

Université
de Toulouse

IMAGERIES NOUVELLES POUR LA PRATIQUE EXEMPLE DES ATTEINTES CORONAIRES DANS LA STÉNOSE AORTIQUE



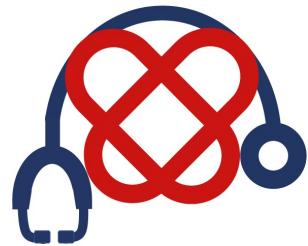
IRM ET MYOCARDE QUELLES INFORMATIONS EN ATTENDRE ?

Olivier LAIREZ
CHU de Toulouse

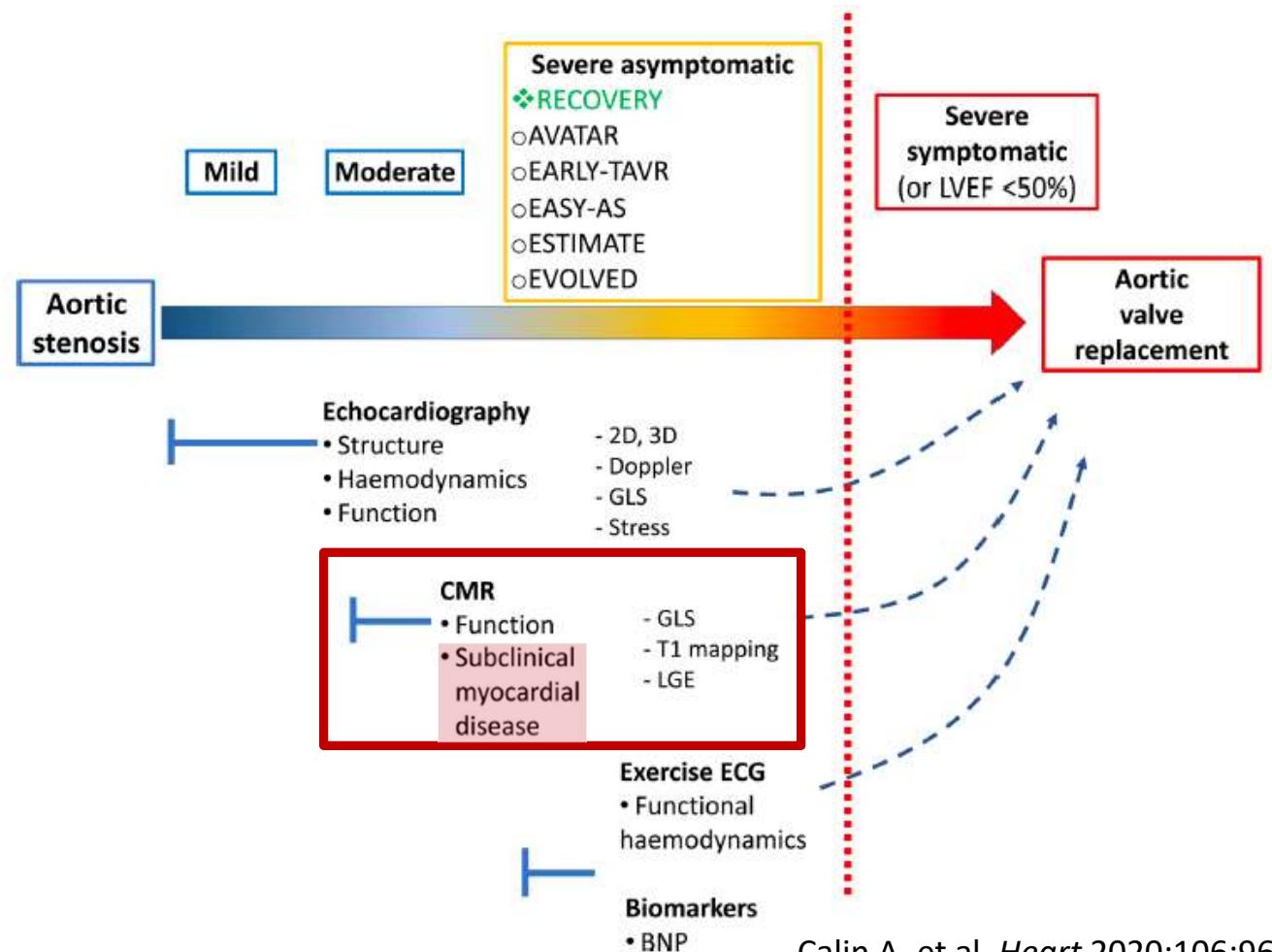
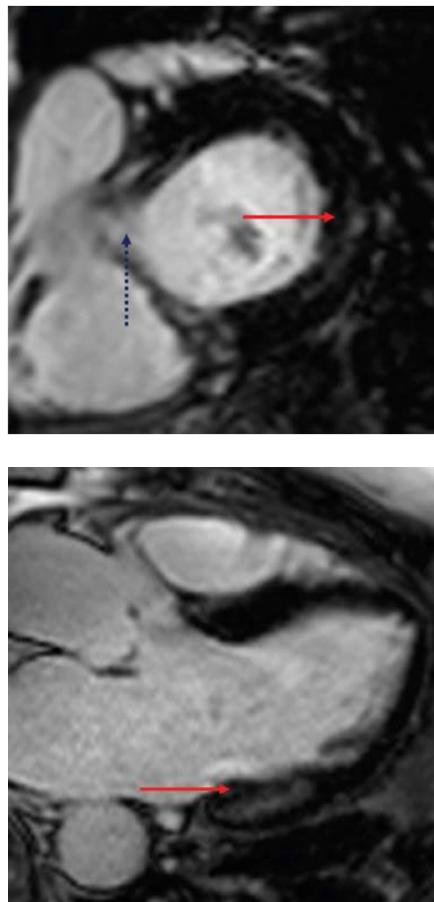
LIENS D'INTÉRÊT

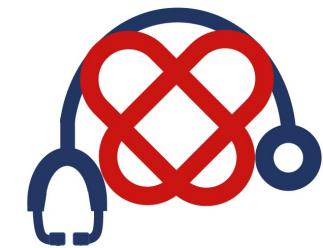
Olivier LAIREZ

Aucun



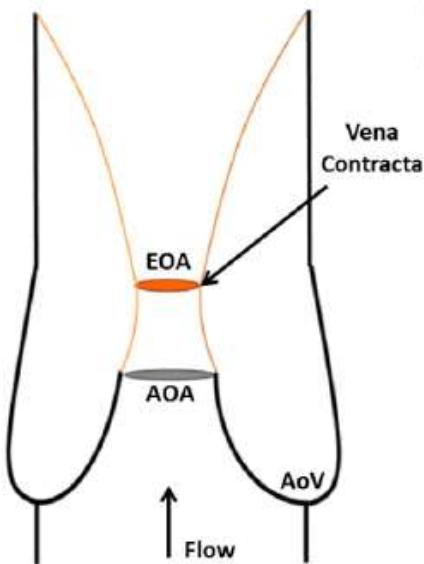
Role of advanced left ventricular imaging in adults with aortic stenosis



