Quantifying myocardial function in valvular heart disease

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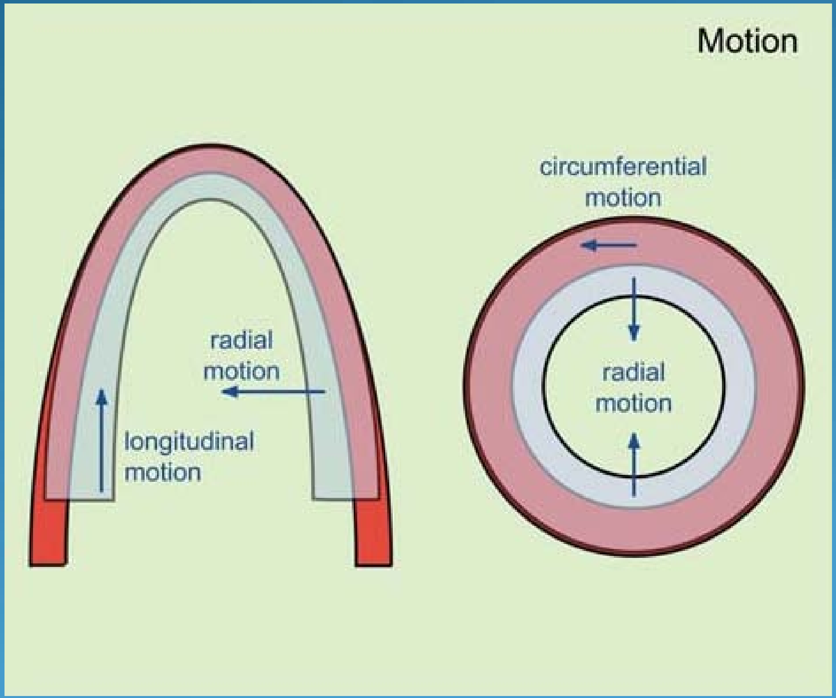
The heart under pathologic conditions

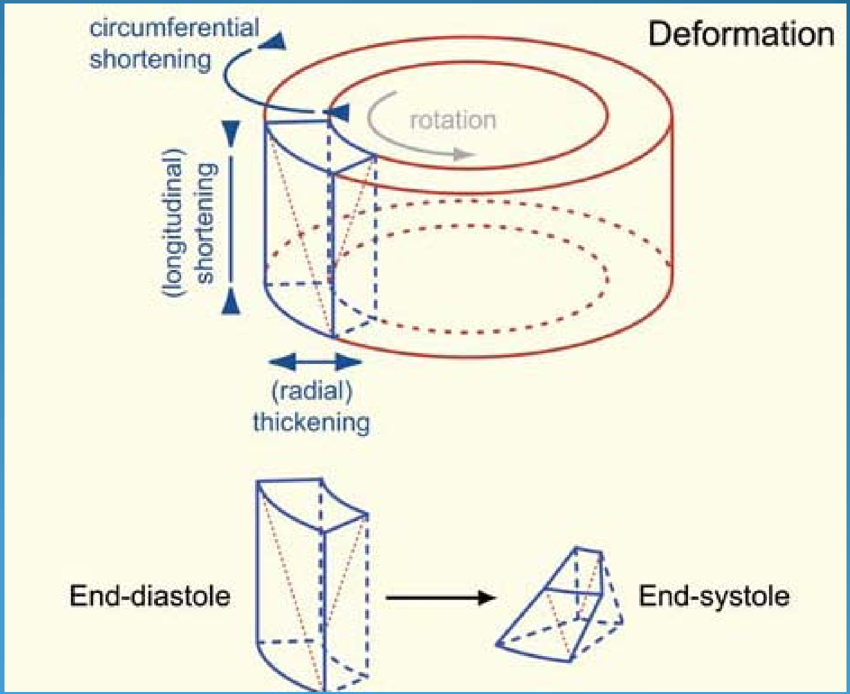
- The heart is a volume pump
- Remodelling process to maintain SV in cases of:
  - Change of loading conditions
  - Local changes in geometry
  - Dyssynchrony
  - Reduced contractility
- ↓ SV ⇨ HF symptoms

Global indices EF and FS in evaluation of myocardial function

- Reflect contractility
- Limitations:
  - Depend on geometric assumptions
  - Load-dependent
  - Assess global function without taking into account segment influence

Quantification of motion and deformation by TDI and STE

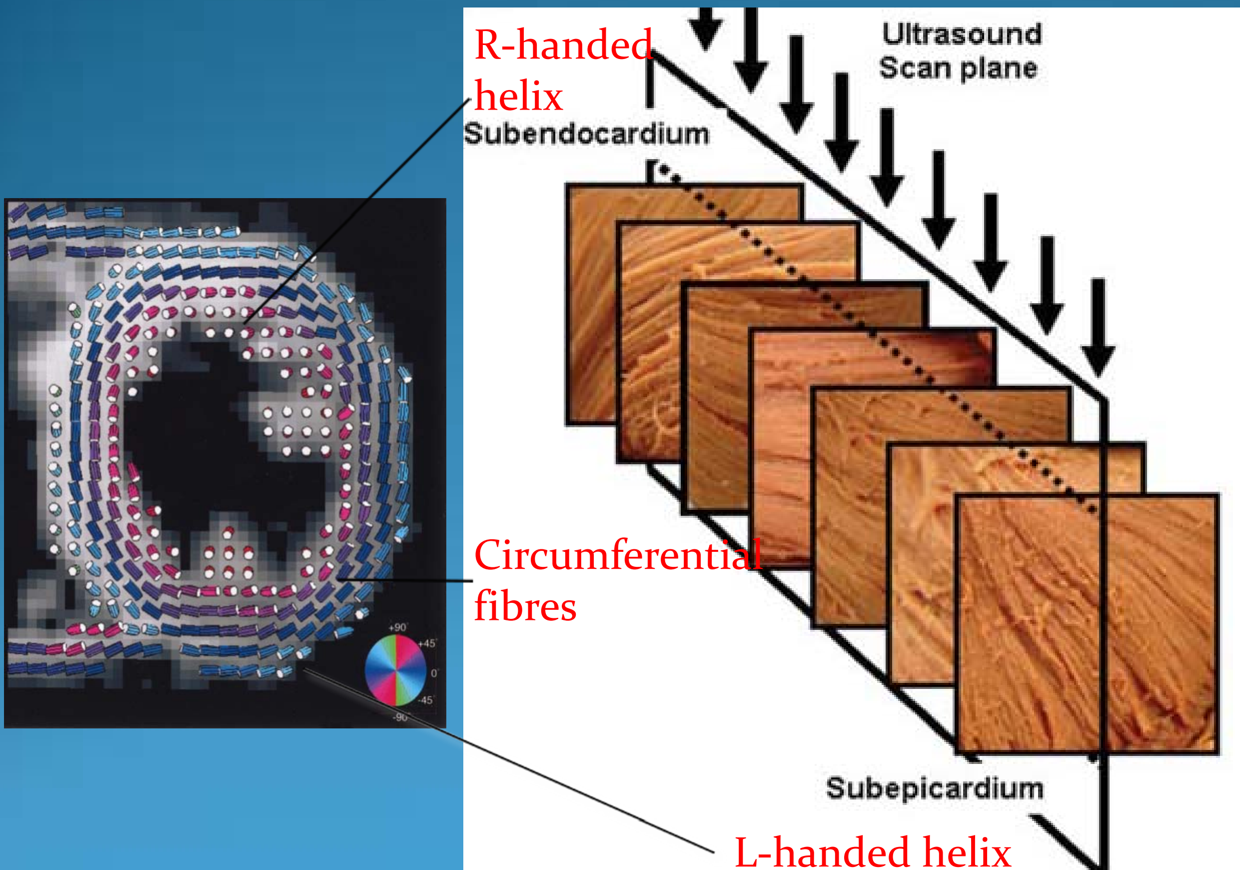




Bijnens B et al. Eur J Echocardiogr 2009;10:216-226.

LV architecture and function

The three strain components are interrelated and cannot be decomposed into different layer functions



Longitudinal mechanics

Radial mechanics

Circumferential mechanics

Mor-Avi et al. Eur J Echocardiogr 2011.

LV mechanics

Longitudinal deformation

Circumferential deformation (shortening)

Radial deformation (wall thickening)

15% shortening of myocytes

>40% radial wall thickening

>60% change in EF

Shear deformation

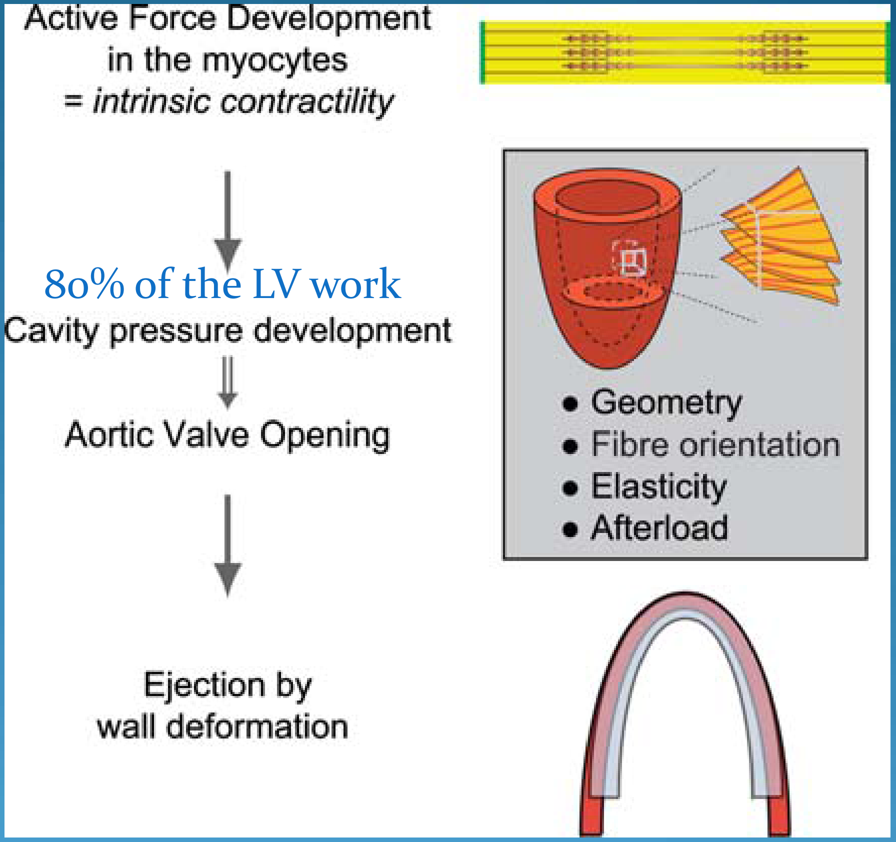
Principle of conservation of mass:

Shortening in longitudinal and circumferential direction ⇨ thickening in the radial direction

LV function

Circumferential fibres (80%) ⇨ isometric work (IVC)

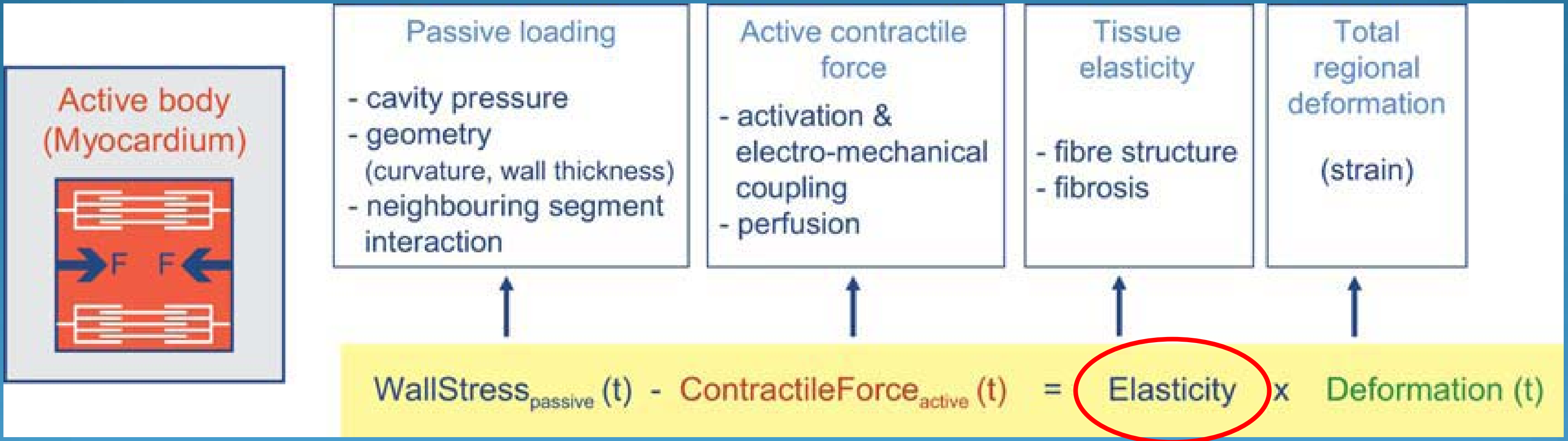
Longitudinal and circumferential fibres ⇨ isotonic work



- Deformation analysis – measures the fraction of the ejection work, but not isometric work of the heart
- The full description of LV work needs to incorporate the measure of load in combination with mathematical models

Bijnens B et al. Eur J Echocardiogr 2009;10:216.

