
# Ecografia e remodeling ventricolare: diagnosi e terapia … nell'infarto miocardico acuto

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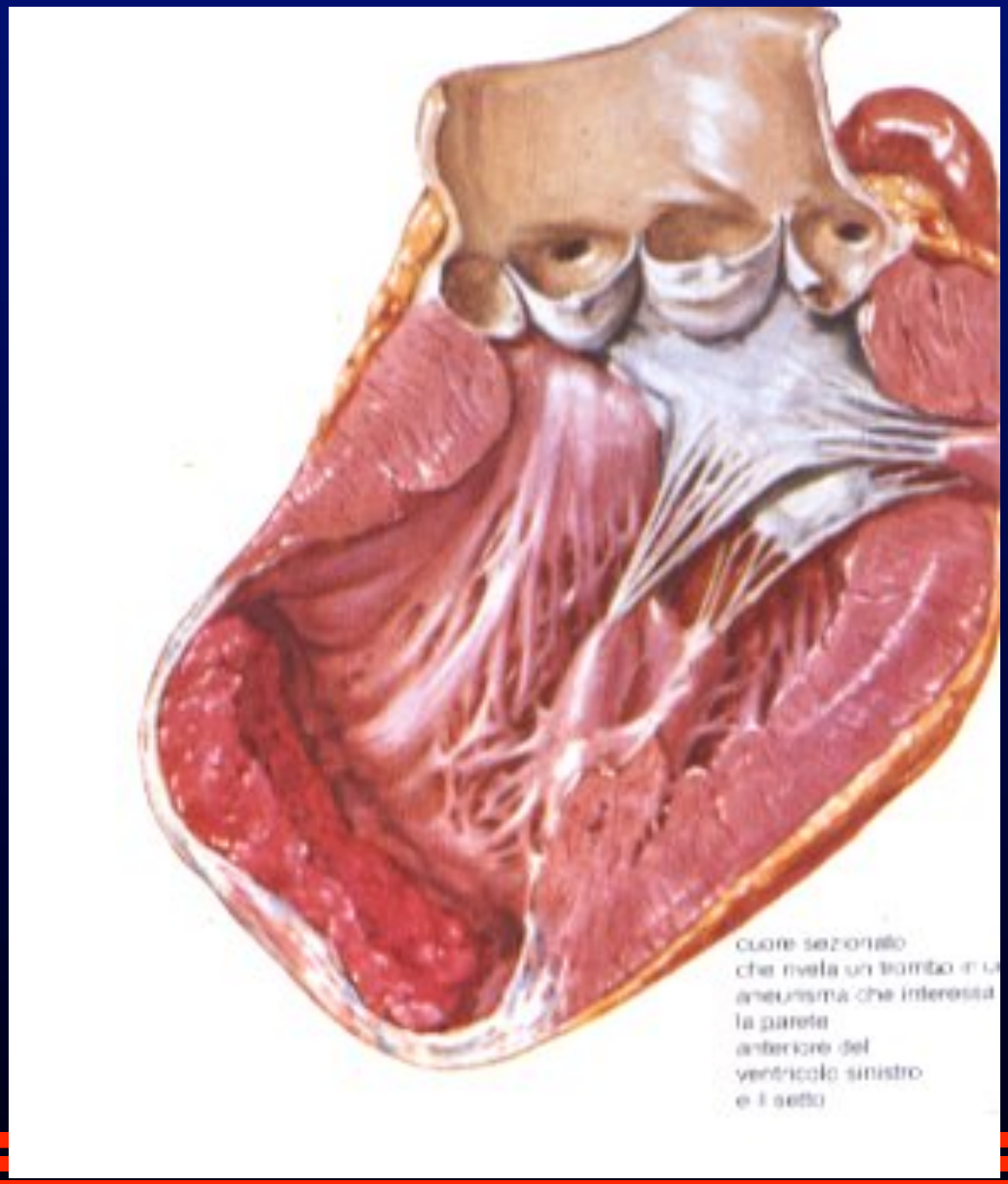
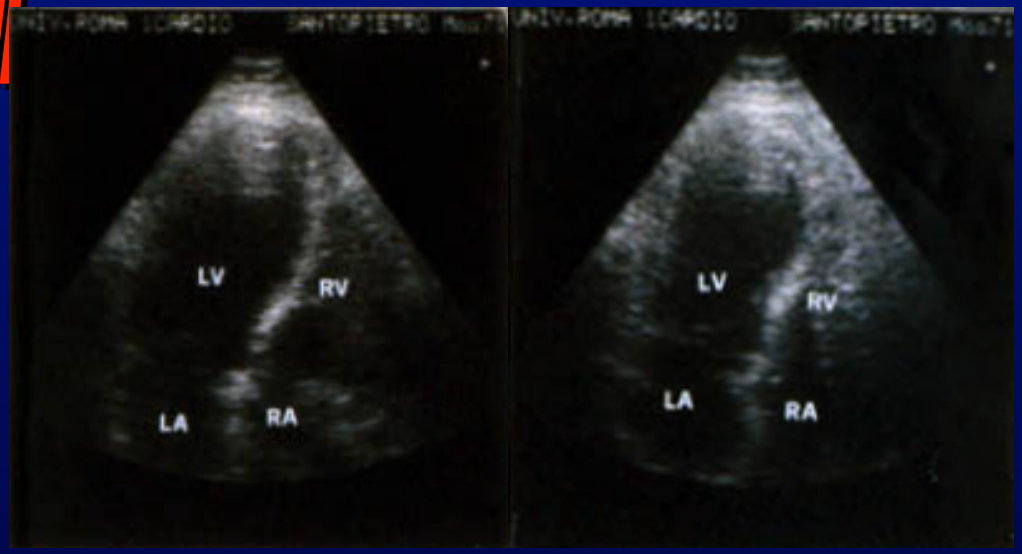
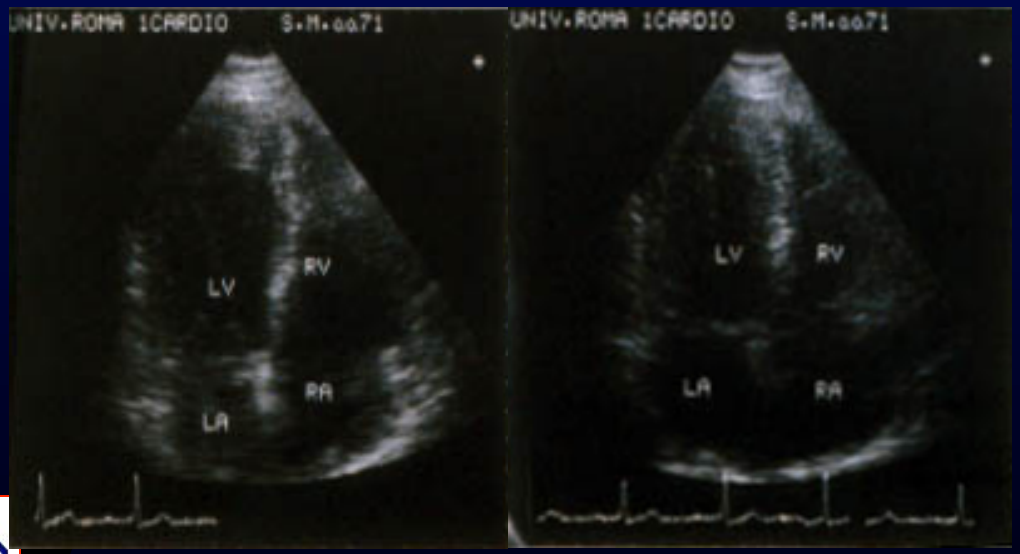
# *Rimodellamento Ventricolare*






1988
W.P. 20 mmHg
I.C. 2,2 l/m2/min
PO2 66 mmHg
SaO2 92,8 %
VTDVS 220 cc
VTSVS 140 cc
FE 36 %
DCO 9,6 cc/min/mmHG




W.P. 10 mmHg
I.C. 2,7 l/m2/min
PO2 80 mmHg
SaO2 95,8 %
VTDVS 190 cc
VTSVS 100 cc
FE 48 %
DCO 15,2 cc/min/mmHg

# *Definition*

"Remodelling" qualifies changes that result in the rearangement of normally existing structures. Although remodelling does not necessarilly define a pathological condition, myocardial remodelling is usually restricted to deseased conditions. The above definition eliminates gestional and developmental aspects and also the so-called physiological cardiac hypertrophy that follows intensive exercising .

# *Rimodellamento Ventricolare*

- *Rimodellamento fisiologico*

  in funzione: a) entità
  b) durata
  c) tipo
  dell'esercizio fisico

- *Rimodellamento Patologico*

**-Aumenti prevalenti di pre-carico**
(iperafflussi, insufficienze valvolari, etc.)

**-Aumenti prevalenti di post-carico**
(iperafflussi, insufficienze valvolari, etc.)

**-Rimodellamento ventricolare post-infartuale**
nuovo adattamento morfologico e funzionale in risposta al sovvertimento della normale topografia e alla distorsione della geometria ventricolare

# RIMODELLAMENTO FISIOLOGICO

NORMALE

ISOMETRICI (atleti di potenza)

ISOTONICI (atleti di resistenza)

RAPPORTO MASSA / VOLUME COSTANTE

GIRAFFA

UOMO

ANFIBIO

# Rimodellamento post-infartuale

Dilatazione ventricolare sinistra con distorsione della geometria ventricolare regionale e globale che si verifica dopo un infarto miocardico

Qualunque modificazione architettonica o strutturale che avviene dopo infarto miocardico sia nelle regioni infartuate che non infartuate

# Rimodellamento post-infartuale

Processo caratterizzato da:

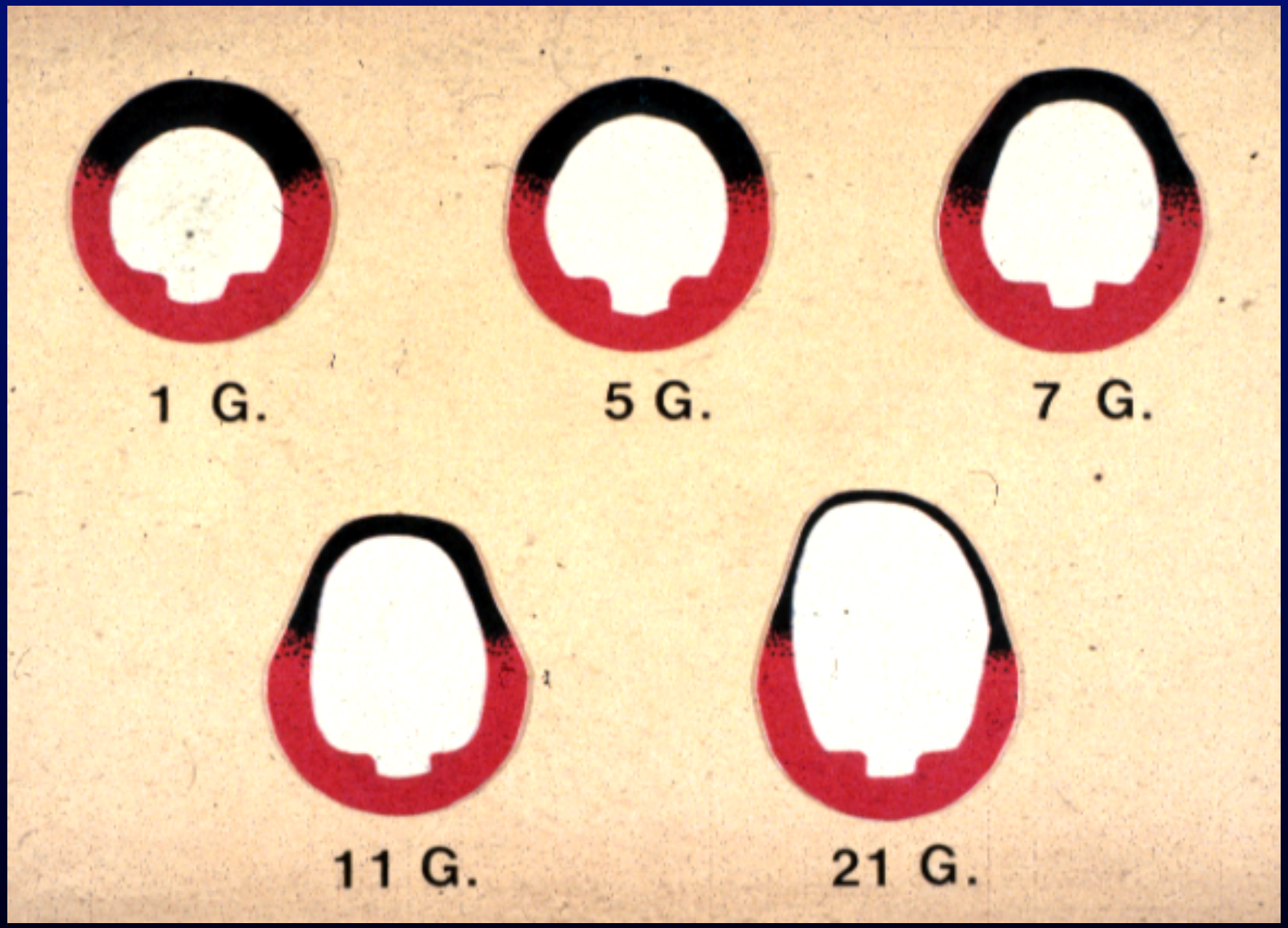
*espansione della zona infartuata*

*ipertrofia compensatoria delle regioni non ischemiche*

# LV Remodeling after AMI

**Definition**

**≥ 20% End Diastolic Volume from day 1 to 6 months follow-up**


1 G.
5 G.
7 G.
11 G.
21 G.

# Timing of LV remodeling

EARLY remodeling (in-hospital):

from 24-48 hours to pre-discharge

LATE remodeling:

from pre-discharge to 6 months

*European Heart Journal (1995) 16 (Supplement N), 31-36*

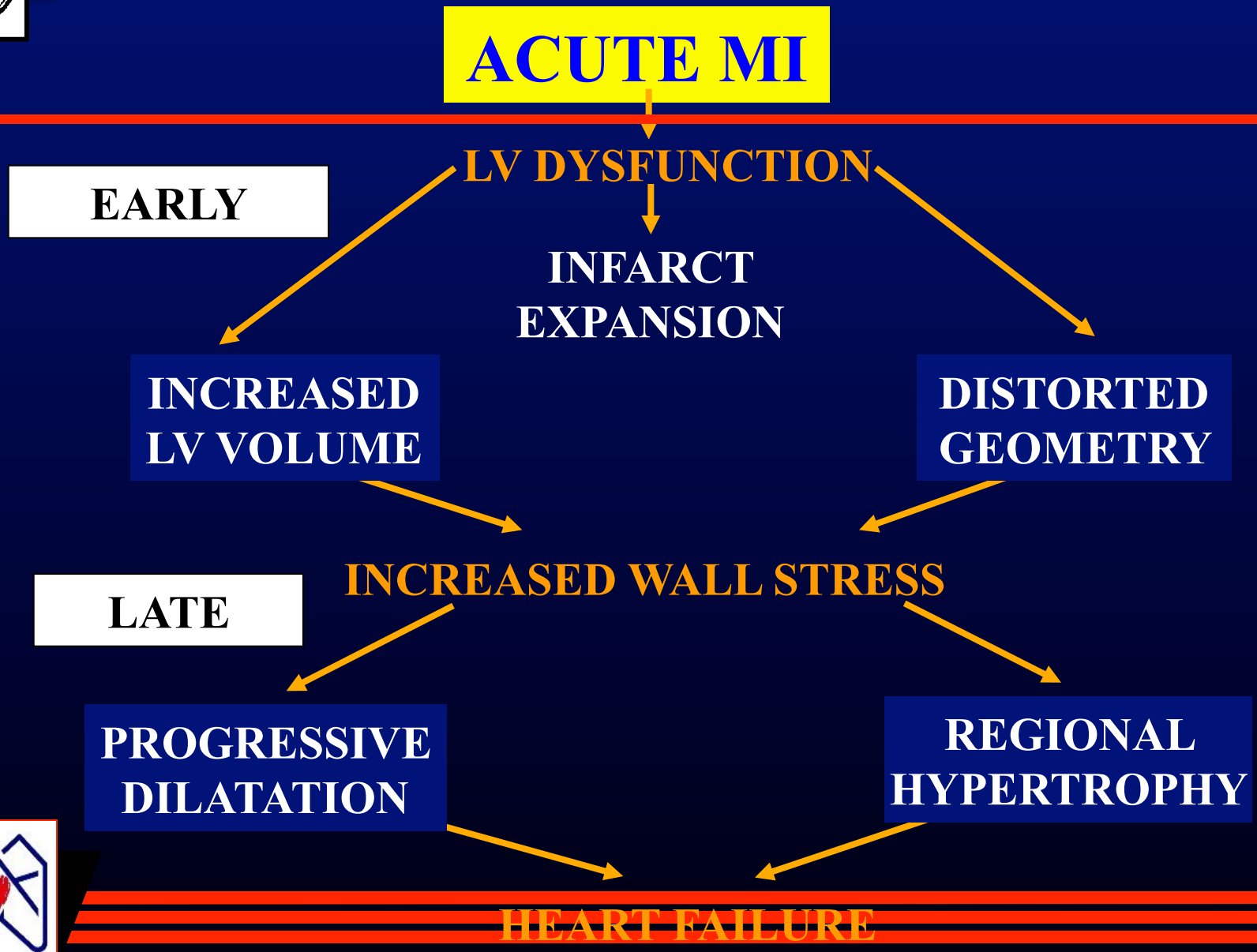
# Post-infarction myocardial remodelling: why does it happen?

G.S. Franchs and C. Chu

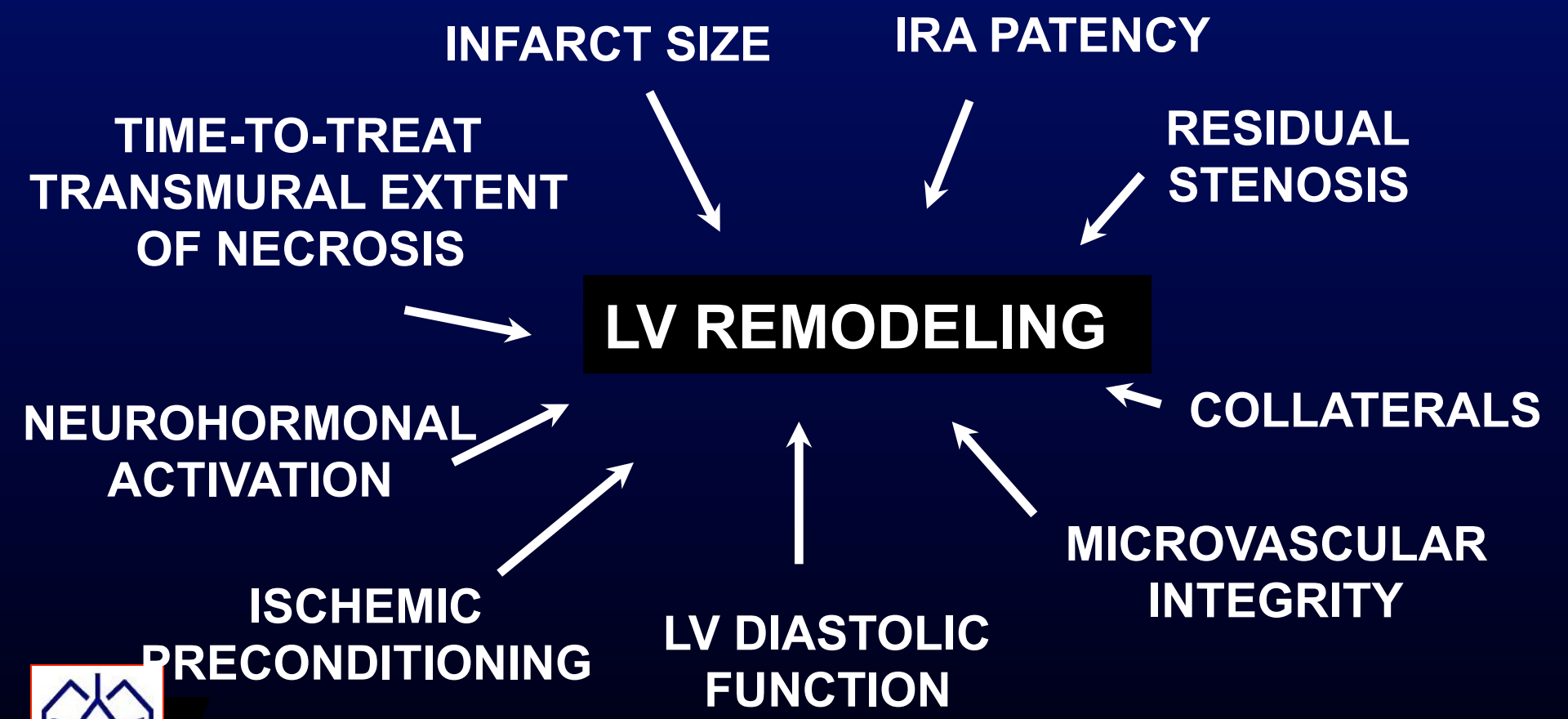
*Cardiovascular Division of Medicine, University of Minnesota Medical School, Minneapolis.*

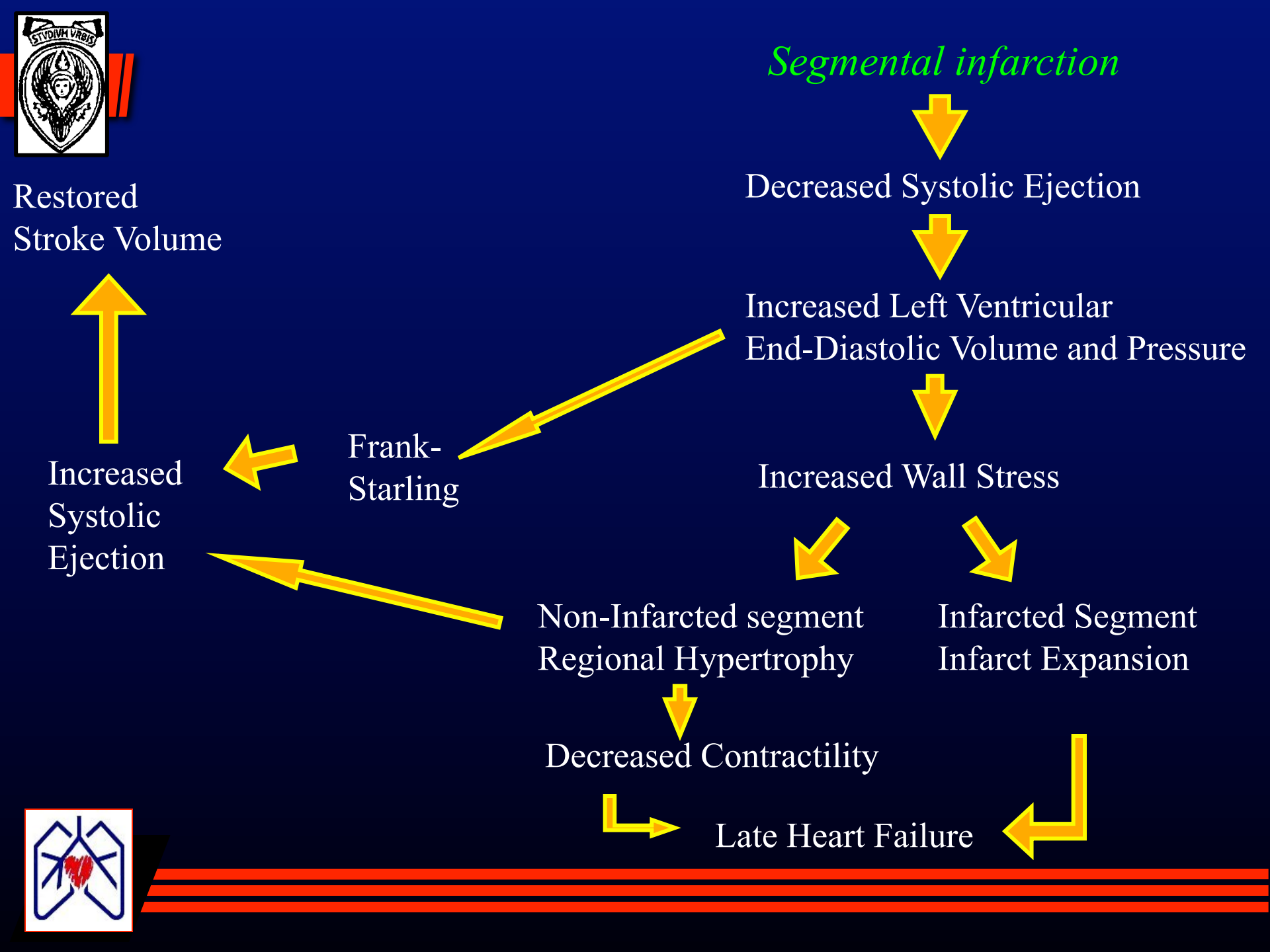
*Ninnesota U.S.A.*

# LV REMODELING AFTER AMI



# INTERACTIVE FACTORS THAT INFLUENCE LV REMODELING




Segmental infarction
Decreased Systolic Ejection
Increased Left Ventricular End-Diastolic Volume and Pressure
Increased Wall Stress
Infarcted Segment Infarct Expansion
Non-Infarcted segment Regional Hypertrophy
Decreased Contractility
Late Heart Failure
Frank-Starling
Increased Systolic Ejection
Restored Stroke Volume

# *Variabili che condizionano entità e progressività del rimodellamento ventricolare*

- *Sede dell'infarto*
- *Dimensioni dell'infarto*
- *Modalità e tempi di cicatrizzazione dell'infarto*
- *Sollecitazioni meccaniche parietali*
  *(stress telediastolico e telesistolico)*

