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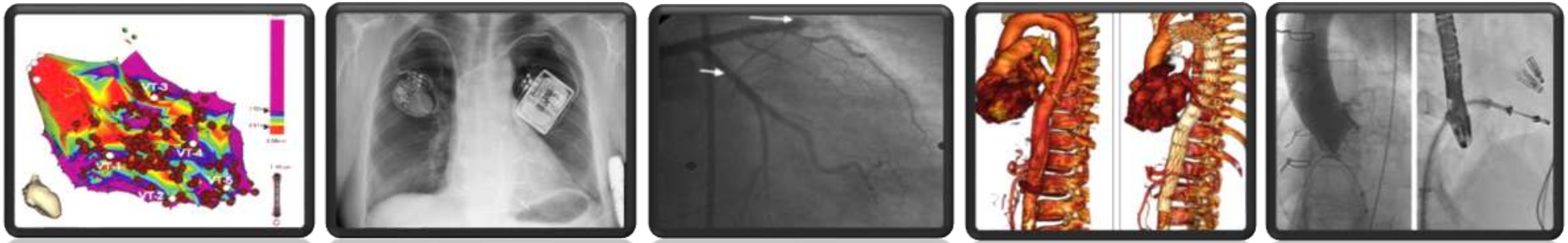
Hormones and Cardiomyopathy



The Invisible Heart: Cardiovascular Disease in Women
14th National Congress of Cardiology
13.-14.12.2025
Baku

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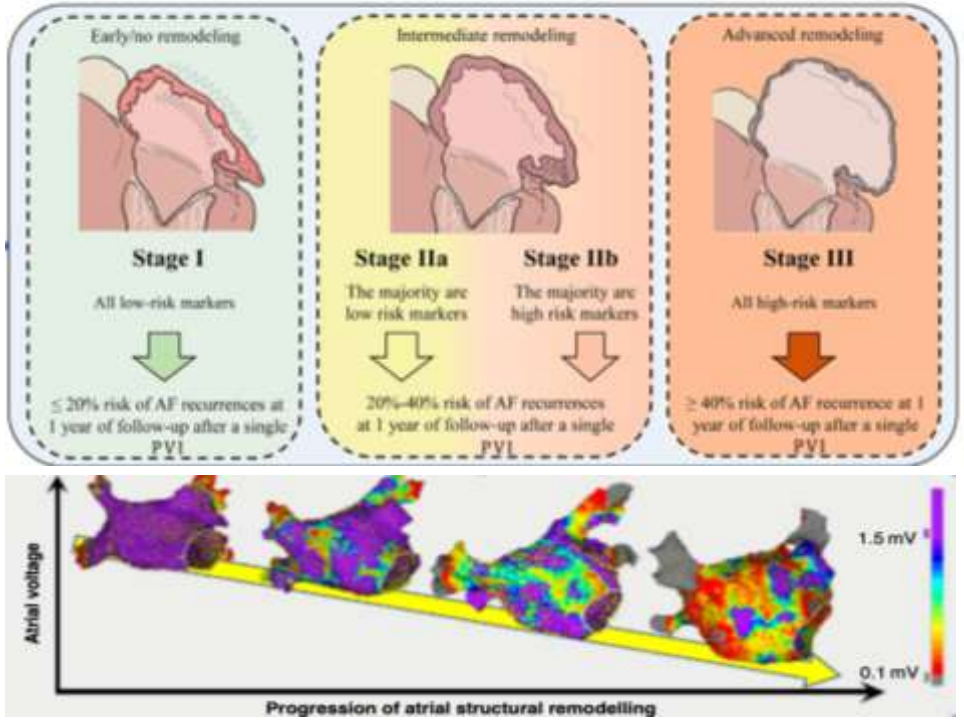
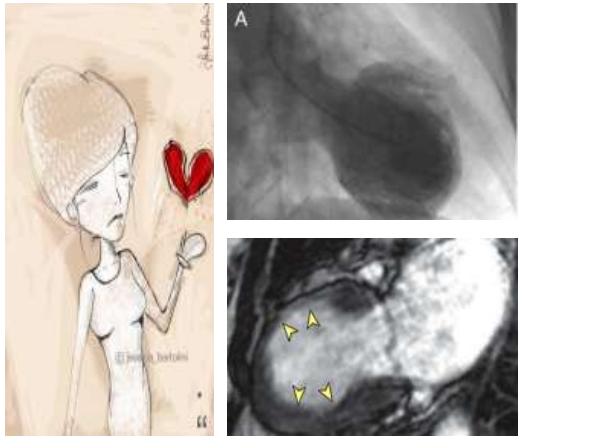
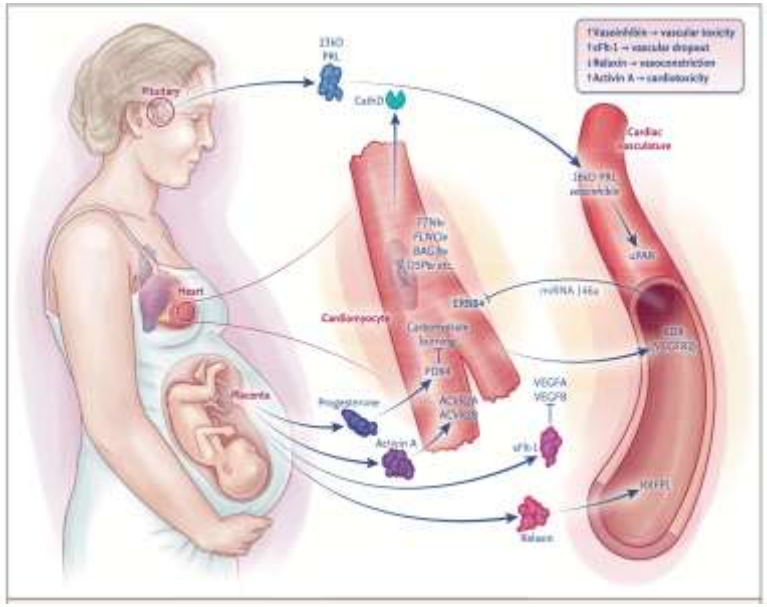
I, *Ibrahim Akin* DO NOT have a financial interest / arrangement or affiliation with one or more organizations that could be perceived as a real or apparent conflict of interest in the context of the subject of this presentation.

Impact of Hormones in Cardiomyopathy

Atrial cardiomyopathy stage







Shoureshi P, et al. J Am Coll Cardiol 2024;83:2214-32
Goette A, et al Europace 2024
Arany Z. NEJM 2024

Atrial Cardiomyopathy

EHRA/HRS/APHRS/SOLAECE expert consensus on Atrial cardiomyopathies: Definition, characterisation, and clinical implication☆,☆☆

1.1. *Definition of atrial cardiomyopathy*

The working group proposes the following working definition of atrial cardiomyopathy: 'Any complex of structural, architectural, contractile or electrophysiological changes affecting the atria with the potential to produce clinically-relevant manifestations' (Table 1).

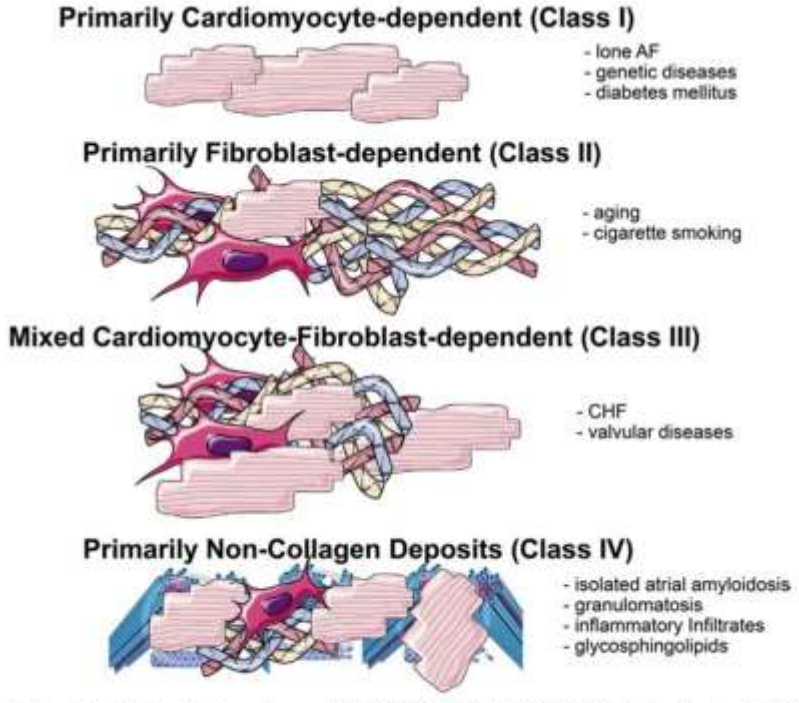
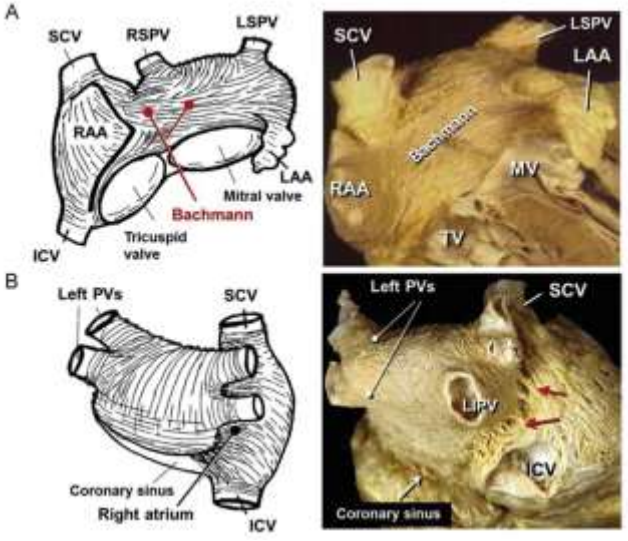
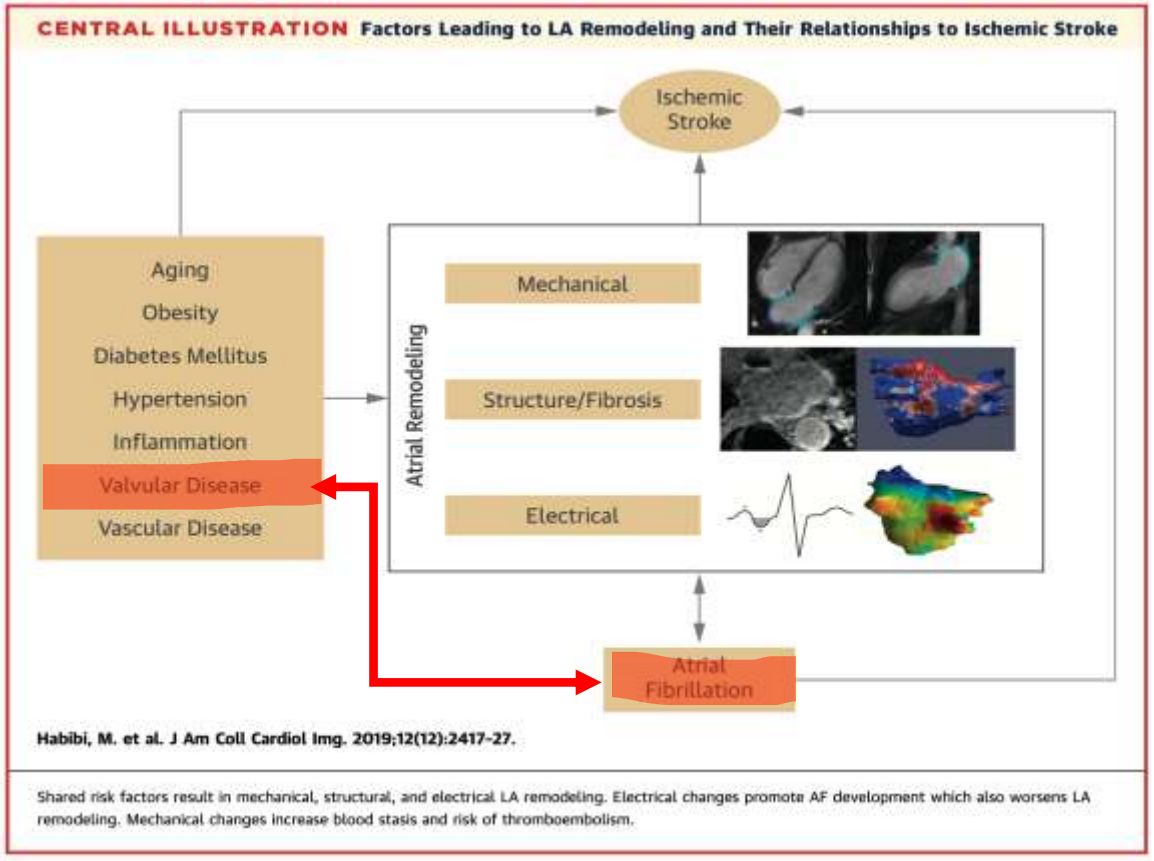


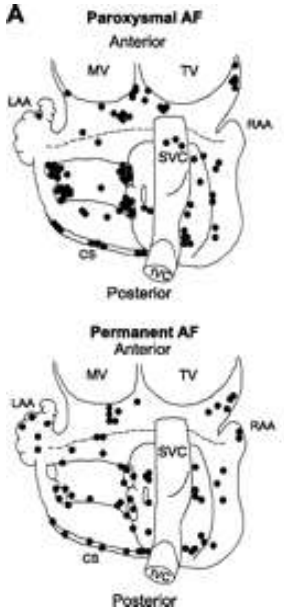
Fig. 1. Histological and pathopysiological classification of atrial cardiomyopathies (EHRA/HRS/APHRS/SOLAECE): EHRAS classification. The EHRAS class may vary over time in the cause of the disease and may differ at various atrial sites. Of note, the nature of the classification is purely descriptive. EHRAS I–IV is not intended to describe disease progression from EHRAS I to EHRAS IV.

Goette A, et al. J Arrhythmia 2016;32:247-78

Atrial Cardiomyopathy – Pathophysiology



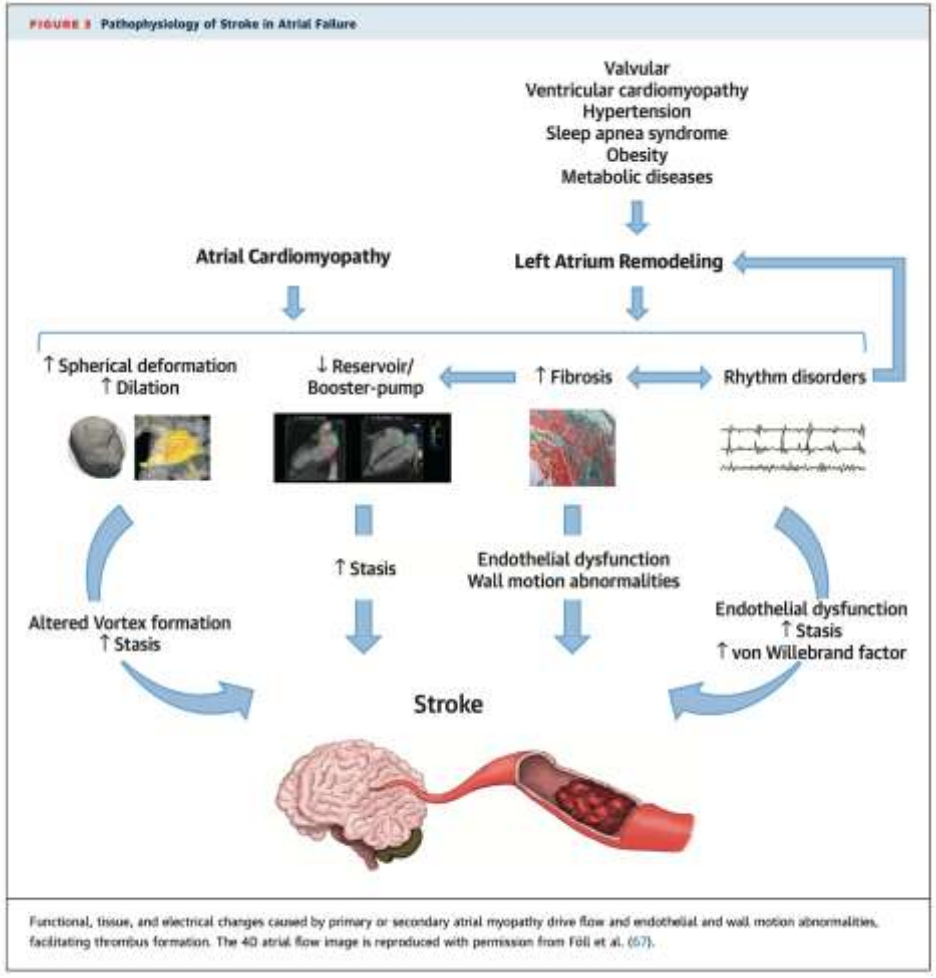




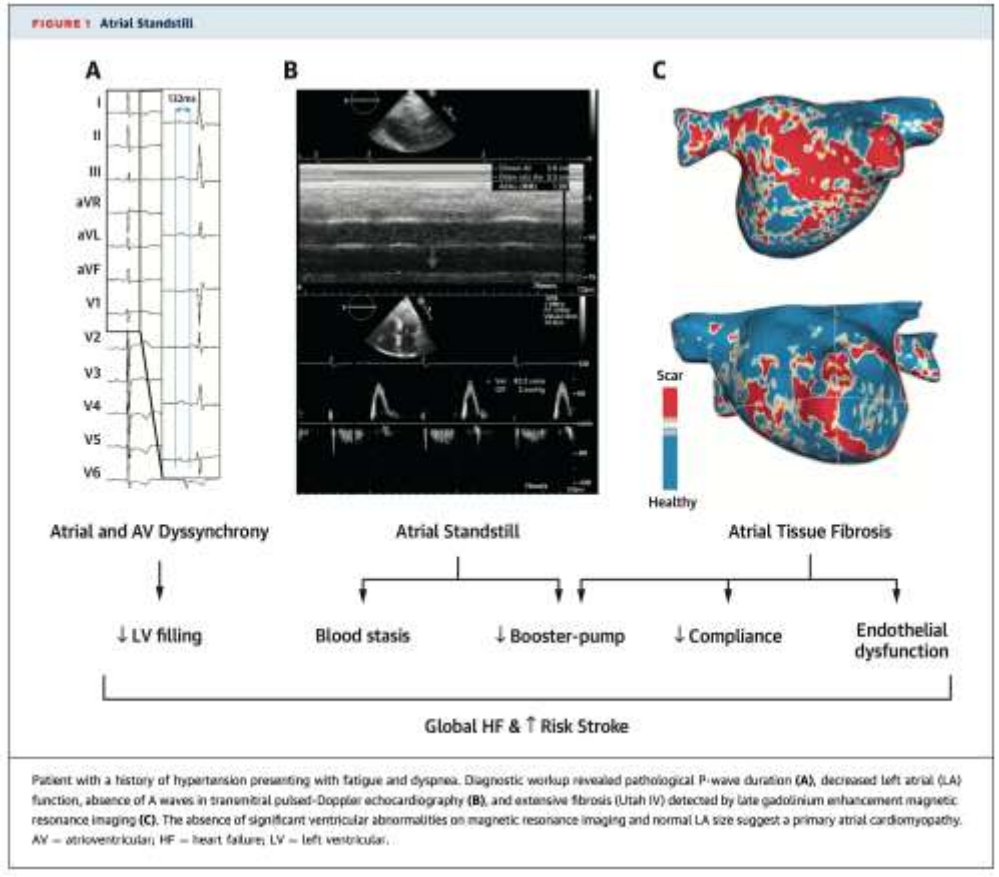
To date the pathogenesis of ACM has not been completely clarified

