McGill University Health Centre

Centre universitaire de santé McGill

Top Cardiology Topics in 2022

Jacqueline Joza, MD MSc FRCPC

Cardiac Electrophysiologist

McGill University Health Centre

Disclosures

Medtronic: Grant – Investigator-initiated external research program

Boston Scientific – Advisory Board

Objectives

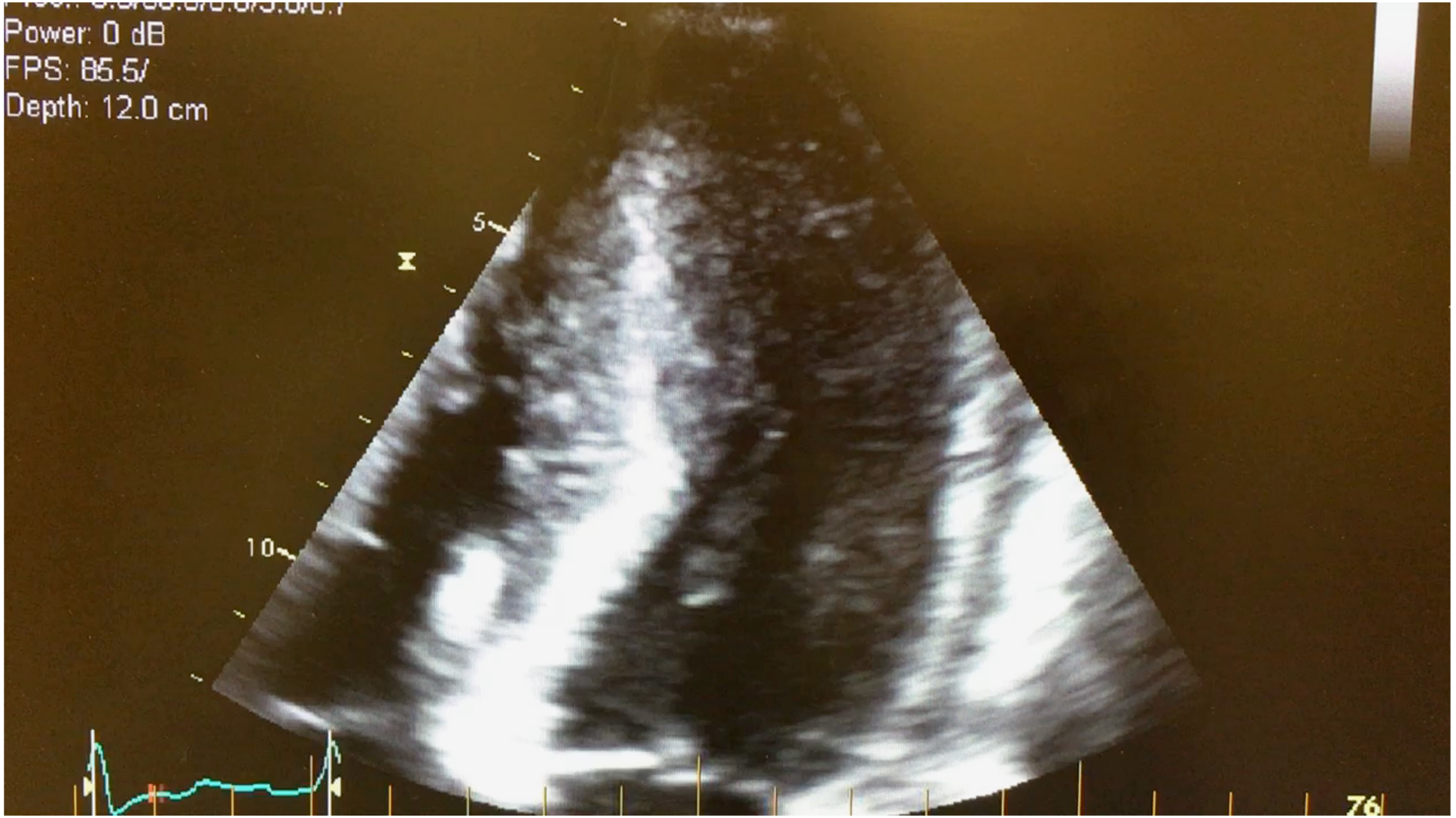
1. Learn about new updates in the treatment of hypertrophic cardiomyopathy

2. Evaluate the role of salt intake in heart failure patients

3. Understand the importance on intervening early in atrial fibrillation

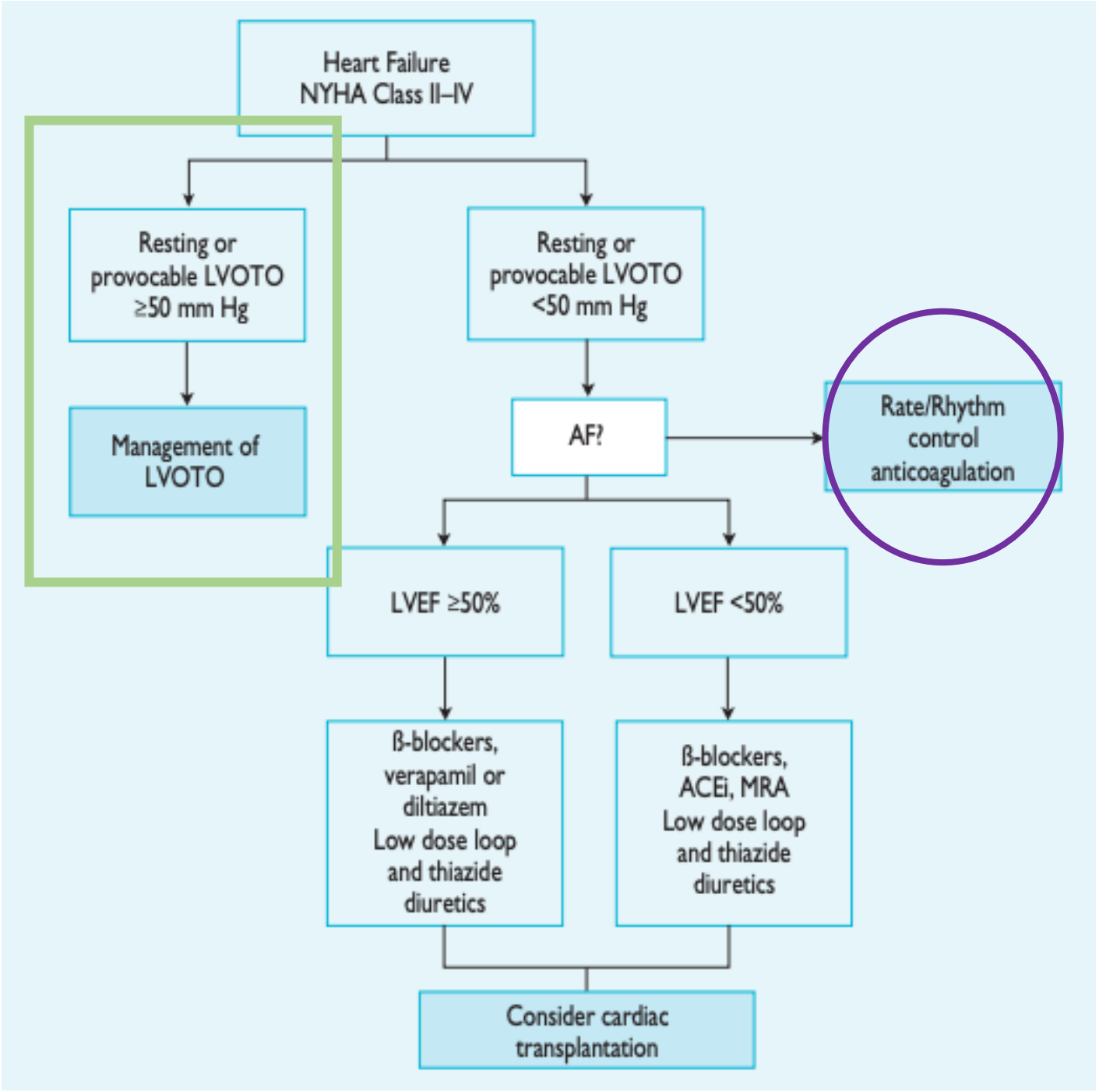
4. Discover updates in lipid lowering treatments

50 year-old man presents with a stroke: an echocardiogram is performed

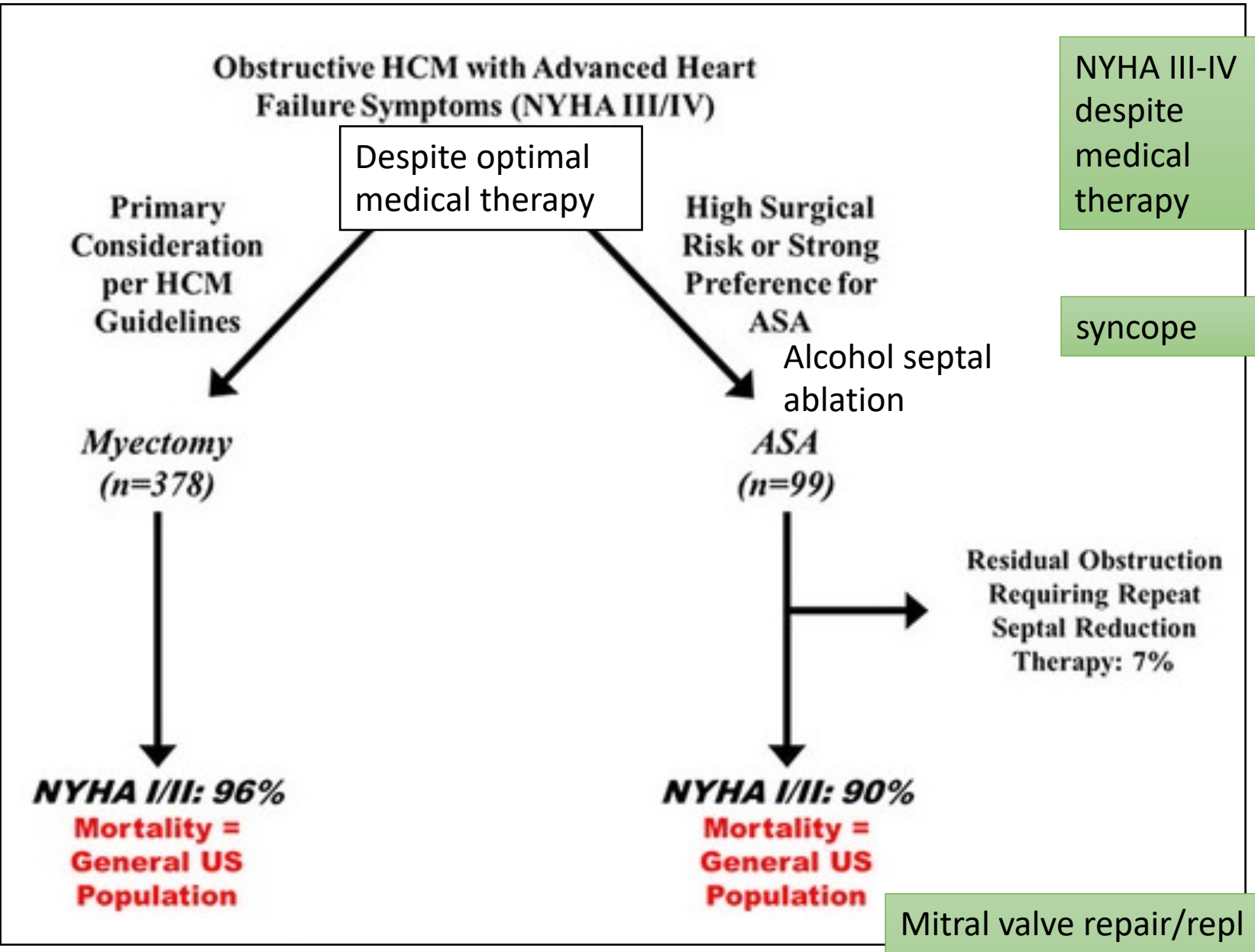


Hypertrophic Cardiomyopathy: with outflow tract obstruction

He describes several years of worsening dyspnea.
-Echo reveals a gradient of 70mmHg across the LV outflow tract at rest. What treatments are available for this patient?