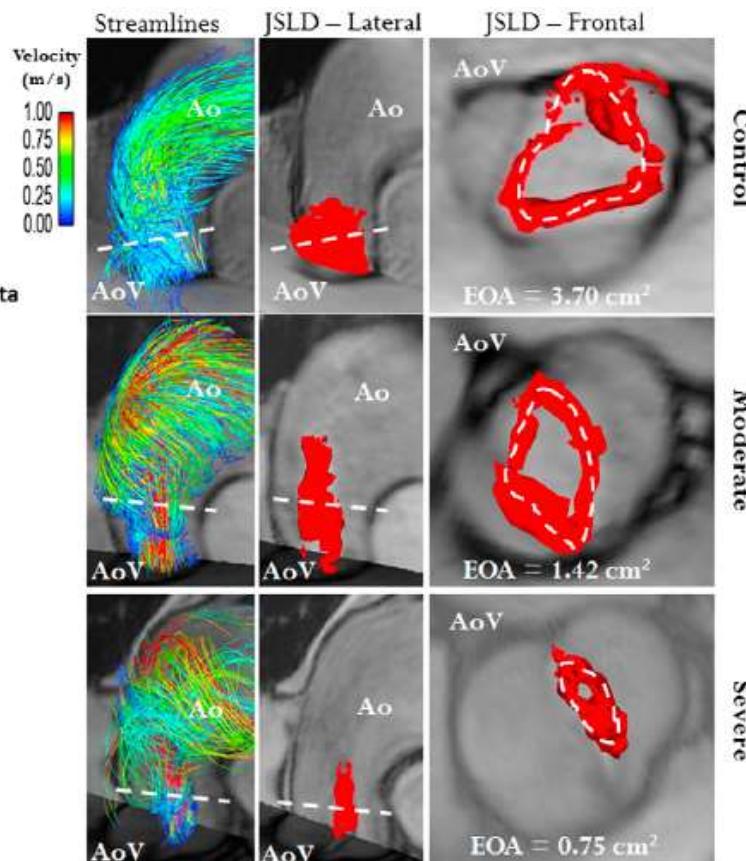


PLACE DU 4D FLOW

A



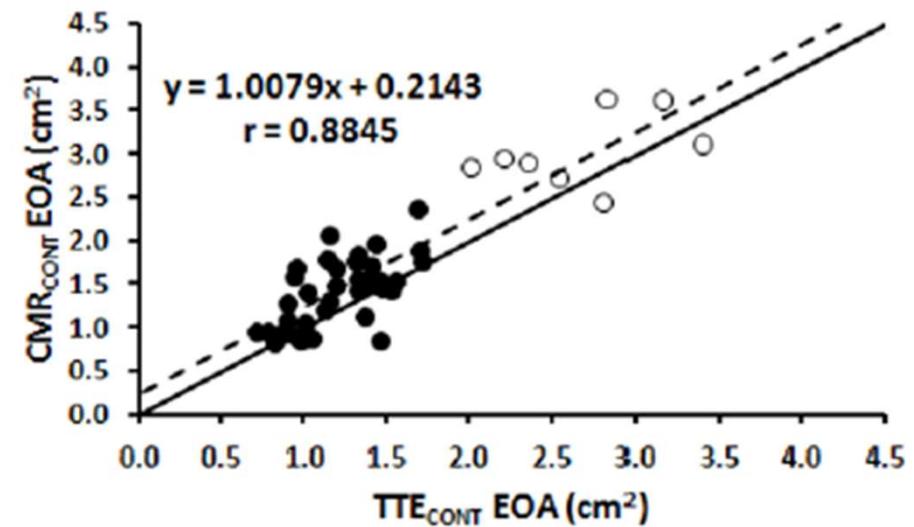
B

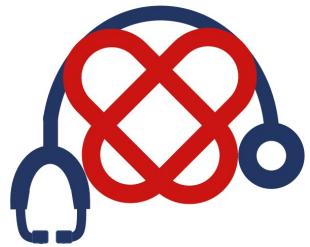


Control

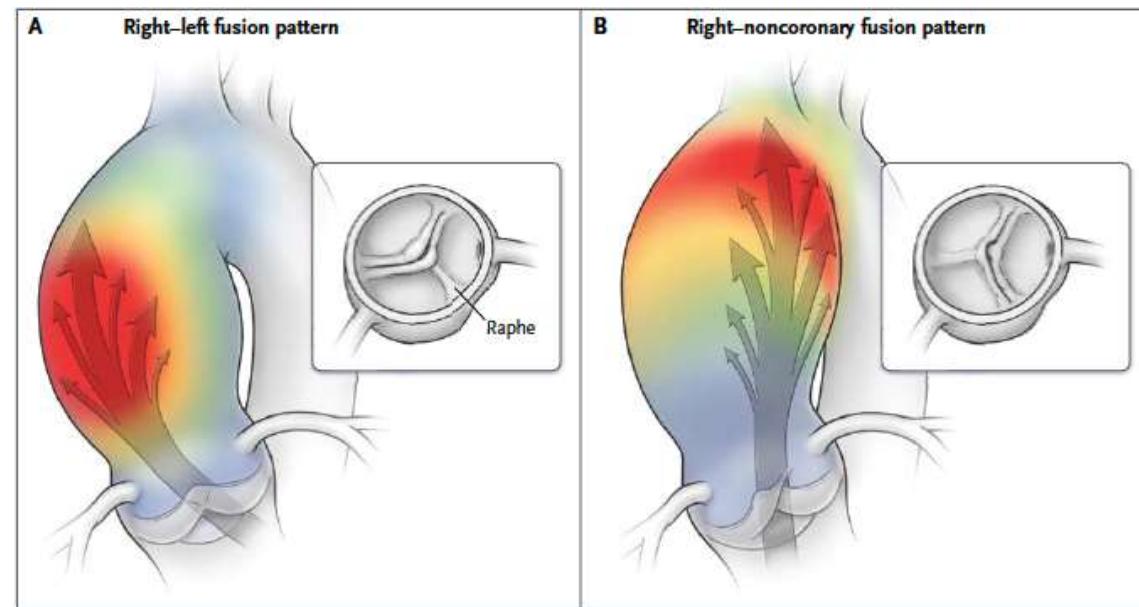
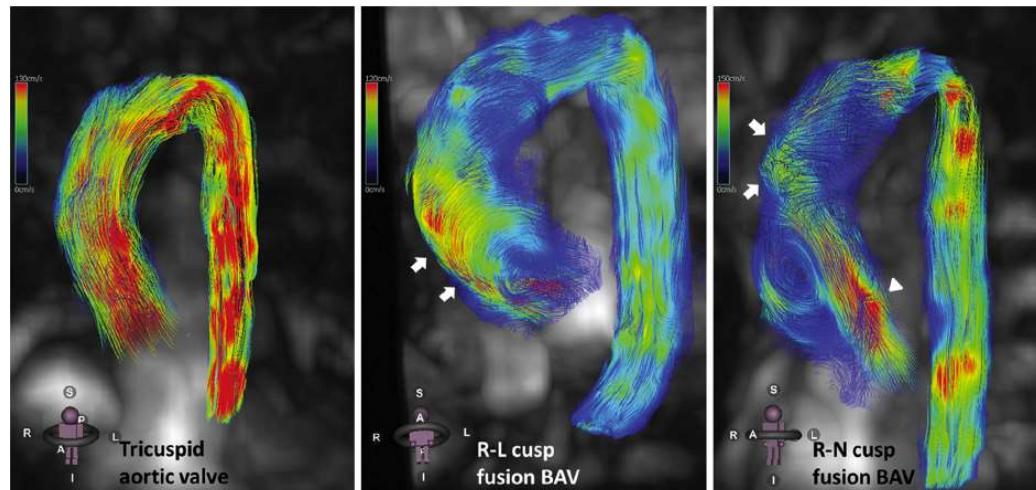
Moderate

Severe



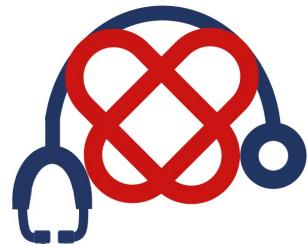


PLACE DU 4D FLOW



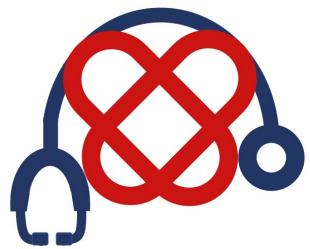


RÉTRÉCISSEMENT AORTIQUE & FIBROSE MYOCARDIQUE

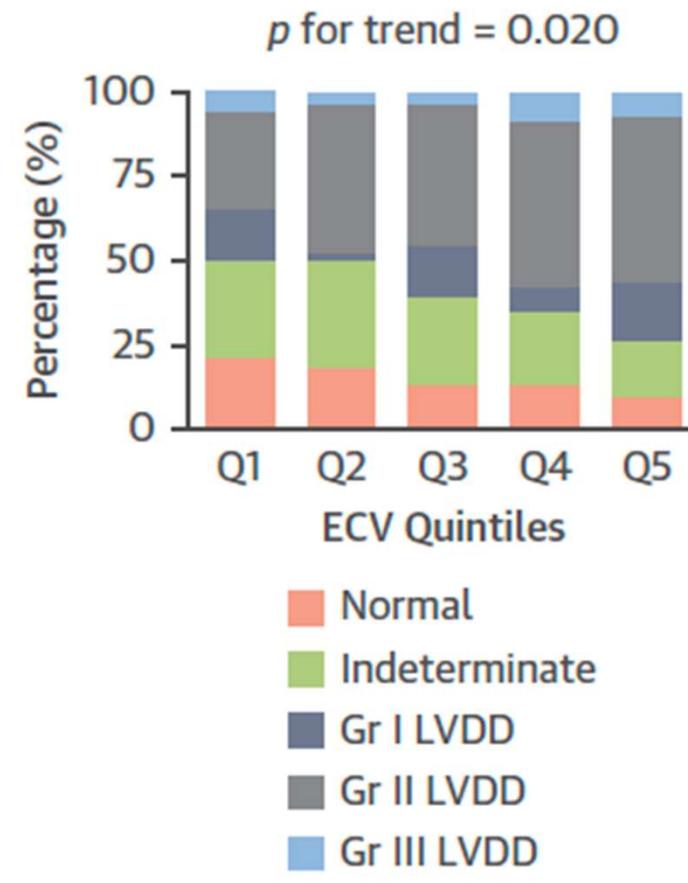
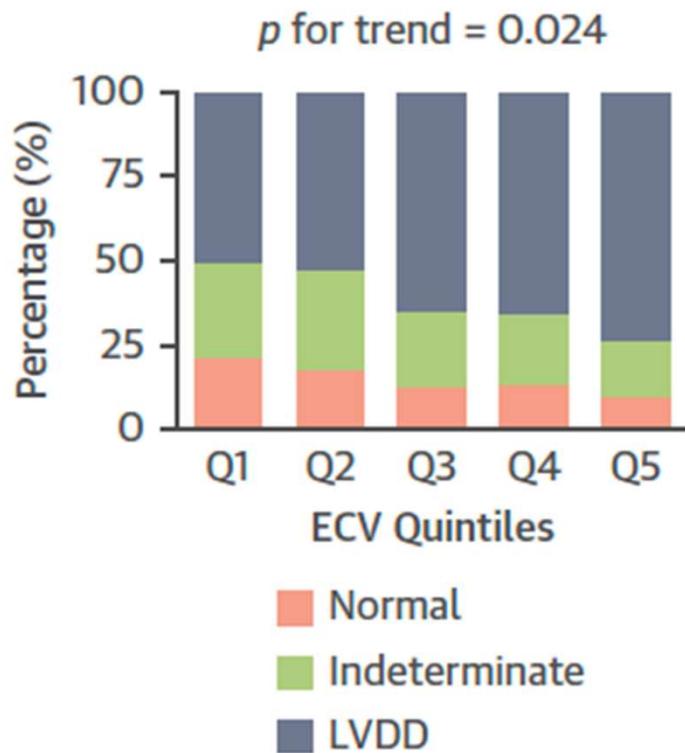


Diffuse Myocardial Fibrosis and Diastolic Function in Aortic Stenosis

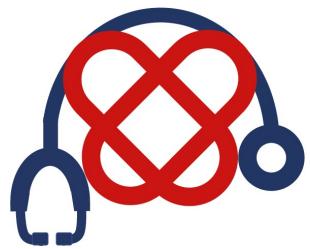
Association Between ECV and Diastolic Function Parameters in the Entire Study Population of Patients With Aortic Stenosis and Control Subjects				
	ECV (per 1% Increase)			
	Unadjusted		Adjusted*	
	OR (95% CI)	p Value	OR (95% CI)	p Value
Diastolic dysfunction†	1.15 (1.04-1.27)	0.005	1.15 (1.04-1.26)	0.006
Septal e' <7 cm/s	1.19 (1.02-1.39)	0.030	1.19 (1.01-1.39)	0.038
E/septal e' ratio >15	1.16 (1.05-1.27)	0.003	1.15 (1.05-1.27)	0.003
TR Vmax >2.8 m/s	1.12 (0.99-1.27)	0.064	1.12 (0.99-1.27)	0.075
LAVI >34 ml/m ²	1.11 (0.98-1.27)	0.113	1.10 (0.97-1.26)	0.145



