

EAE TEACHING COURSE 2010

Belgrade, Serbia

October 22-23, 2010

ACUTE MYOCARDIAL INFARCTION

andother causes of

ACUTE CHEST PAIN

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Cardiac

Coronary ArteryDisease (STEMI, NSTEMI, UA) Myocarditis Pericarditis Myopericarditis Cardiomyopathy Valvular disease Tako-Tsubosyndrome

<u>Vascular</u> ←

AorticDissection

AorticAneurysm Pulmonary Embolism

PulmonaryHypertension CerebrovascularDisease

Pulmonary

Pleuritis or Pneumonia Tracheobronchitis Pneumothorax Mediastinitis or Mediastinal Emphysema

Gastrointestinal

Gastritis/Pepticulcer Esophagealdiseases Reflux Spasm Esophagitis Galbladderdisease Pancreatitis

<u>Musculoskeletal</u>

Cervical DiskDisease Arthritisoftheshoulder Costochondritis IntercostalMuscleCramps SubacromialBursitis Rib fracture Herpes zoster

Psychogenic

Chest Pain

Anxietydisorders Panicdisorder Hyperventilation Somatoformdisorders

INITIAL STRATEGY

HISTORY

Chest pain characteristics and associated symptoms

Atypical complaints: younger (25-40 yrs) and older (>75), women, pts with diabetes, chronic renal failure or dementia

PHYSICAL EXAMINATION

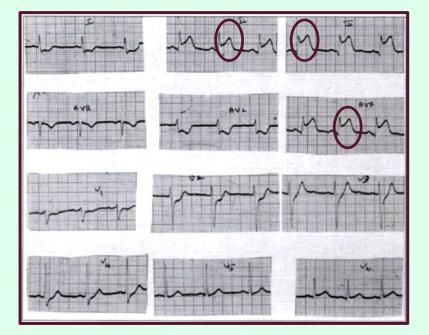
Usually normal HF, hemodynamic instability, new MR murmur _____ high-risk Should target potential non-cardiac causes for the symptoms (prominent murmurs, friction rub, fever, abnormal lung sounds, chest pain after palpation)

ECG

The easiest, simplest, most important tool One of the "vital signs" for pts with chest pain Performed/interpreted within 10 minutes of presentation

INITIAL STRATEGY

ST-segment elevation on initial ECG



Evaluation for immediate reperfusion therapy

An obvious non-cardiac cause for the symptoms



Treatment based on the alternative diagnosis

RECOMMENDATIONS FOR ECHOCARDIOGRAPHY IN PATIENTS WITH CHEST PAIN

Diagnosis of underlying disease in patients with chest pain and
 Clinical evidence of valvular, pericardial, or myocardial disease

Evaluation of chest pain in patients with suspected aortic dissection

Evaluation of chest pain in patients with suspected acute myocardial ischemia, when baseline ECG and other laboratory markers are non-diagnostic and when study can be obtained during pain or within minutes after its abatement

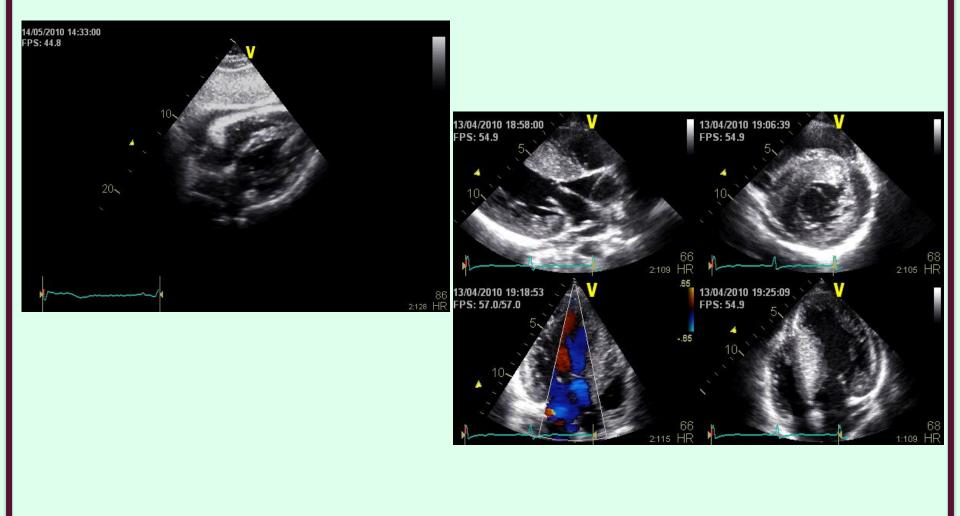
Evaluation of patients with chest pain and hemodynamic instability unresponsive to simple therapeutic measures

 $^{\odot}$ 2003 by the American College of Cardiology Foundation and the American Heart Association, Inc.

ACC/AHA PRACTICE GUIDELINES—FULL TEXT

ACC/AHA/ASE 2003 Guideline Update for the Clinical Application of Echocardiography

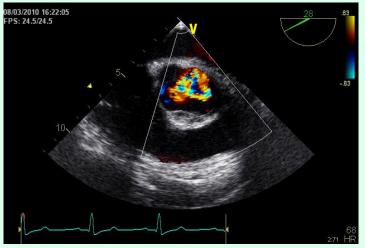
IMAGING To confirm /exclude other diagnosis