# 1) SEDE NECROSI

**INFARTI IN SEDE ANTERIORE** → **COINVOLGIMENTO REGIONI APICALI**

↓

Più estesa compromissione

↓

**MAGGIORE FREQUENZA COMPLICANZE**

↕

Minore Spessore pareti

Più ampio Raggio di curvatura → Maggiore stress

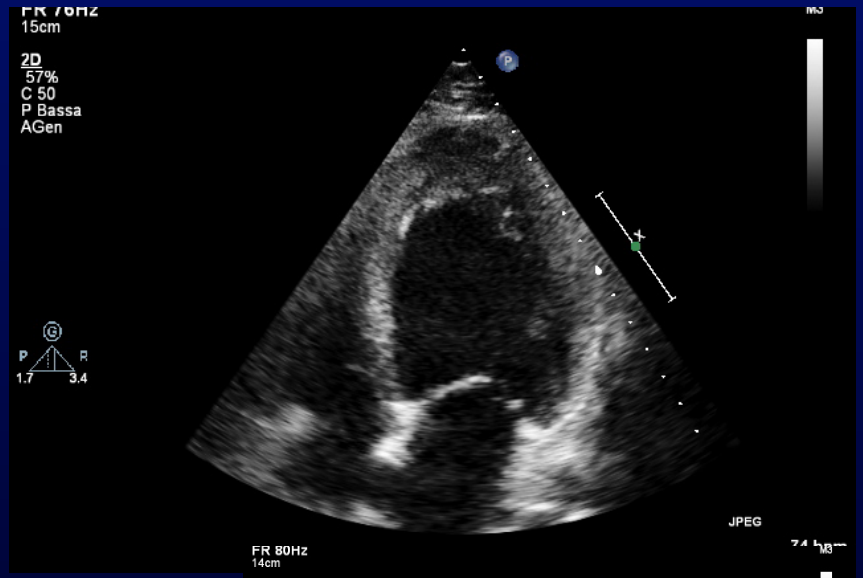
↓

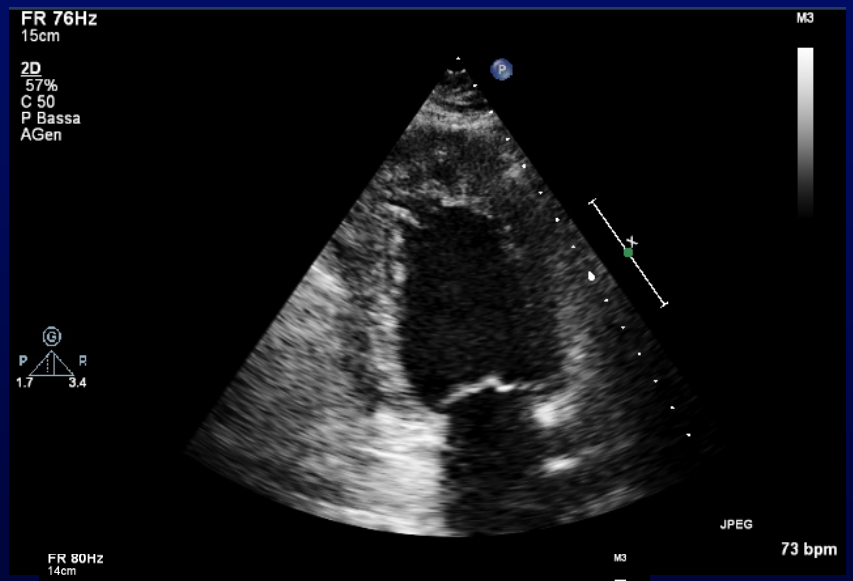
Maggiore sollecitazione meccanica

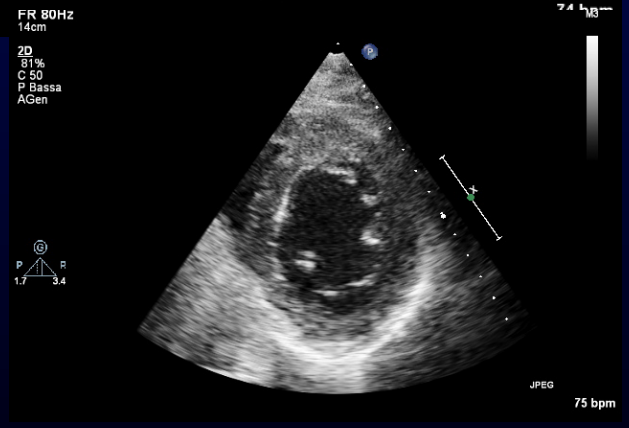
↓

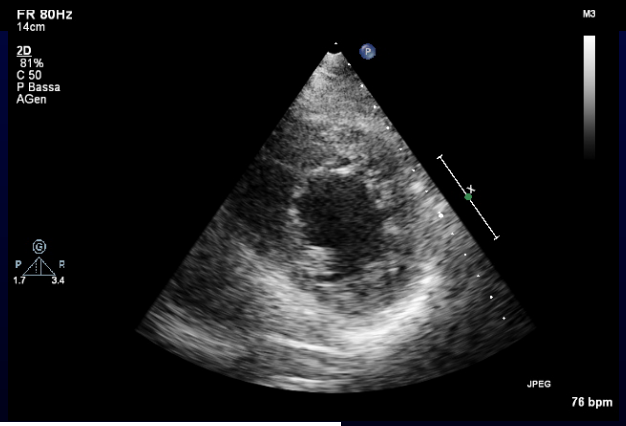
**DILATAZIONE FINO ALL' ANEURISMA**

# *2D Echo*
# antero-apical MI



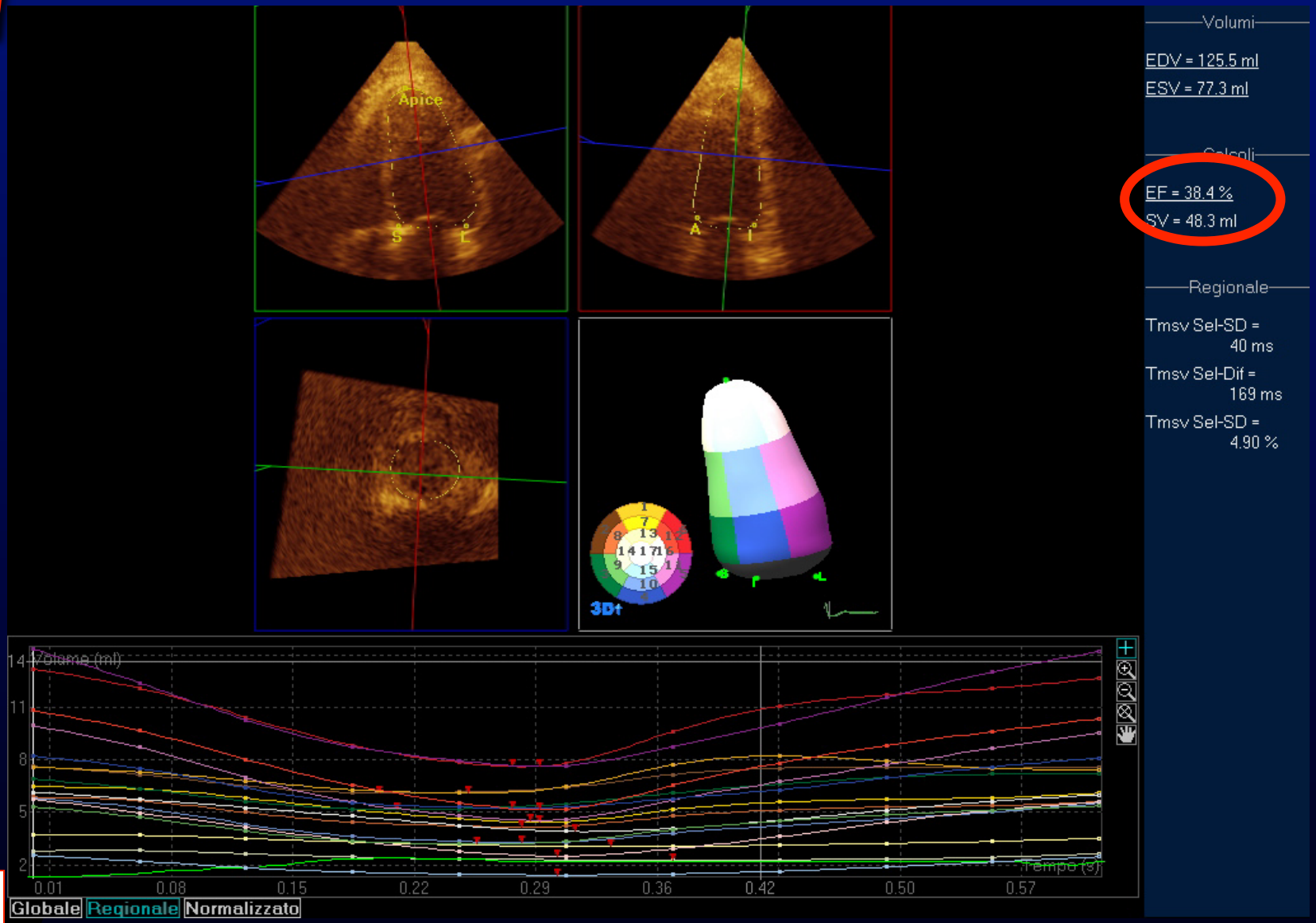



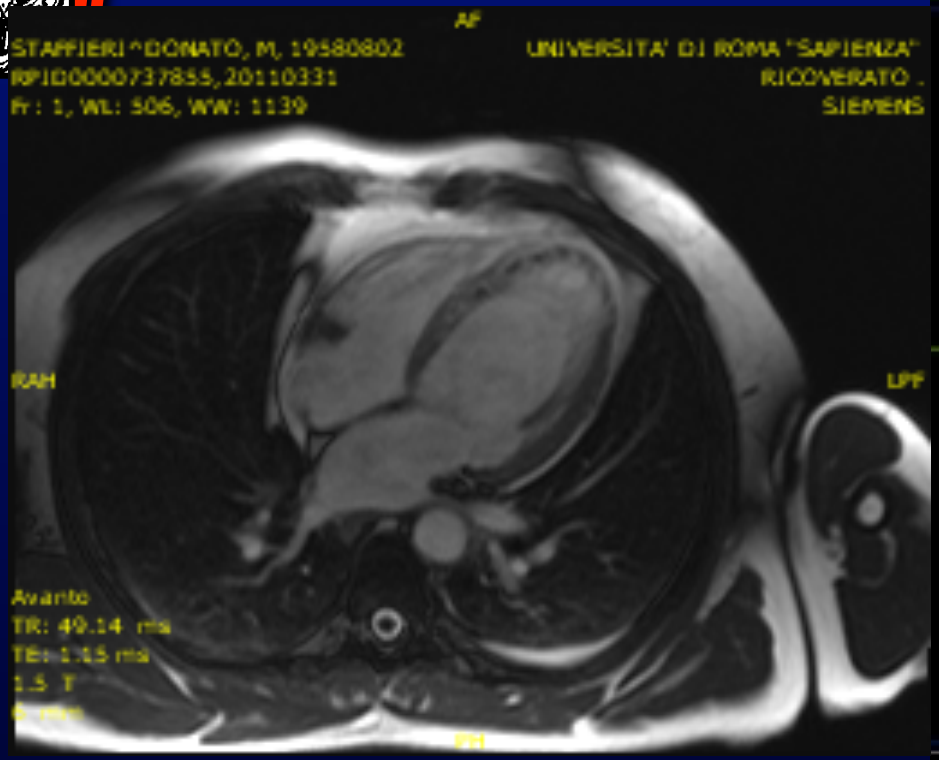



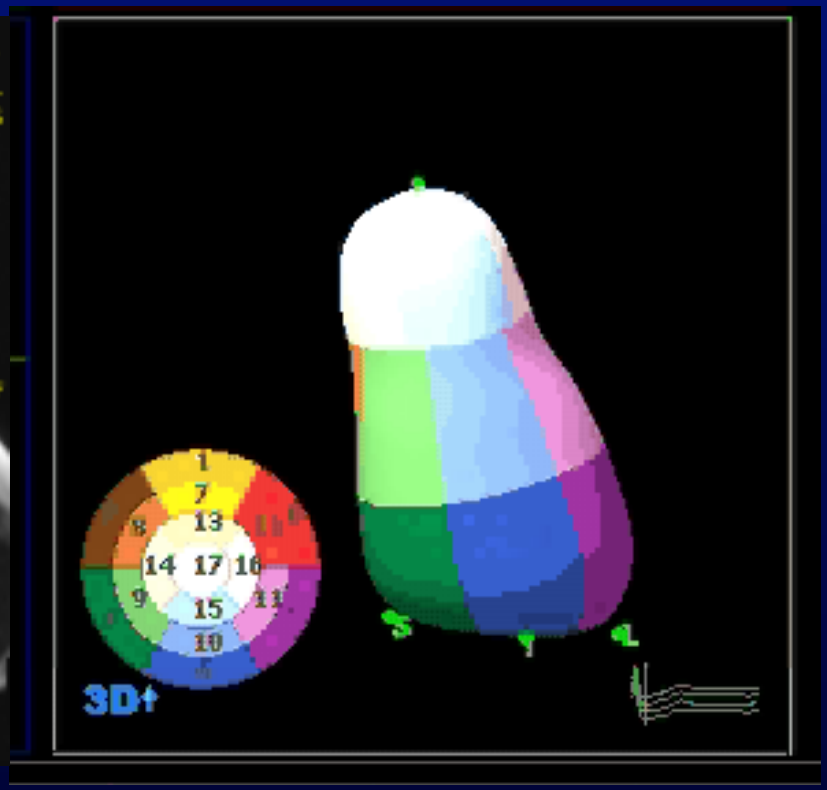




**2D ECHO qualitative EF: 35%**

# 3D ECHO: regional wall motion assessment






CMR: FE: 38% ⟷ ECHO 3D: FE: 38%

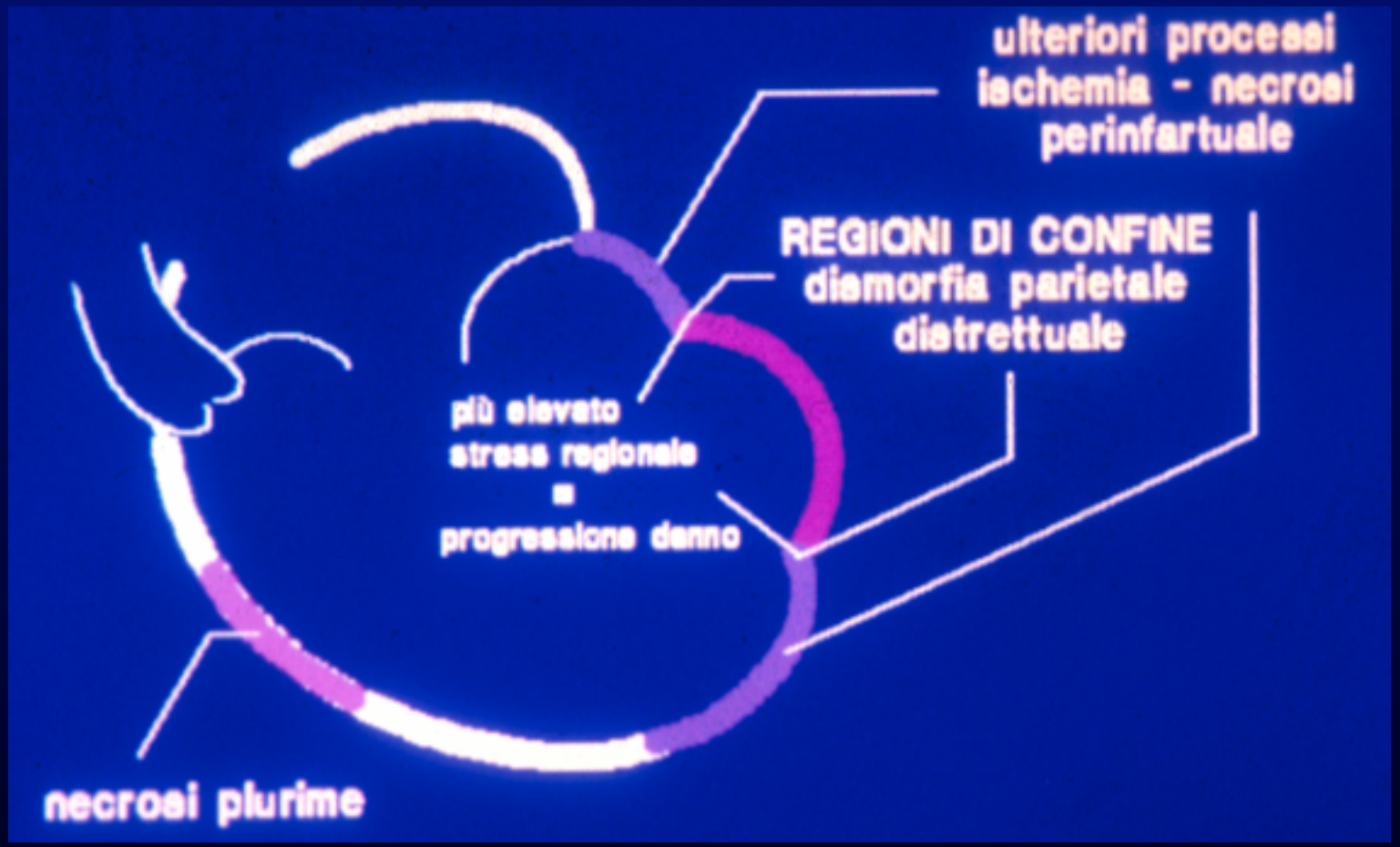
# *Variabili che condizionano entità e progressività del rimodellamento ventricolare*

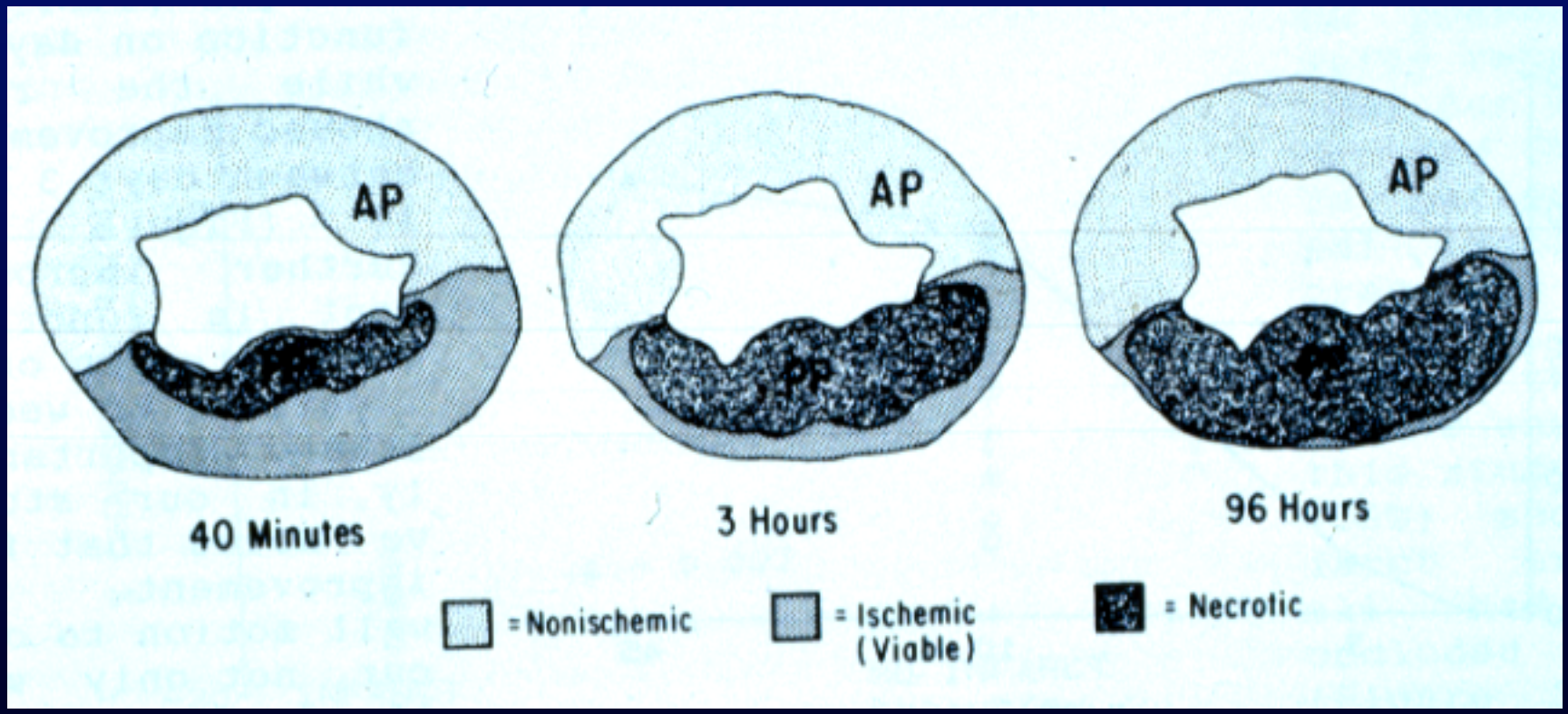
- *Sede dell'infarto*
- *Dimensioni dell'infarto*
- *Modalità e tempi di cicatrizzazione dell'infarto*
- *Sollecitazioni meccaniche parietali*
  *(stress telediastolico e telesistolico)*

# Ecocardiografia nella valutazione dell'estensione della necrosi

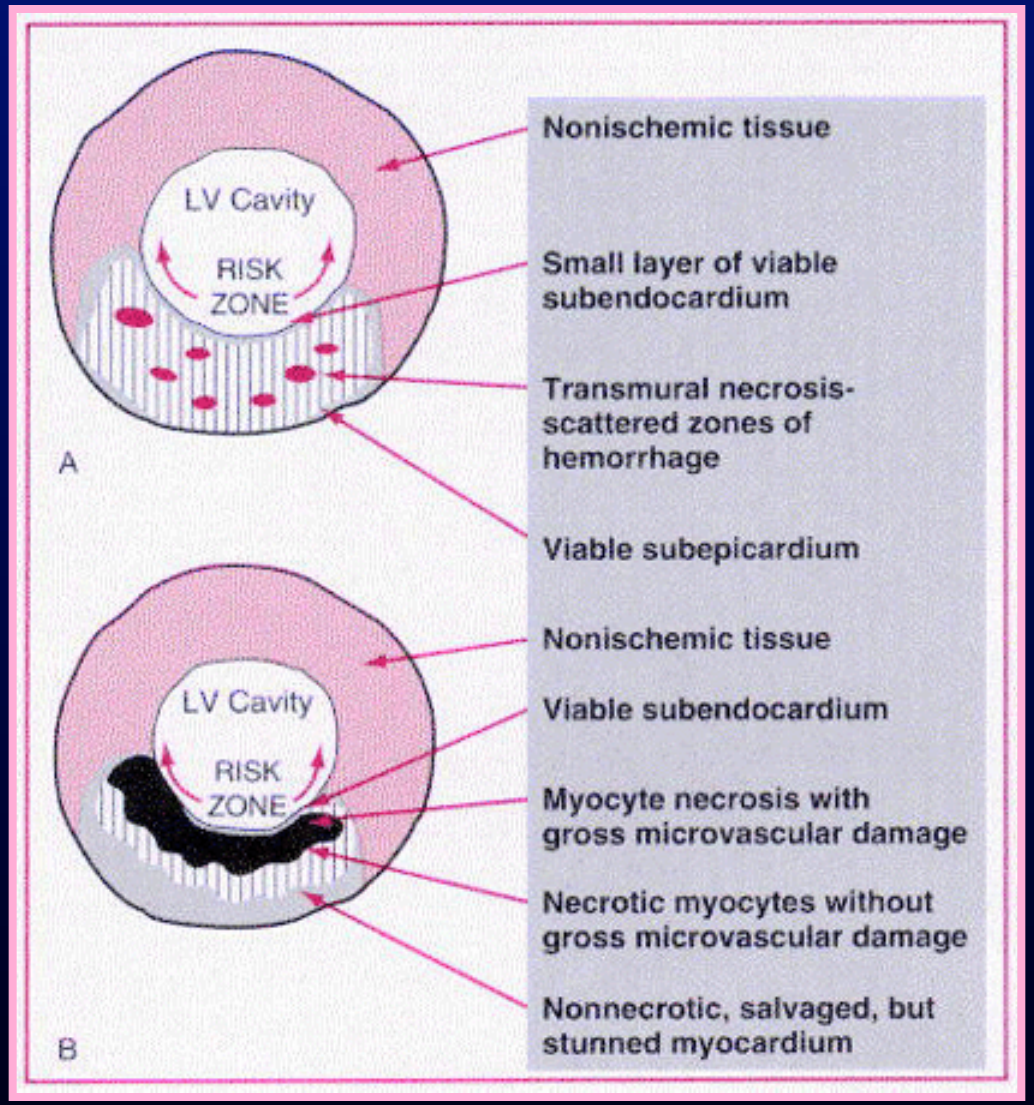
- **Inadeguatezza del parametro FE**
- **Inadeguatezza del parametro eco-score**
  *(se si prendono in considerazione le areee ipercinetiche)*
- **Alterazioni cinetiche non specifiche di danno ischemico necrotico**
- **Limitazioni nella valutazione dell'ispessimento sistolico**
- **Problematica del miocardio stunned ed ibernato**
  *(necessità di studi seriati)*
- **Impossibilità di valutare la transmuralità della necrosi**

# ESTENSIONE CIRCONFERENZIALE



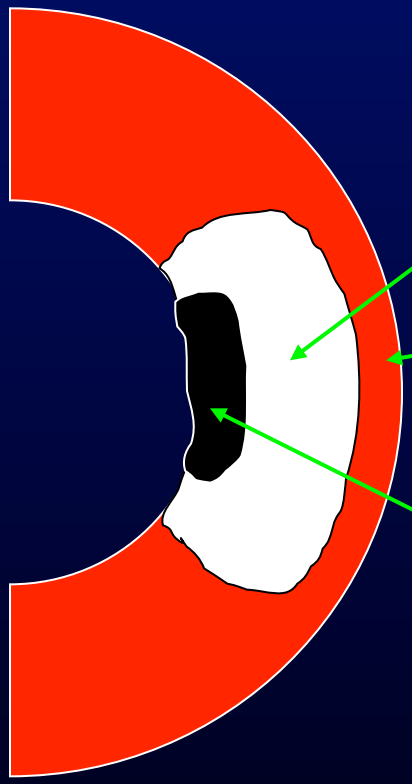

AP
AP
AP
40 Minutes
3 Hours
96 Hours
= Nonischemic
= Ischemic (Viable)
= Necrotic

# Ischemic-necrotic wavefront




From Braunwald E, Heart Disease, 6th Ed

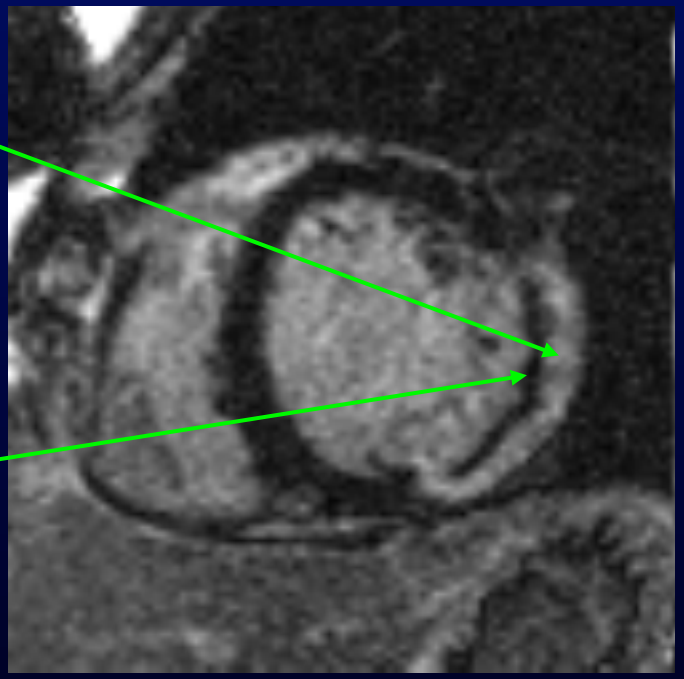
# Acute Myocardial Infarcts



Necrotic myocytes without gross microvascular damage

Non-necrotic, salvaged myocardium

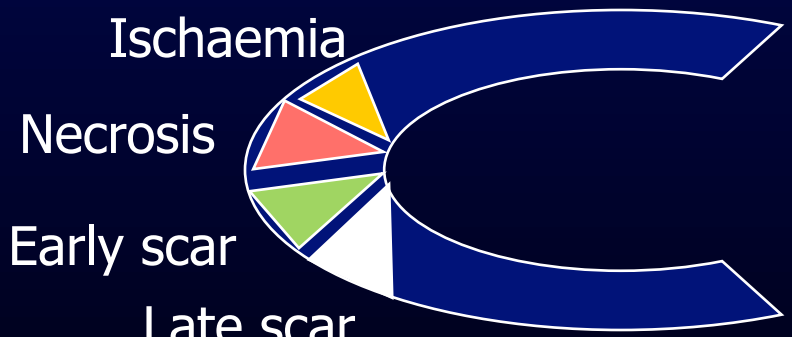
Myocyte necrosis with gross microvascular damage



# Transmural extension

Grade


Type



## Estensione Circonferenziale

Ecocardiografia

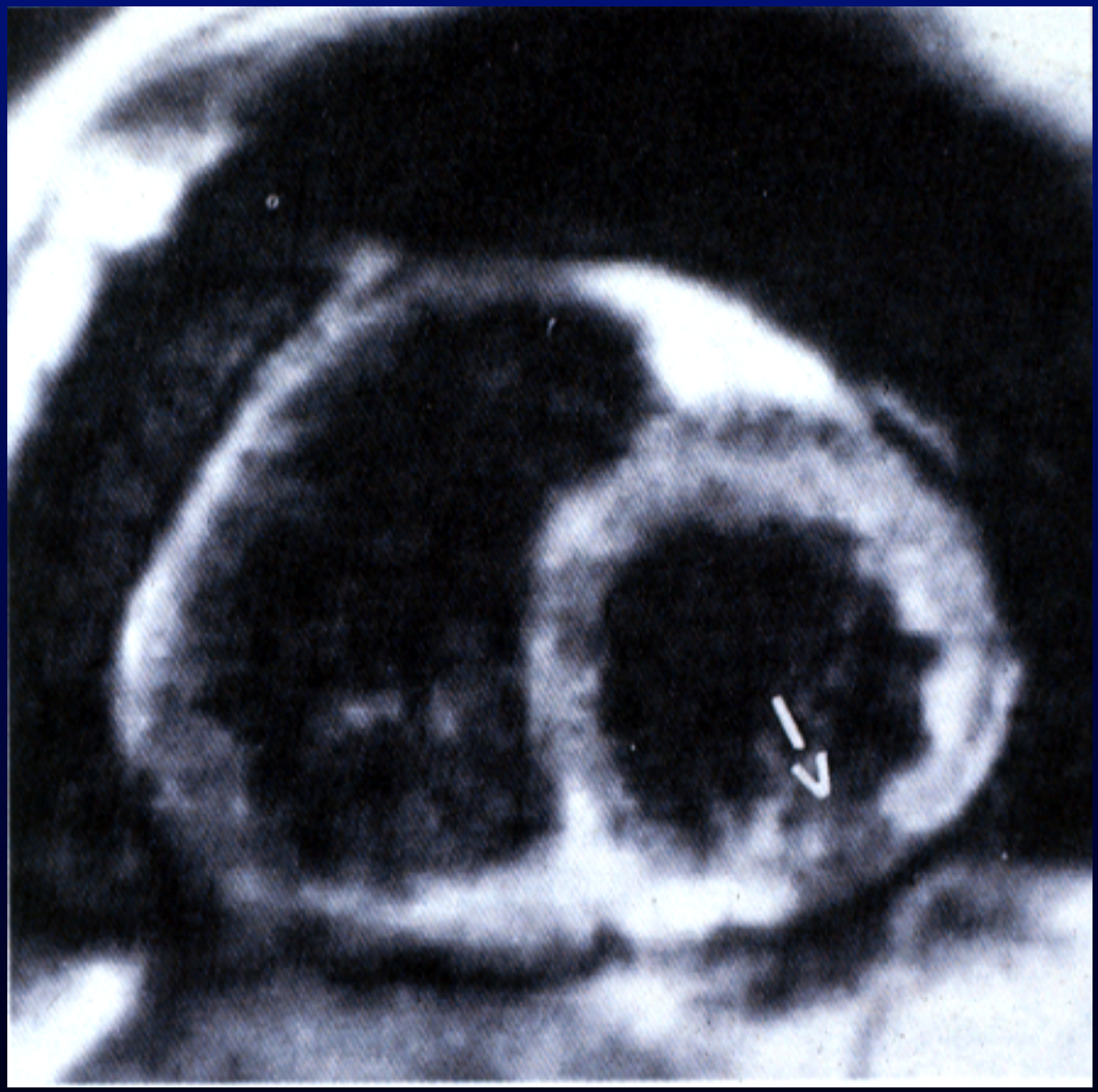
Numero settori con cinesi compromessa

## Estensione Transmurale

Caratterizzazione Tissutale

Ecocardiografia

Risonanza Magnetica

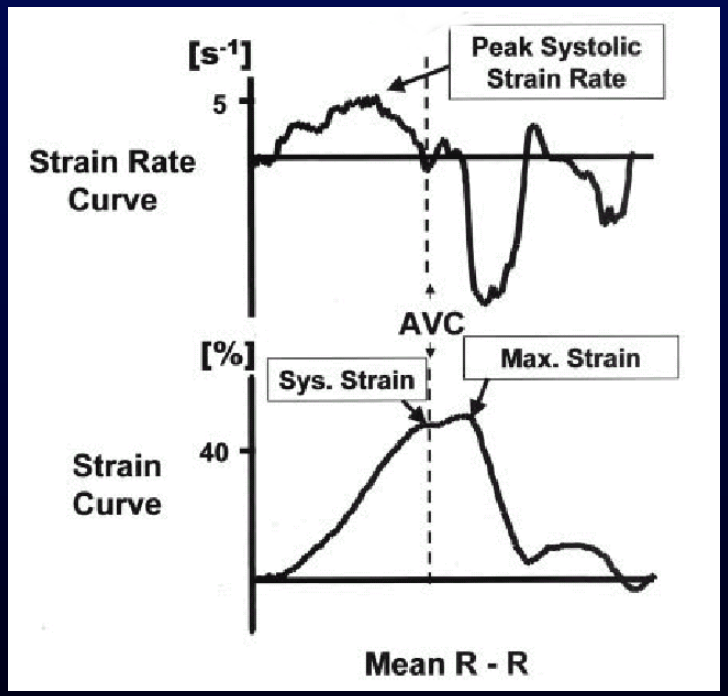

I
V

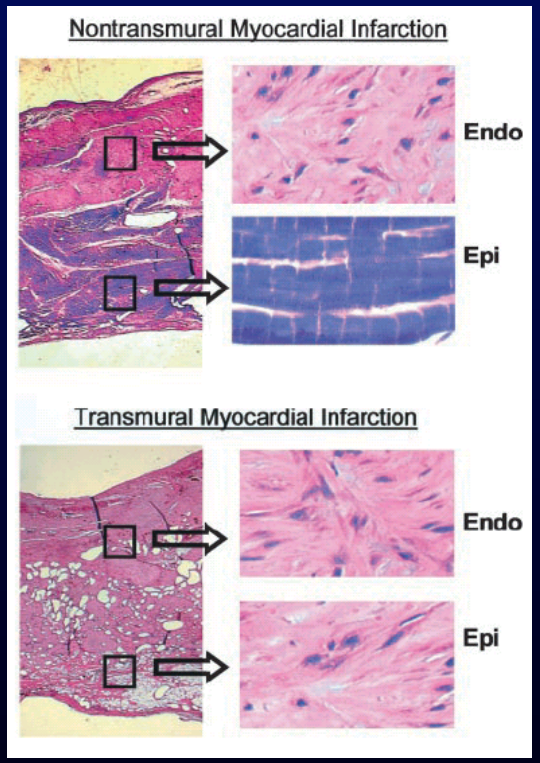
# Tissue deformation imaging:

**Strain rate-imaging** quantifies both the amount of myocardial deformation (strain) and the rate of local myocardial deformation (strain-rate)

**Defining the Transmurality of a Chronic Myocardial Infarction by Ultrasonic Strain-Rate Imaging Implications for Identifying Intramural Viability. An Experimental Study**

Weidemann et al, **Circulation. 2003**




MRI ? TDI

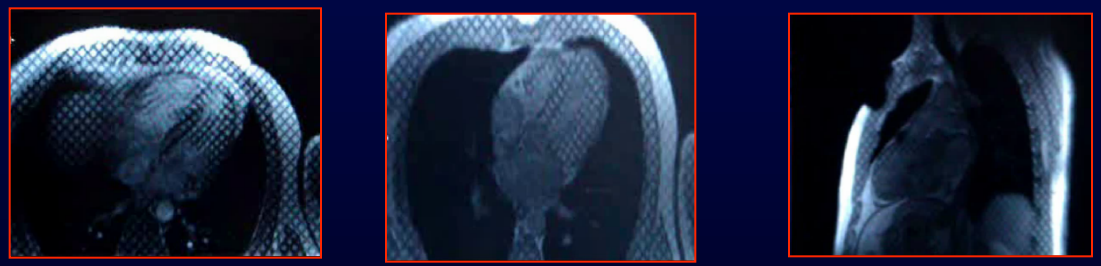
# Our experience: correlation MRI *vs* TDI derived strain

MRI Study

"On-line" analysis

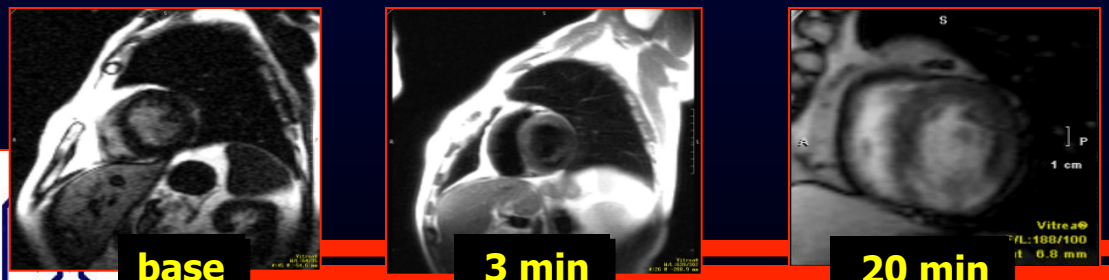
**MRI SHORT AXIS BASAL-MIDWALL-APICAL VIEWS**

**MRI 4-5-2 CHAMBERS APICAL VIEW**



Functional Study

(cine-GRE sequences)




**Contrast perfusional study (Gadolinium-diethylenetriamine pentaacetic acid)(Gd-DTPA)**

# Our experience: correlation MRI *vs* TDI derived strain

MRI study

**"Off-line" analysis**

- % THICKENING= $\frac{\text{SYSTOLIC THICKENING – DIASTOLIC THICKENING}}{\text{DIASTOLIC THICKENING}}$ X 100

- NON HYPERENHACEMENT (mm)

- NON HYPOENHACEMENT (mm)

- $\frac{\text{EARLY HYPOENHANCEMENT/ WALL THICKNESS}}{\text{LATE HYPERNHANCEMENT / WALL THICKNESS}}$ X 100

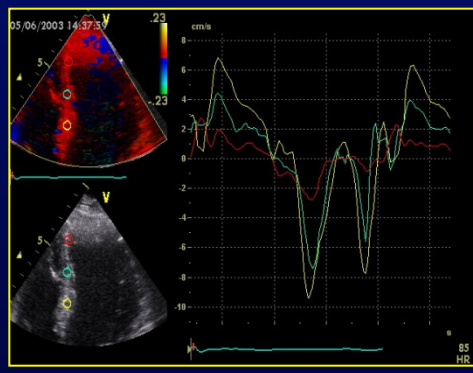
40 myocardial segments showed early hypoenhancement and /or delayed hyperenhancement

# Materials and Methods

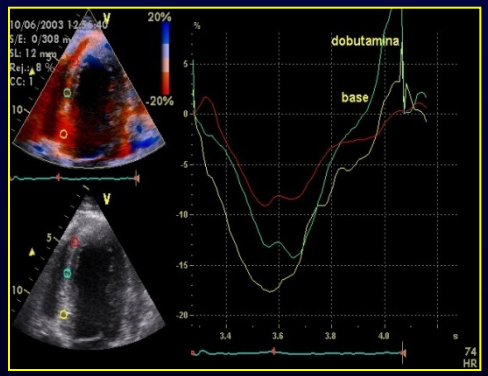
Echographic study using TDI

**"Off-line" analysis**

**Velocimetric Study** → **Velocity graphics** →



**Wall Strain Study** → **"Strain" graphics** →



# Ventricular remodeling

| | Non Remodelled | Remodelled |
|---|---|---|
| **Patients (n)** | 9 | 3 |
| **Strain** | -12.9 ± 4.4 | -4.8 ± 2.8 |
| **Hypo (mm)** | 0.4± 1.5 | 1.0 ± 2.2 |
| **Hyper (mm)** | 7.3 ± 2.3 | 6.7± 3.8 |
| **Non Hypo (mm)** | 9.5± 1.7 | 8.4 ± 3.8 |
| **Non Hyper (mm)** | 2.6 ± 2.8 | 2.7 ± 2.6 |
| **Hypo/Hyper (%)** | 18.6 ± 18.8 | 31.9 ± 39 |

# Ventricular remodeling

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|---|---|---|
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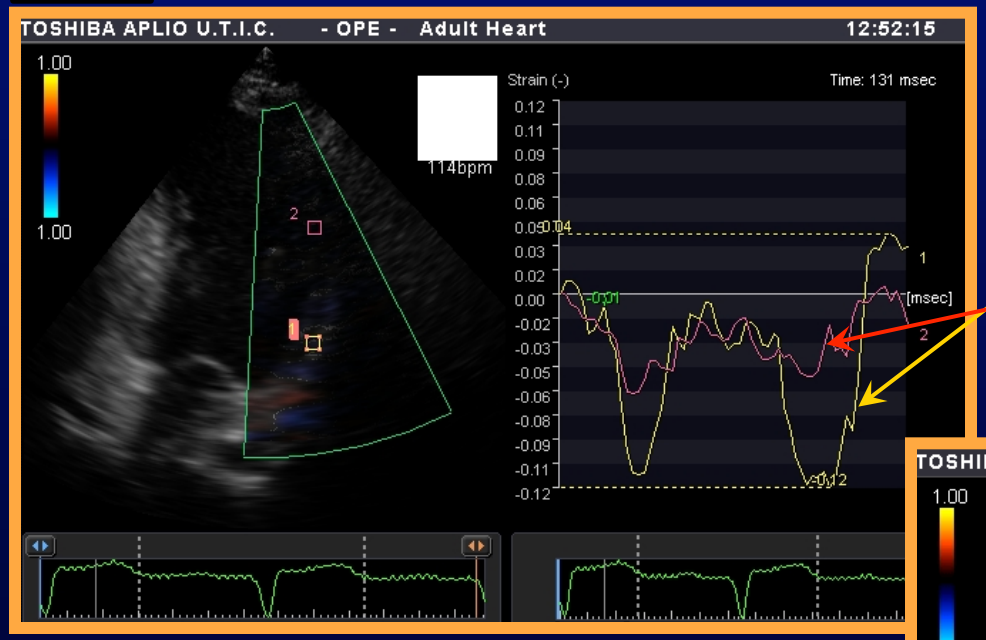
| **Hypo/Hyper (%)** | 18.6 ± 18.8 | 31.9 ± 39 |

# What about delayed hyperenhancement ?

# *Our experience…*

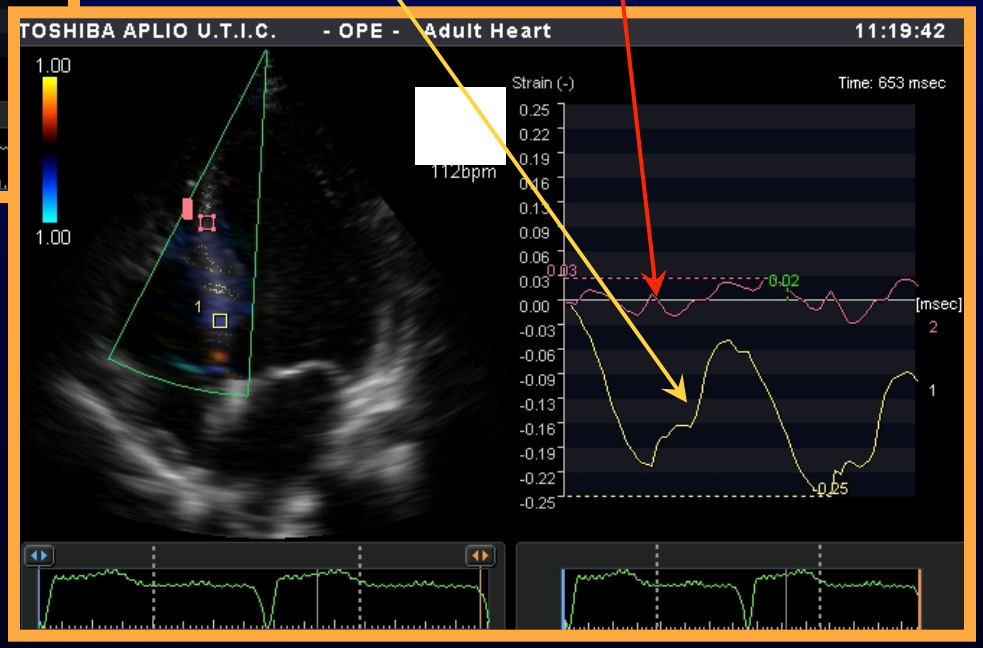
## *MRI delayed hyperenhancement vs TDI strain*




2 chamber view in antero-apical STEMI: anterior wall strain curves.

middle segment

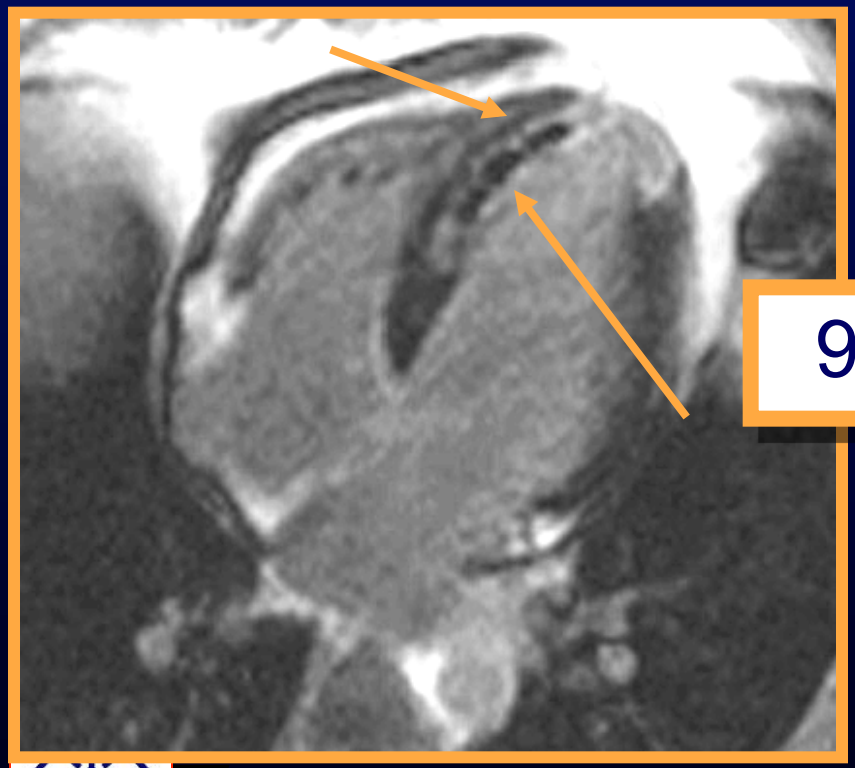
basal segment



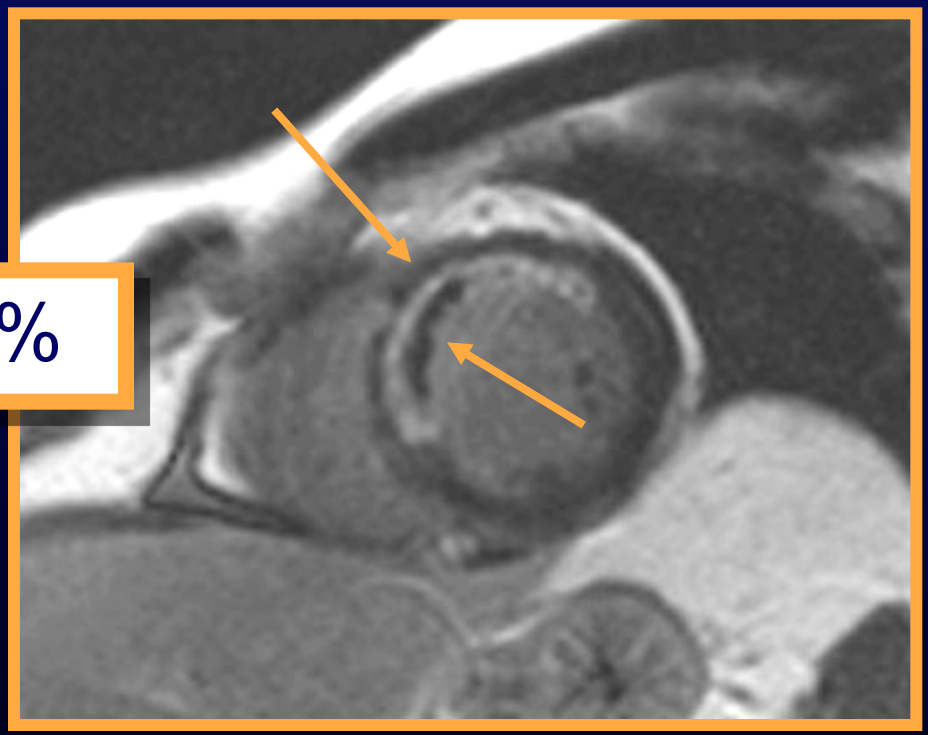

4 chamber view in antero-apical STEMI: septal strain curves.