ESC Guidelines Hypertrophic Cardiomyopathy 2020



Carey Kimmelstiel. Circulation: Cardiovascular Interventions. Volume: 12, Issue: 7

NYHA III-IV despite medical therapy

syncope

Septal myectomy > ASA

Mitral valve repair/repl if mod-severe regurg from septal anterior motion

Septal reduction therapy to improve symptoms is recommended in patients with a resting or maximum provoked LVOT gradient of ≥50 mm Hg, who are in NYHA functional Class III–IV, despite maximum tolerated medical therapy.	I	B
Septal reduction therapy should be considered in patients with recurrent exertional syncope caused by a resting or maximum provoked LVOTO gradient ≥50 mm Hg despite optimal medical therapy.	IIa	C
Septal myectomy, rather than SAA, is recommended in patients with an indication for septal reduction therapy and other lesions requiring surgical intervention (e.g. mitral valve repair/replacement, papillary muscle intervention).	I	C
Mitral valve repair or replacement should be considered in symptomatic patients with a resting or maximum provoked LVOTO gradient ≥50 mm Hg and moderate-to-severe mitral regurgitation not caused by SAM of the mitral valve alone.	IIa	C

ESC Guidelines Hypertrophic Cardiomyopathy 2020

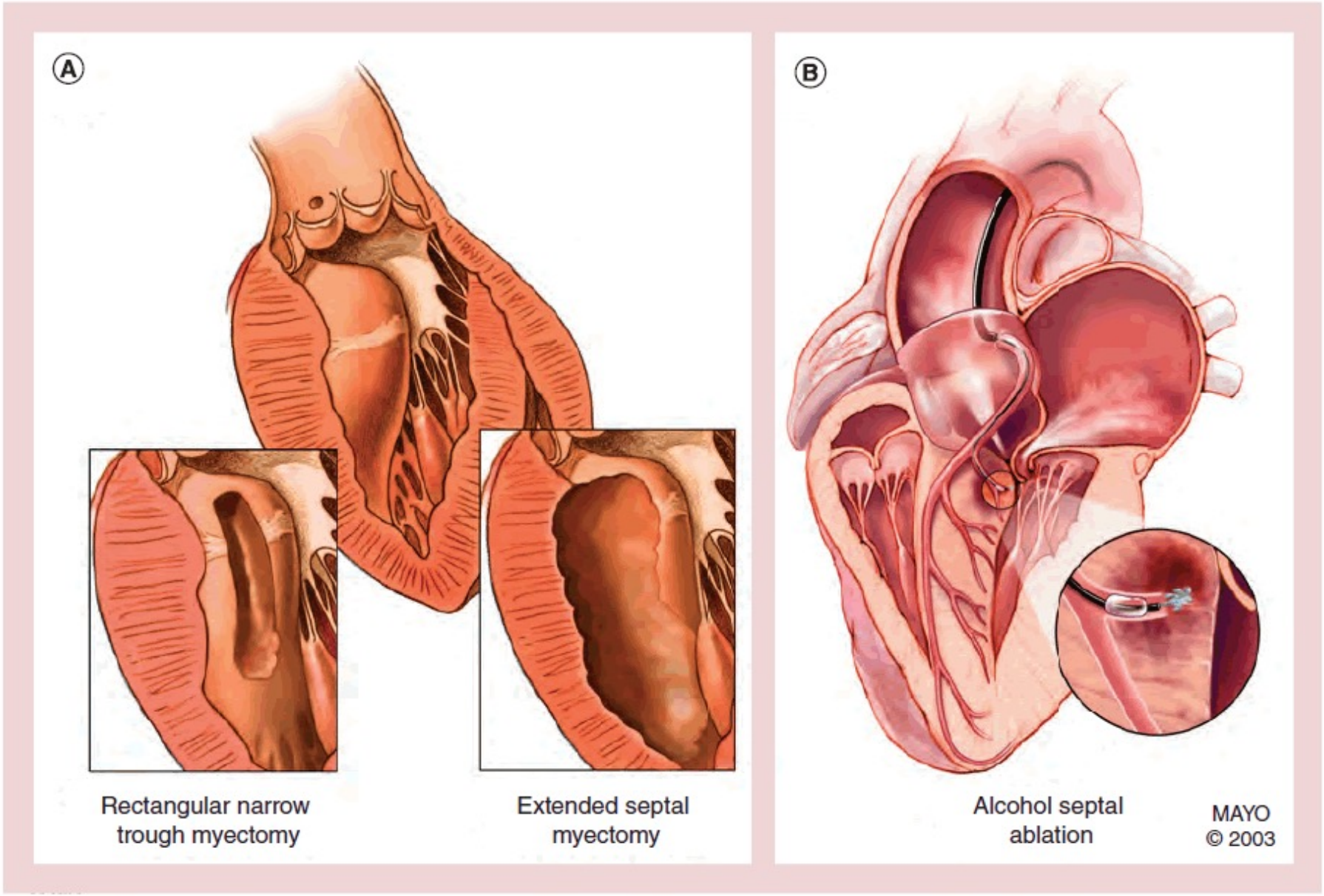


Figure 2. Septal reduction therapies. (A) Comparison of surgical techniques: Morrow trough myectomy (lower left) and extended myectomy (lower right). **(B)** Alcohol septal ablation performed via the first septal perforator. Reproduced with permission from [43] and the Mayo Foundation.

Concerns:
-Post-alcohol ablation or myectomy ventricular arrhythmias lead to increased risk of SCD
-Increased need for pacemaker implantation for heart block
-Complex with expertise required
-Post-op mortality 5.9%

Geske Interventional Cardiology. 2014 vol 6 issue 2

Myosin Inhibition in Patients With Obstructive Hypertrophic Cardiomyopathy Referred for Septal Reduction Therapy

VALOR-HCM: MAVACAMTEN to treat pts with obstructive HCM

Desai et al. JACC vol 80 No 2. 2022

Milind Y. Desai, MD, MBA,[a,b,c] Anjali Owens, MD,[d] Jeffrey B. Geske, MD,[e] Kathy Wolski, MPH,[b,c] Srihari S. Naidu, MD,[f] Nicholas G. Smedira, MD, MBA,[a,g] Paul C. Cremer, MD, MS,[b,c] Hartzell Schaff, MD,[h] Ellen McErlean, RN, MSN,[b,c] Christina Sewell, RN,[b,c] Wanying Li, PhD,[i] Lulu Sterling, PhD,[i] Kathy Lampl, MD,[i] Jay M. Edelberg, MD, PhD,[i] Amy J. Sehnert, MD,[i] Steven E. Nissen, MD[b,c]

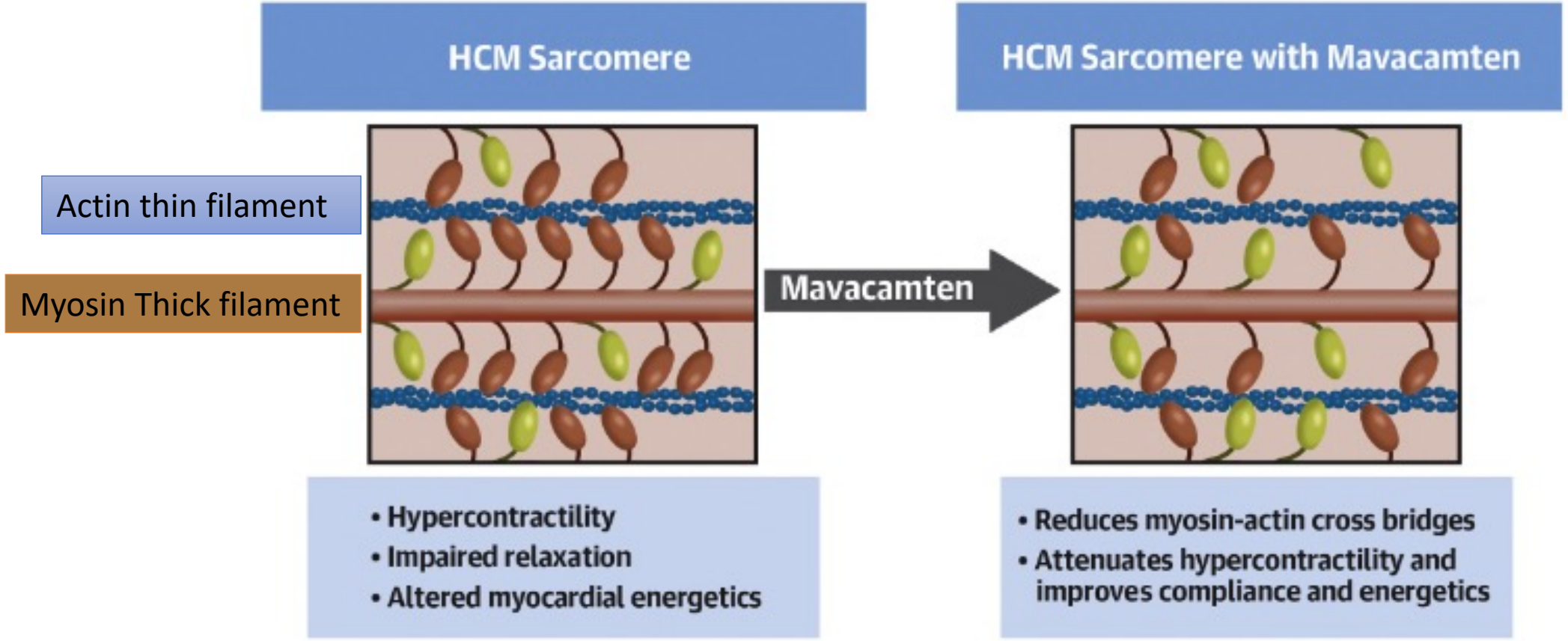
LVOT ≥ 50mmHg at rest or provocation who met criteria for septal reduction therapy

