Universitätsspital Basel

Cardio Update 23.05.2024

Imaging Pearls

PD Dr. Philip Haaf

CARDIOVASCULAR FLASHLIGHT

https://doi.org/10.1093/eurheartj/ehad208

Online publish-ahead-of-print 8 April 2023

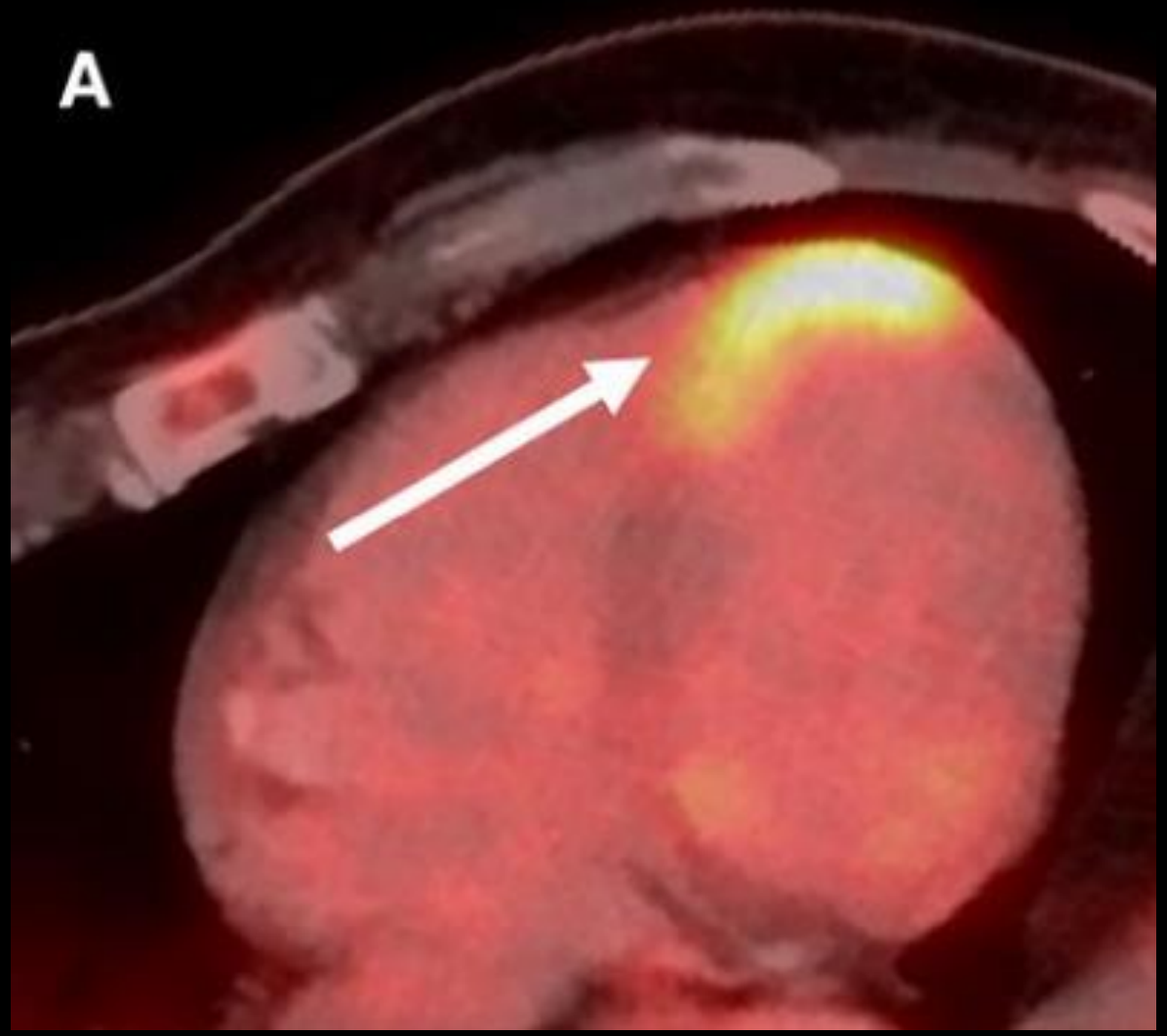
Martin T. Freitag [1]*, Philip Haaf [2], and Michael J. Zellweger[2]

