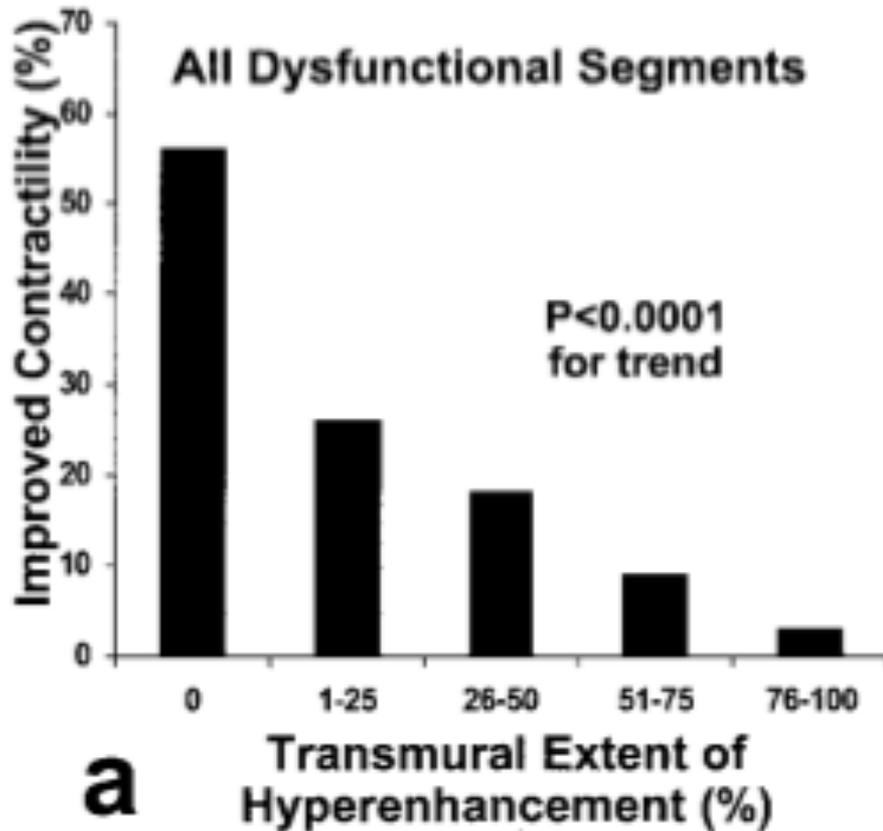
4 – LATE ENHANCEMENT E TERAPIA



RIVASCOLARIZZAZIONE IN
CMIO ISCHEMICA



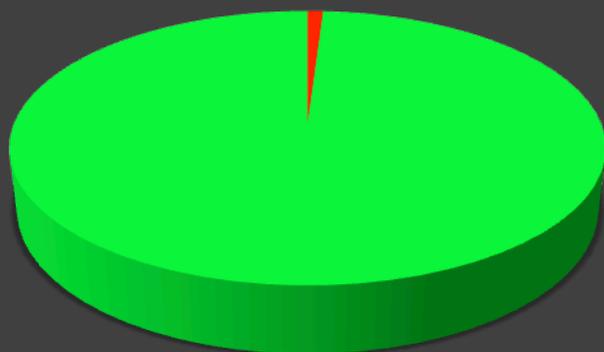
RISPOSTA ALLA TERAPIA BETA-BLOCCANTE (CMD ISCHEMICA E NON ISCHEMICA)