Mavacamten 5mg Qdaily, titrated up to 15mg based on LVOT gradient and LVEF

Placebo

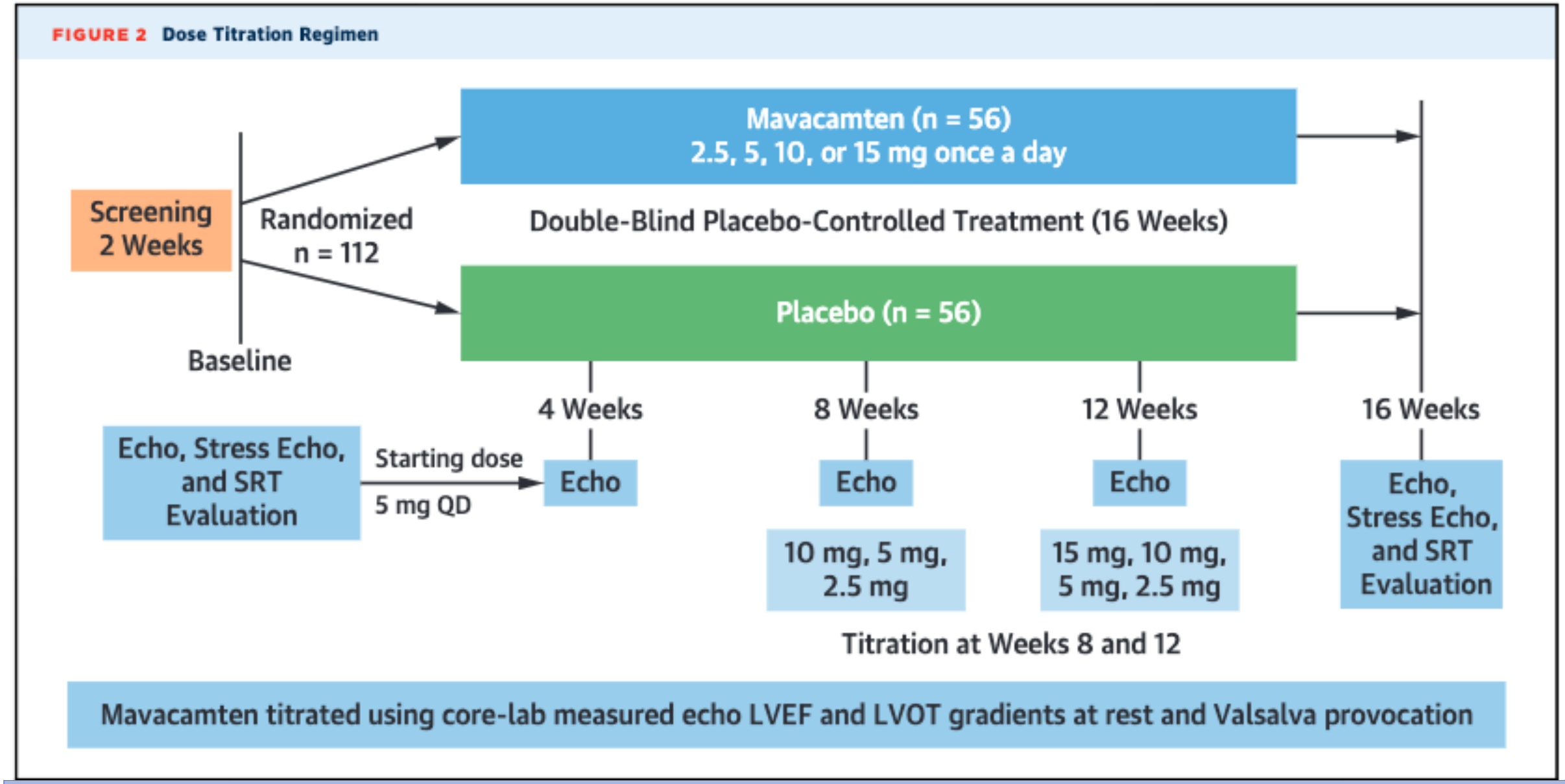
16 Weeks

Primary endpoint: composite of the proportion of pts proceeding with septal reduction therapy OR who remained guideline-eligible after 16 weeks of treatment



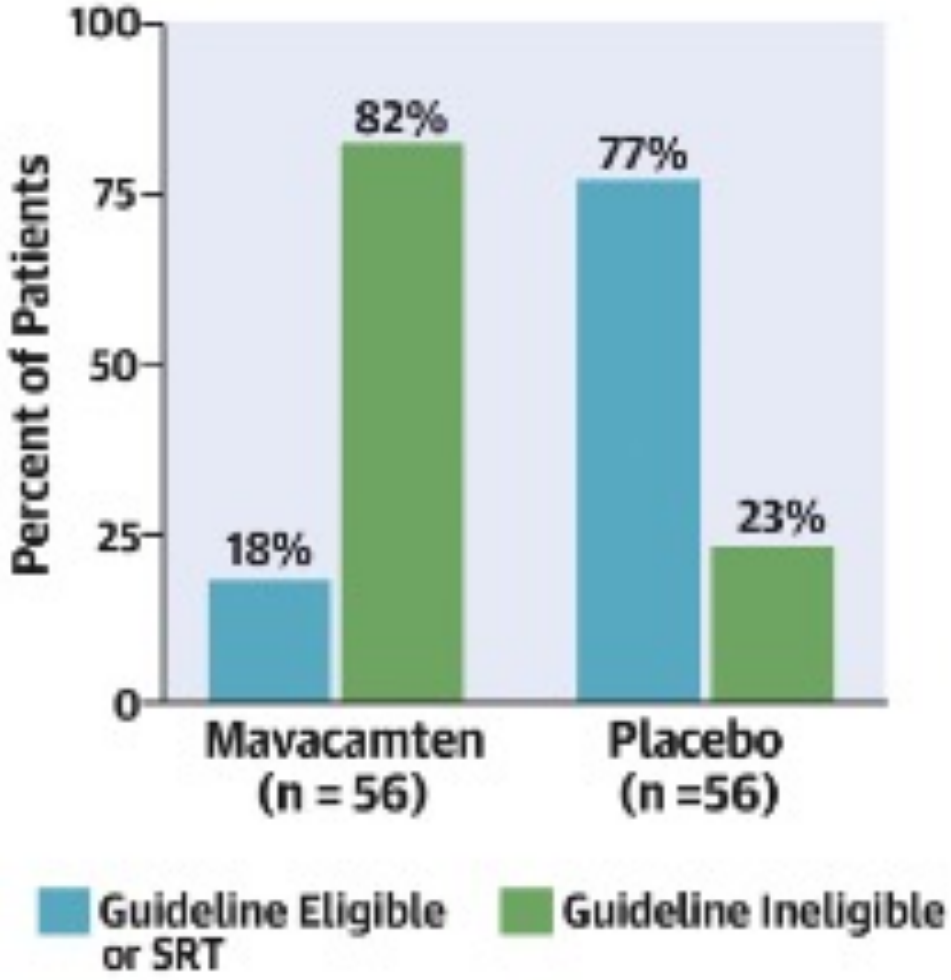
Mavacamten is a small molecule modulator of beta-cardiac myosin, that reversibly **inhibits myosin binding to actin,** directly inhibiting sarcomere force output to reduce contractility and improve ventricular compliance.

Safety outcomes: concern for a reduction in contractility (or LVEF); nausea known

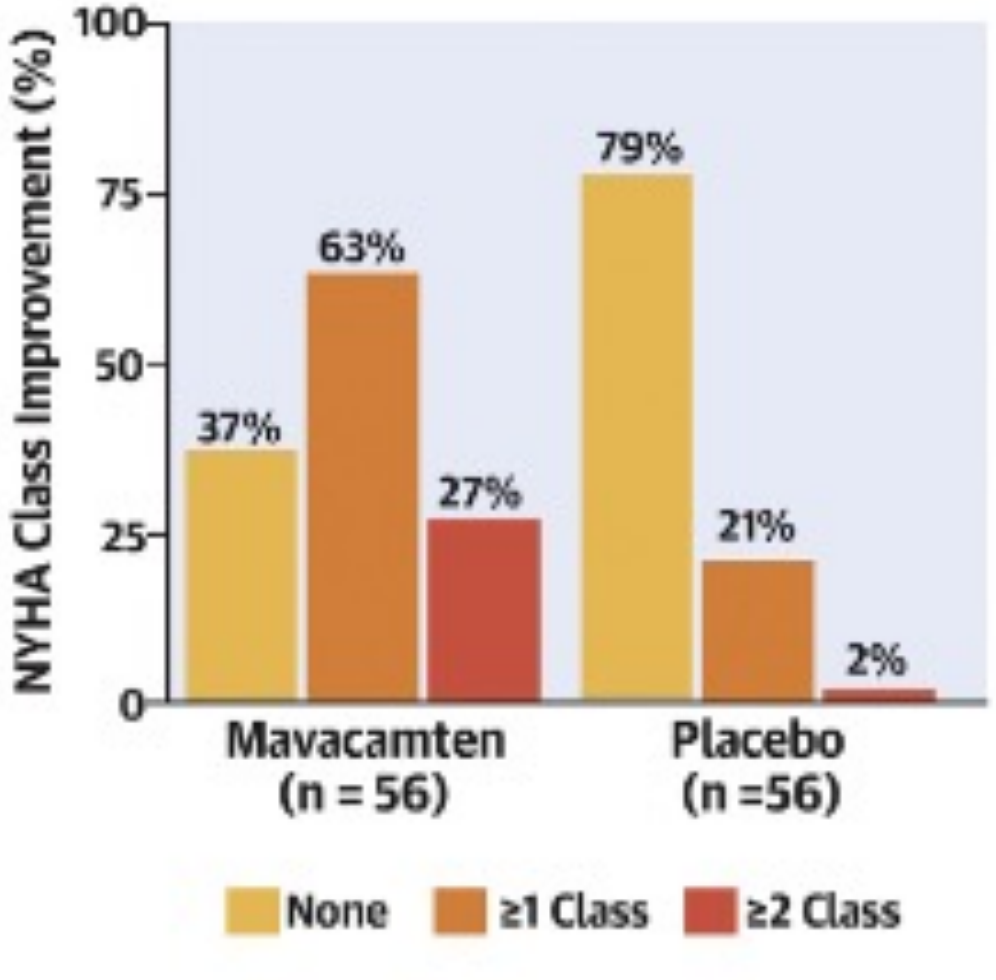


112 obstructive HCM patients enrolled: mean age 60 ± 12 years. 51% men; 93% NYHA III/IV
Mean post exercise LVOT gradient of 84 ± 35mmHg

Patients Who Underwent SRT or Remained Guideline Eligible for SRT

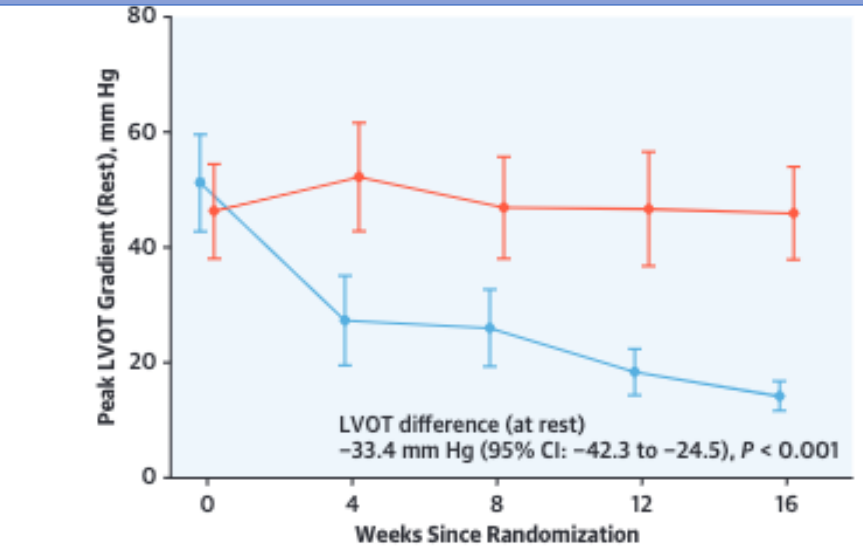


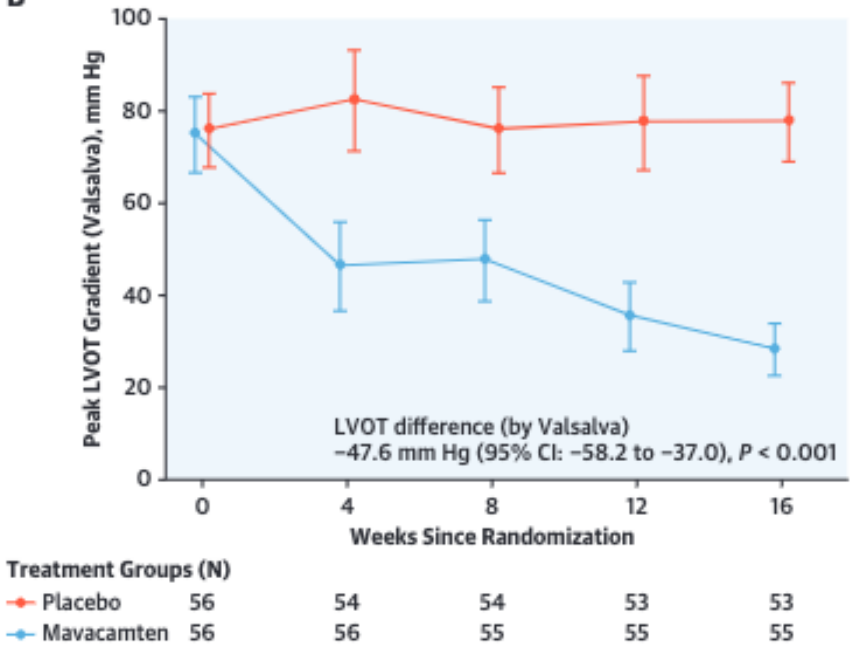
Patients Who Improved by 0, ≥1, or ≥2 NYHA Class



Desai MY, et al. J Am Coll Cardiol. 2022;80(2):95–108.