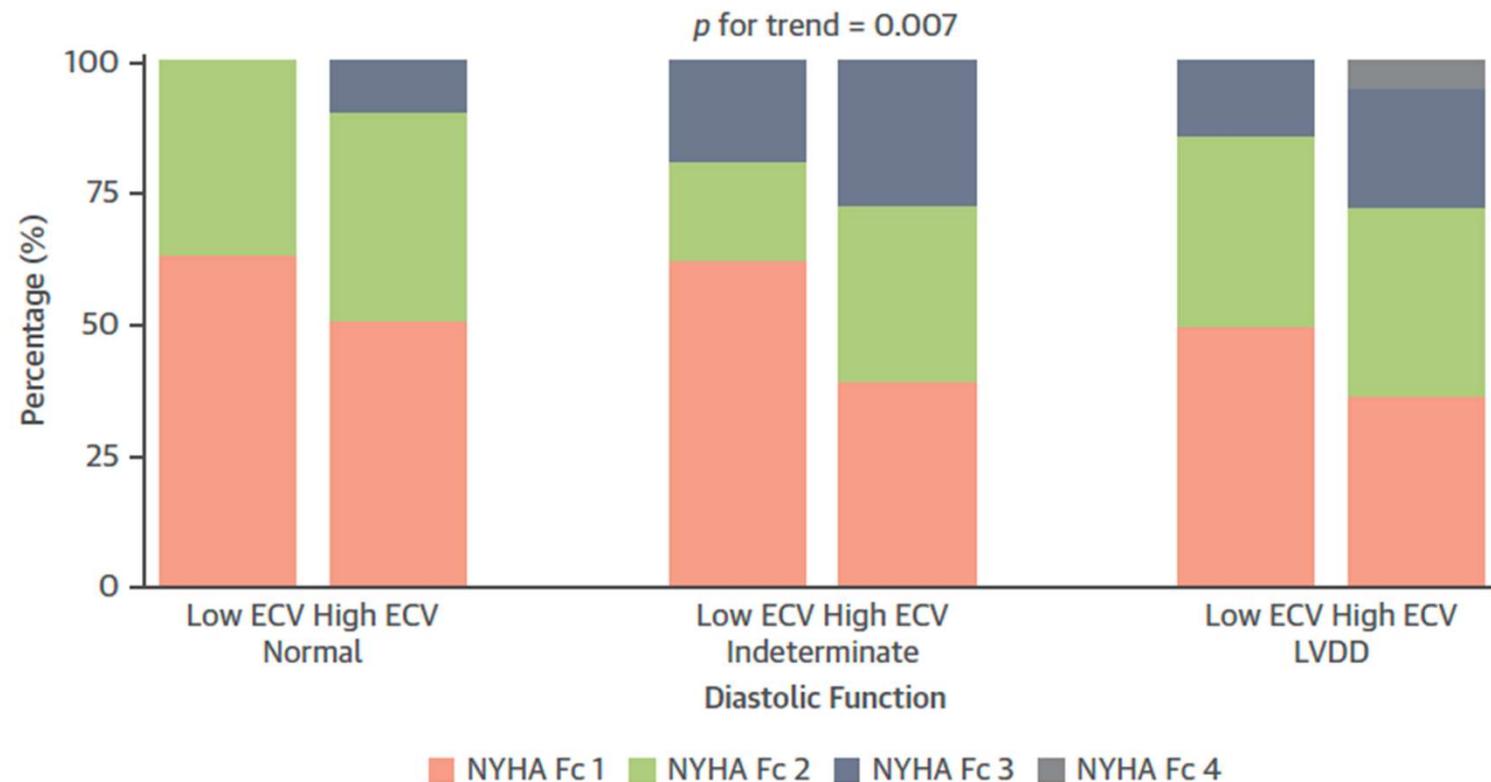
Diffuse Myocardial Fibrosis and Diastolic Function in Aortic Stenosis



Lee HJ, et al. *J Am Coll Cardiol Img* 2020;13:2561–72



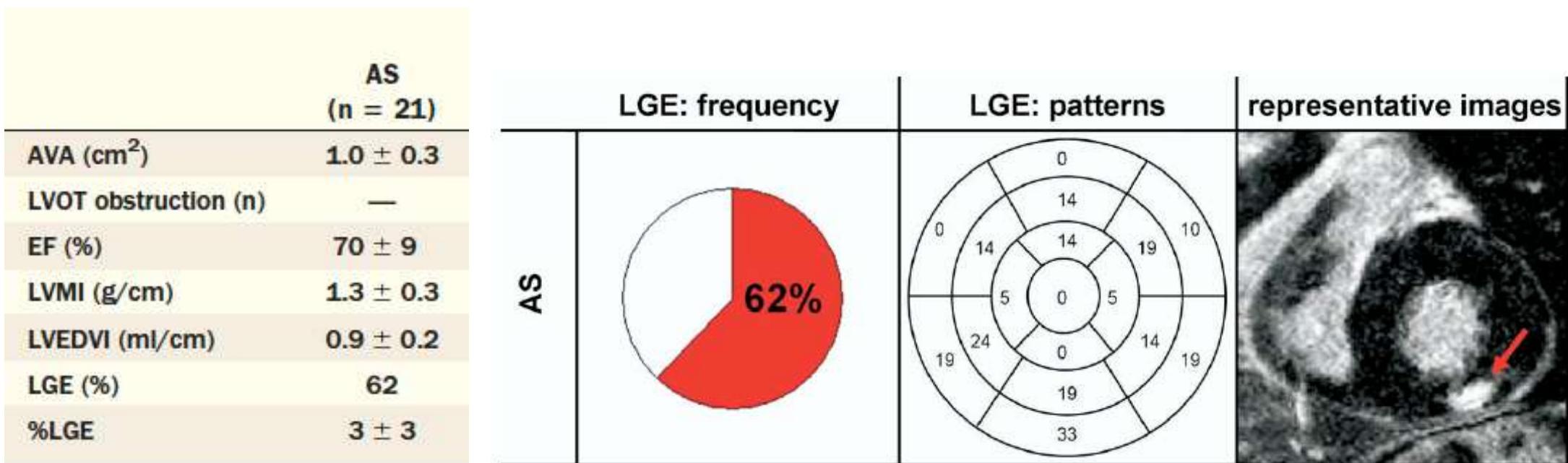
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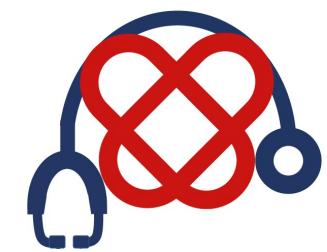




Noninvasive Detection of Fibrosis Applying Contrast-Enhanced Cardiac Magnetic Resonance in Different Forms of Left Ventricular Hypertrophy

Relation to Remodeling

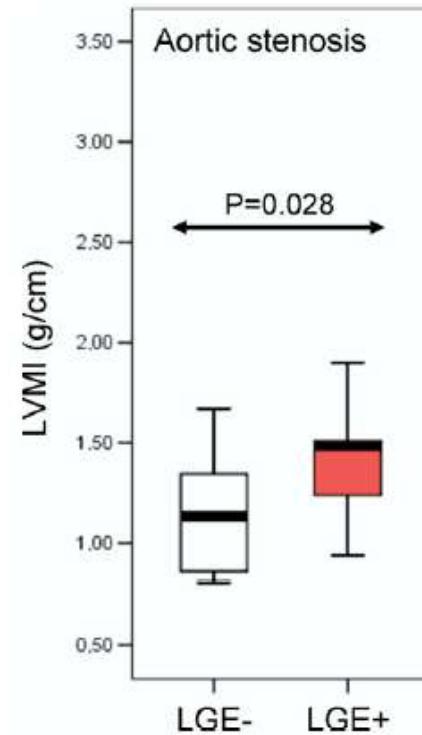
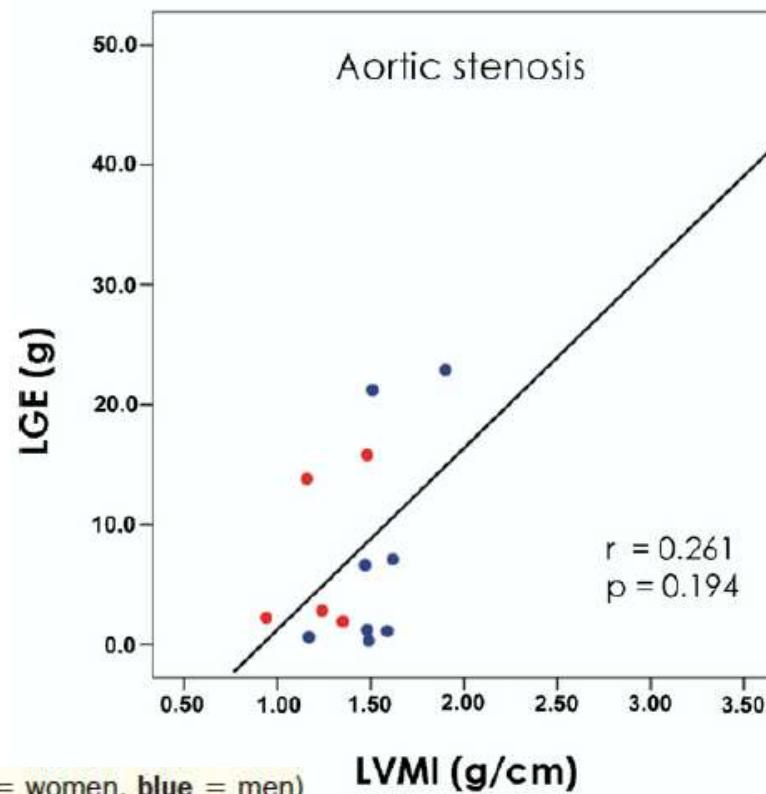


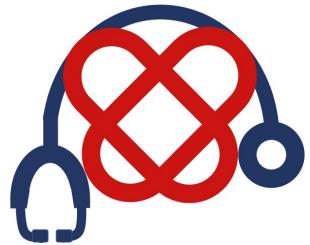


Noninvasive Detection of Fibrosis Applying Contrast-Enhanced Cardiac Magnetic Resonance in Different Forms of Left Ventricular Hypertrophy

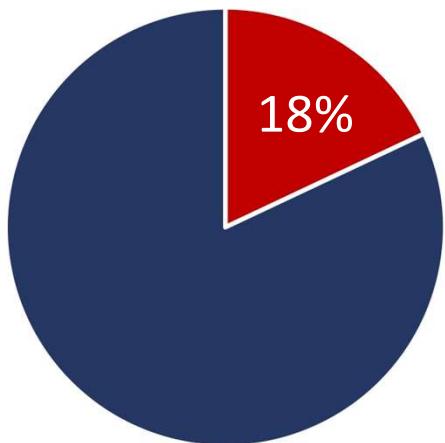
Relation to Remodeling

AS (n = 21)	
AVA (cm ²)	1.0 ± 0.3
LVOT obstruction (n)	—
EF (%)	70 ± 9
LVMI (g/cm)	1.3 ± 0.3
LVEDVI (ml/cm)	0.9 ± 0.2
LGE (%)	62
%LGE	3 ± 3



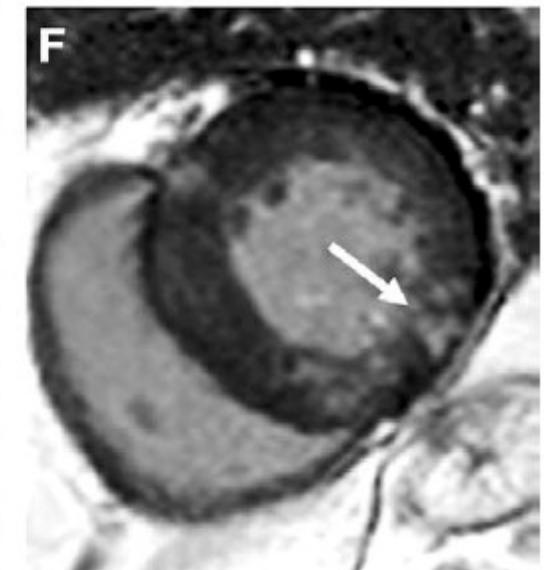
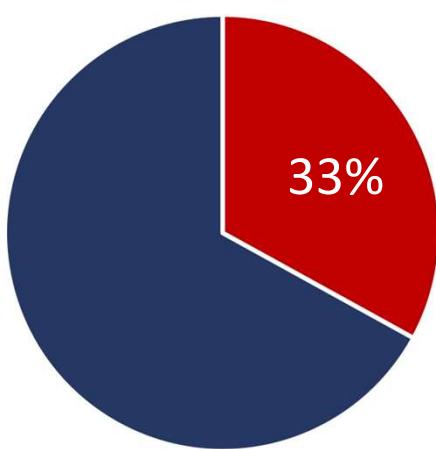