RISPOSTA ALLA CRT

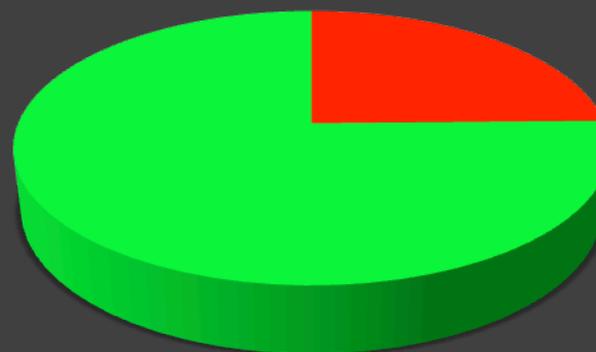
LE%



■ SI 1%
■ NO

RESPONDER

LE%



■ SI' 24,7%
■ NO

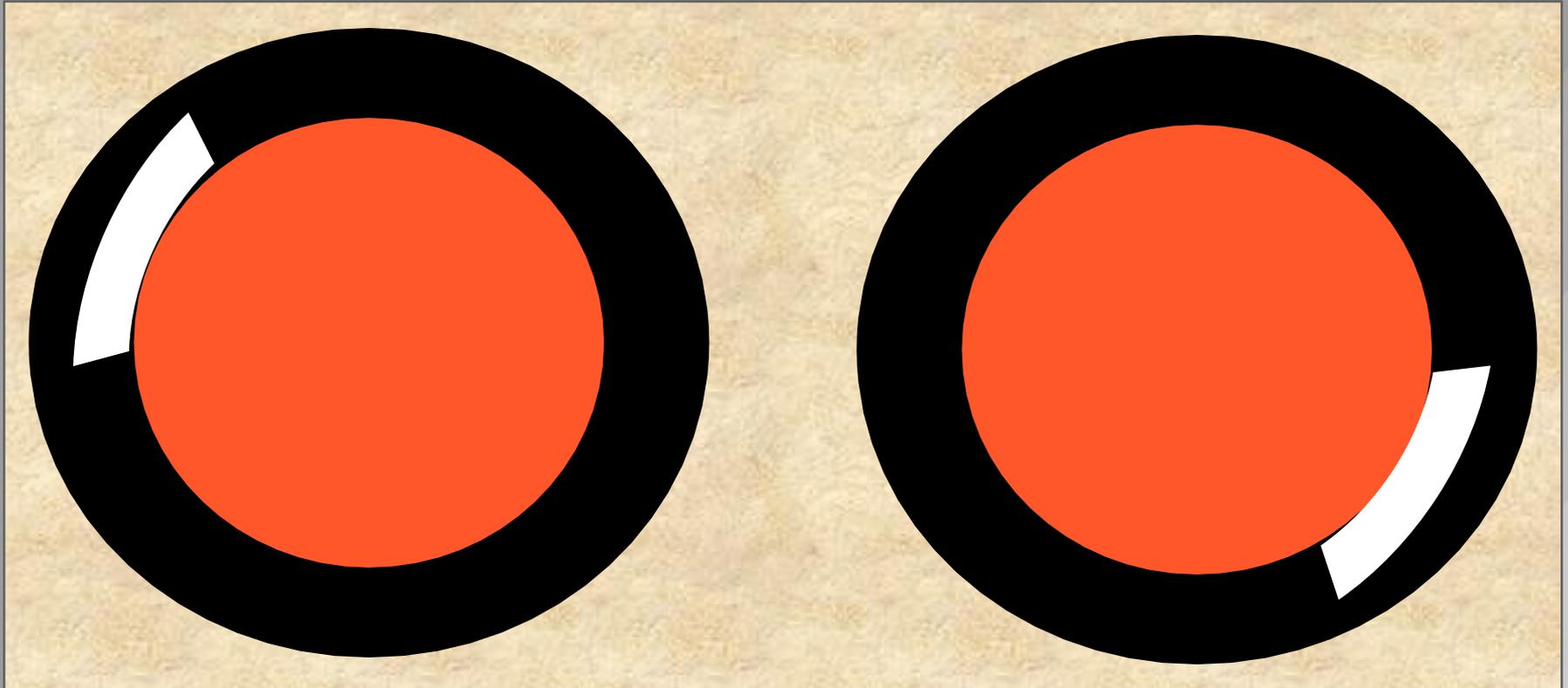
NON RESPONDER

$P < 0,01$

LE < 15%
Se = 85% Spec = 90%
Identificazione non-responder



RISPOSTA ALLA CRT

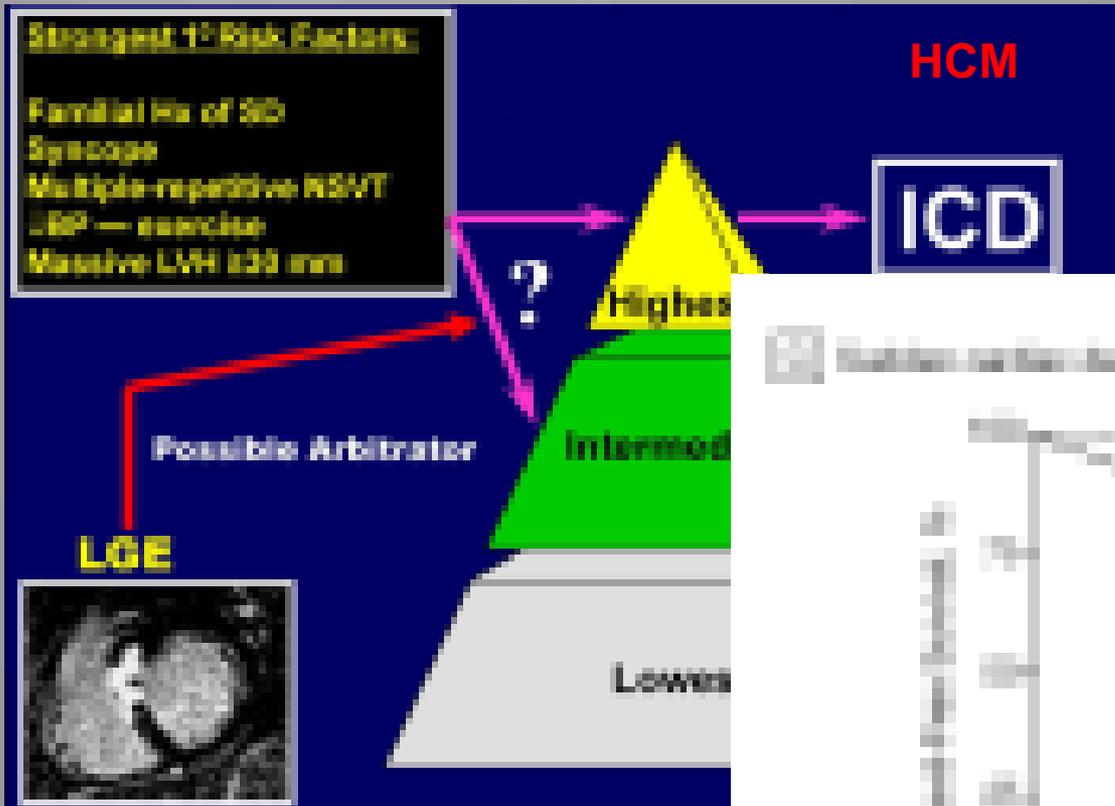