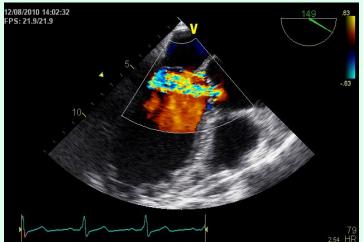
To confirm /exclude other diagnosis

AORTIC PATHOLOGY



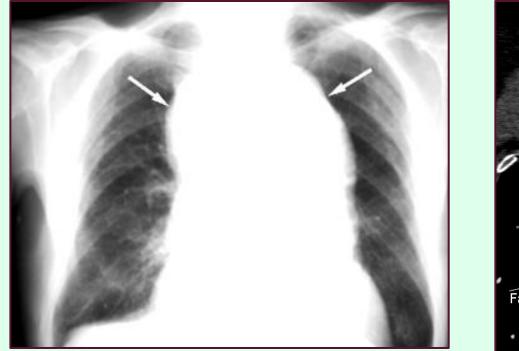






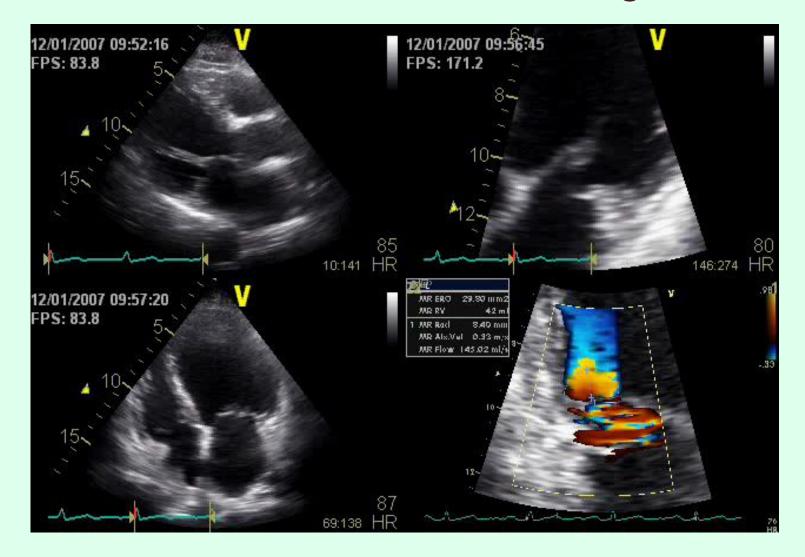
IMAGING To confirm /exclude other diagnosis