Myocardial Scar and Mortality in Severe Aortic Stenosis



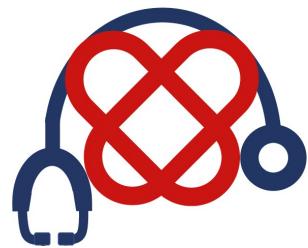
LGE infarct pattern

LGE non infarct pattern





HISTOIRE NATURELLE



Progression of Hypertrophy and Myocardial Fibrosis in Aortic Stenosis

	Natural History Cohort, n=61	AVR Cohort, n=38
Age, y	61±12	66±8
Male sex, n (%)	40 (66)	29 (76)
Body mass index, kg/m ²	28.3±5.6	27.3±3.6
Body surface area, m ²	1.88±0.21	1.86±0.16
Echocardiography		
Aortic stenosis severity, n (%)		
Mild	26 (43)	0
Moderate	21 (34)	0
Asymptomatic severe	14 (23)	0
Symptomatic severe	0	38 (100)

LV Assessment	Baseline Values	Annualized Absolute Change, units/y	P Value
Indexed left ventricular end-diastolic volume, mL/m ²	70±12	-1 (-4, 2)	0.015
Ejection fraction, %	75±8	0 (-2, 4)	0.23
Left ventricular mass index, g/m ²	75±20	3 (1, 5)	<0.0001
Maximum left ventricular wall thickness, mm	12±3	0.5 (0, 1)	<0.0001
Infarct late gadolinium enhancement, n (%)	8 (13)
Infarct late gadolinium enhancement mass, g	7.6±4.5	-0.1 (-1.4, 0.7)	0.56
Midwall late gadolinium enhancement, n (%)	16 (26)
Midwall late gadolinium enhancement mass, g	2.5 (0.8, 4.8)	1.6 (0.4, 4.1)	<0.0001

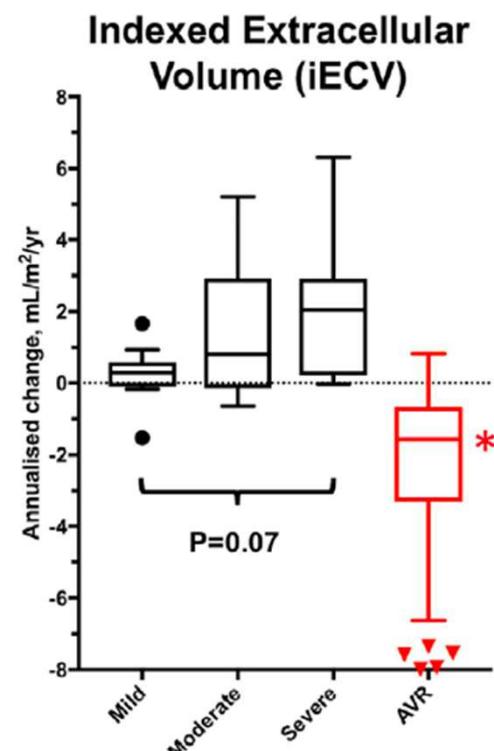
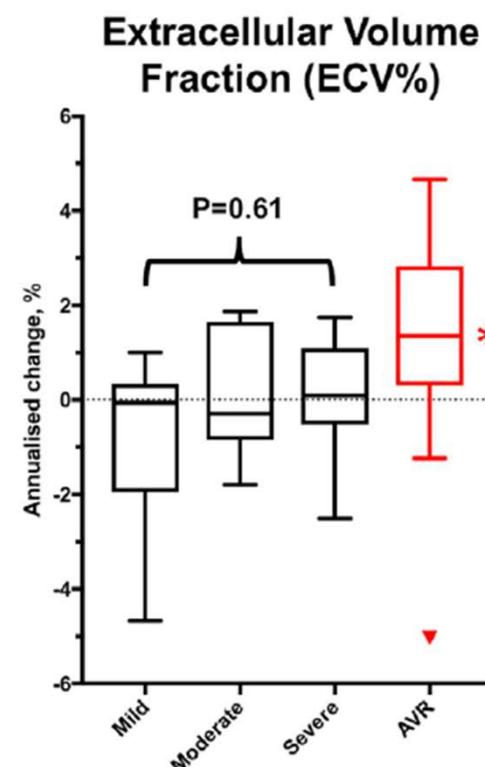
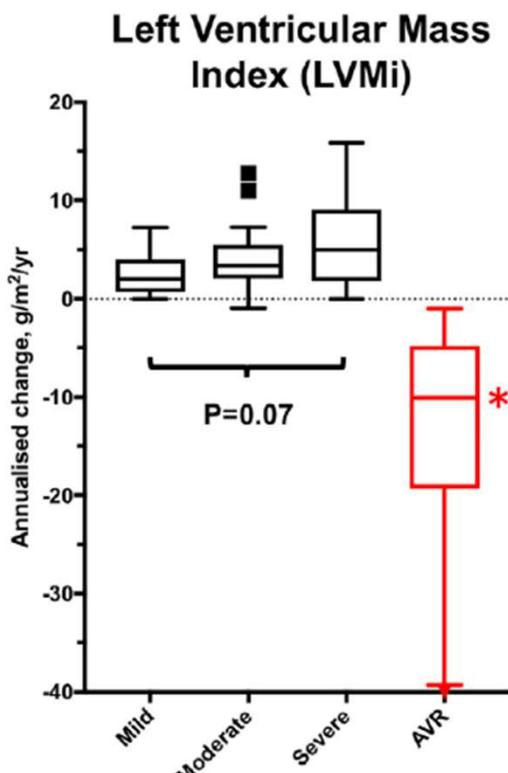


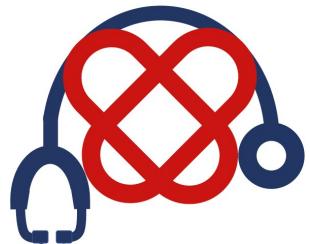
Progression of Hypertrophy and Myocardial Fibrosis in Aortic Stenosis

LV Assessment	Aortic Valve Stenosis Severity			P Value
	Mild, n=26	Moderate, n=21	Severe, n=14	
Infarct late gadolinium enhancement, n (%)	2 (8)	3 (14)	3 (21)	...
Midwall late gadolinium enhancement, n (%)	1 (4)	10 (48)	5 (36)	...
T1 mapping measures				
Extracellular volume fraction, %/y	0 (-1.9, 0.8)	0 (-0.8, 1.7)	0 (0.5, 0.9)	0.61
Total extracellular volume, mL/y	0.7 (0.0, 1.0)	1.5 (-0.2, 6.8)	3.7 (0.4, 6.0)	0.08
Indexed extracellular volume, mL/m ² per year	0.3 (-0.1, 0.6)	0.8 (-0.1, 2.9)	2.0 (0.2, 2.9)	0.07



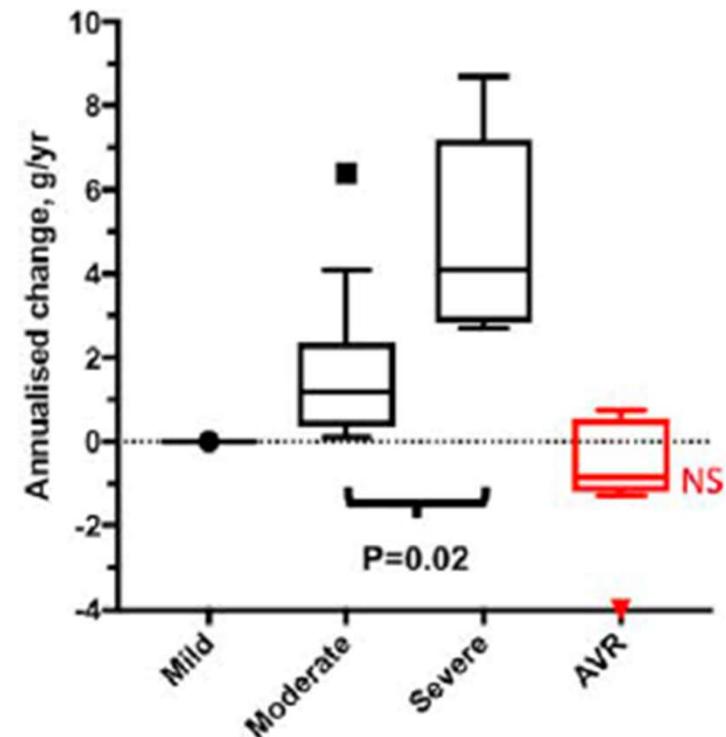
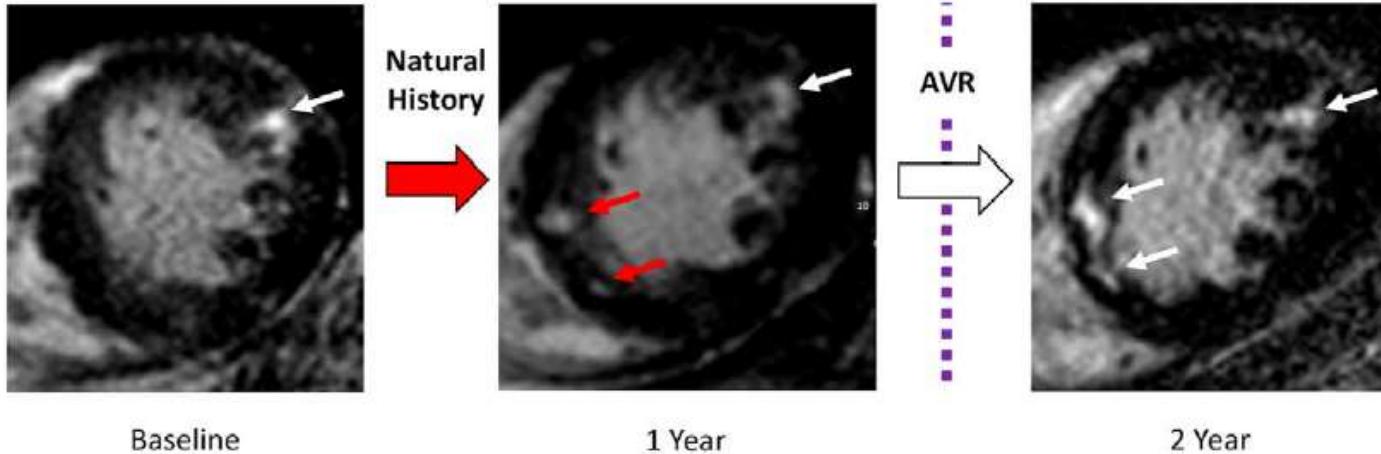
Progression of Hypertrophy and Myocardial Fibrosis in Aortic Stenosis