Goette A, et al. J Arrhythmia 2016;32:247-78
Habibi M, et al. J Am Coll Cardiol 2019;12:2417-27

Atrial Cardiomyopathy – Stroke / Heart Failure



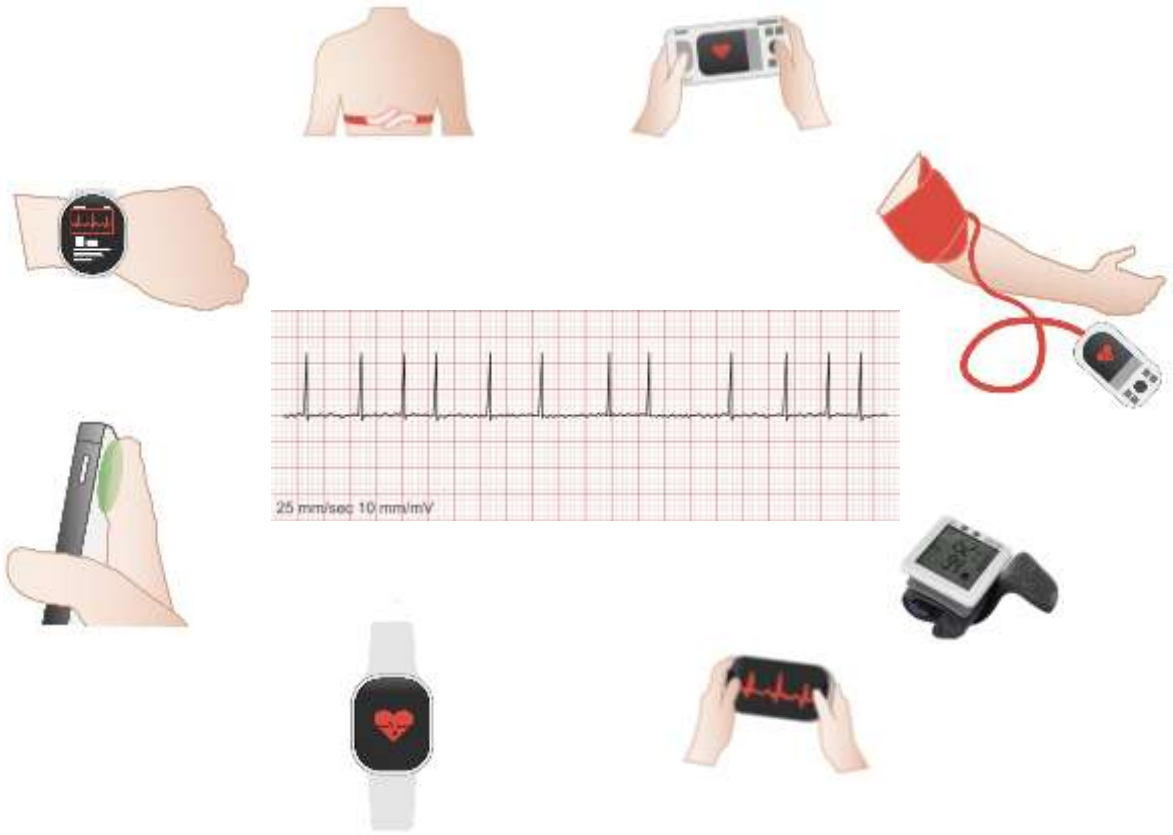
Functional, tissue, and electrical changes caused by primary or secondary atrial myopathy drive flow and endothelial and wall motion abnormalities, facilitating thrombus formation. The 4D atrial flow image is reproduced with permission from Föll et al. (67).

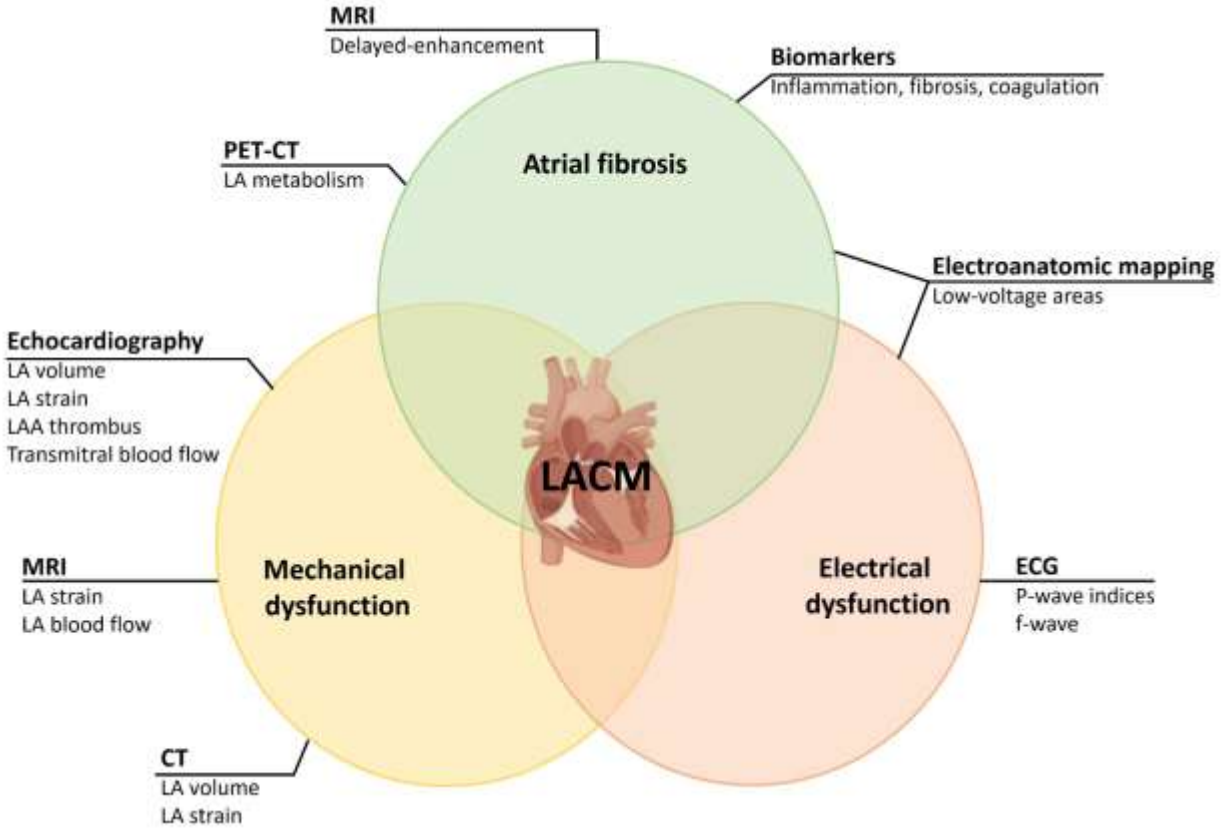


Patient with a history of hypertension presenting with fatigue and dyspnea. Diagnostic workup revealed pathological P-wave duration **(A)**, decreased left atrial (LA) function, absence of A waves in transmitral pulsed-Doppler echocardiography **(B)**, and extensive fibrosis (Utah IV) detected by late gadolinium enhancement magnetic resonance imaging **(C)**. The absence of significant ventricular abnormalities on magnetic resonance imaging and normal LA size suggest a primary atrial cardiomyopathy. AV = atrioventricular; HF = heart failure; LV = left ventricular.

Bisbal F, et al. J Am Coll Cardiol 2020;75:222-32
Van Gelder, et al. Eur Heart J 2024

Arrhythmie-induced Cardiomyopathy – Diagnostic

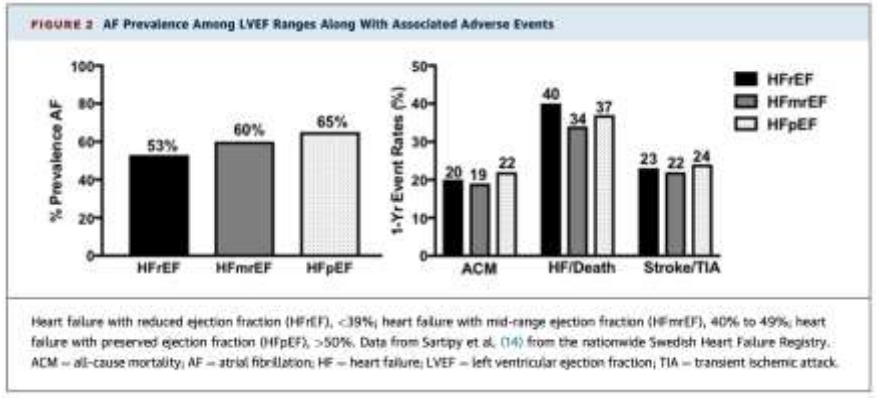


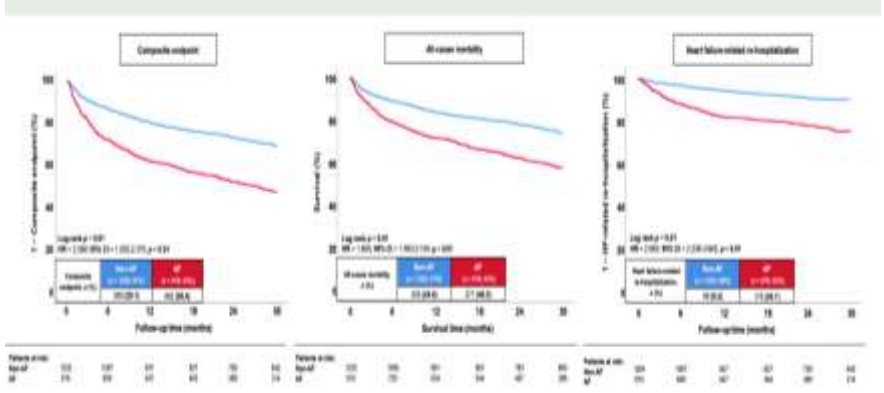


There are still no established criteria for the diagnosis of atrial cardiomyopathy

Kreimer F, et al. Front Cardiovasc Med 2022

AF in Different Form of HF





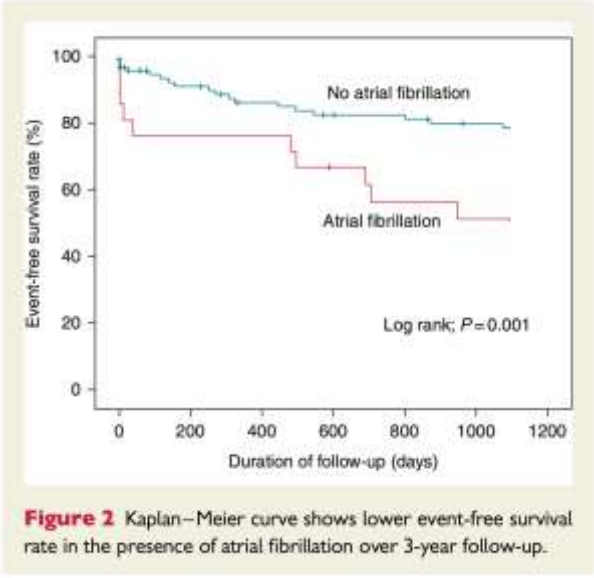
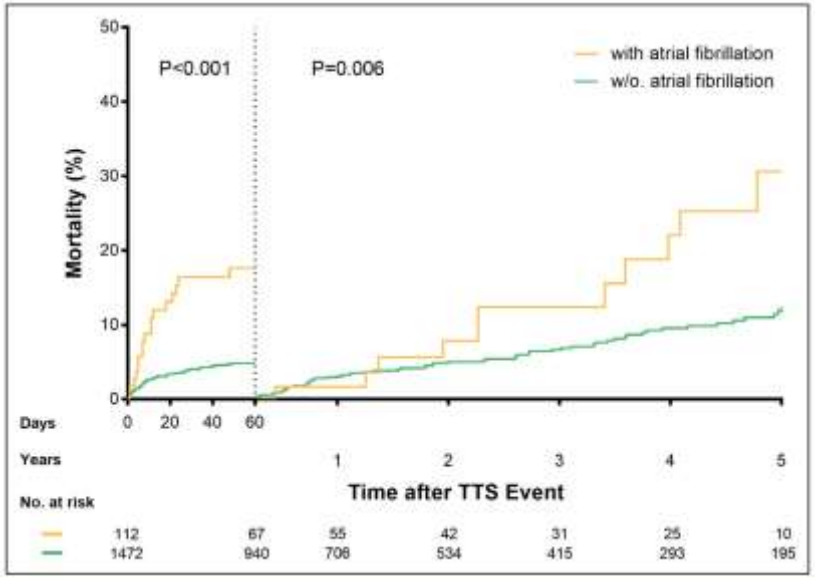
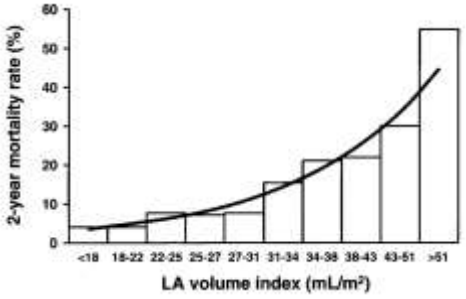


Figure 2 Kaplan–Meier curve shows lower event-free survival rate in the presence of atrial fibrillation over 3-year follow-up.

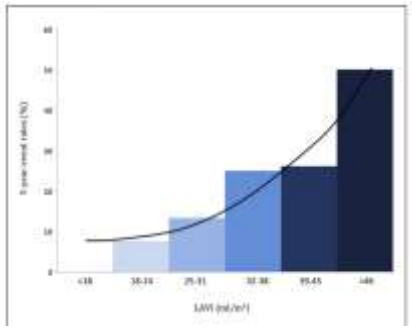


Schupp T, … Akin I. Eur J Prev Cardiol 2024
El-Battrawy I, … Akin I. Europace 2017;19:1288-92
El-Battrawy I, … Akin I. JAHA 2021;10:e14059
Carlise MA, et al. JACC Heart Fail 2019;7:447-56

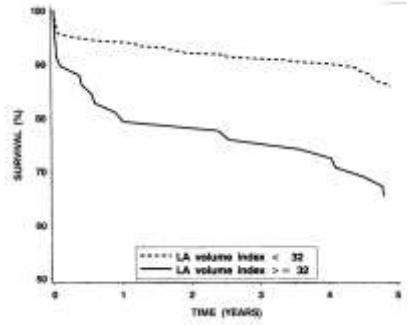
ACM – Impact on different Cohorts



ACS



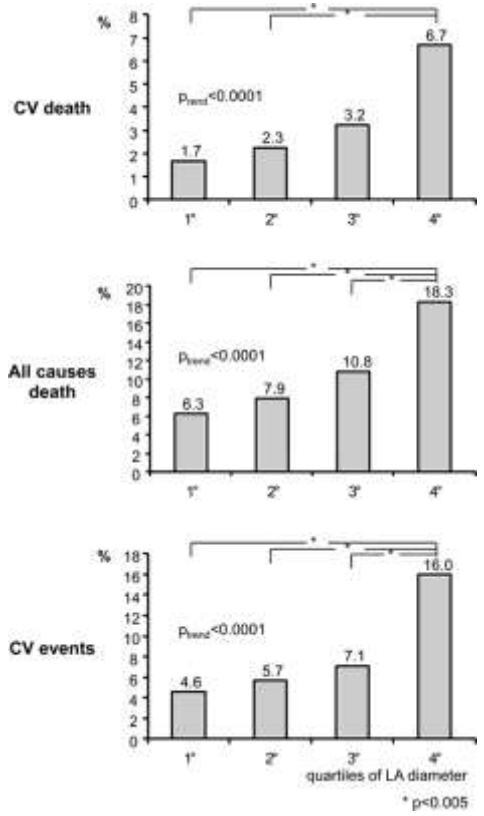
Diabetics



CCS

Marker	Model 1*
Individual markers	
PTFV$_1$ alone‡ (n=4954)	1.08 (1.04–1.11)
Left atrial dimension alone§ (n=4919)	1.15 (1.03–1.29)
NT-proBNP alone‖ (n=3992)	1.17 (1.11–1.24)
AF alone¶ (n=5120)	2.18 (1.85–2.57)

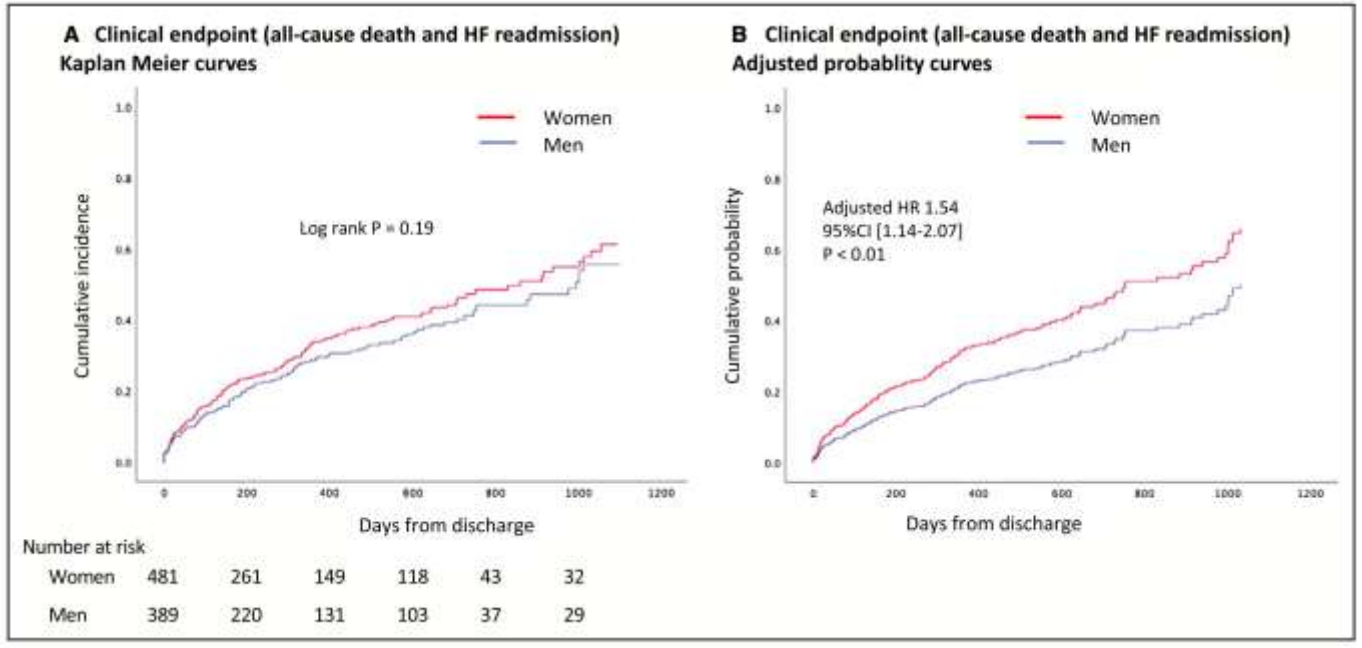
Stroke



Beinart R, et al. J Am Coll Cardiol 2004;44:327-34
Poulsen MK, et al. J Am Coll Cardiol 2013;62:2416-21
Moller JE, et al. Circulation 2003;107:2207-12
Kamel H, et al. Stroke 2018;49:980-6
Bombelli L, et al. Hypertension 2014