# MRI QUANTIFICATION

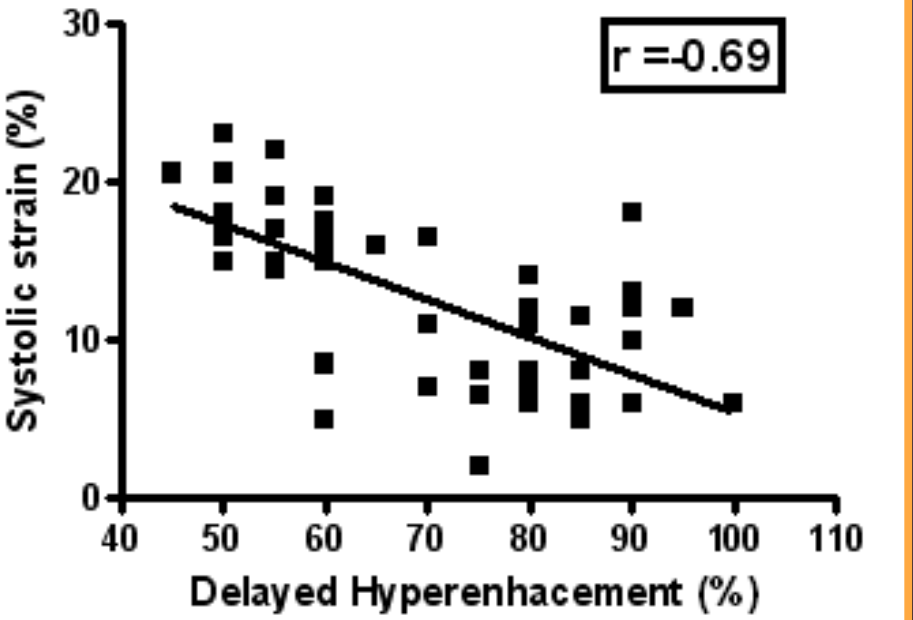
Antero-apical STEMI: delayed hyperenhancement.



90%



# RESULTS

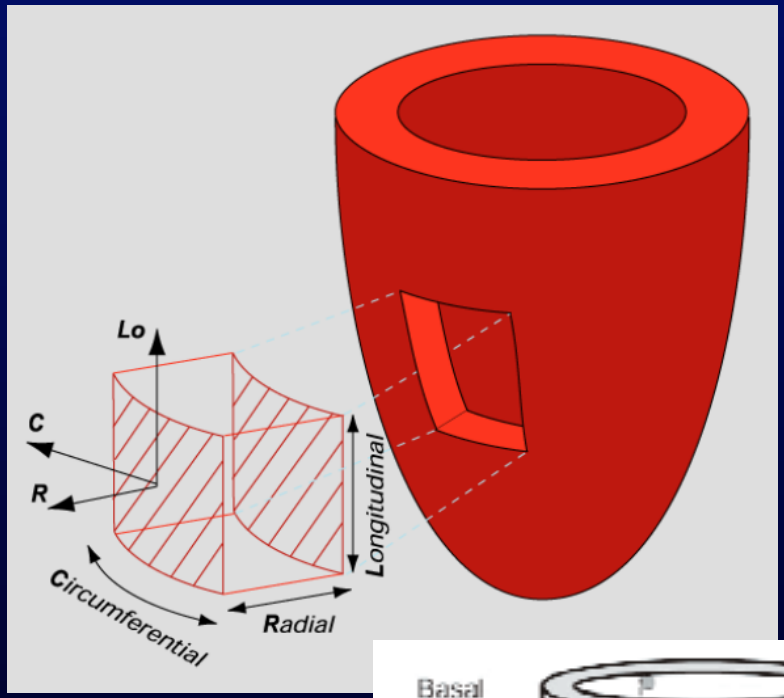



**Significant systolic strain-delayed hyperenhancement correlation.**
**(r=0.69; p<0.0001)**

# *FINAL CONSIDERATIONS*

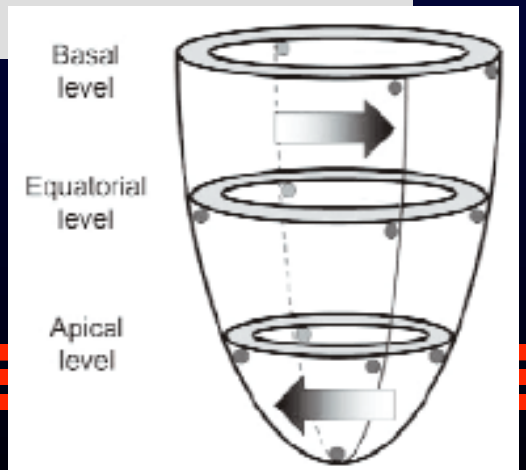
1. A significative **INVERSE CORRELATION** between the TRANSMURALITY of MYOCARDIAL NECROSIS and SYSTOLIC STRAIN was found;
2. In the clinical setting of ST-elevation myocardial infarction **SYSTOLIC STRAIN** may represent a PREDICTIVE INDEX of the TRANSMURAL EXTENSION of MYOCARDIAL NECROSIS.

# *Multi-dimensional deformation*
## *new models of myocardial deformation*



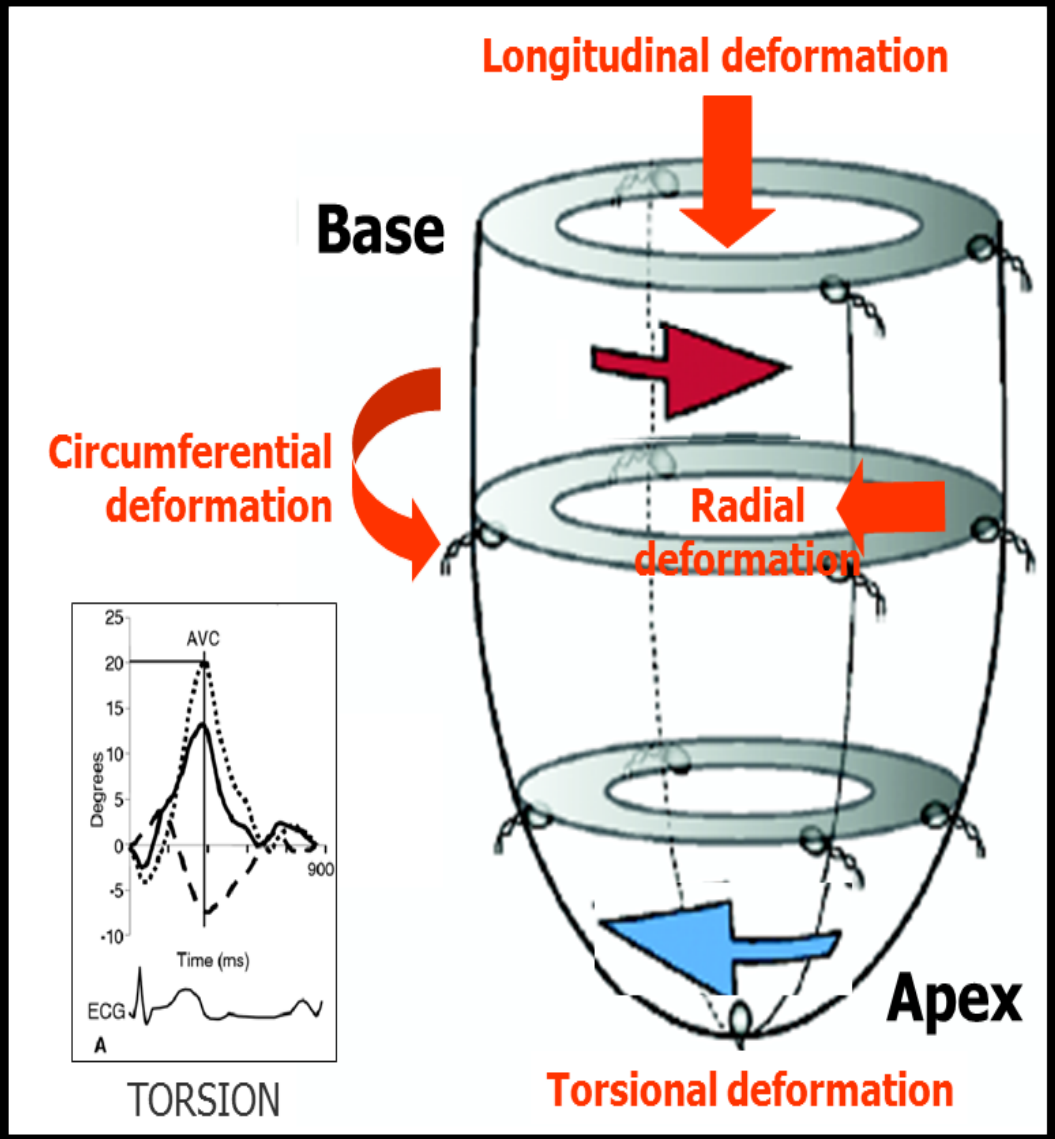

Longitudinal:
sub-endocardium fibers base/ apex shortening

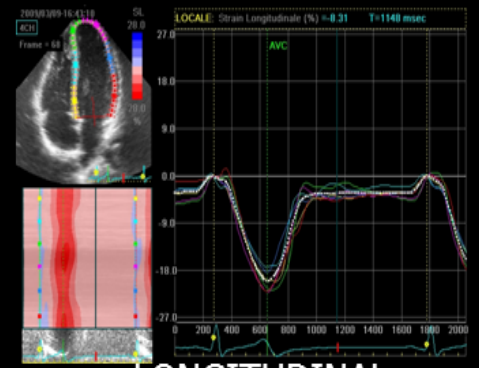
**Circumferential:**
**circular border fibers shortening**

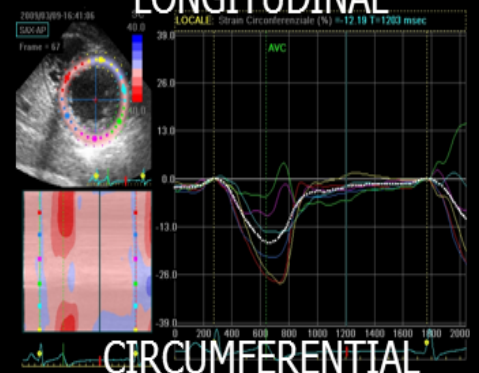


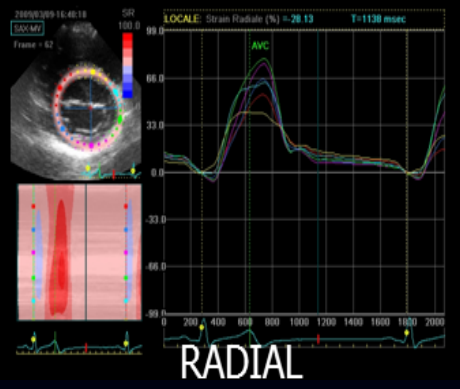

**Radial: wall thickening**

# Speckle Tracking Echo: analysis of strain



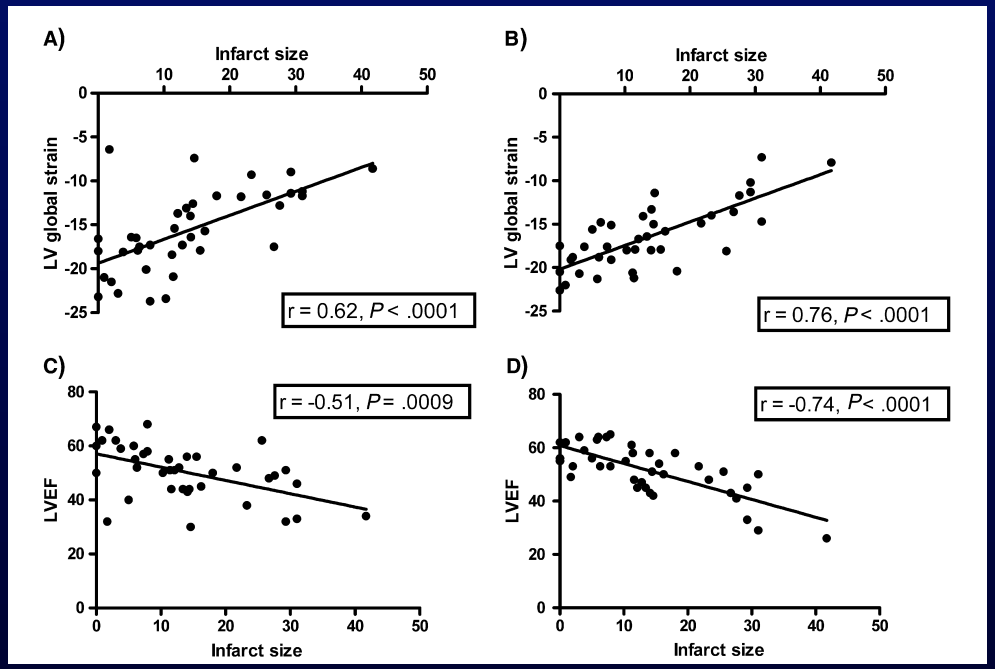



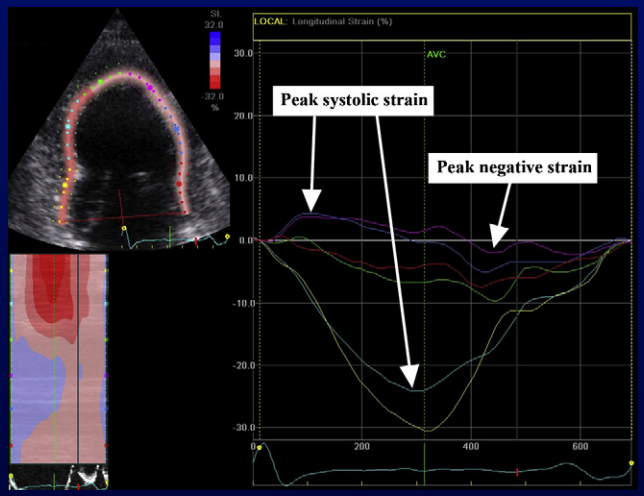



# Comparison of Left Ventricular Ejection Fraction and Left Ventricular Global Strain as Determinants of Infarct Size in Patients with Acute Myocardial Infarction

Benthe Sjøli, MD, Stein Ørn, MD, Bjørnar Grenne, MD, Trond Vartdal, MD, Otto A. Smiseth, MD, PhD, Thor Edvardsen, MD, PhD, and Harald Brunvand, MD, PhD, *Olso, Norway*




- LV global strain is a more precise diagnostic predictor of large infarcts compared with LVEF and is more reproducible.
- Global strain measured after revascularization demonstrates advantages over LVEF in the evaluation of LV injury in patients with ST-elevation myocardial infarction.

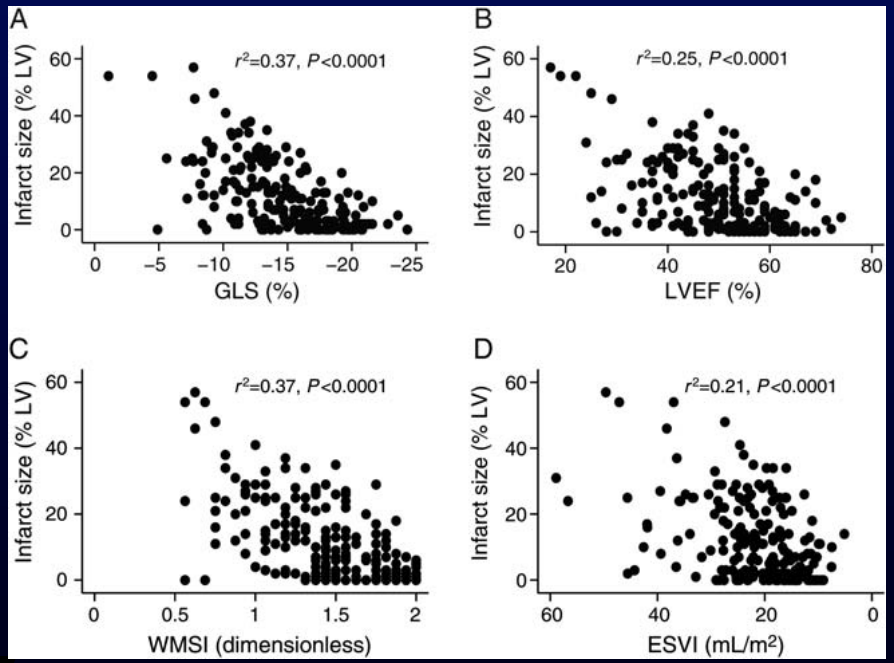
J Am Soc Echocardiogr 2009;22:1232-8

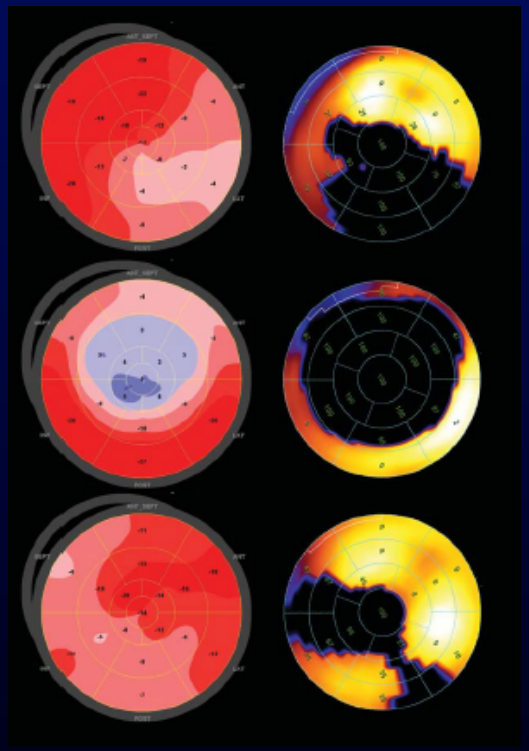
European Journal of Echocardiography

# Global longitudinal strain by speckle tracking for infarct size estimation

Kim Munk[1,*], Niels Holmark Andersen[1], Søren Steen Nielsen[2], Bo Martin Bibby[3], Hans Erik Bøtker[1], Torsten Toftegaard Nielsen[1] and Steen Hvitfeldt Poulsen[1]

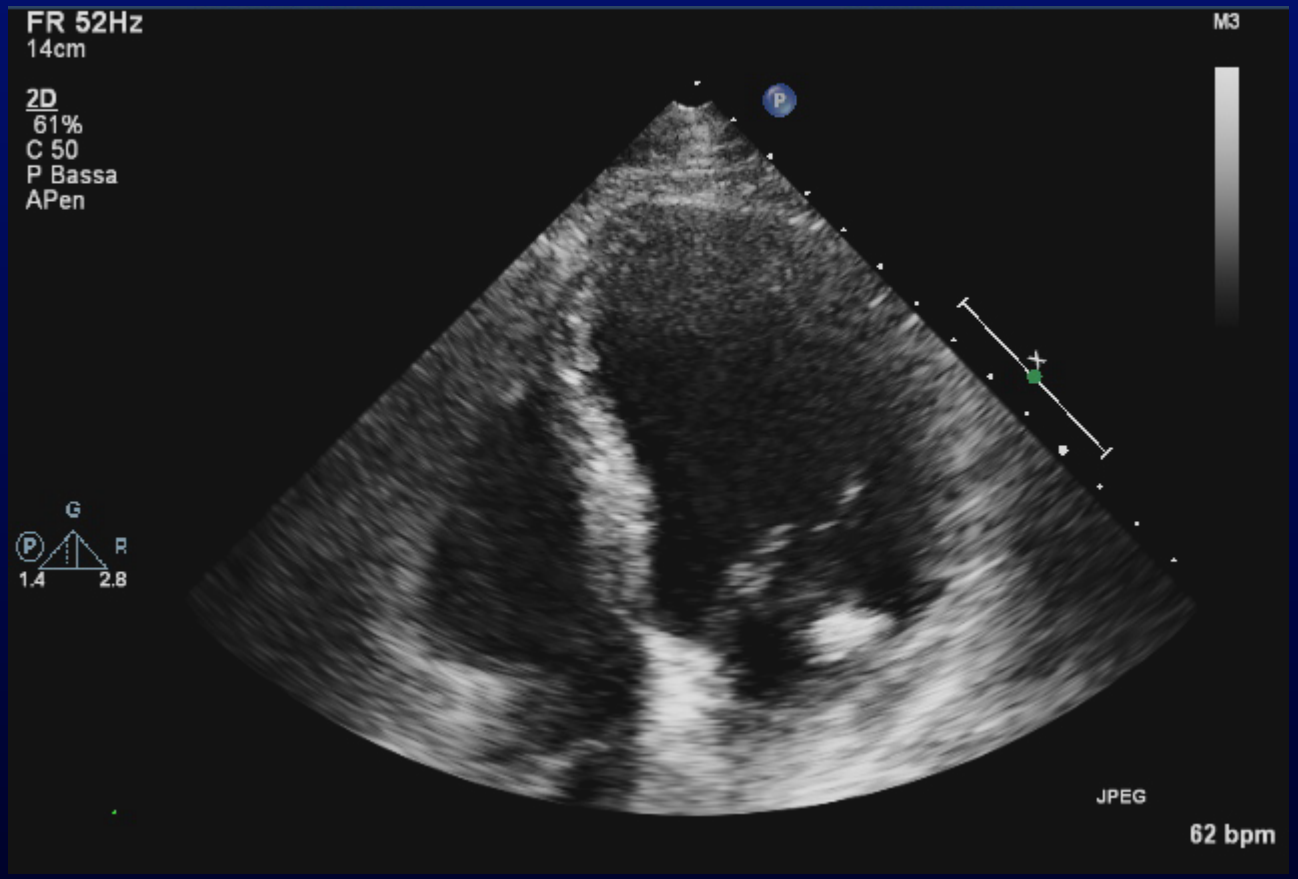
Linear regression showed good correlation between GPLS and Infarct Size (assessed by SPECT)



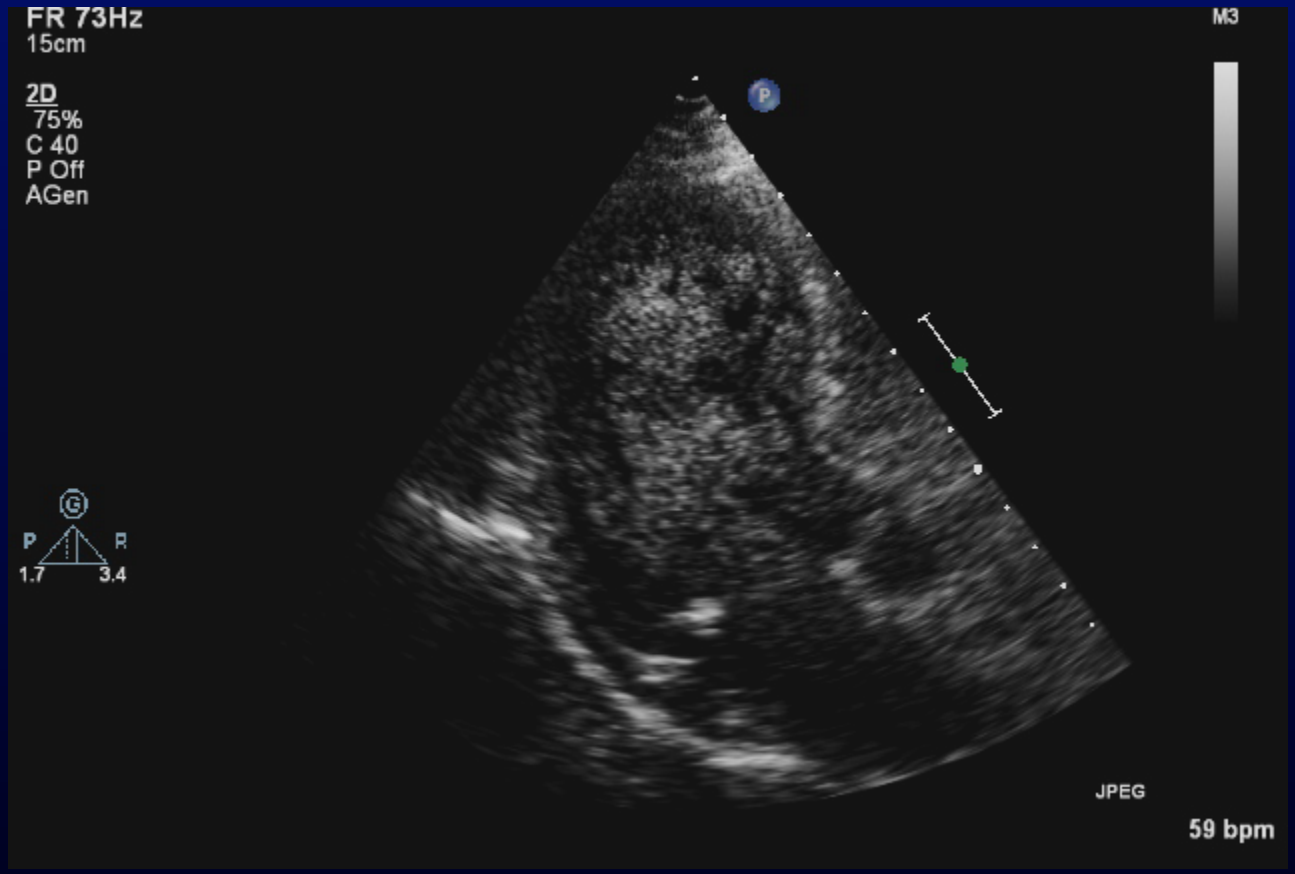



Eur J Echocardiography (2011) 12, 156–165

# Our experience: a pt with acute apical MI

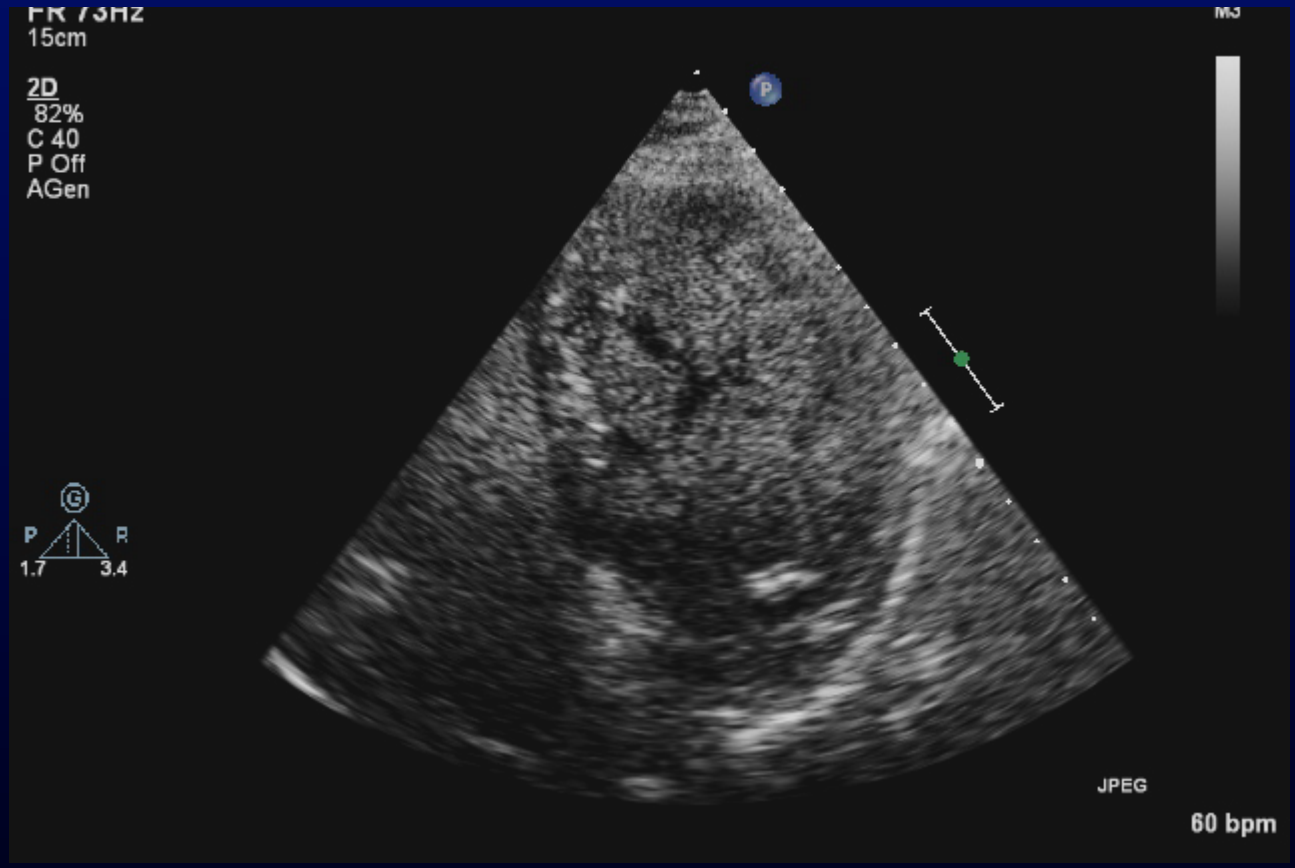


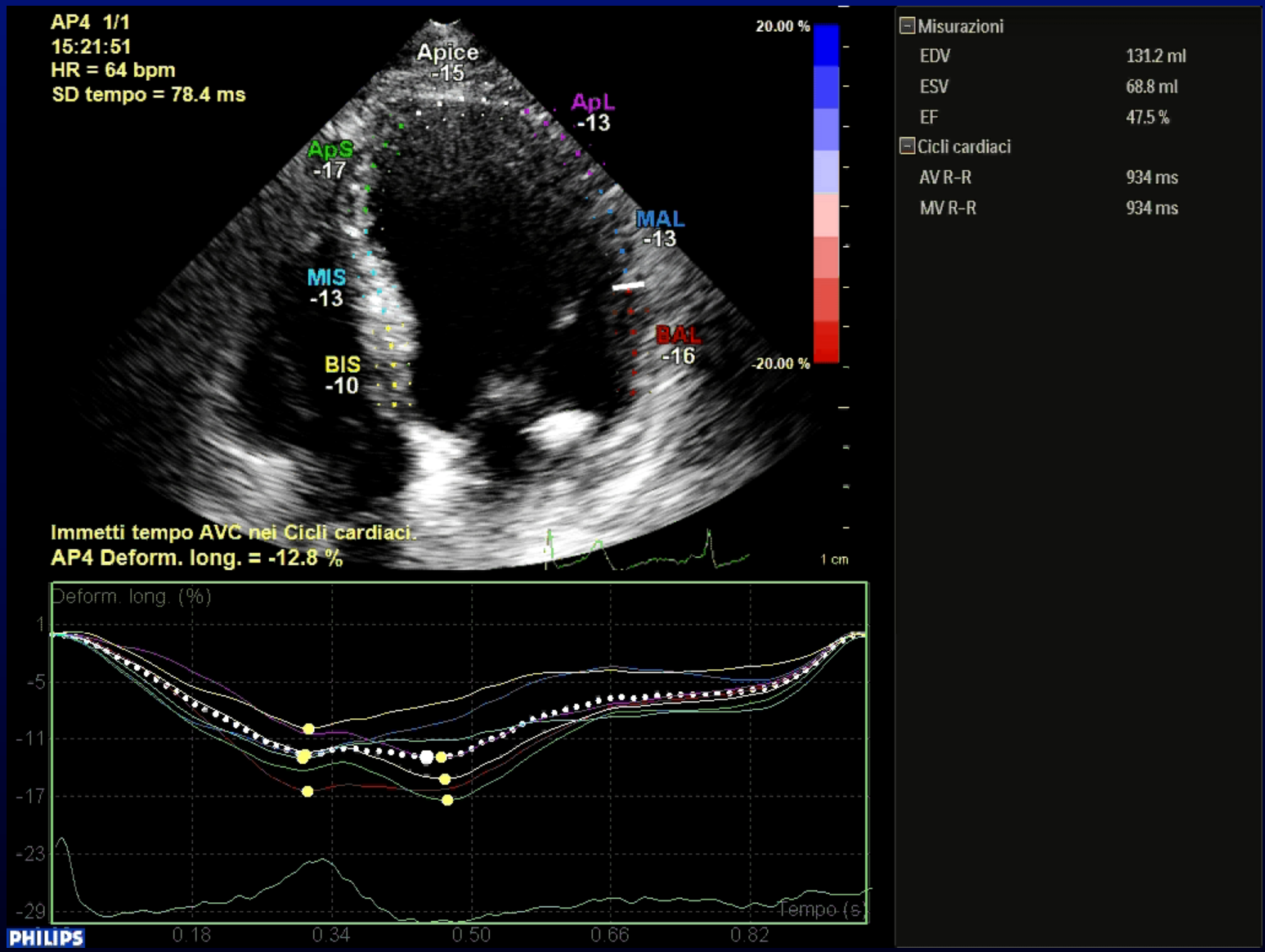
# Acoustic window improved by CE



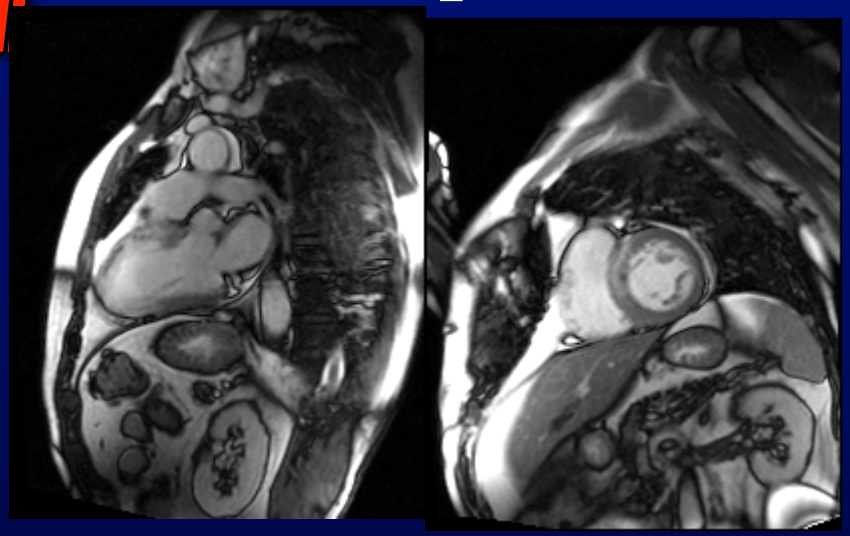
# Acoustic window improved by CE

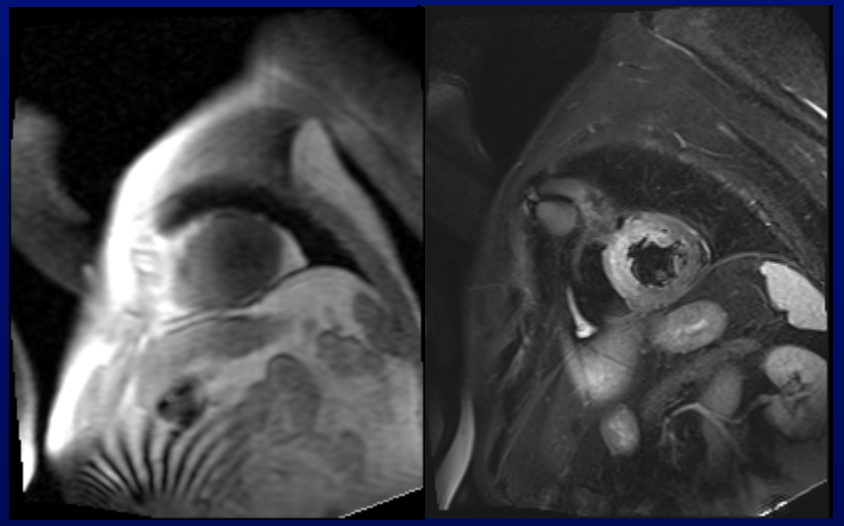
Wide apical WMA




AP4 1/1
15:21:51
HR = 64 bpm
SD tempo = 78.4 ms
Apice
-15
ApL
-13
ApS
-17
MAL
-13
MIS
-13
BAL
-16
BIS
-10
20.00 %
-20.00 %
Immetti tempo AVC nei Cicli cardiaci.
AP4 Deform. long. = -12.8 %
1 cm
Deform. long. (%)
1
-5
-11
-17
-23
-29
Tempo (s)
0.18
0.34
0.50
0.66
0.82
PHILIPS
Misurazioni
EDV 131.2 ml
ESV 68.8 ml
EF 47.5 %
Cicli cardiaci
AV R-R 934 ms
MV R-R 934 ms