AORTIC DISSECTION

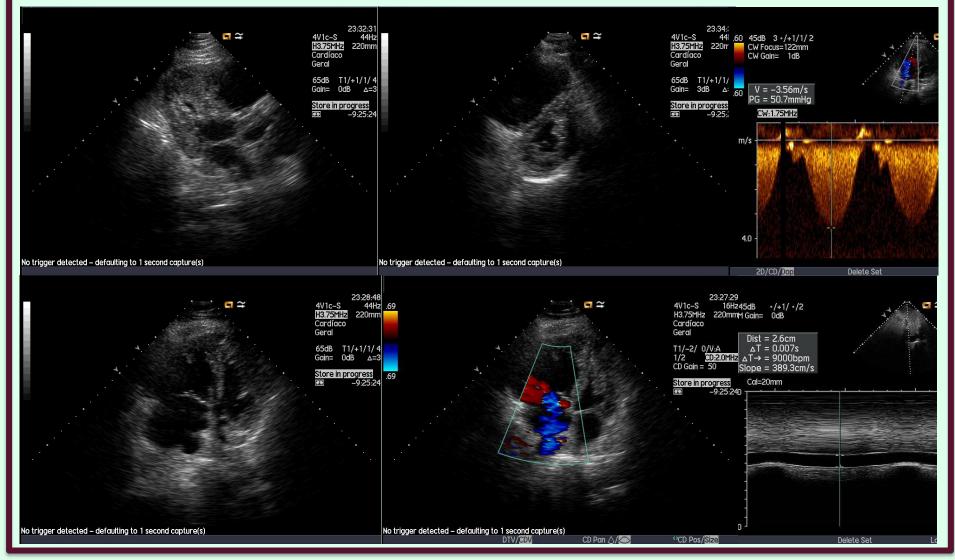




IMAGING To confirm /exclude other diagnosis

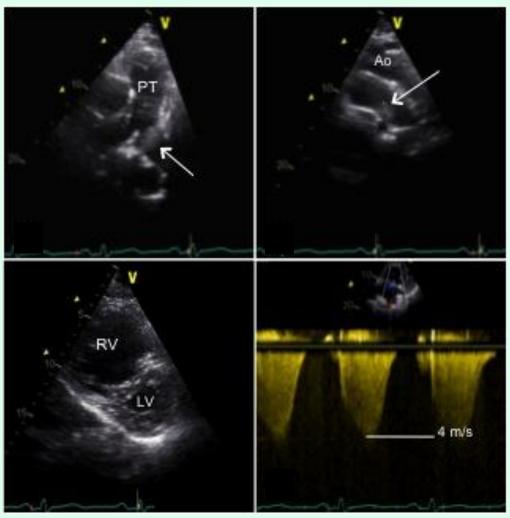


To confirm /exclude other diagnosis



To confirm /exclude other diagnosis

PULMONARY EMBOLISM

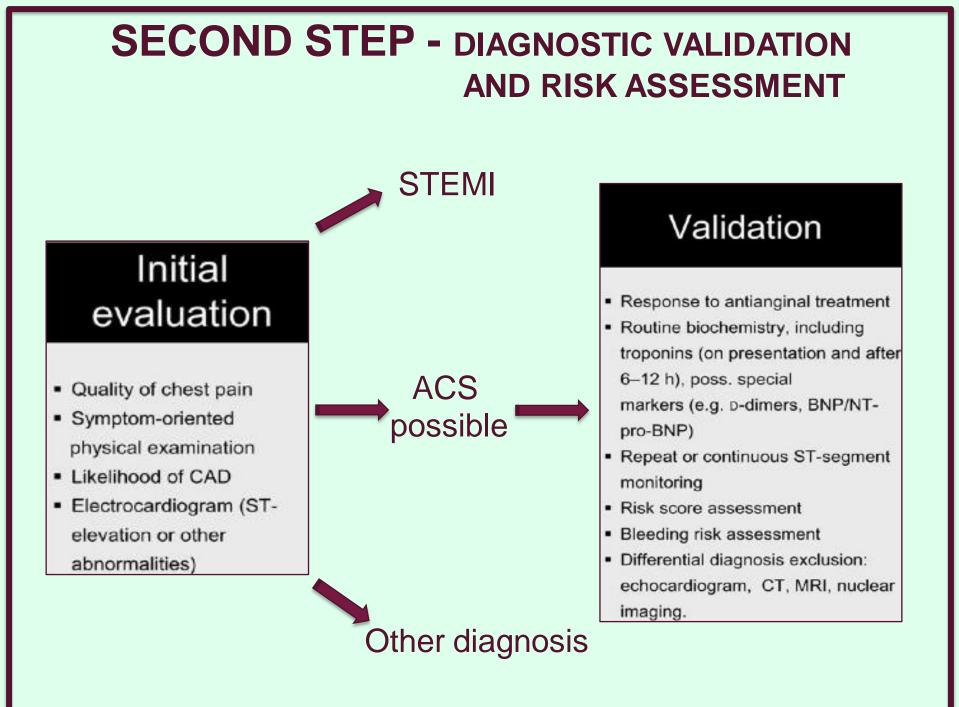


IMAGING - Role of Multislice CT

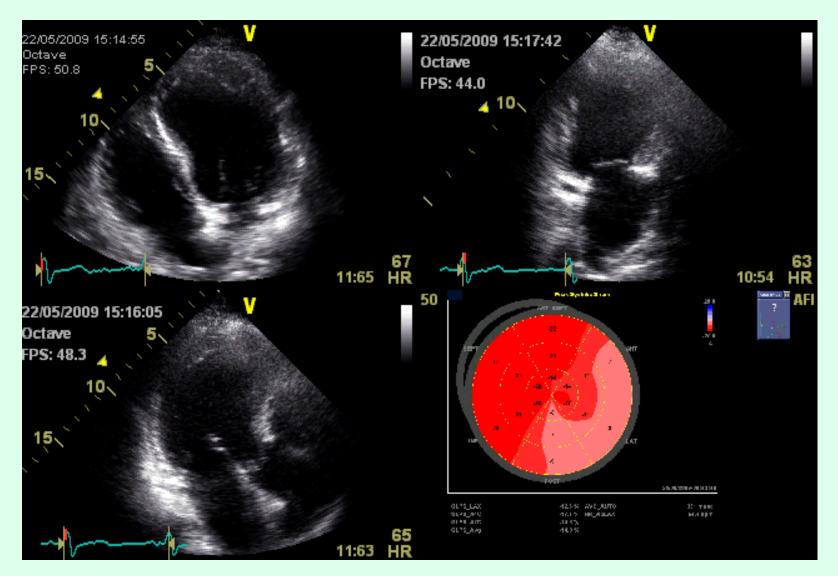
Patients with chest pain in the Emergency Room

PULMONARY EMBOLISM

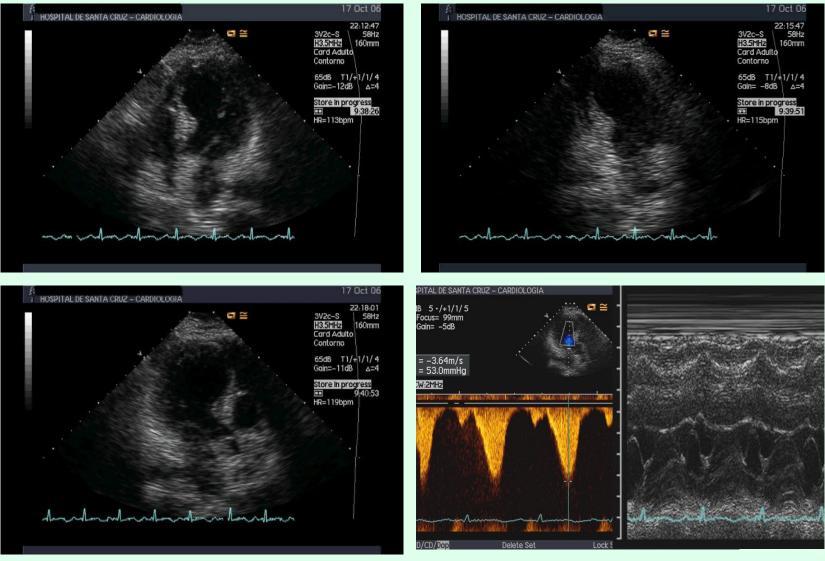




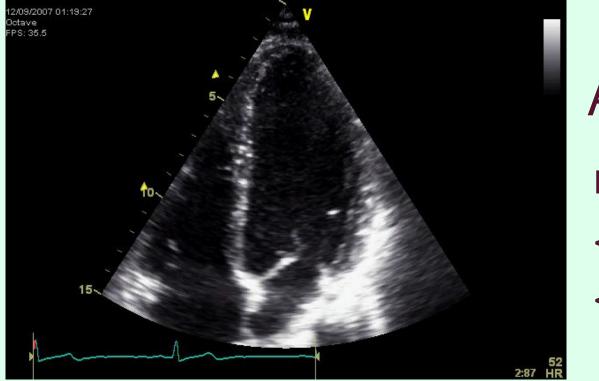
INFARCT-RELATED ARTERY



IMAGING To confirm /exclude other diagnosis



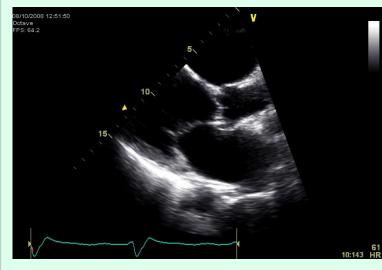
Regional Asynergy (WMA)