LE SIV $\leq 40\%$
Se = 1 Spec = 1
Identificazione responder

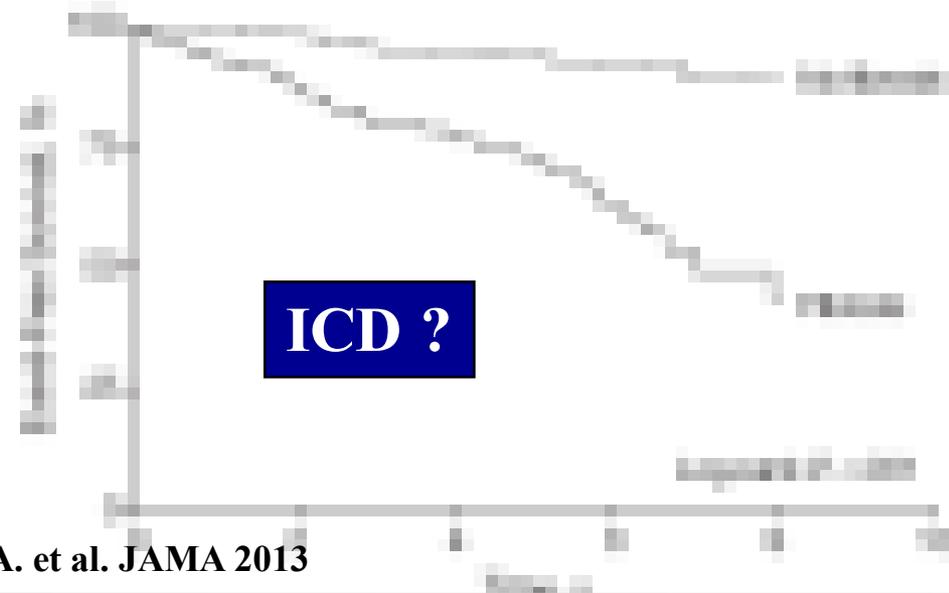
LE infero-laterale 50%
Se = 0.71 Spec = 0.87
Identificazione non responder



IMPIANTO DI ICD



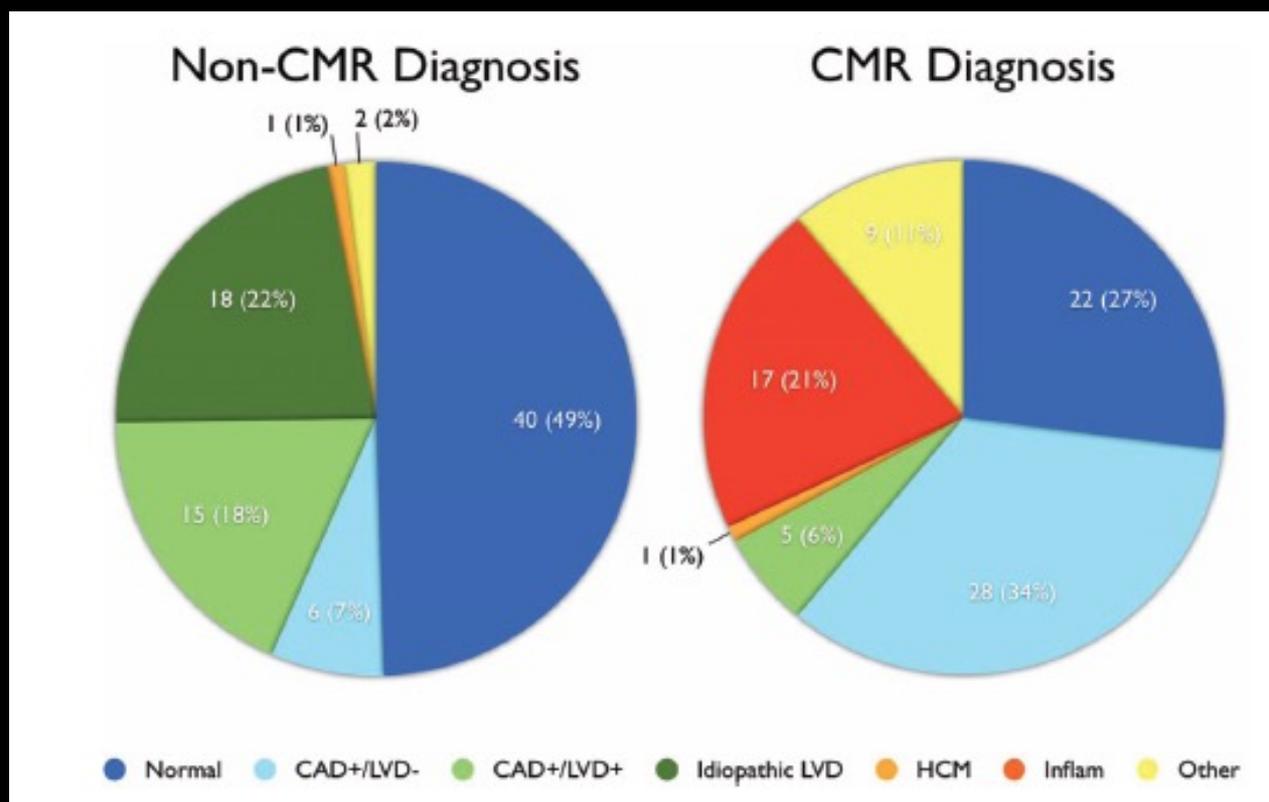
Number of SCD deaths decreased over time