# Acute phase

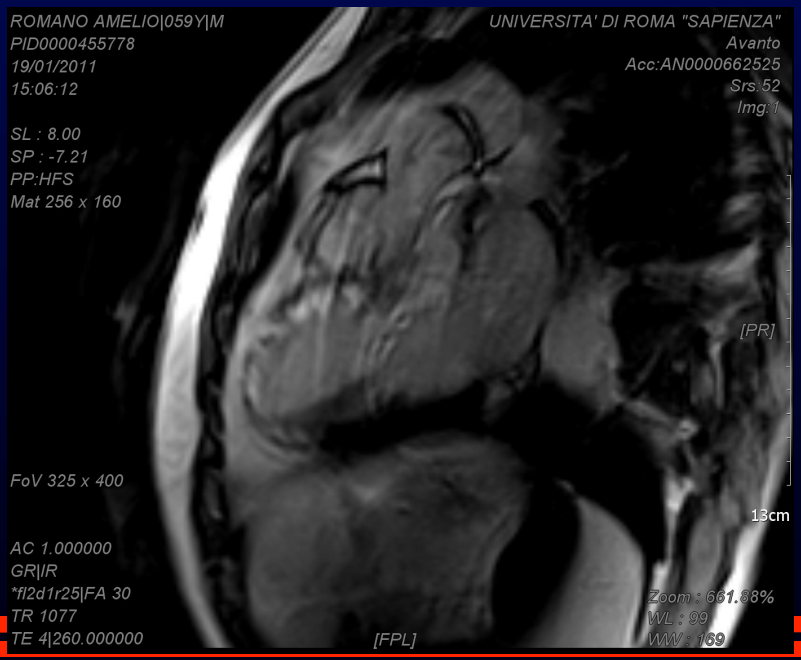


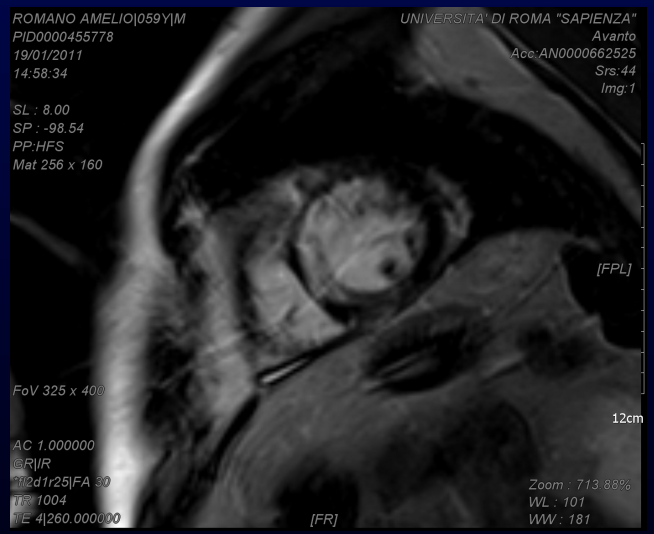


First pass

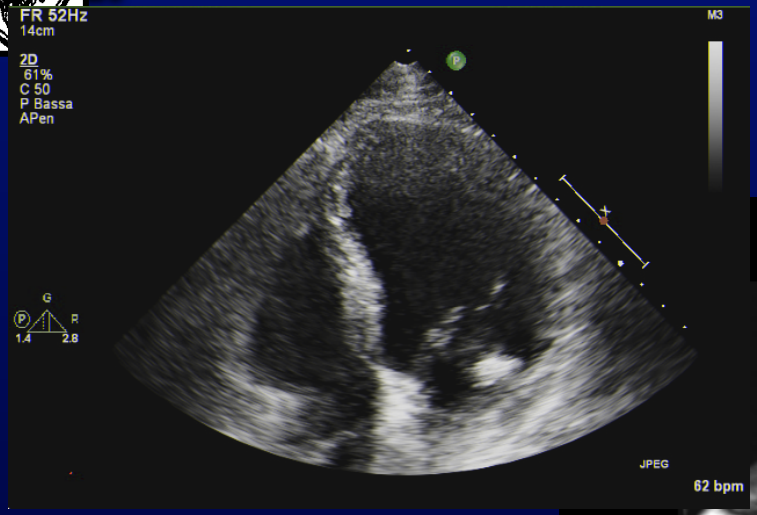
edema

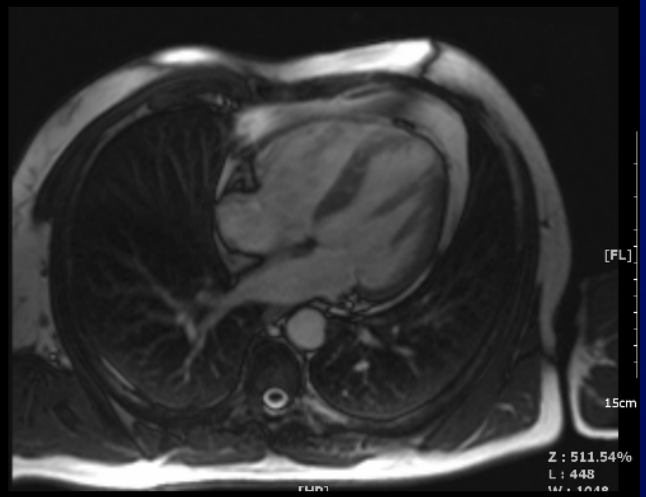
LGE

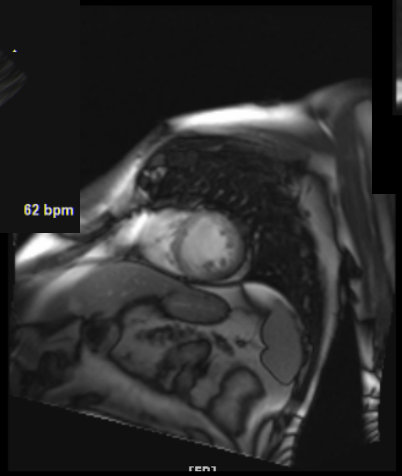


# ..6 months later



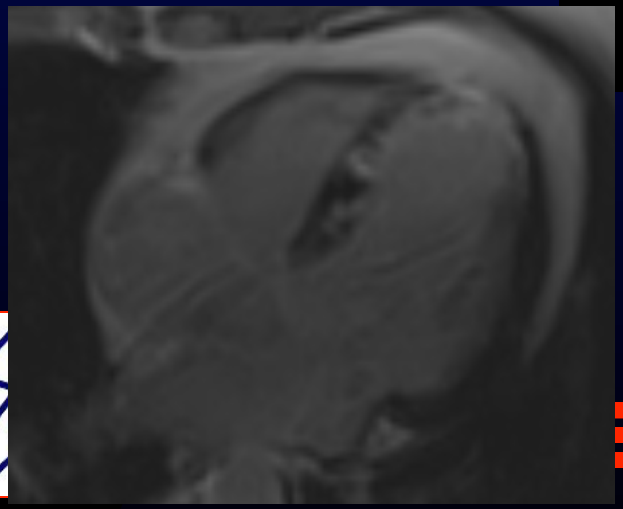



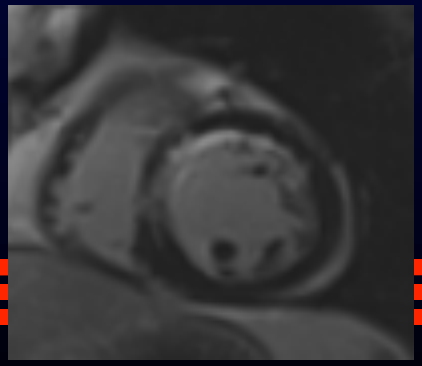



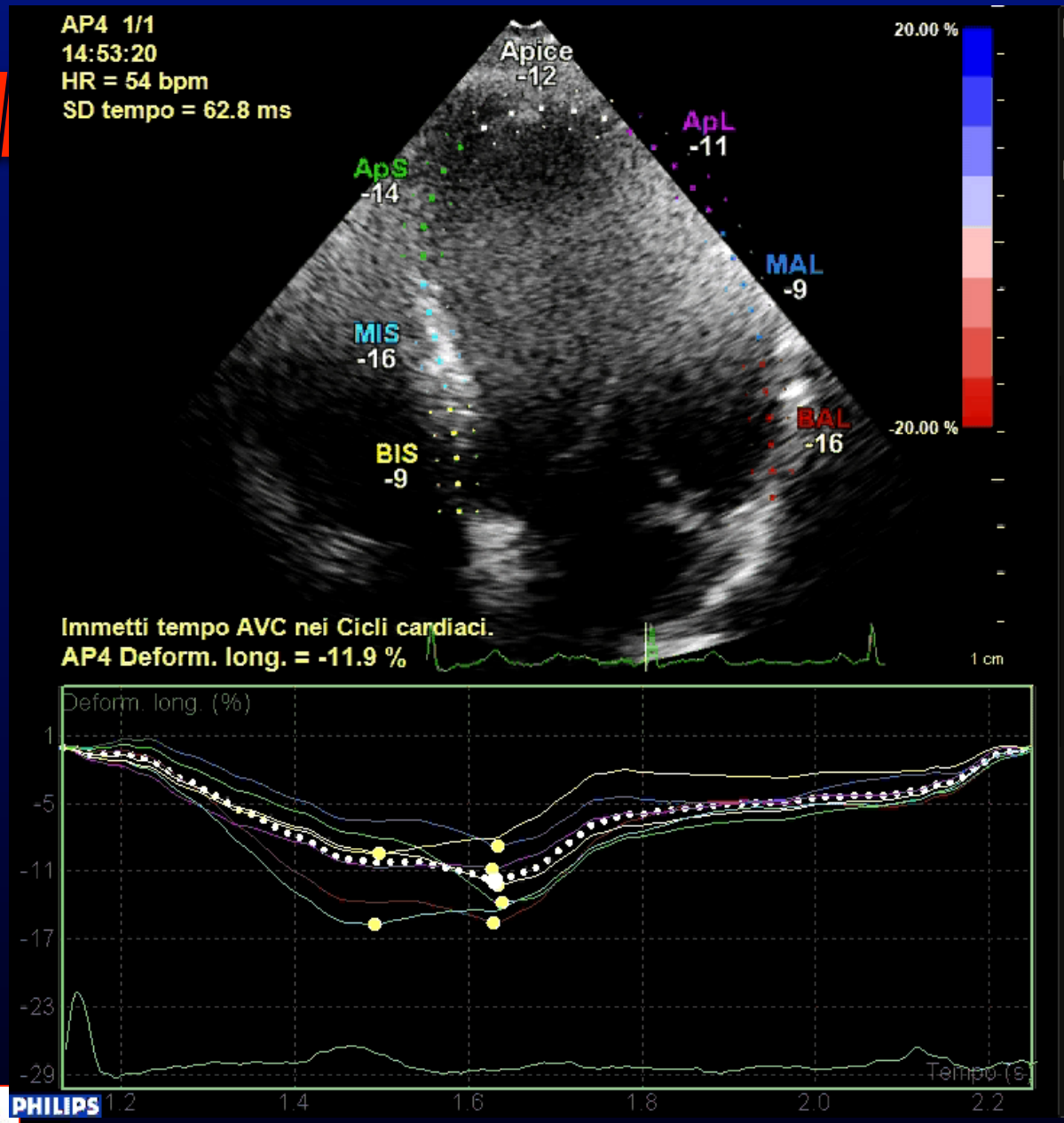



Delayed hypertenhancement

Delayed hypertenhancement








Misurazioni

| | |
|---|---|
| EDV | 176.5 ml |
| ESV | 99.0 ml |
| EF | 43.9 % |

Cicli cardiaci

| | |
|---|---|
| AV R-R | 1118 ms |
| MV R-R | 1118 ms |

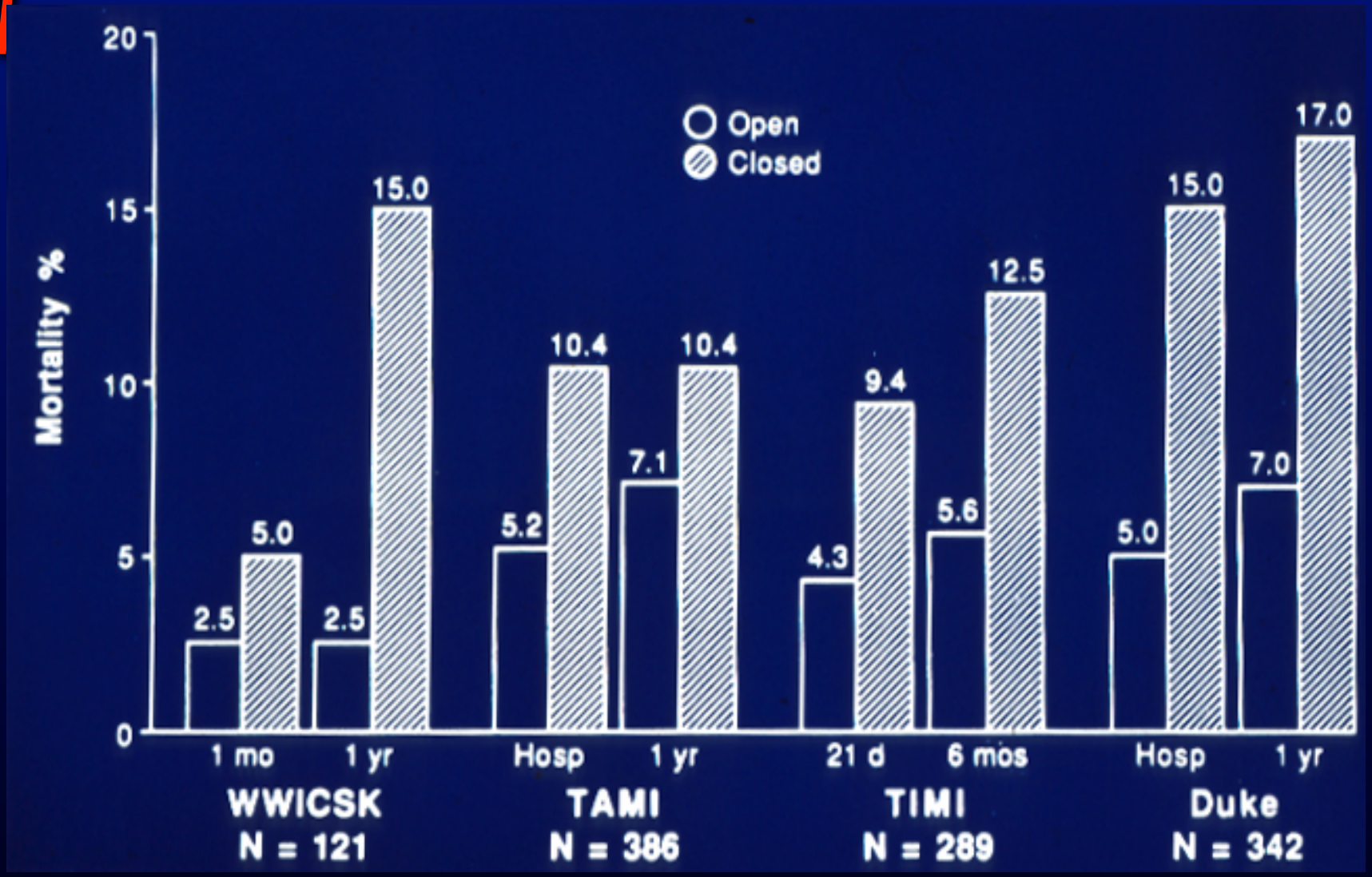
# *Variabili che condizionano entità e progressività del rimodellamento ventricolare*

- *Sede dell'infarto*
- *Dimensioni dell'infarto*
- *Modalità e tempi di cicatrizzazione dell'infarto*
- *Sollecitazioni meccaniche parietali*
  *(stress telediastolico e telesistolico)*

# Effect of Infarct Artery Patency on Prognosis After Acute Myocardial Infarction

Gervasio A. Lamas, MD; Greg C. Flaker, MD; Gary Mitchell, MD; Sidney C. Smith, Jr, MD; Bernard J. Gersh, MD, ChB, Dphil; Chuan Chuan Wun, PhD; Lemuel Moyè, MD, PhD; Jean L. Rouleau, MD; John D. Rutherford, MD; Marc A. Pfeffer, MD, PhD; Eugene Braunwald, MD; for the Survival and Ventricular Enlargement Investigators

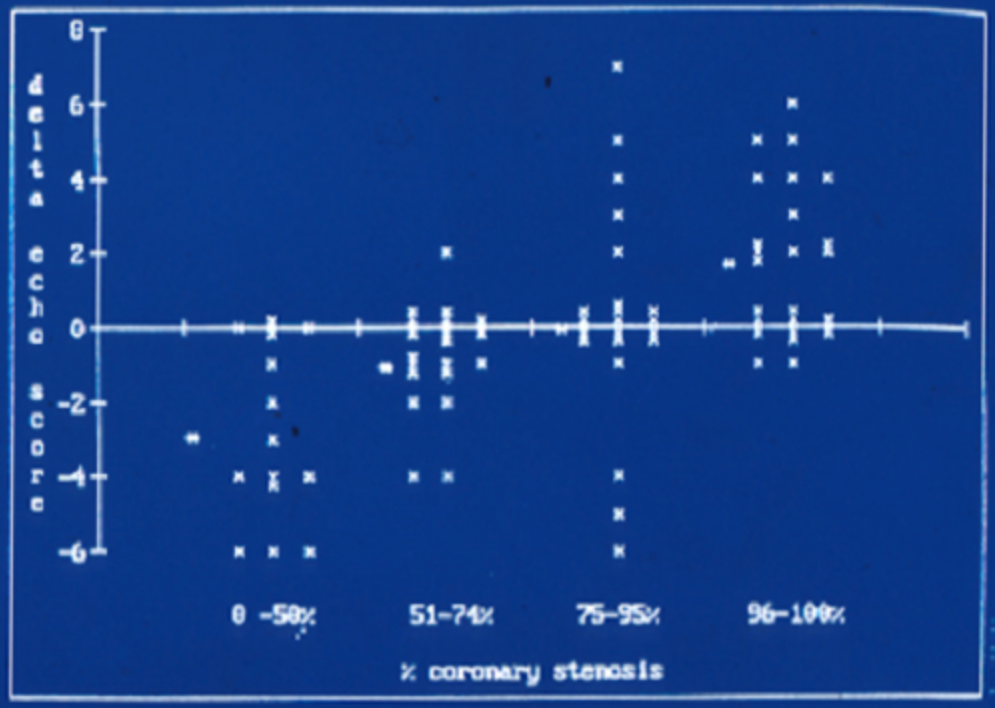
(Circulation, 1995;92:1101-1109)


Mortality %
20
15
10
5
0
Open
Closed
2.5
5.0
2.5
15.0
5.2
10.4
7.1
10.4
4.3
9.4
5.6
12.5
5.0
15.0
7.0
17.0
1 mo
1 yr
Hosp
1 yr
21 d
6 mos
Hosp
1 yr
WWICSK
N = 121
TAMI
N = 386
TIMI
N = 289
Duke
N = 342

# Influence of Reperfusion Induced by Thrombolytic Treatment on Natural History of Left Ventricular Regional Wall Motion Abnormality in Acute Myocardial Infarction

Maria Penco, MD, Silvio Romano, MD, Luciano Agati, MD, Alessandra Dagianti, MD, Antonio Vitarelli, MD, Francesco Fedele, MD, and Armando Dagianti, MD

(Am J Cardiol 1993;71:1015-1020)



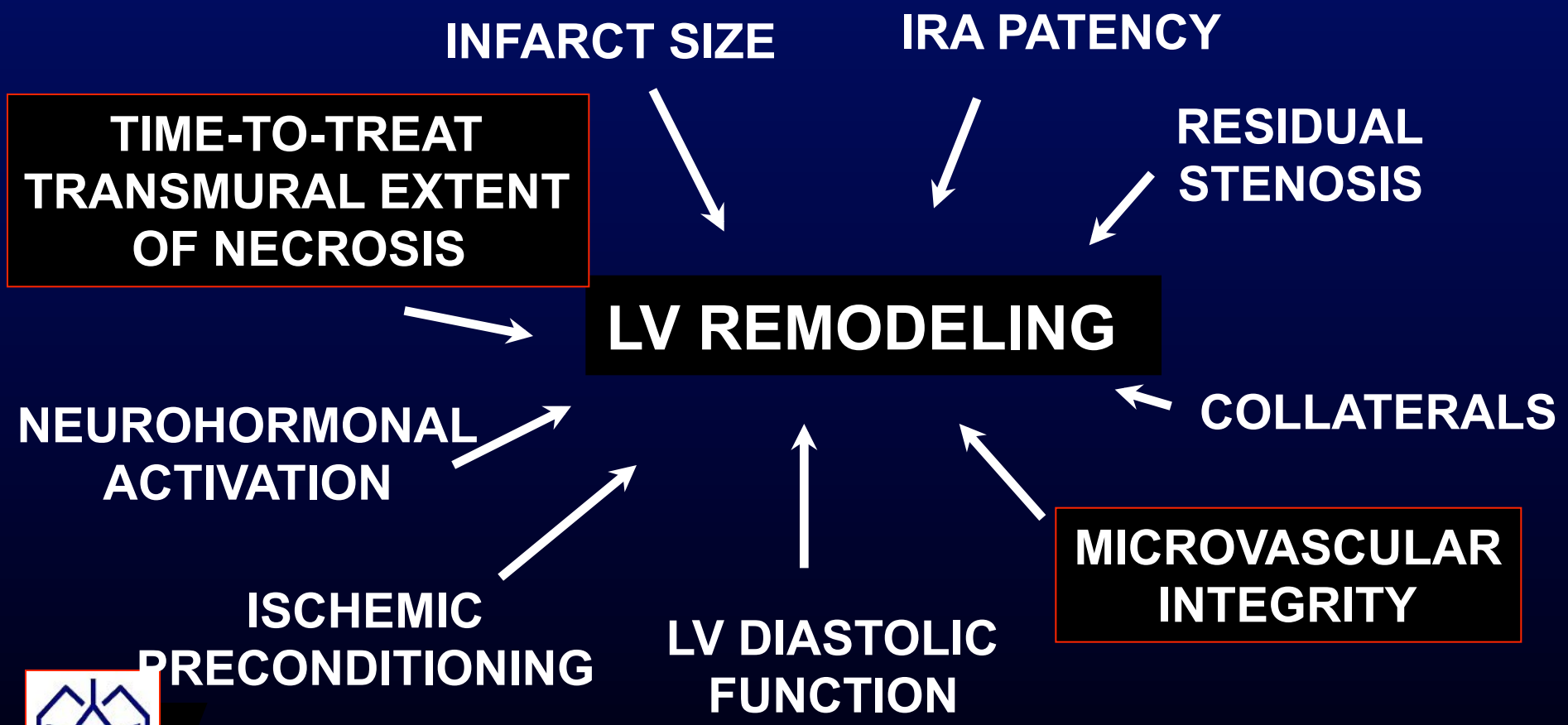
# Clinical Implications of the "No Reflow" Phenomenon

## A Predictor of Complications and Left Ventricular Remodelling in Reperfused Anterior Wall Myocardial Infarction

Hiroshi Ito, MD; Atsushi Maruyama, MD; Katsuomi Iwakura, MD; Shin Takiuchi, MD; Torhu Masuyama, MD; Masatsugu Hori, MD; Yorihiko Higashino, MD; Kenshi Fujii, MD Takazo Minamino, MD.

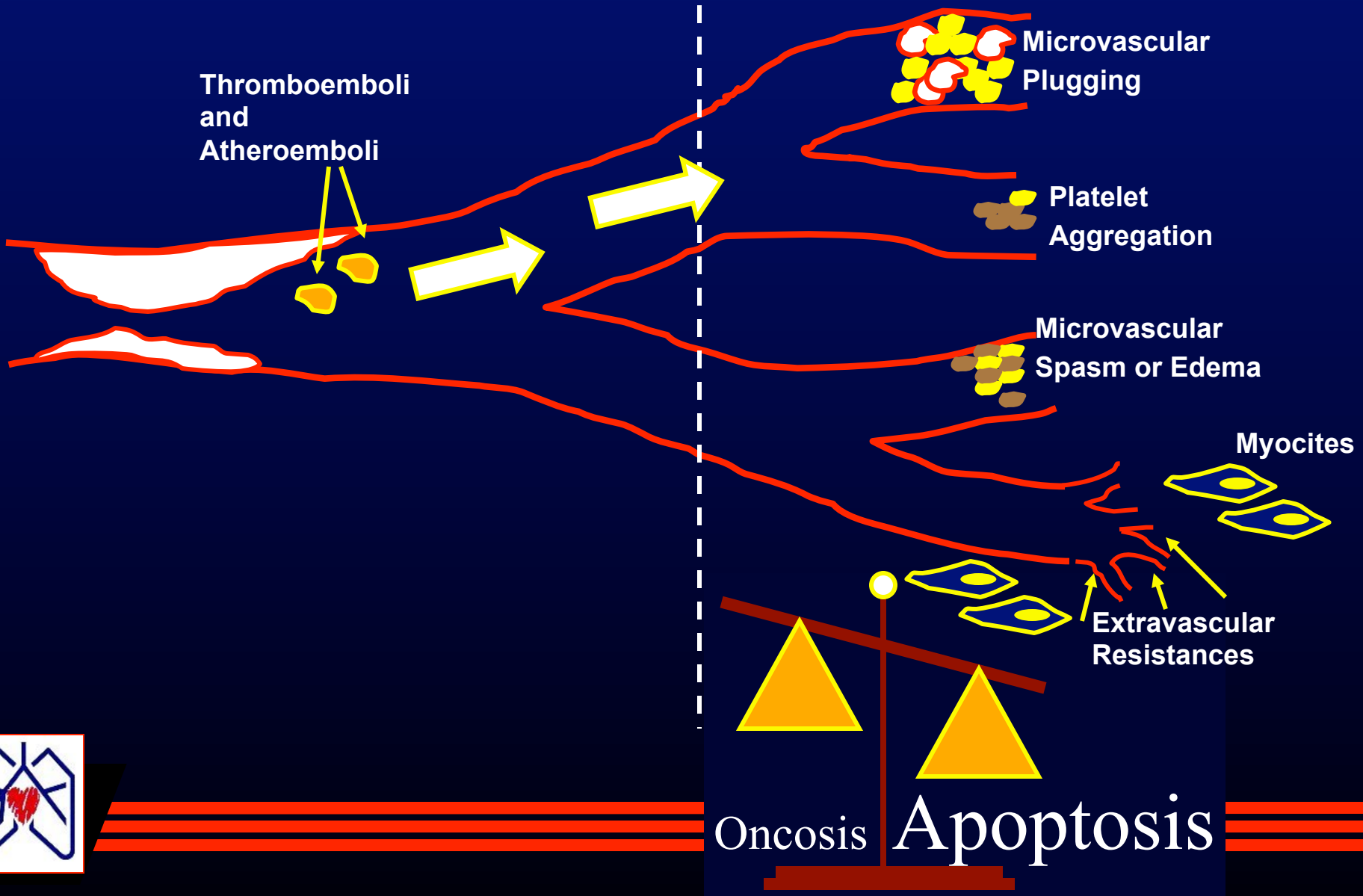
(*Circulation, 1996; 93: 223-228*)

# INTERACTIVE FACTORS THAT INFLUENCE LV REMODELING



# Epicardial Vessel

# Microvasculature



# Cardioprotection in AMI

**Proven Benefit in AMI**

Early reperfusion—attain and maintain coronary patency
Stents > PCI > thrombolysis
Agents to help maintain patency:
Aspirin
Low-molecular-weight heparin
Glycoprotein IIb/IIIa inhibitor with PCI
Intravenous beta-blocker (given early)

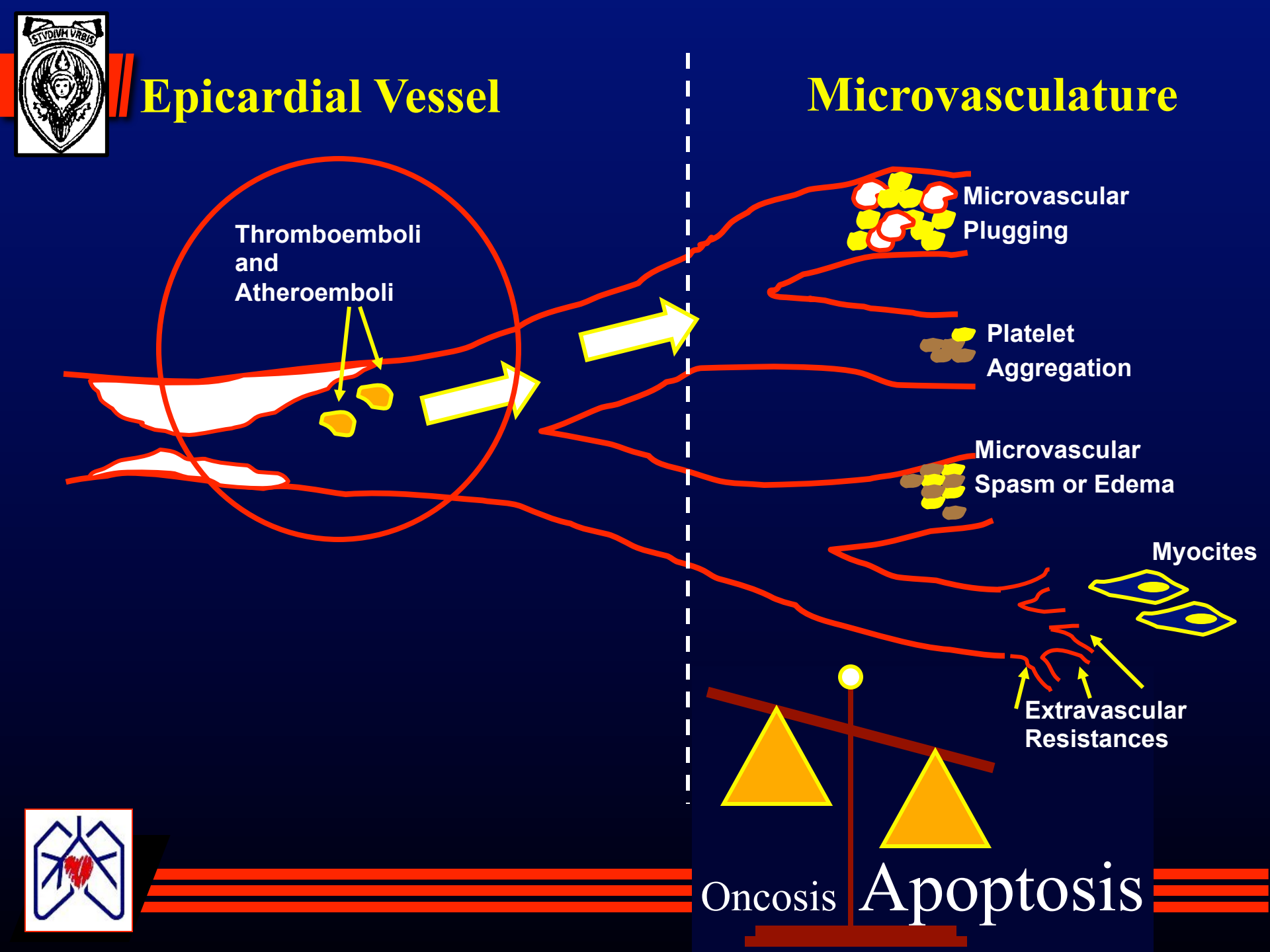
**Possible Benefit as Early Adjunctive Therapy**

Glucose-insulin-potasium or glucose-insulin
Adenosine (anterior wall myocardial infarctions)
Cariporide
Thrombectomy devices
Hypothermia (in anterior wall AMI with temperature 35°C)
Hyperoxemic solutions

**No Consistent Benefits as Early Adjunctive Therapy in Clinical Trials**

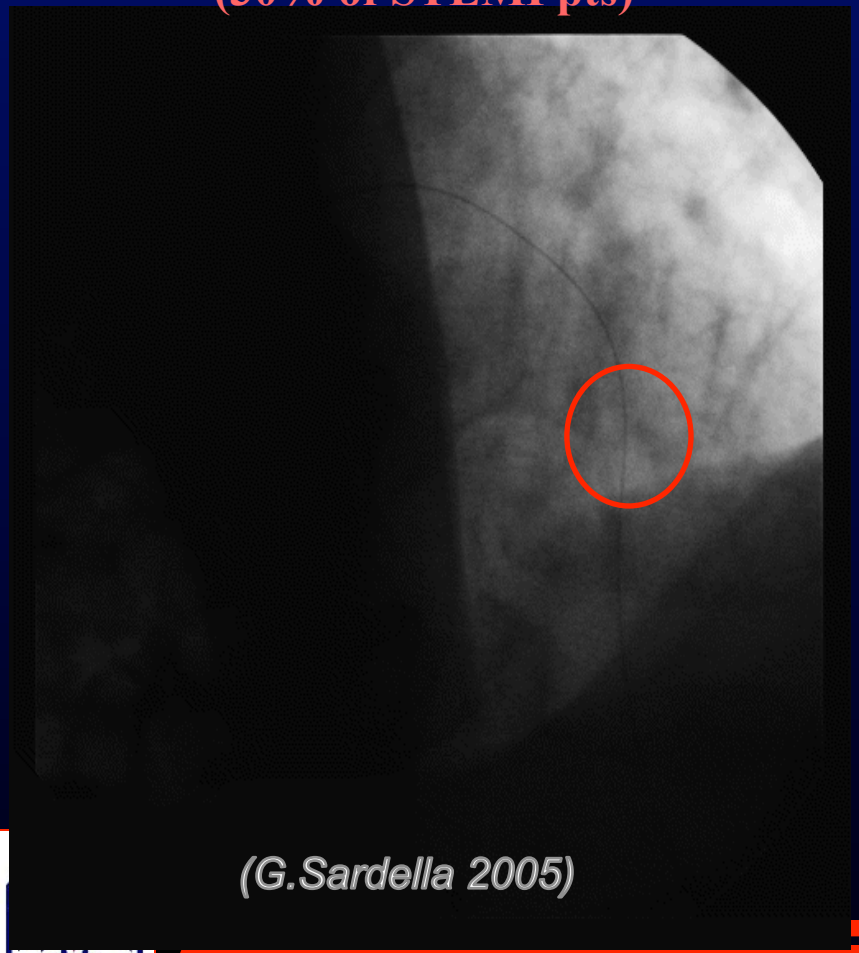
Inhibitors of neutrophil adhesion
Calcium channel blockers
hSOD (superoxide dismutase)
Reothrex
Trimetazidine (antioxidant)
Molsidomine (nitric oxide donor)
Fluosol
Hyaluronidase (pre-thrombolytic study)
Corticosteroids
Beta blockers (given late and without reperfusion)
Complement inhibition (no effect on infarct size)