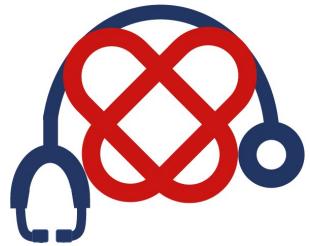




Progression of Hypertrophy and Myocardial Fibrosis in Aortic Stenosis

Late gadolinium enhancement



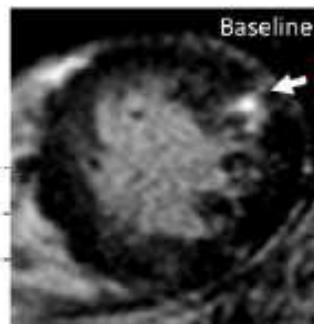


Progression of Hypertrophy and Myocardial Fibrosis in Aortic Stenosis

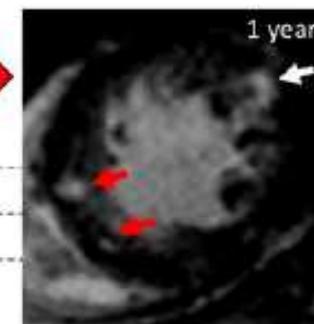
Natural History Group

Patient A

V max (m/s)	4.9
LVMi (g/m ²)	148
LGE (g)	5.0
iECV (mL/m ²)	42.0



Baseline



1 year

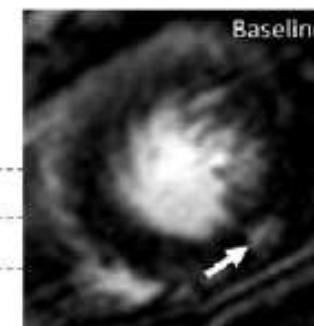
AVR Group

Increasing aortic stenosis severity with time

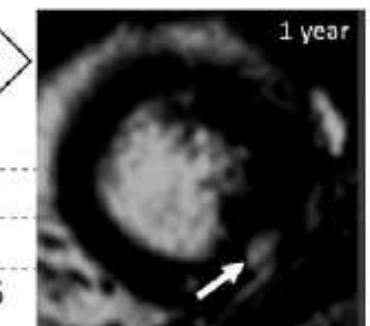
AVR

Patient B

V max (m/s)	5.3
LVMi (g/m ²)	116
LGE (g)	2.6
iECV (mL/m ²)	31.9



Baseline



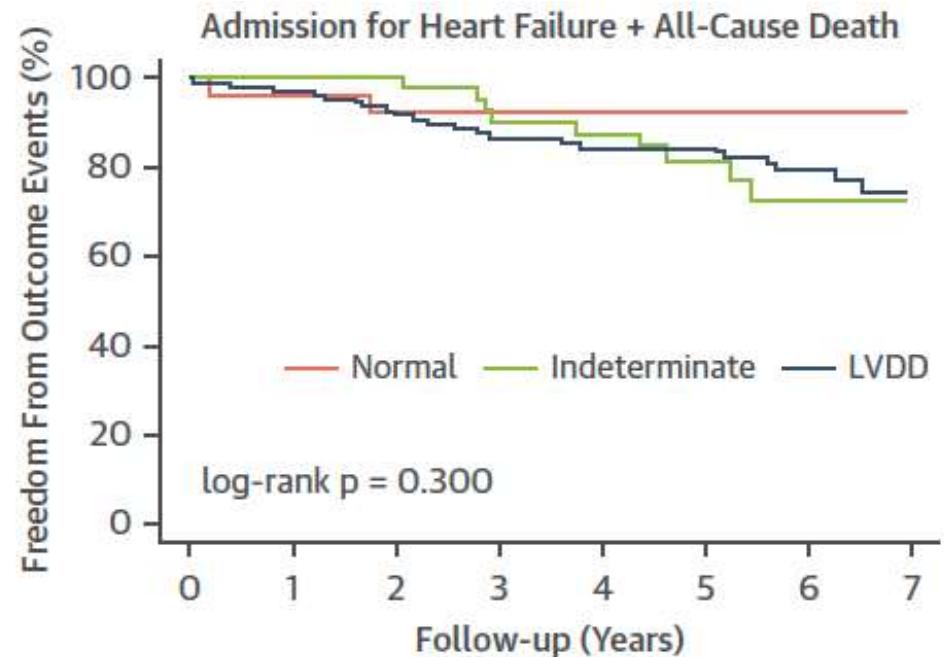
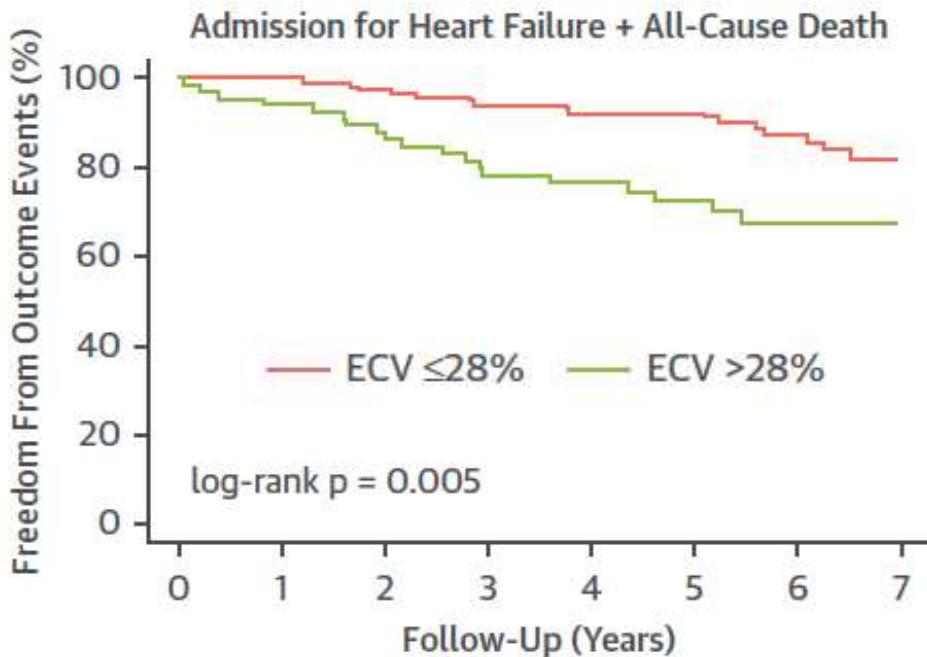
1 year



IMPACT SUR LE PRONOSTIC



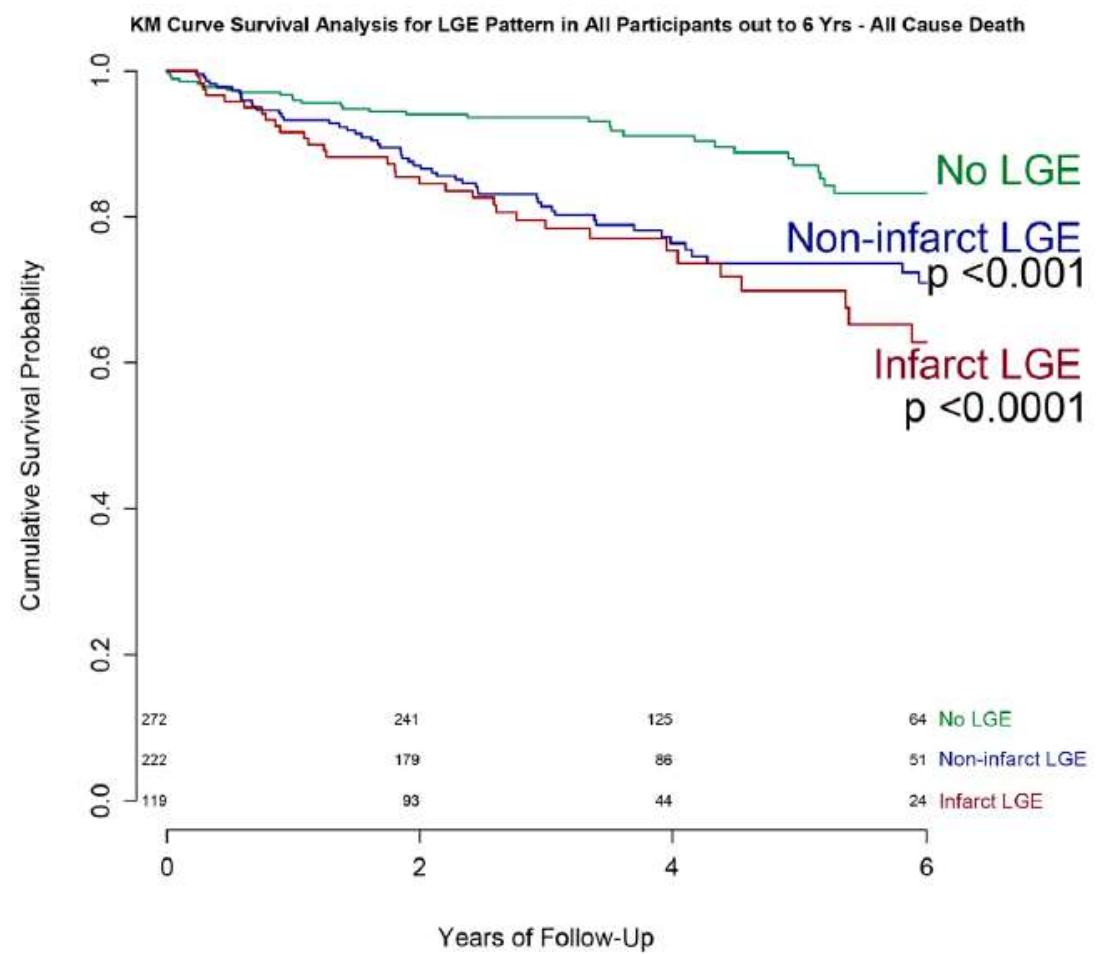
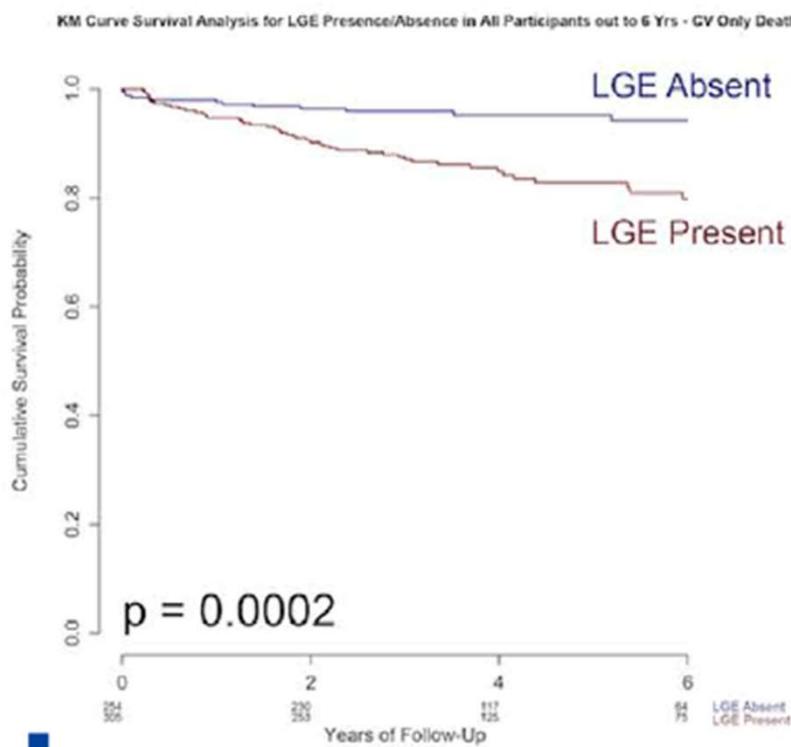
Diffuse Myocardial Fibrosis and Diastolic Function in Aortic Stenosis