Relation between intrinsic function (contractility) and deformation



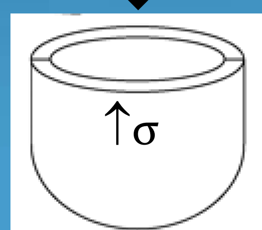
Bijnens B et al. Eur J Echocardiogr 2009;10:216-226.

LV remodelling

Volume overload
MR

- LV dilatation
- ↑ Stroke Volume

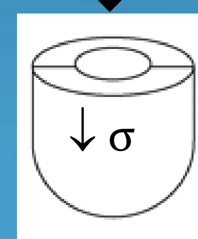
Difficulties to cope with high P



$$\sigma = P \times r / 2h$$

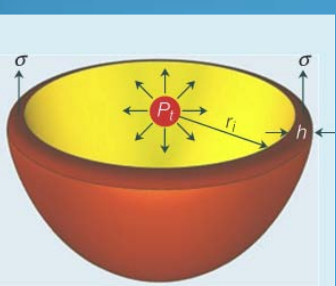
Pressure overload
AS

- LV hypertrophy
- Same Stroke Volume

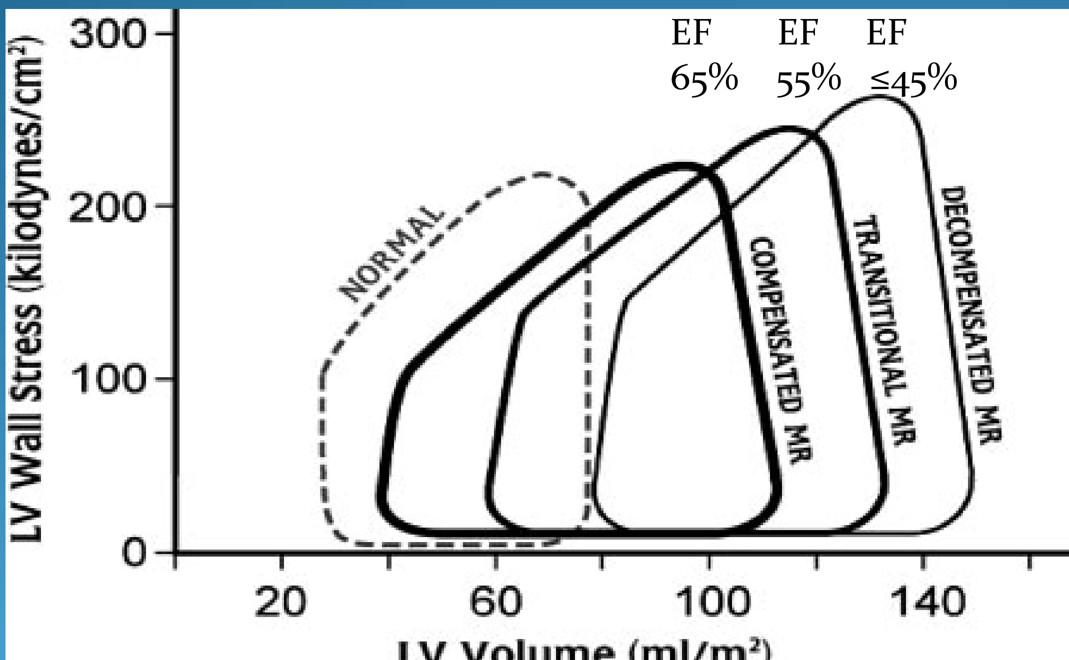


Copes with high P

$$\sigma = P \times r / 2h$$



Hemodynamic stages in chronic MR



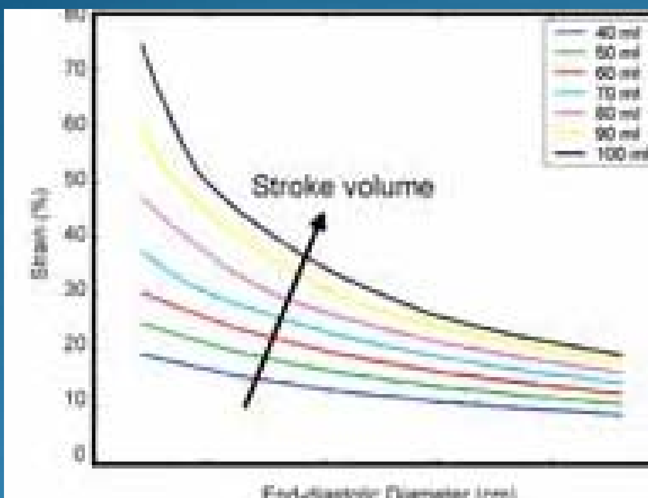
1. ↑ EDV, ↑ SV
normal preload, afterload, contractility, EF

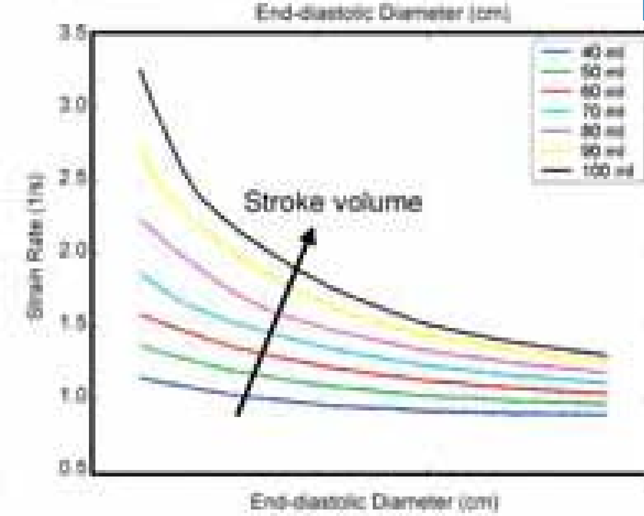
2. ↓ contractility,
↑afterload

3. ↑↑ LV dilation,
↑ diastolic P,
↑ systolic wall stress,
irreversible LV dysfunction

Gaasch W and Meyer T. Circulation 2008;118:2298.

Deformation / geometry in MR





Constant contractility

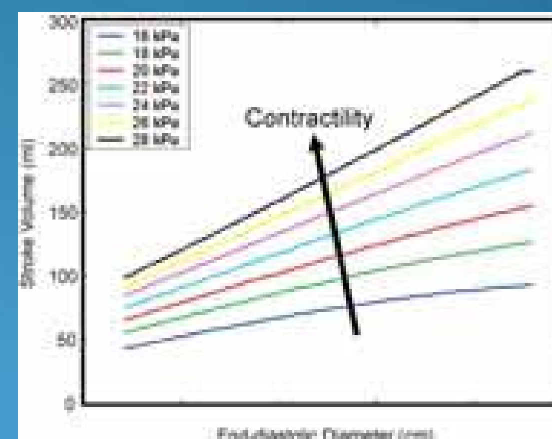
S and SR:

- ↓ with ↑ EDD (certain SV)
- ↑ with ↑ SV (certain LV dimensions)

⇨ ↑ in deformation with ↑ SV is compensated by the ↓ due to ↑ EDD

Reduced contractility ⇨

↓ S and SR

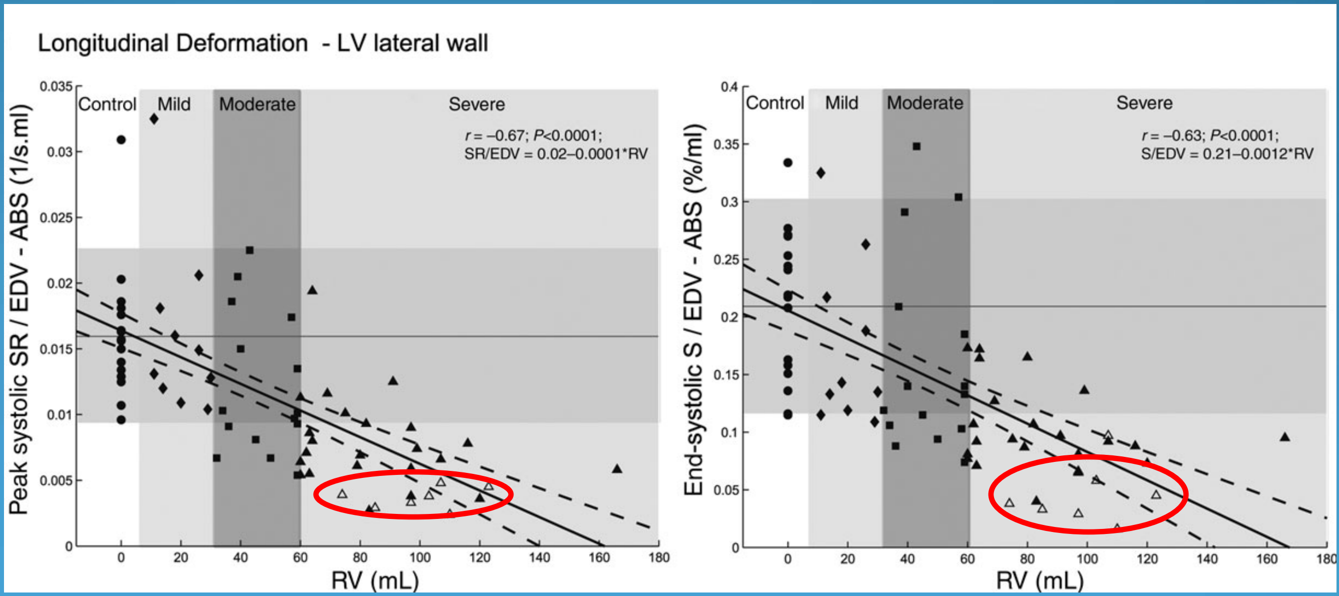


Marciniac A et al. Eur Heart J 2007;28:2627.