Modified from Kloner and Rezkalla, *J Am Coll Cardiol* 2004

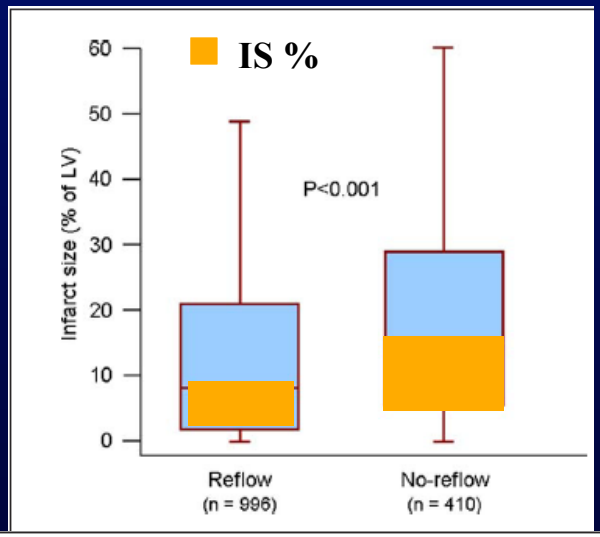

Epicardial Vessel
Microvasculature
Thromboemboli
and
Atheroemboli
Microvascular
Plugging
Platelet
Aggregation
Microvascular
Spasm or Edema
Myocites
Extravascular
Resistances
Oncosis
Apoptosis

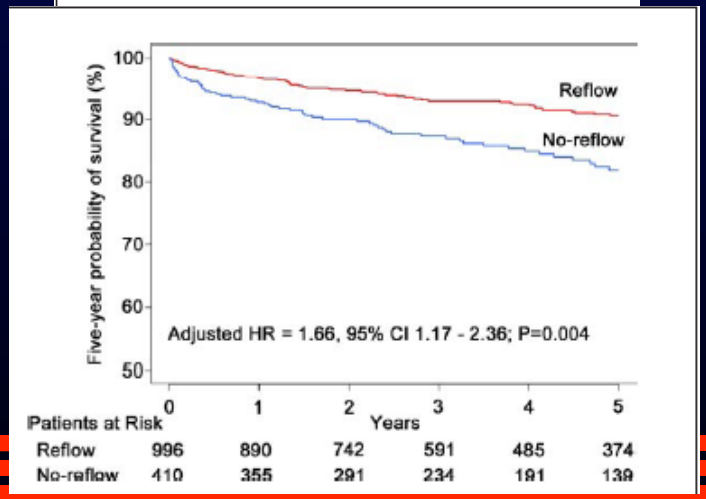
# Distal Embolization as "NO-FLOW" determinant

- In STEMI the " no-flow" phenomenon is caused by the distal embolization after the IRA reopening (30% of STEMI pts)




- Impact of "NR" on IS and Mortality




( G. Ndrepepa JACC 2010;55;2383-2389 )

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ISSN 0735-1097/09/$36.00
doi:10.1016/j.jacc.2008.10.017



# Thrombus Aspiration During Primary Percutaneous Coronary Intervention Improves Myocardial Reperfusion and Reduces Infarct Size

## The EXPIRA (Thrombectomy With Export Catheter in Infarct-Related Artery During Primary Percutaneous Coronary Intervention) Prospective, Randomized Trial

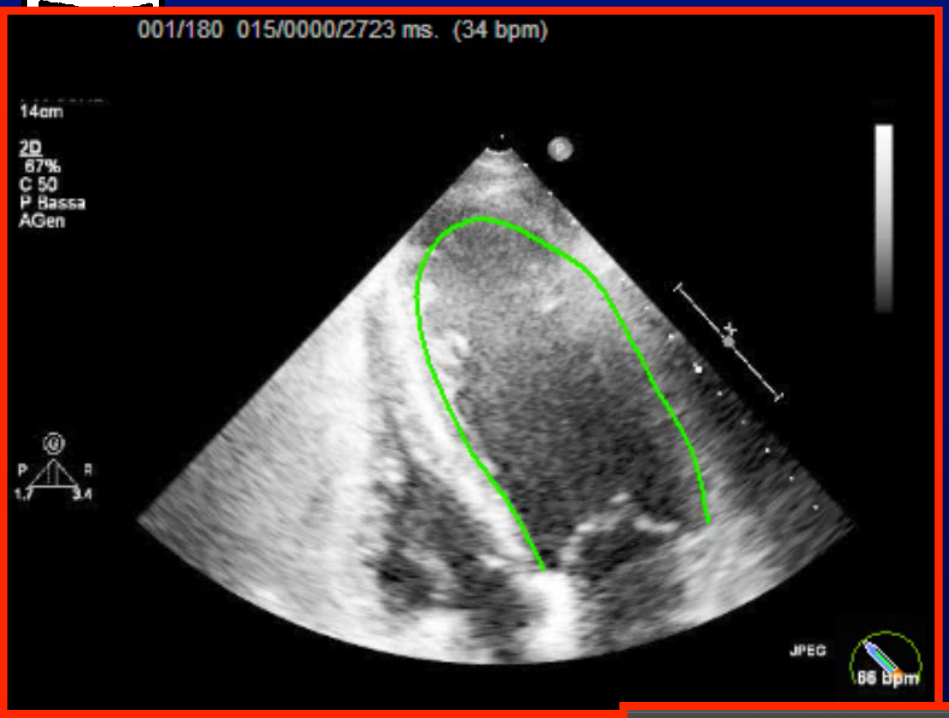
Gennaro Sardella, MD, FACC, FESC,* Massimo Mancone, MD,* Chiara Bucciarelli-Ducci, MD,*‡ Luciano Agati, MD,* Raffaele Scardala, MD,* Iacopo Carbone, MD,† Marco Francone, MD,† Angelo Di Roma, MD,* Giulia Benedetti, MD,* Giulia Conti, MD,* Francesco Fedele, MD*

*Rome, Italy; and London, United Kingdom*

# Microvascular Flow Assessment in ACS: Methods

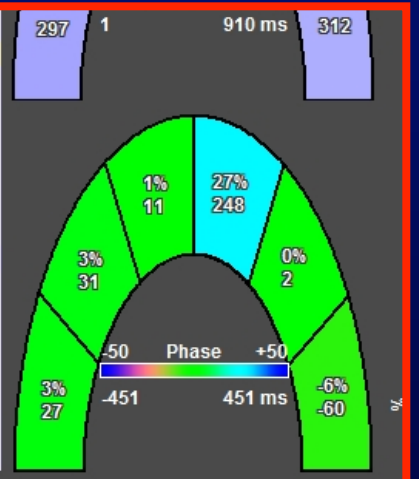
ST Segment Changes
TIMI grade
Blush grade
SPECT
MCE
MRI
2D strain

# EXPIRA trial
# Echo sub-study

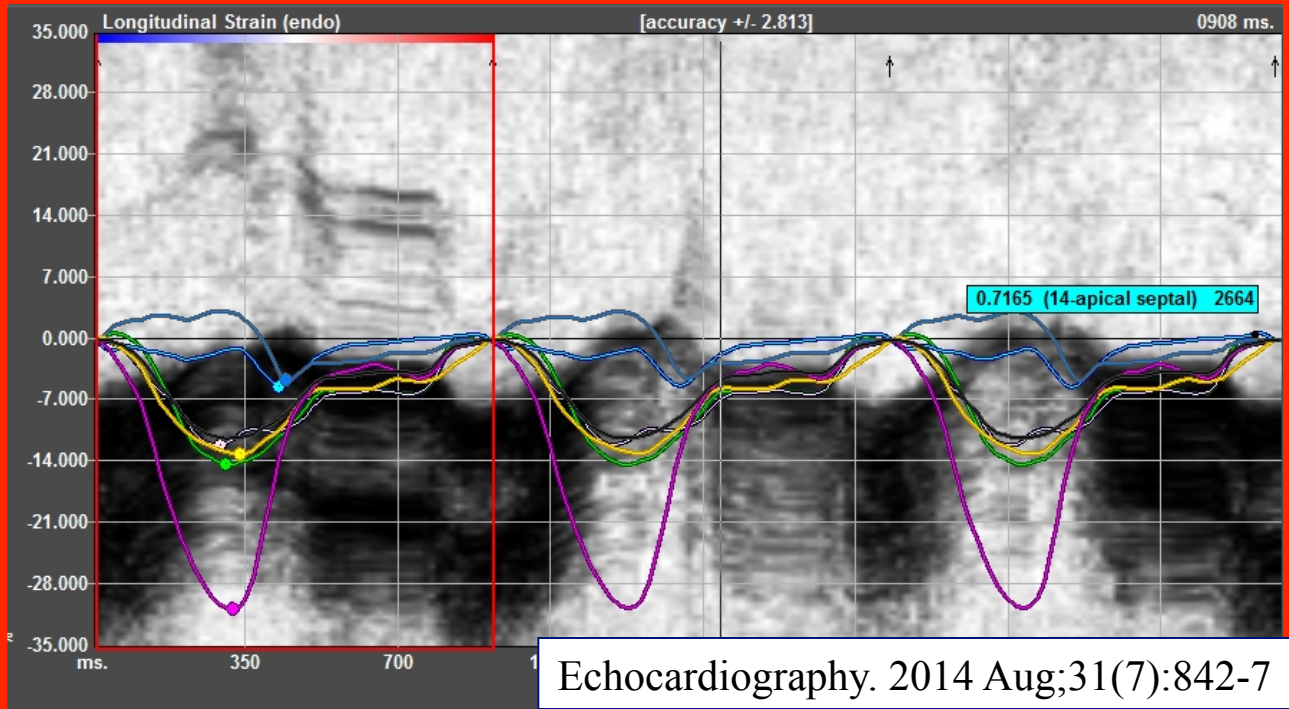



| Longitudinal Strain (endo) | | |
|---|---|---|
| Seg. | Pk % | TPk ms |
| 03-basal septal | -14.4181 | 297 |
| 09-mid septal | -12.1745 | 282 |
| 14-apical septal | -5.3933 | 418 |
| 16-apical lateral | -4.5801 | 433 |
| 12-mid lateral | -13.1805 | 327 |
| 06-basal lateral | -31.1076 | 312 |
| Average | -13.4757 | 345 |

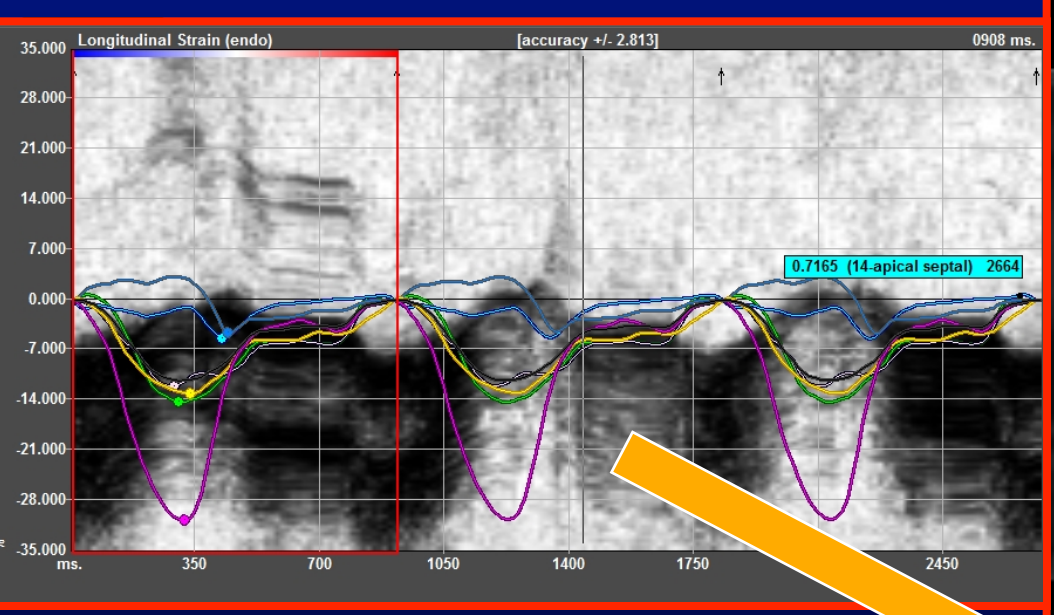
Maximum Opposing Wall Delay: 151

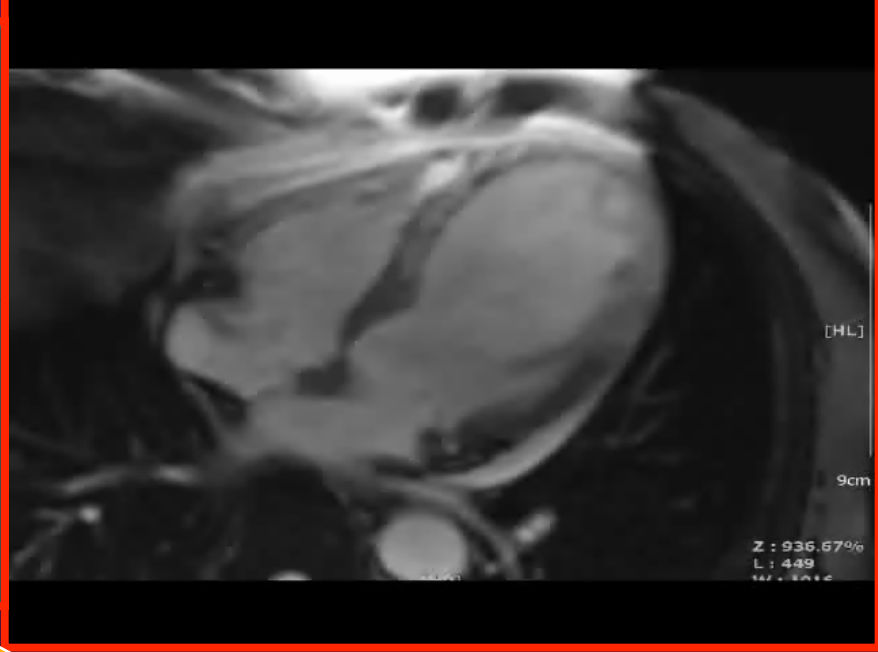



Apical strain impairment




Echocardiography. 2014 Aug;31(7):842-7

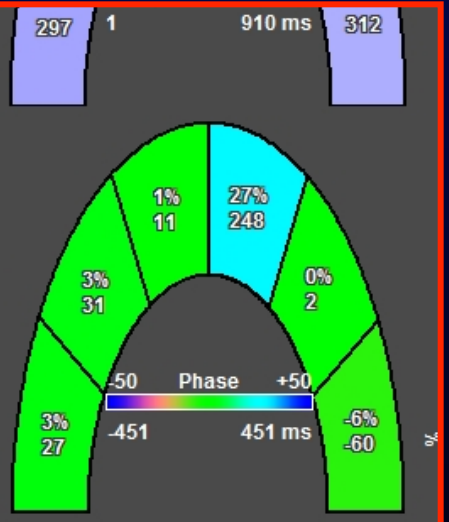


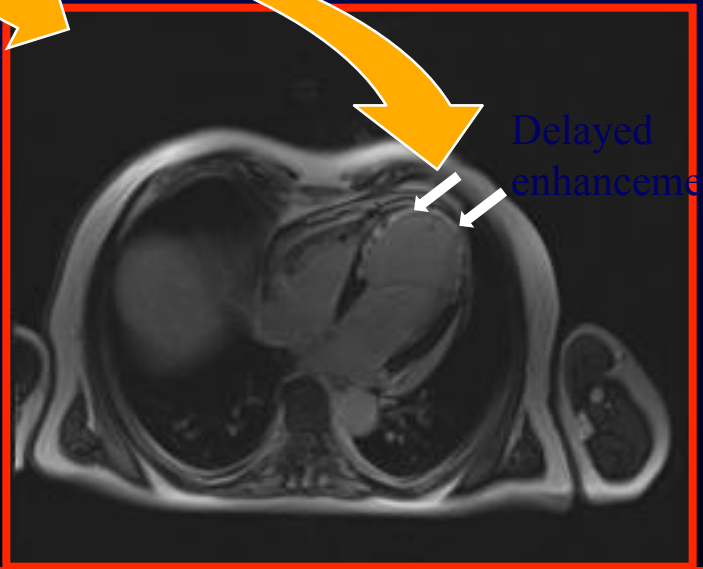




# 2D strain correlation with MRI

| Longitudinal Strain (endo) | | |
|---|---|---|
| Seg. | Pk % | TPk ms |
| 03-basal septal | -14.4181 | 297 |
| 09-mid septal | -12.174 | 282 |
| 14-apical septal | -5.3933 | 418 |
| 16-apical lateral | -4.5801 | 433 |
| 12-mid lateral | -13.1805 | 327 |
| 06-basal lateral | -31.1076 | 312 |
| Average | -13.4757 | 345 |

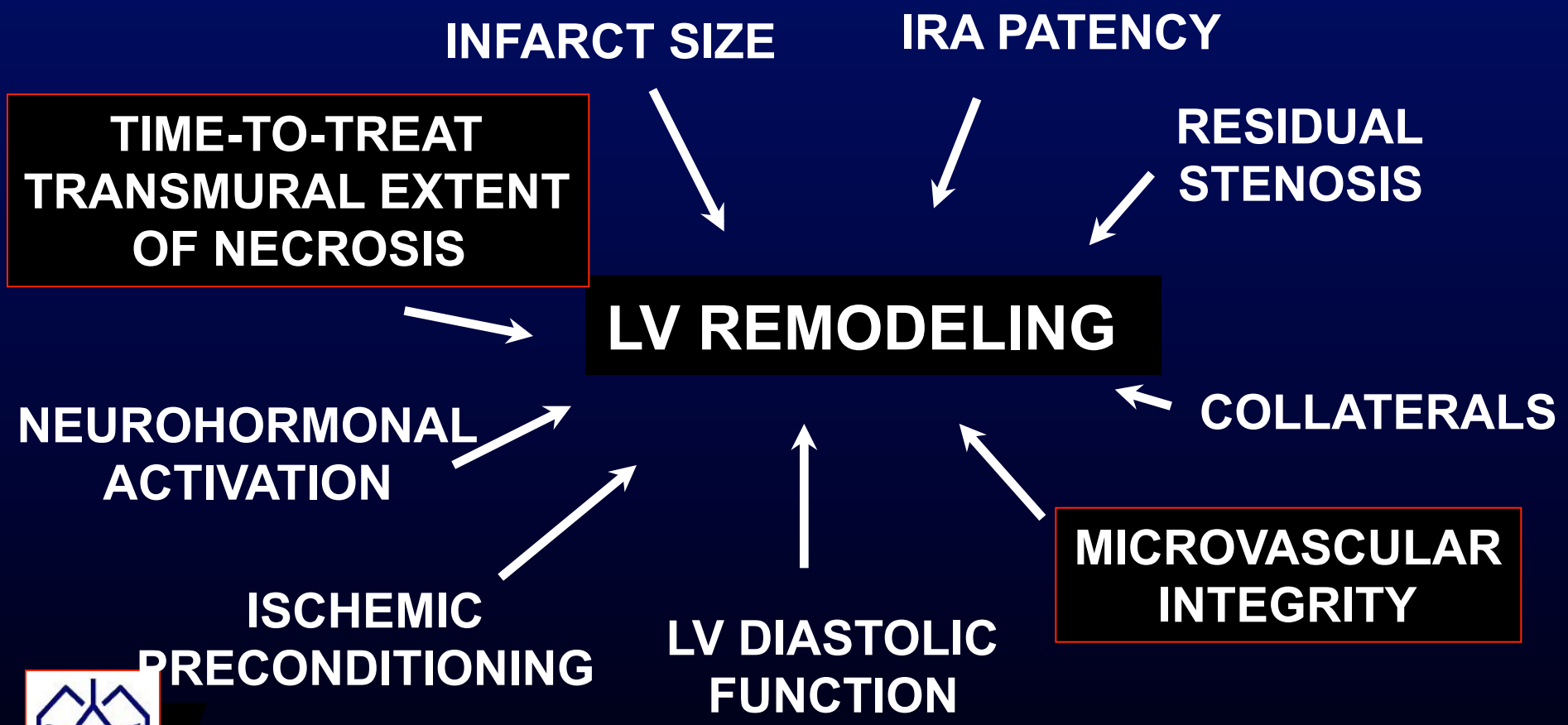
Maximum Opposing Wall Delay: 151




Echocardiography. 2014 Aug;31(7):842-7

# INTERACTIVE FACTORS THAT INFLUENCE LV REMODELING



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ISSN 0735-1097/08/$34.00
doi:10.1016/j.jacc.2007.09.051

# The Extent of Microvascular Damage During Myocardial Contrast Echocardiography Is Superior to Other Known Indexes of Post-Infarct Reperfusion in Predicting Left Ventricular Remodeling

## Results of the Multicenter AMICI Study

Leonarda Galiuto, MD, PHD, FACC,* Barbara Garramone, MD,* Antonio Scarà, MD,* Antonio G. Rebuzzi, MD,* Filippo Crea, MD, FACC,* Giuseppe La Torre, MD, MSC,† Stefania Funaro, MD,‡ Mariapina Madonna, MD,§ Francesco Fedele, MD,§ Luciano Agati, MD,§ on behalf of the AMICI Investigators

*Rome and Campobasso, Italy*

**Table 2** Univariate and Multivariate Analysis of the Day 1 Predictors of Left Ventricular Remodeling at Follow-Up

| Variable | Odds Ratio* (95% CI) | p Value | Odds Ratio† (95% CI) | p Value |
|---|---|---|---|---|
| Age >65 yrs | 2.44 (0.97–6.19) | 0.059 | | |
| Male gender | 1.18 (0.35–3.96) | 0.784 | | |
| Hypertension | 0.58 (0.23–1.41) | 0.231 | | |
| Diabetes | 2.06 (0.66–6.32) | 0.208 | | |
| Dyslipidemia | **2.8 (1.14–6.85)** | **0.024** | | |
| Smoker | 0.58 (0.23–1.35) | 0.195 | | |
| Positive family history of CAD | 0.55 (0.19–1.62) | 0.277 | | |
| Time to treat | 0.94 (0.84–1.04) | 0.245 | | |
| ST-segment reduction cutoff <70% | 1.15 (0.40–3.25) | 0.79 | | |
| CK peak | 0.99 (0.37–2.71) | 0.996 | | |
| Initial TIMI score = 0 | 1.06 (0.74–1.52) | 0.743 | | |
| Initial MBG = 0 | 0.55 (0.25–1.21) | 0.138 | | |
| Final TIMI score <3 | **5.84 (2.04–16)** | **0.001** | 5.6 (1.40–22) | 0.015 |
| Final MBG <3 | 1.14 (0.36–3.67) | 0.819 | | |
| Multivessel disease | **1.75 (1.02–2.99)** | **0.042** | | |
| WMA cutoff >44% | **5.78 (2.09–15.8)** | **0.001** | | |
| WMSI cutoff >1.9 | **5.82 (2.19–15.4)** | **<0.0001** | | |
| CD cutoff >25% | **6.48 (2.48–16)** | **<0.0001** | 7 (1.86–27) | 0.04 |
| CSI cutoff >1.8 | **8.85 (3.26–24)** | **<0.0001** | | |
| EF cutoff <44.5% | **4.29 (1.71–10)** | **0.002** | | |
| ESV cutoff <54.4 ml | 1.08 (0.45–2.58) | 0.864 | | |
| EDV cutoff <101 ml | 1.95 (0.81–4.70) | 0.136 | | |
| Hosmer-Lemeshow test | | | Chi-square = ( | |

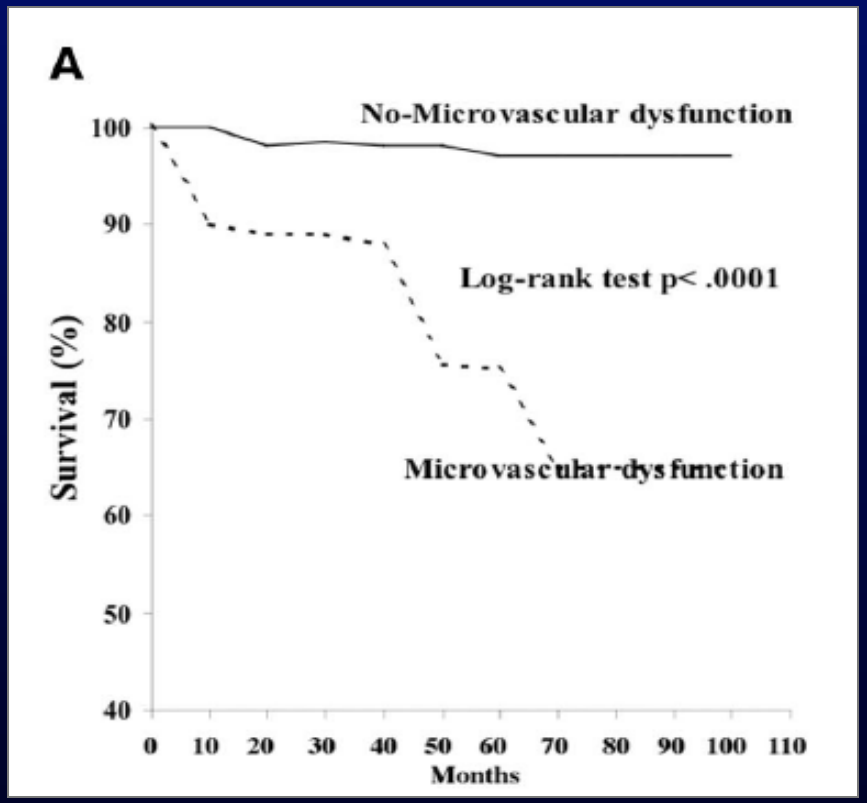
JACC Vol. 51, No. 5, 2008
February 5, 2008:552–9

**Table 3** Univariate and Multivariate Analysis of the Day 1 Predictors of Left Ventricular Remodeling at Follow-Up in Patients With Grade 3 TIMI Flow

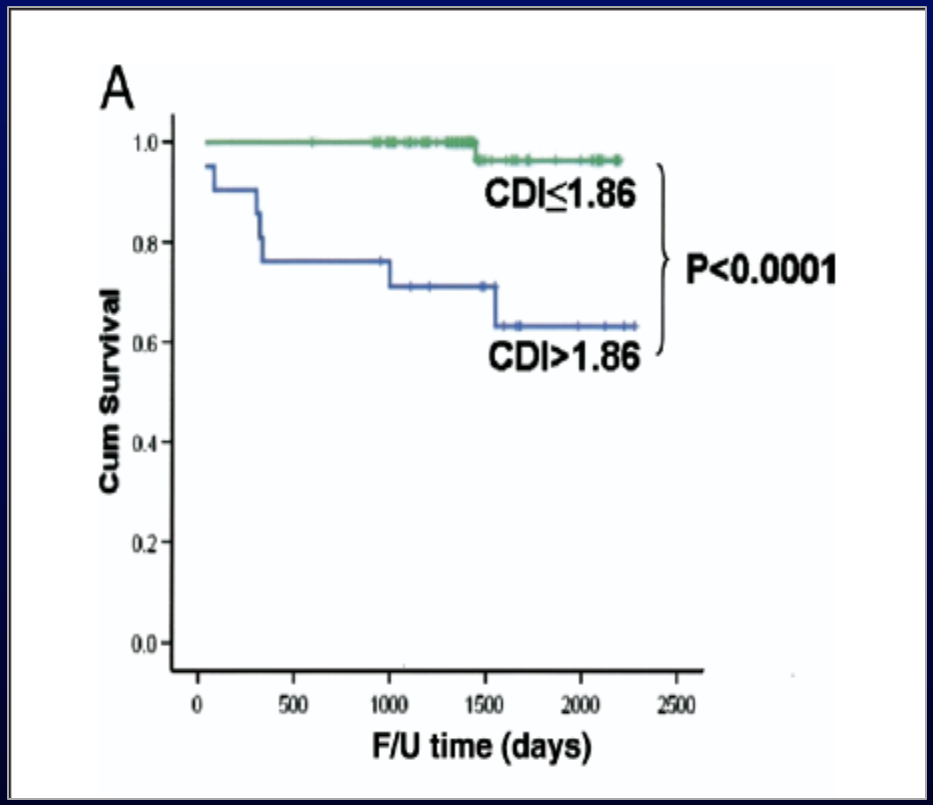
| Variable | Odds Ratio* (95% CI) | p Value | Odds Ratio† (95% CI) | p Value |
|---|---|---|---|---|
| Age >65 yrs | 1.23 (0.55–4.12) | 0.121 | | |
| Male gender | 1.18 (0.35–3.96) | 0.784 | | |
| Hypertension | **0.25 (0.74–0.821)** | **0.024** | | |
| Diabetes | 2.62 (0.67–10.16) | 0.163 | | |
| Dyslipidemia | **4.32 (1.28–14.50)** | **0.018** | | |
| Smoker | 0.59 (0.184–1.93) | 0.388 | | |
| Positive family history of CAD | 0.40 (0.08–1.95) | 0.257 | | |
| Time to treat | 0.98 (0.86–1.10) | 0.757 | | |
| ST-segment reduction cutoff <70% | 1.00 (0.98–1.02) | 0.648 | | |
| CK peak | 1 (1–1) | 0.821 | | |
| Initial TIMI score 0 | 1.26 (0.81–1.26) | 0.303 | | |
| Initial MBG 0 | 0.97 (0.41–2.30) | 0.955 | | |
| Final MBG <3 | 1.16 (0.46–2.82) | 0.738 | | |
| Multivessel disease | **2.46 (1.16–5.21)** | **0.019** | | |
| WMA cutoff >44% | **1.03 (1.00–1.07)** | **0.029** | | |
| WMSI cutoff >1.9 | **7.4 (1.32–41)** | **0.02** | | |
| CD cutoff >25% | **8.75 (3.22–46)** | **0.0001** | 12.7 (2.65–61.2) | |
| CSI cutoff >1.8 | **1.05 (1.01–1.09)** | **0.005** | | |
| EF cutoff <44.5% | **0.90 (0.83–0.97)** | **0.013** | | |
| ESV cutoff <54.4 ml | 0.98 (0.95–1.01) | 0.40 | | |
| EDV cutoff <101 ml | 0.96 (0.93–0.99) | 0.015 | | |
| Hosmer-Lemeshow test | | | Chi-square = 0.37 | 0.840 |

JACC Vol. 51, No. 5, 2008
February 5, 2008:552–9

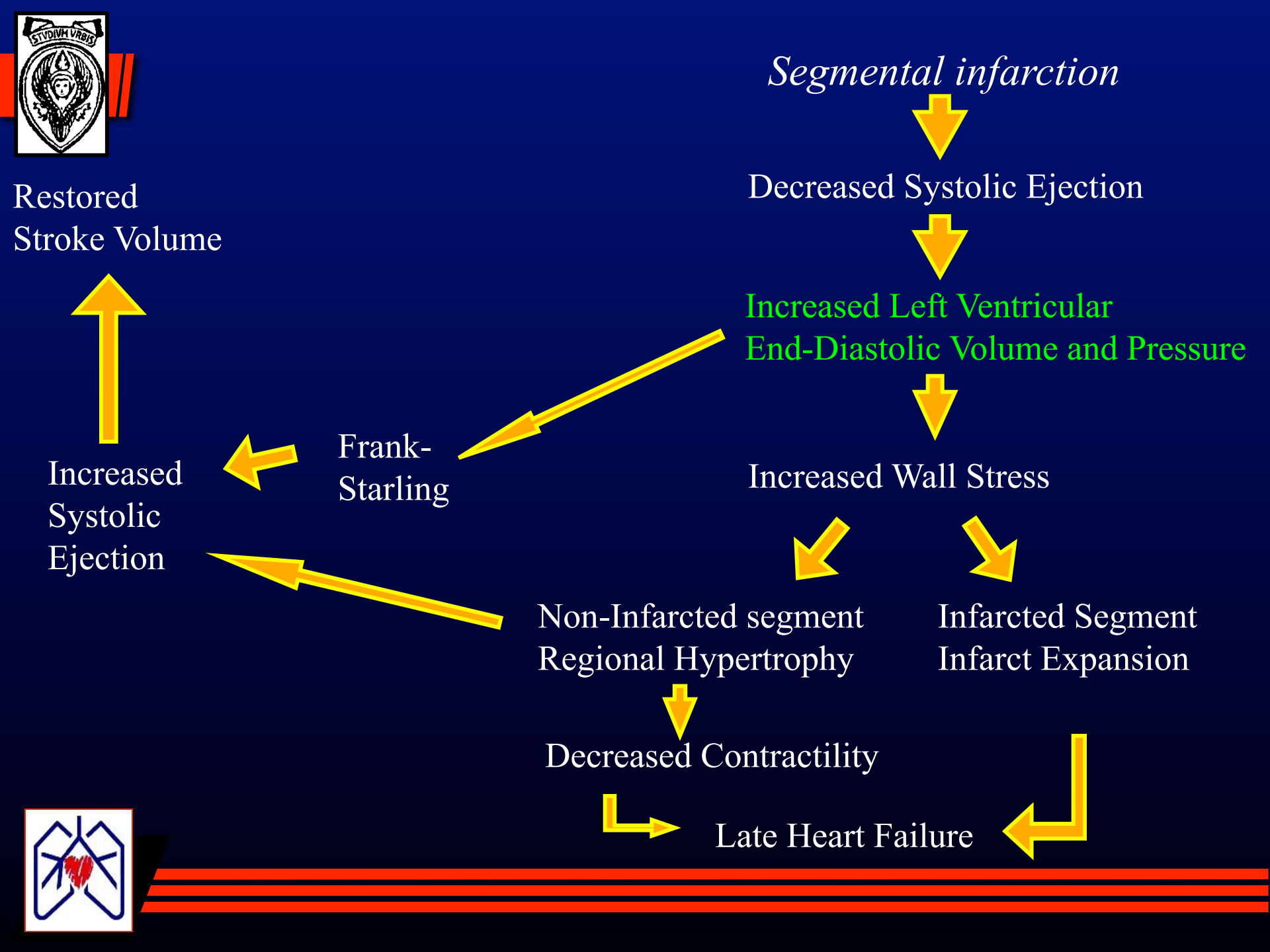
# Long Term Follow-up




Bolognese L et al. Circulation 2004;109:1121



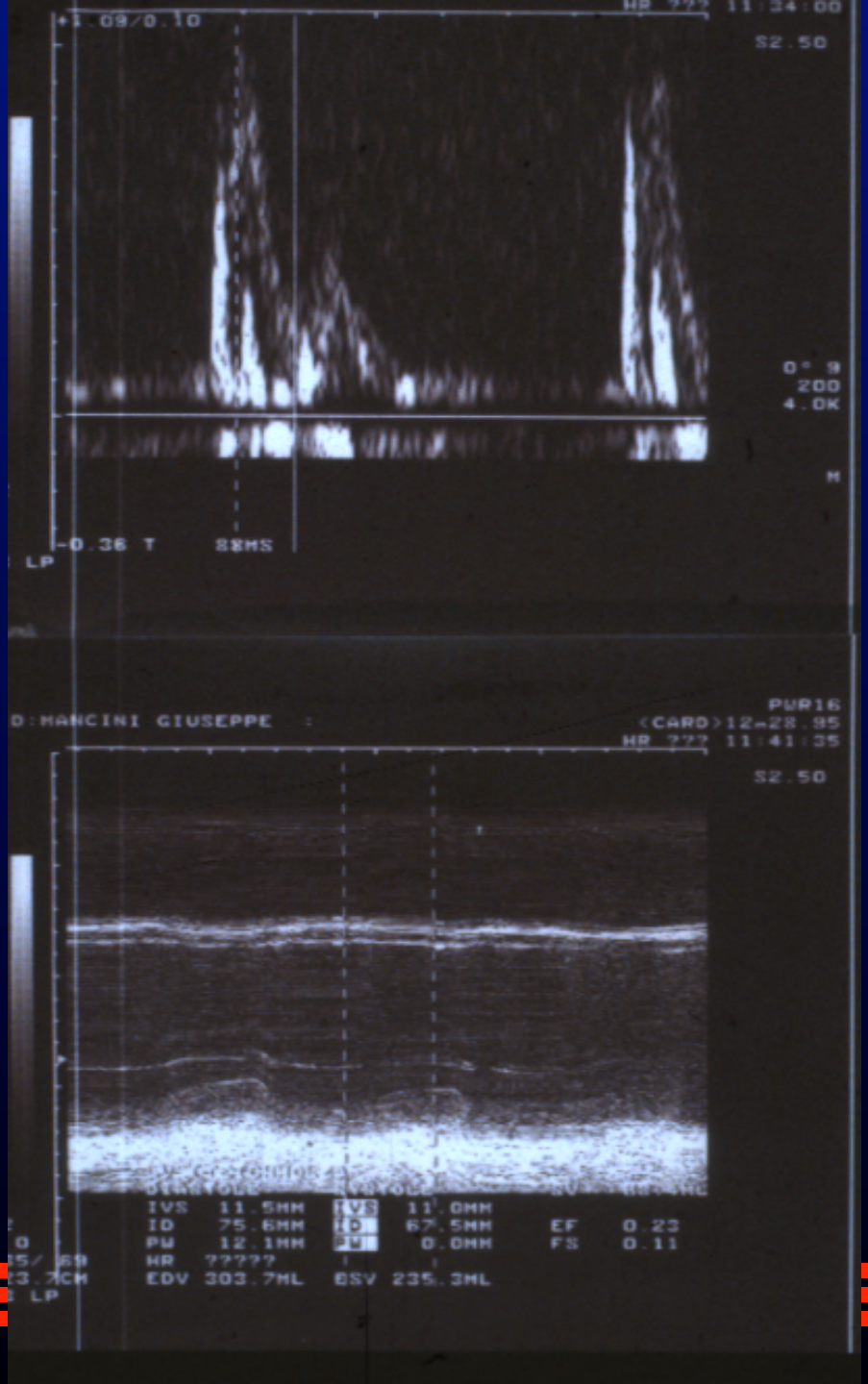

Dwidedi G, Senior R et al JACC 2007;24:327

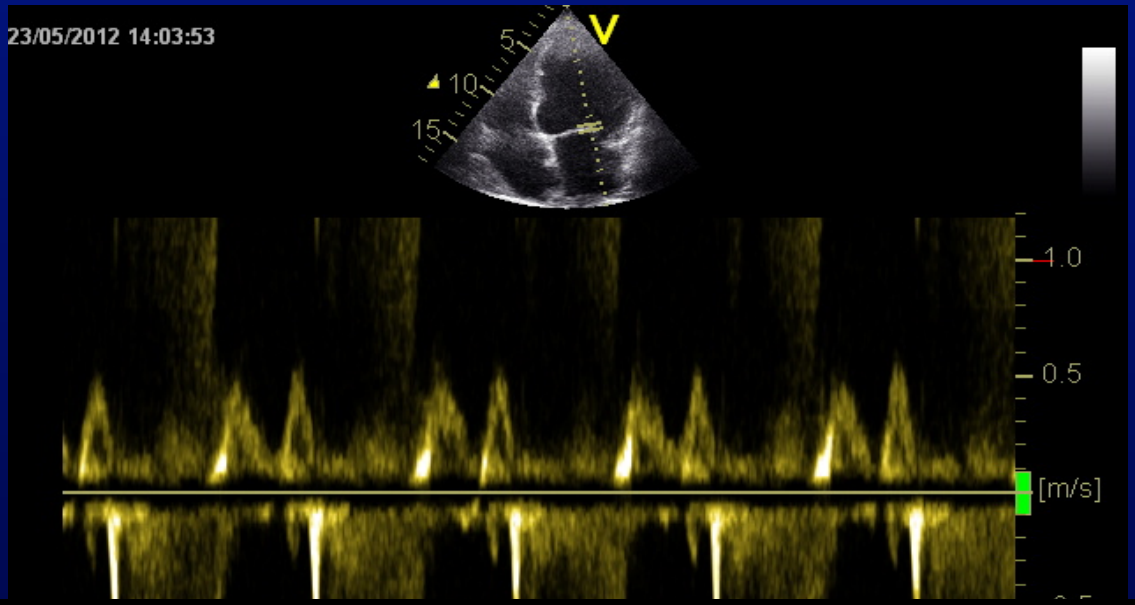

Segmental infarction
Decreased Systolic Ejection
Increased Left Ventricular
End-Diastolic Volume and Pressure
Increased Wall Stress
Infarcted Segment
Infarct Expansion
Non-Infarcted segment
Regional Hypertrophy
Decreased Contractility
Late Heart Failure
Frank-
Starling
Increased
Systolic
Ejection
Restored
Stroke Volume