Myocardial Scar and Mortality in Severe Aortic Stenosis

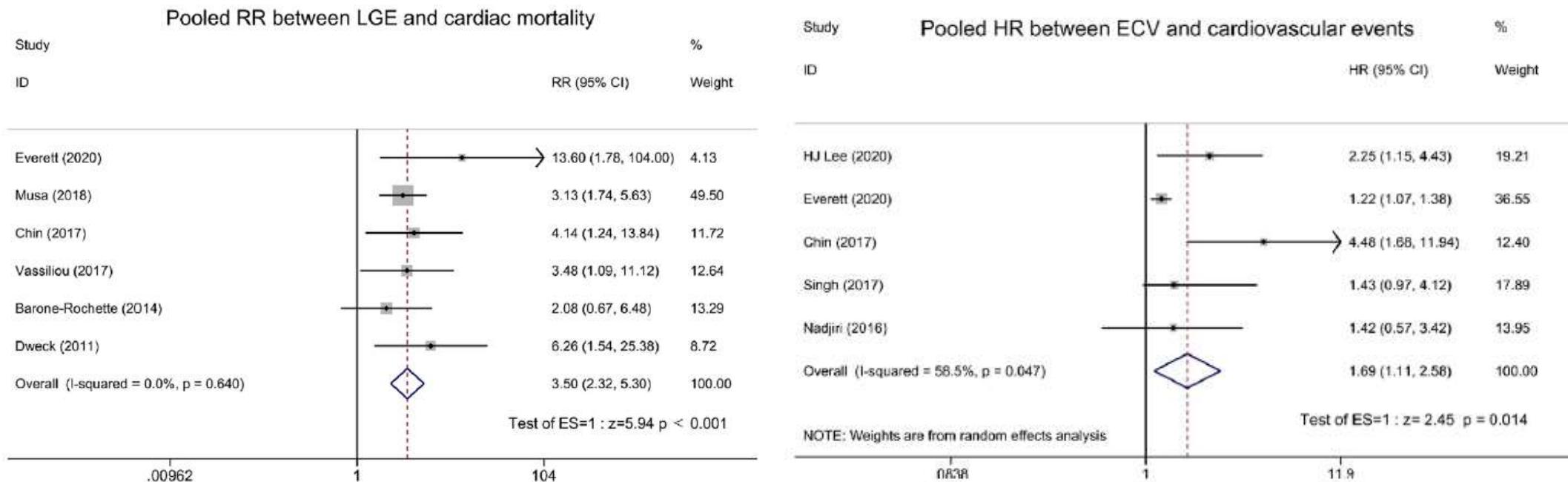
Cardiovascular Mortality



Musa T, et al. *Circulation*. 2018;138:1935–1947

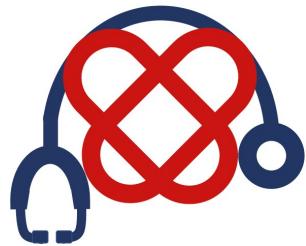


Prognostic value of cardiac magnetic resonance in patients with aortic stenosis: A systematic review and meta-analysis





LA PLACE DE L'AMYLOSE

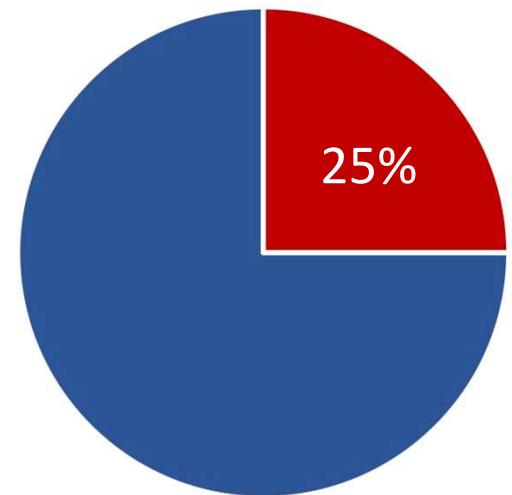


Senile systemic amyloidosis affects 25% of the very aged: A population-based autopsy study

Population finlandaise

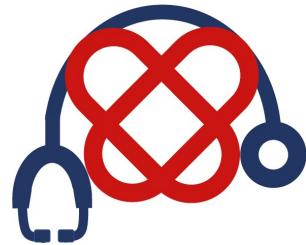
256 autopsies de sujets de plus de 85 ans

Âge de 85 à 106 ans (moyenne 93 ans)

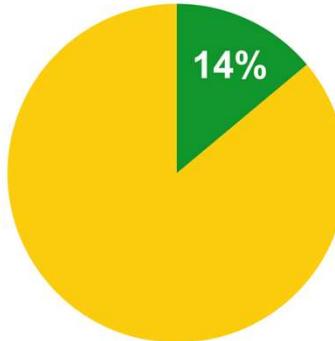
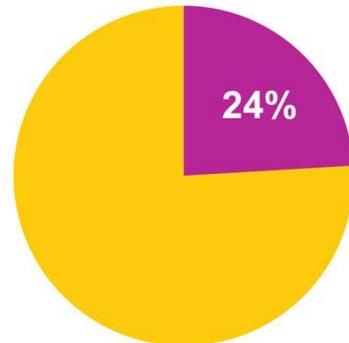


Amylose cardiaque à transthyrétine :

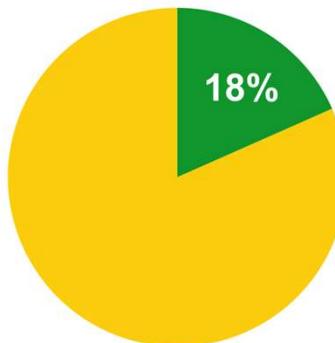
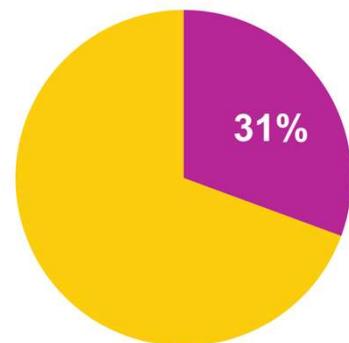
- 25% (63/256) de la population de l'étude
- 17% (11/65) des 85–89 ans
- 23% (29/127) des 90–94 ans
- 32% (18/56) des 95–100 ans
- 63% (5/8) des > 100 ans



PRÉVALENCE DU RAO DANS L'AMYLOSE CARDIAQUE



Whole population



Patients over 80 years

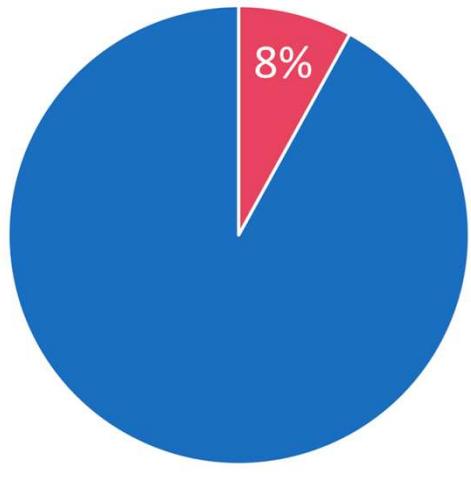
■ Aortic stenosis

■ Severe aortic stenosis

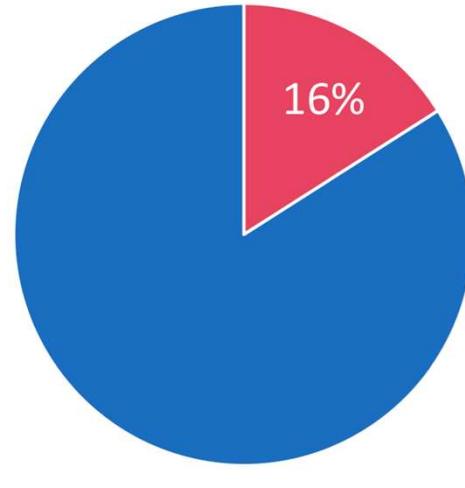
■ No aortic stenosis



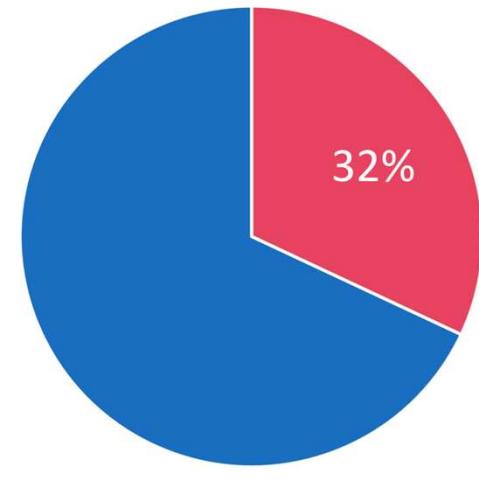
Cardiac amyloidosis is prevalent in older patients with aortic stenosis and carries worse prognosis



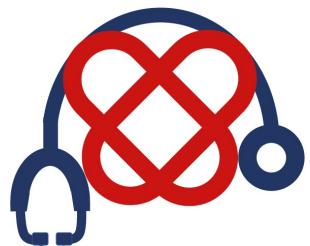
Population totale
(n=113)