Sex Differences in HFpEF – PURSUIT-HFpEF

Variable	Women	Men	*P* Value
Number	481*	389*	
Age, y	82.23 (6.63)	79.75 (6.93)	<0.001
Body mass index	21.73 (4.82)	22.19 (3.80)	0.124
Body weight, kg	47.77 (11.81)	58.92 (11.49)	<0.001
Obesity (body mass index ≥25)	90 (19.0)	82 (21.4)	0.387
Systolic blood pressure, mm Hg	118.61 (18.12)	119.44 (17.88)	0.502
Diastolic blood pressure, mm Hg	66.20 (11.91)	65.34 (12.01)	0.296
Heart rate, bpm	72.56 (13.47)	70.09 (13.13)	0.007
NYHA class			0.011
NYHA I	153 (32.4)	155 (40.4)	
NYHA II	268 (56.8)	207 (53.9)	
NYHA III	43 (9.1)	17 (4.4)	
NYHA IV	8 (1.7)	5 (1.3)	
Frail†	186 (38.8)	73 (18.8)	<0.001
HFA-PEFF score			0.758
Low (0–1)	6 (1.3)	4 (1.1)	
Intermediate (2–4)	132 (28.0)	115 (31.3)	
High (5–6)	317 (69.7)	248 (67.6)	
History			
Hypertension	395 (82.5)	340 (87.6)	0.037
Dyslipidemia	207 (43.3)	149 (38.7)	0.188
Diabetes mellitus	149 (31.2)	138 (35.9)	0.147
Anemia	330 (68.8)	284 (73.4)	0.136
Atrial fibrillation	178 (37.2)	153 (39.3)	0.528



Sotomi Y, et al. J Am Heart Assoc 2021

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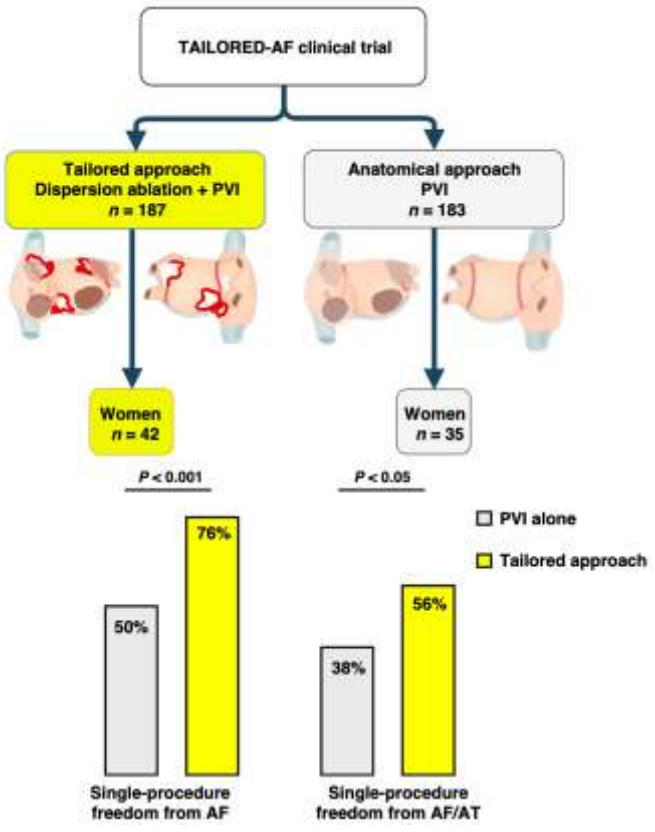
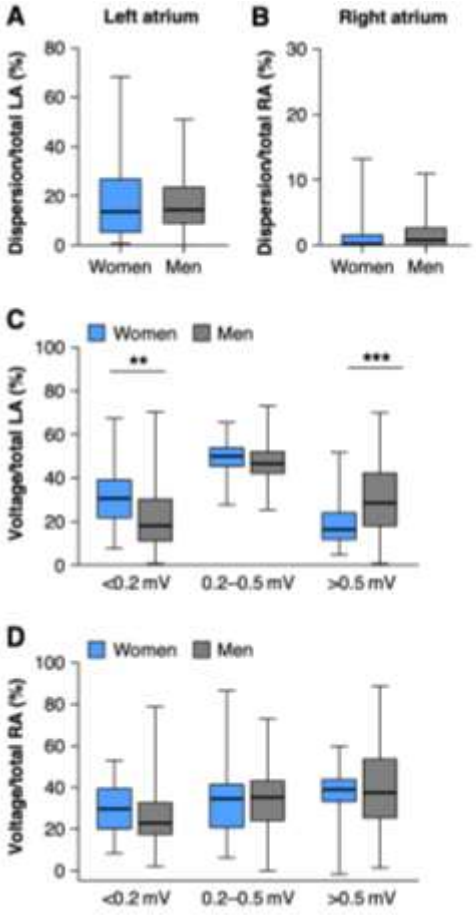
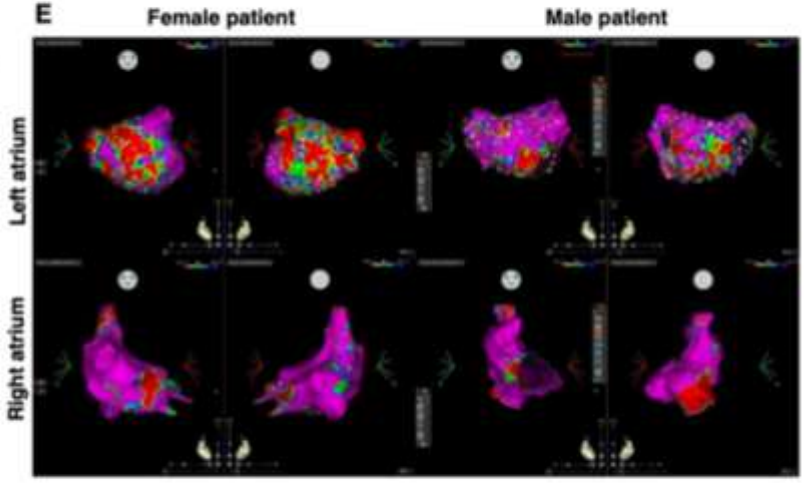
ACM Differences Male and Female – TAILORED AF (persistent AF)

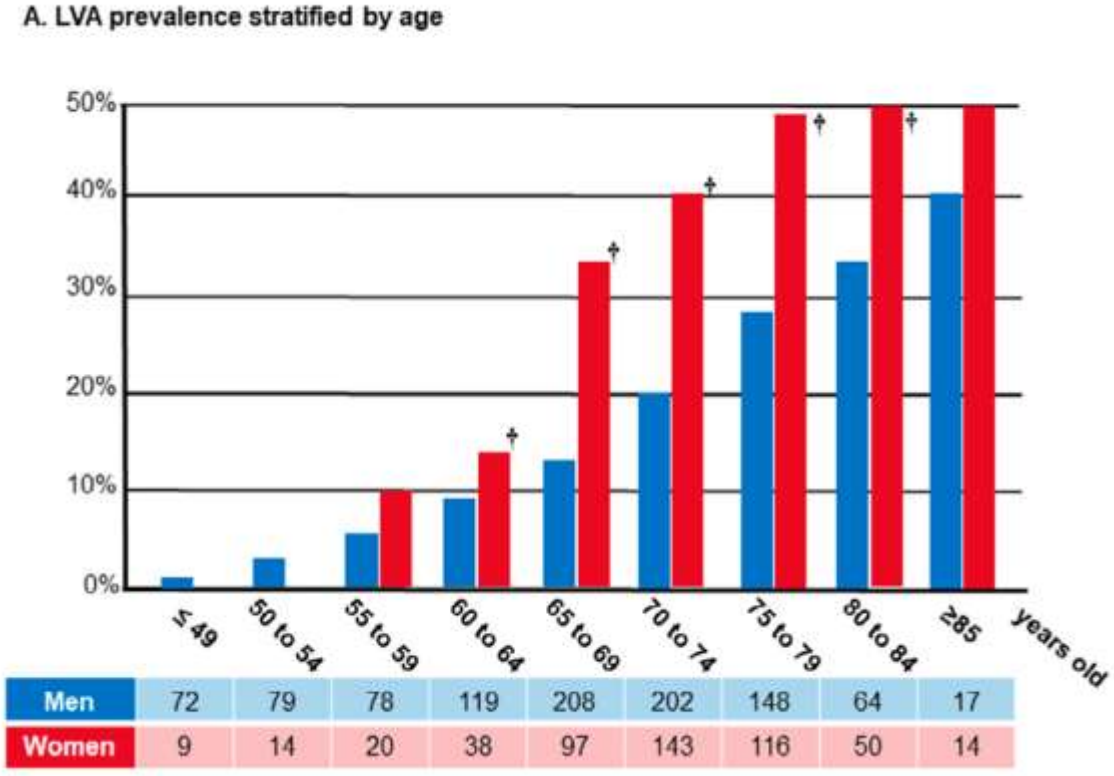






Deisenhofer I, et al. Europace 2025

Gender differences in Atrial fibrosis

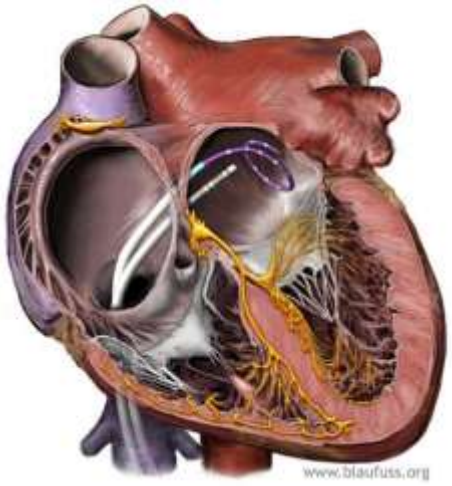


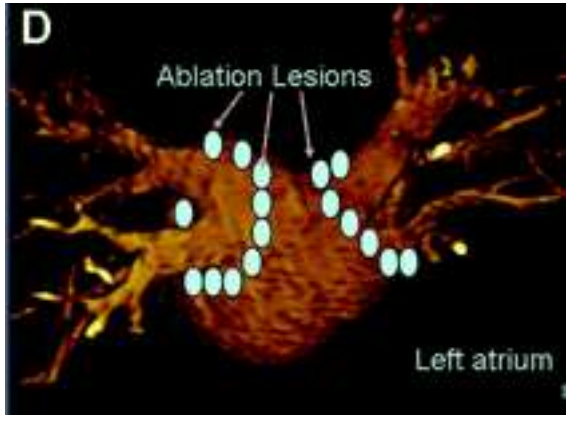
	≤ 49	50 to 54	55 to 59	60 to 64	65 to 69	70 to 74	75 to 79	80 to 84	≥85
Men	72	79	78	119	208	202	148	64	17
Women	9	14	20	38	97	143	116	50	14

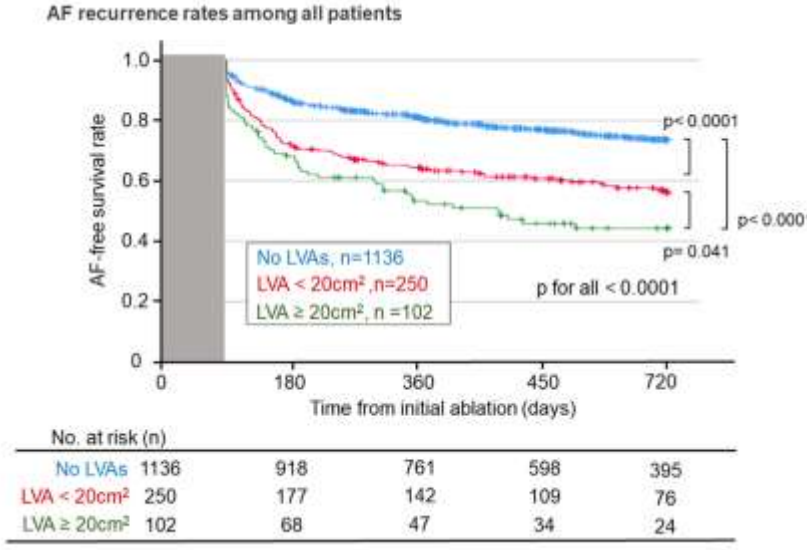
cardiac hypertrophy.[16] In addition, hormonal change during menopause influence myocardial remodeling in women, including a decrease in nitric oxide with menopause,[17] post-menopausal activation of the renin-angiotensin-aldosterone system in response to low estrogen,[17] increased expression of protein kinase A.[18] Our finding that LVAs in women

Masuda M, et al. Am J Cardiol 2023

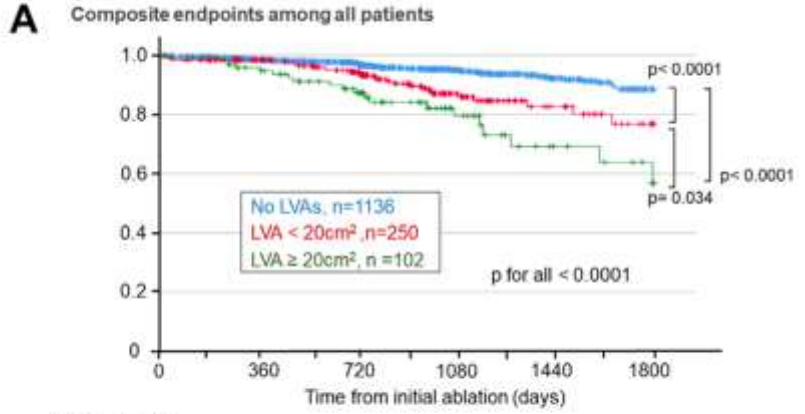
Clinical Impact of Atrial Cardiomyopathy on AF Recurrence after PVI







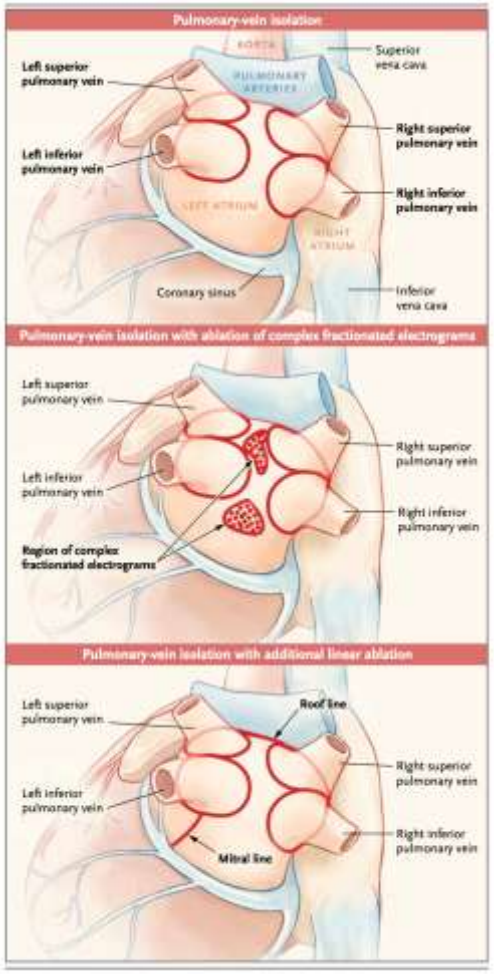
No. at risk (n)					
No LVAs	1136	918	761	598	395
LVA < 20cm²	250	177	142	109	76
LVA ≥ 20cm²	102	68	47	34	24



No. at risk (n)						
No LVAs	1136	955	737	430	231	109
LVA < 20cm²	250	212	167	78	37	14
LVA ≥ 20cm²	102	84	62	31	14	9

Masuda M, et al. Haert Rhythm 2024;21:378-86

Ablation strategy in pers. AF and outcome – STAR AF II Trial / CAPLA Trial / DECAAF Trial / DECAAF II Trial



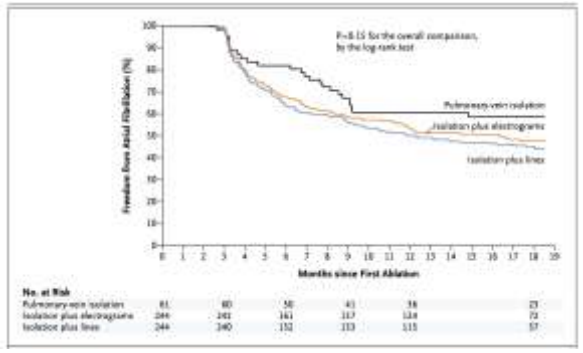
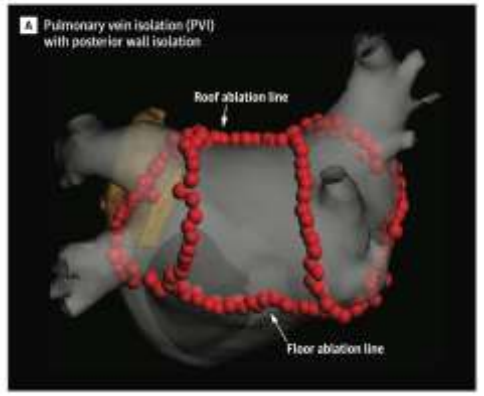


Figure 1. Pulmonary Vein Isolation With and Without Posterior Wall Isolation



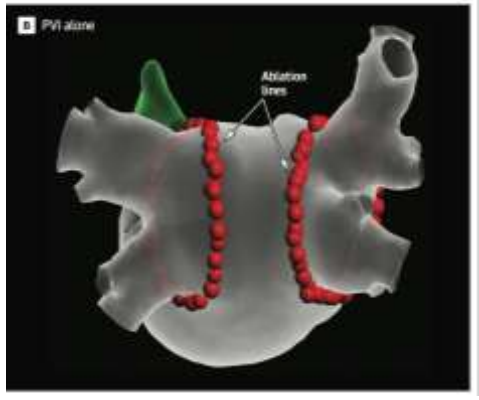


Figure 3. Any Atrial Arrhythmia Recurrence, Without Antiarrhythmic Medication, After a Single Ablation Procedure

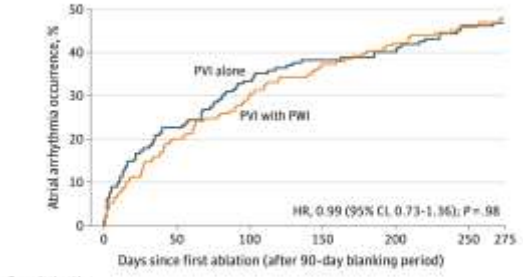
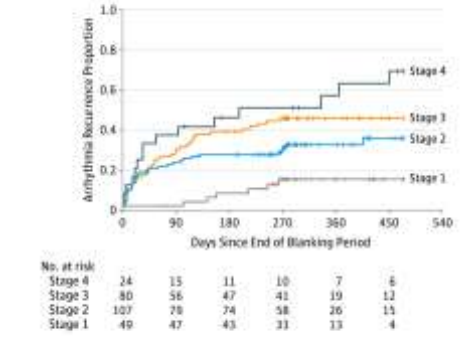
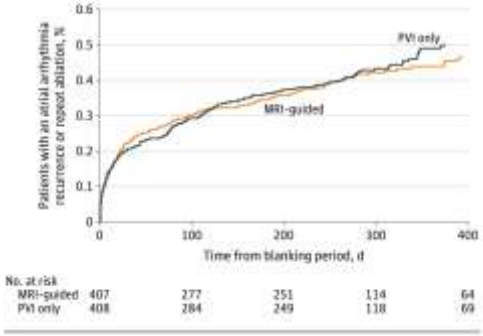


Figure 4. Cumulative Incidence of Arrhythmia Recurrence Without Covariate Adjustment Through Day 475 After the Blanking Period



Small vertical ticks on curves indicate where a patient's follow-up has completed without recurrent atrial fibrillation.