Geometry compensated deformation indices – Strain/EDV, SR/EDV

- TDI: 54 asymptomatic patients
- Mild, moderate, severe non-ischemic MR
- Strain/EDV and SR/EDV were significantly reduced in patients with severe MR and with ESD ≥ 4.5 cm – distinguish reduced contractility

● - controls
♦ - mild MR
■ - moderate MR
▲- severe MR (ESD<4.5 cm)
△ - severe MR (ESD ≥4.5 cm)



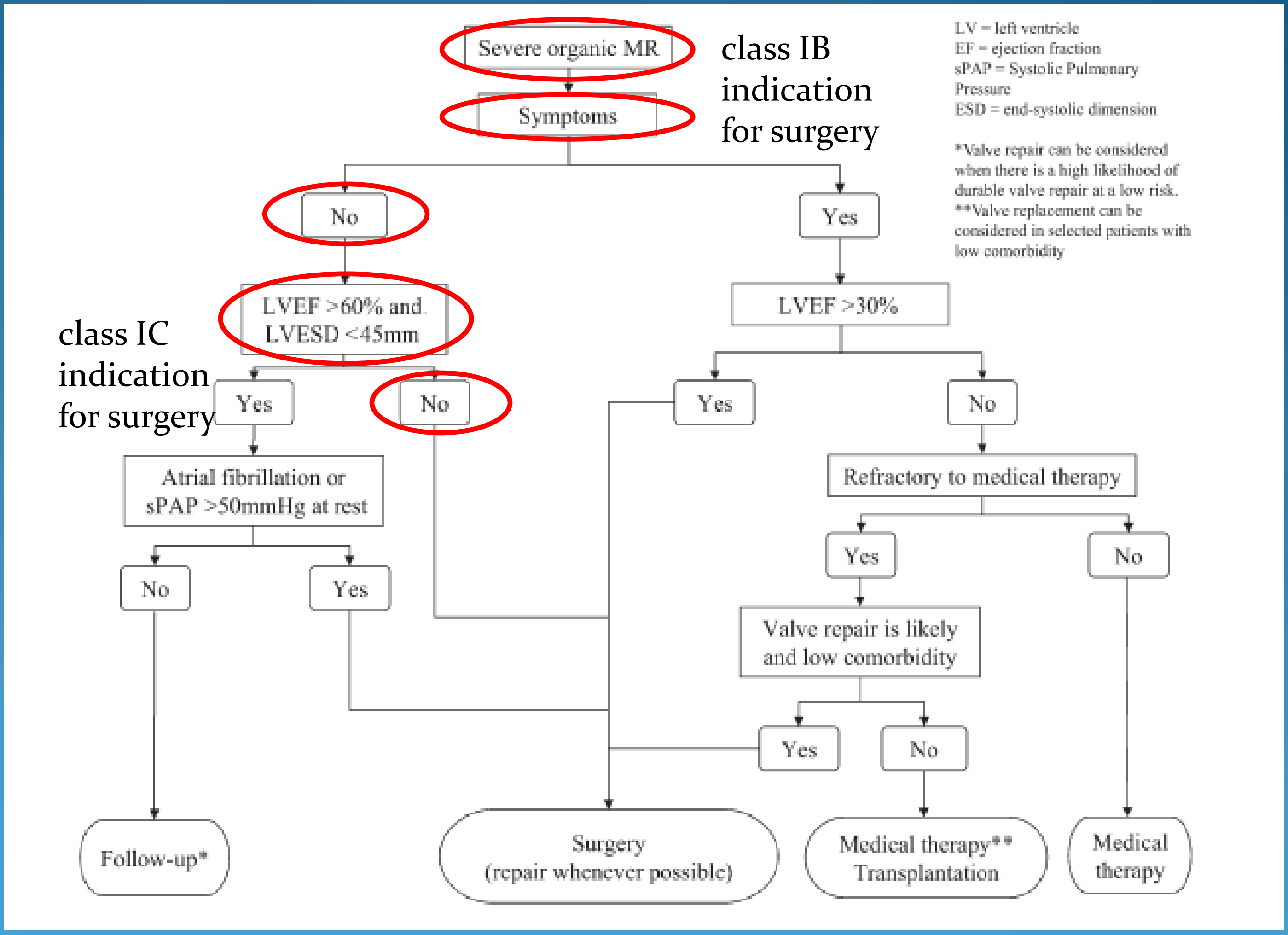
Marciniac A et al. Eur Heart J 2007;28:2627.

ESC Guidelines

Asymptomatic patients
+
Preserved LV function

Optimal timing of surgery?

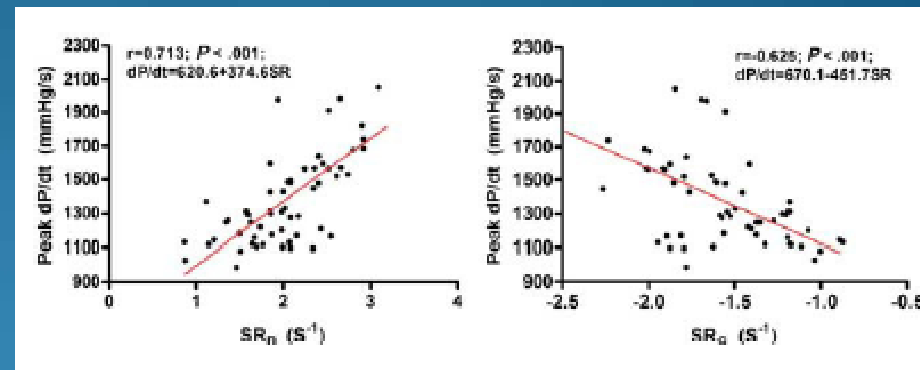
Major challenge - to detect early contractile dysfunction to prevent irreversible changes



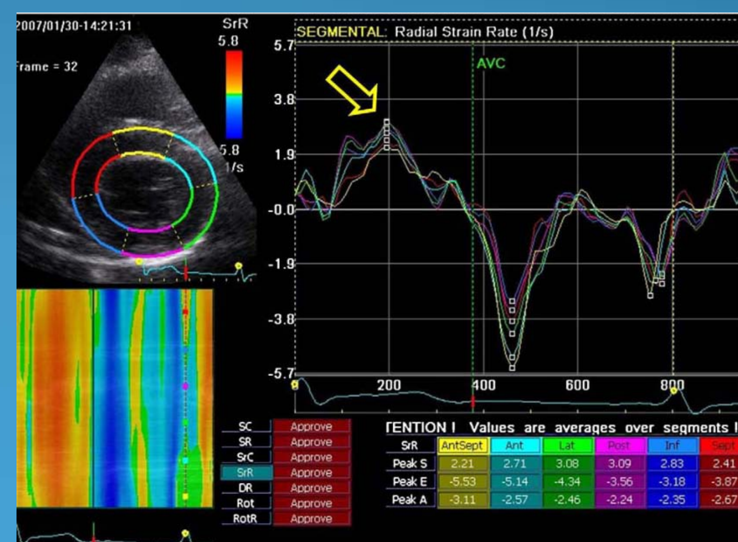
Guidelines on the management of valvular heart disease. Eur Heart J 2007.

Long-axis function is depressed earlier in chronic severe MR

- STE in 59 pts with EF ≥50%; 34 contr.
  - Group 1 - N contractile function: dP/dt ≥1300 mmHg (invasive)
  - Group 2 - contractile dysfunction: dP/dt <1300 mmHg
- ↓ SR_L in all pts
  - no correlation with dP/dt
- ↓ SR_R and SRc in patients with contractile dysfunction
  - correlation with dP/dt



SR_R >2.0 /s predicts dP/dt ≥1300 mmHg (sens. 77%, spec. 73%)

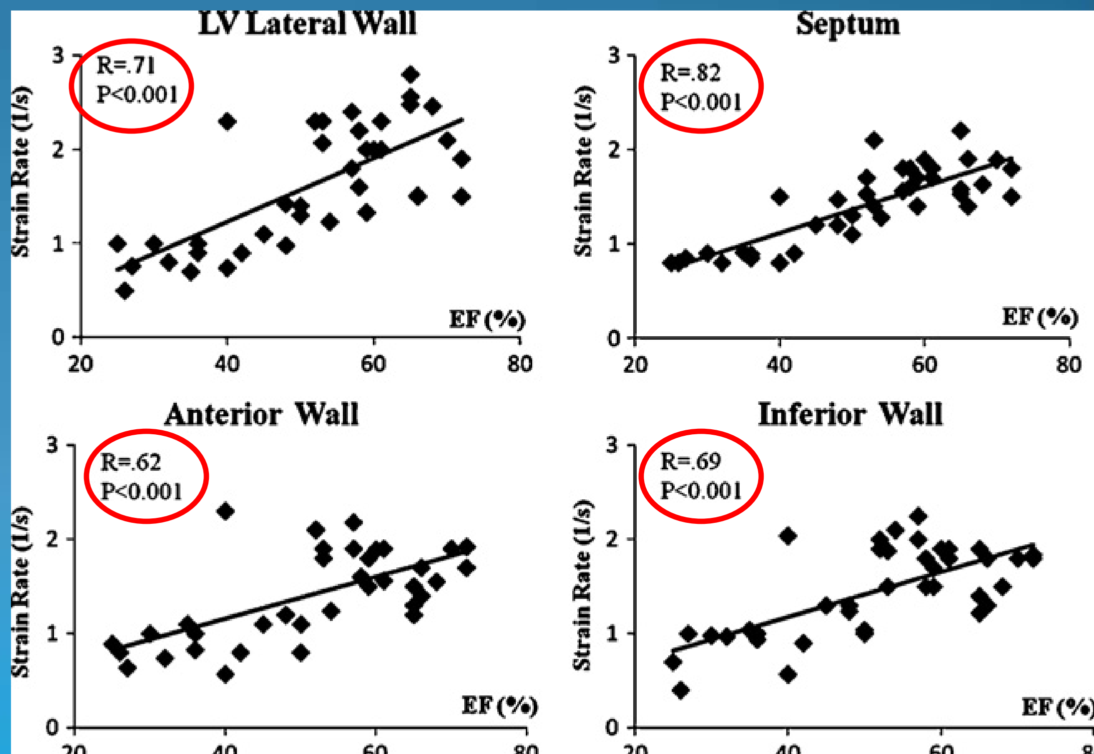


Kim et al. Am Heart J 2009;157:345.

Low pre-op SR predicts decline in EF after MV repair

- TDI: 62 patients
- MV repair
- EF post-op at 12 months
  - Group 1: >50% and
  - Group 2: ≤ 50%:
    - 2A - NYHA I-II
    - 2B - NYHA III-IV
- EF ↓ in all pts right after MV repair but:
  - Group 1 – EF improved at 12 m
  - Group 2A and 2B - EF ↓ more at 12 m
- Positive correlation

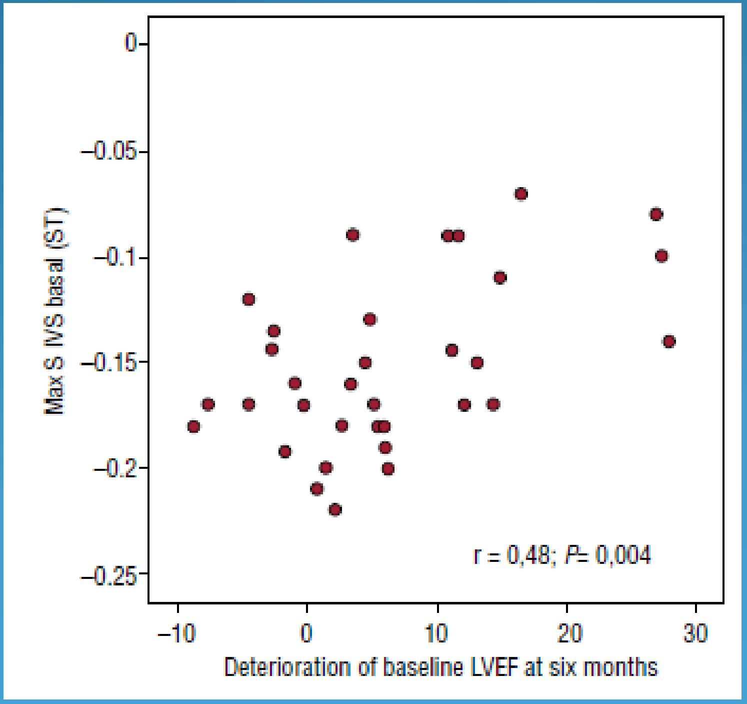
pre-op SR / post-op EF (12 m)

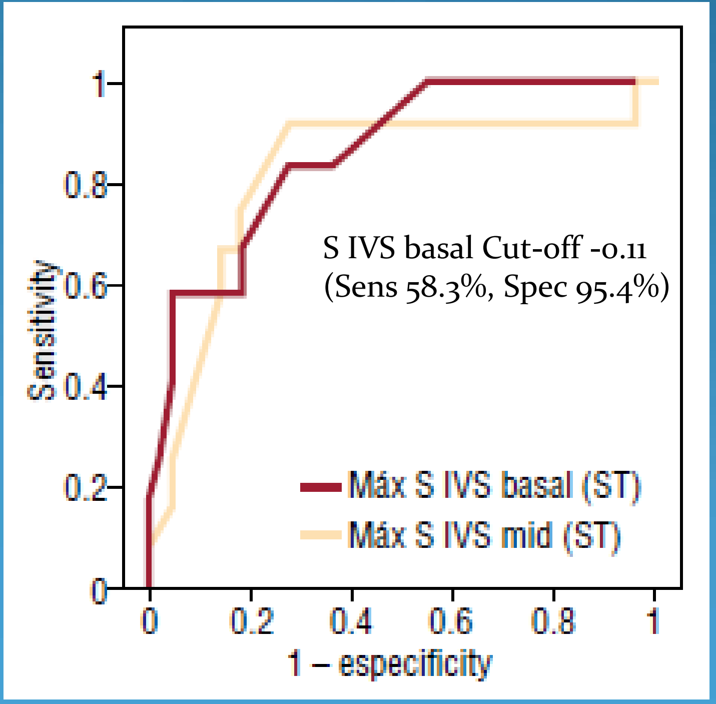


Marciniak et al. Eur J Cardio-thoracic Surg 2011;40:1131.