Gulati A. et al. JAMA 2013

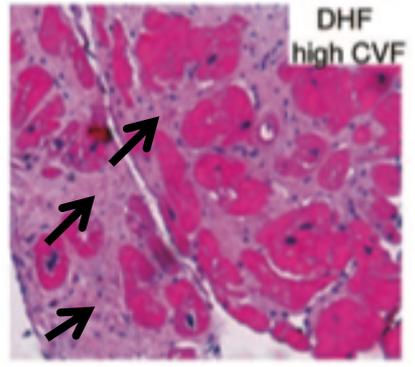
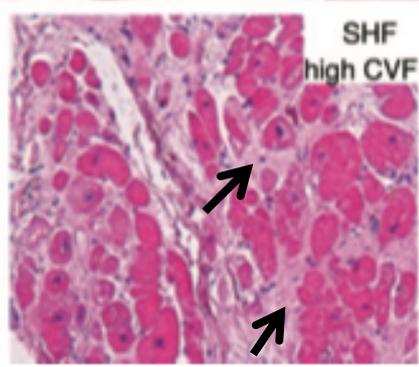
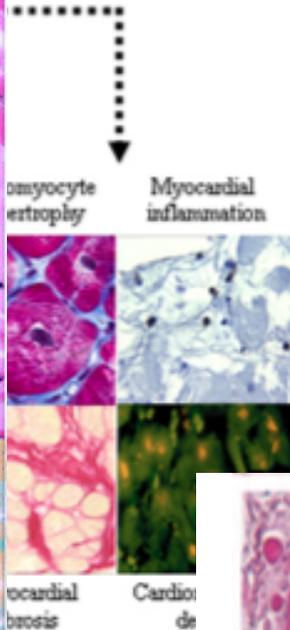
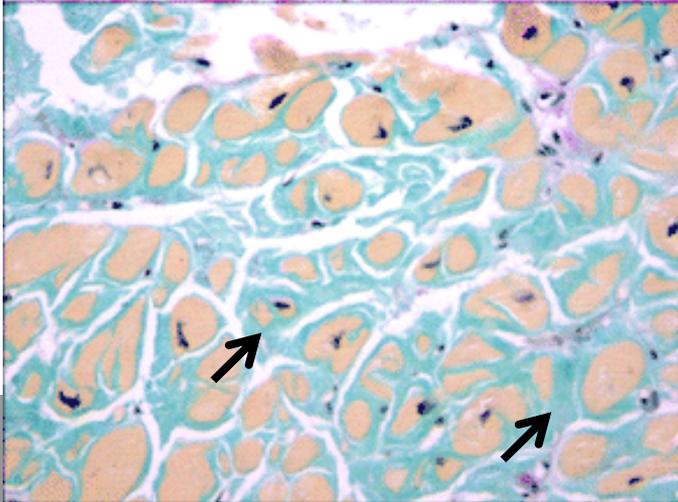
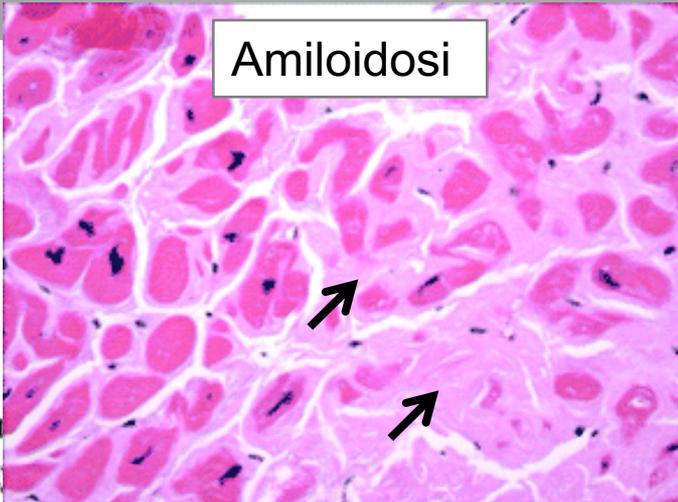
Utility of Cardiovascular Magnetic Resonance in Identifying Substrate for Malignant Ventricular Arrhythmias