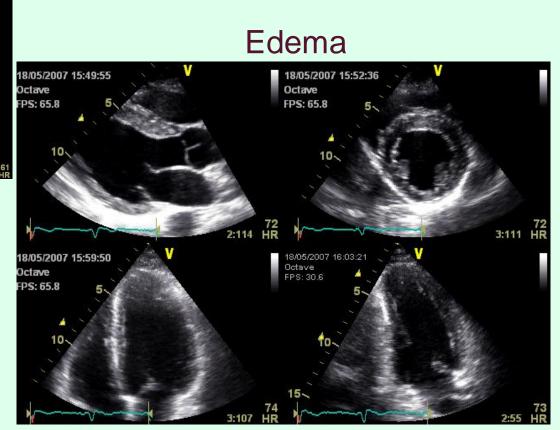


Absent if: Resting CF ↓ <50% < 20% wall thickness < 1-6% LV mass

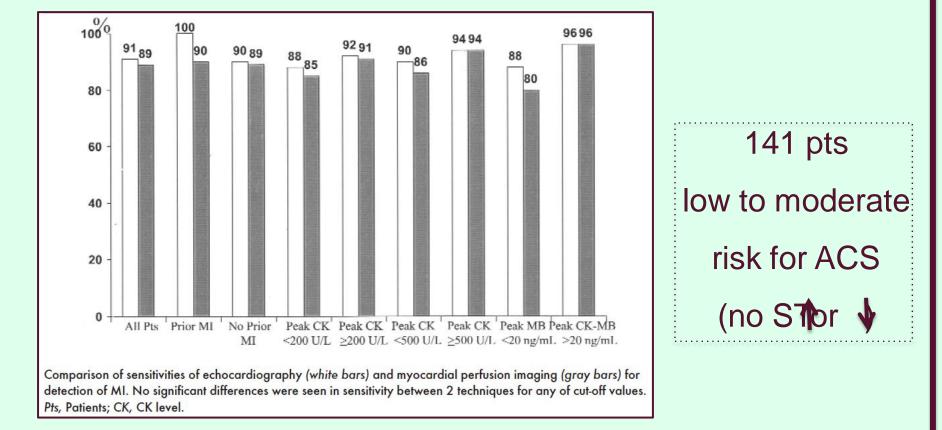
Differentiation from acute and old MI

Fibrosis / Scar



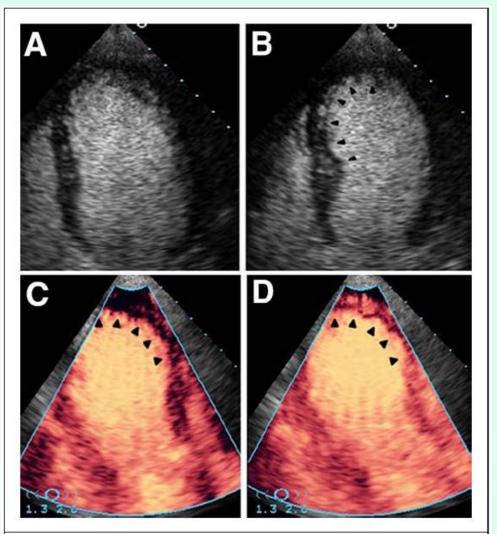


Comparison of 2D Echo and MPI for diagnosing AMI in emergency department patients



Kontos, M et al. Am Heart J 2002;143:659-67

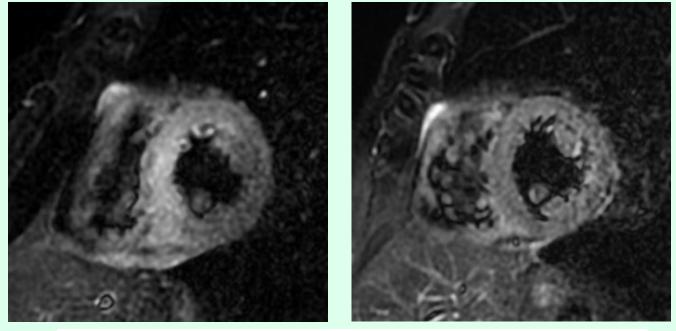
Regional Asynergy (WMA)



Wei K. J Am Coll CardiolImg 2010;3:197-203

Role of MRI

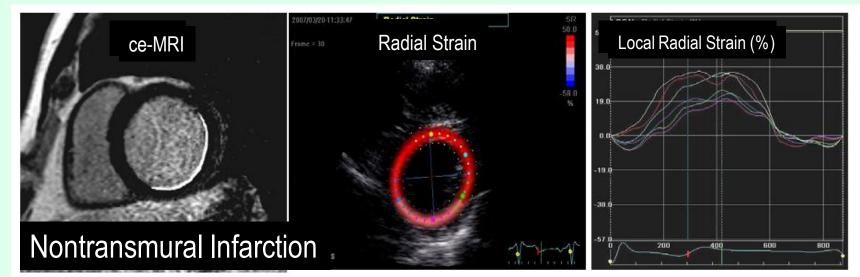
Differentiate Acute from Chronic infarction



Delayed Enhancement and T2-Weighted Cardiovascular Magnetic Resonance Imaging Differentiate Acute From Chronic Myocardial Infarction

> Abdel-Aty H, Zagrosek A, Schulz-Menger J, et al. Circulation 2004;109:2411–16. Cury R, Shash K, Naguerney et al. Circulation 2008;118:837–844.

IMAGING Transmurality of MI



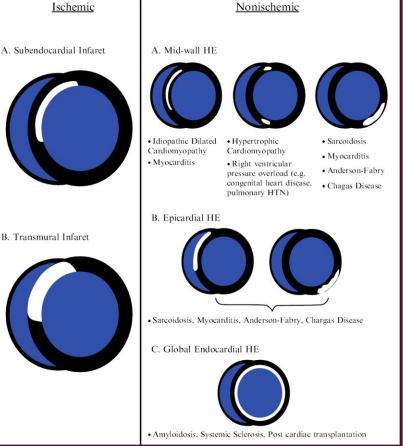


Becker et al, JACC April 2008

Role of MRI

HE Patterns for Ischemic and Non-ischemic Disorders

"wave front phenomenon" of ischemic cell death Early No reperfusion reperfusion Late Infarct reperfusion expansion **Aneurysm Rupture Remodeling HF**



Karamitsos et al. JACC 2009; 54: 1407

BIOMARKERS

All patients with suspected ACS should undergo serial cardiac biomarker sampling

Current recommendations indicate troponin as the preferred biomarker

The diagnosis of NSTE-ACS should never be made only on the basis of cardiac biomarkers, whose elevation should be interpreted in the context of other clinical findings