Eur Heart J 2023

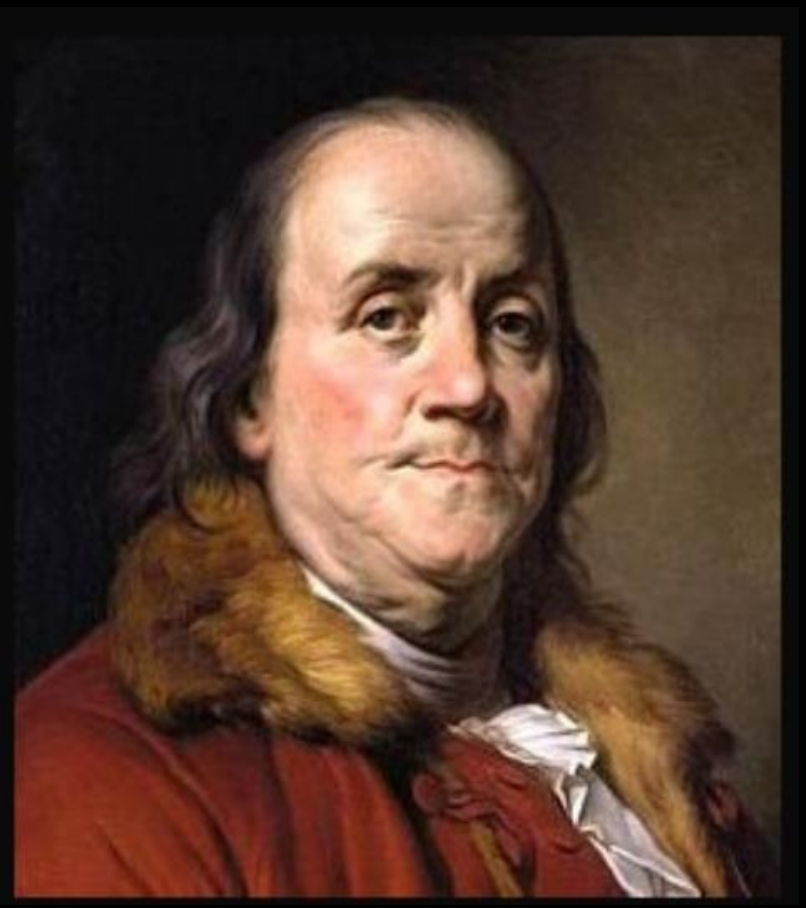
Whole body ^{18}F-FDG-PET/CT

It depends on…

the preparation?



Vorbereitung ist alles…



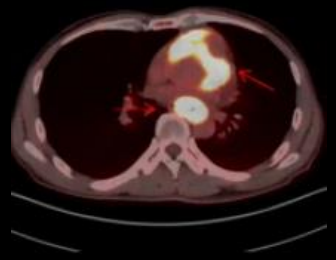
By failing to prepare, you are preparing to fail.

Benjamin Franklin

Radioactive Glucose Tracer (FDG)

“Oncologic” preparation

4-6 h fasting

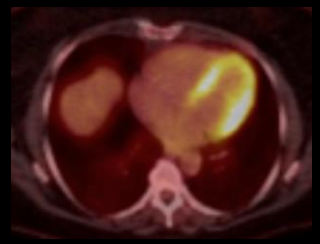


Multiple cardiac metastases

“Cardiac sarcoidosis”

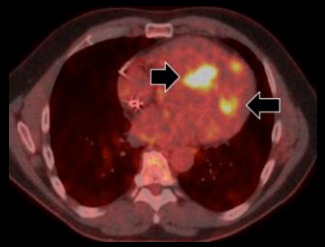
72 h fasting, Heparin iv

(no carbohydrates)



Inadequate fasting

Non-diagnostic scan



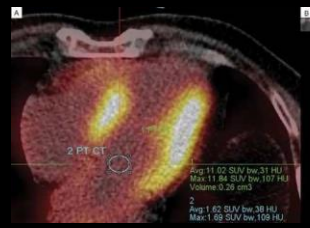
72 h fasting

diagnostic scan

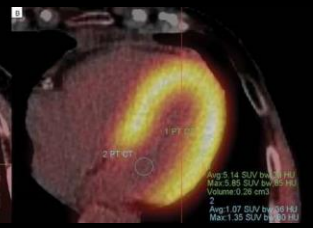
“Viability preparation”

Glucose loading

(high-glucose drink/infusion)



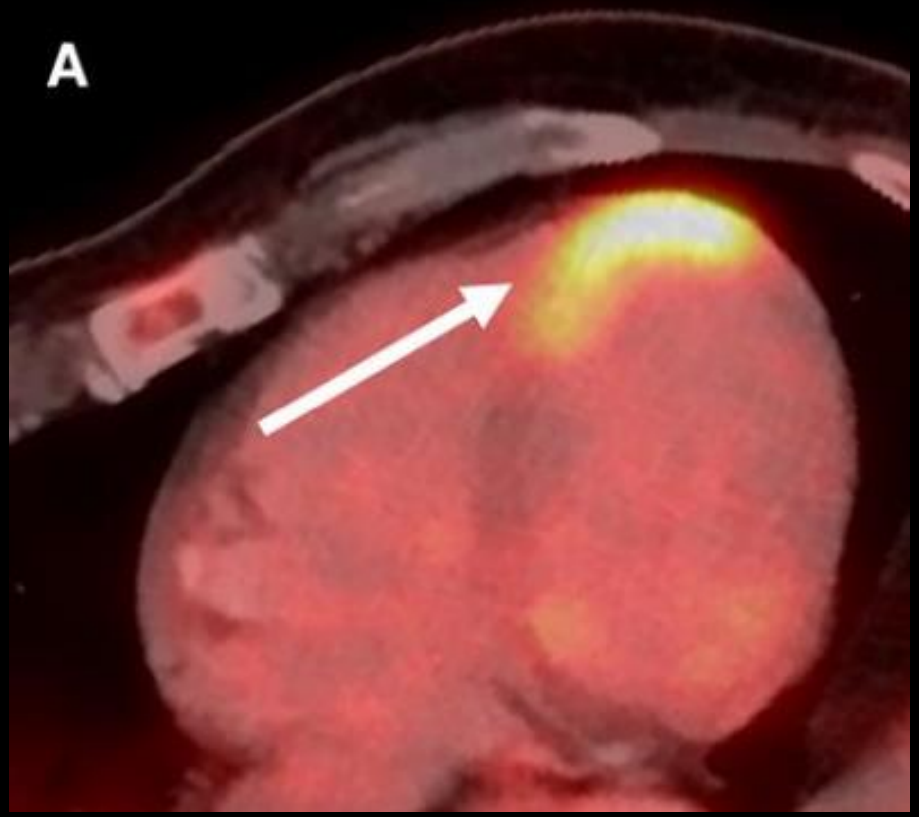
Non-viable



Viable

Oncologic whole body ^{18}F-FDG-PET/CT (standard staging method)

patient fastened as per protocol for 6 h with blood glucose 5.3 mmol/L prior to injection of ^{18}F-FDG



A 69-year-old man

Whole body ^{18}F-FDGPET/CT for staging of recurrent seminoma.