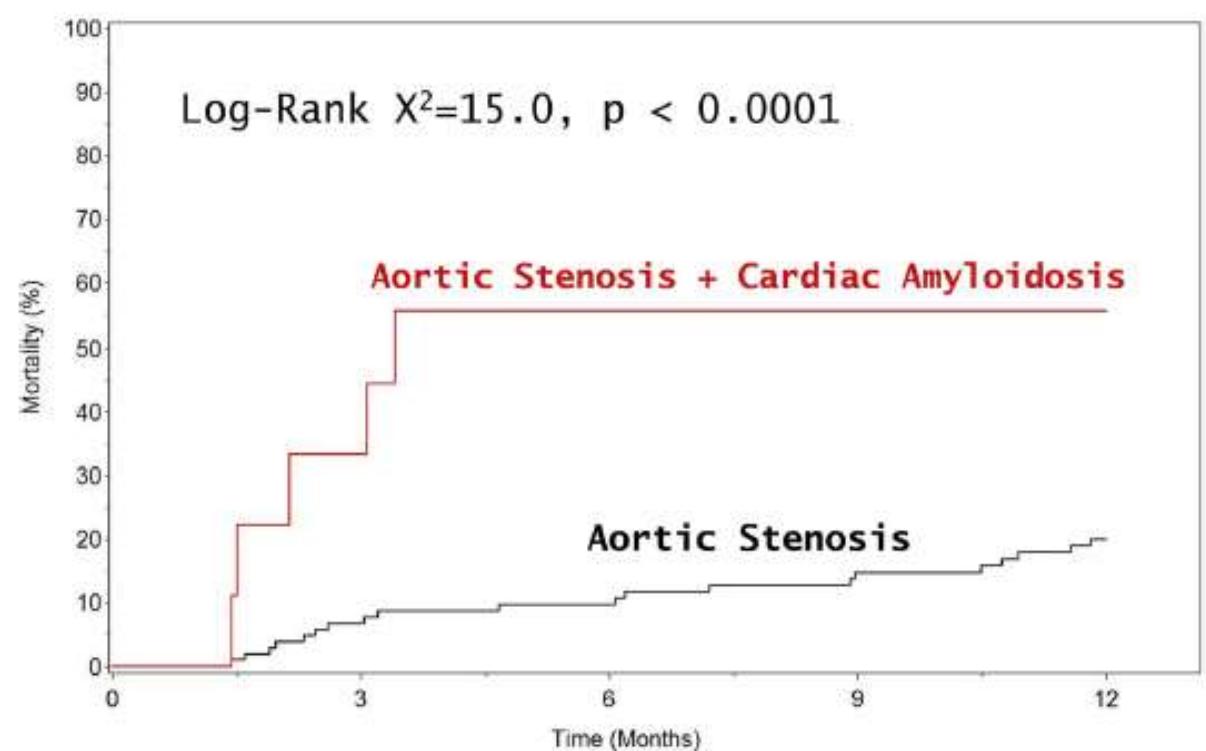
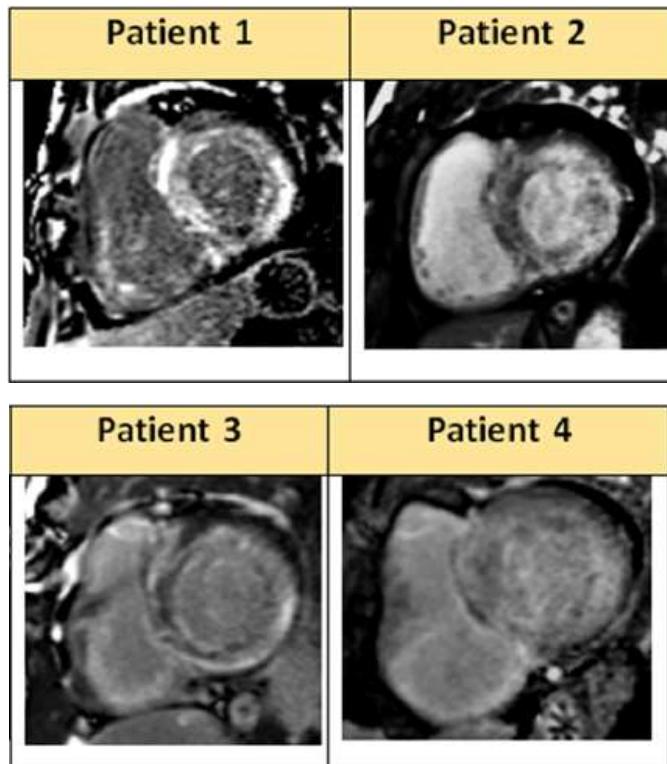
Patients > 74 ans
(n=57)



Hommes > 74 ans
(n=25)



Cardiac amyloidosis is prevalent in older patients with aortic stenosis and carries worse prognosis

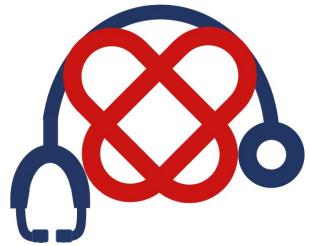




SYNTÈSE

RÉTRÉCISSEMENT AORTIQUE &

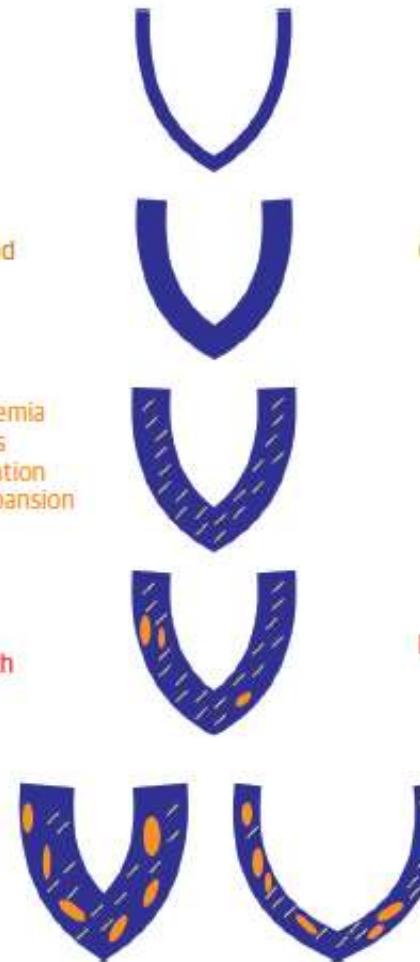
FIBROSE MYOCARDIQUE EN IRM



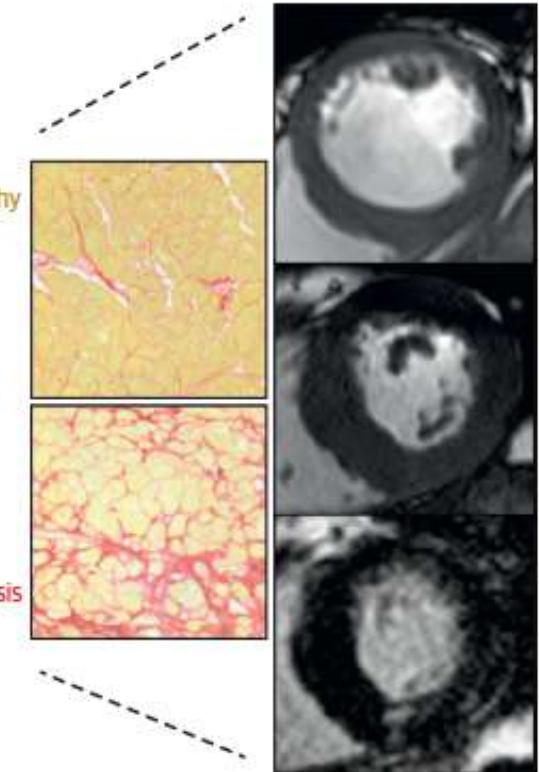
PHYSIOPATHOLOGIE



Increased afterload
↓
Supply-demand ischemia
Mechanical stress
Myofibroblast infiltration
Extracellular matrix expansion
↓
Myocyte cell death

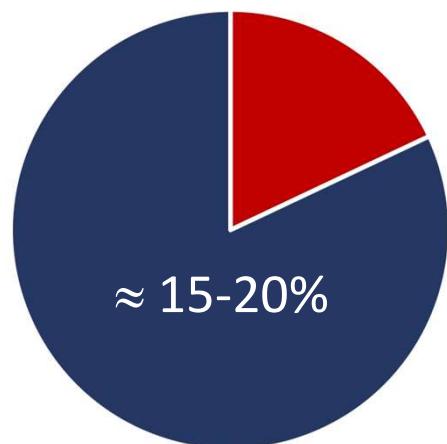


Cellular hypertrophy
↓
Diffuse fibrosis
↓
Replacement fibrosis
↓
Heart failure
Cardiac death