Longitudinal Strain predicts a decrease in EF after MVR

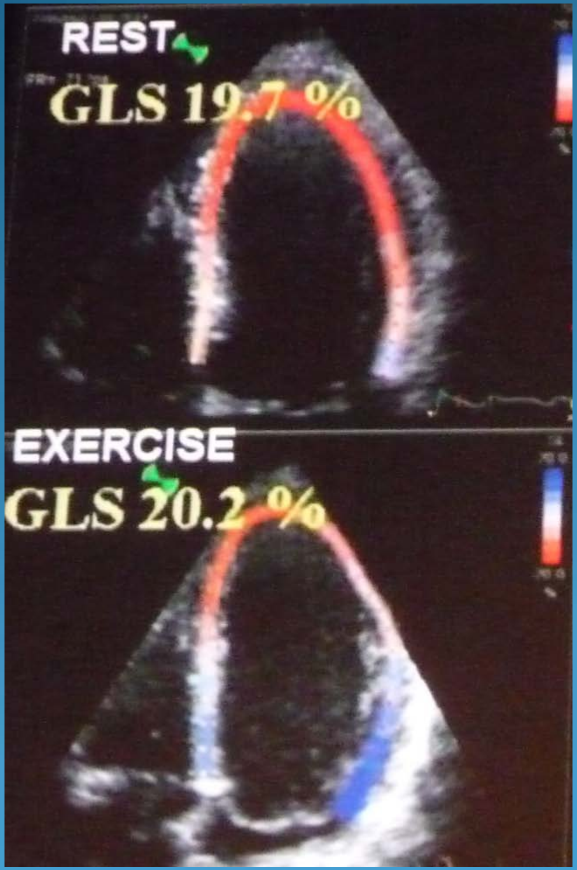
- 38 pts with severe MR, mean EF 62.4%
- MVR
- 2 groups: ↓ EF ≤10% and >10% at 6 months
- Measurements by STE - ↑ predictive value than measurements by TDI
- LS IVSbasal (ST) – the most powerful predictor of post-op EF





Agustín J et al. Rev Esp Cardiol 2010;63:544.

Longitudinal dysfunction during exercise predicts postoperative dysfunction



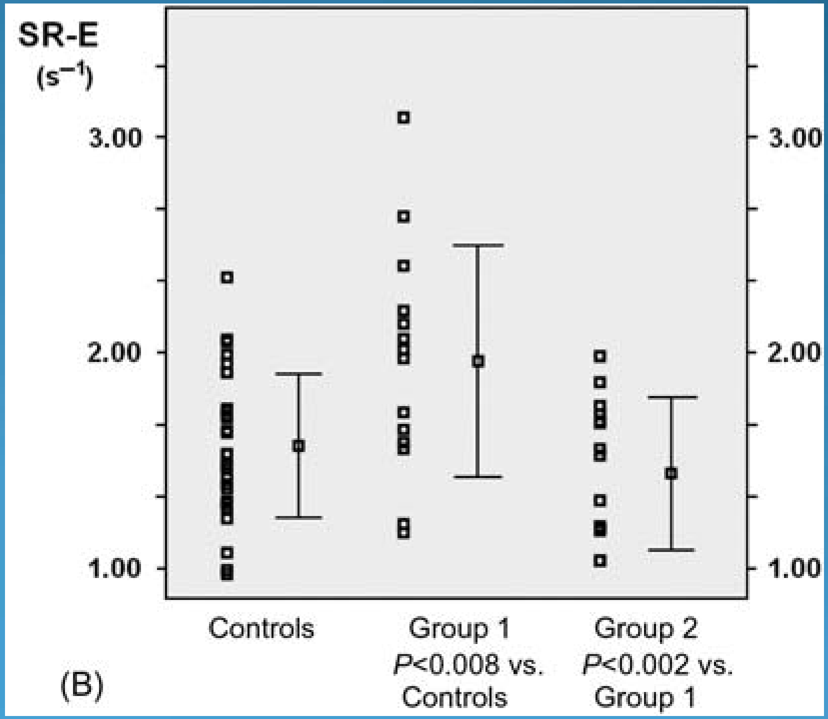
- 71 patients with asymptomatic degenerative MR and normal EF
- 23 controls
- Lower GLS at rest indicates subclinical LV dysfunction
- Lower changes in GLS at peak exercise (<1.9%) - associated with decrease in EF <50% after surgery

Lancellotti P et al. JASE 2008;21:1331.

Diastolic deformation in chronic MR

- TDI: 30 pts with moderate to severe MR
  - Group 1: asymptomatic, compensated LV
  - Group 2: have ≥1: > NYHA class I, LV ESD ≥40 mm, EF ≤60%
- 30 controls
- SR-E ↑ in pts with preserved LV function - enhanced by ↑ LA pressure, ↑ LV compliance, ↑ restoring forces
- SR-E ↓ in pts with LV systolic dysfunction - ↓ restoring forces, ↑ LV stiffness and advanced remodelling

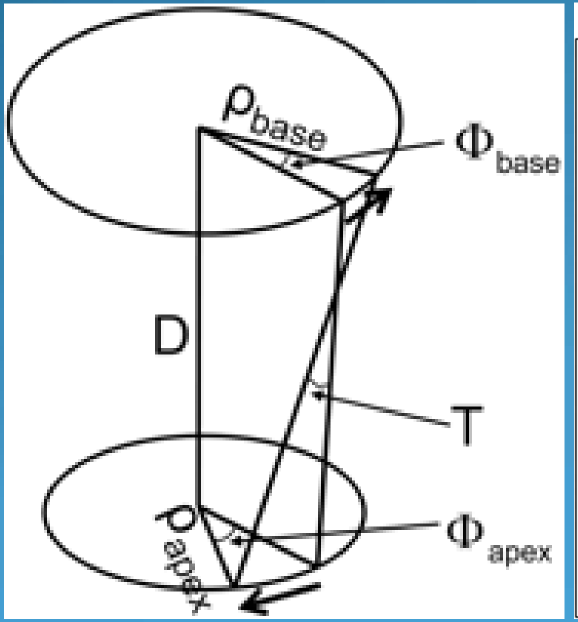
➜ Biphasic pattern



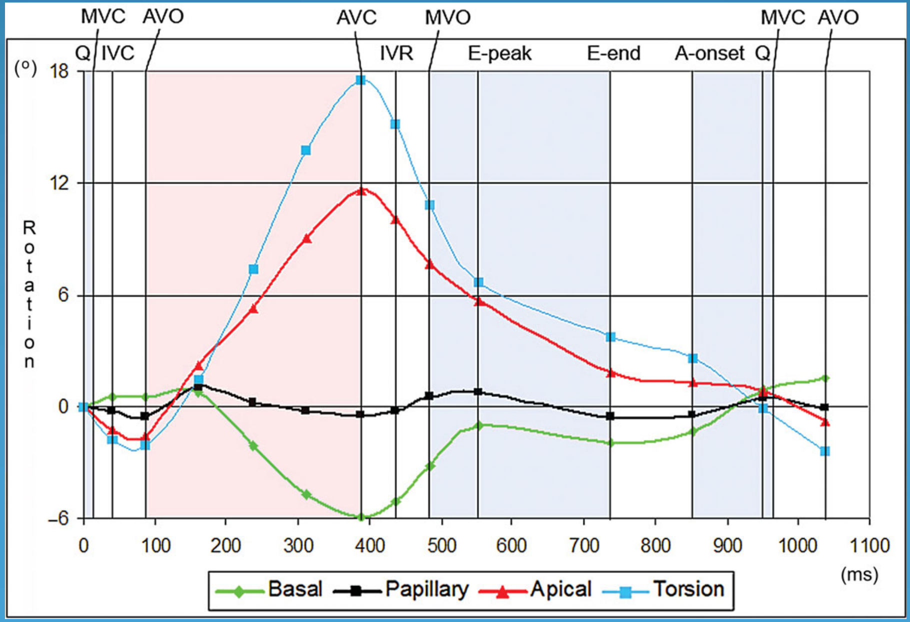
Borg et al. Eur J Echocardiogr 2010;11:523.

LV rotation and twist/torsion

- Role in LV systolic function – in generating SV, fiber stress and fiber shortening
- Role in LV diastolic LV function – contribute to suction and filling



Rüssel et al. J Am CollCardiol Img 2009.

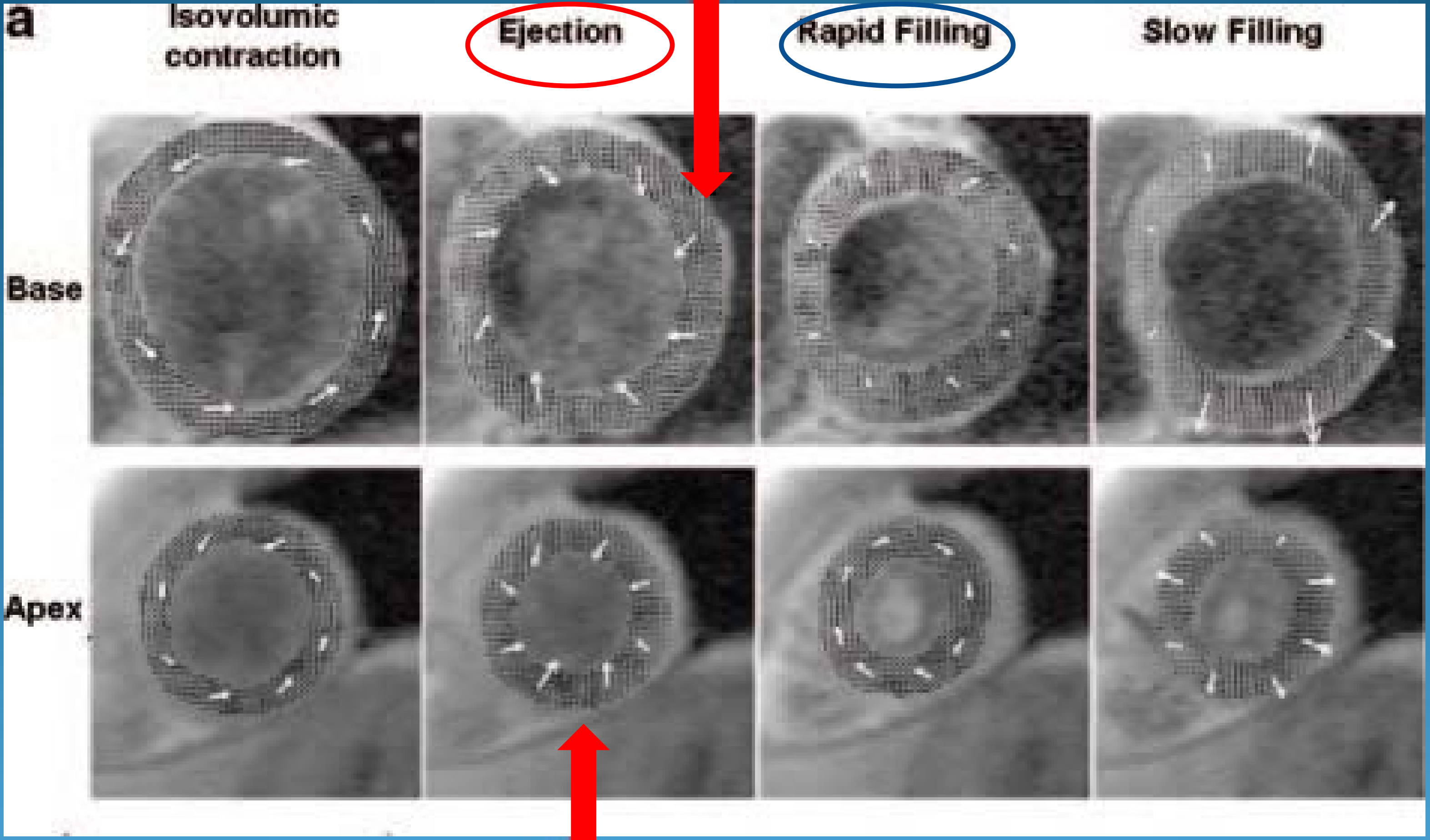


Gustaffson et al. Eur J Echocardiogr 2009.