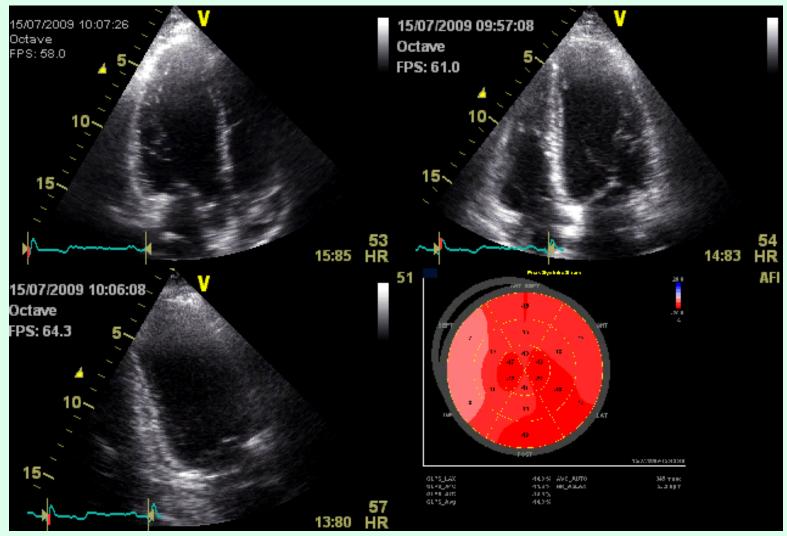
Tn (+) INCIDENC		E CLINICALSIGNIFICANCE	
Acute PE 1	32%	↑ Riskofin-hospitalmortality	
AcutePericarditis ₂	22%	correlates with recent infection	
HF Severebutstable	≤ 15%	Independent predictor of long-term	
Acute, left3,4,5	≤55%	survival or readmission for HF	
Myocarditis ₆	≥34%	Recent onset of HF	
Sepsis6,7	≤85%	TRisk of in-hospital mortality	
Renal failure8	≤20%	Poor long-term outcome	
1. Giannitis E et al. <i>Circulation</i> 20 2. Bonnefoy E et al. <i>Eur Heart J 2</i> 3. La Vecchia L et al. <i>Am J Card</i> 4. Cummins B et al. <i>Am Heart J</i>	00; 102: 211 2000; 21: 832 io/1997; 80: 88	5. Missov E et al. <i>Circulation</i> 1997; 96: 2953 6. Arlati S et al. <i>Intensive Care Med</i> 2000; 26: 31 7.ElstKM et al. <i>ClinChem</i> 2000; 46: 650 8. Khan NA et al. <i>Circulation</i> 2005; 112: 3088	

PARAMETERS USED FOR ASSESSMENT OF PROGNOSIS AFTER ACUTE MI

LV EF \rightarrow *mortality*

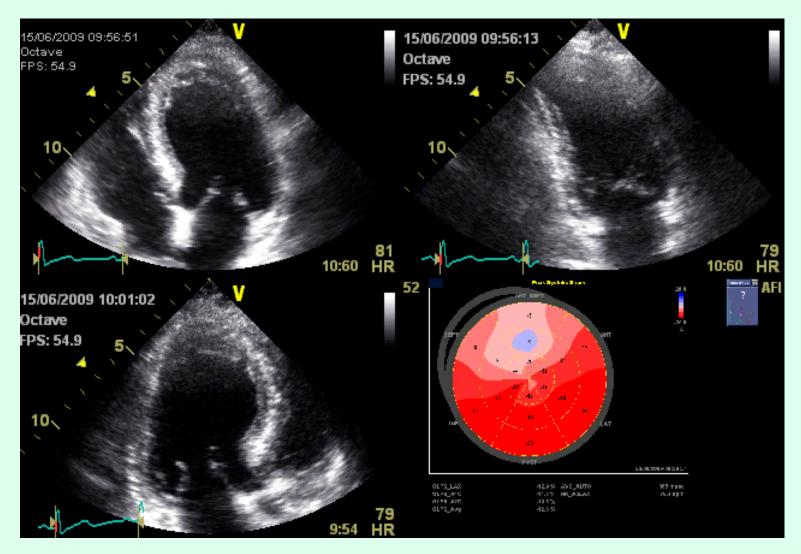
Large asynergic zone (high WMSi) \rightarrow shock, HF **Infarct expansion** \rightarrow LV remodelling, aneurysm **End-systolic volume** *mortality* **Short DT** \rightarrow *LV remodelling, mortality* **E/e' ratio** \rightarrow cardiac events, mortality **Stress Echo**→ cardiac events, mortality **Viability** \rightarrow LV dilation, mortality **Remote dyssynergy** \rightarrow *multivessel CAD* Mitral regurgitation → heart failure, *mortality* **Left atrial enlargement** \rightarrow *LV remodelling, aneurysm* **Right ventricular dysfunction** \rightarrow *mortality*

INFARCT-RELATED ARTERY



The LV global strain shows a good correlation with LV infarct size

INFARCT-RELATED ARTERY



The LV global strain shows a good correlation with LV infarct size

PREDICTORS OF LV REMODELING AFTER AMI Pre-existing hypertension Infarct size Large asynergic zone (high WMSi) Cardiac enzyme indexEjection Fraction Anterior location Infarct expansion Transmurality of infarction Mostly Short early mitral DT interrelated End-systolic volume Patency of the IRA TIMI grade <3 flow risk factors **Microvascular obstruction** Mitral regurgitation Left atrial enlargement Viability