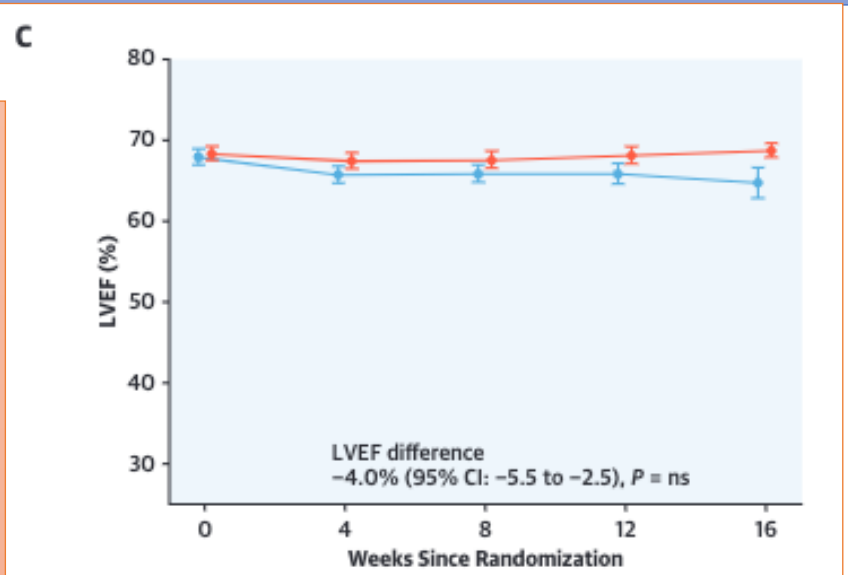
LVOT gradients significantly improved:-33mmHg improvement (rest) and -47mmHg (valsalva)

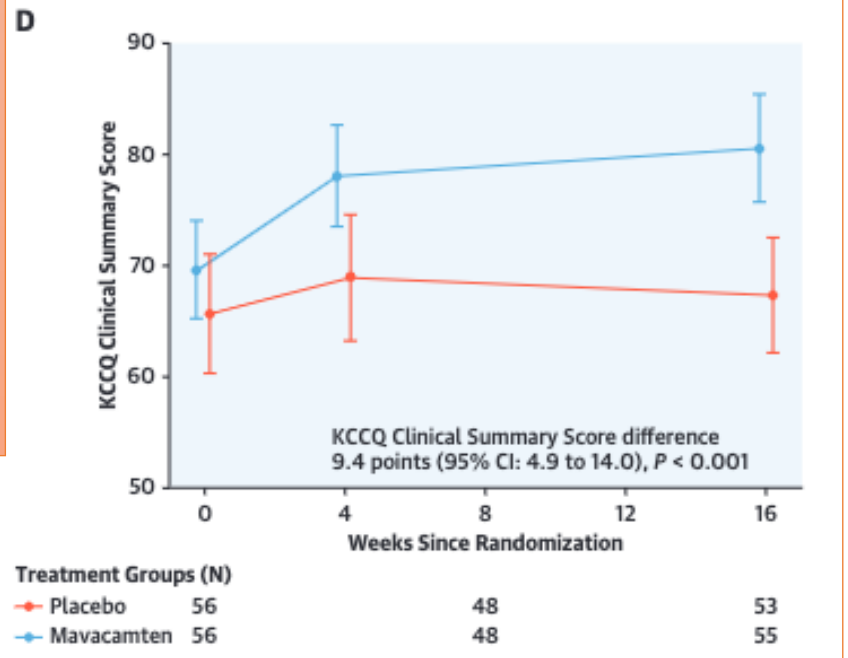




LVEF remained stable in both groups

Cardiomyopathy QOL score KCCQ showed significant improvement





2. 65 year-old female with ischemic cardiomyopathy, on optimal medical therapy.
LVEF 30%.

What do you advise her regarding his salt intake?

Sodium restriction has been universally recommended in heart failure management due to the belief that sodium consumption leads to edema and fluid retention
-Observational studies have not shown clear benefits of sodium restriction to specific targets, and randomized trials have been lacking

THE LANCET

Volume 399, Issue 10333, 9 April 2022, Pages 1391-1400

Articles

Reduction of dietary sodium to less than 100 mmol in heart failure (SODIUM-HF): an international, open-label, randomised, controlled trial

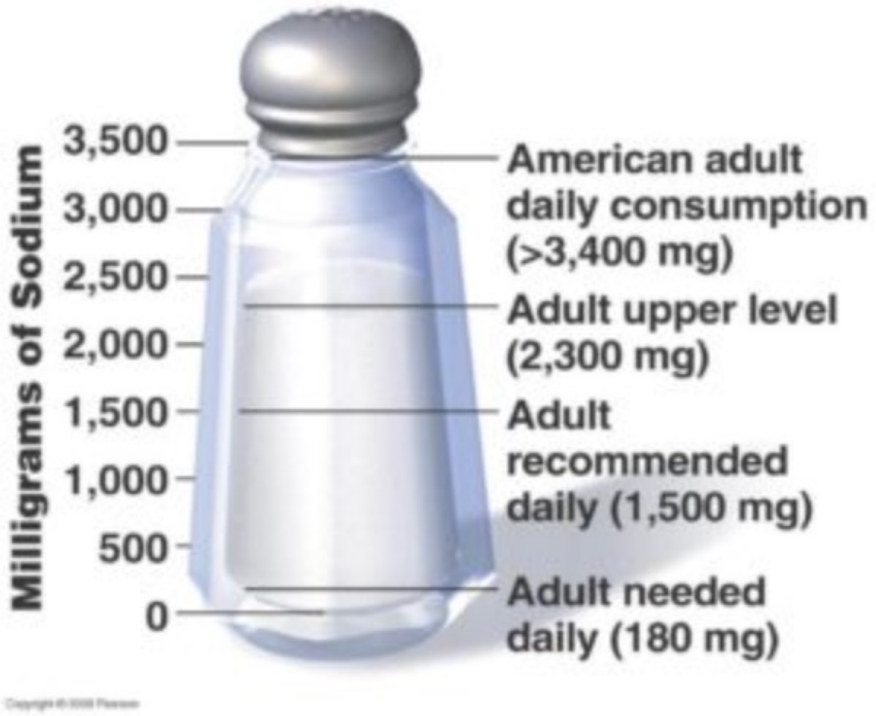
Prof Justin A Ezekowitz MBBCh [a], Prof Eloisa Colin-Ramirez PhD [b], Prof Heather Ross MD [c], Prof Jorge Escobedo MD [d], Prof Peter Macdonald MD [e], Prof Richard Troughton MD [f], Clara Saldarriaga MD [g], Wendimagegn Alemayehu PhD [a], Finlay A McAlister MD [a], JoAnne Arcand PhD [h], Prof John Atherton PhD [i], Prof Robert Doughty MD [j], Milan Gupta MD [k], Jonathan Howlett MD [l], Shahin Jaffer MD [m], Andrea Lavoie MD [n], Mayanna Lund MBChB [o], Prof Thomas Marwick PhD [p], Prof Robert McKelvie MD [q], Prof Gordon Moe MD [r] …Shelley Zieroth

Patients with chronic heart failure on optimally tolerated guideline-directed medical therapy were included.
Exclusions: mean dietary sodium intake <1500mg/d, Na < 130M, GFR<20, CV hospitalization in last month

At baseline: median sodium intake was 2286mg/day for low sodium diet group and 2119mg/day for usual diet. Randomized to:

A. Low sodium group: <1500mg/d with a normocaloric diet of 15-20% protein, 50-55% carbs, and 25-30% fat (7% saturate fat). + counseling by dietitians, physicians or nurses with daily sample menus.

B. Usual care: general advice to restrict dietary sodium



- 1/4 teaspoon salt = 575 mg. sodium
- 1/2 teaspoon salt = 1,150 mg. sodium
- 3/4 teaspoon salt = 1,725 mg. sodium
- 1 teaspoon salt = 2,300 mg. sodium

	Low sodium diet group (n=397)	Usual care group (n=409)
Age, years	66 (57–73)	67 (58–75)
Sex		
Female	127 (32%)	141 (34%)
Male	270 (68%)	268 (66%)
Geographical region		
Canada	230 (58%)	241 (59%)
Australia and New Zealand	79 (20%)	78 (19%)
Mexico, Chile, and Colombia	88 (22%)	90 (22%)
Diagnosed with heart failure for ≥1 year	269 (68%)	282 (69%)
Hospitalised for heart failure in past 12 months	129 (32%)	141 (34%)
Ejection fraction	36 (28–48)	35 (27–50)
NYHA functional class		
1	2 (1%)	6 (1%)
2	293 (74%)	283 (69%)
3	98 (25%)	119 (29%)
4	3 (1%)	0
Medical history		
Hypertension	246 (62%)	258 (63%)
Coronary artery disease	187 (47%)	186 (45%)
Peripheral arterial disease	33 (8%)	42 (10%)
Cerebrovascular disease (transient ischaemic attack or stroke)	45 (11%)	41 (10%)
Atrial fibrillation or flutter	156 (39%)	173 (42%)
Diabetes (type 1 or 2)	132 (33%)	156 (38%)
Chronic obstructive pulmonary disease	64 (16%)	72 (18%)
Previous ventricular fibrillation or tachycardia	65 (16%)	59 (14%)
(Continued from previous column)		
Laboratory values		
BNP, pg/mL†	194 (74–470)	222 (85–541)
NT-proBNP, pg/mL†	763 (228–1161)	934 (418–2169)
eGFR, mL/min per 1·73 m²‡	61 (46–75)	58 (42–71)
Serum sodium, mmol/L	139 (137–141)	139 (137–141)
Serum potassium, mmol/L	4 (4–5)	4 (4–5)
Medical and device therapy		
Any RAAS inhibitor (ACE, ARB, or ARNI)	314 (79%)	335 (82%)
β blocker	351 (88%)	351 (86%)
ACE or ARB	256 (64%)	284 (69%)
Sacubitril–valsartan	63 (16%)	53 (13%)
Mineralocorticoid antagonist	237 (60%)	224 (55%)
Implantable cardioverter-defibrillator§	104 (26%)	81 (20%)
Pacemaker	36 (9%)	29 (7%)
Cardiac resynchronisation therapy	41 (10%)	33 (8%)

Data are median (IQR) or n (%). ACE=angiotensin converting enzyme. ARB=angiotensin receptor blocker. ARNI=angiotensin receptor blocker neprilysin inhibitor. BMI=body-mass index. BNP=b-type natriuretic peptide. eGFR=estimated glomerular filtration rate. NT-proBNP=N-terminal b-type natriuretic peptide. NYHA=New York Heart Association. RAAS=renin-angiotensin-aldosterone system. *Available in 118 patients. †Within 90 days of enrolment, and BNP records were available for 263 patients (n=127 in low sodium diet group, n=136 in usual care group) and NT-proBNP records were available for 62 patients (n=27 low sodium diet group, n=35 usual care group). ‡Significant difference between groups; p=0·036. §Significant difference between groups; p=0·037.