No prior known CAD but reports stable angina pectoris.

Strong ^{18}F-FDG-uptake in the apex of the heart.

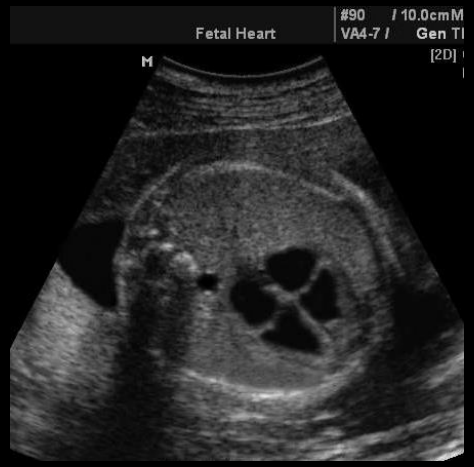
Differential Diagnosis?

Management?

Cardiometabolism

Fetal heart

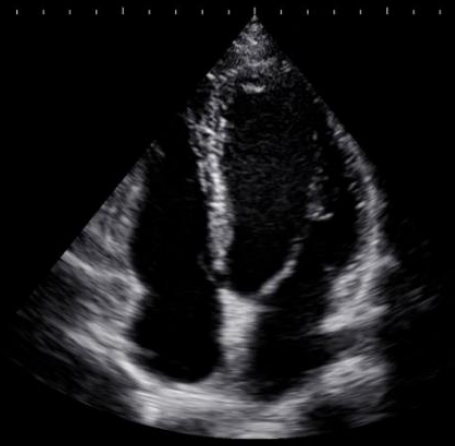
$O_2\downarrow$



- Primarily relies on glucose rather than free fatty acids for energy metabolism
- This metabolic pathway is well-suited for the fetal heart, which may experience fluctuations in oxygen availability during development.

Adult “mature” heart

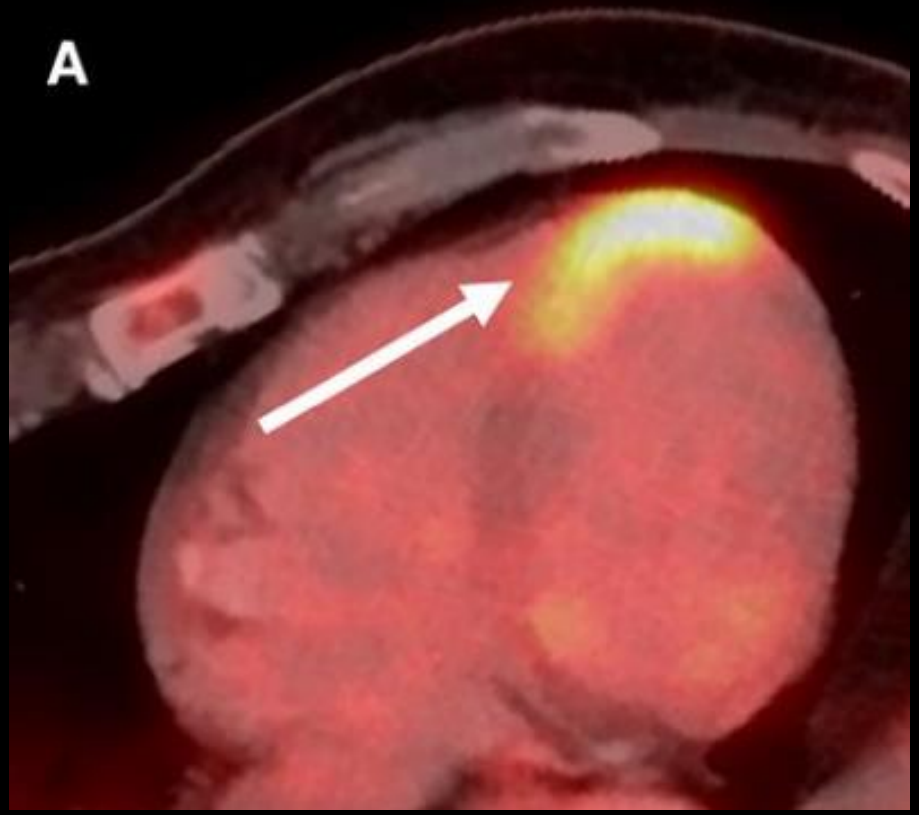
$O_2\uparrow$



- Primarily relies on fatty acids rather than glucose for energy metabolism
- This preference for fatty acid oxidation is a key characteristic of mature cardiac metabolism (Krebs cycle to generate ATP).