Twist (°) =
A rot – B rot

Torsion (°/cm) =
Twist / D

Dominant contraction of the R-handed helix (subendocardium)



Co-contraction of the L-handed helix (subepicardium)

Buckberg et al. Circulation 2008;118:2571.

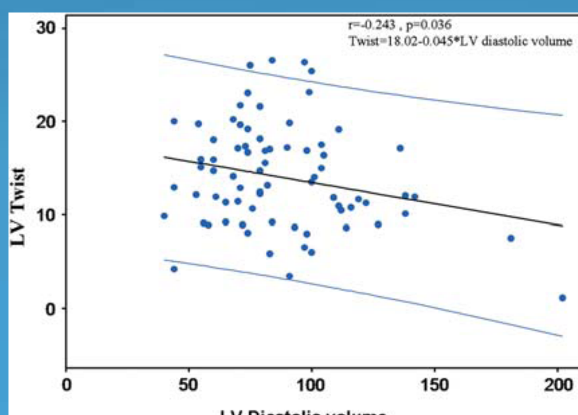
Reduced endocardial LV rotation detecs latent LV dysfunction in MR

- 83 pts with mild, moderate and severe MR
- EF>60%, LVESD<40 mm
- Moderate MR – highest rotational profile
- Severe MR – lowest rotational profile

➜ Biphasic pattern

Table 2 Left ventricle rotation-related parameters

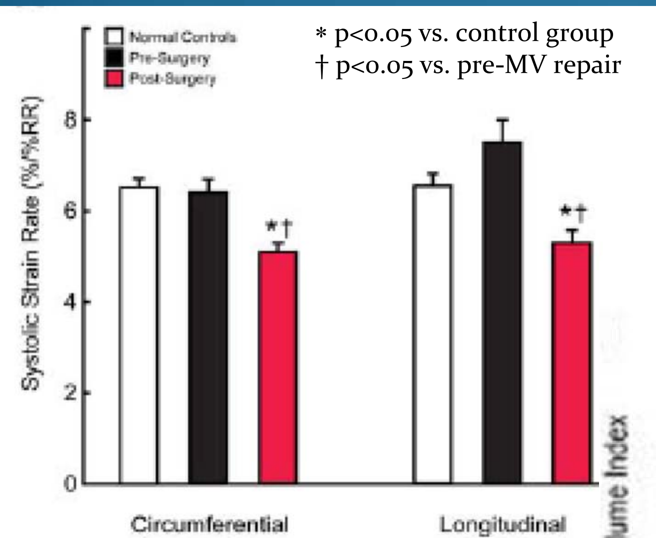
	Controls (*n* = 41)	Mild MR (*n* = 22)	Moderate MR (*n* = 12)	Severe MR (*n* = 9)	ANOVA *P*-value
Endocardial rotation					
ROT-API (°)	6.58 ± 3.17***	7.47 ± 3.11	10.77 ± 4.32**	6.11 ± 4.39*	0.003
ROT-BAS (°)	−6.06 ± 2.91	−7.81 ± 2.76	−7.07 ± 2.50	−5.33 ± 4.64	0.1
Twist (base-apex) (°)	12.65 ± 5.19	15.28 ± 4.08	17.83 ± 5.20‡	11.43 ± 6.09†	0.005
Torsion (°/cm)	1.6 ± 0.71	1.83 ± 0.62	2.26 ± 0.66¥	1.39 ± 0.80‖	0.015



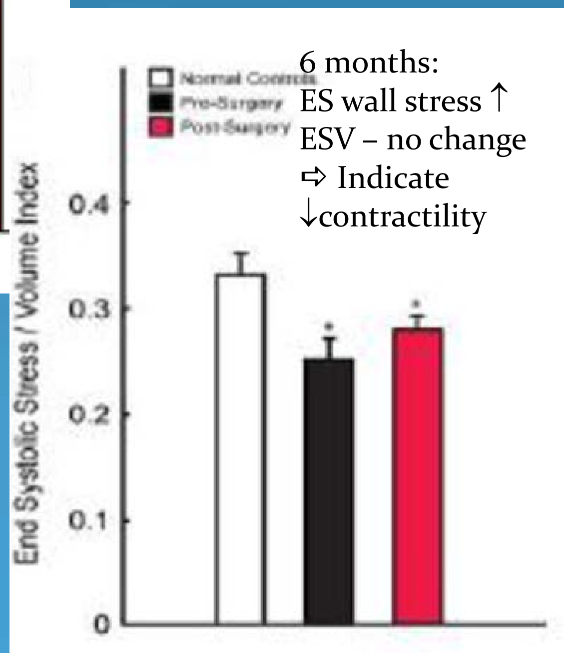
- Correlations endocardial twist/EDV and torsion/EDV, ESV⇨ suggest influence of ↑ preload and SDF despite preserved EF

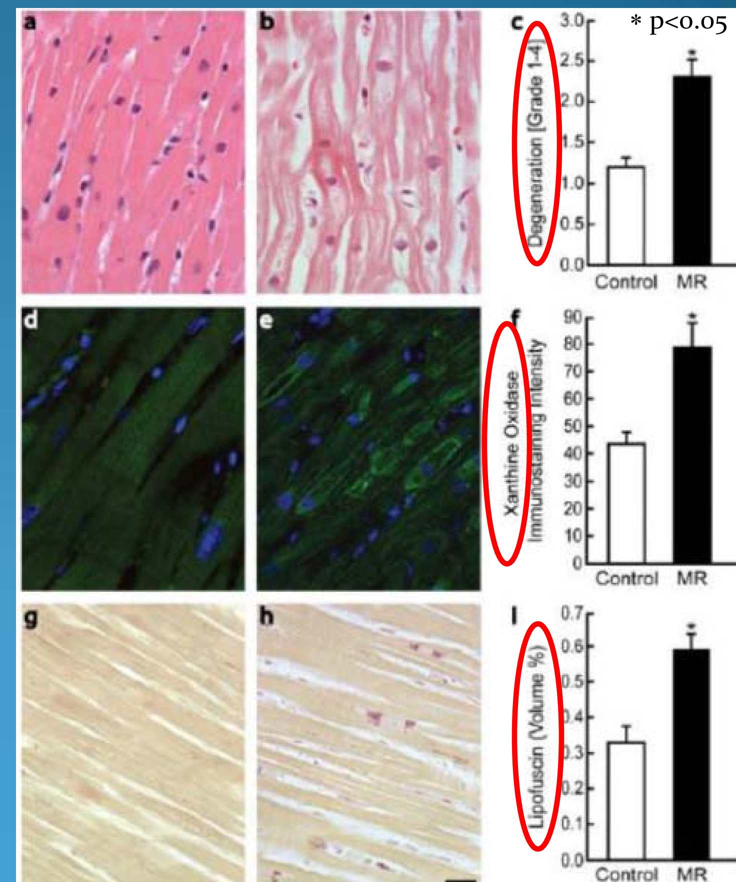
Moustafa et al. Eur J Echocardiogr 2011;12:291.

Increased oxidative stress and lipofuscin accumulation - LV contractile dysfunction



- 27 pts, severe MR, EF>60%, MV repair, 6 m
- 40 controls
- Strain and SR – MRI with tissue tagging
- Biopsy

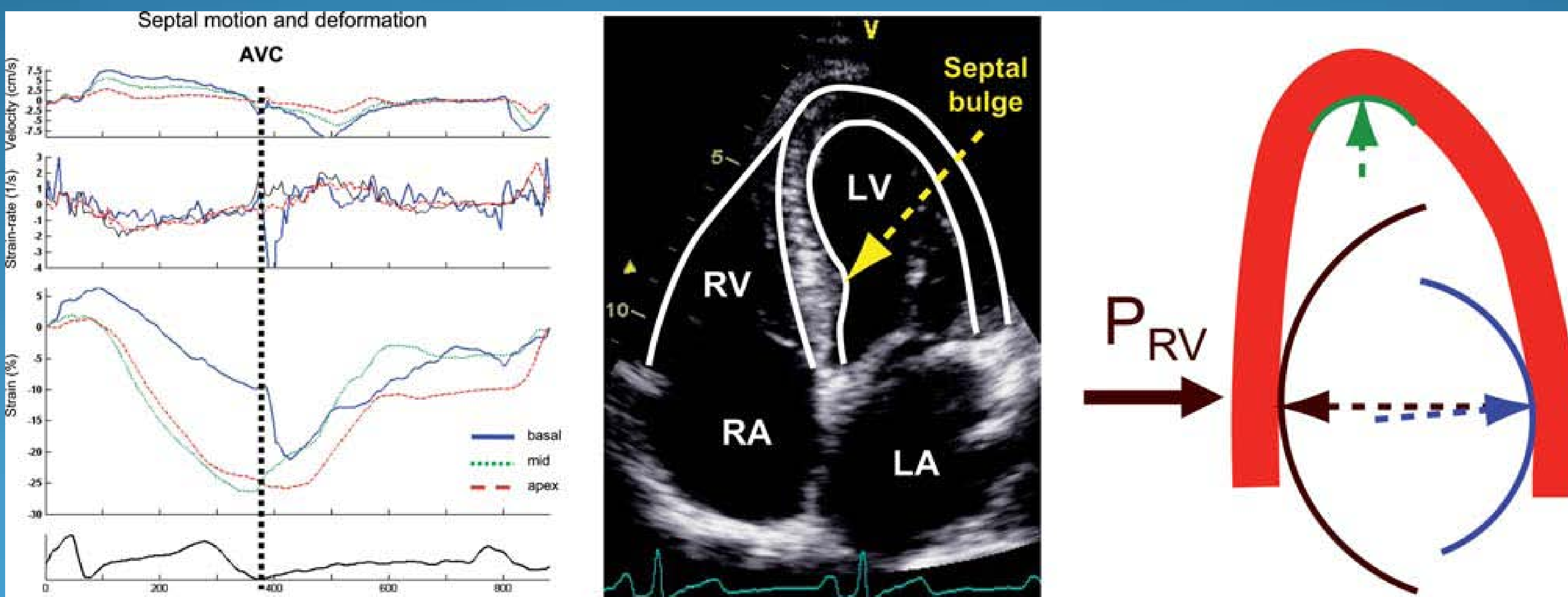




Ahmed et al. J Am Coll Cardiol 2010;55:671.