Markers of neurohormonal activation

LV Dyssynchrony acutely after MI Predicts LV Remodeling

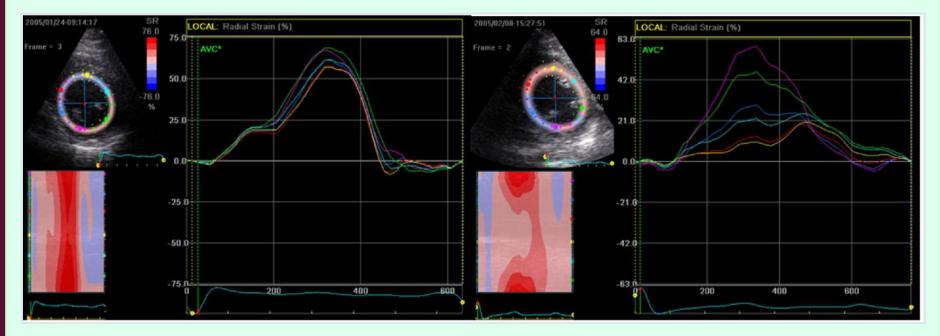
•178 pts; primary PCI

•2D-echo + radial strain analysis (speckle-tracking) within 48h + 6 mo f-up

•LV Remodeling: >15% increase of LVESV

		No LV Remodeling ($n = 2$	142) LV Remodeling (n = 36)	p Value
Peak cTnT level, μ g/l, (25th, 75th	percentiles)	5.2 (1.9, 9.8)	10.1 (6.3, 15.3)	<0.001
Peak CPK level, U/I (25th, 75th percentiles)		1,893 (868, 3,236)	3,877 (1,816, 5,597)	<0.00
	No LV Remo	odeling (n = 142)	LV Remodeling (n = 36)	p Value
Baseline				
LVESV, ml	64 (54, 70)		76 (54, 91)	NS
LVEDV, ml	128 (106, 148)		139 (108, 160)	NS
LVEF, %	47 (42, 52)		47 (42, 51)	NS
WMSI	1.50 (1.25, 1.63)		1.56 (1.38, 1.69)	< 0.05
LA dimension, mm	38 (34, 42)		41 (37, 43)	NS
E/E' ratio	11.7 (9.7, 15.7)		14.8 (12.3, 18.4)	< 0.05
MR, moderate-severe (%)	6 (4)		2 (6)	NS
LV dyssynchrony, ms	31 (12, 77)		148 (134, 180)	< 0.001

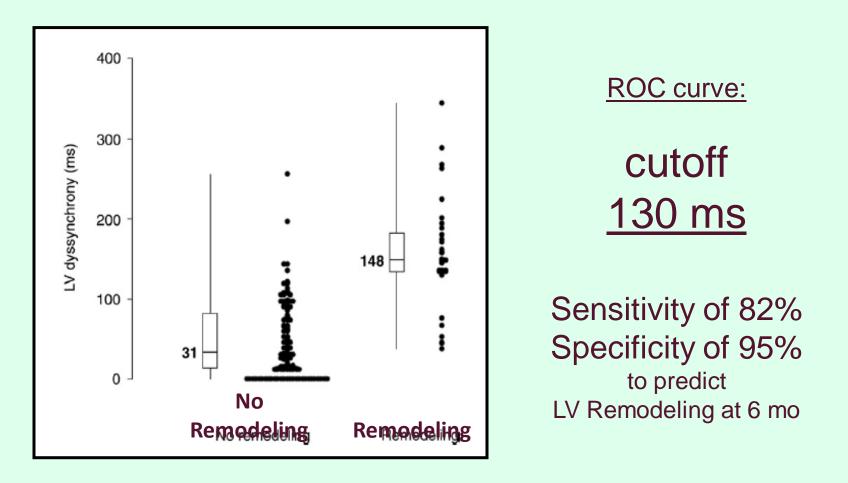
LV Dyssynchrony acutely after MI predicts LV remodeling



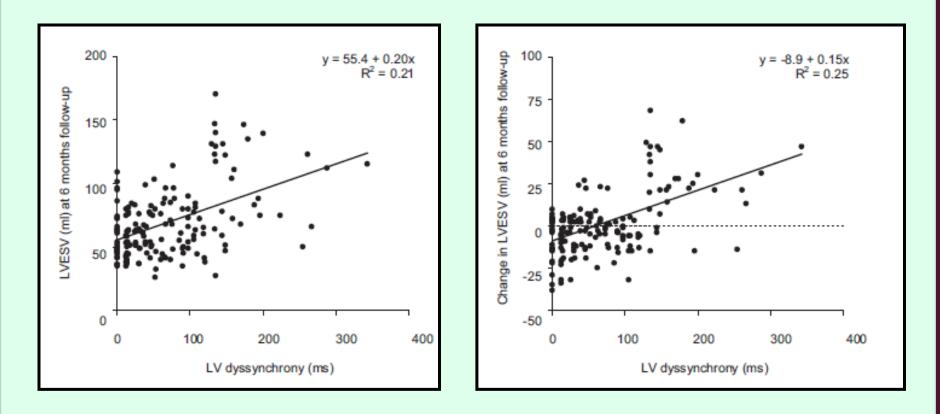
No dyssynchrony at baseline No remodeling at follow-up

Dyssynchrony at baseline Remodeling at follow-up

EXTENT OF LV DYSSYNCHRONY WAS SIGNIFICANTLY LARGER IN PATIENTS WITH LV REMODELING DURING FOLLOW-UP VERSUS THOSE WITHOUT LV REMODELING



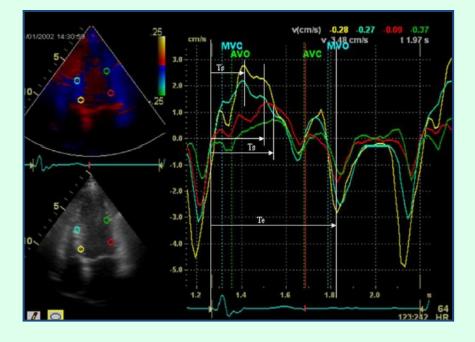
CORRELATION BETWEEN LV DYSSYNCHRONY AT BASELINE AND LVESV AND CHANGE IN LVESV AT 6-MONTH F-UP

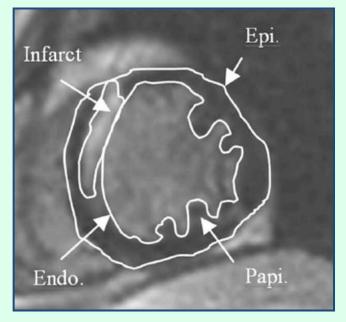


Resynchronization therapy??-

Further studies needed !!!