Oncologic whole body ^{18}F-FDG-PET/CT

patient fastened as per protocol for 6 h with blood glucose 5.3 mmol/L prior to injection of ^{18}F-FDG



A 69-year-old man

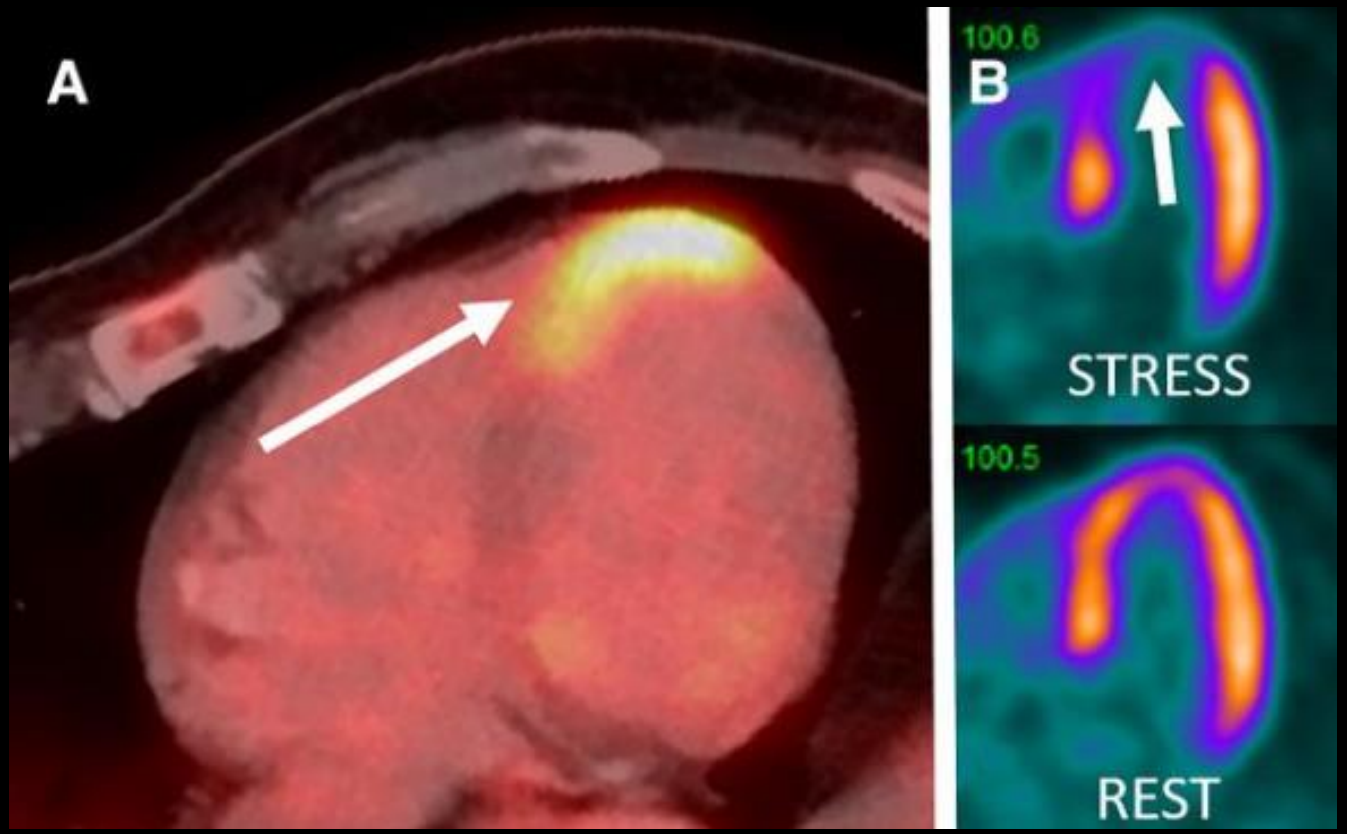
Whole body ^{18}F-FDGPET/CT for staging of recurrent seminoma.

No prior known CAD but reports stable angina pectoris.

The CT images showed calcified coronary arteries. He was referred for an ^{82}Rb-PET/CT scan

^{18}F-FDG-PET/CT

^{82}Rb-PET/CT



Severe ischemia in the territory of LAD territory matching the region of the ^{18}F-FDG-uptake

Normal uptake at rest.