Table 1: **Baseline clinical and demographic characteristics**

2014-2020: 806 patients were randomly assigned to low-sodium diet or usual care

<u>Primary endpoint:</u>
Composite of CV hospitalization, ER visit for CV cause, or all cause mortality

Results: Reduced dietary sodium vs. usual care in patients with heart failure (intention-to-treat analysis)

Outcomes	Events/100 patient-y		At 12 mo
	Reduced sodium†	Usual care†	RRR (95% CI)‡
Primary composite§	17.2	19.2	1% (–41 to 32)‖
CV hospitalization	11.4	13.8	6% (–48 to 40)
			RRI (CI)‡
CV emergency department visit	4.7	3.9	6% (–51 to 125)
All-cause mortality	6.0	4.3	34% (–36 to 171)

Conclusions:

-992 patients were needed to provide 80% power to detect a 30% RRR in the primary composite outcome, assuming a 25% event rate in the usual care group

This study **failed to show any benefit** of recommending to restrict sodium intake to 1500g/day

-The lack of benefit may be due to

-limited statistical power related to early termination of the trial (Covid-19 reduced hospital admissions)

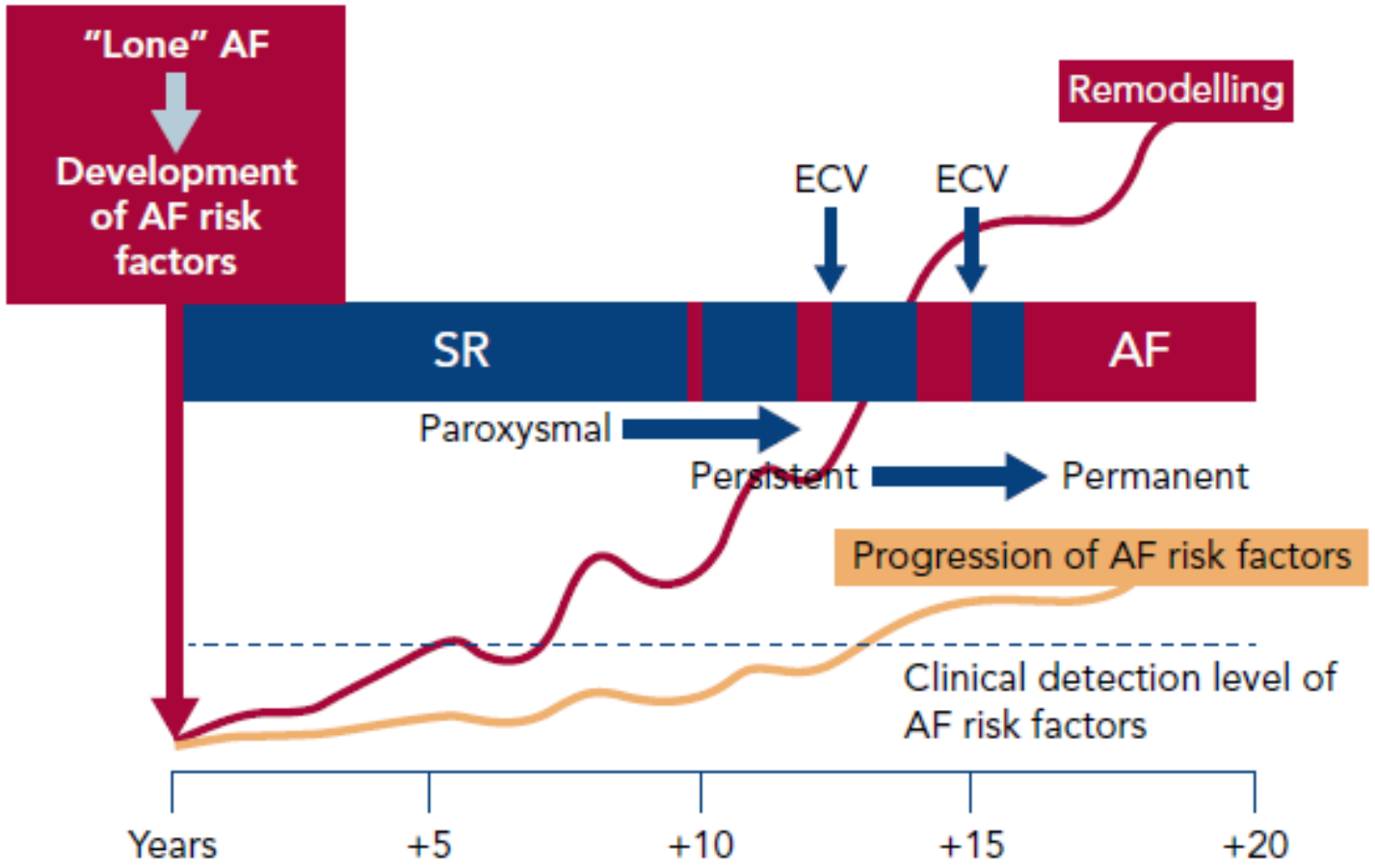
-The median daily sodium intake in the usual care group exceeded that of the reduced sodium group by only 415mg/d – (even the control group restricted sodium by a fair amount)

-Findings do not support a goal of restricting dietary sodium in pts with HF compared to a more general low sodium recommendation

-However, we cannot conclude that sodium restriction is not an important part of HF management

3. 55M with a 3rd episode of atrial fibrillation episode. This was despite being on metoprolol 25mg po BID. Seen in the Emergency department where he is given flecainide 100mg BID in addition to his metoprolol. **What is his risk of progression to persistent atrial fibrillation?**

Figure 2: Time-dependent Atrial Remodelling and Development of Atrial Fibrillation



An initial strategy of rhythm control has been associated with reduced risk of death from cv causes with reduced rates of stroke among patients in whom AFib had been diagnosed within the previous year

ORIGINAL ARTICLE

Progression of Atrial Fibrillation after Cryoablation or Drug Therapy

Jason G. Andrade, M.D., Marc W. Deyell, M.D., Laurent Macle, M.D., George A. Wells, Ph.D., Matthew Bennett, M.D., Vidal Essebag, M.D., Ph.D., Jean Champagne, M.D., Jean-Francois Roux, M.D., Derek Yung, M.D., Allan Skanes, M.D., Yaariv Khaykin, M.D., Carlos Morillo, M.D., et al., for the EARLY-AF Investigators*

November 7, 2022
DOI: 10.1056/NEJMoa2212540

303 pts with paroxysmal AF on a rate-controlling medication were randomized

154 patients underwent AF ablation (cryo-ablation); 18 underwent repeat ablation

149 were assigned first-line antiarrhythmic drug
-36 and 18 pts crossed over to ablation in 1st and 2nd year

3 year follow-up with Implantable loop recorder

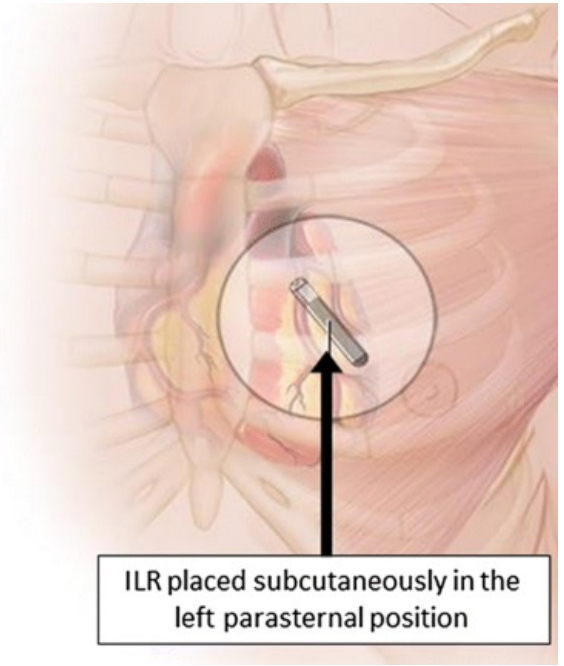
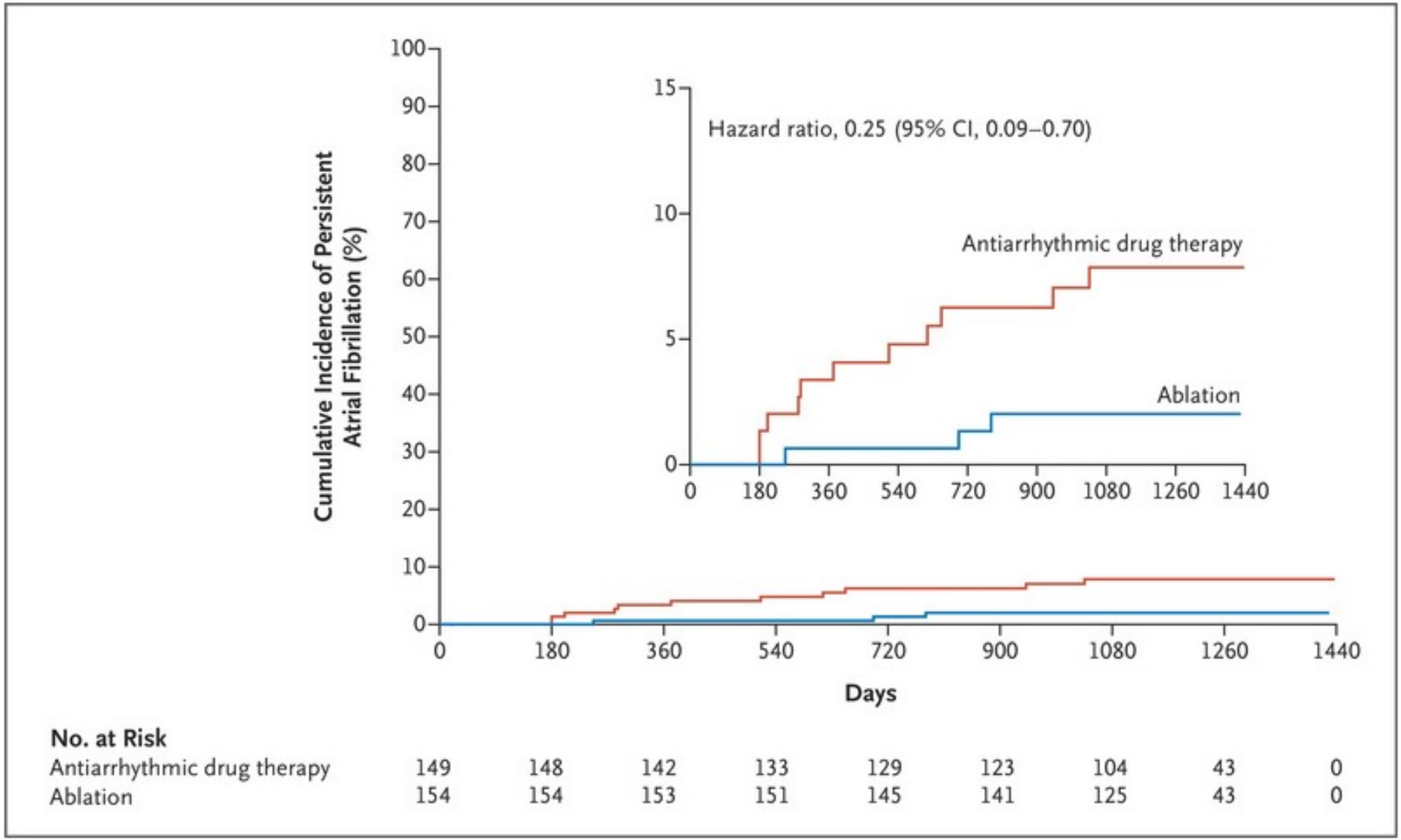


Figure 2. Cumulative Incidence of First Episode of Persistent Atrial Fibrillation.



Conclusion

Initial treatment of paroxysmal atrial fibrillation with catheter ablation was associated with a lower incidence of persistent atrial fibrillation or recurrent atrial tachyarrhythmia over 3 years of follow-up than initial use of antiarrhythmic drugs.