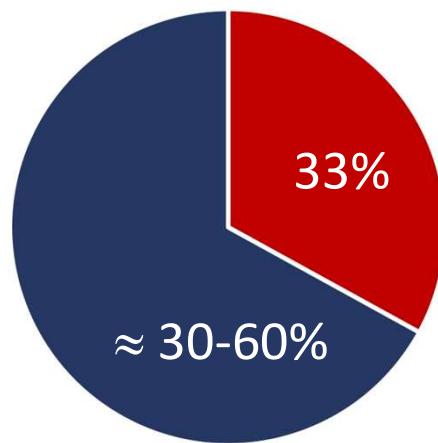




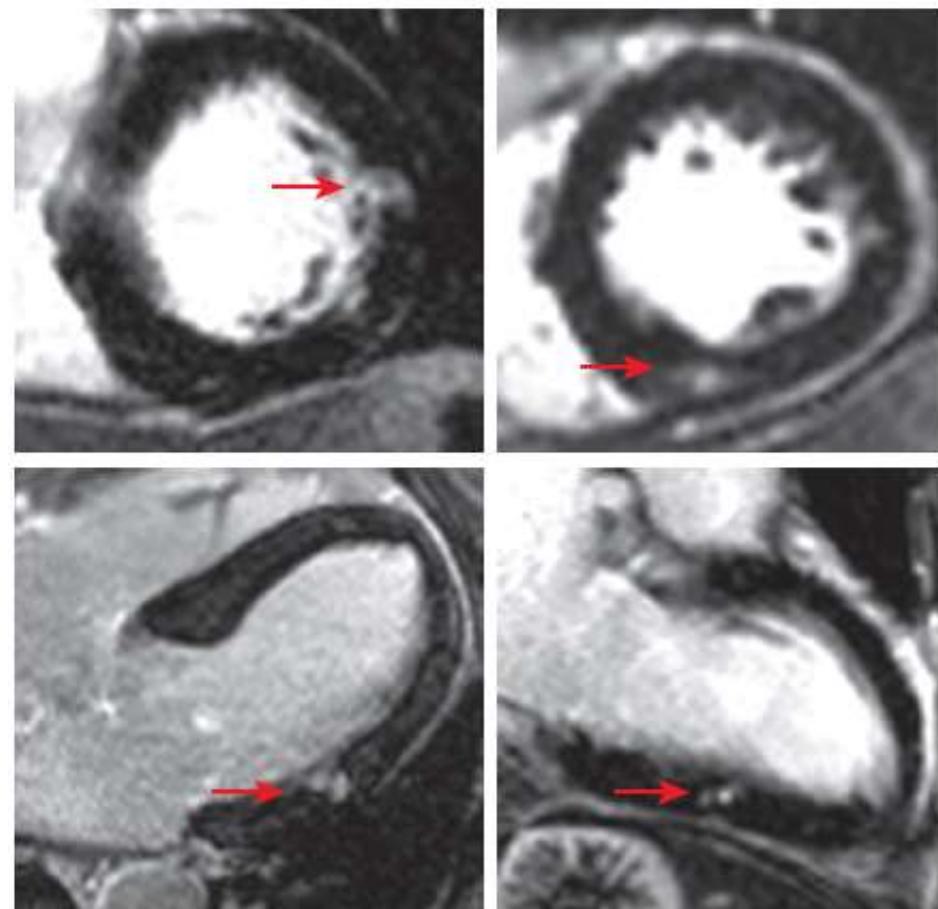
PRÉVALENCE & SÉMIOLOGIE

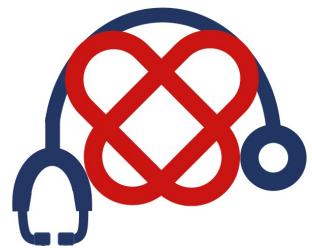


LGE infarct pattern

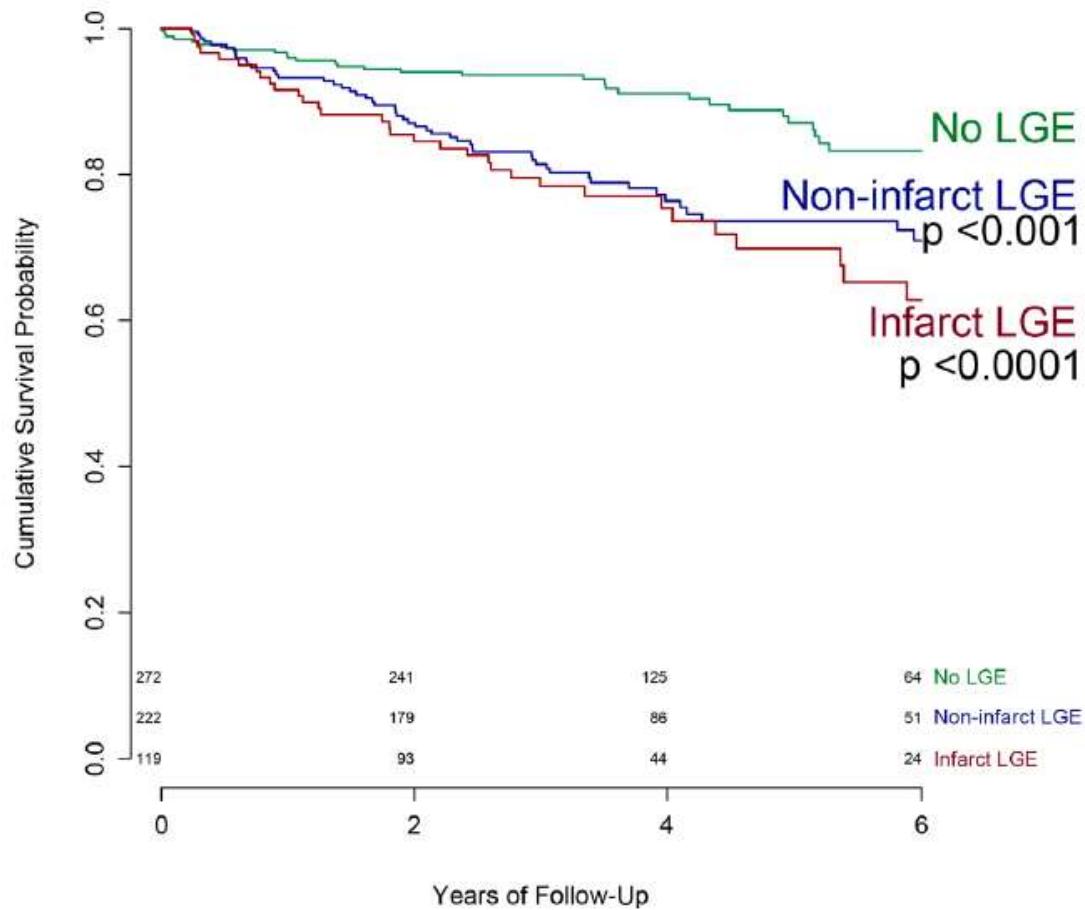


LGE non infarct pattern



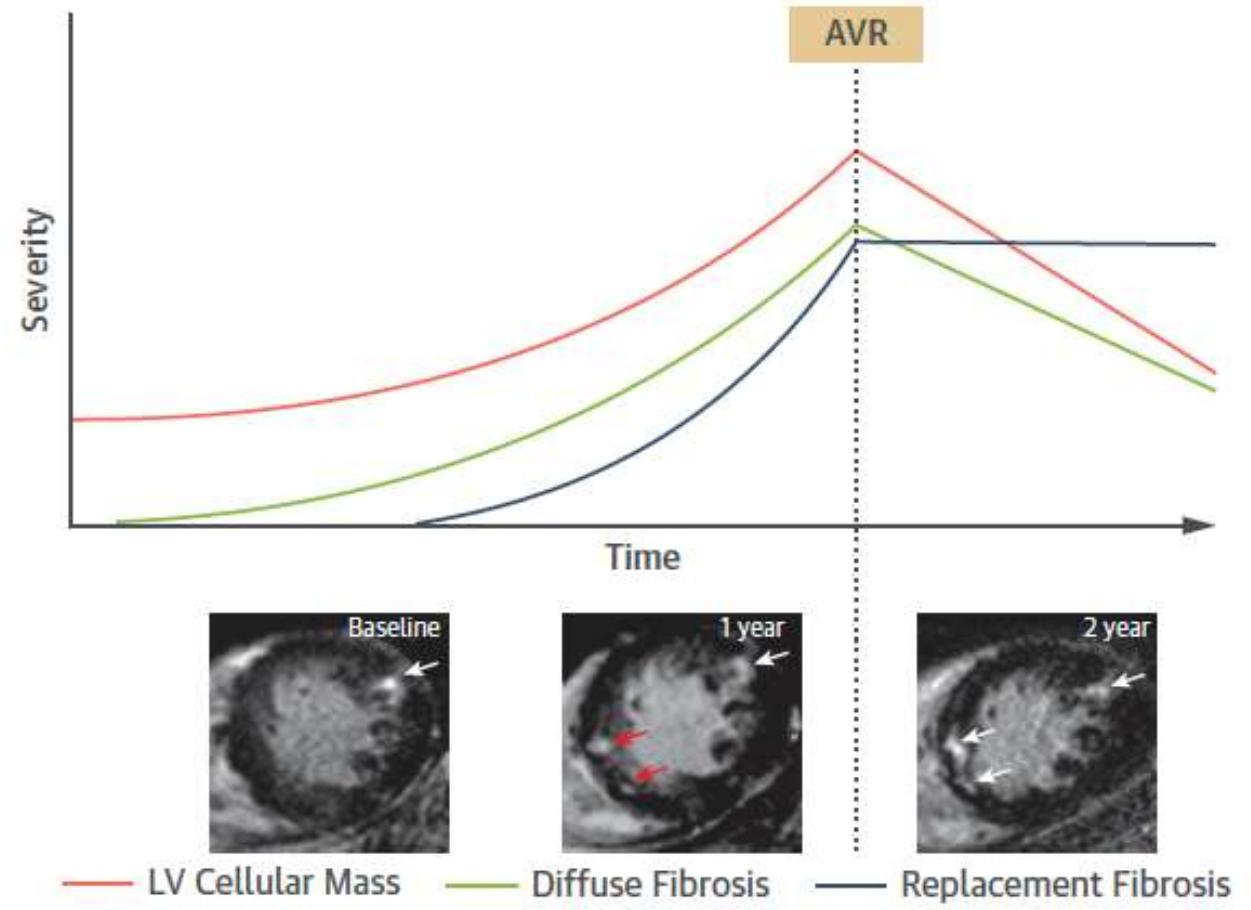
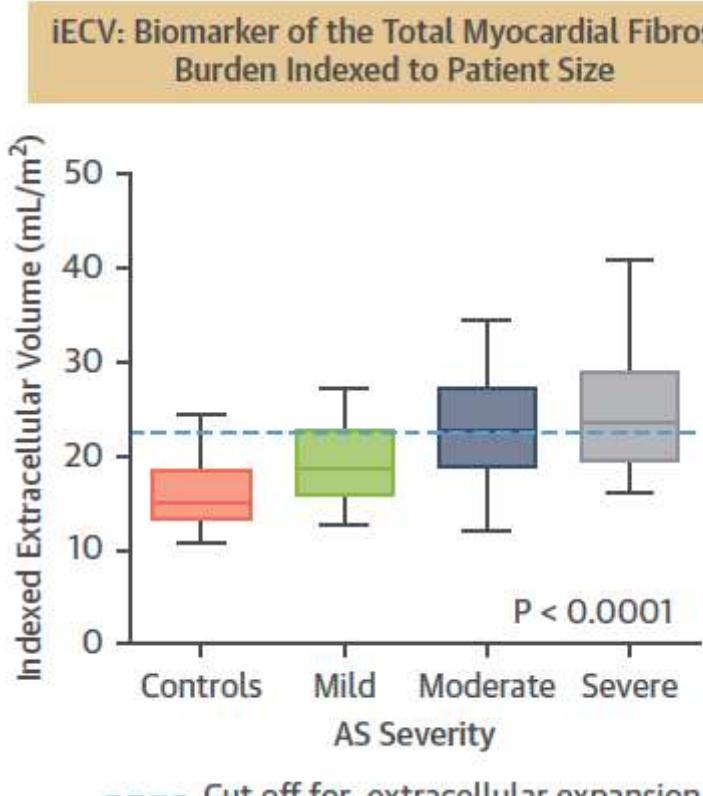


PRONOSTIC

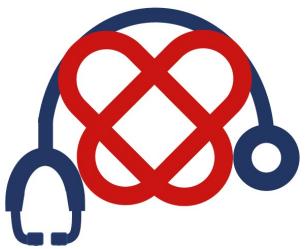




HISTOIRE NATURELLE



Bing R, et al. J Am Coll Cardiol Img. 2019;12:283–96



PERSPECTIVES