James A. White, MD, FRCPC; Nowell M. Fine, MD; Lorne Gula, MD, MSc; Raymond Yee, MD; Allan Skanes, MD; George Klein, MD; Peter Leong-Sit, MD; Heather Warren, MD; Terry Thompson, PhD; Maria Drangova, PhD; Andrew Krahn, MD





5 – QUANTIFICAZIONE DELLO SPAZIO EXTRACELLULARE

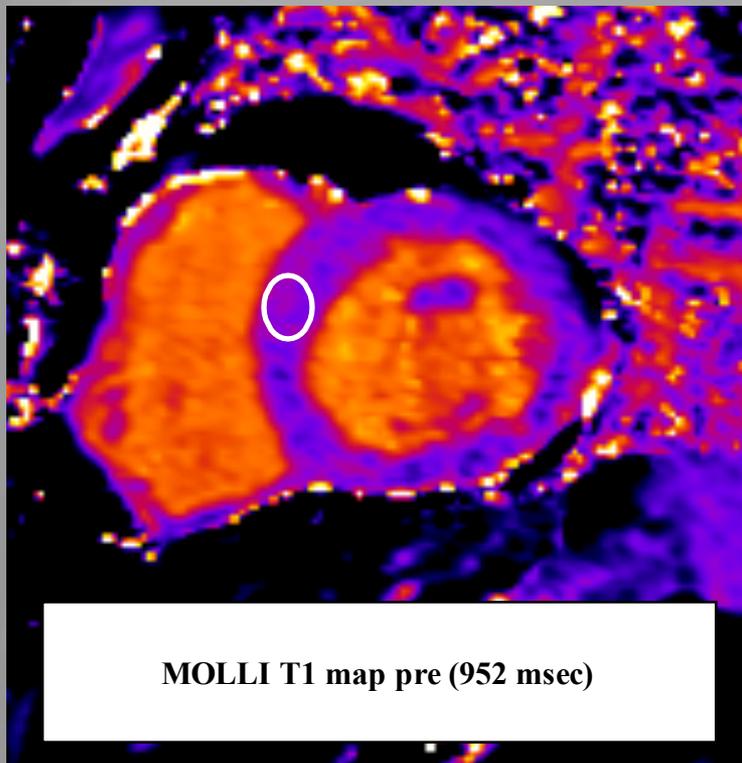


Van Heerebeek L. et al Circulation 2006



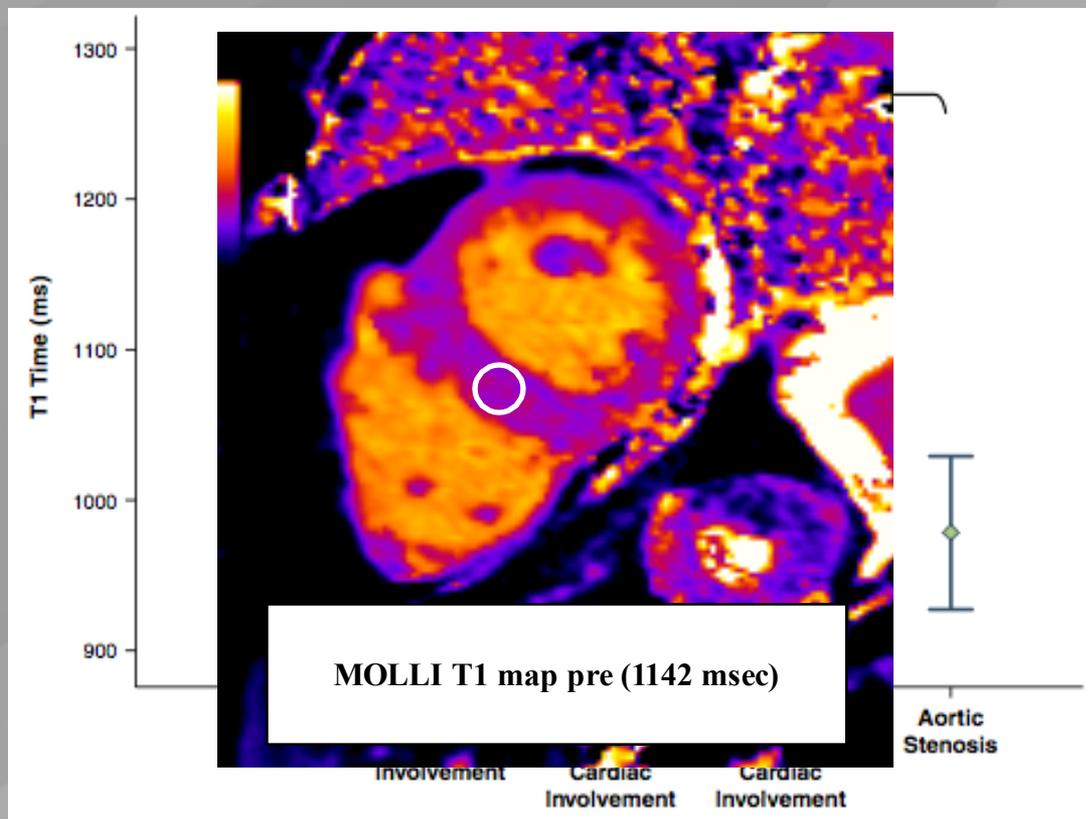
NUOVE TECNICHE DI MAPPAGGIO T1 – T1 NATIVO PRECONTRASTO