Figure 2. Primary Composite of Atrial Arrhythmia Recurrence or Repeat Ablation



Verma A, et al. N Engl J Med 2015;372:1812-22
Kister PM, et al. JAMA 2023;329:127-35
Marrouche NF, et al. JAMA 2014;311:498-506
Marrouche NF, et al. JAMA 2022;327:2296-305

EAST AFNET 4 Trial

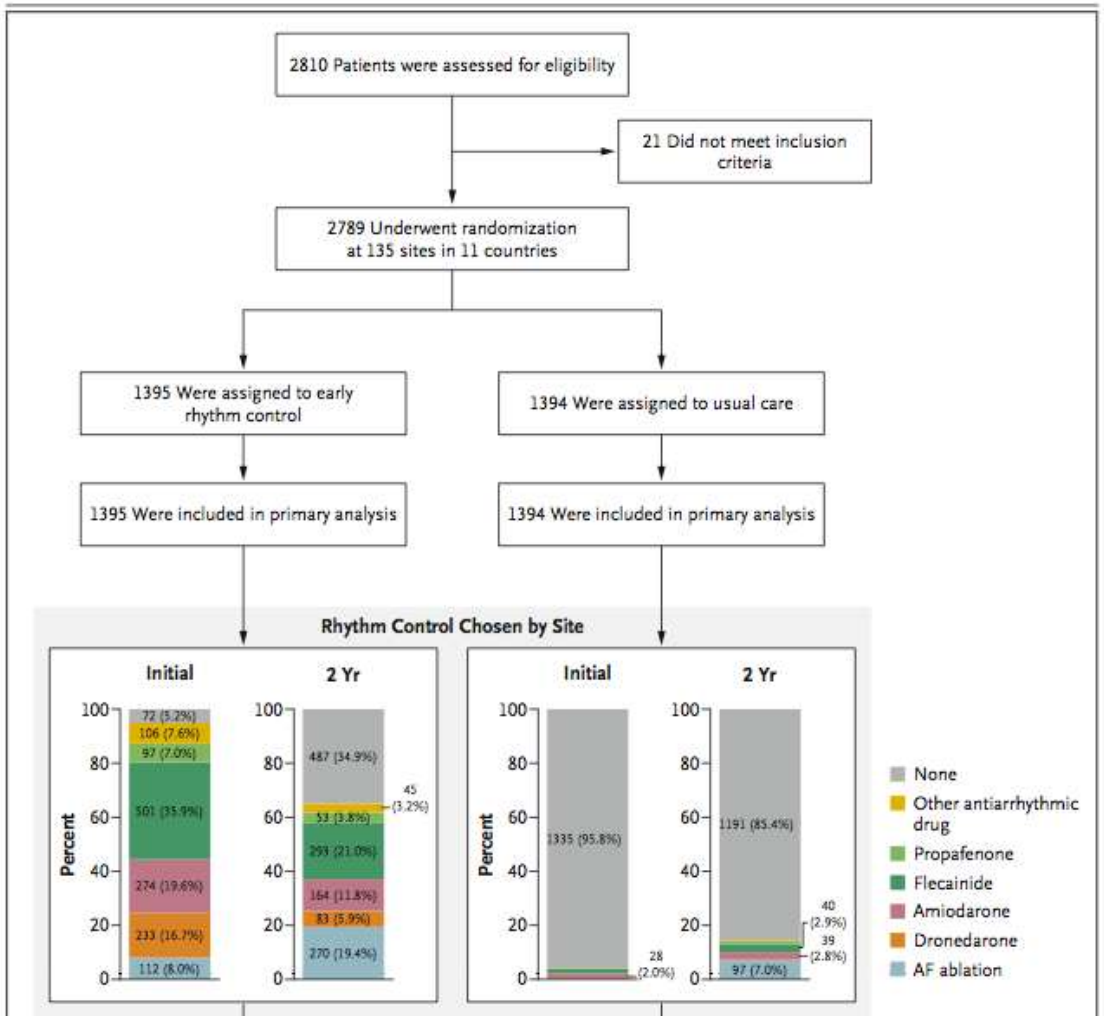
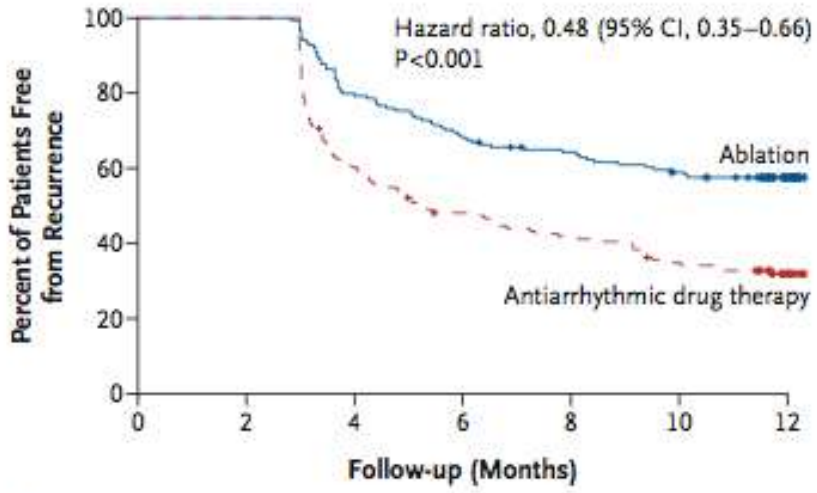


Table 2. Efficacy Outcomes.*

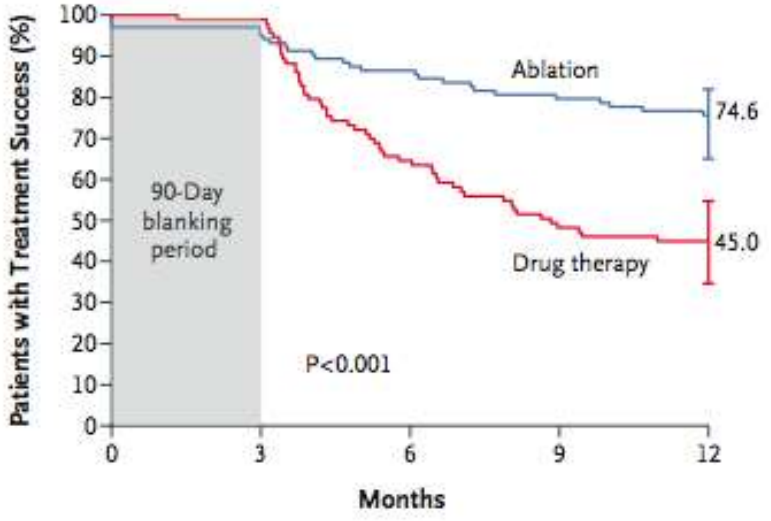
Outcome	Early Rhythm Control	Usual Care	Treatment Effect
First primary outcome — events/person-yr (incidence/100 person-yr)	249/6399 (3.9)	316/6332 (5.0)	0.79 (0.66 to 0.94)†
Components of first primary outcome — events/person-yr (incidence/100 person-yr)			
Death from cardiovascular causes	67/6915 (1.0)	94/6988 (1.3)	0.72 (0.52 to 0.98)‡
Stroke	40/6813 (0.6)	62/6856 (0.9)	0.65 (0.44 to 0.97)‡
Hospitalization with worsening of heart failure	139/6620 (2.1)	169/6558 (2.6)	0.81 (0.65 to 1.02)‡
Hospitalization with acute coronary syndrome	53/6762 (0.8)	65/6816 (1.0)	0.83 (0.58 to 1.19)‡
Second primary outcome — nights spent in hospital/yr	5.8±21.9	5.1±15.5	1.08 (0.92 to 1.28)§
Key secondary outcomes at 2 yr			
Change in left ventricular ejection fraction — %	1.5±9.8	0.8±9.8	0.23 (−0.46 to −0.91)¶
Change in EQ-5D score‖	−1.0±21.4	−2.7±22.3	1.07 (−0.68 to 2.82)¶
Change in SF-12 Mental Score**	0.7±10.6	1.6±10.1	−1.20 (−2.04 to −0.37)¶
Change in SF-12 Physical Score**	0.3±8.5	0.1±8.2	0.33 (−0.39 to 1.06)¶
Change in MoCA score	0.1±3.3	0.1±3.2	−0.14 (−0.39 to 0.12)¶
Sinus rhythm — no. of patients with feature/total no. (%)	921/1122 (82.1)	687/1135 (60.5)	3.13 (2.55 to 3.84)††
Asymptomatic — no. of patients with feature/total no. (%)‡‡	861/1159 (74.3)	850/1171 (72.6)	1.14 (0.93 to 1.40)††

Kirchhof P, et al N Engl J Med 2020

EARLY AF Trial and STOP AF Trial



No. at Risk							
Ablation	154	154	123	105	96	86	55
Antiarrhythmic drug therapy	149	149	89	69	60	49	27



No. at Risk					
Ablation	104	99	88	81	70
Drug therapy	99	93	60	44	39

Andrade JG, et al N Engl J Med 2021
Wazni OM, et al N Engl J Med 2021

Early PVI and Impact on AF Progression (EARLY-AF)

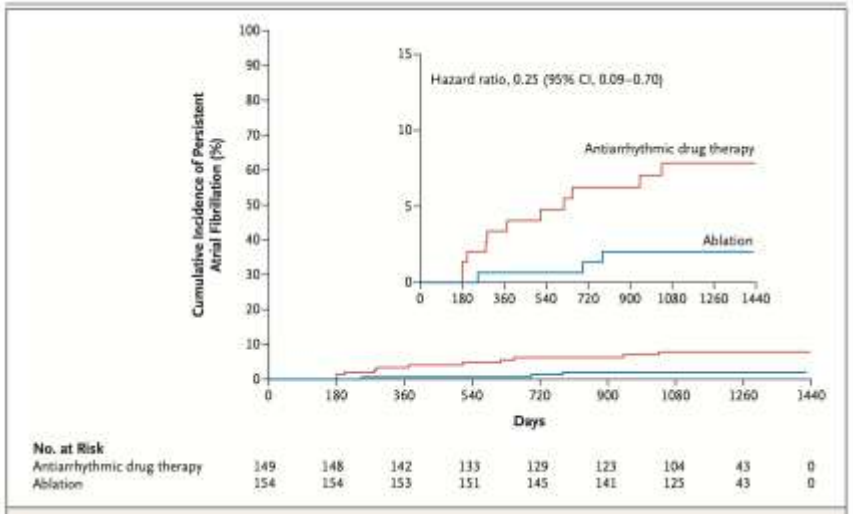


Table 1. Main End Points of Interest.*

End Point	Ablation Group (N=154)	Antiarrhythmic Drug Group (N=149)	Hazard Ratio (95% CI)
	number (percent)		
Progression to persistent atrial fibrillation from 91 days after treatment initiation to final follow-up	3 (1.9)	11 (7.4)	0.25 (0.09–0.70)
Recurrence of any atrial tachyarrhythmia			
From 91 days to 12 mo after treatment initiation†	66 (42.9)	101 (67.8)	0.48 (0.35–0.66)
From 91 days to 36 mo after treatment initiation	87 (56.5)	115 (77.2)	0.51 (0.38–0.67)
Hospitalization			
No. of patients with event (%)	8 (5.2)	25 (16.8)	0.31 (0.14–0.66)
No. of events	9	29	
Median no. of events per patient among those with an event (IQR)	1 (1–1)	1 (1–1)	

Patient with paroxysmal AF

Catheter ablation[a] (Class I)

Patient with persistent AF

Catheter ablation[a] (Class IIb)

Patient with permanent AF

Rate control target = resting heart rate <110 b.p.m. (lenient control), with stricter control with continuing symptoms (Class IIa)

Atrioventricular node ablation and CRT (Class IIa)

Andrade LG, et al. N Engl J Med 2023
Van Gelder, et al. Eur Heart J 2024

PVI in Idiopathic HF – CAMERA MRI Trial

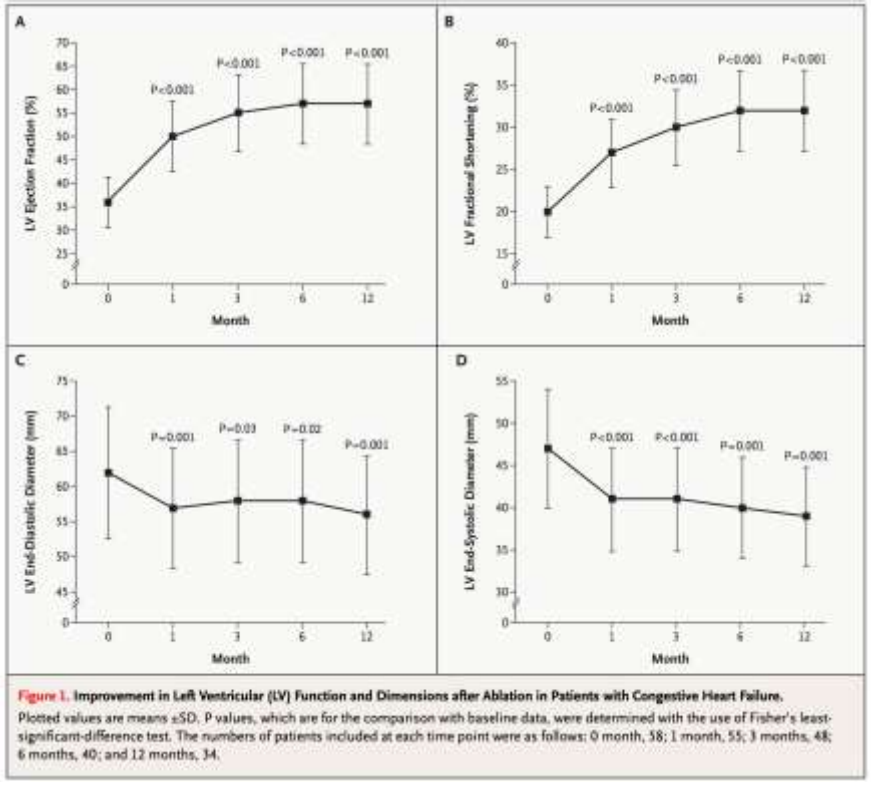
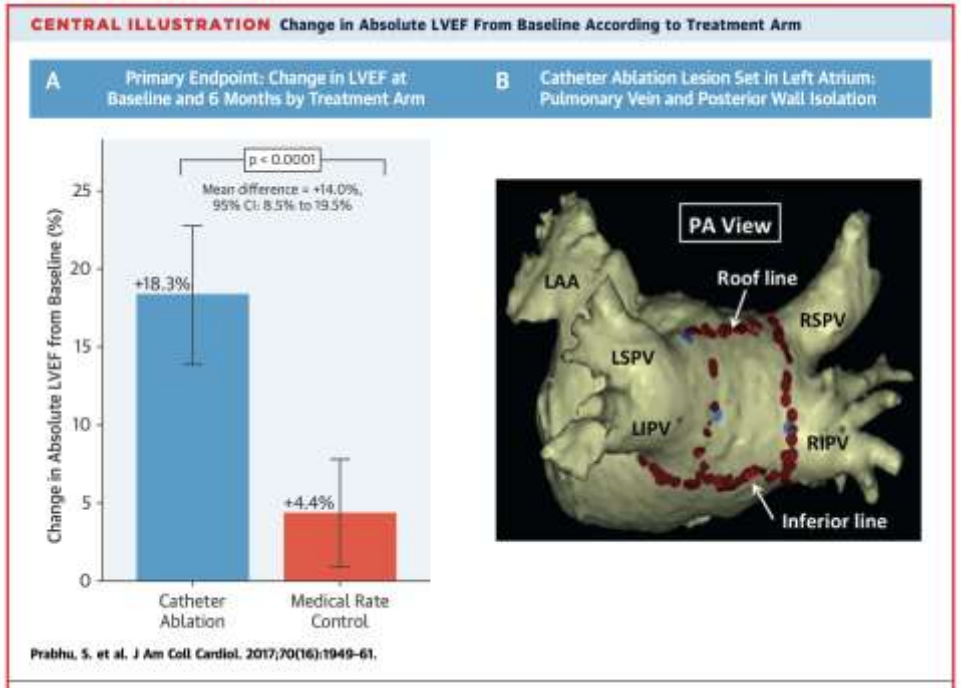
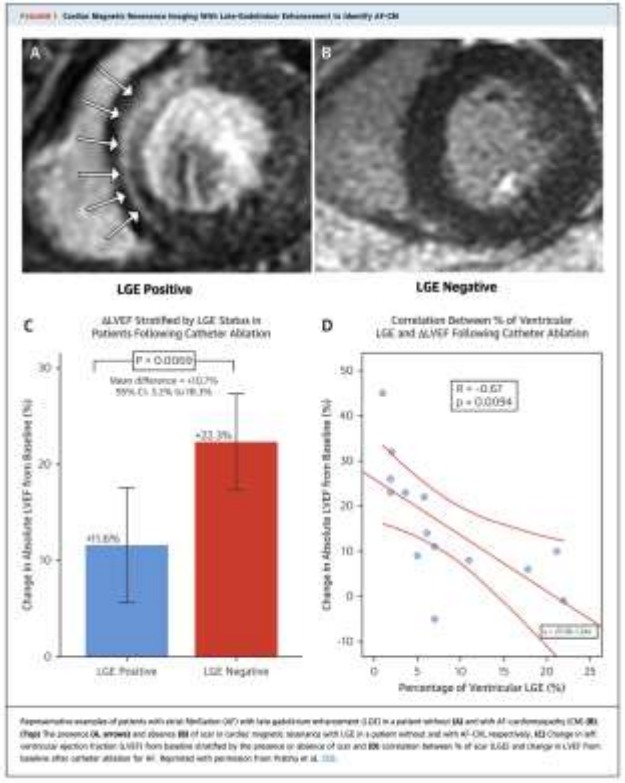


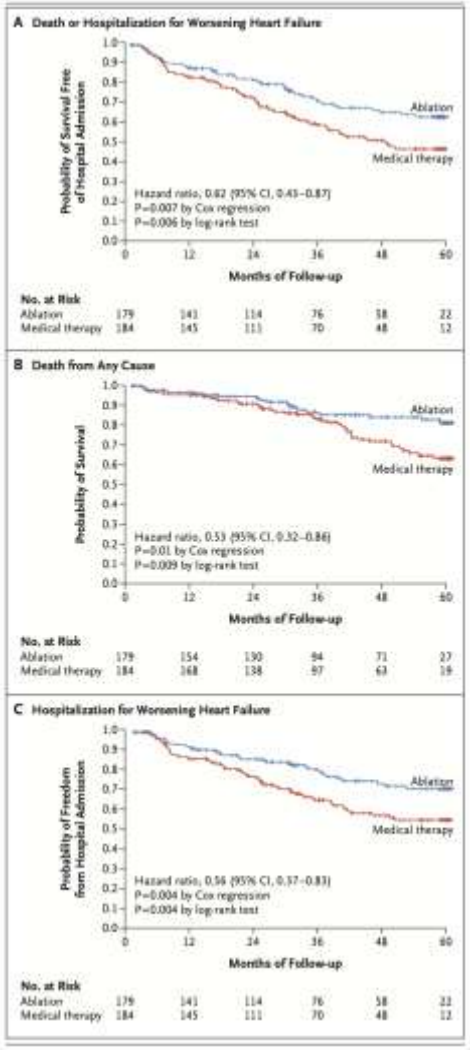
Figure 1. Improvement in Left Ventricular (LV) Function and Dimensions after Ablation in Patients with Congestive Heart Failure.
Plotted values are means ±SD. P values, which are for the comparison with baseline data, were determined with the use of Fisher's least-significant-difference test. The numbers of patients included at each time point were as follows: 0 month, 58; 1 month, 55; 3 months, 48; 6 months, 40; and 12 months, 34.