58 year old M

Diabetic x 3 years

Non ST segment elevation MI at age 52 with 2 sents placed in the right coronary artery

On atorvastatin 40mg Qd; LDL remains 2.6

What is the next step?

Table 1. Who to screen for dyslipidemia in adults at risk

Who to screen
Men 40 years of age or older; women 40 years of age or older (or postmenopausal)
• Consider earlier in ethnic groups at increased risk such as South Asian or indigenous individuals
All patients with any of the following conditions, regardless of age
• Clinical evidence of atherosclerosis
• Abdominal aortic aneurysm
• Diabetes mellitus
• Arterial hypertension
• Current cigarette smoking
• Stigmata of dyslipidemia (corneal arcus, xanthelasma, xanthoma)
• Family history of premature CVD*
• Family history of dyslipidemia
• CKD (eGFR $\leq$ 60 mL/min/1.73 m^2 or ACR $\geq$ 3 mg/mmol)
• Obesity (BMI $\geq$ 30)
• Inflammatory diseases (RA, SLE, PsA, AS, IBD)
• HIV infection
• Erectile dysfunction
• COPD
• History of hypertensive disorder of pregnancy

Pharmacological Treatment Indications

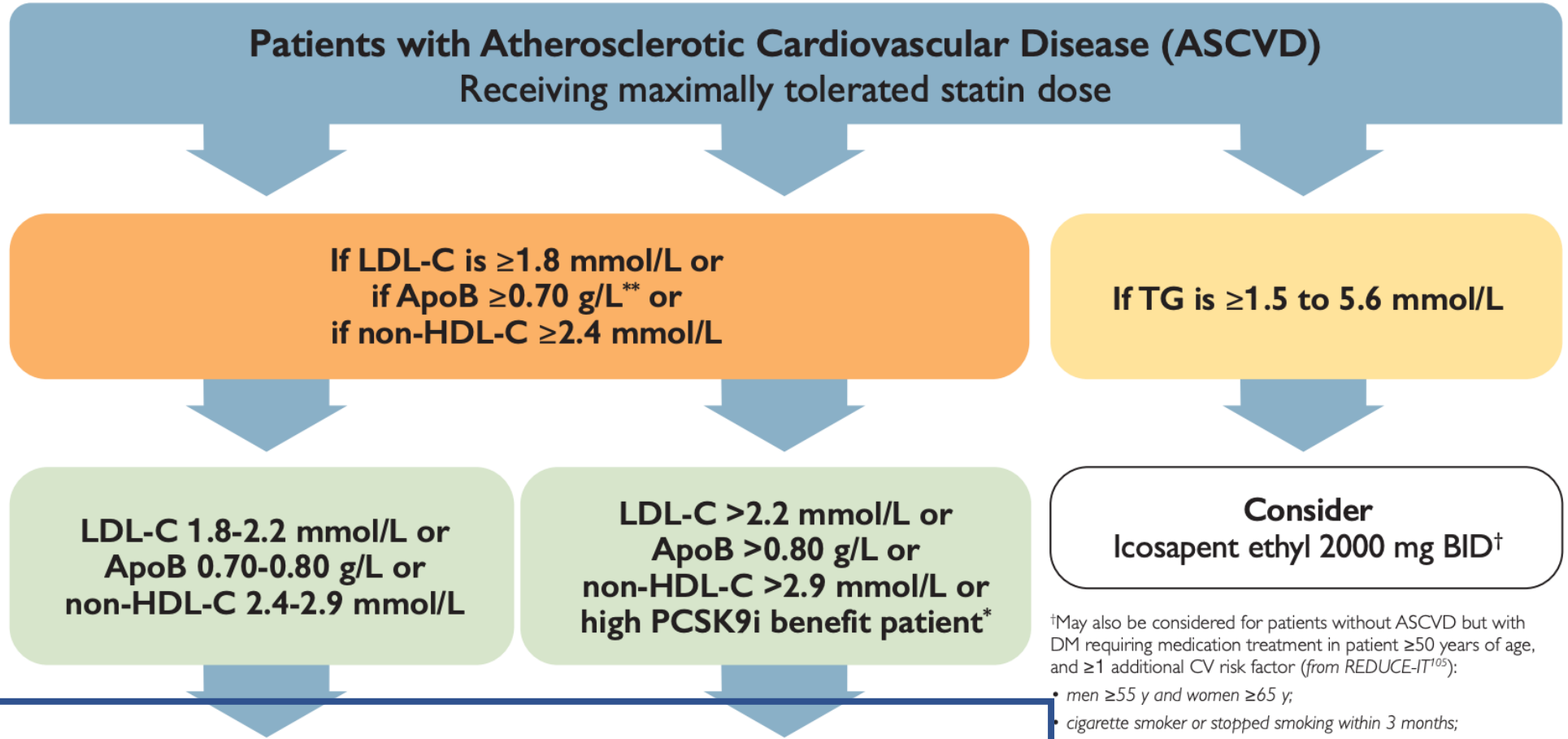
Category	Consider Initiating pharmacotherapy if:	Recommendation
Primary Prevention†	**High*** **FRS ≥20%**	We recommend use of high-intensity statin therapy in addition to appropriate health behaviour modifications as initial therapy for all eligible patients to prevent CVD. For patients who do not tolerate a high-intensity statins, we recommend the maximally tolerated statin dose.
	Intermediate Risk* **FRS 10-19.9% and** LDL-C ≥3.5 mmol/L **or** Non-HDL-C ≥4.2 mmol/L **or** ApoB ≥1.05 g/L **or** Men ≥50 yrs and women ≥60 yrs with one additional risk factor: low HDL-C, IFG, high waist circumference, smoker, or HTN **or** with presence of other risk modifiers: hsCRP ≥2.0 mg/L, CAC >0 AU, family, history of premature CAD, Lp(a), ≥50 mg/dL (≥100 nmol/L)	
	Low-Risk*†‡ **FRS <10%**	
Statin Indicated Conditions	Atherosclerotic Cardiovascular Disease (ASCVD): • Myocardial infarction (MI), acute coronary syndromes (ACS) • Stable angina, documented coronary artery disease using angiography • Stroke, TIA, document carotid disease • Peripheral arterial disease, claudication, and/or ABI <0.9 • Abdominal aortic aneurysm (AAA) -- abdominal aorta >3.0 cm or previous aneurysm surgery	
	Most patients with diabetes: • Age ≥40y • Age ≥30y & diabetes x ≥15y duration • Microvascular disease	
	Chronic Kidney Disease • Age ≥50y and eGFR <60 mL/min/1.73 m2 or ACR >3 mg/mmol	
	LDL-C ≥5.0 mmol/L(or ApoB ≥1.45 g/L or non-HDL-C ≥5.8 mmol/L) (familial hypercholesterolemia or genetic dyslipidemia)	

**Screening should be repeated every 5 years for men and women aged 40 to 75 years using the modified FRS (Framingham Risk Score) or CLEM to guide therapy to reduce major CV events. A risk assessment might also be completed whenever a patient's expected risk status changes.*

†Calculate risk using the FRS

†‡Refer to page 19 for low-risk individuals who may benefit from statin therapy.

Treatment Approach for Patients with a Statin Indicated Condition



*Patients shown to derive largest benefit form intensification of statin therapy with PCSK9 inhibitor therapy are identified in Table 3.

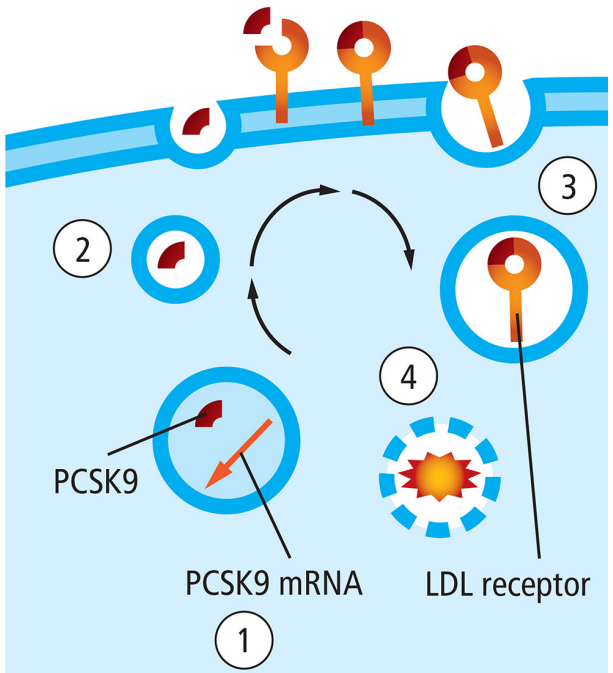
**At low levels of LDL-C or non-HDL-C, measurement of apoB is more accurate than other markers.

†May also be considered for patients without ASCVD but with DM requiring medication treatment in patient ≥50 years of age, and ≥1 additional CV risk factor (*from REDUCE-IT*[105]):

- *men ≥55 y and women ≥65 y;*
- *cigarette smoker or stopped smoking within 3 months;*
- *hypertension (≥140 mmHg systolic OR ≥90 mmHg diastolic) or on BP medication;*
- *HDL-C ≤1.04 mmol/L for men or ≤1.3 mmol/L for women;*
- *hsCRP >3.0 mg/L;*
- *Renal dysfunction: eGFR >30 and <60 mL/min;*
- *Retinopathy;*
- *Micro- or macroalbuminuria;*
- *ABI <0.9 without symptoms of intermittent claudication)*

Action of PCSK9 protein

1. PCSK9 is produced using mRNA
2. PCKS9 is secreted
3. PCKS9 binds to LDL receptor
4. LDL receptor is degraded



ASGPR = asialoglycoprotein receptor; PCSK9 =
RISC = RNA-induced silencing complex; siRNA =

The PCSK9 protein binds to and degrades the LDL receptor.
This causes LESS LDL receptors at the surface
Less LDL will be hepatically cleared

PCSK9 inhibitors work by blocking the PCSK9 proteins that break down those LDL receptors.