NORMALE



Valore normale a 1.5 T = 950-960 msec.

AMILOIDOSI



Karamitsos et al. JACC Cardiovasc Imaging 2013

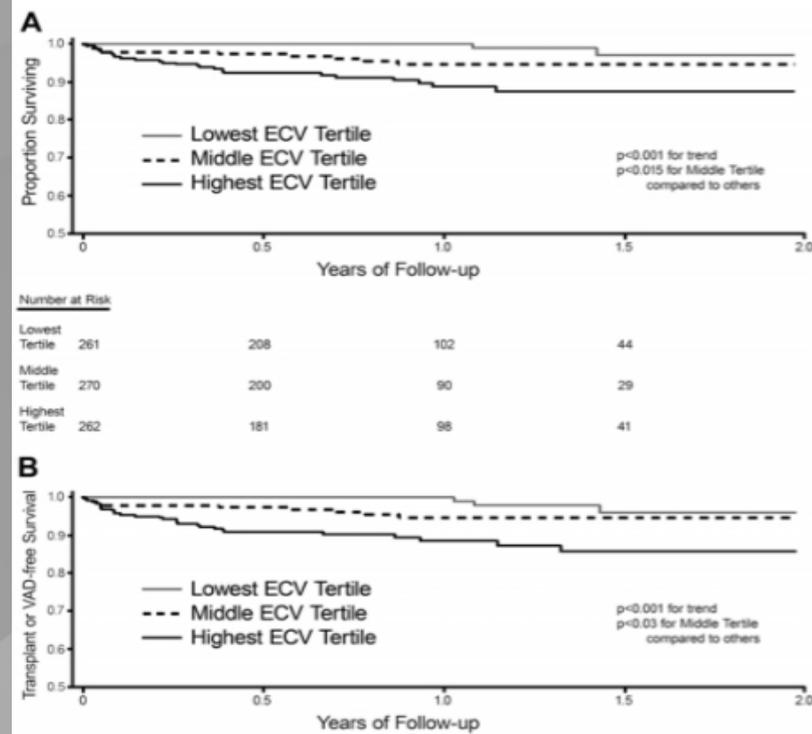
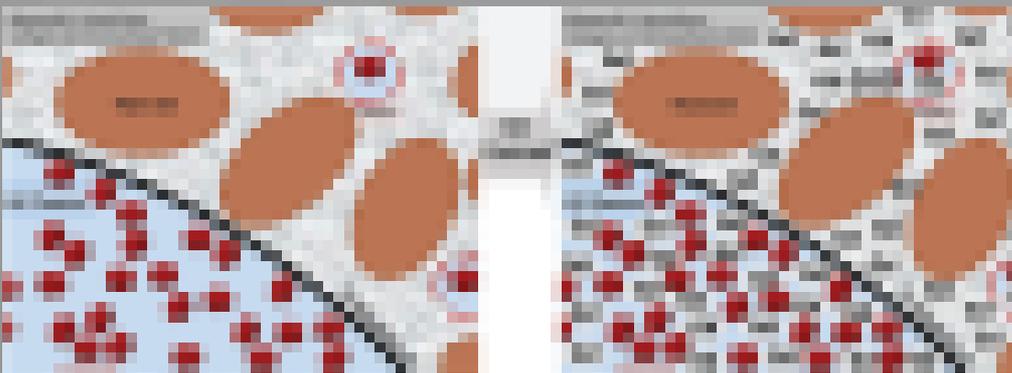


NUOVE TECNICHE DI MAPPAGGIO T1 – EXTRACELLULAR VOLUME FRACTION (ECV)