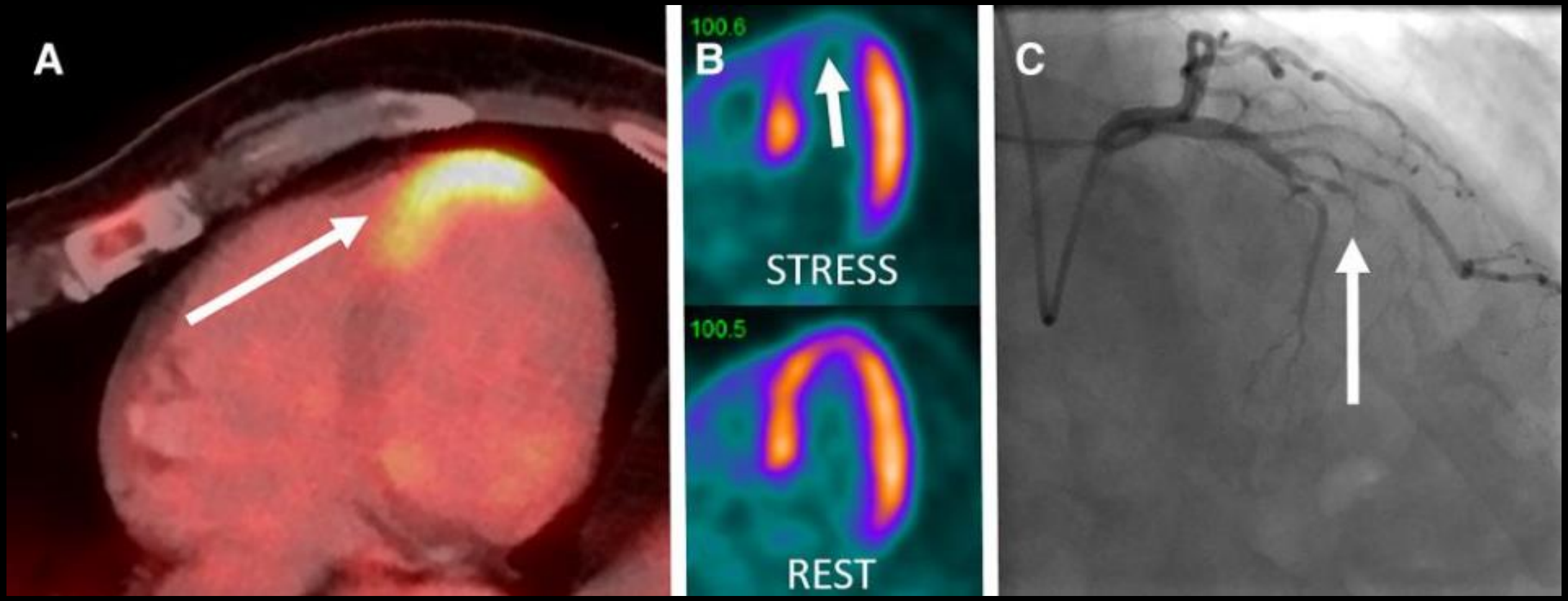
No myocardial infarction.

The patient was referred for invasive angiography.

^{18}F-FDG-PET/CT

^{82}Rb-PET/CT

Invasive angiography

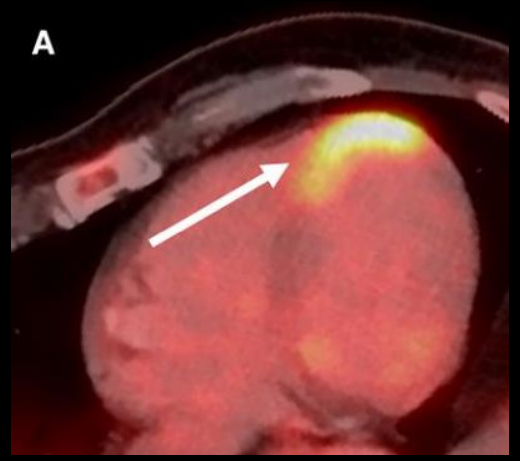


occlusion of the apical LAD with retrograde perfusion by RCA → successful revascularization.

“Lipid shift” of the heart toward anaerobic metabolism

- from fatty acid oxygenation (lipid metabolism) to glycolysis (glucose metabolism) -

Adult heart “under pressure” (such as ischaemia, i.e. hypoxia)



Fatty oxygenation is impaired (due to hypoxia)

→ metabolic adaptations (de-differentiation of cardiomyocytes may occur)

→ heart increases glucose utilization to maintain ATP production, to preserve cardiac function and to improve cell survival

→ Glycolysis is anaerobic (no oxygen required) but is less efficient in generating ATP → hypokinesia/akinesia

CARDIOVASCULAR FLASHLIGHT

https://doi.org/10.1093/eurheartj/ehad208
Online publish-ahead-of-print 8 April 2023

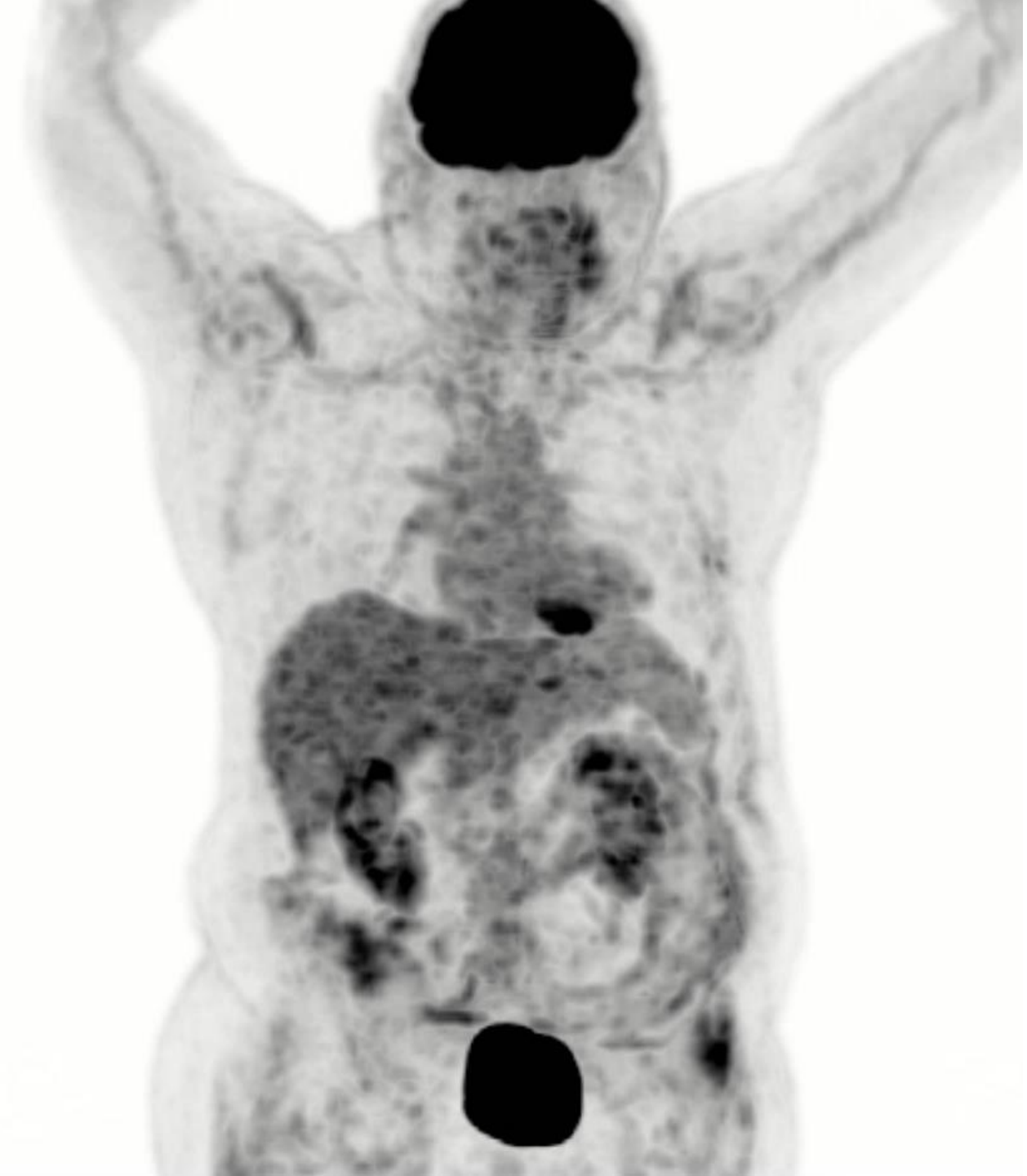
Severe myocardial ischemia detected in routine oncological positron emission tomography/computed tomography with ^{18}F-fluorodeoxyglucose