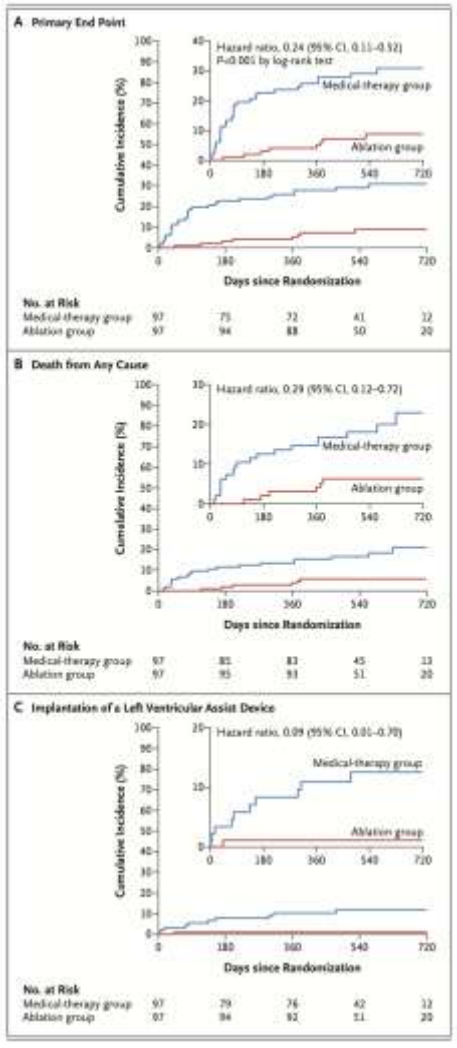


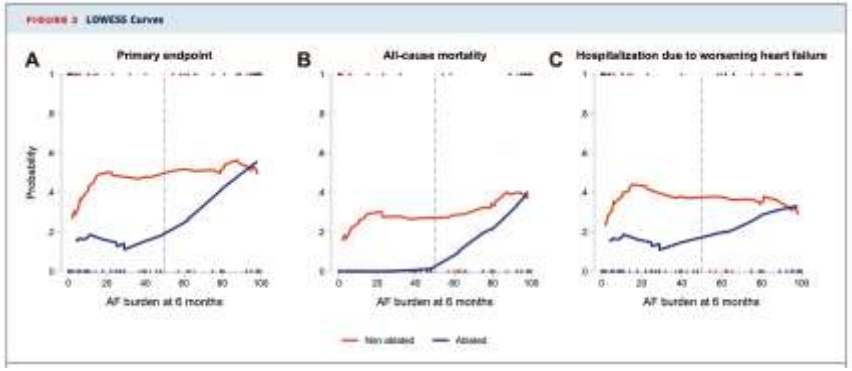


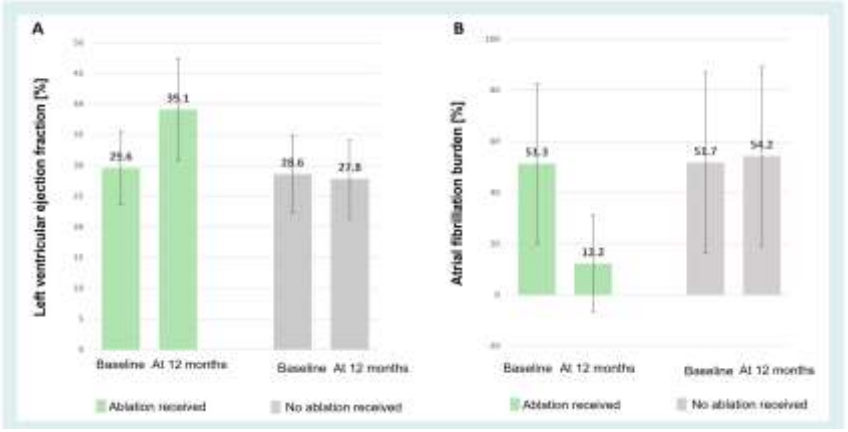
Hsu LF, et al. N Engl J Med 2004;351:2373-83
Huizar JF, et al. J Am Coll Cardiol 2019;73:2328-44

Ablation in HFrEF - CASTLE AF / CASTLE HTX



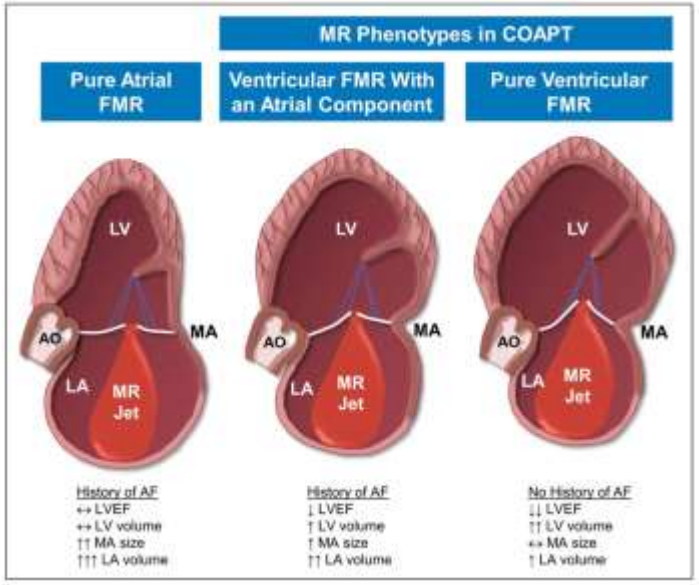






Marrouche NF, et al. N Engl J Med 2018;378:417-27
Brachmann J, et al. JACC EP 2021;7:594-603
Sohns C, et al. Circ EP 2023;389:1380-9
Sohns C, et al. N Engl J Med 2023;389:1380-9

Arrhythmia-induced Cardiomyopathy – Atrial and Ventricular FMR and Association with Atrial Fibrillation



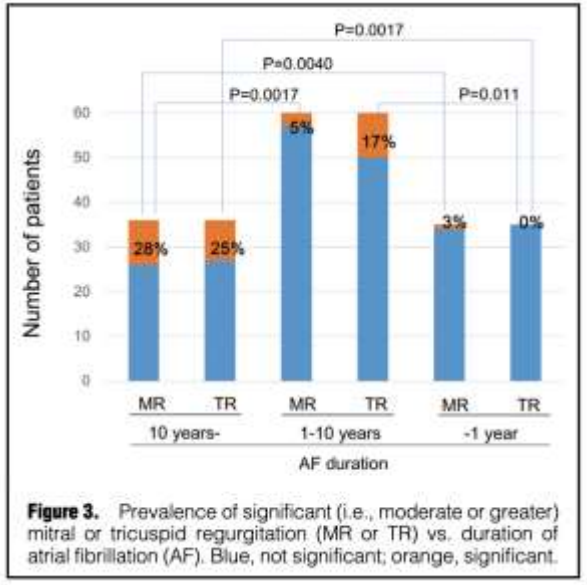


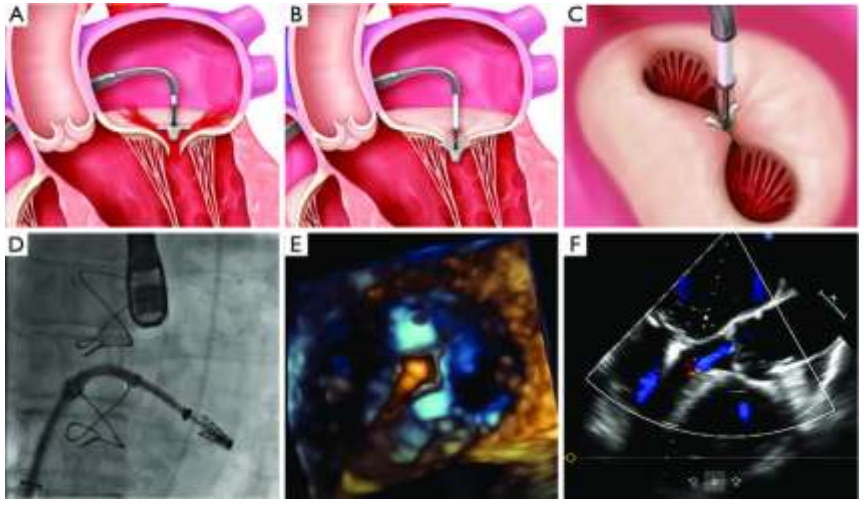
Figure 3. Prevalence of significant (i.e., moderate or greater) mitral or tricuspid regurgitation (MR or TR) vs. duration of atrial fibrillation (AF). Blue, not significant; orange, significant.

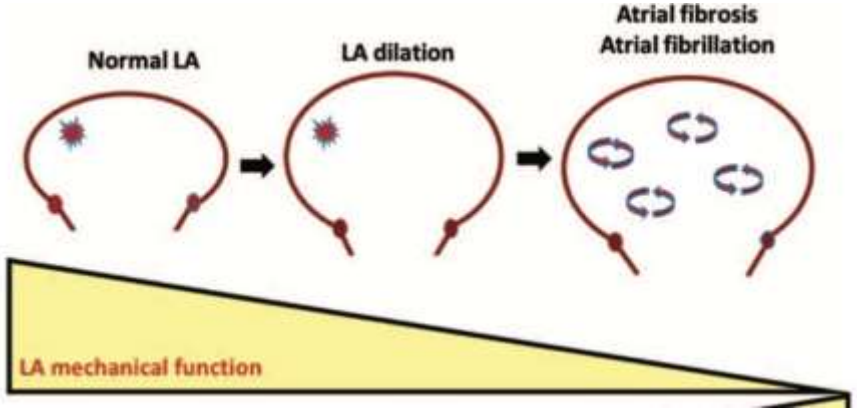
Table 1. Baseline Demographic and Clinical Characteristics of the Patients with Heart Failure and Moderate-to-Severe Functional Mitral Regurgitation.*

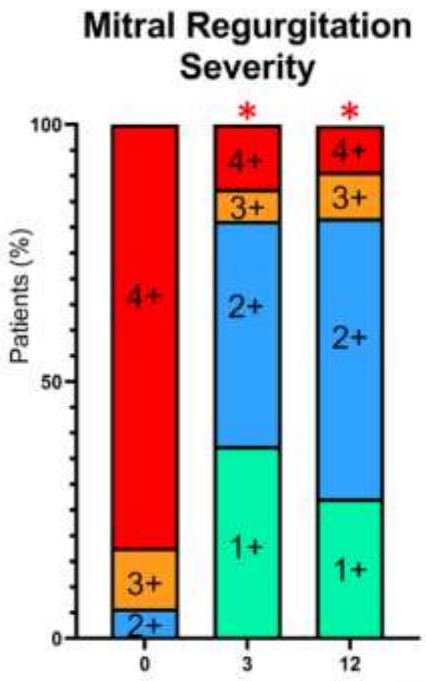
Characteristic	Device Group (N=250)	Control Group (N=255)
Age — yr	70.0±10.4	69.4±10.7
Male sex — no. (%)	195 (78.0)	211 (82.8)
Diabetes — no. (%)	91 (36.4)	85 (33.3)
Hypertension — no. (%)	141 (56.4)	127 (49.8)
Previous myocardial infarction — no. (%)	144 (57.6)	135 (52.9)
Previous PCI — no. (%)	118 (47.2)	125 (49.0)
Previous CABG — no. (%)	69 (27.6)	64 (25.1)
Previous stroke or TIA — no. (%)	29 (11.6)	30 (11.8)
Peripheral vascular disease — no. (%)	38 (15.2)	27 (10.6)
History of atrial fibrillation or flutter — no. (%)	118 (47.2)	125 (49.0)

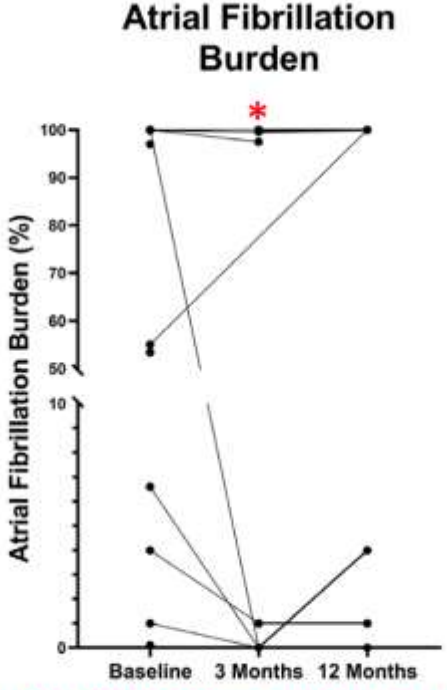
Gertz ZM, et al. Circ Cardiovasc Interv 2021
Shoureshi P, et al. J Am Coll Cardiol 2024;83:2214-32
Abe Y, et al. Circ J 2018;82:1451-58
Anker S, et al N Engl J Med 2024

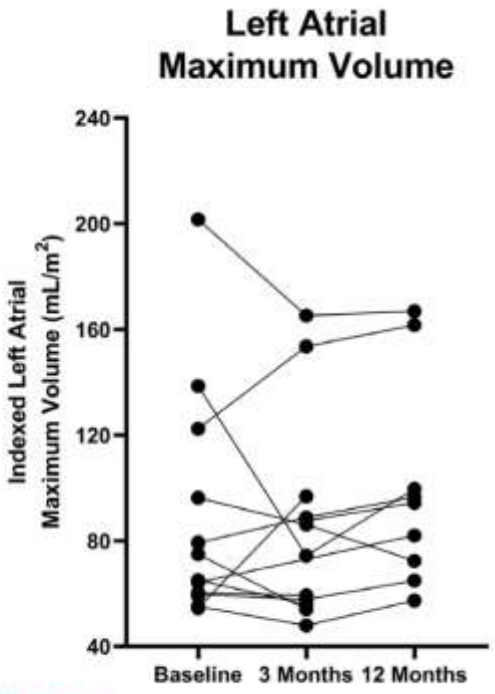
M-TEER and Impact on AF







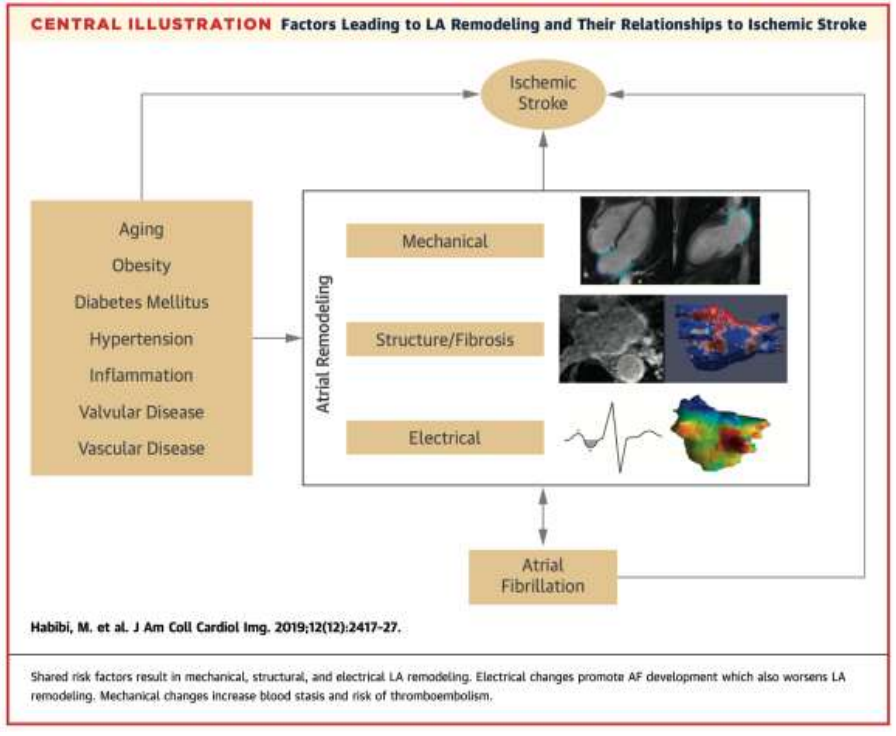


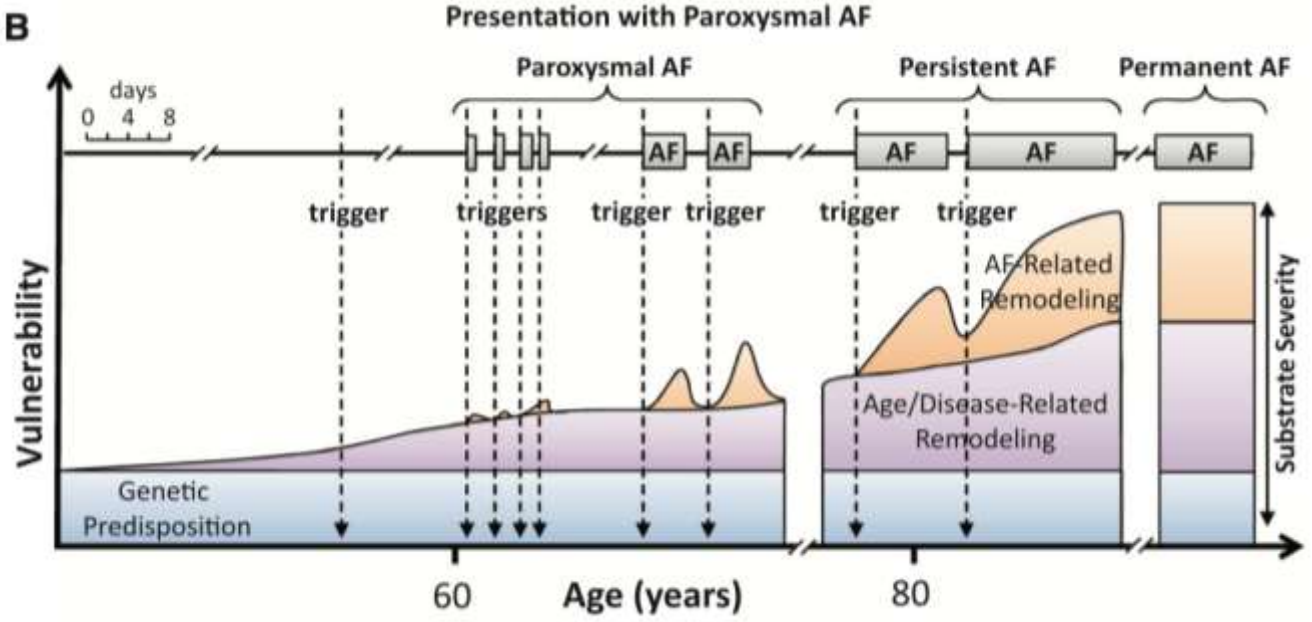


*p<0.05 for comparison to baseline

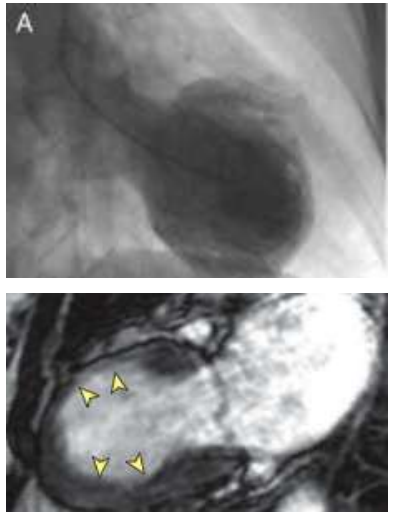
Trankle CR, et al. PACE 2022
Triposkiadis F, et al. Eur J Heart Fail 2016