NORMALE



PATOLOGICO

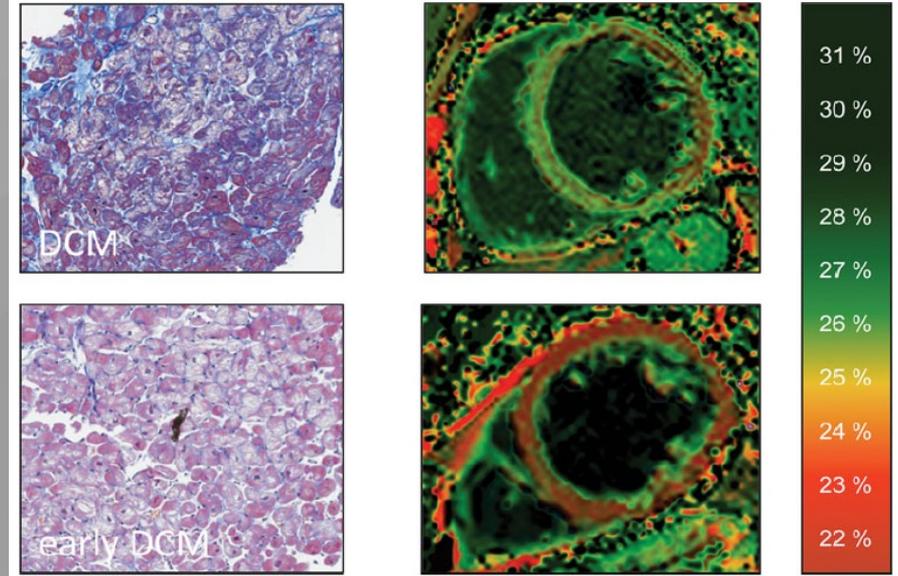
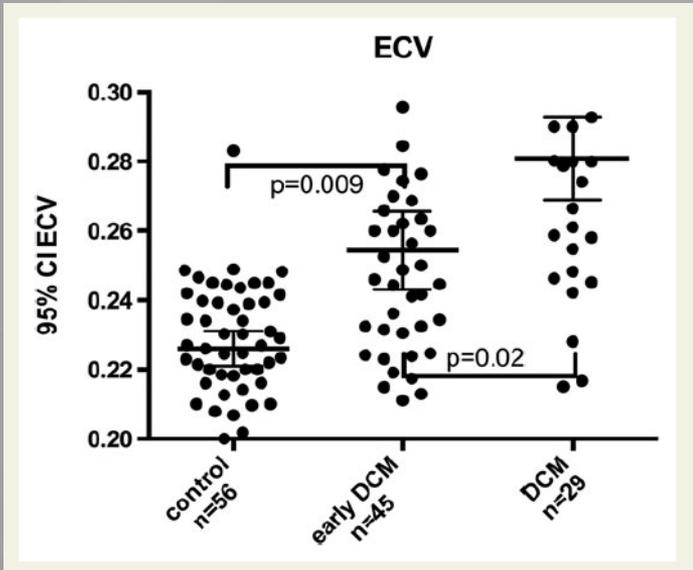


- 793 pz
- Cmio ipertrofica ed amiloidosi escluse
- Prognosi –va in pz con i valori più elevati di ECV
- Dati preliminari, necessari ulteriori studi

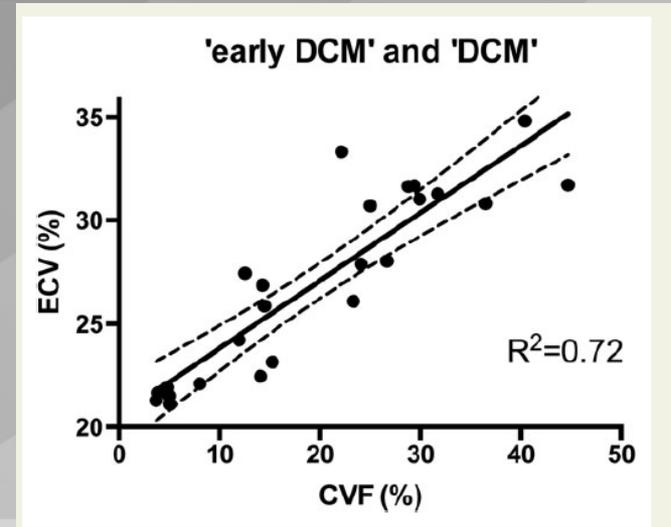


ECV e biopsia miocardica

Siepen EHJ cardiov Imaging 2014



Nella CMD, la fibrosi interstiziale è un marker precoce di disfunzione sistolica con progressivo incremento nelle fasi avanzate della malattia





Studio farmacologico di fase I, pazienti con amiloidosi, monitorati anche con RMC ed ECV

ClinicalTrials.gov

A service of the U.S. National Institutes of Health

A Study to Evaluate the Safety of GSK2398852 When Co-administered With GSK2315698 in Patients With Systemic Amyloidosis

This study is currently recruiting participants.

Verified June 2013 by GlaxoSmithKline

Sponsor:

GlaxoSmithKline

Collaborators:

Imperial College London

Heart Hospital

Royal Free Hospital

Quintiles London

Information provided by (Responsible Party):

GlaxoSmithKline

ClinicalTrials.gov Identifier:

NCT01777243

First received: January 24, 2013

Last updated: June 27, 2013

Last verified: June 2013

[History of Changes](#)

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[No Study Results Posted](#)

[Disclaimer](#)

[How to Read a Study Record](#)

depleter) and GSK2398852 (anti-SAP mAb), and circulating SAP concentrations will be assessed. Dose escalation in Part A will continue to the highest well tolerated dose or the highest allowable dose. Subjects will be closely monitored and will undergo Equilibrium contrast Magnetic Resonance Imaging (EqMRI) including organ volume, Elastography and Liver Biopsy if required.