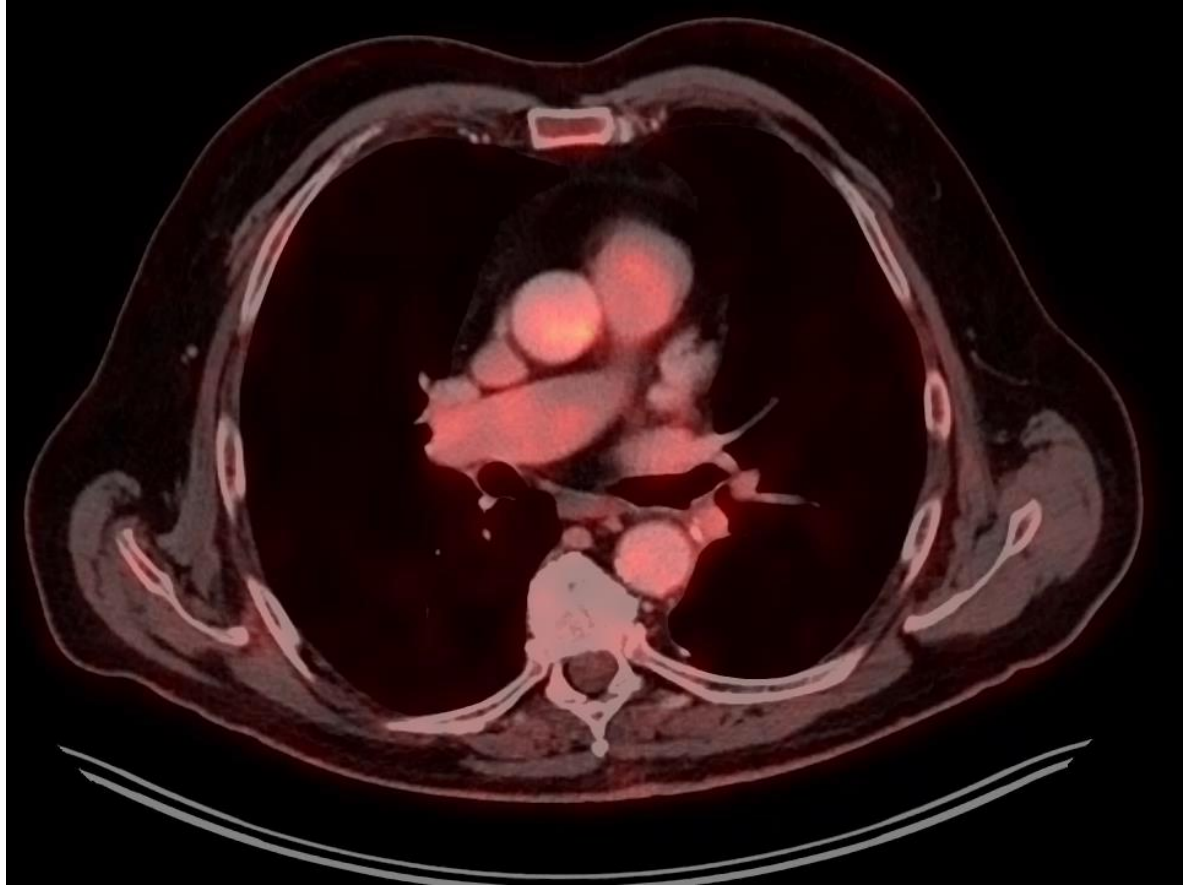
Martin T. Freitag [1]*, Philip Haaf [2], and Michael J. Zellweger[2]

Eur Heart J 2023

Severe ischemia and hibernating myocardium may be detected in routine ^{18}F-FDG-PET/CT scans without stress testing under specific circumstances ('lipid-shift' + coronary pattern of increased ^{18}F-FDG-uptake).