# Early and Late Changes in Left Ventricular Filling After Acute Myocardial Infarction and The Effect of Infarct Size

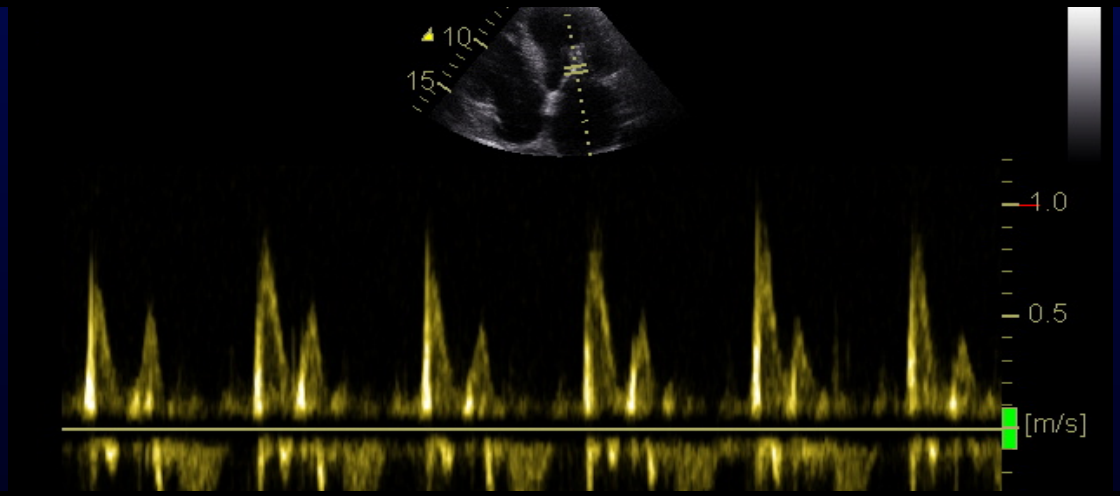
Athanase Pipilis, MD; Theo E. Meyer, FCP(SA), Dphil, Oliver Ormerod, DM, MRCP; Marcus Flather, MRCP and Peter Sleight, MD

*(Am J Cardiol 1992; 70:1397-1401)*

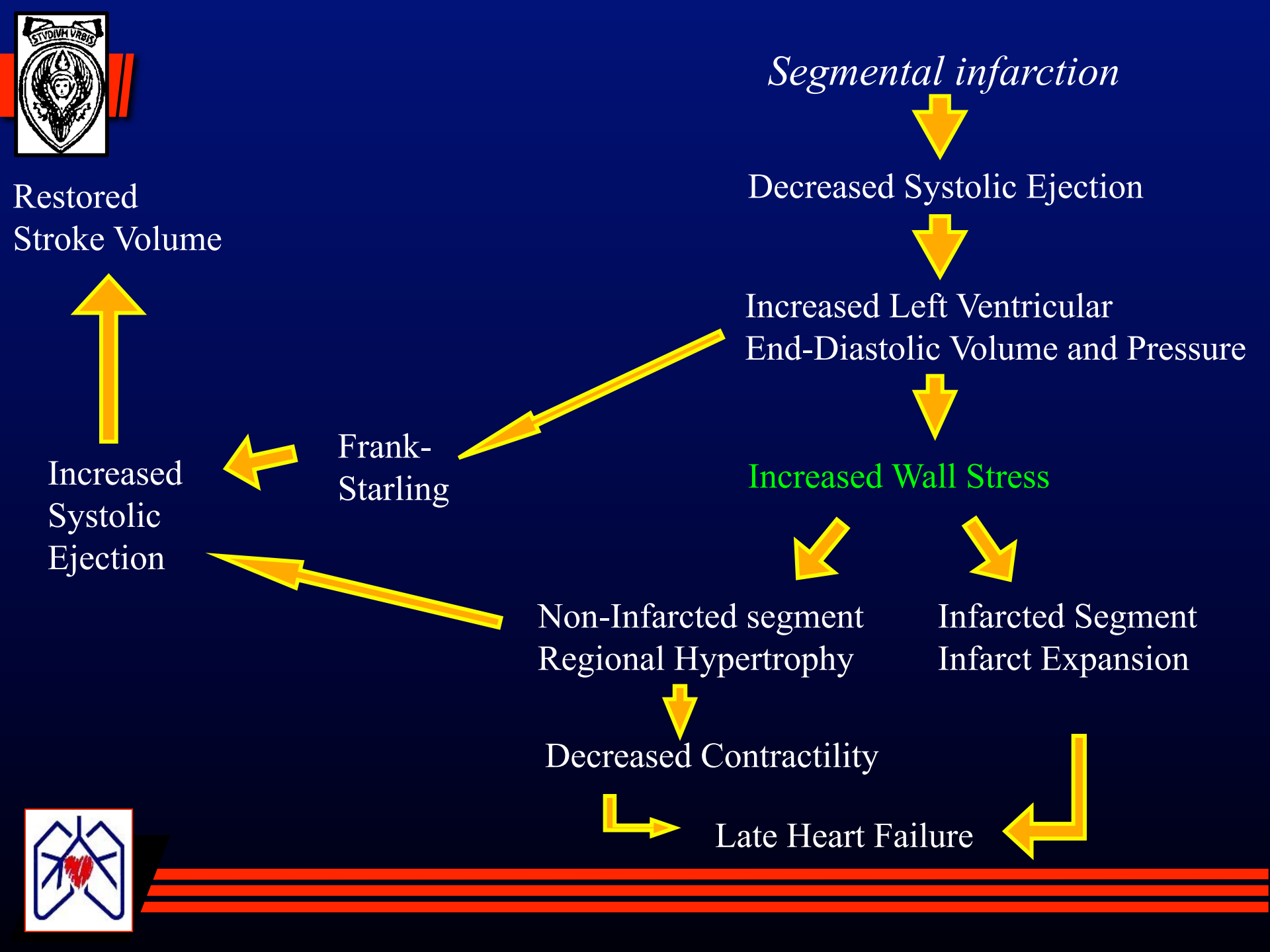

+1.09/0.10
S2.50
0° 9
200
4.0K
M
-0.36 T 88MS
LP
D:MANCINI GIUSEPPE
PWR16
(CARD)12-28.95
HR 222 11:41:35
S2.50
IVS 11.5MM
ID 75.6MM
PW 12.1MM
HR ?????
EDV 303.7ML
IVS 11.0MM
ID 67.5MM
PW 0.0MM
ESV 235.3ML
EF 0.23
FS 0.11
LP



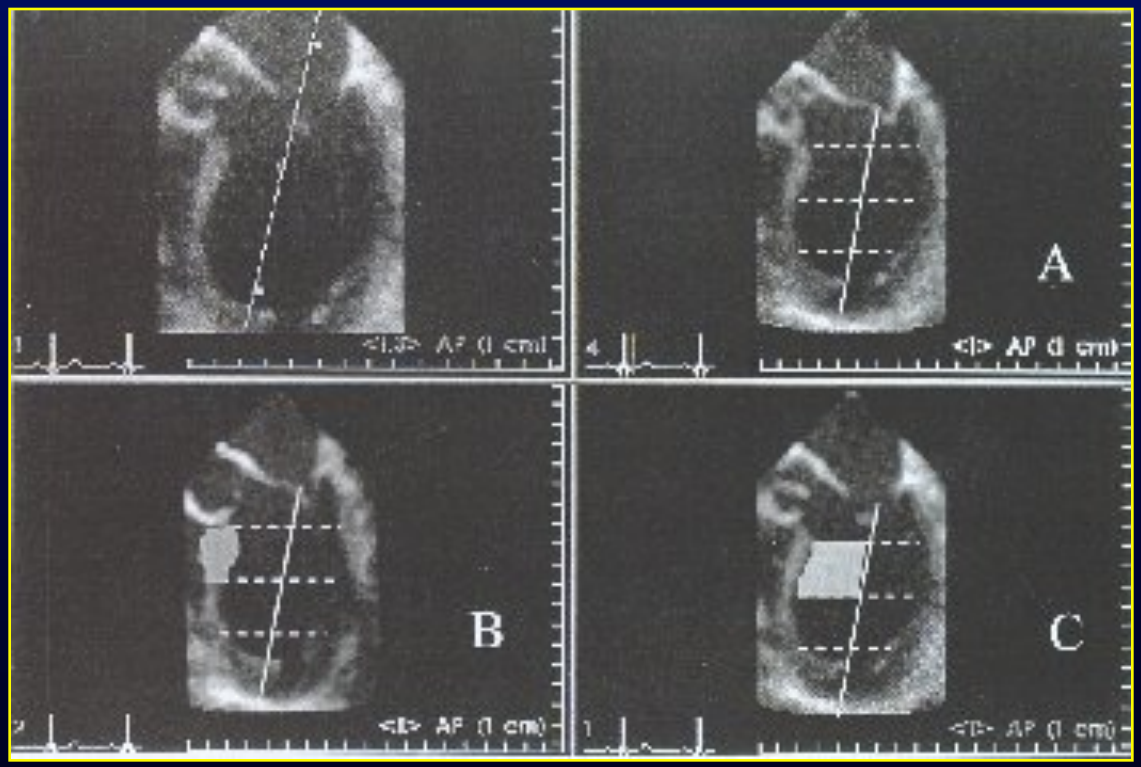

Early mild impaired relaxation



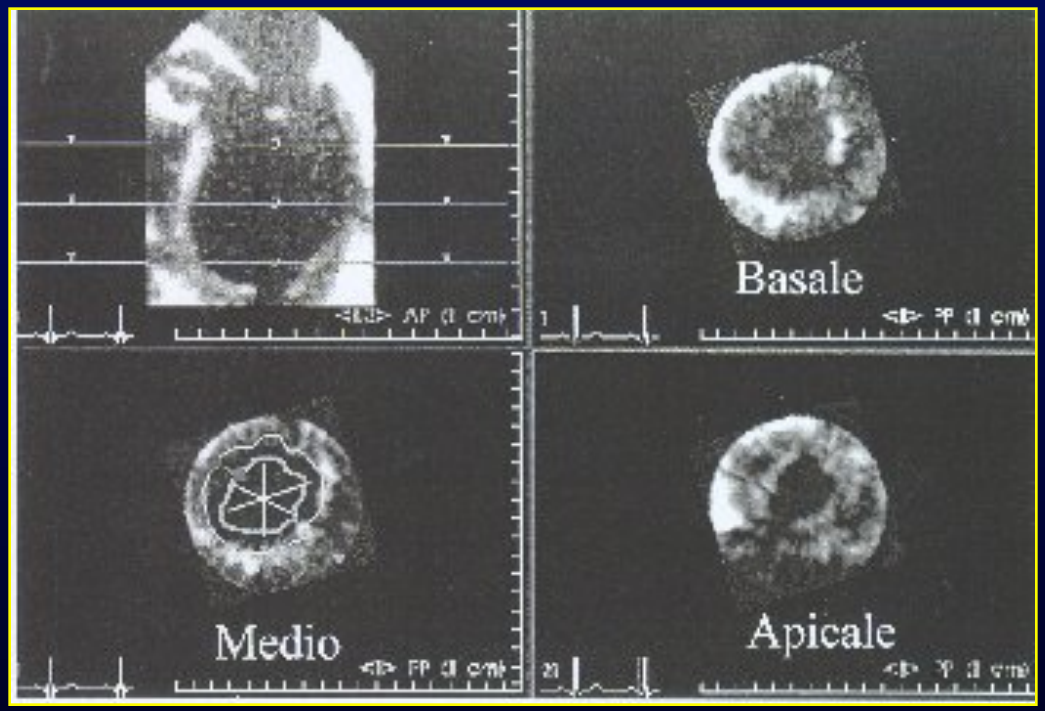

Restrictive physiology, altered compliance

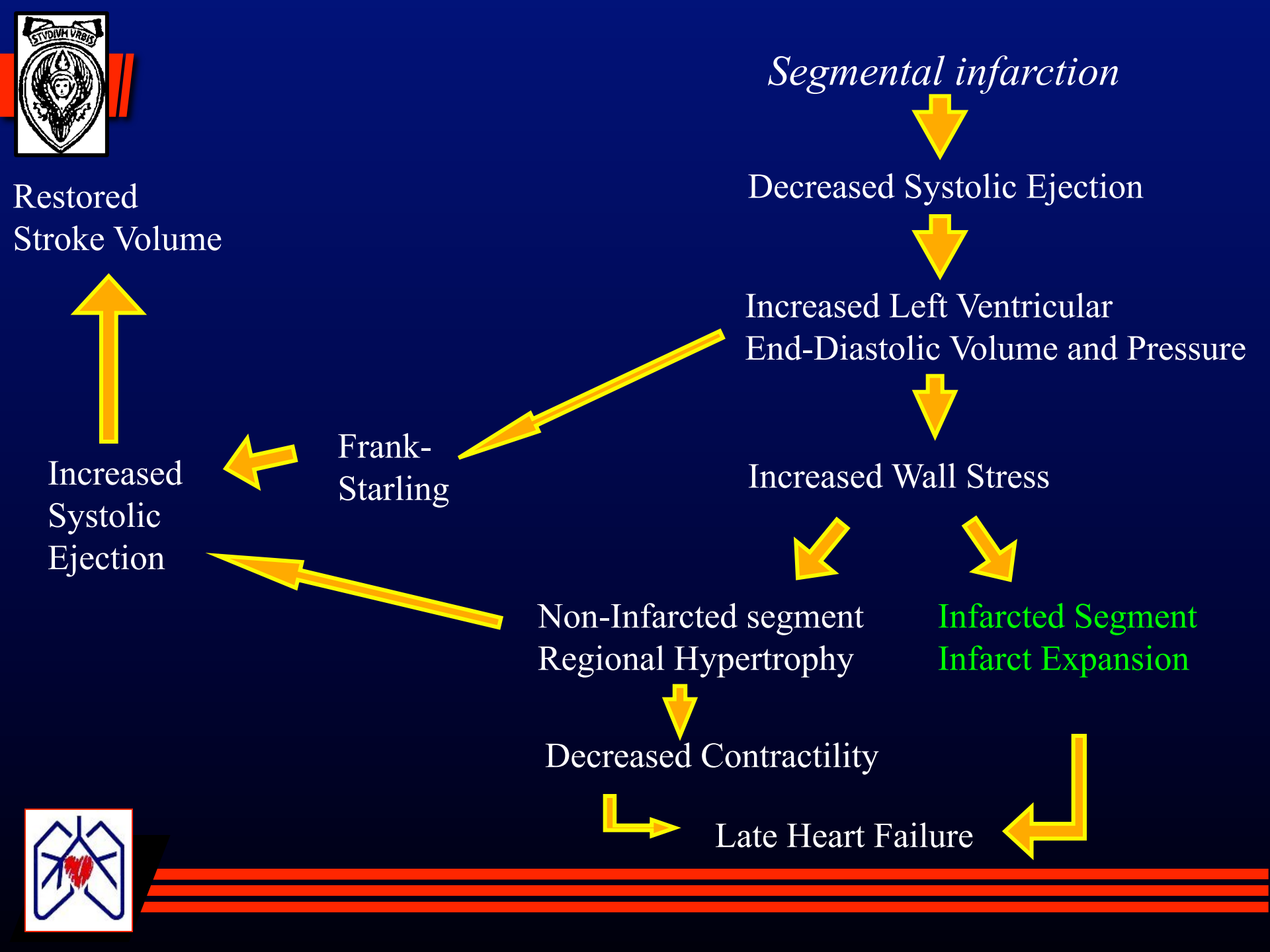

Segmental infarction
Decreased Systolic Ejection
Increased Left Ventricular End-Diastolic Volume and Pressure
Increased Wall Stress
Infarcted Segment Infarct Expansion
Non-Infarcted segment Regional Hypertrophy
Decreased Contractility
Late Heart Failure
Frank-Starling
Increased Systolic Ejection
Restored Stroke Volume

# *Calcolo dello stress circonferenziale mediante ecocardiografia tridimensionale*



# *Calcolo dello stress meridiano mediante ecocardiografia tridimensionale*

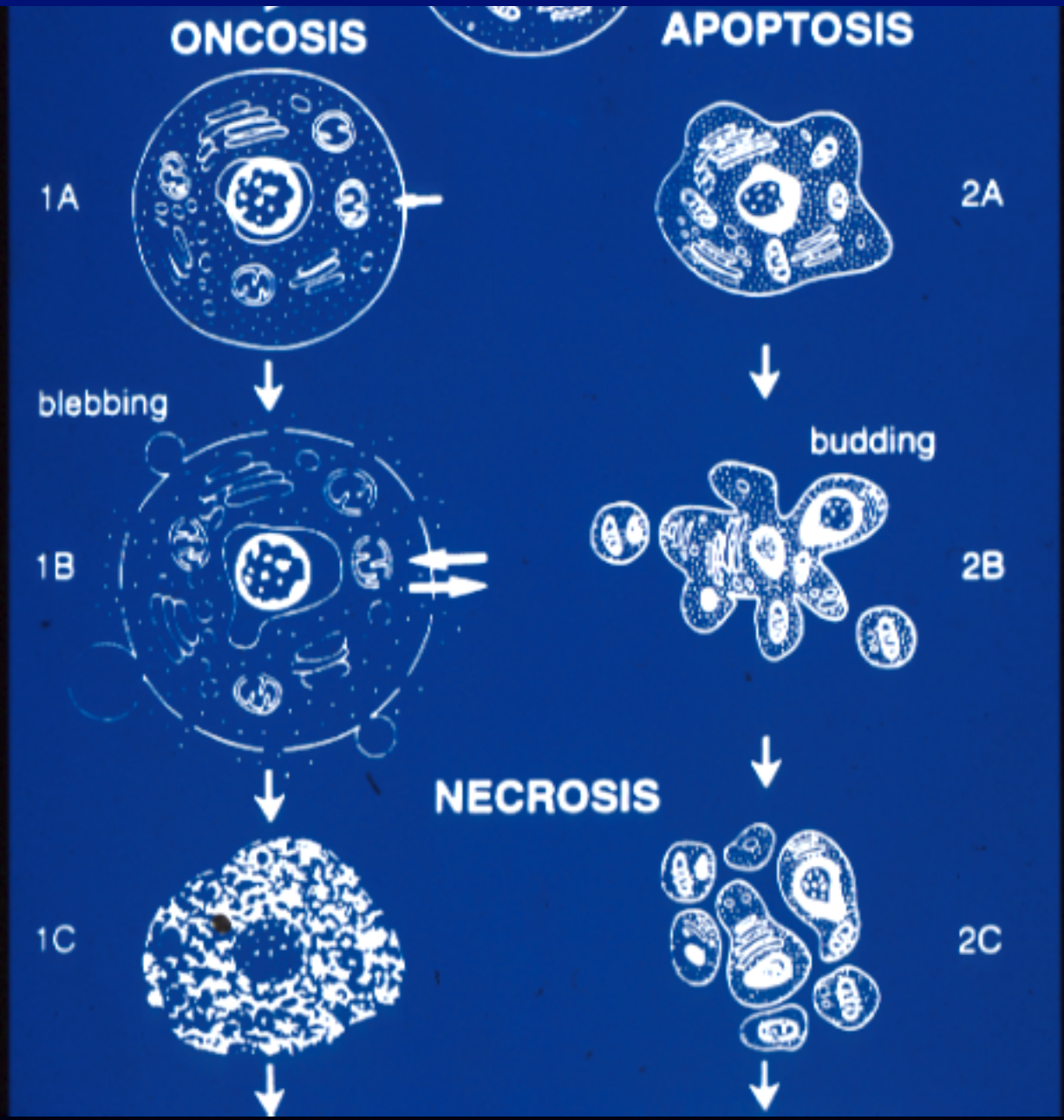



Segmental infarction
Decreased Systolic Ejection
Increased Left Ventricular End-Diastolic Volume and Pressure
Increased Wall Stress
Infarcted Segment
Infarct Expansion
Non-Infarcted segment
Regional Hypertrophy
Decreased Contractility
Late Heart Failure
Frank-Starling
Increased Systolic Ejection
Restored Stroke Volume

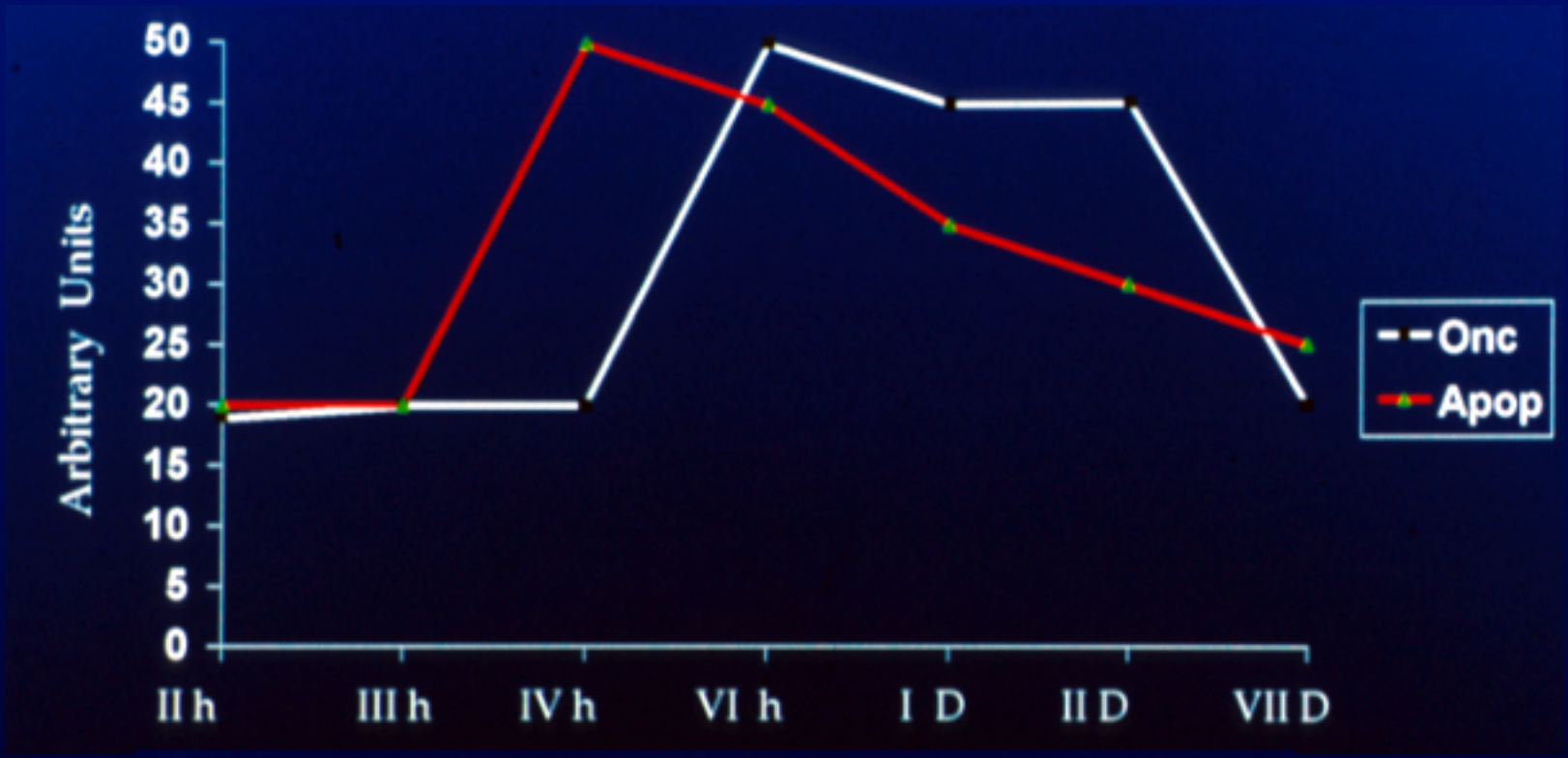
# Cellular Mechanisms of Myocardial Infarct Expansion

Harlan F. Weisman, MD; David E. Bush, MD; John A. Mannisi, MD; Myron L. Weisfeldt, MD and Bernardine Healy, MD

(*Circulation 1988; 78: 186-201*)


ONCOSIS
APOPTOSIS
1A
2A
blebbing
budding
1B
2B
NECROSIS
1C
2C

# *Myocyte Cell Death in the Infarcted Heart*




*Oncosis* ⇨ *90.000 myocytes*

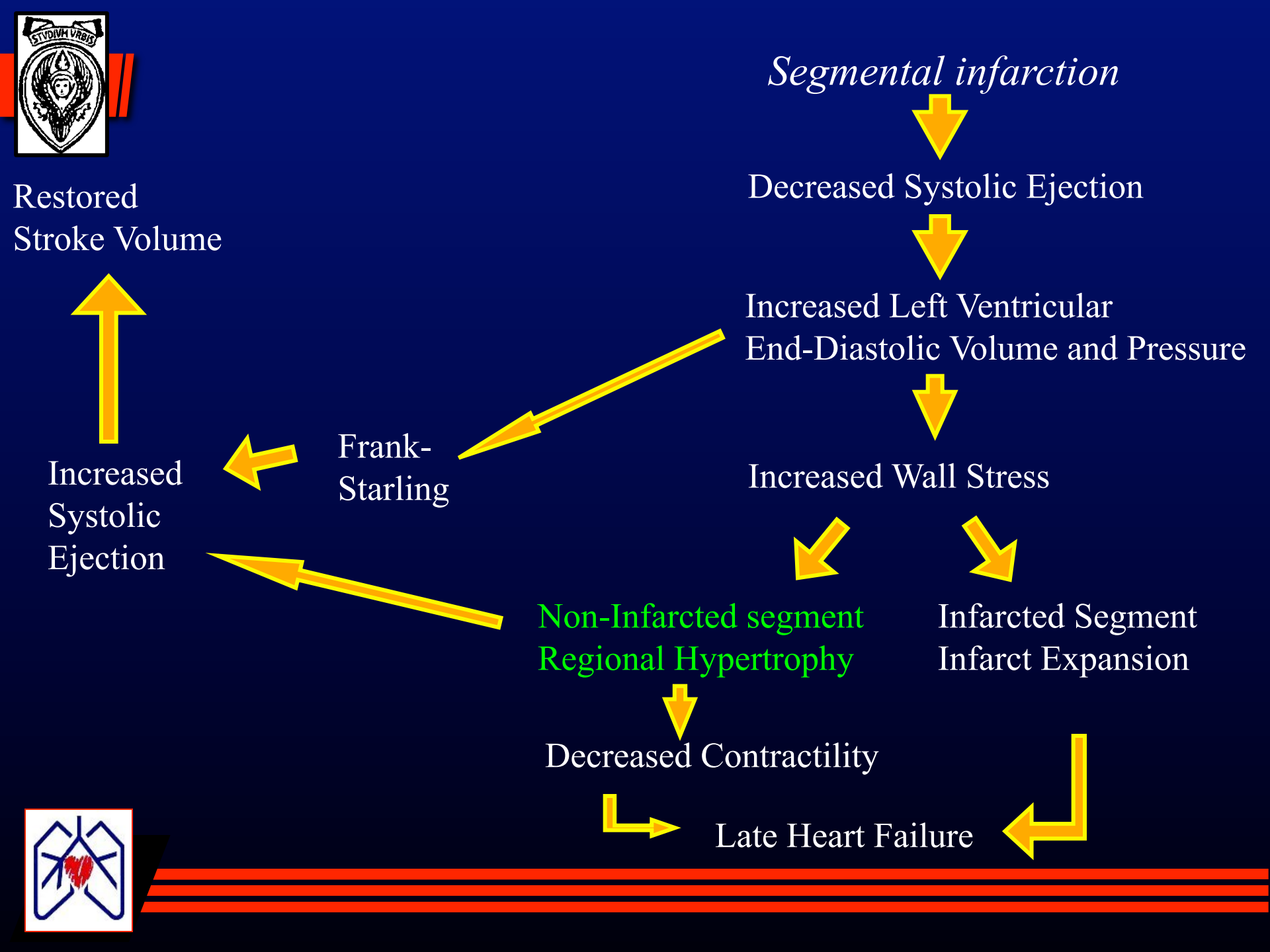
*Apoptosis* ⇨ *2.800 .000 myocytes* ] *Necrosis*

# Stretch-Induced Programmed Myocyte Cell Death

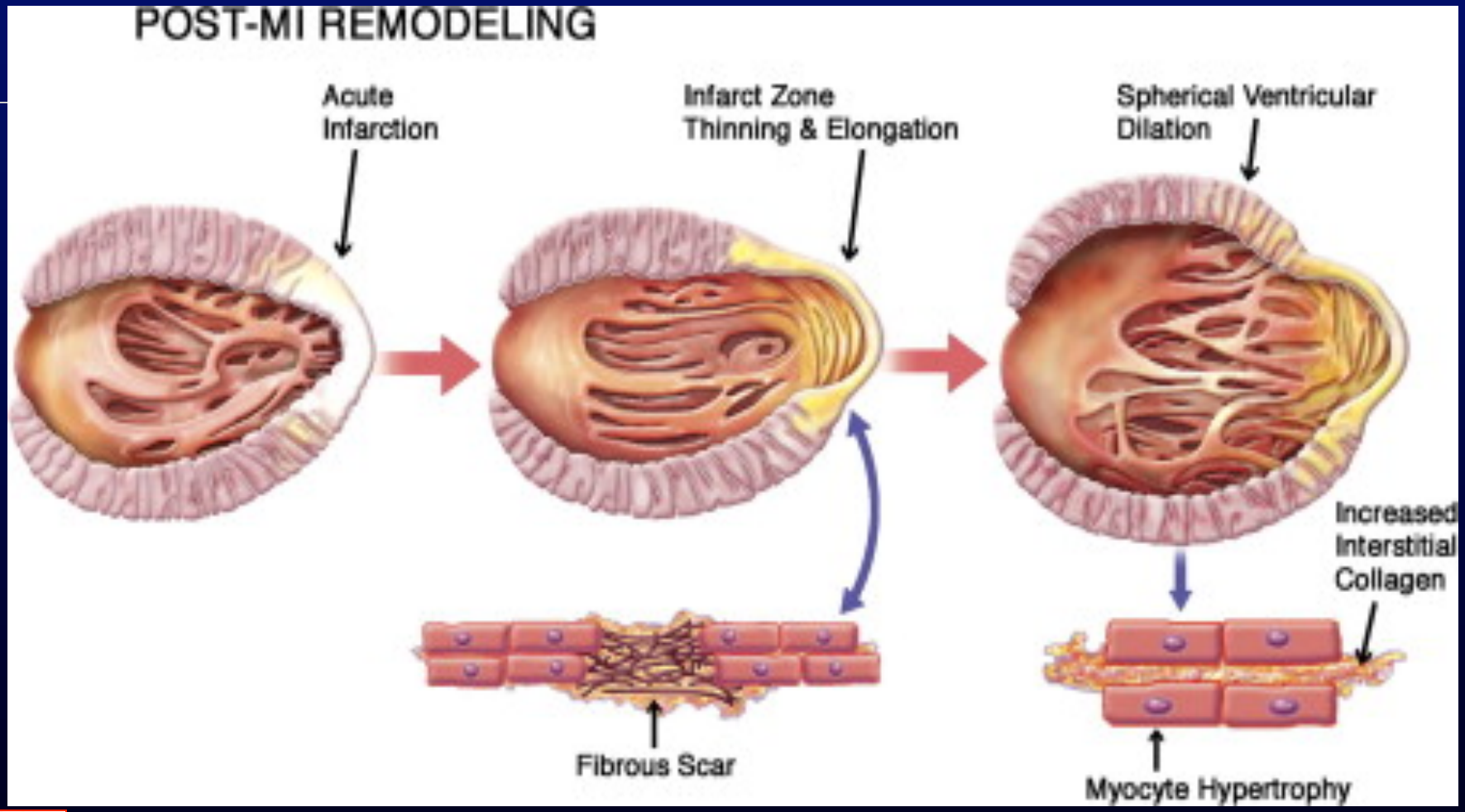
Wei Cheng,* Baosheng Li,* Jan Kajstura,* Peng Li,* Micheal s. Wolin,†
Edmund H. Sonnenblick,*†Thomas H. Hintze,† Giorgio Olivetti,* and Piero Anversa*†

Department of *Medicine and Physiology, New York Medical College, Valhalla, New York 10595: and †Department of Medicine, Albert Einstein College of Medicine, New York 10461.