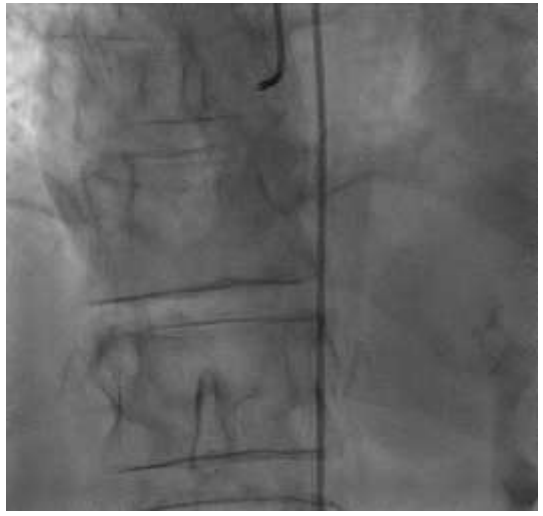
Conclusion – Atrial Cardiomyopathy

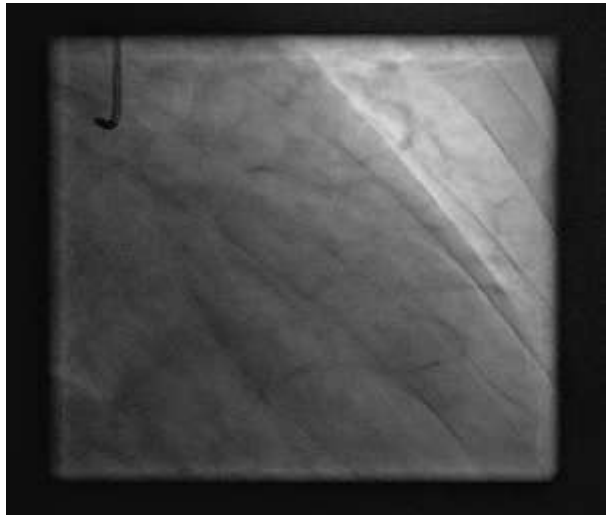


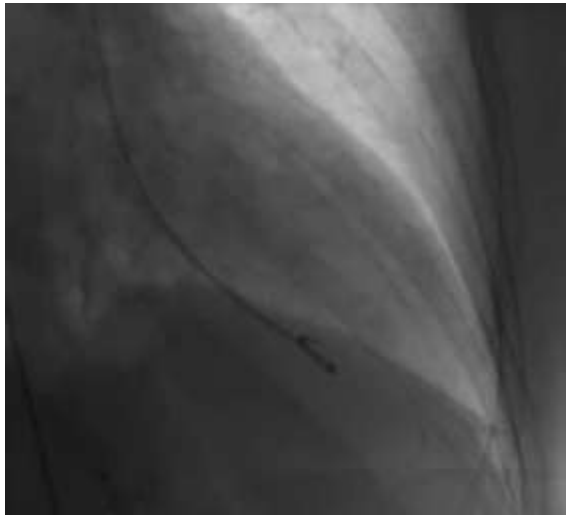


Stress-Cardiomopathy









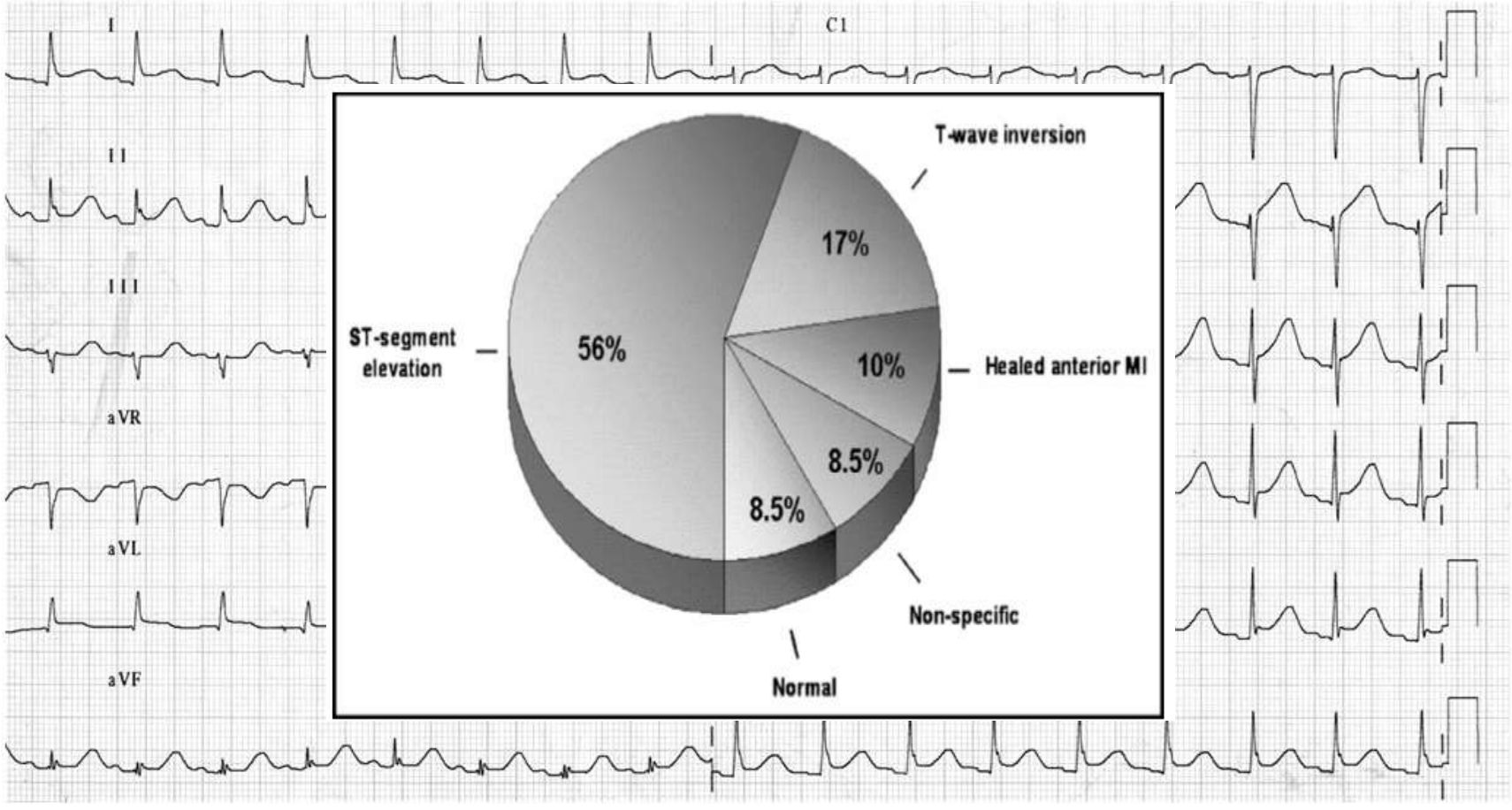
El-Battrawy I,... Akin I. JAHA 2019
El-Battrawy I,...Akin I. Heart Fail Rev 2020
Sattler K, ... Akin I. Int J Cardiol 2017
Sattler K, ... Akin I. Can J Cardiol 2020
Aweimer A,..Akin I et al. J Intern Med 2021
Hohneck A,... Akin I. Intern Med J 2019
Bill V,... Akin I. Angiology 2018

Stress-Cardiomopathy

Characteristic	Takotsubo Cardiomyopathy		Acute Coronary Syndrome	P Value†
	Total Cohort (N=1750)	Matched Cohort (N=455)	Matched Cohort (N=455)	
Female sex — no. (%)	1571 (89.8)	411 (90.3)	411 (90.3)	1.00
Age — yr	66.4±13.1	67.7±12.5	68.7±12.3	0.19
Chest pain — no./total no. (%)	1229/1619 (75.9)	322/438 (73.5)	361/403 (89.6)	<0.001
Dyspnea — no./total no. (%)	760/1620 (46.9)	208/439 (47.4)	128/363 (35.3)	0.001
Median troponin (IQR) — factor × ULN‡	7.70 (2.22–24.00)	7.68 (2.38–24.21)	8.30 (1.80–36.40)	0.62
Median creatine kinase (IQR) — factor × ULN	0.85 (0.52–1.48)	0.87 (0.55–1.42)	1.12 (0.60–2.97)	<0.001
Median brain natriuretic peptide (IQR) — factor × ULN§	6.12 (2.12–15.70)	5.89 (1.68–13.92)	2.91 (0.88–8.26)	<0.001
ST-segment change — no./total no. (%)				
Elevation	690/1578 (43.7)	185/420 (44.0)	233/455 (51.2)	0.03
Depression	121/1578 (7.7)	35/420 (8.3)	122/392 (31.1)	<0.001
Heart rate — beats/min	87.5±21.8	87.3±21.8	76.1±18.3	<0.001
Systolic blood pressure — mm Hg	130.6±28.8	131.6±31.4	131.5±28.2	0.96
Left ventricular ejection fraction — %¶	41.1±11.8	40.7±11.2	51.5±12.3	<0.001
Left ventricular end diastolic pressure — mm Hg	21.3±8.0	22.1±7.7	20.1±7.8	0.001
Coexisting medical condition — no./total no. (%)				
Coronary artery disease	245/1597 (15.3)	96/455 (21.1)	455/455 (100.0)	<0.001
Neurologic or psychiatric disorder‖	714/1525 (46.8)	252/452 (55.8)	115/448 (25.7)	<0.001
Acute neurologic disorder	143/1528 (9.4)	41/452 (9.1)	4/448 (0.9)	<0.001
Past or chronic neurologic disorder	293/1512 (19.4)	98/452 (21.7)	62/448 (13.8)	0.002
Acute psychiatric disorder	149/1525 (9.8)	57/452 (1.3)	6/448 (1.3)	<0.001
Past or chronic psychiatric disorder	444/1512 (29.4)	165/451 (36.6)	61/448 (13.6)	<0.001

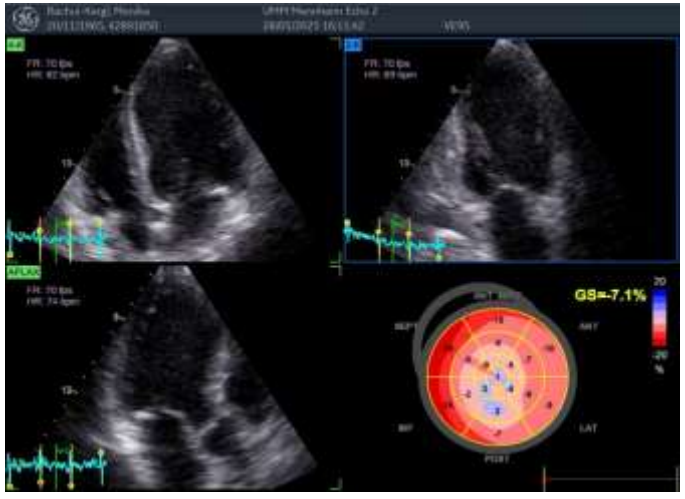
Templin C, et al. NEJM 2015

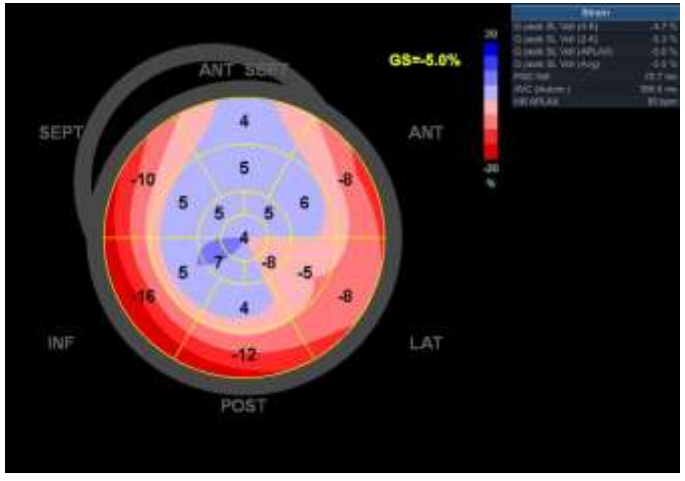
Stress-Cardiomopathy



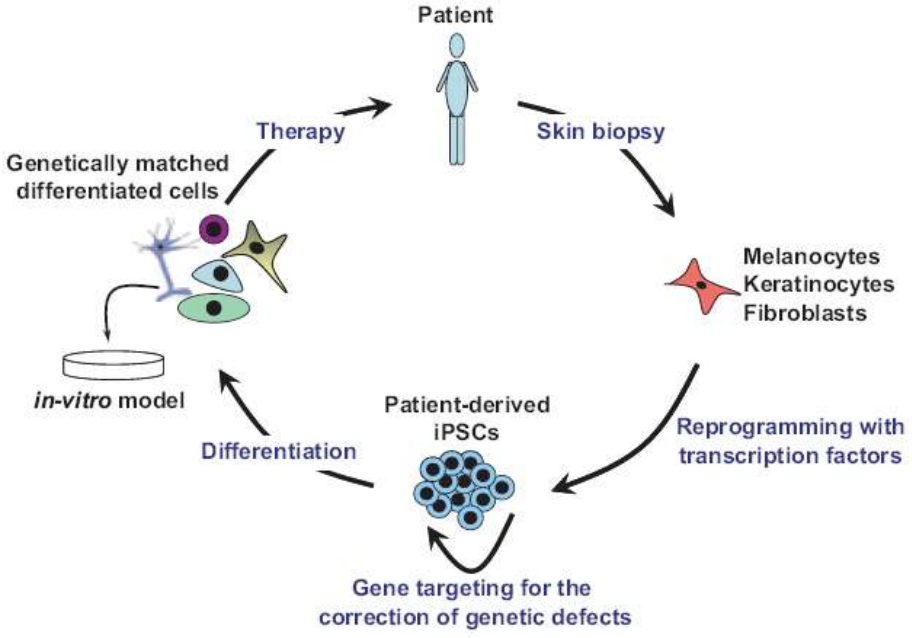
Sharkey SW, et al. Am J Cardiol 2008;101:1723-28

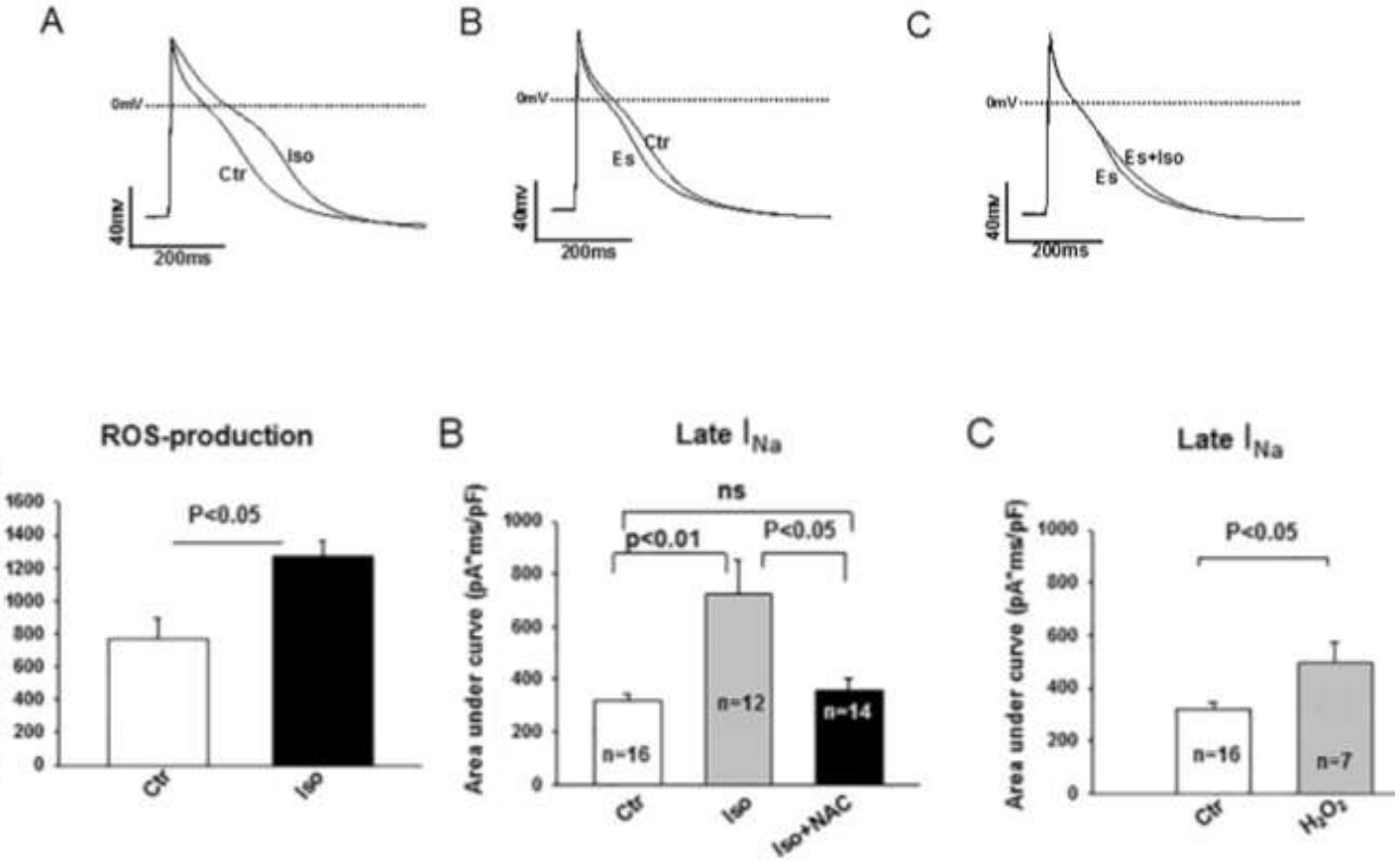
Stress-Cardiomopathy





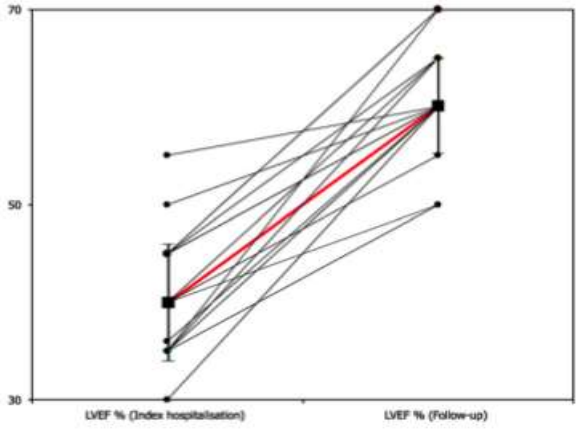
Stress-Cardiomopathy - hiPSC





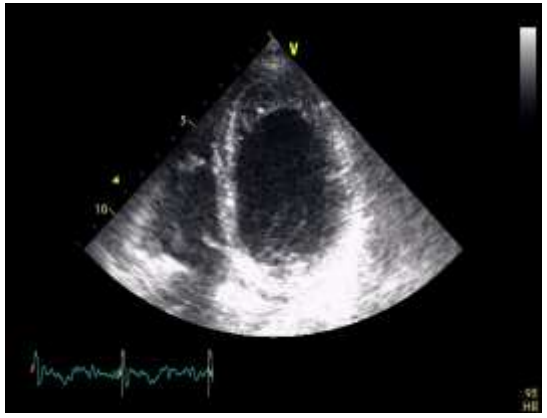
El-Battrawy I,... Akin I. Int J Cardiol 2018

Stress-Cardiomopathy



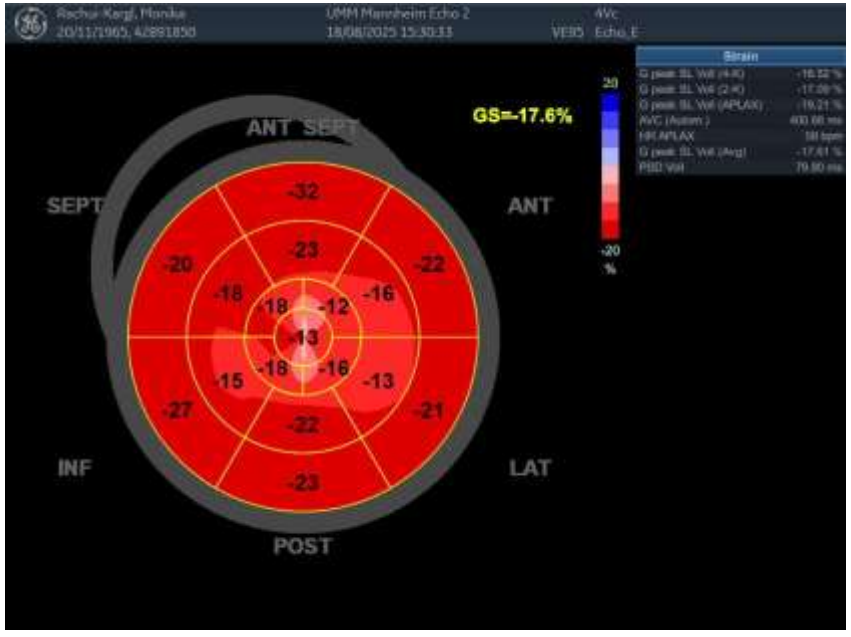
- Heart Failure (37%)
- LV-EF 29-37%
- Arrythmias (1.5-15%)
- LV-Rupture (<0.5%)