Might be interesting for Cardio-Oncology…





- Severe 3-vessel coronary artery calcification
- Infero/septobasal hypermetabolism

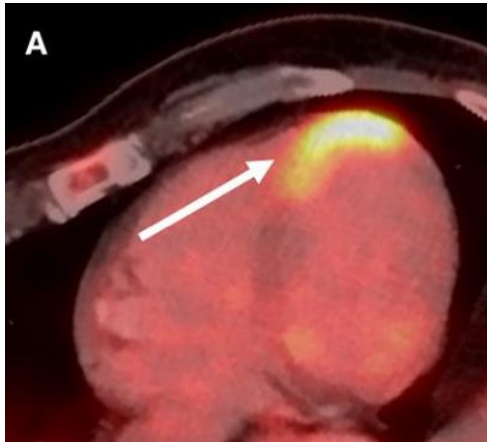
Proximal RCA stenosis!?

Journal of
Clinical Medicine

Article

Myocardial ^{18}F-FDG Uptake Pattern for Cardiovascular Risk Stratification in Patients Undergoing Oncologic PET/CT

high-risk ^{18}F-FDG-PET/CT myocardial uptake pattern:

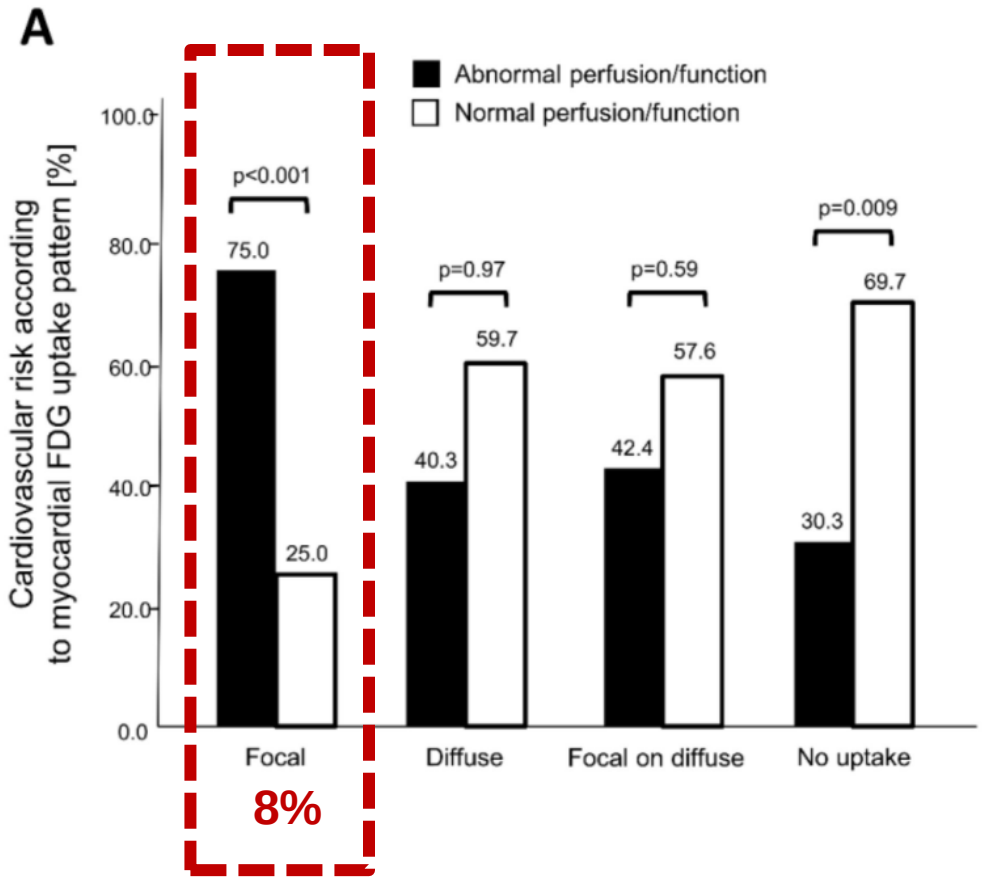


focal ^{18}F-FDG uptake →

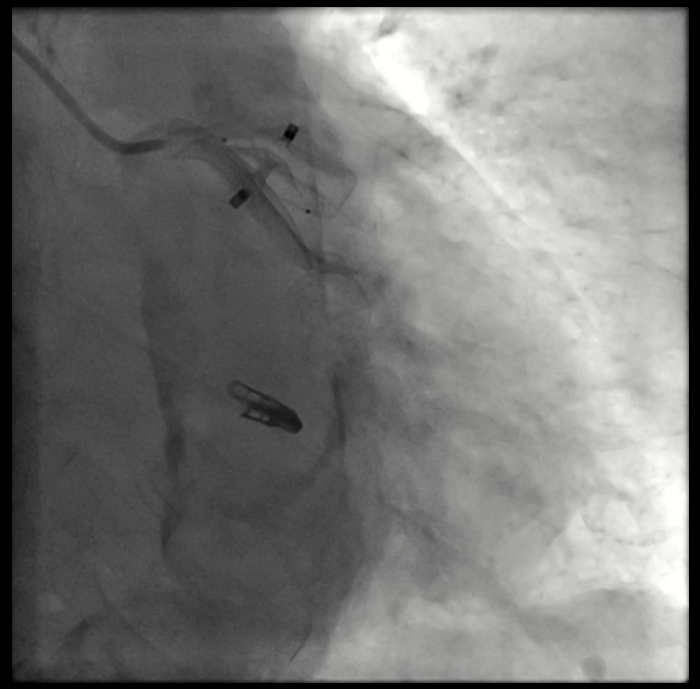
75% abnormal perfusion/function (OR 5.3, p=0.003)