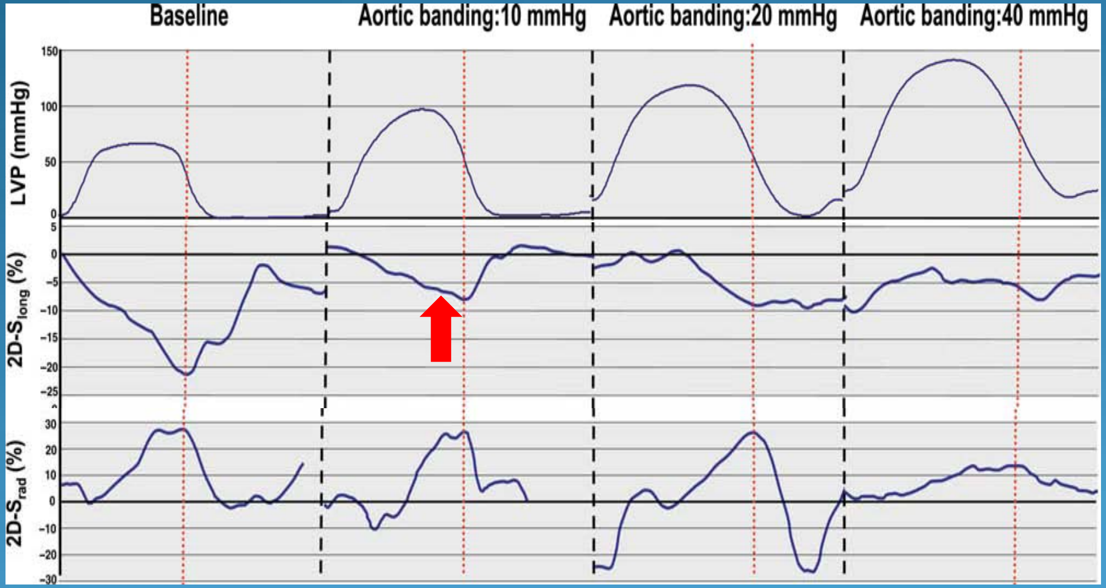
AS – pressure overload

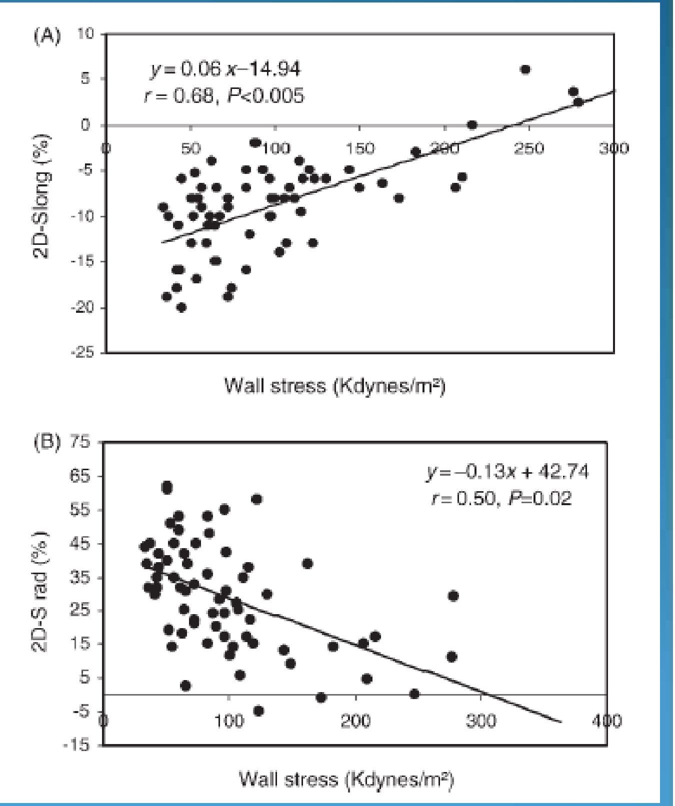
- LV remodelling with initial basal septal hypertrophy ⇦ ↑ wall stress as a result of the flat curvature of the LV
- ↓ S and SR
- Postsystolic deformation



Bijnens et al. Eur J Echocardiogr 2009;10:216.

Early impairment of longitudinal function and preserved radial function in acute aortic banding

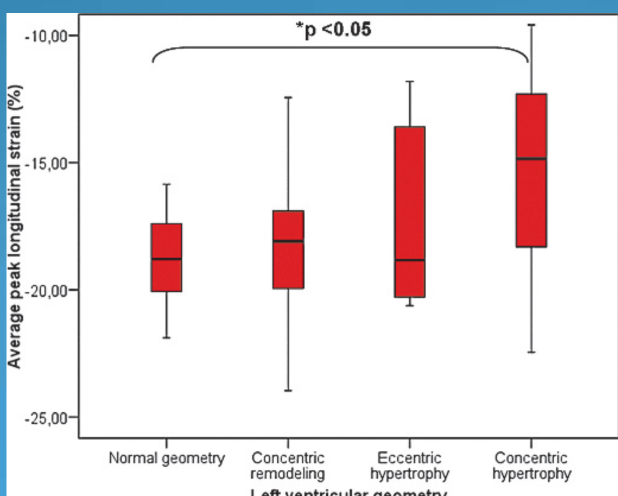




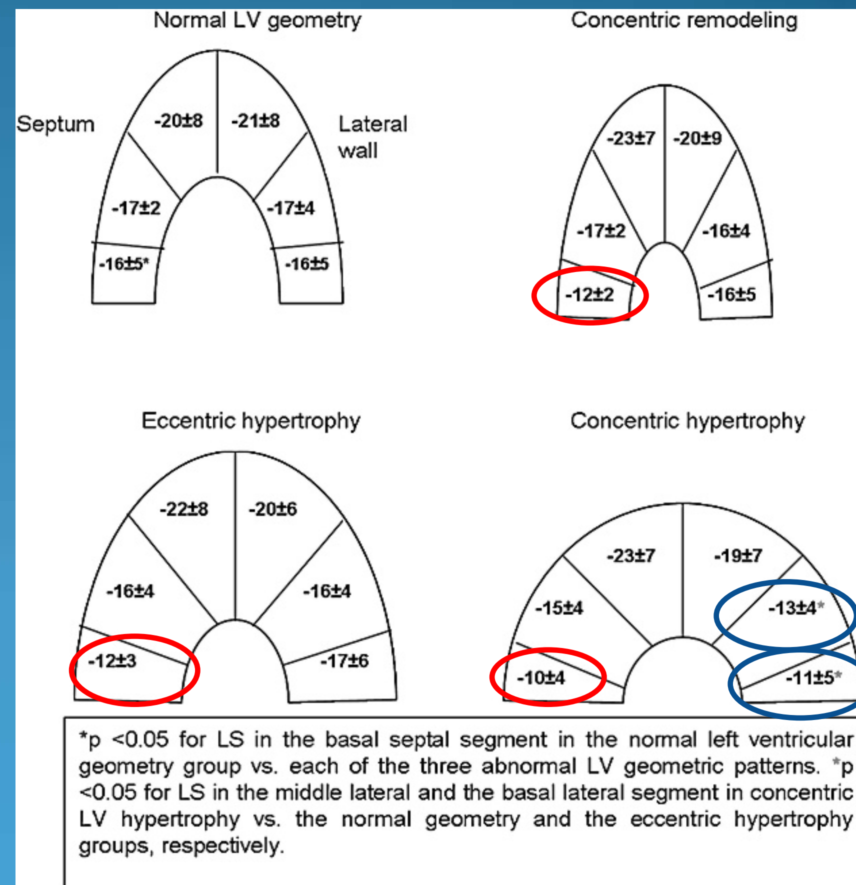
Donal et al. Eur J Echocardiogr 2009;10:914.

AS: relation deformation/geometry

- 70 pts: 40 asymptomatic and 30 symptomatic
  - *Normal LV mass*: normal geometry; concentric remodelling
  - *LV hypertrophy*: eccentric; concentric
- No differences in CS and RS
- ↓ LS is associated with ↑mass index and relative wall thickness, severity of AS

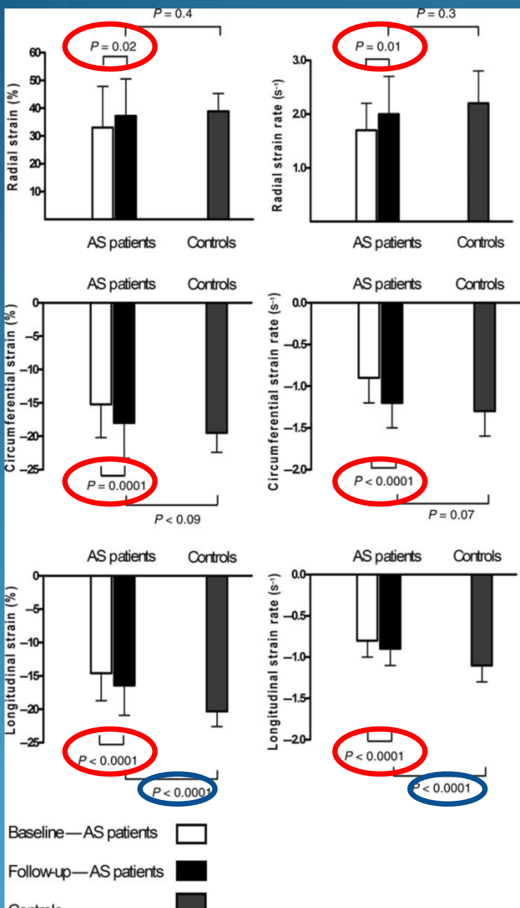


Segmental variations in LS



Cramariuc D, et al. Heart 2010;96:106.

Improvement in impaired S and SR after AVR



- 73 pts with severe AS
  - Preserved EF
  - AVR
  - FU 17 months
- 40 controls

	ΔGlobal LV afterload	
	r	P-value
Radial strain (%)	0.166	0.198
Radial strain rate (s^{-1})	0.221	0.085
Circumferential strain (%)	0.403	0.001
Circumferential strain rate (s^{-1})	0.327	0.009
Longitudinal strain (%)	0.426	0.001
Longitudinal strain rate (s^{-1})	0.269	0.034

- Sign. improvement in L-, C- and R- S/SR after AVR
- L-S/SR remain ↓
- Improvement in L-S/SR, and C-S/SR – related to LV afterload reduction
- No change in EF

Delgado et al. Eur Heart J 2009;30:3037.

Impact of global LV afterload on LV function

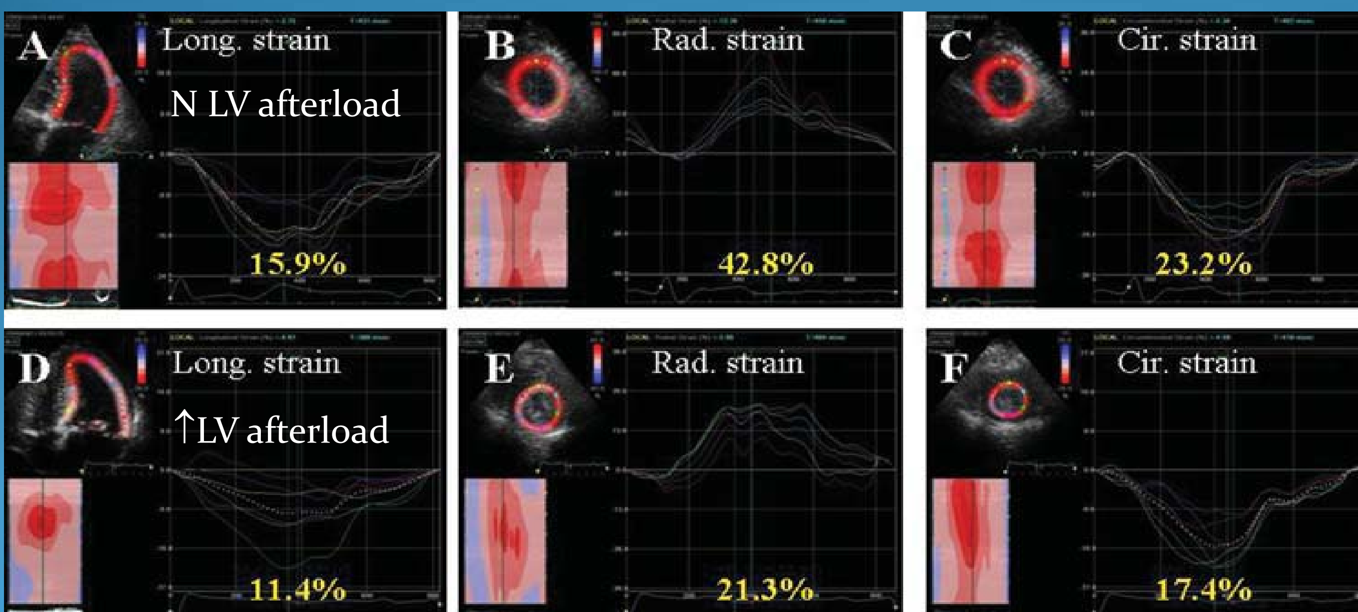
- 173 pts with severe asymptomatic AS, EF ≥55%
- ↑ global LV afterload - 28% of pts
  - Impairs predominantly short-axis function
  - Prevalent in low-flow AS (22% of pts)

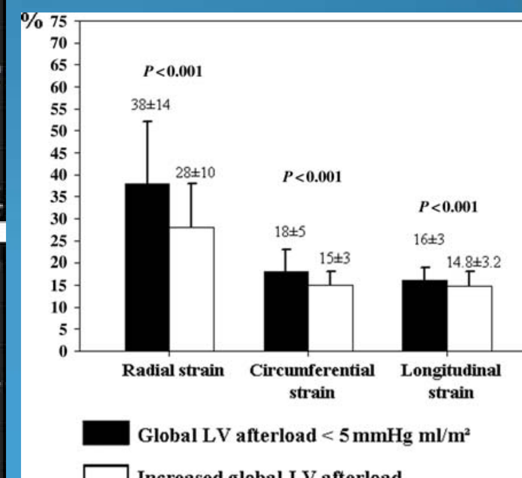
Global LV load:

- Valvular – AS

+

- Arterial - ↓systemic arterial compliance





Lancellotti et al. Eur J Echocardiogr 2010;11:537.