LV systolic dyssynchrony predicts cardiac remodeling after MI



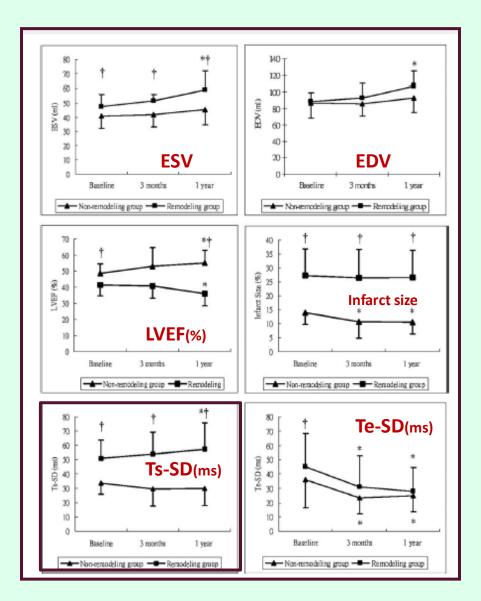


- •47 pts; 35 with early PCI
- •2D-echo + TDI for dyssynchrony

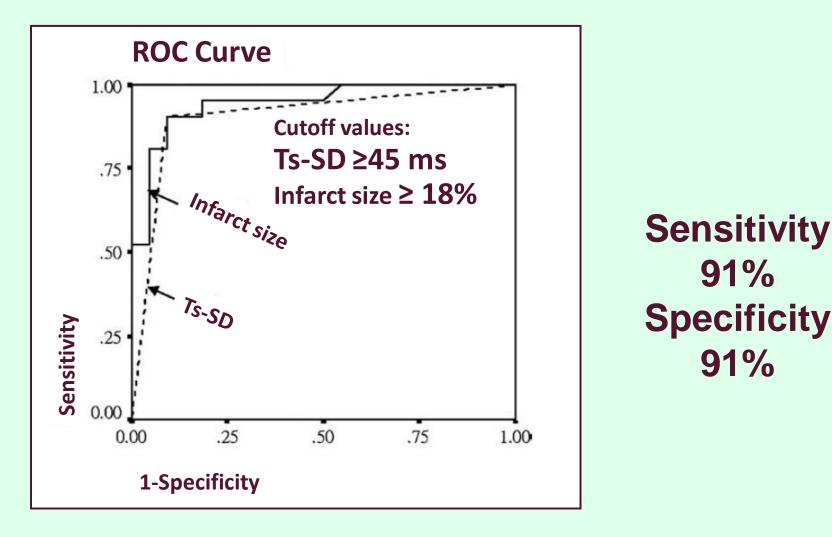
2-6d, 3 mo, 12 m after MI

- •Ce-MRI for infarct size
- •LV Remodeling: >10% increase of LVESV

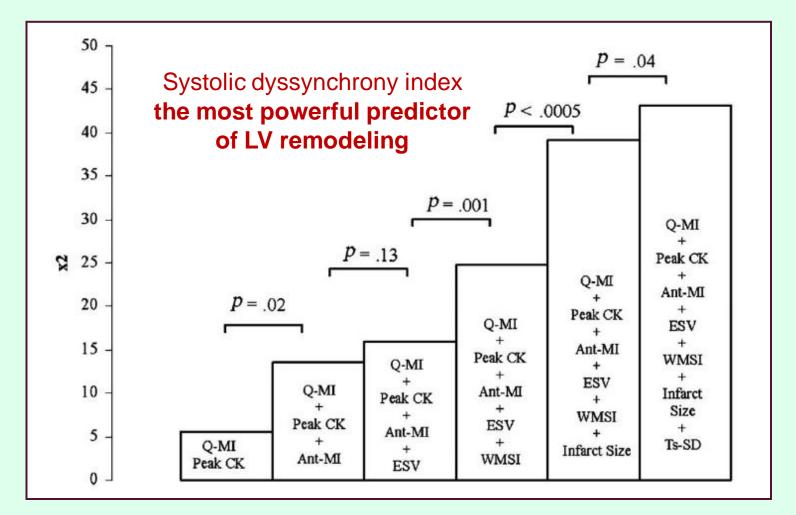
Serial changes of ESV, EDV, LVEF, Ts –SD and Te-SD in the remodeling and non-remodeling groups



PREDICTIVE VALUE OF SYSTOLIC DYSSYNCHRONY BY TDI AND INFARCT SIZE BY CE-MRI



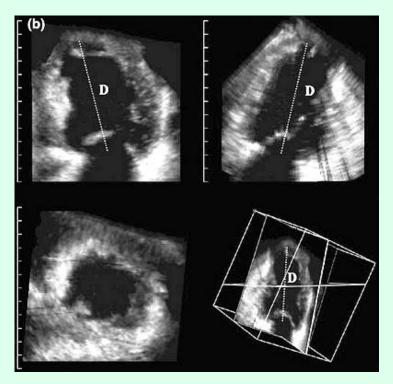
INCREMENTAL PREDICTIVE VALUE OF INFARCT SIZE BY CE-MRI AND SYSTOLIC DYSSYNCHRONY BY TDI



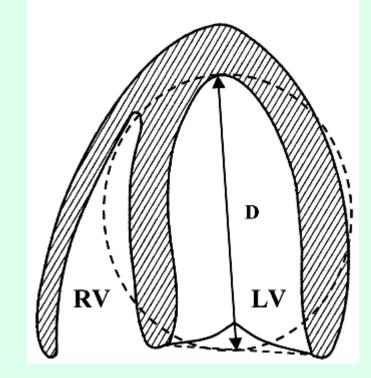
Early Identification of LV Remodeling After MI by 3D Echo