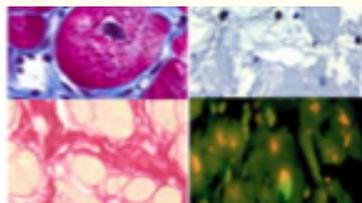
New Targets to Treat the Structural Remodeling of the Myocardium

Arantxa González, PhD,* Susana Ravassa, PhD,* Javier Beaumont, PhD,* Begoña López, PhD,*
Javier Díez, MD, PhD*†

Triggering myocardial injury



Compensatory mechanisms *



Deterioration of LV morphology and function



Clinical cardiac decompensation



Recognizing Hospitalized Heart Failure as an Entity and Developing New Therapies to Improve Outcomes

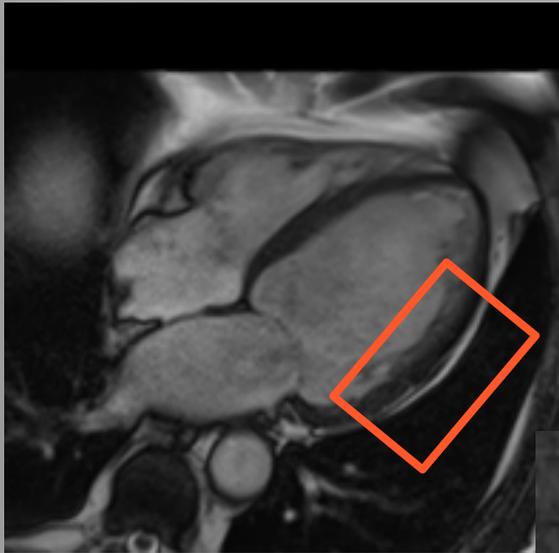
Academics', Clinicians', Industry's, Regulators', and Payers' Perspectives

Mihai Gheorghiade, MD^{a,*}, Ami N. Shah, MD^a,
Muthiah Vaduganathan, MD, MPH^b, Javed Butler, MD, MPH^c,
Robert O. Bonow, MD, MS^a, Giuseppe M.C. Rosano, MD, PhD^d,
Scott Taylor, RPh, MBA^e, Stuart Kupfer, MD^f, Frank Misselwitz, MD, PhD^g,
Arjun Sharma, MD^h, Gregg C. Fonarow, MDⁱ

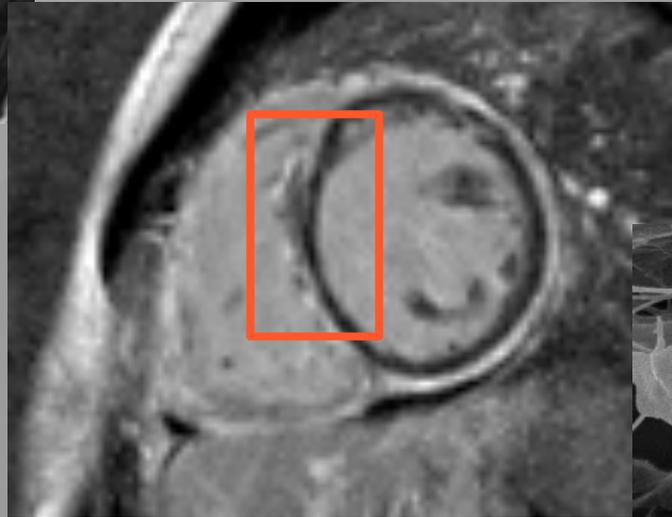




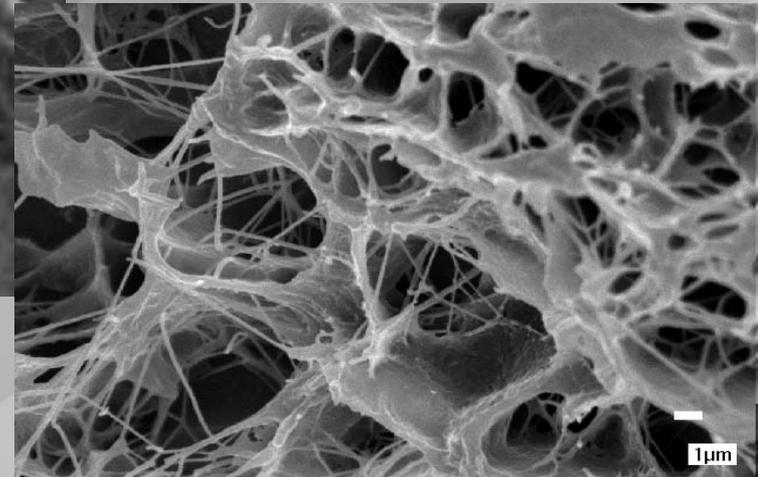
PERCHE' LA RM...



Funzione cardiaca



**Caratterizzazione tissutale
– Fibrosi sostitutiva**



Fibrosi interstiziale