Sudden cardiac death due to ventricular fibrillation	1 (4%)
Atrial fibrillation	4 (15%)
Cardiogenic shock	2 (7%)
Pulmonary oedema	4 (15%)
Left ventricular outflow tract obstruction	3 (11%)
Apical thrombus	2 (7%)
Recurrence of TC	2 (7%)
Rehospitalisation for cardiac complaints	8 (30%)
Cumulative deaths	4 (15%)
Cardiac deaths	2 (7%)
Cumulative morbidity and mortality	14 (52%)

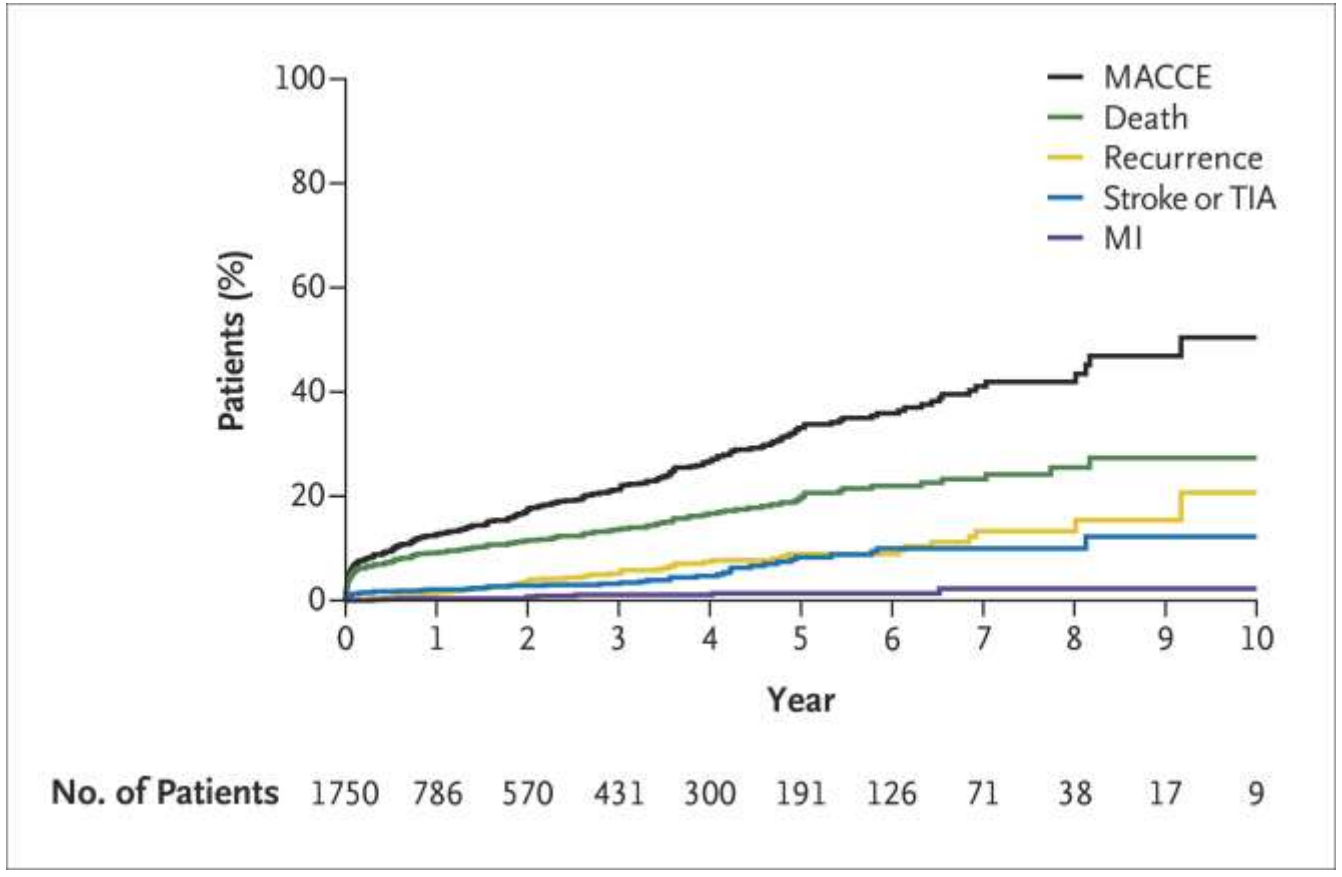


Ionescu CN, et al. Heart, Lung and Circulation 2010;19:601-5

Stress-Cardiomopathy



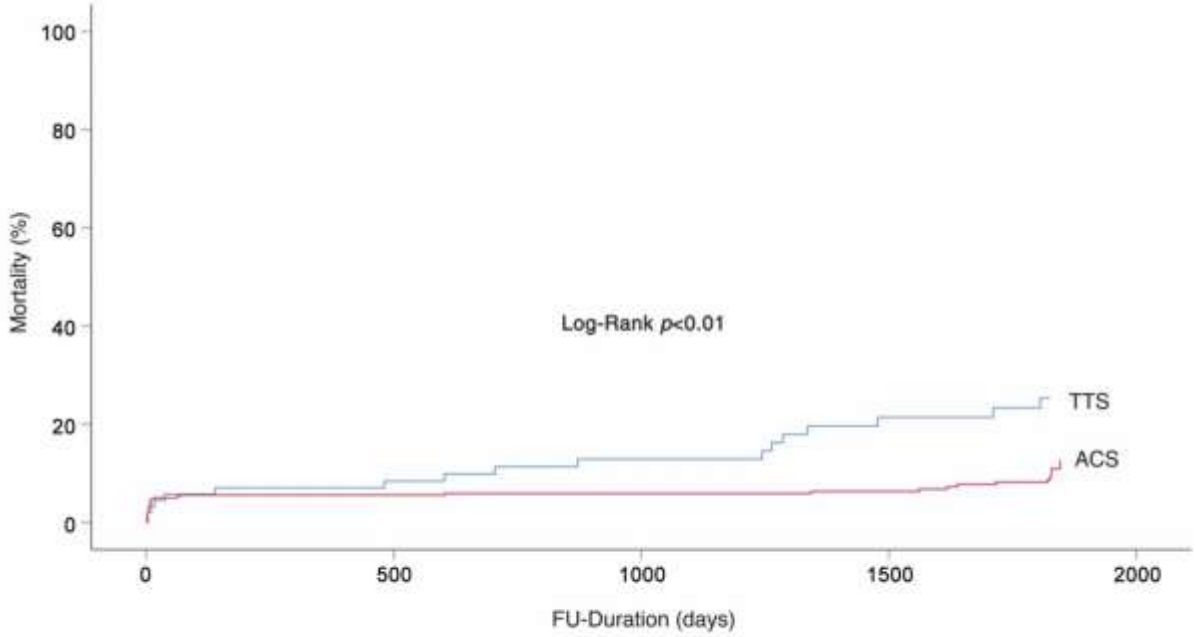
Stress-Cardiomopathy



Templin C, et al. NEJM 2015

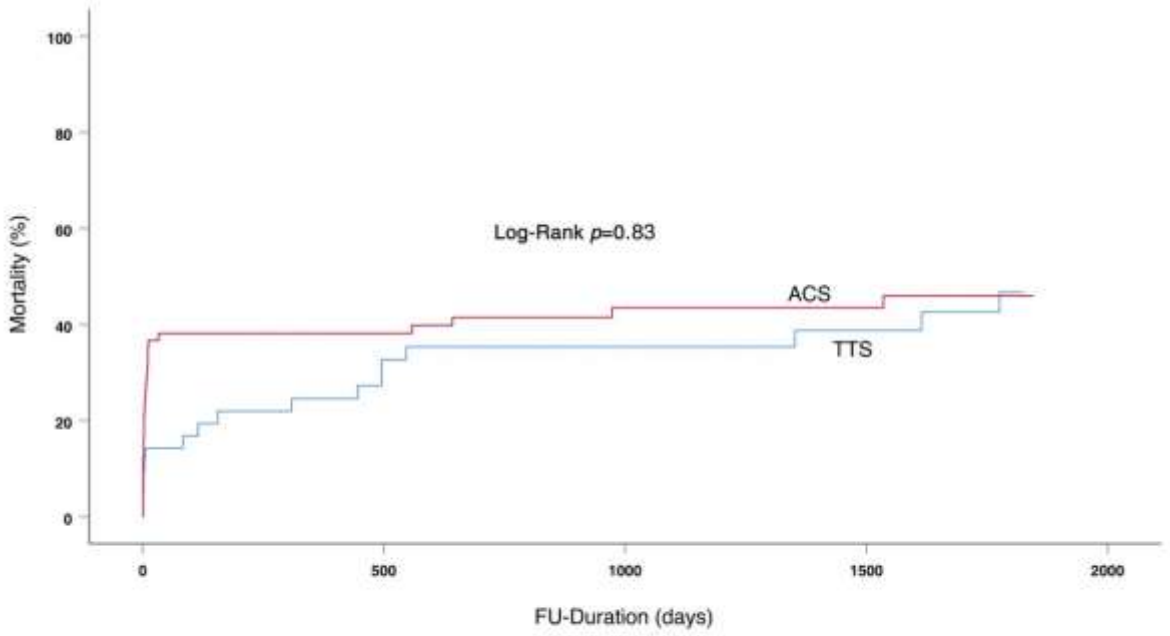
Stress-Cardiomopathy

Index LV-EF > 35%



Patients at risk						
TTS	94	88	85	84	79	77
ACS	432	409	408	408	407	399

Index LV-EF < 35%

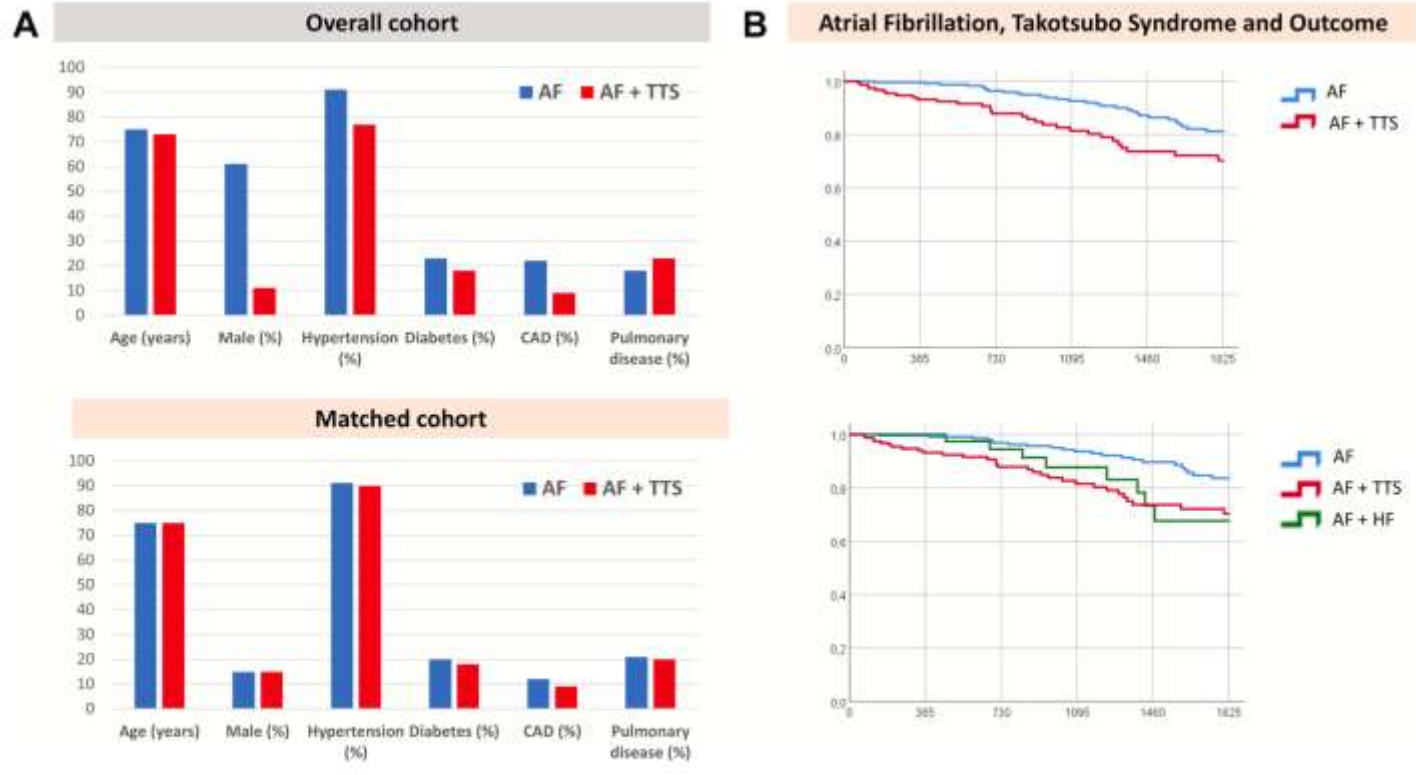


Patients at risk						
TTS	42	34	30	33	29	25
ACS	80	50	48	47	47	46

Abumayyaleh M, … Akin I. In Vivo 2020

AF and TTS

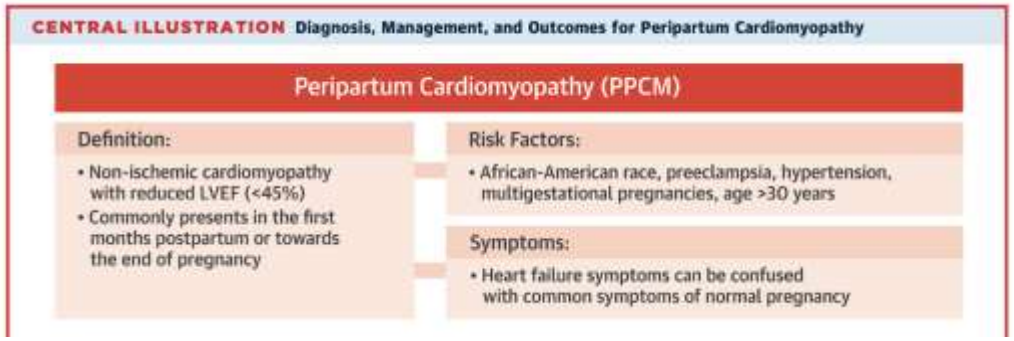
FIGURE 1 Histograms Reporting Main Differences Between Groups



(A) Overall cohort (above). (B) Matched cohort (below). Kaplan-Meier curves for long-term mortality in the matched group for AF-TTS patients and AF (B, above) and then splitting the latter for the presence/absence of heart failure (B, below). AF = atrial fibrillation; HF = heart failure; TTS = Takotsubo syndrome.