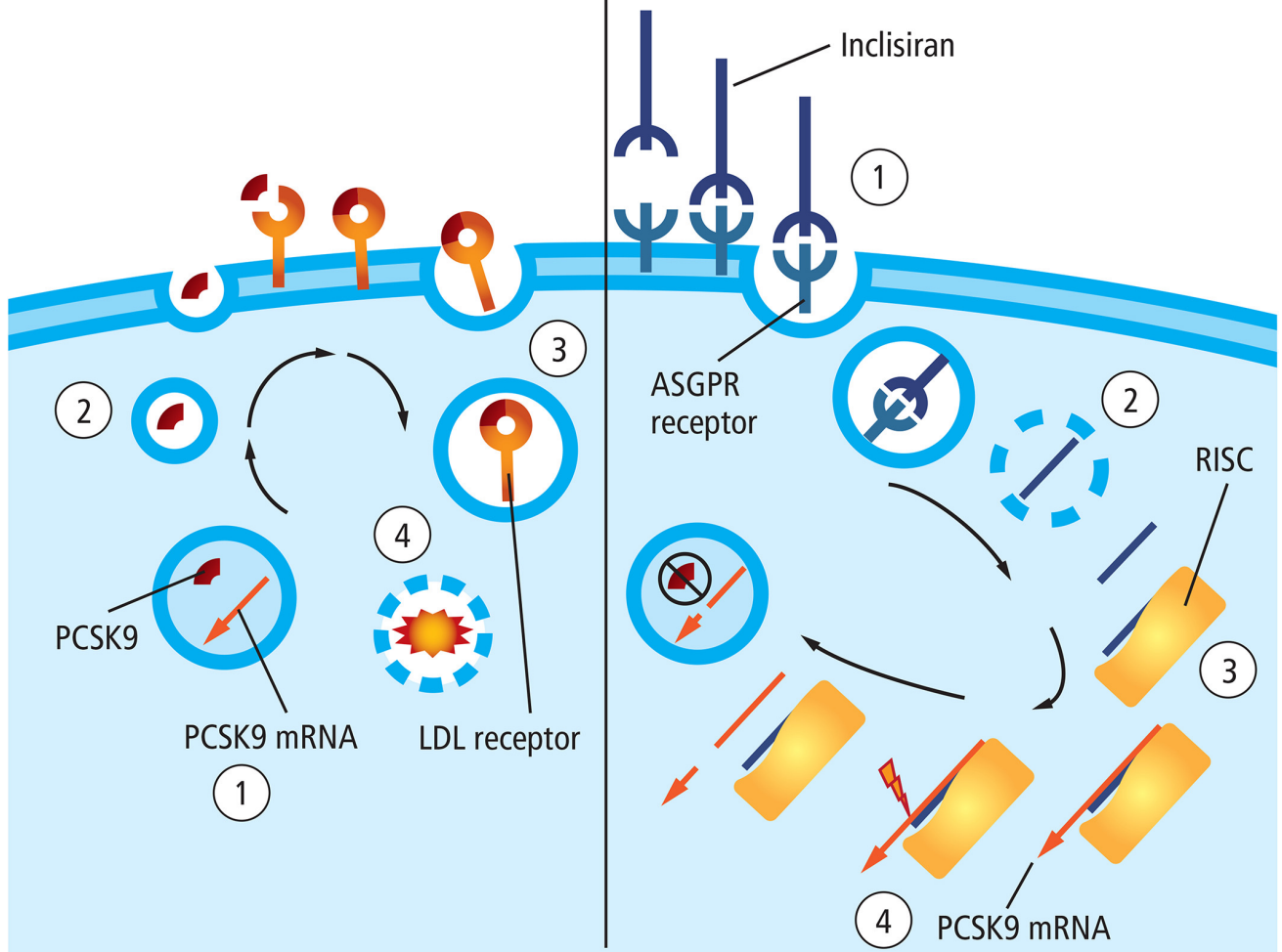
What if we move upstream, to prevent the PCSK9 protein from even being formed in the first place?

Action of PCSK9 protein

1. PCSK9 is produced using mRNA
2. PCKS9 is secreted
3. PCKS9 binds to LDL receptor
4. LDL receptor is degraded

Action of inclisiran

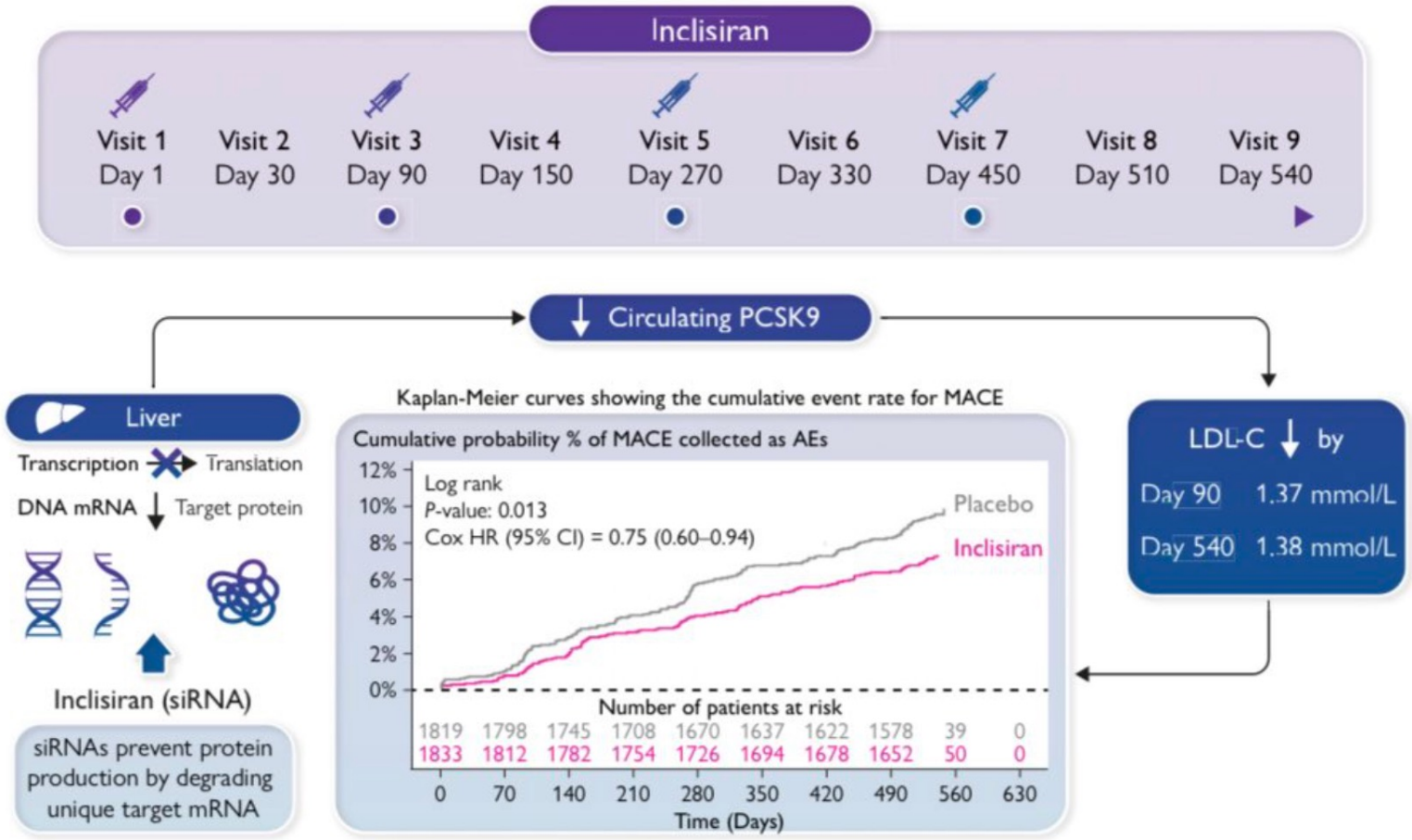
1. Inclisiran binds to ASGPR receptor and is endocytosed
2. Endosome is degraded releasing siRNA
3. siRNA loaded into RISC complex
4. Complex binds to PCSK9 mRNA and destroys it



ASGPR = asialoglycoprotein receptor; PCSK9 = proprotein convertase subtilisin-kexin type 9; RISC = RNA-induced silencing complex; siRNA = small interfering RNA.

New long-term Leqvio® (inclisiran) data from Novartis show sustained efficacy and safety over four years

ORION -3 Study Presented at AHA, 2022



Inclisiran and risk of reported MACE from the patient-level pooled ORION-9, ORION-10 and ORION-11 trials.

Rivaroxaban for rheumatic heart disease associated atrial fibrillation - INVICTUS

Ganesan Karthikeyan, Stuart Connolly*, Mpiko Ntsekhe, Bongani Mayosi* and Salim Yusuf**, for the INVICTUS Steering Committee and investigators

28th August 2022

**PI, **Steering committee chair*

- Rheumatic heart disease affects > 40 million people – mainly young and low income countries
- About 20% of symptomatic RHD patients have AF
- No Randomized trials of anticoagulation in RHD-AF
- Less than ½ the patients are prescribed a and just 1/3rd
- achieve therapeutic INRs in these countries (very challenging)

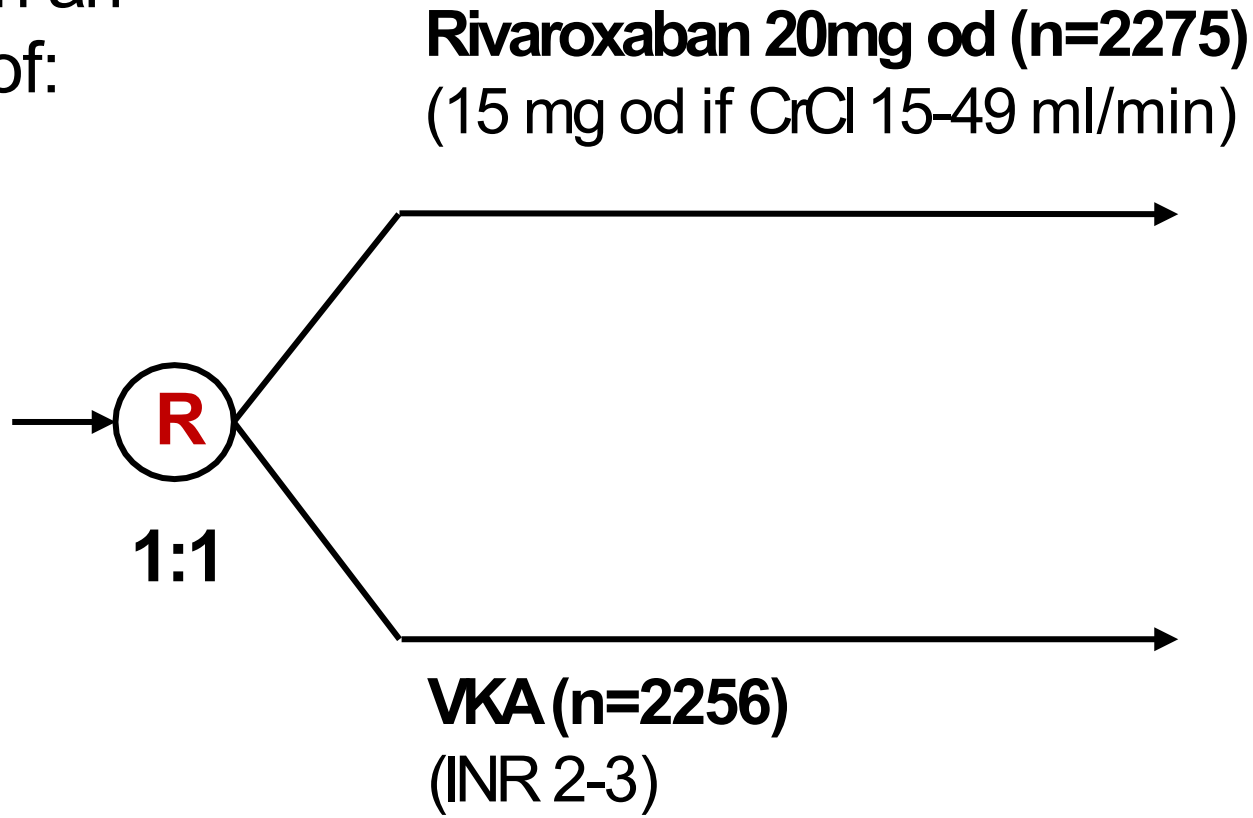
- **An anticoagulant that does not need monitoring would be of great benefit**

Patients with RHD-AF or AFL with an additional RF for stroke: either of:

- **Moderate Mitral stenosis**
  (valve area ≤2cm²)

- **CHA_2DS_2VASc score** ≥2

  -Or LA clots
  -Or Spontaneous echo contrast on echo



Am Heart J 2020;225:69-77

Outcomes and analysis

- **Primary outcome**: Composite of **stroke/systemic embolism***, MI, death due to vascular or unknown cause

  *Original primary outcome

- **Primary safety** outcome: ISTH major bleeding

- 4500 patients, 80% power for non-inferiority, with margin at HR of 1.186 (upper bound of the 97.5% CI)