Pradoxical low-flow, low gradient AS despite preserved EF – more advanced stage of the disease

512 pts with severe AS, EF ≥50%

- N LV flow output: SVi>35 ml/m²
- Low flow: SVi ≤35 ml/m² (35% pts)

Results from severe AS + :

1. ↓ Systemic arterial compliance (SVi/PP)
2. LV concentric remodelling
3. ↓ contractility

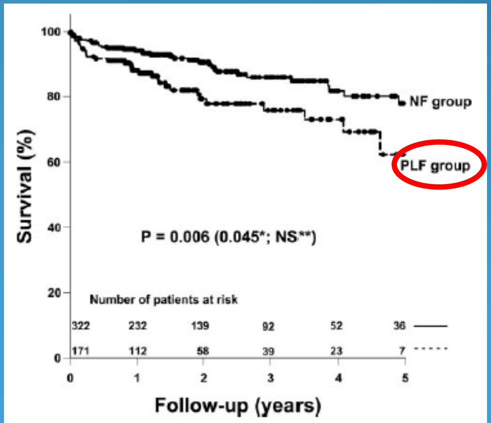
↑Global LV afterload

Valvulo-arterial impedance (Zva):

$\frac{SAP + MPG}{SVi}$ (mmHg/ml/m²)

	NF Group (n=331)	PLF Group (n=181)	*P*
LV global afterload			
Valvulo-arterial impedance, mm Hg · mL⁻¹ · m⁻²‡	4.1±0.7	5.3±1.3	<0.001
Valvulo-arterial impedance ≥5.5, %‡	3	37	<0.001

⇨ Reduction of SV and CO



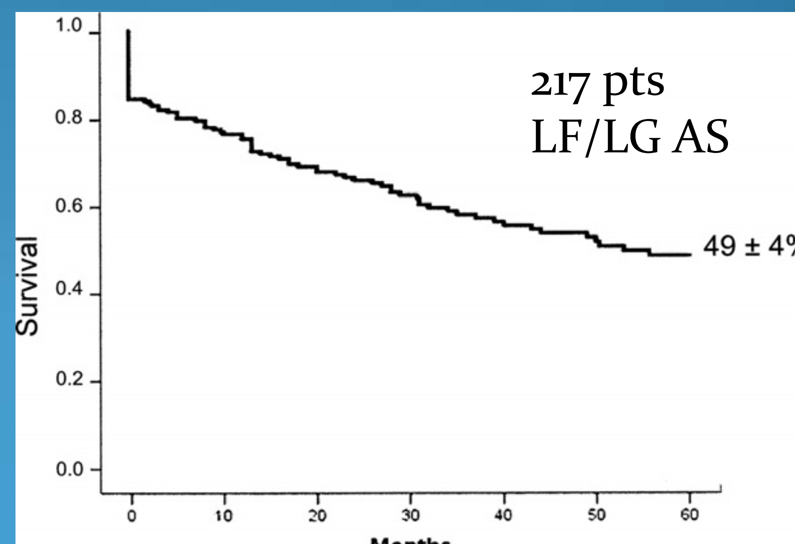
Hachicha et al. Circulation 2007;115:2856.

Low flow, low gradient AS

AVA < 1 cm² (<0.6 cm²/m²), LV EF ≤40%, Mean PG ≤40 mmHg (ESC)

Secondary LV dysfunction to severe AS

- ~5-10% of AS patients
- Dobutamine-echo:
  - DD from pseudosevere AS
- The worst prognosis



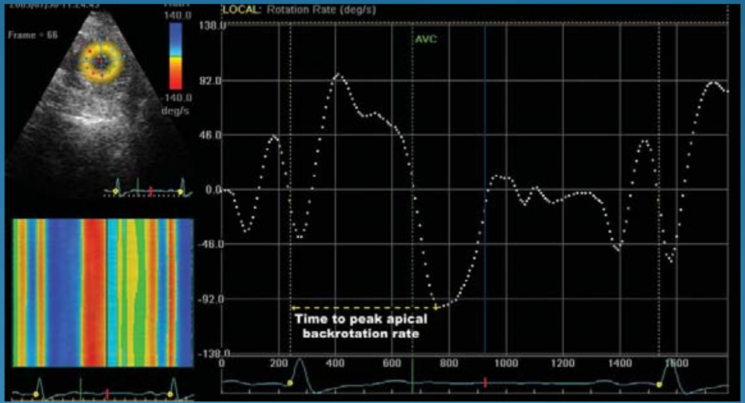
Levy et al. J Am Coll Cardiol 2008;51:1466.

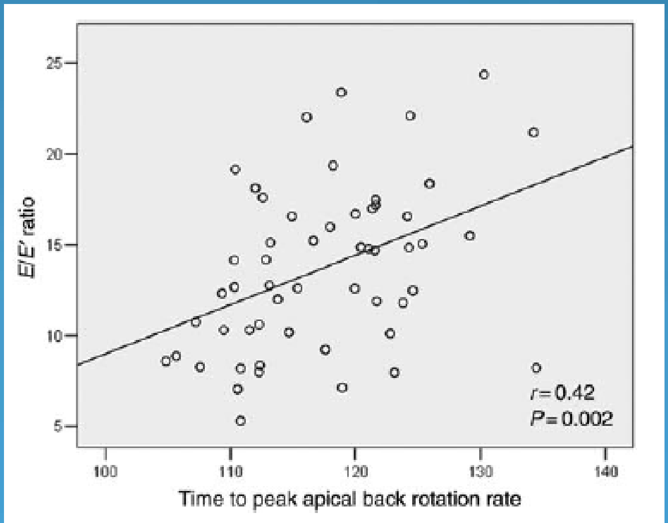
Torsional dynamics in AS

- 61 pts with severe AS and preserved EF, HF NYHA I-III
- 40 controls

	Controls ($n = 40$)	AS ($n = 61$)	P-value
LV peak apical rotation (°)	15.7 ± 5.9	21.0 ± 7.6	<0.001
LV peak basal rotation (°)	−6.2 ± 2.9	−6.7 ± 3.2	0.4
LV twist (°)	20.8 ± 6.8	26.5 ± 9.1	0.001
LV twist rate (°/s)	118 ± 35	137 ± 55	0.006
LV peak systolic torsion (°/cm)	2.7 ± 0.9	3.4 ± 1.3	0.002
LV peak untwisting rate (°/s)	−143 ± 48	−158 ± 59	0.18
Time to peak LV untwisting rate	115 ± 7	115 ± 6	0.8
LV peak apical back rotation rate (°/s)	−93 ± 47	−115 ± 55	0.04
Time to peak apical back rotation rate	113 ± 8	117 ± 7	0.004
LV peak basal back rotation rate (°/s)	64 ± 20	70 ± 23	0.18
Time to peak basal back rotation rate	113 ± 6	113 ± 7	0.9

correlation: E/E', LAVi, BNP





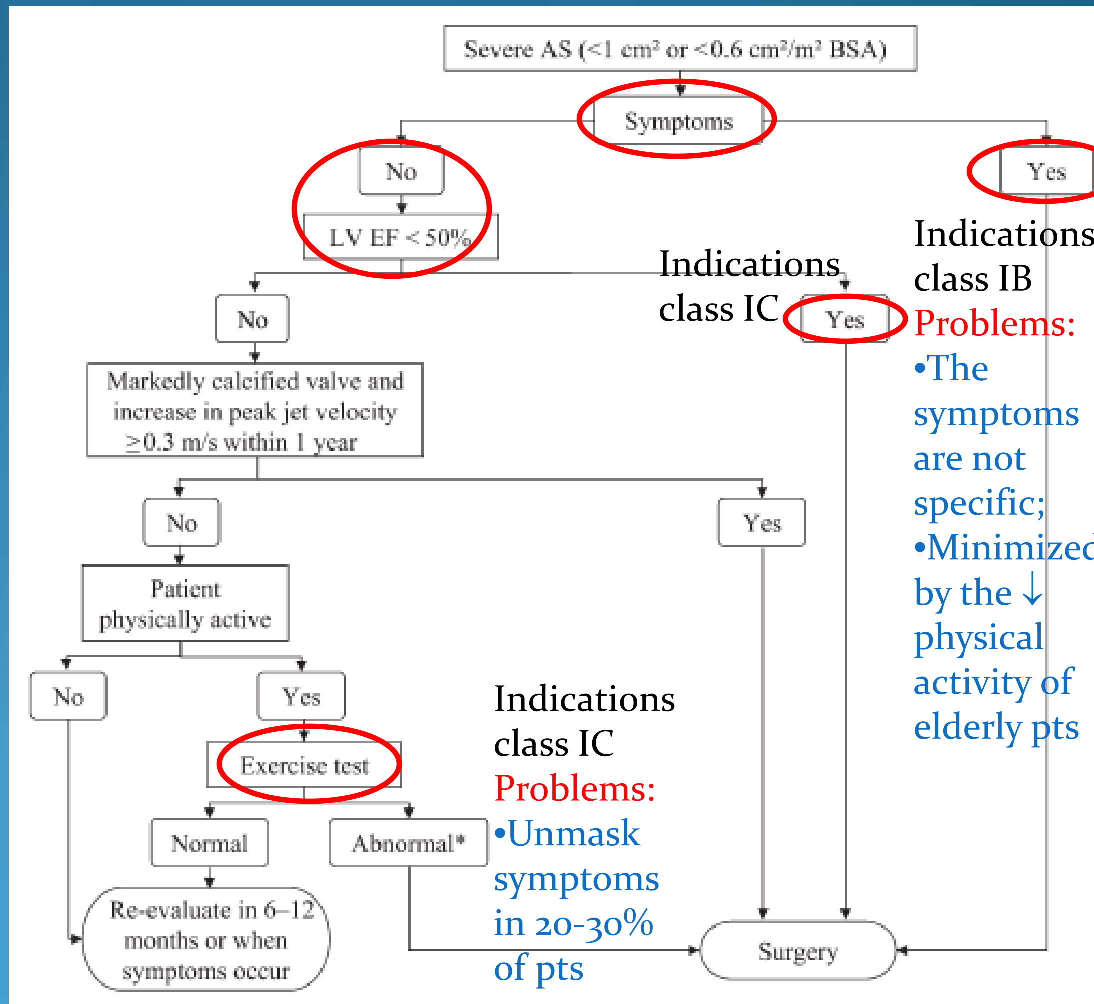
- ↑Apical rotation ⇦ subendocardial ischemia - ↓counteraction
- ↑ and delayed apical back rotation rate
- Impaired untwisting - potential role in development and progression of diastolic dysfunction

Popescu et al. Eur J Echocardiogr 2010;11:406.