Arcari L, … Akin I…et al. JACC EP 2025

Peripartum Cardiomyopathy - PPCM



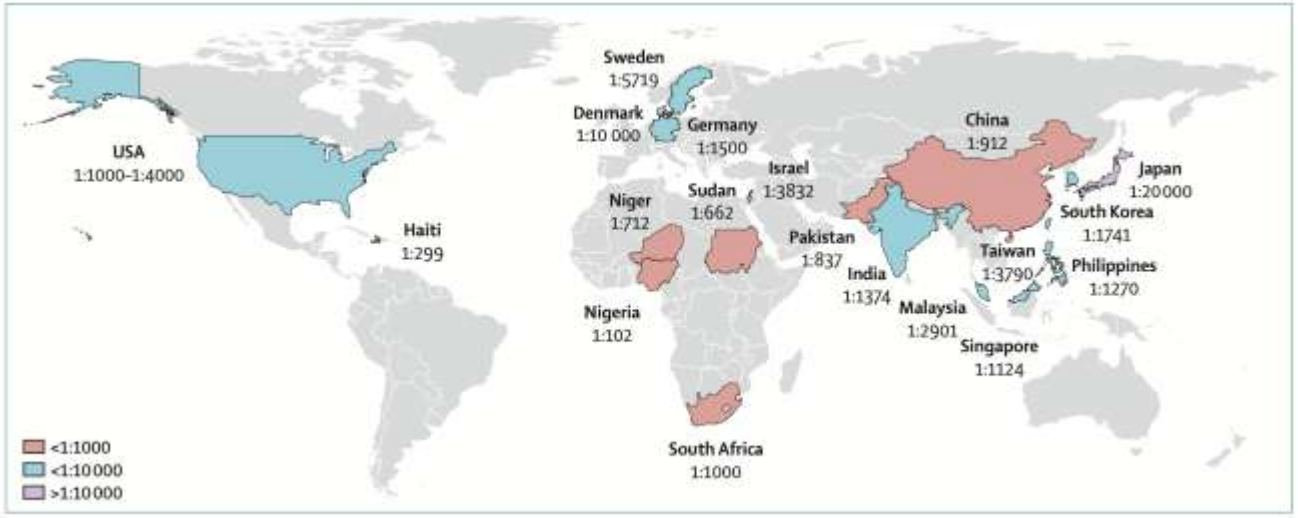


Figure 1: Global incidence

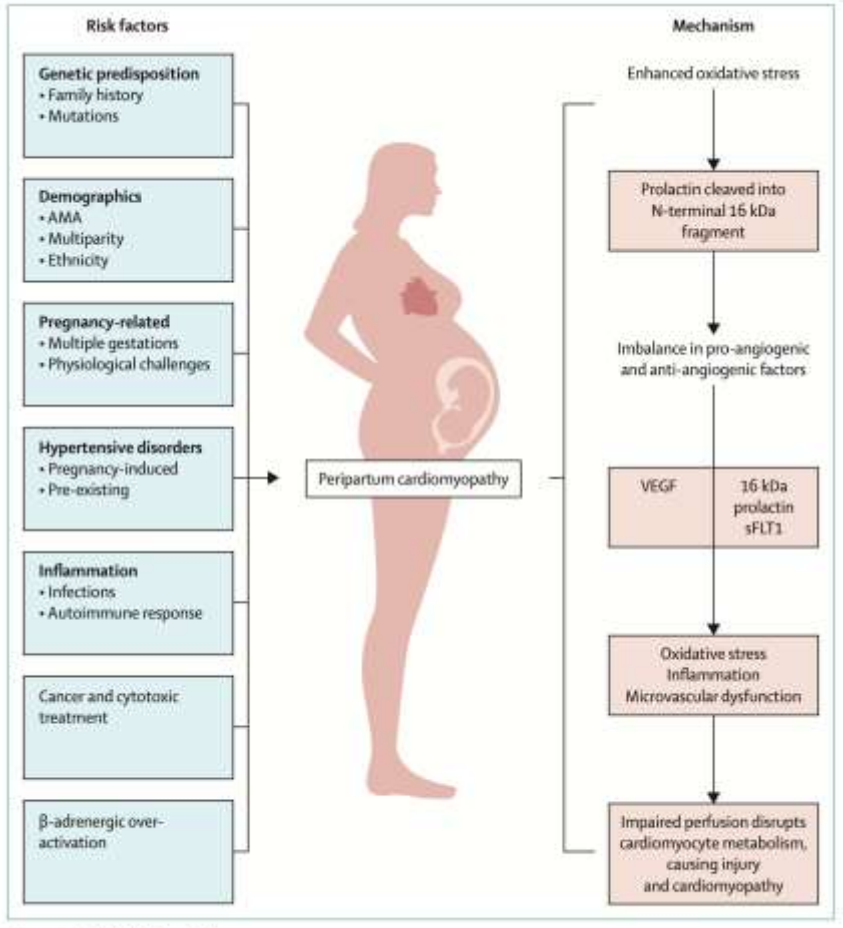
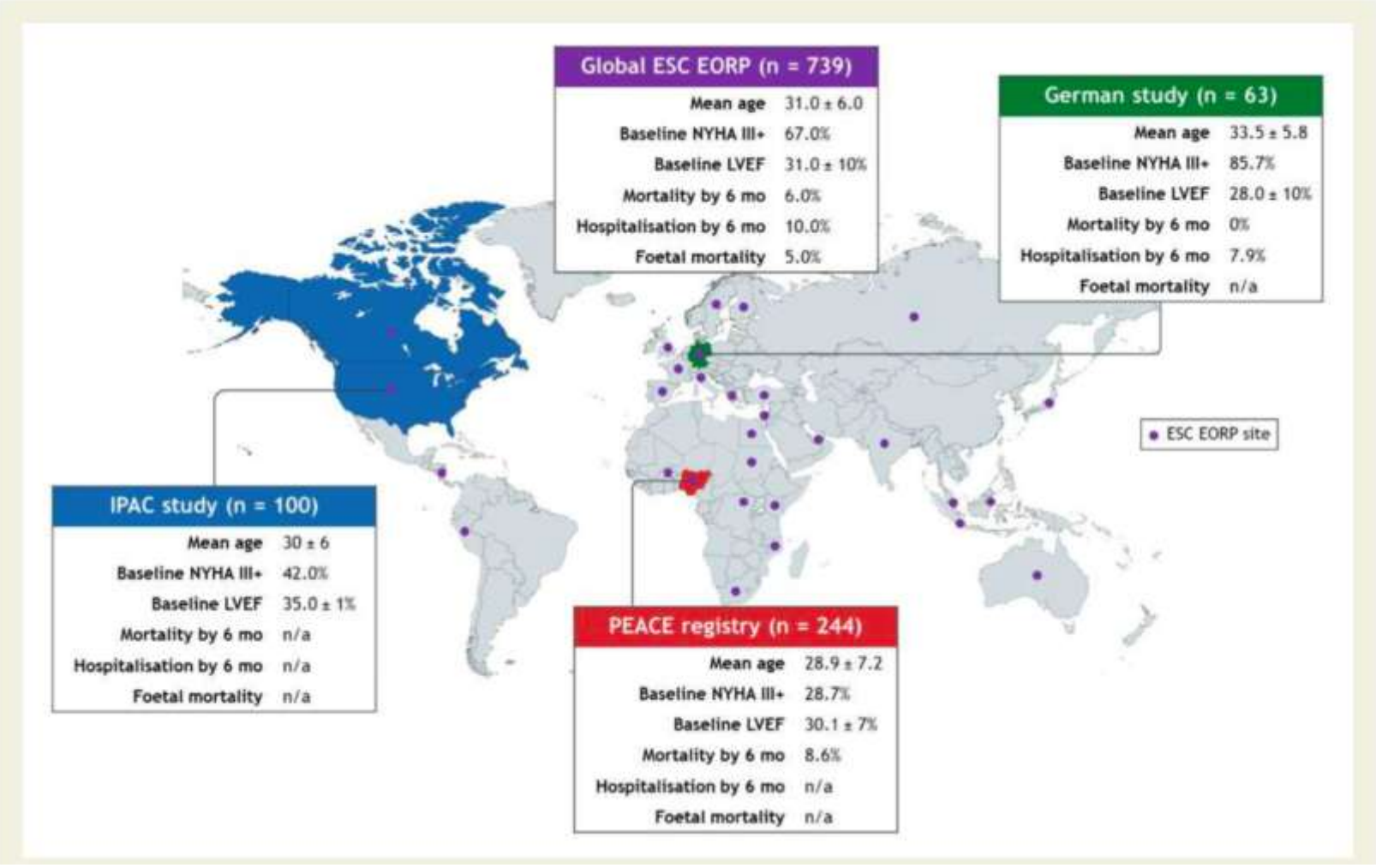


***Figure 2:* Multiple hit model**
AMA=advanced maternal age. sFLT1=soluble fms-like tyrosine kinase-1. VEGF=vascular endothelial growth factor.

Sliwa K, et al. Lancet 2025

Peripartum Cardiomyopathy - PPCM



Sliwa K, et al. EHJ 2021

Peripartum Cardiomyopathy - PPCM

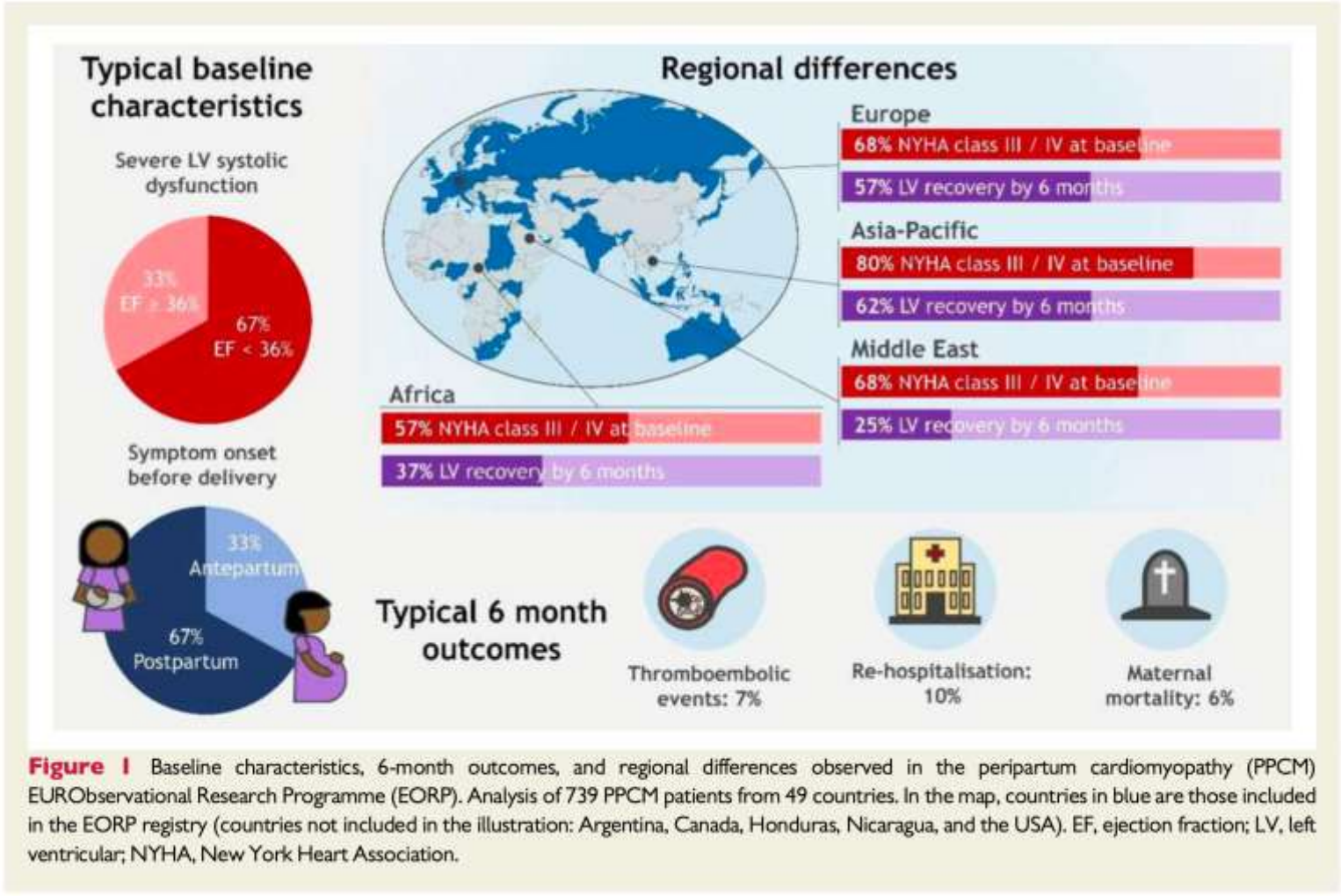


Figure 1 Baseline characteristics, 6-month outcomes, and regional differences observed in the peripartum cardiomyopathy (PPCM) EURObservational Research Programme (EORP). Analysis of 739 PPCM patients from 49 countries. In the map, countries in blue are those included in the EORP registry (countries not included in the illustration: Argentina, Canada, Honduras, Nicaragua, and the USA). EF, ejection fraction; LV, left ventricular; NYHA, New York Heart Association.

Sliwa K, et al. EHJ 2021

Peripartum Cardiomyopathy - PPCM

	History	Onset	Biomarkers	Echocardiography or CMRI	Differentiation from peripartum cardiomyopathy
Peripartum cardiomyopathy	No known cardiac disease; no heart failure signs or symptoms before pregnancy	Towards the end of pregnancy and in the months following delivery	Elevated natriuretic peptides	Reduced systolic left ventricular function; LVEF <45%	NA
Myocarditis	Previous viral infection (eg, respiratory)	Acute or subacute onset after viral infection	Elevated troponin; elevated C-reactive protein	Normal or reduced systolic left ventricular function; typical myocardial LGE pattern; pericardial effusion	CMRI (LGE pattern); myocardial biopsy
Pre-existing idiopathic or familial DCM or acquired cardiomyopathy	Heart failure signs or symptoms or known heart disease before pregnancy	During the second trimester of pregnancy	Elevated natriuretic peptides	Reduced systolic left ventricular function; right ventricular dysfunction possible; typical myocardial late enhancement pattern (DCM)	History; echocardiography; CMRI (LGE pattern)
Takotsubo syndrome	Chest pain; stressful delivery or emergency due to fetal complications	Acute onset during delivery or immediately after	Elevated natriuretic peptides	Regional wall motion irregularities with typical anatomical patterns	History; echocardiography
Pregnancy-associated myocardial infarction	Chest pain; epigastric pain	Acute onset during pregnancy or after delivery	Elevated troponin	Regional wall motion irregularities; ischaemic myocardial scar	History; echocardiography; coronary angiography; CMRI (LGE pattern)
Pulmonary embolism	Chest pain; unilateral leg swelling; acute dyspnoea	Acute onset during pregnancy or after delivery	Elevated natriuretic peptides or troponin; elevated D-dimer	Right ventricular dysfunction; right ventricular dilation; left ventricular function usually normal	CT; ventilation or perfusion scan
Amniotic fluid embolism	Chest pain during or immediately after delivery; acute dyspnoea	Acute onset during delivery or immediately after	Elevated natriuretic peptides possible	Reduced systolic right ventricular function; right ventricular dilatation	History; echocardiography
Hypertensive heart disease or severe pre-eclampsia	Pre-existing or new-onset hypertension; proteinuria	During the second trimester of pregnancy	Elevated natriuretic peptides	Left ventricular hypertrophy; diastolic dysfunction; transient left ventricular dysfunction	History; echocardiography
Hypertrophic cardiomyopathy	Familial predisposition	During the second trimester of pregnancy	Elevated natriuretic peptides	Left ventricular hypertrophy; typical myocardial LGE pattern; LVOTO (HOCM)	History; echocardiography; CMRI (LGE pattern)
HIV/AIDS cardiomyopathy	HIV infection; AIDS	During the second trimester of pregnancy	Elevated natriuretic peptides	Reduced systolic left ventricular function; left ventricle or right ventricle often not dilated	HIV serology; test
Pre-existing (unknown) congenital heart disease	Heart failure signs or symptoms before pregnancy; known heart disease; previous cardiac surgery	During the second trimester of pregnancy	Elevated natriuretic peptides	(Corrected) Congenital heart defects; cardiac shunts	History; echocardiography
Pre-existing valvular heart disease	Heart failure signs or symptoms before pregnancy; known heart disease	During the second trimester of pregnancy	Elevated natriuretic peptides	Valvular stenosis or regurgitation; prosthetic heart valves	History; echocardiography

CMRI=cardiac magnetic resonance imaging. DCM=dilative cardiomyopathy. HOCM=hypertrophic obstructive cardiomyopathy. LGE=late gadolinium enhancement. LVEF=left ventricular ejection fraction. LVOTO=left ventricular outflow tract obstruction. NA=not applicable.

Table: Differential diagnosis of peripartum cardiomyopathy.

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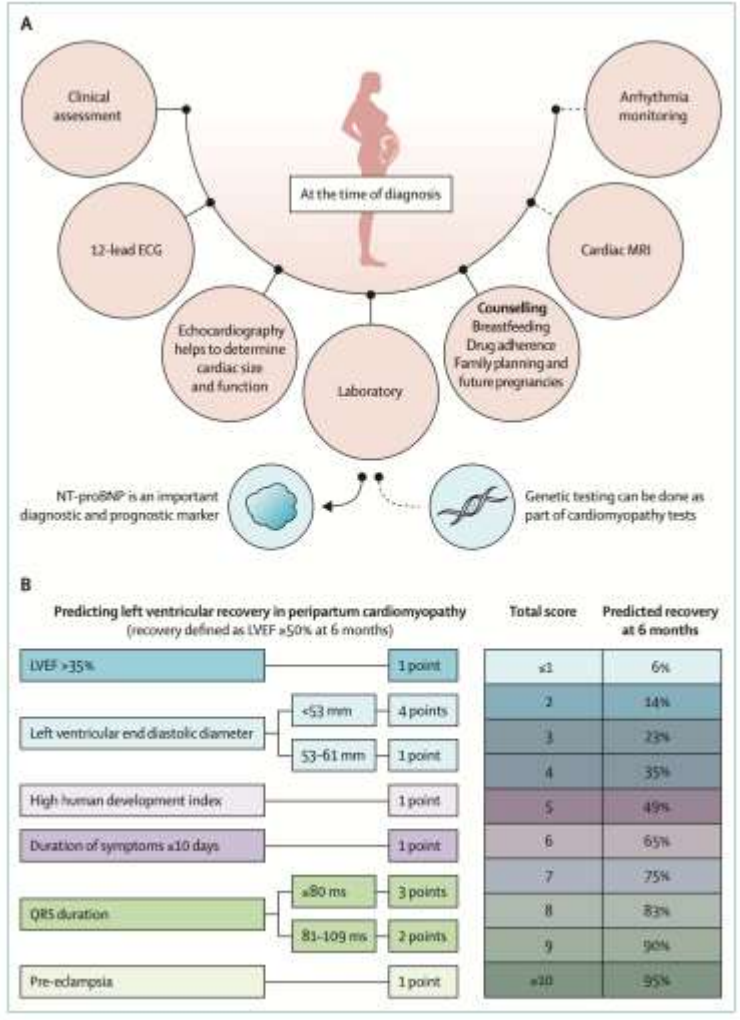

BOARD scheme

Bromocriptine (dose according to severity of the disease)	**Oral heart failure drugs** (β blockers, ACE inhibitor or ARB, MRA)	**Anticoagulation** (at least in prophylactic dose)	**Relaxants** (intravenous vasodilators if SBP >110 mm Hg)	**Diuretics** (in case of fluid overload)

Drug	Heart failure and absence of complete left ventricular recovery	Complete and sustained recovery (LVEF >55% and NYHA functional class I)
β blocker	Essential for all patients in standard or maximally tolerated dosages	Continue all drugs for at least 6–12 months or lifelong after full recovery; individual approach or discuss with patient. Discontinue stepwise and monitor symptoms and left ventricular function: 1) MRA 2) SGLT2 inhibitor 3) ARNI, ACE inhibitor, or ARB 4) β blocker
ARNI	Essential for all patients in standard or maximally tolerated dosages	
ACE inhibitor	Recommended for all patients in standard or maximally tolerated dosages when ARNI is not available or contraindicated	
ARB	Recommended in patients who do not tolerate ACE inhibitor	
MRA	Recommended for all patients in standard or maximally tolerated dosages; preferably eplerenone due to less hormonal side-effects and less blood pressure reduction compared with spironolactone	
SGLT2 inhibitor	Essential for all patients in standard dosage (dapagliflozin 10 mg or empagliflozin 10 mg once daily)	
Ivabradine	Recommended in patients in sinus rhythm with a persisting heart rate >70 bpm at rest despite maximal tolerated β blocker dose	Discontinue if heart rate <50 bpm or in case of complete recovery
Diuretics	Recommended in patients with fluid overload	Taper dose or discontinue if no signs of fluid overload; maintain only if part of antihypertensive therapy

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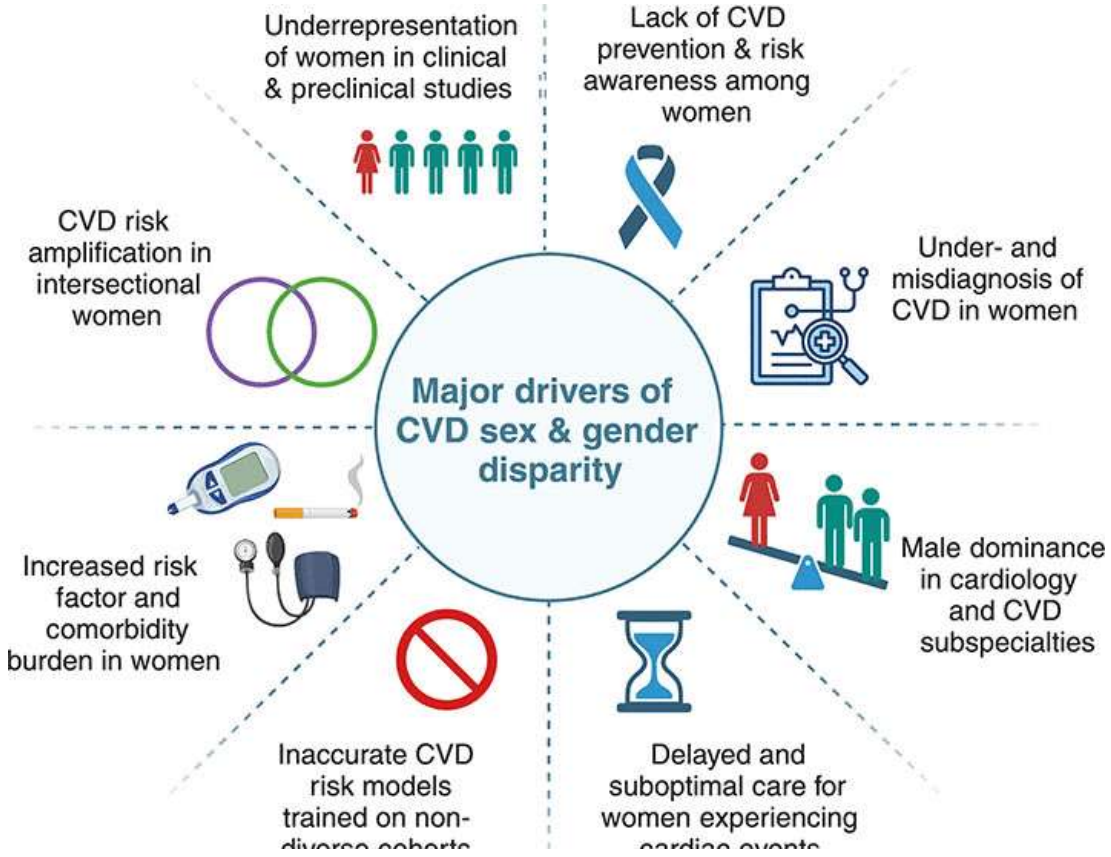
Summary


Underrepresentation of women in clinical & preclinical studies
Lack of CVD prevention & risk awareness among women
CVD risk amplification in intersectional women
Under- and misdiagnosis of CVD in women
Major drivers of CVD sex & gender disparity
Increased risk factor and comorbidity burden in women
Male dominance in cardiology and CVD subspecialties
Inaccurate CVD risk models trained on non-
Delayed and suboptimal care for women experiencing

Thank you