• 33 pts

Serial 3D-echo: 6d, 3mo, 6mo, 12mo post MI
LV Remodelling: >20% increase of EDV

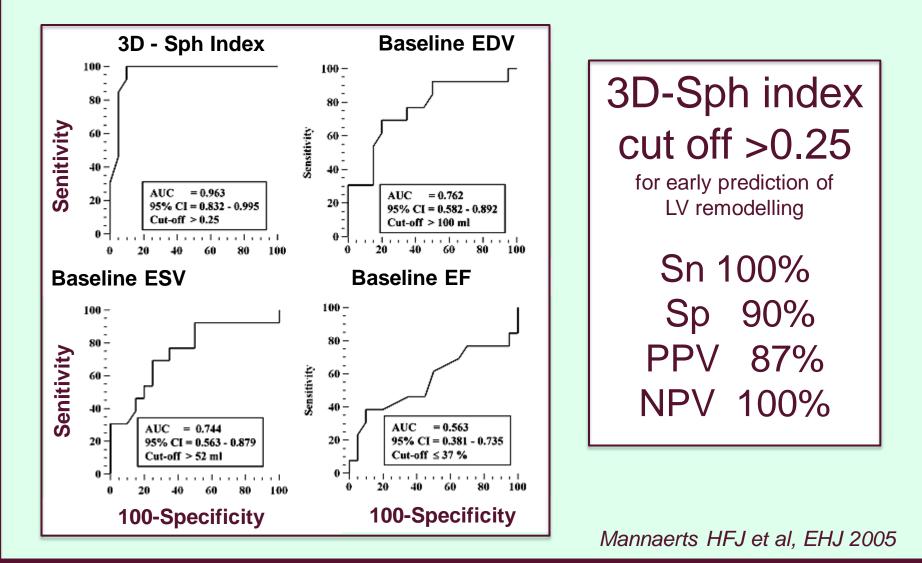


3D Sphericity Index EDV/(4/3 x π x (D/2)³)



Mannaerts HFJ et al, EHJ 2005

VALUE OF 3D SPHERICITY INDEX FOR EARLYPREDICTION OF POST MI LV REMODELING

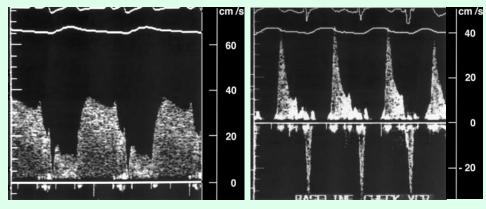


CORONARY FLOW PATTERN OF INFARCT-RELATED ARTERY

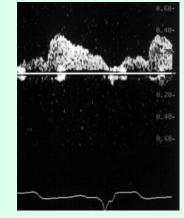
Markers of "no-reflow"

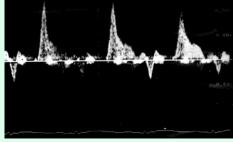
Rapid deceleration of diastolic flow and systolic retrograde flow

CathLab



TT Doppler

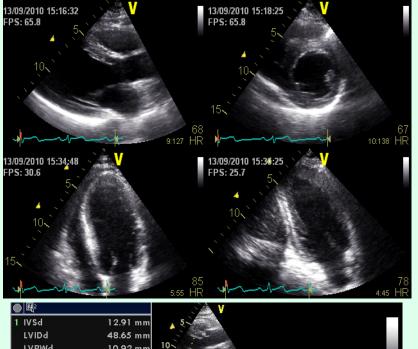


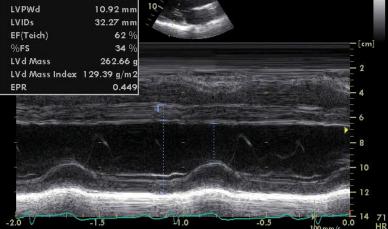


T Kawamotoetal. *Circulation1999;100:339-45* Tani T etal. CardiovasvularUltrasound 2005; 3:22

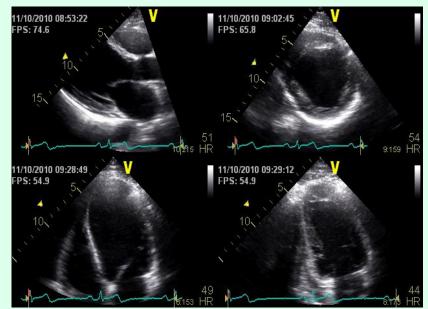
CLINICAL CASE

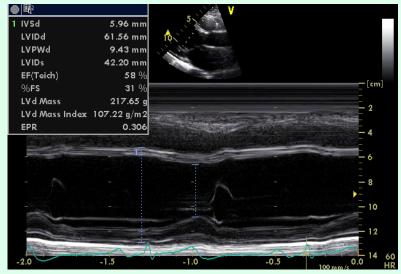
ACUTE





1 MONTHF-UP



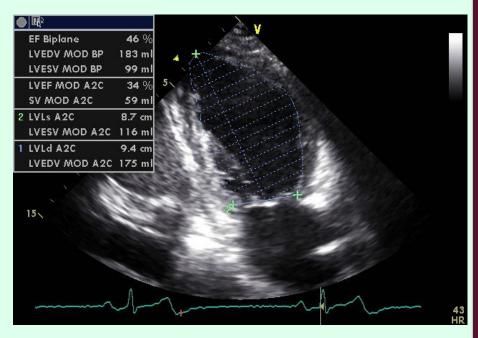


CLINICAL CASE

ACUTE

EF Biplane 40 % LVEDV MOD BP 101 ml LVESV MOD BP 60 ml LVEF MOD A2C 40 % 5 SV MOD A2C 41 ml 2 LVLs A2C 8.9 cm LVESV MOD A2C 61 ml 1 LVLd A2C 9.3 cm LVEDV MOD A2C 102 ml 15 84 HR

1 MONTHF-UP



CLINICAL CASE

ACUTE

1 MONTHF-UP