Centres and countries

-138 centres in 24 countries

-Africa, Asia, and Latin America

-PHRI, Hamilton Coordinating Centre

Baseline characteristics

	Rivaroxaban (n=2275)	**VKA (n=2256)**
Age, years, mean (2 decades younger than other AF/OAC trials)	50.7	50.3
Female sex, n (%)	1648 (72.4)	1626 (72.1)
Mitral valve stenosis, n (%): 2/3rd had severe MS	**1927 (85.5)**	**1903 (85.2)**
Congestive HF, n (%)	879 (38.6)	866 (38.4)
Hypertension, n (%)	522 (22.9)	535 (23.7)
Diabetes, n (%)	158 (6.9)	132 (5.9)
Stroke, n (%)	248 (10.9)	257 (11.4)
Coronary artery disease, n (%)	32 (1.4)	20 (0.9)
CHA_2DS_2-VASc score 0-1, n (%)	**978 (43)**	**993 (44)**

Drug compliance and INR control

Drug compliance by visit (%)		
Visit	**Riva**	**VKA**
I year	88.7	98
II year	84.4	97.7
III year	81.2	97.1
IV year	79	96.4

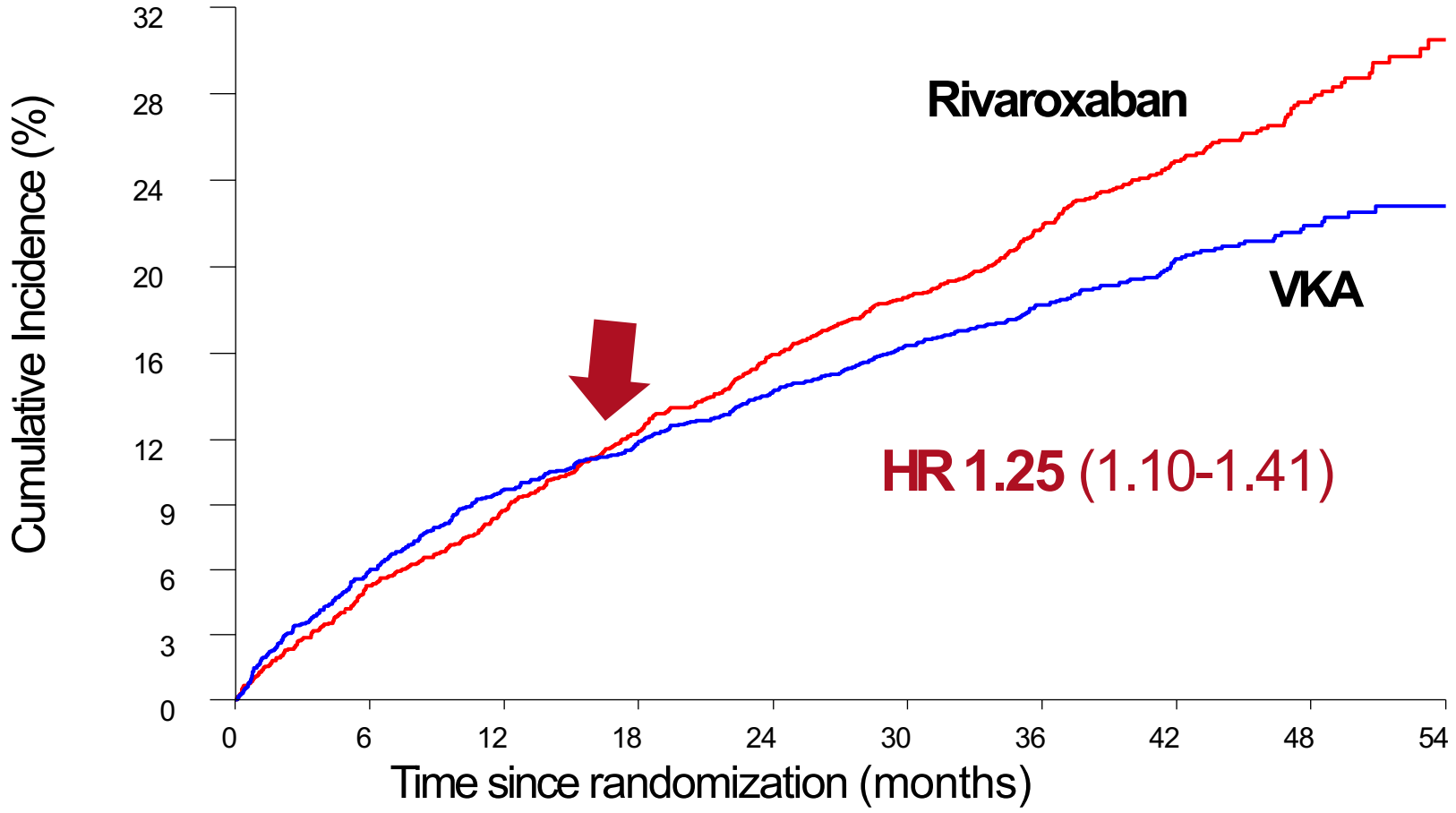
INR control by visit	
Visit	**INRs between 2-3, (%)**
Baseline	**33.2**
I year	59.0
II year	**65.3**
III year	65.1
IV year	**64.1**

Permanent discontinuation: Riva 23% (1/3rd underwent mechanical valve replacement, and therefore started VKA), VKA 6% (excellent compliance with VKA)

-Improved INRs to 65% at 2 years, and maintained thereafter

Results: Stroke, SE, MI or death (vascular or unknown)

4531 patients, 97% follow-up, average of 3.1 years



Proportional hazards assumption NOT met

Outcomes % per year (n)	**Rivaroxaban** (n=2275)	**VKA** (n=2256)	**HR** (95% CI)	**RMST difference,** days (95% CI)	**p value (RMST)**
Primary composite	**8.2** (560)	**6.5** (446)	**1.25** (1.10-1.41)	**-76** (-121, -31)	0.001
Death	**8.0** (552)	**6.4** (442)	**1.23** (1.09-1.40)	**-72** (-117, -28)	0.001
Ischemic stroke	**1.1** (74)	**0.7** (48)	**1.53** (1.06-2.20)	**-23** (-40, -6)	0.01

Few systemic embolism (16), and MI (8) events. RMST: restricted means survival time

Primarily driven by a difference in death: reduction on those on VKA. No difference in stroke or systemic embolism, but there were lsignificantly less ischemic strokes with VKA as compared to rivaroxaban

On-treatment analysis showed similar results

Difference in death unanticipated

- Not explained by difference in stroke
  VKA prevented 26 ischemic strokes vs. 110 deaths
- Difference in mortality driven entirely by **HF and sudden deaths**
- No difference in bleeding
- More frequent healthcare contact in the VKA arm for INR
- No difference in HF hospitalization, need for valve replacement or valvuloplasty, or HF meds

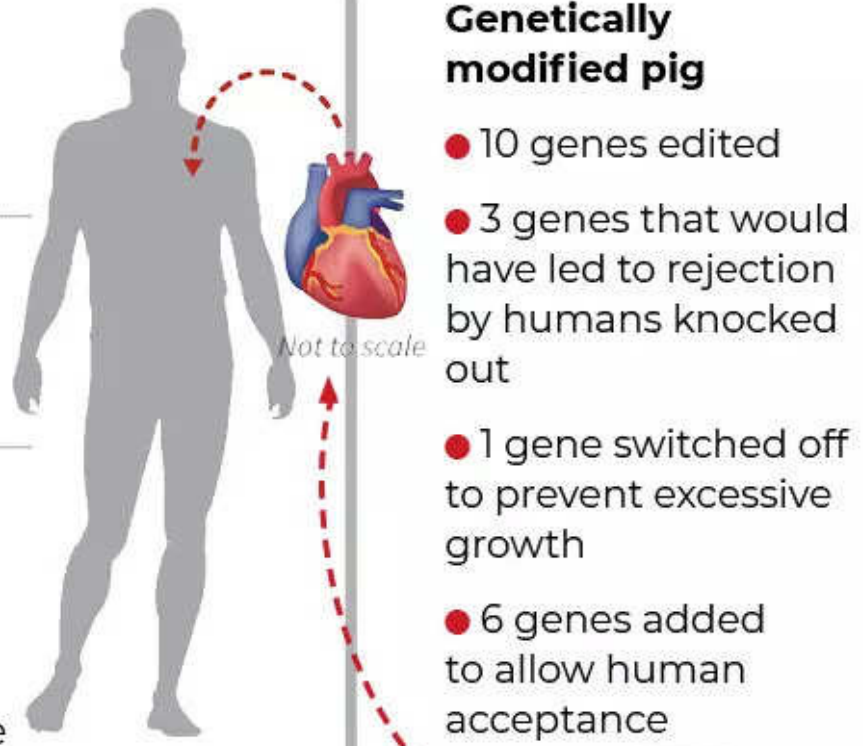
Conclusions

- In RHD-AF, compared to rivaroxaban, VKAs reduced ischemic stroke and mortality, without increasing the risk of major bleeding
- VKAs should remain the standard of care for RHD-AF
- Mortality benefit of VKA in RHD requires further study

World's first pig heart transplant into a human

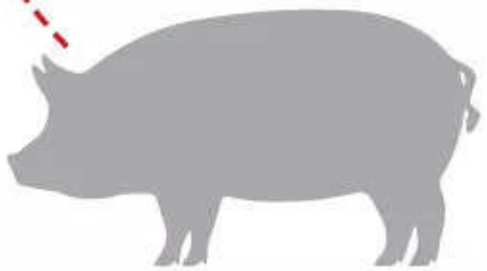
- "Historic procedure" at the University of Maryland Medical School on Jan 7, 2022

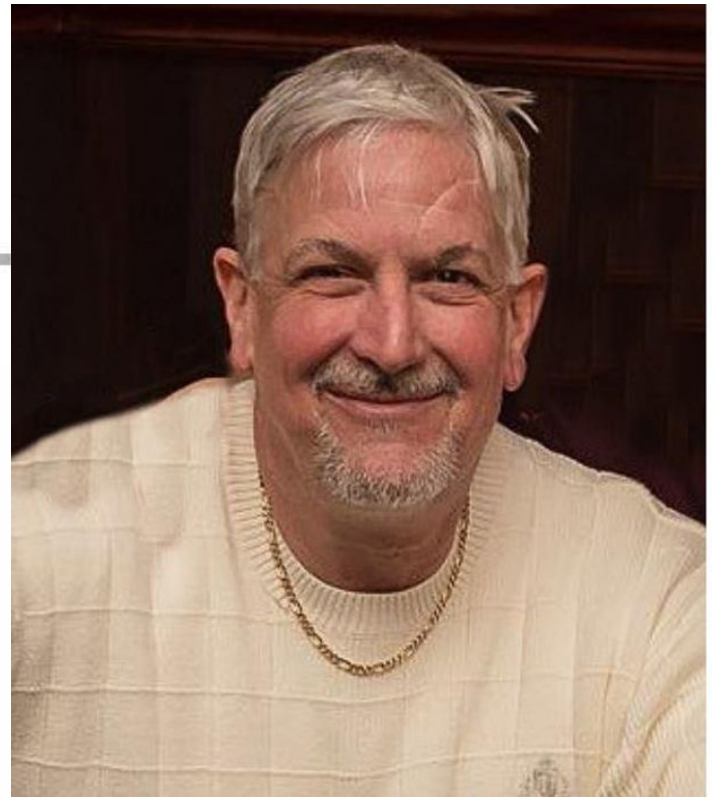
- Patient: David Bennett, 57 years old
- Ineligible for human transplant
- Use of pig heart was considered last-ditch effort to save his life
- Bennett is now recovering and being carefully monitored



Genetically modified pig

- 10 genes edited
- 3 genes that would have led to rejection by humans knocked out
- 1 gene switched off to prevent excessive growth
- 6 genes added to allow human acceptance





David Bennett – 57 years old – died March 2022

Dr. Barley Griffith – performed the surgery January 7 2022

Conclusions

Updates in Cardiology 2022:

-New treatment for obstructive HCM; what about non-obstructive HCM?
-Salt – maybe not as important as previously thought in Heart Failure pts
-AF progression: stop its progression with ablation
-New therapies in LDL lowering –
-Vitamin K antagonists still the winner for rheumatic heart disease –