Indications for AVR in AS

Exercise test

1. Change of mean pressure gradient
2. Study of LV systolic function
    - EF
    - L Strain



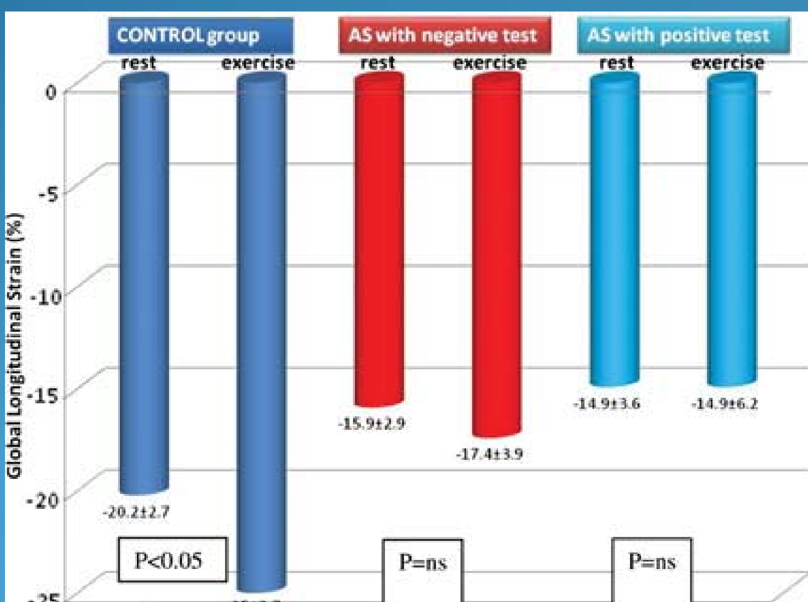
Guidelines on the management of valvular heart disease. Eur Heart J 2007.

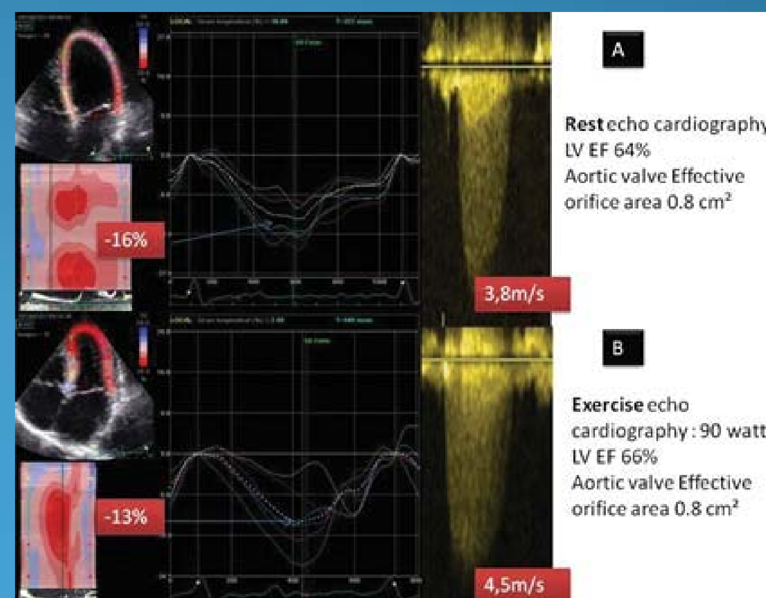
Nonuniformity in changes in PG and GLS during exercise

- 207 asymptomatic pts, moderate-severe AS, EF ≥55%
- Abnormal test in 28% of pts
- Associated with:
  - ↓LS at rest <-15.5%,
  - ↑ in MPG ≥14 mmHg,
  - exercise-induced Δ GLS <-1.4%

4 categories of pts according to changes in LS and MPG:

Low Δ MPG + No CR n=50 Δ MPG < 14 mmHg Δ GLS < -1.4%	Low Δ MPG + CR n=58 Δ MPG < 14 mmHg Δ GLS > -1.4%
Abn Ex test= 36 %	Abn Ex test = 14%
High Δ MPG + CR n=49 Δ MPG > 14 mmHg Δ GLS > -1.4%	High Δ MPG + No CR n=50 Δ MPG > 14 mmHg Δ GLS < -1.4%
Abn Ex test = 31%	Abn Ex test = 54%

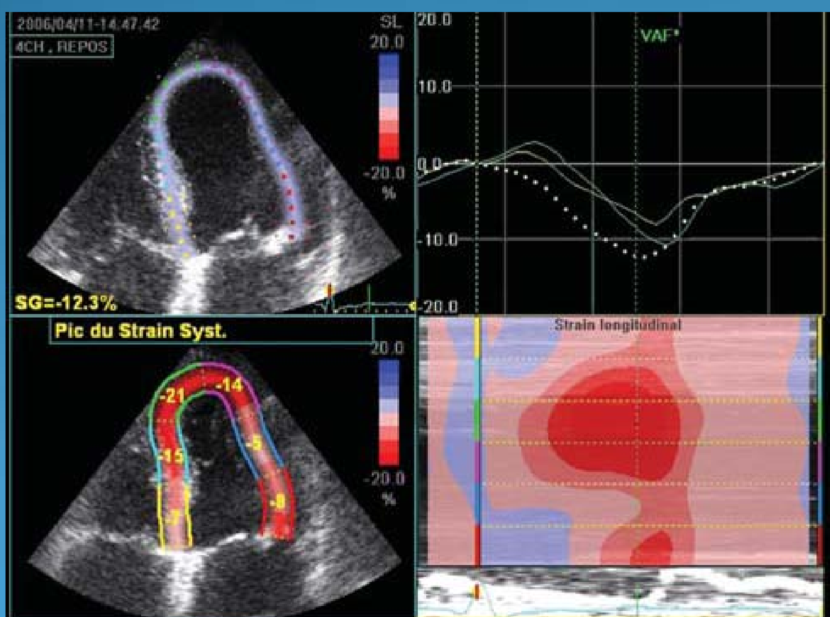




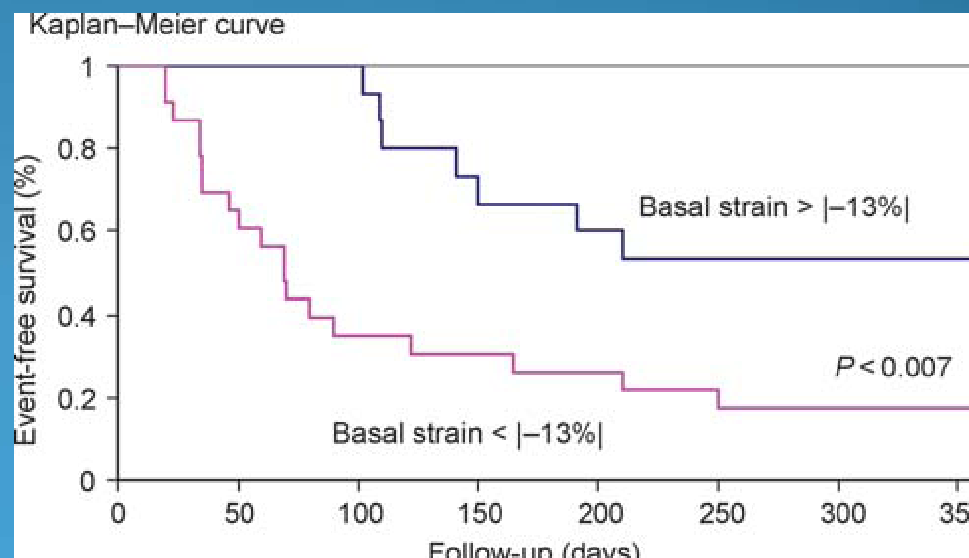
Donal et al. Eur J Echocardiogr 2011;12:235.

Impaired longitudinal deformation impacts on exercise tolerance and prognosis in AS

- 60 asymptomatic pts with severe AS and EF >55%
- 60 controls
- GLS < -18% and basal LS < -13% - predict an abnormal exercise response
- ↓ LS – marker of cardiovascular events
- ↓ basal LS < -13% - the best predictor of CV events within 12 m (AVR, CVD, nonfatal CVE)



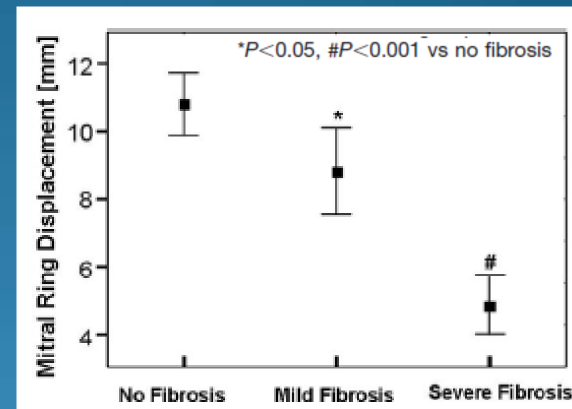
Severe AS SG – 12% SB –7%

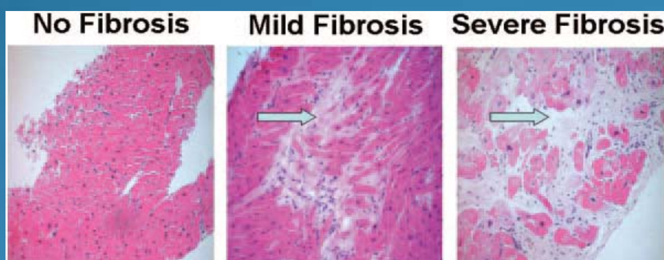


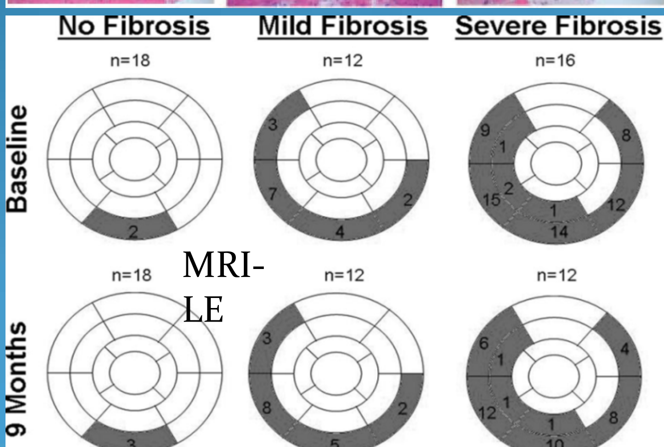
Lafitte et al. Eur J Echocardiogr 2009;10:414.

Fibrosis in AS

- 58 symptomatic pts with severe AS, AVR, FU at 9 months
- TDI – SR and S, MRI-LE - fibrosis
- Biopsy - **Myocardial fibrosis:**
  - Subendocardial
  - Starts from the basal segments
  - Irreversible
  - Impact on the postoperative outcome







	No Fibrosis (n=22)	Mild Fibrosis (n=15)	Severe Fibrosis (n=21)	P
Radial strain, %	31 (11)	29 (9)	26 (10)	0.119
Radial strain rate, s^{-1}	1.7 (0.4)	1.7 (0.4)	1.6 (0.3)	0.247
Septal longitudinal strain, %	−19 (5)	−15 (6)	−10 (5)	<0.001
Septal longitudinal strain rate, s^{-1}	−1.2 (0.2)	−0.9 (0.3)	−0.6 (0.2)	<0.001

Longitudinal function parameters:

SR, S, Mitral ring displacement –

related to the severity of fibrosis and the clinical outcome after AVR

Weidemann et al. Circulation 2009;120:577.

Conclusions

- Deformation parameters are load dependent and should be related to LV geometry and shape
- Adaptive mechanisms in valve disease have to be known
- Longitudinal deformation can be used in early detection of LV dysfunction and in prediction of exercise response and postoperative outcome
- Deformation parameters during exercise: useful in prediction of CVE and postoperative EF
- LV torsion is a valuable addition in detecting early LV dysfunction
- Further studies are needed for implementation of deformation parameters in the assessment of valve disease