(*J. Clin. Invest. 1995. 96: 2247-2259)*


Segmental infarction
Decreased Systolic Ejection
Increased Left Ventricular End-Diastolic Volume and Pressure
Increased Wall Stress
Infarcted Segment Infarct Expansion
Non-Infarcted segment Regional Hypertrophy
Decreased Contractility
Late Heart Failure
Frank-Starling
Increased Systolic Ejection
Restored Stroke Volume

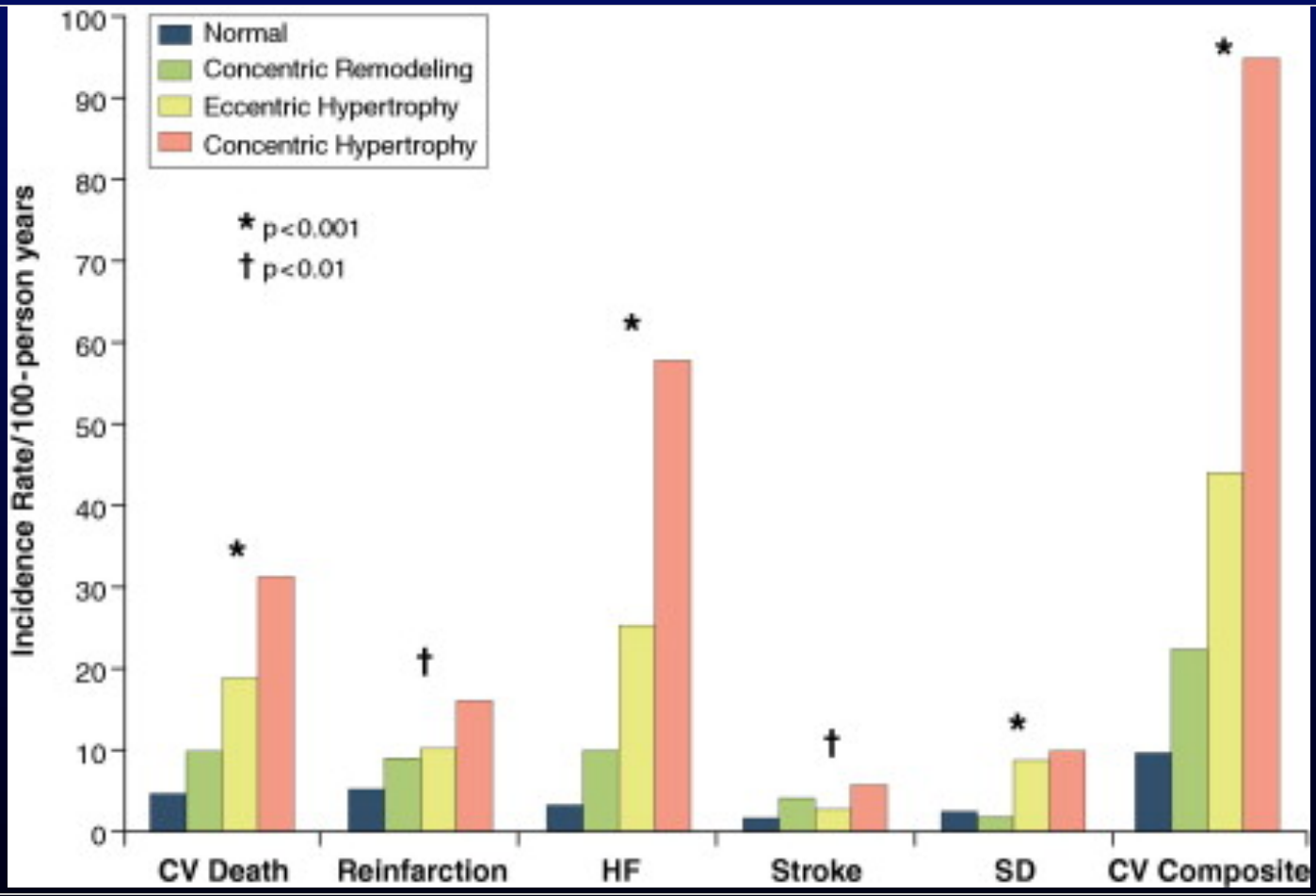
# Left Ventricular Remodeling in Heart Failure: Current Concepts in Clinical Significance and Assessment



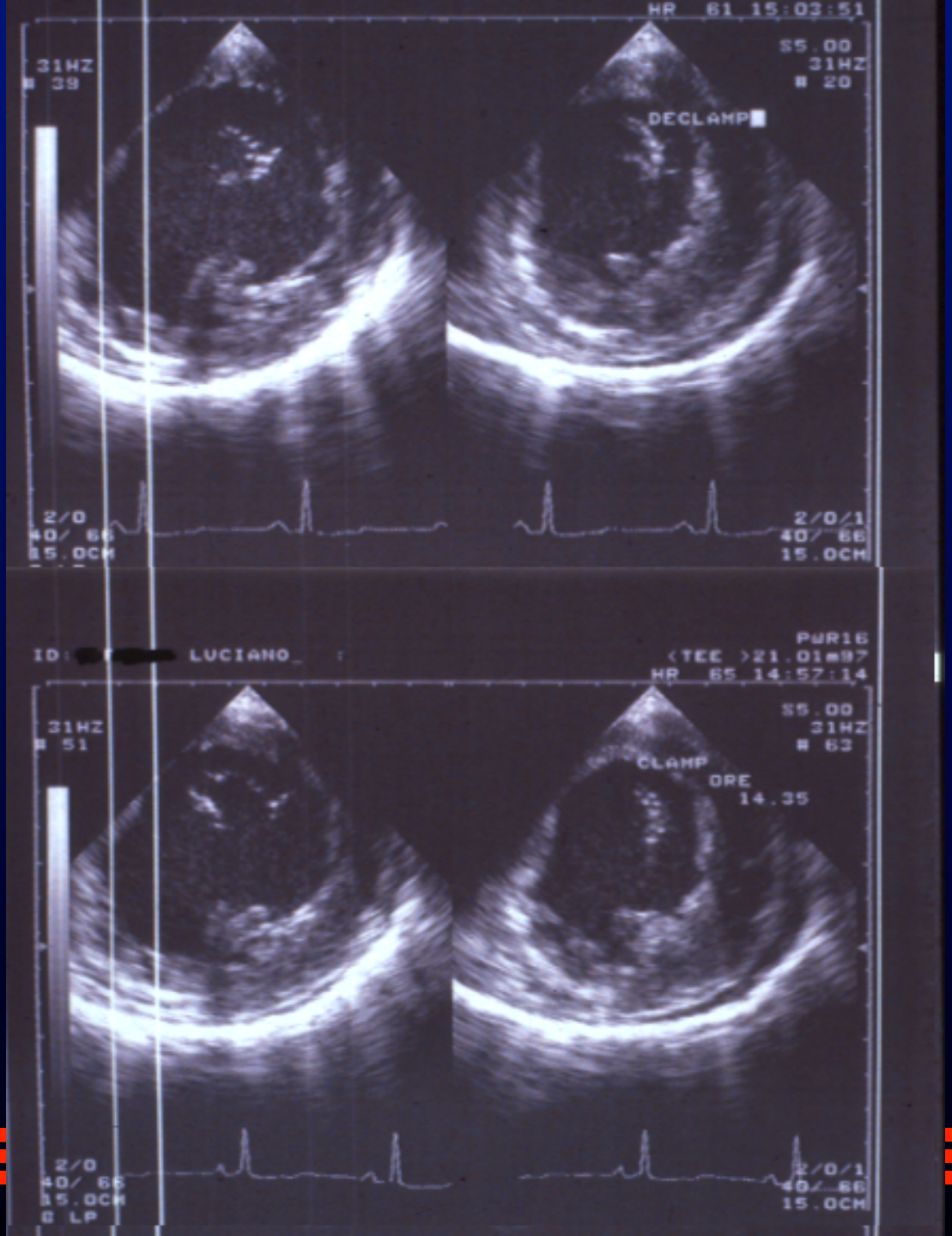

J Am Coll Cardiol Img. 2011

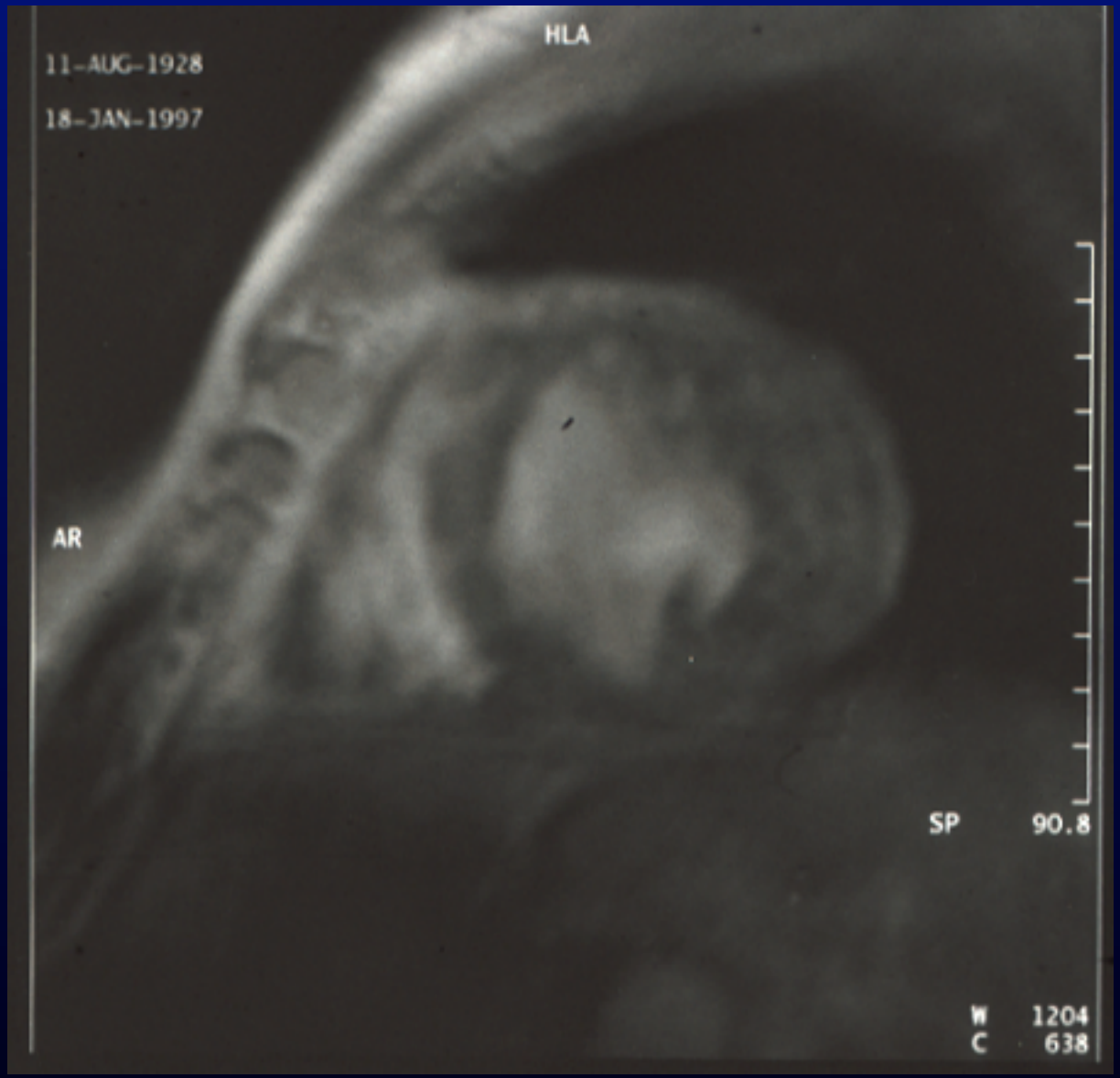
# Prognostic Implications of Left Ventricular Mass and Geometry Following Myocardial Infarction

The VALIANT (VALsartan In Acute myocardial iNfarcTion) Echocardiographic Study

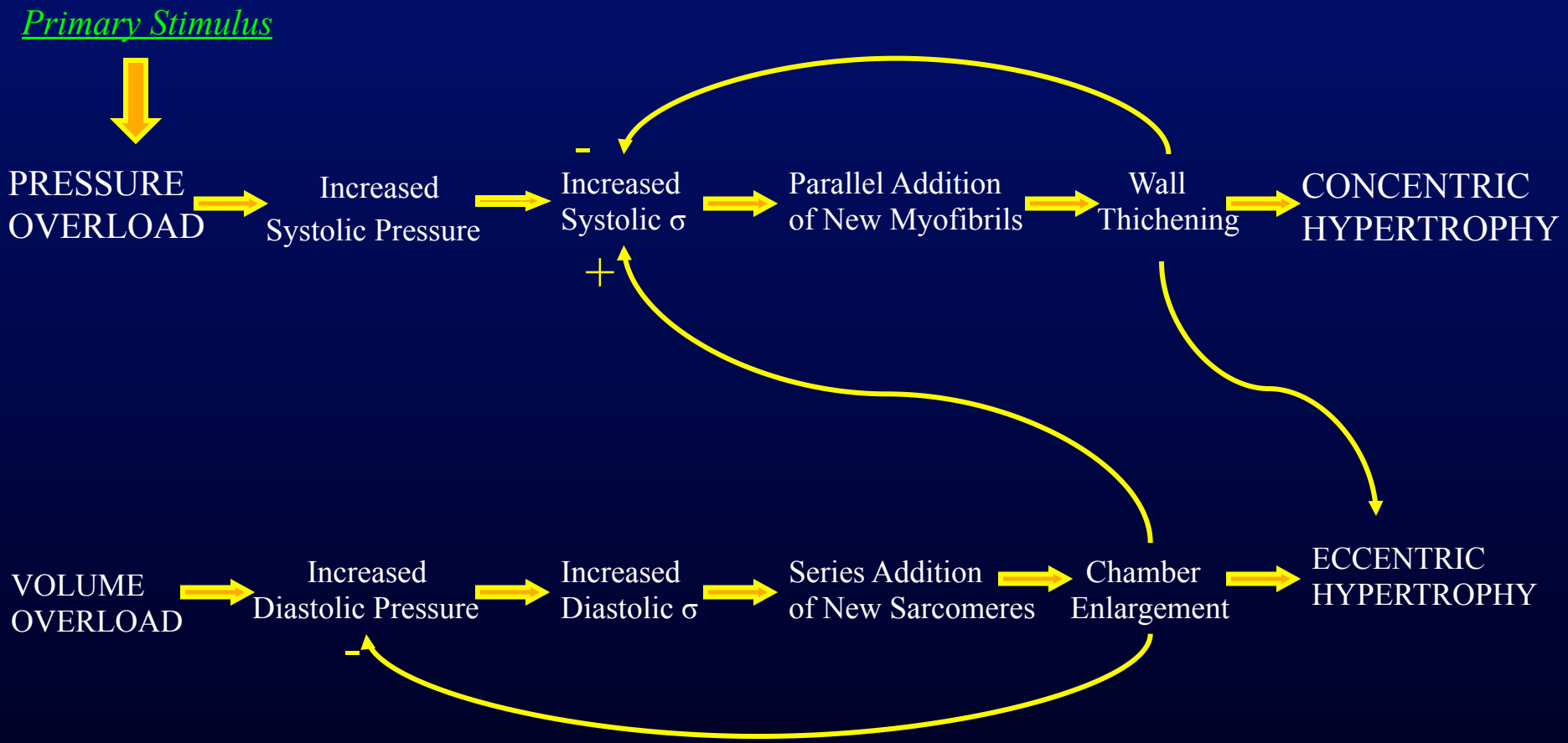



Verma et al. JACC: CARDIOVASCULAR IMAGING VOL. 1, NO. 5, 2008


HR 61 15:03:51
31HZ
# 39
55.00
31HZ
# 20
DECLAMP
2/0
40/ 66
15.0CM
2/0/1
40/ 66
15.0CM
ID: LUCIANO
PWR16
(TEE )21.01m97
HR 65 14:57:14
31HZ
# 51
55.00
31HZ
# 63
CLAMP
ORE
14.35
2/0
40/ 66
15.0CM
C LP
2/0/1
40/ 66
15.0CM


HLA
11-AUG-1928
18-JAN-1997
AR
SP 90.8
W 1204
C 638

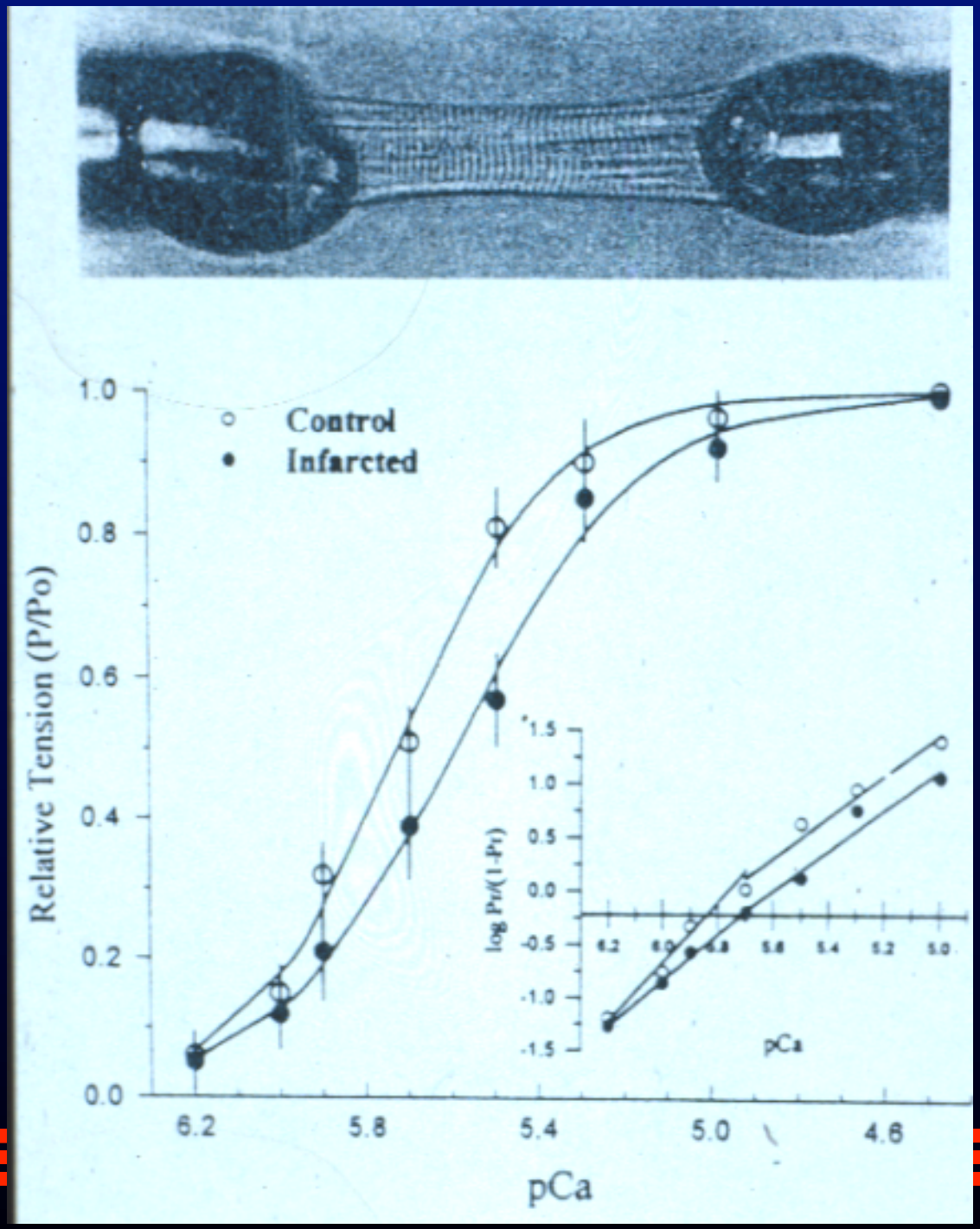
# HYPOTHESIS

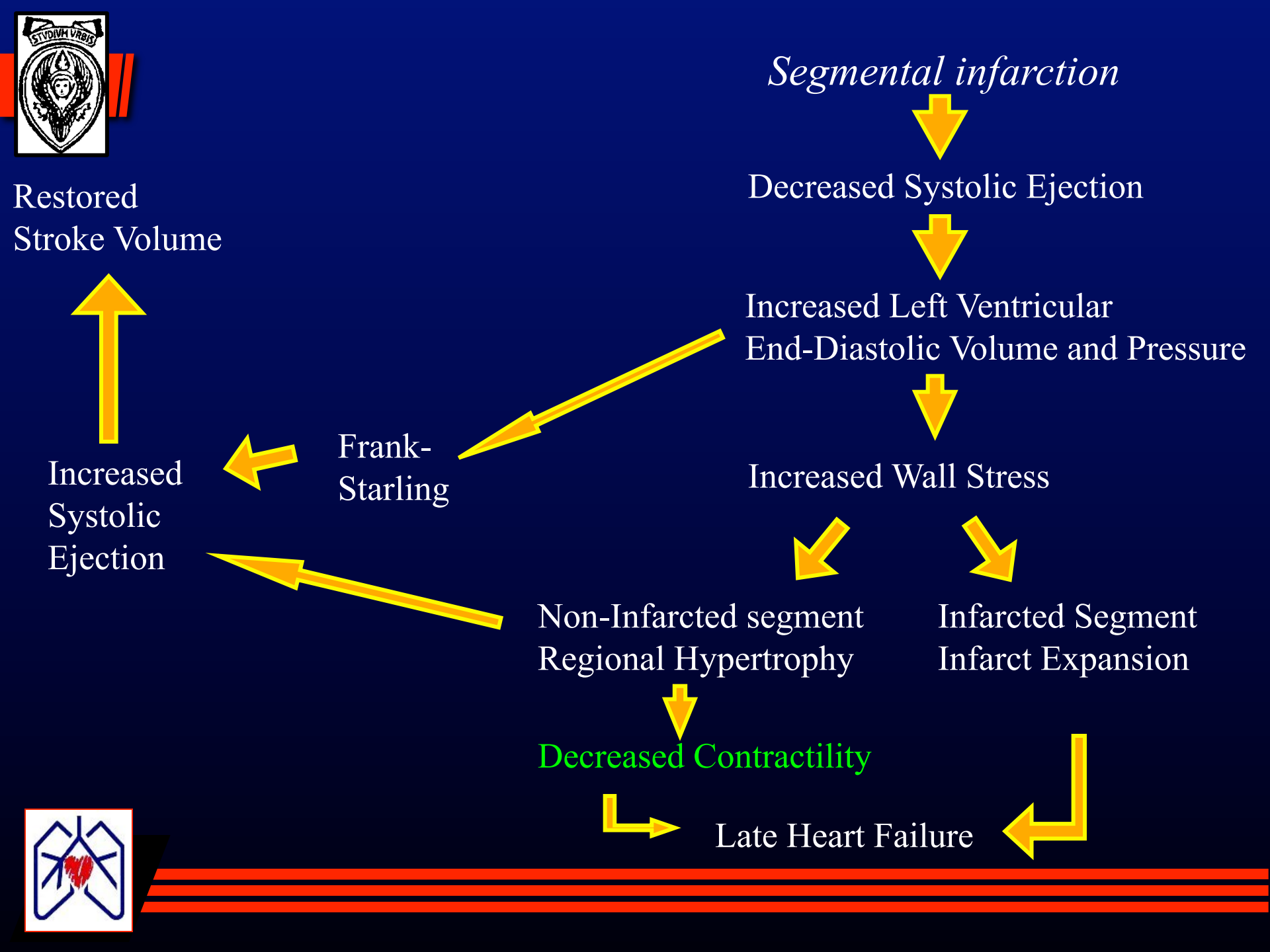

Primary Stimulus
PRESSURE OVERLOAD
Increased Systolic Pressure
Increased Systolic σ
Parallel Addition of New Myofibrils
Wall Thichening
CONCENTRIC HYPERTROPHY
VOLUME OVERLOAD
Increased Diastolic Pressure
Increased Diastolic σ
Series Addition of New Sarcomeres
Chamber Enlargement
ECCENTRIC HYPERTROPHY

# Functional Significance of Hypertrophy of the Noninfarcted Myocardium After Myocardial Infarction in Humans

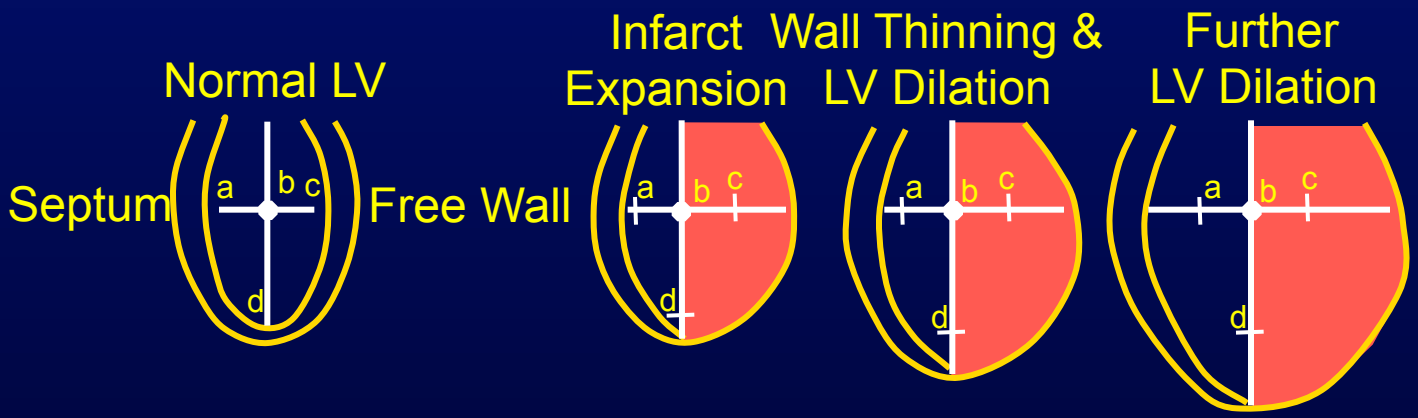
Leonard E. Ginzton, MD; Richard Conant, PhD, derek M. Rodrigues, MD and Michael M. Laks, MD

*(Circulation 1989; 80: 816-822)*


Control
Infarcted
Relative Tension (P/Po)
1.0
0.8
0.6
0.4
0.2
0.0
6.2
5.8
5.4
5.0
4.6
pCa
log Pr/(1-Pr)
1.5
1.0
0.5
0.0
-0.5
-1.0
-1.5
pCa


Segmental infarction
Decreased Systolic Ejection
Increased Left Ventricular End-Diastolic Volume and Pressure
Increased Wall Stress
Non-Infarcted segment Regional Hypertrophy
Infarcted Segment Infarct Expansion
Decreased Contractility
Late Heart Failure
Frank-Starling
Increased Systolic Ejection
Restored Stroke Volume

# Ventricular Remodeling Following MI

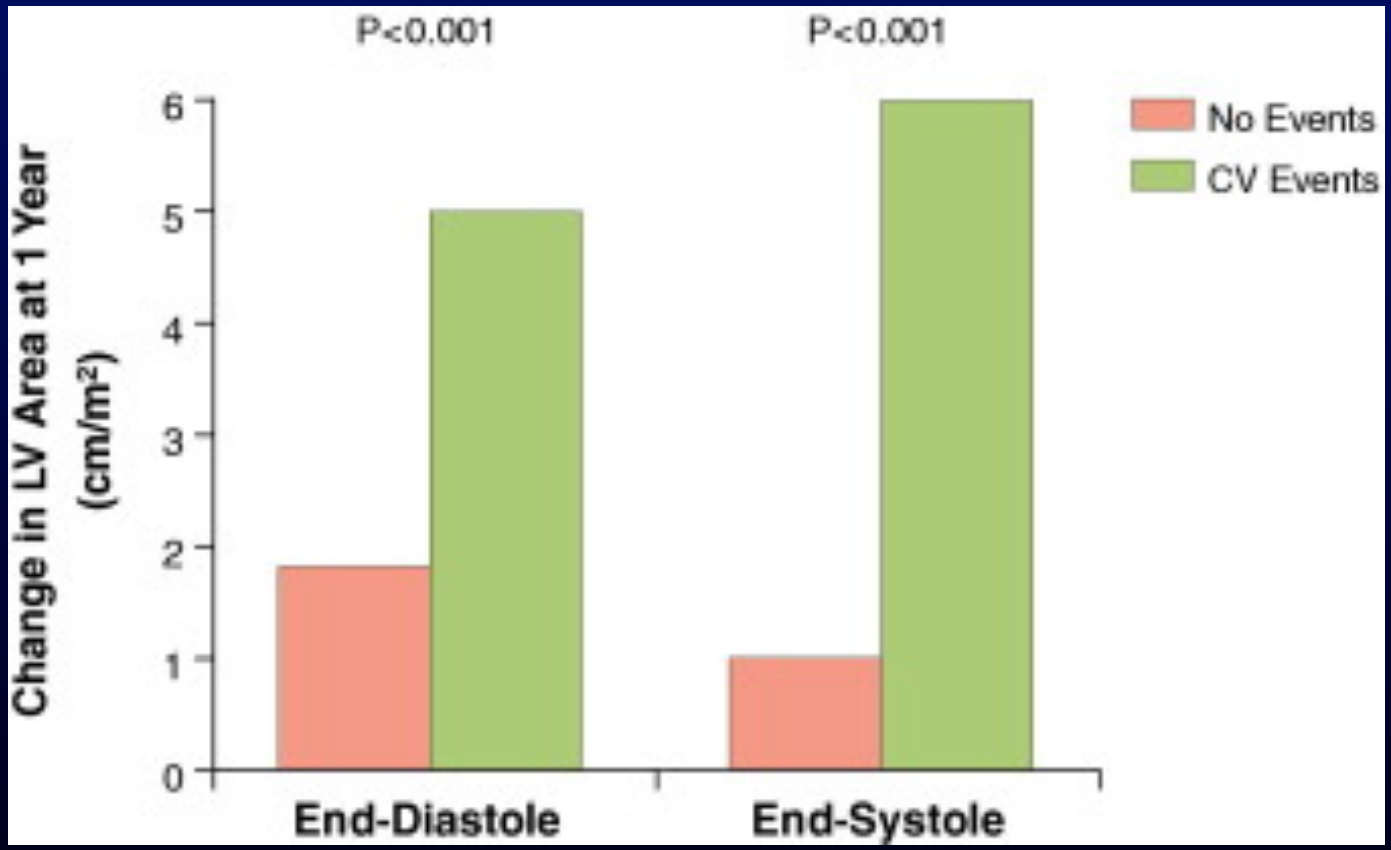



## Volume Changes Occurring in Left Ventricle

Pfeffer MA. *Am J Cardiol.* 1991;68:17D-25D.

# Quantitative Two-dimensional Echocardiographic Measurements Are Major Predictors of Adverse Cardiovascular Events After Acute Myocardial Infarction

## The Protective Effects of Captopril




Sutton et al, Circulation 1994;89:68-75

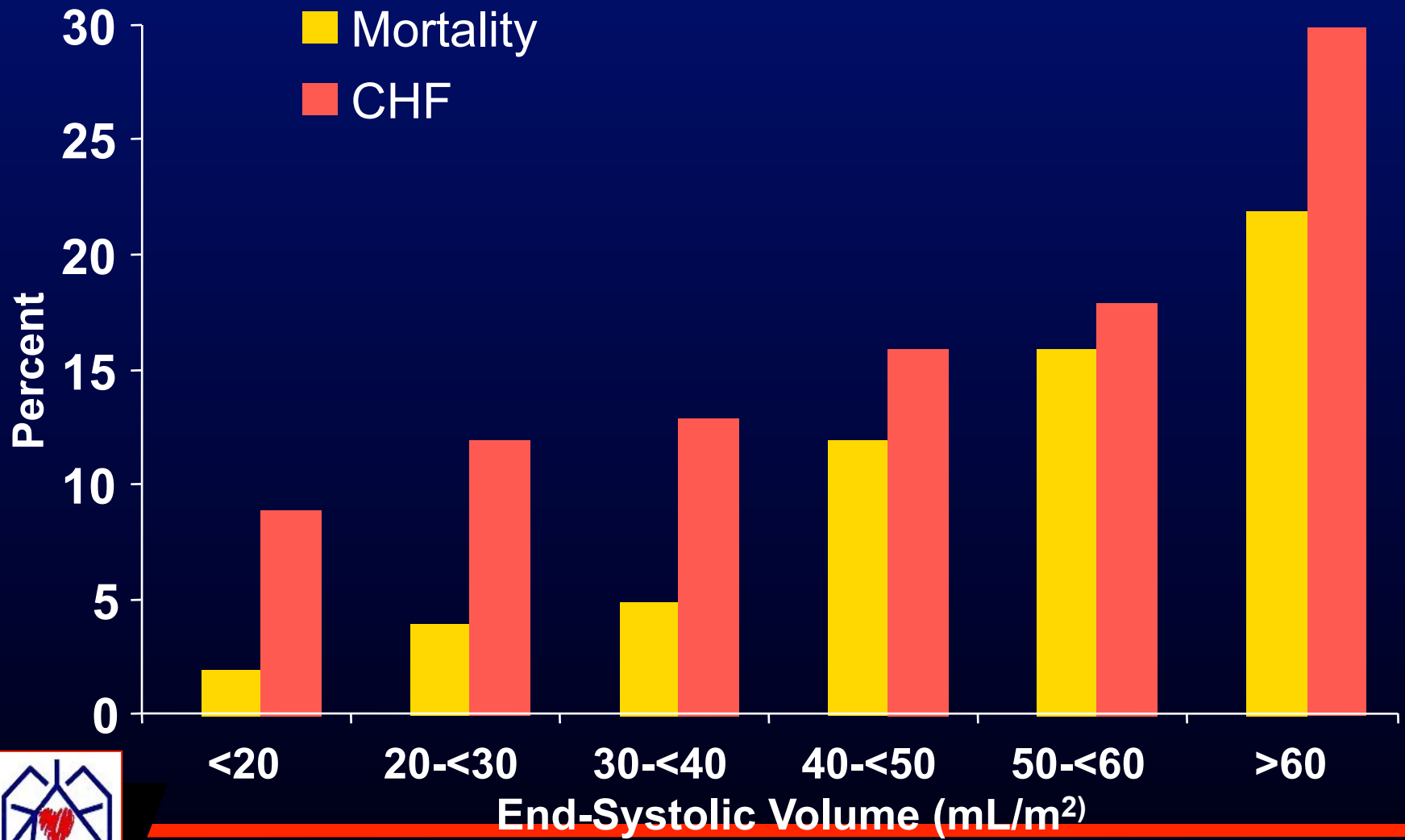
# Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction

HARVEY D. WHITE, M.B., F.R.A.C.P., ROBIN M. NORRIS, M.D., F.R.A.C.P., MICHAEL A. BROWN, M.B., F.R.A.C.P., PETER W. T. BRANDT, M.B., F.R.A.C.R., RALPH M. L. WHITLOCK, M.B., F.R.A.C.P., AND CHRISTOPHER J. WILD, PH.D.

ABSTRACT Impairment of left ventricular function is the major predictor of mortality after acute myocardial infarction, but it is not known whether this is best described by ejection fraction or by end-systolic or end-diastolic volume. We measured volumes, ejection fractions, and severity of coronary arterial occlusions and stenoses in 605 male patients under 60 years of age at 1 to 2 months after a first (n = 443) or recurrent (n = 162) myocardial infarction and followed these patients for a mean of 78 months for survivors (range 15 to 165 months). There were 101 cardiac deaths, 71 (70%) of which were sudden (instantaneous or found dead). Multivariate analysis with log rank testing and the Cox proportional hazards model showed that end-systolic volume ($\chi^2$ = 82.9) had greater predictive value for survival than end-diastolic volume ($\chi^2$ = 59.0) or ejection fraction ($\chi^2$ = 46.6), whereas stepwise analysis showed that once the relationship between survival and end-systolic volume had been fitted, there was no additional significant predictive information in either end-diastolic volume or ejection fraction. Severity of coronary occlusions and stenoses showed additional prediction of only borderline significance (p = .04 in one analysis), but continued cigarette smoking did remain an independent risk factor after stepwise analysis. For a subset of patients (n = 200) who had taken part in a randomized trial of coronary artery surgery after recovery from infarction, surgical "intention to treat" showed no predictive value. We conclude that for prediction, end-systolic volume is the primary predictor of survival after myocardial infarction, being superior to ejection fraction when ejection fraction is low (<50%) or when end-systolic volume is high (<100 ml). Treatment of infarction should be aimed at limitation of infarct size and prevention of ventricular dilation.

*Circulation 76, No. 1, 44–51, 1987.*

# Relation Between Post-MI End Systolic Volume and Natural History Outcomes



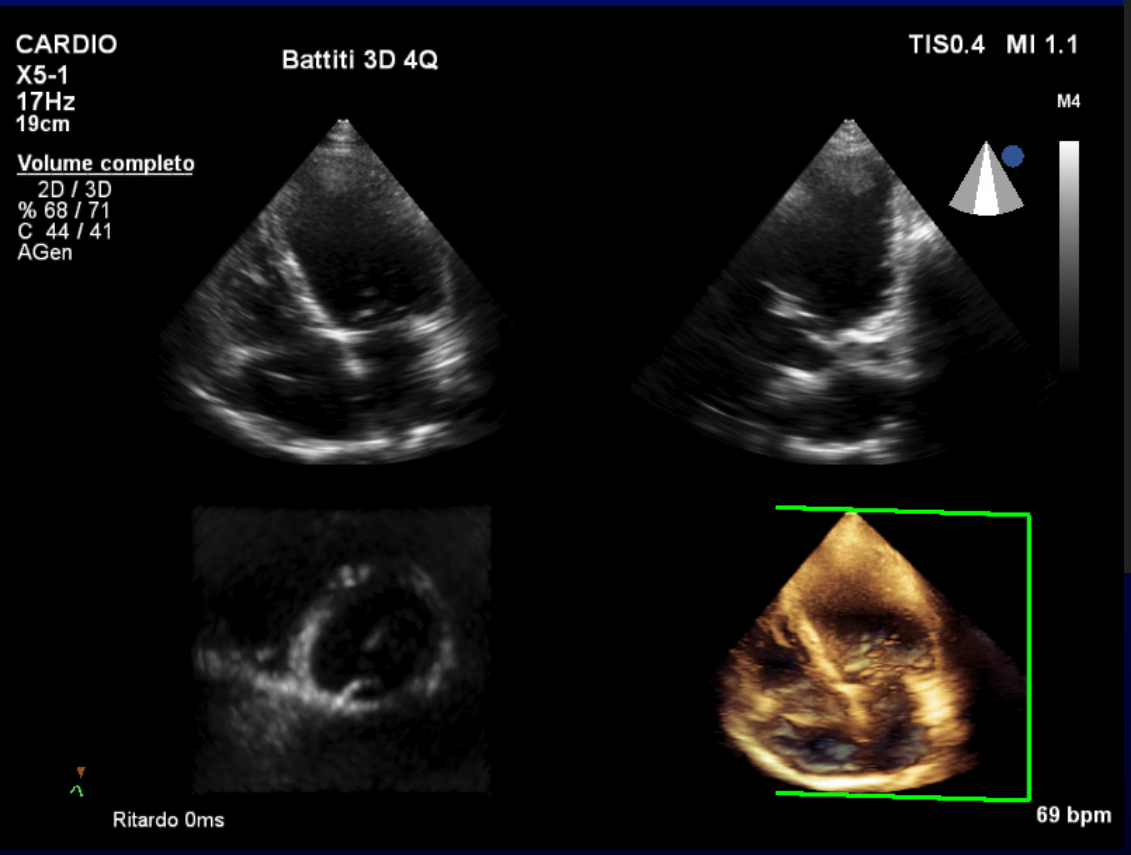

Migrino RO et al. *Circulation.* 1997;96:116-121.

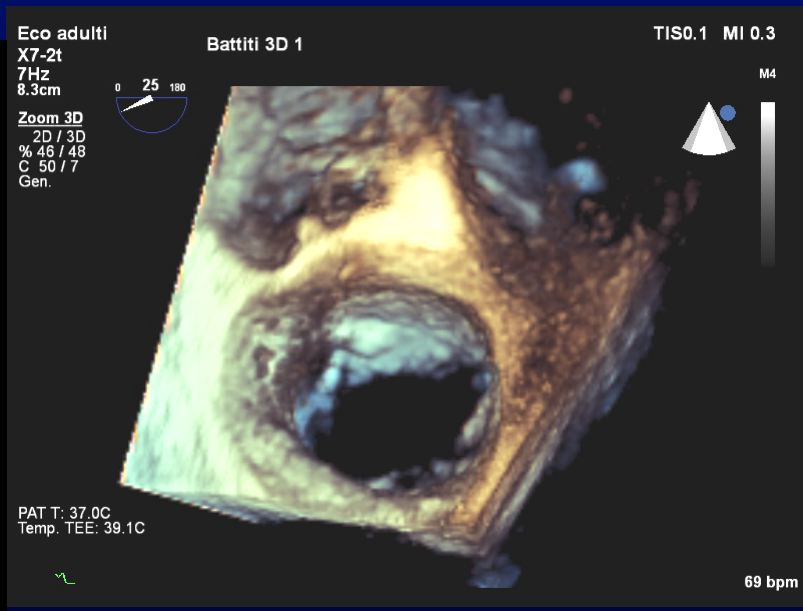
# Pathogenesis of mitral regurgitation in acute Myocardial infarction: importance of changes in Left ventricular shape and regional function

Jan M. Van Dantzig, MD; Ben J. Delemarre, MD; Rudolph W. Koster, MD; Hans Bot, PhD and Cees A. Visser, MD *Amsterdam, The Netherlands*

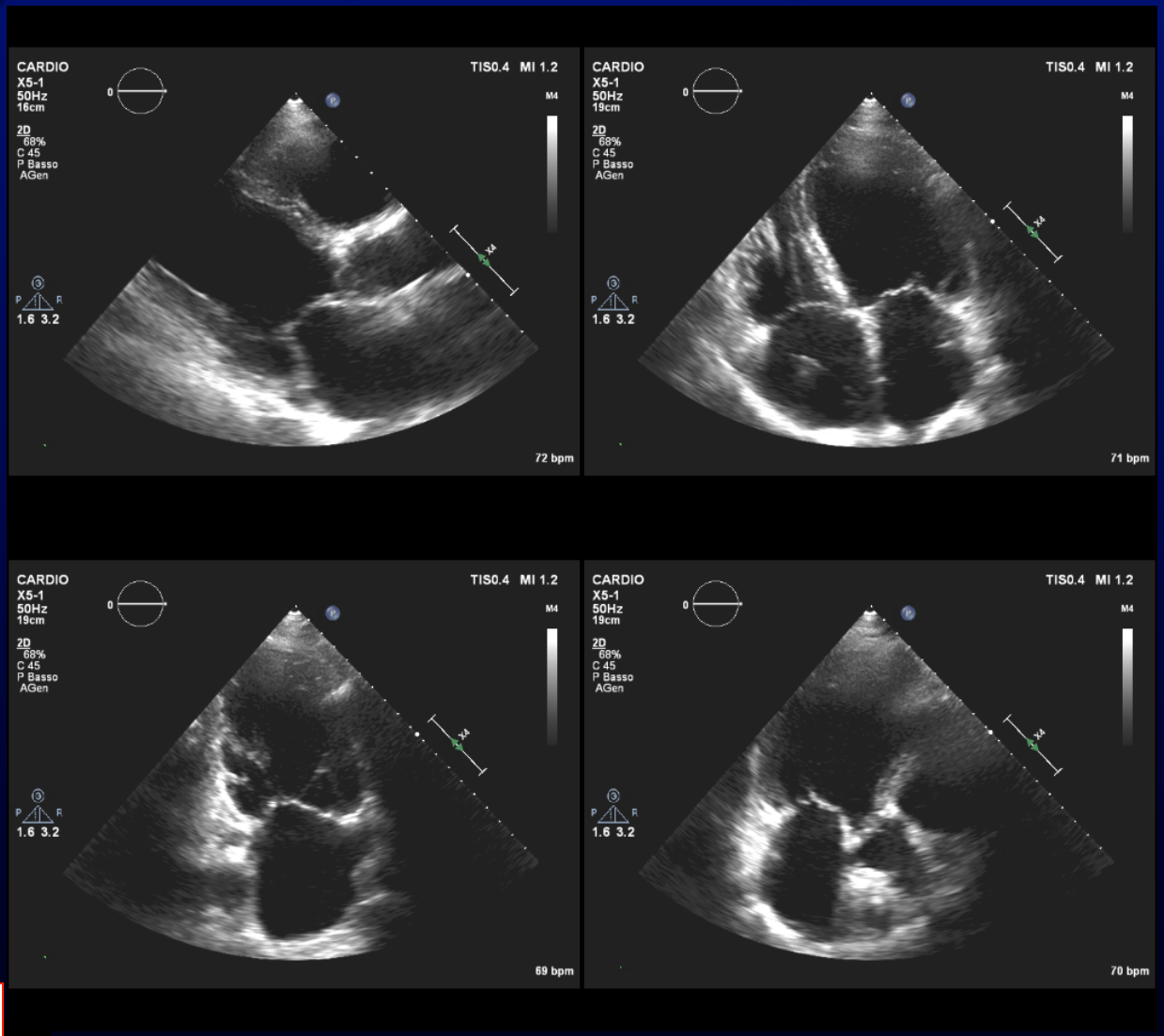
*(Am Heart J 1996; 131:865-871)*

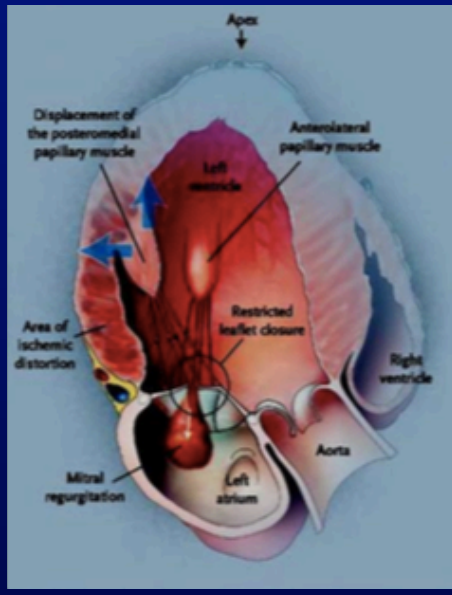
# *3D Full Volume and RT3D evaluation: insights on LV function and MV morphology*



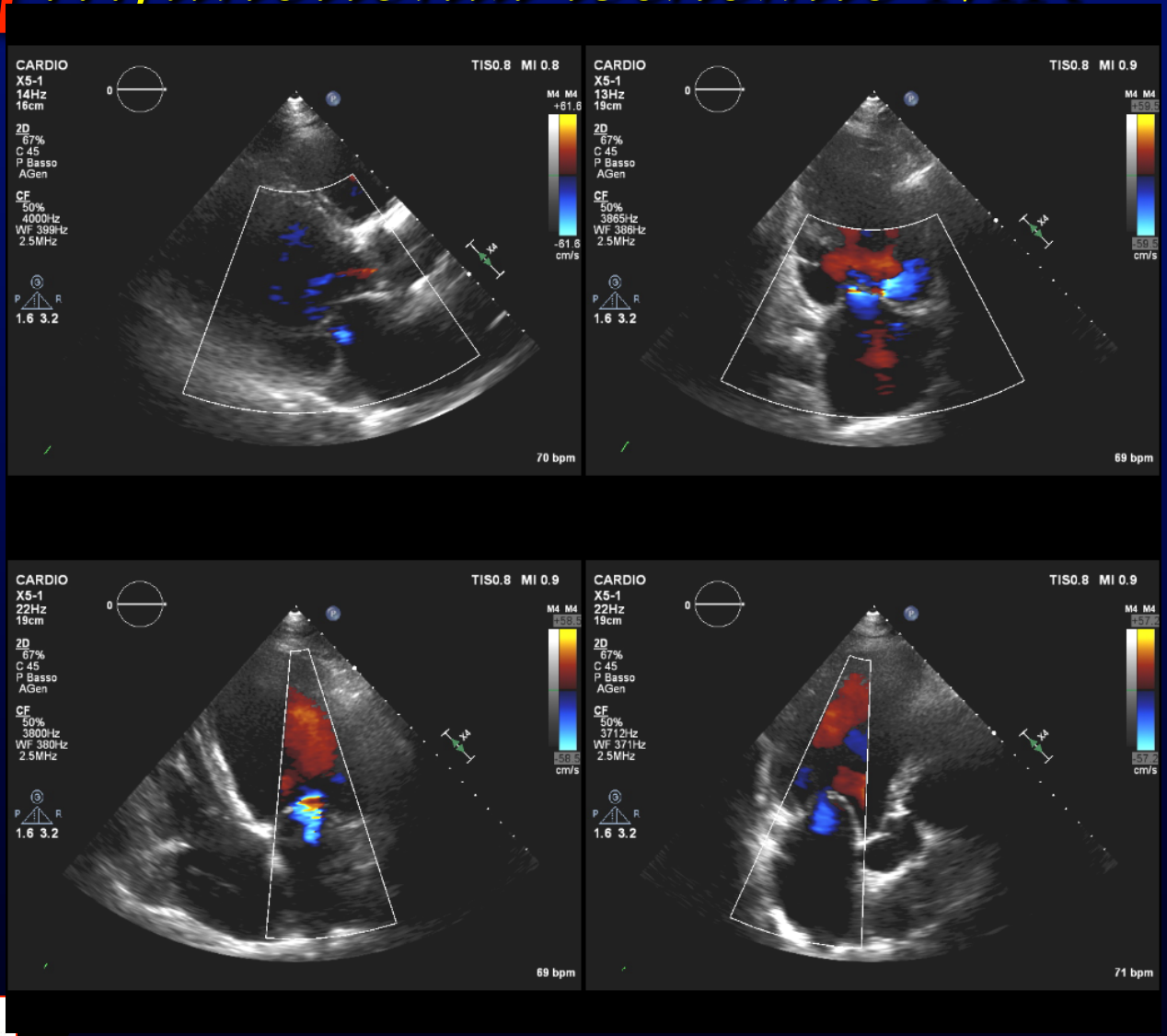



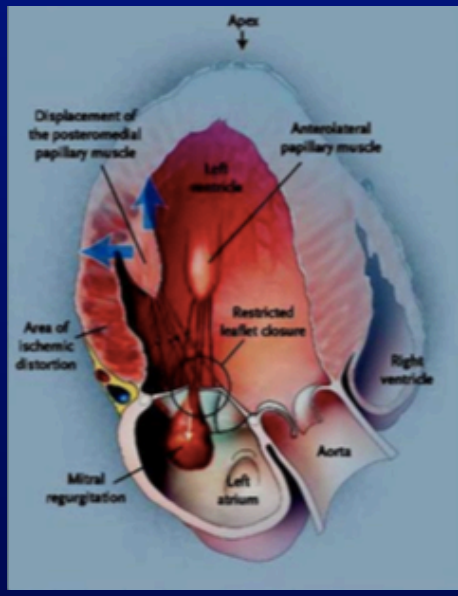
# Pt with previous inferior MI



# …functional ischemic MR

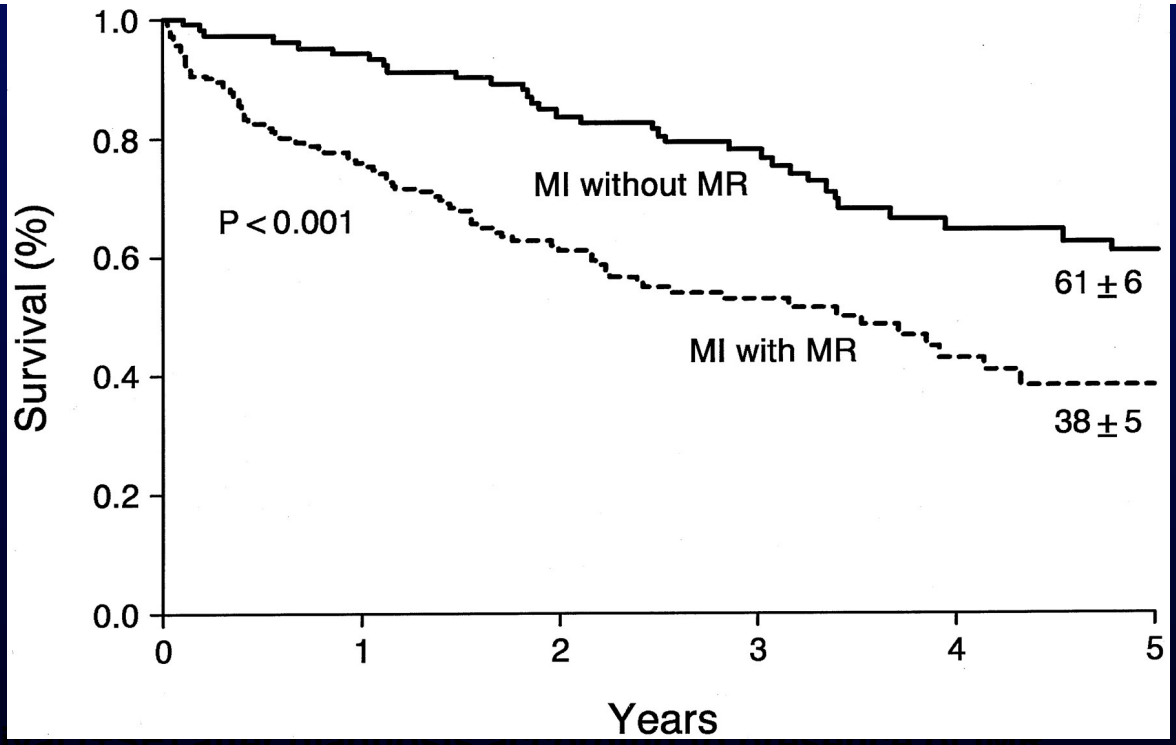


# Ischemic Mitral Regurgitation

## Long-Term Outcome and Prognostic Implications With Quantitative Doppler Assessment

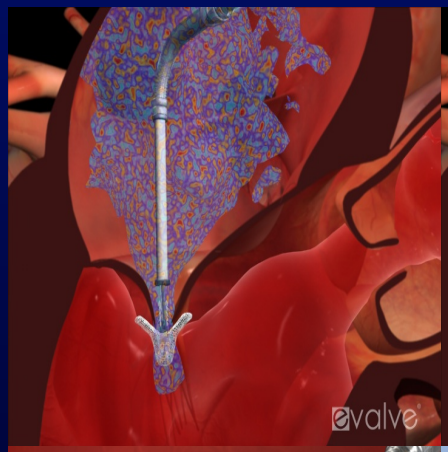
Francesco Grigioni, MD; Maurice Enriquez-Sarano, MD; Kenton J. Zehr, MD;
Kent R. Bailey, PhD; A. Jamil Tajik, MD

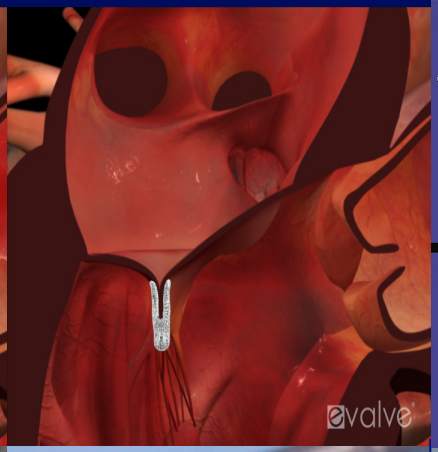


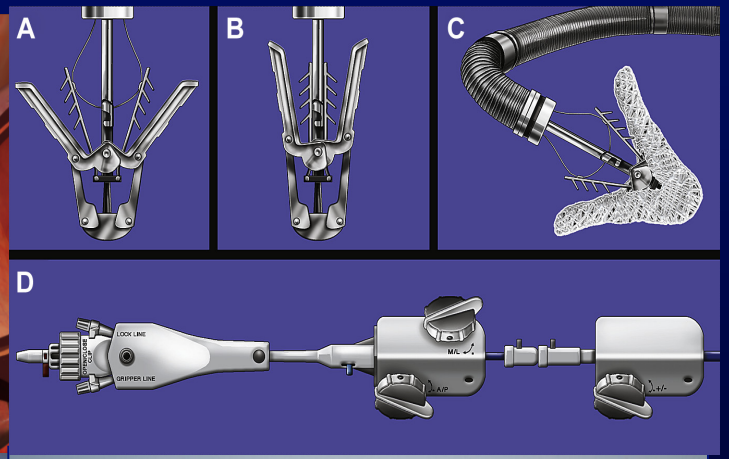

Survival (%) after diagnosis according to presence of MR.

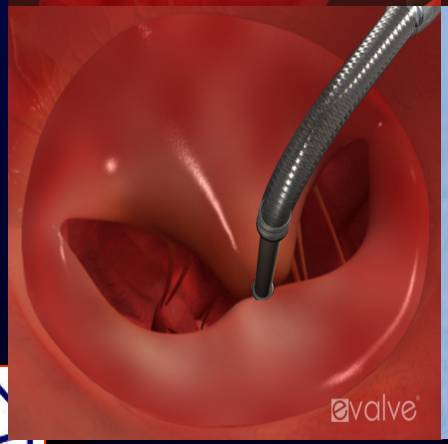
Grigioni F et al. Circulation. 2001;103:1759-1764

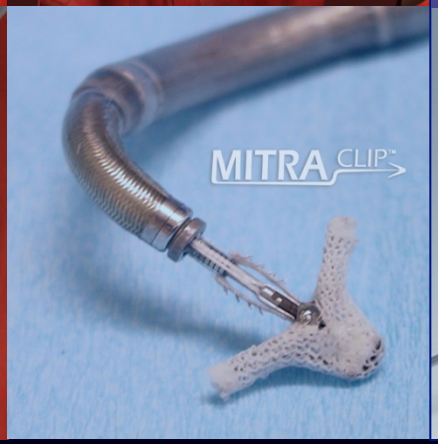
# Percutaneous Mitral Valve Repair




# MitraClip: essential role of echo

- Patient selection
- Guidance to Procedure
- Identification/Assessment of Complications
- Evaluation of Final Results
- Follow up

# *Indications for Mitral clipping*



LV function, who cannot be revascularized or who present with cardiomyopathy, are questionable. Repair may be considered in selected patients if comorbidity is low, in order to avoid or postpone transplantation. In the other patients, optimal medical treatment is currently the best option, followed, in the event of failure, by extended HF treatment [cardiac resynchronization therapy (CRT); ventricular assist devices; cardiac restraint devices; heart transplantation].

The percutaneous mitral clip procedure may be considered in patients with symptomatic severe secondary MR despite optimal medical therapy (including CRT if indicated), who fulfil the echo criteria of eligibility, are judged inoperable or at high surgical risk by a team of cardiologists and cardiac surgeons, and who have a life expectancy greater than 1 year (recommendation class IIb, level of evidence C).

There is continuing debate regarding the management of moderate ischaemic MR in patients undergoing CABG. In such cases, valve repair is preferable. In patients with low EF, mitral valve surgery is more likely to be considered if myocardial viability is present and if comorbidity is low. In patients capable of exercising, exercise echocardiography should be considered whenever possible. Exercise-induced dyspnoea and a large increase in MR severity and systolic pulmonary artery pressure favour combined surgery.

There are no data to support surgical correction of mild MR.

#### 6.2.6 Medical treatment

Optimal medical therapy is mandatory: it should be the first step in the management of all patients with secondary MR and should be given in line with the guidelines on the management of HF.[13] This includes ACE inhibitors and beta-blockers, with the addition of an aldosterone antagonist in the presence of HF. A diuretic is required in the presence of fluid overload. Nitrates may be useful for treating acute dyspnoea, secondary to a large dynamic component.

The indications for resynchronization therapy should be in accordance with related guidelines.[13] In responders, CRT may immediately reduce MR severity through increased closing force and resynchronisation of papillary muscles.[159] A further reduction in MR and its dynamic component can occur through a reduction in tethering force in relation to LV reverse remodelling.

## 7. Mitral stenosis

Rheumatic fever, which is the predominant aetiology of MS, has greatly decreased in industrialized countries; nevertheless, MS still results in significant morbidity and mortality worldwide.[1,3] Percutaneous mitral commissurotomy (PMC) has had a significant impact upon the management of rheumatic MS.

### 7.1 Evaluation

The patient with MS may feel asymptomatic for years and then present with a gradual decrease in activity. The diagnosis is usually established by physical examination, chest X-ray, ECG, and echocardiography.

The general principles for the use of invasive and non-invasive investigations follow the recommendations made in the *General comments* (Section 3).[12]

Specific issues in MS are as follows:

- Echocardiography is the main method used to assess the severity and consequences of MS, as well as the extent of anatomic lesions.

  Valve area should be measured using planimetry and the pressure half-time method, which are complementary. Planimetry, when it is feasible, is the method of choice, in particular immediately after PMC. Continuity equation and proximal isovelocity could be used when additional assessment is needed. Measurements of mean transvalvular gradient, calculated using Doppler velocities, are highly rate- and flow-dependent, but are useful to check consistency in the assessment of severity, particularly in patients in sinus rhythm. MS does not usually have clinical consequences as long as when valve area is >1.5 cm$^2$ (*Table 4*).[15]

  A comprehensive assessment of valve morphology is important for the treatment strategy. Scoring systems have been developed to help assess suitability, taking into account valve thickening, mobility, calcification, subvalvular deformity, and commissural areas.[15,160,161]

  Echocardiography also evaluates pulmonary artery pressures, associated MR, concomitant valve disease, and LA size. Due to the frequent association of MS with other valve diseases, a comprehensive evaluation of the tricuspid and aortic valves is mandatory. TTE usually provides sufficient information for routine management.

  TOE should be performed to exclude LA thrombus before PMC or after an embolic episode, if TTE provides suboptimal information on anatomy or, in selected cases, to guide the procedure.

  3DE improves the evaluation of valve morphology (especially visualization of commissures),[162] optimizes accuracy and reproducibility of planimetry, and could be useful for guiding (TOE) and monitoring (TTE) PMC in difficult cases.

  Echocardiography also plays an important role in monitoring the results of PMC during the procedure.
- Stress testing is indicated in patients with no symptoms or symptoms equivocal or discordant with the severity of MS. Dobutamine or, preferably, exercise echocardiography may provide additional information by assessing changes in mitral gradient and pulmonary pressures.[21]

### 7.2 Natural history

Survival in asymptomatic patients is usually good up to 10 years, progression being highly variable with sudden deterioration, which is usually precipitated by pregnancy or complications such as AF or embolism.[163] Symptomatic patients have a poor prognosis without intervention.[12]

### 7.3 Results of intervention

#### 7.3.1 Percutaneous mitral commissurotomy

Technical success and complications are related to patient selection and the operator's experience.[164] Good initial results, defined as valve area >1.5 cm$^2$ with no MR >2/4, are achieved in over 80% of cases. Major complications include procedural mortality 0.5–4%, haemopericardium 0.5–10%, embolism 0.5–5%, and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**… really so effective?**

# Rimodellamento Ventricolare Post - Infartuale

## Trattamento

# *"Prevenzione Primaria"*

*E' possibile prevenire o minimizzare il processo di rimodellamento:*

- **limitando l'insulto iniziale attraverso riperfusione miocardica precoce;**
- **avviando un trattamento farmacologico precocemente ed estensivamente a tutti i pazienti dopo IMA.**

# *"Prevenzione Secondaria"*

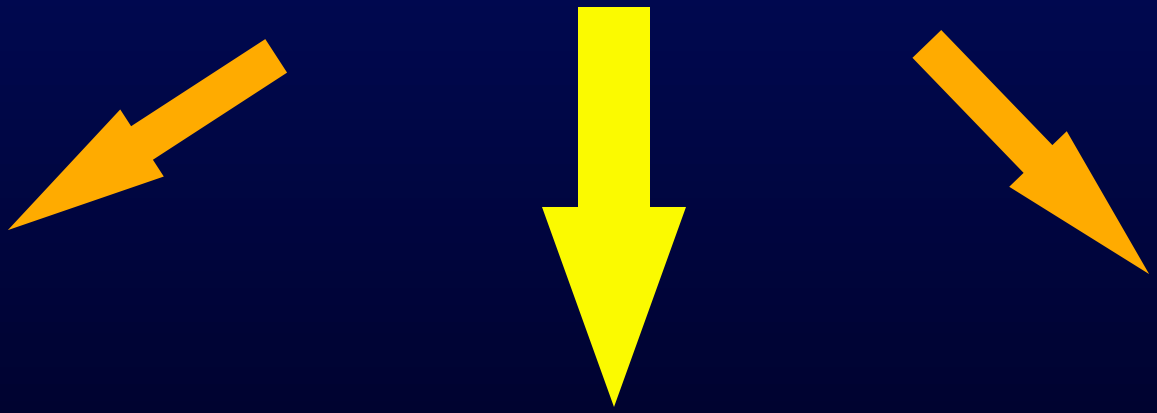
- **Trattamento farmacologico avviato più tardivamente in pazienti selezionati in base alla presenza di insufficienza cardiaca, disfunzione ventricolare sinistra, etc.**

# Rimodellamento Ventricolare Post - Infartuale

## Quale Trattamento Farmacologico?

*Effetto emodinamico*

*Effetto cardioprotettivo*



*Effetti spesso disgiunti*

# TERAPIA FARMACOLOGICA INSUFFICIENZA CARDIACA

Situazione attuale

Antagonisti recettoriali vasopressina

Antagonisti adrenergici

**Statine**

**Ace-inibitori**

**Beta-bloccanti**

**Diuretici**

**Inibitori Recettori Angiotensina**

**Antialdosteronici**

**Digitale**

Antagonisti dell' endotelina

Antagonisti delle citokine

# Spironolattone: *potenziali meccanismi d'azione*

25 mg/die

**Diuresi e sodiuresi**

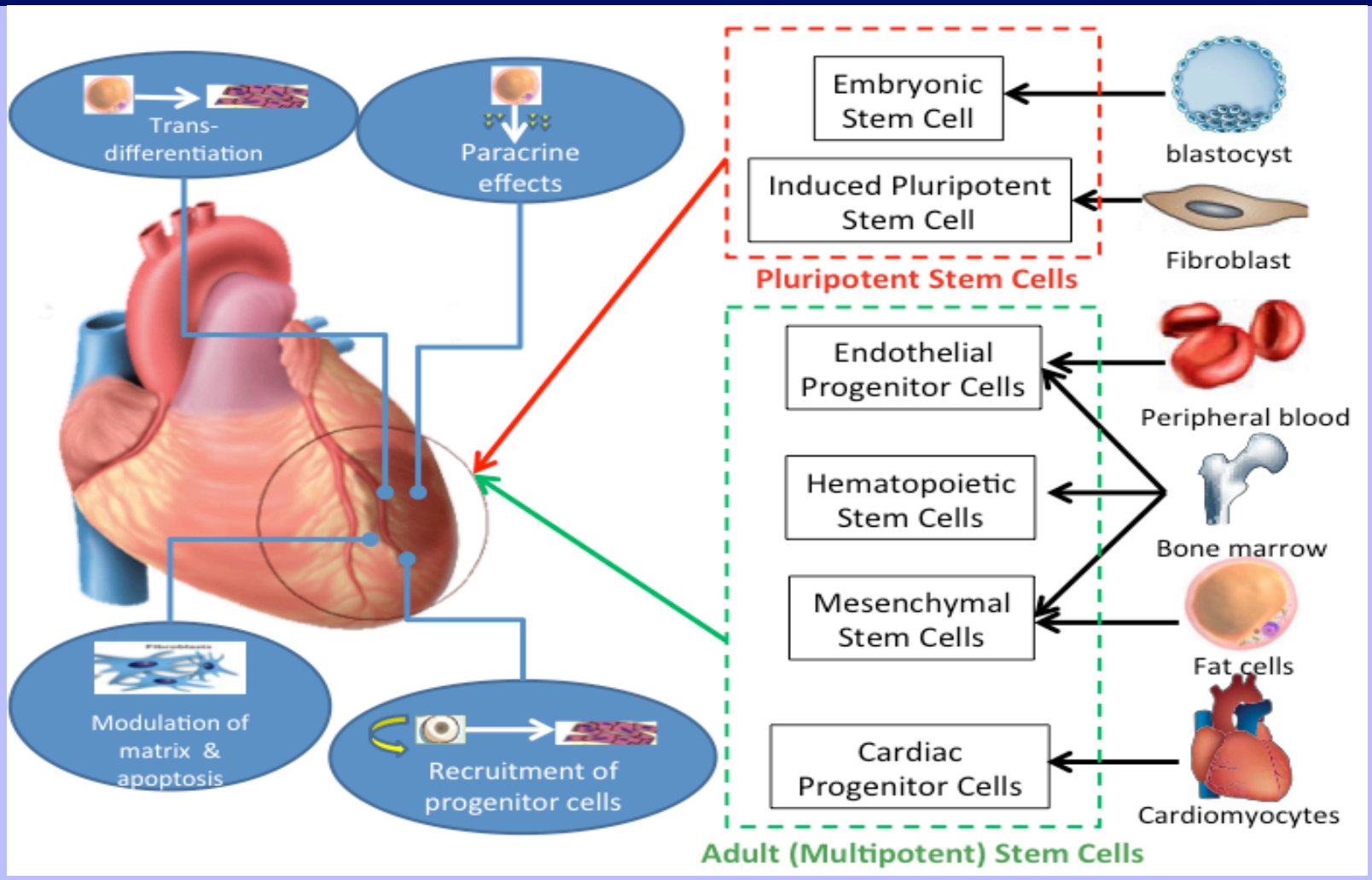
*Effetto emodinamico non significativo (soprattutto se paragonato ai diuretici dell'ansa utilizzati)*

**Blocco effetto aldosterone**
**↓ formazione di collagene**
**↓ fibrosi miocardica e vascolare**
**↓ perdita di potassio**
**↓ rischio morte improvvisa**

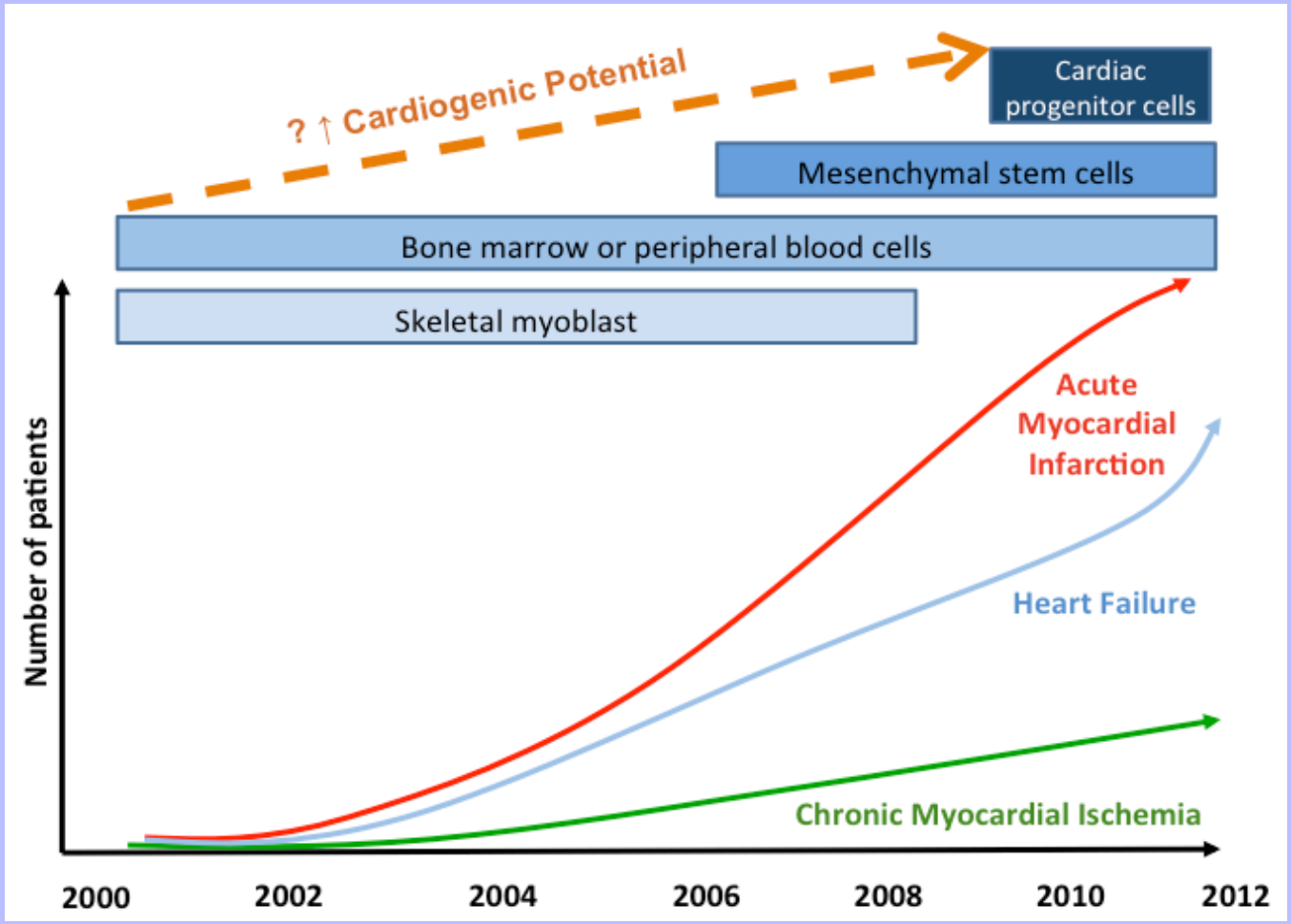
Effetto additivo a quello degli Ace-inibitori

***Weber K.T. N Engl J Med 1999, 341***

# New emerging therapies: cardiac stem cells



# Cardiac stem cells: increasing use in humans




Thank you
for your attention