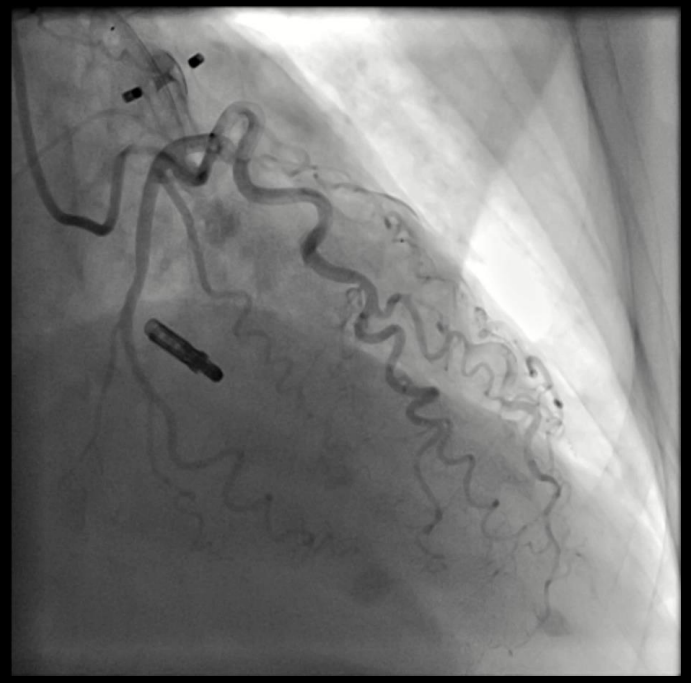
332 patients with ^{18}F-FDG PET

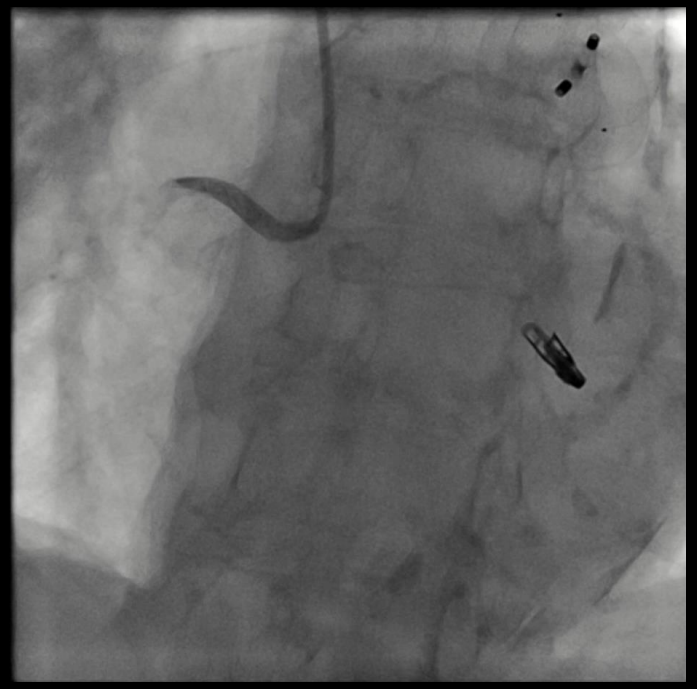
and SPECT-MPI within 6 months

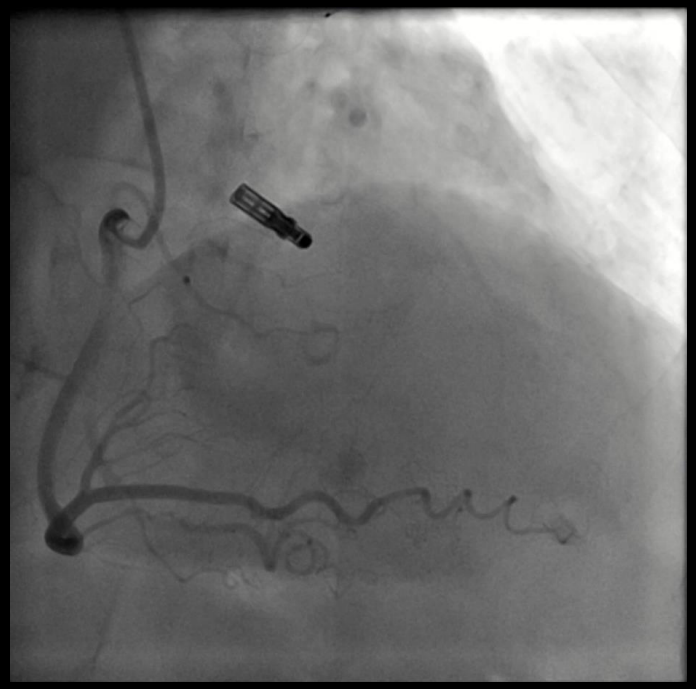


Haider et al. J Clin Med 2020

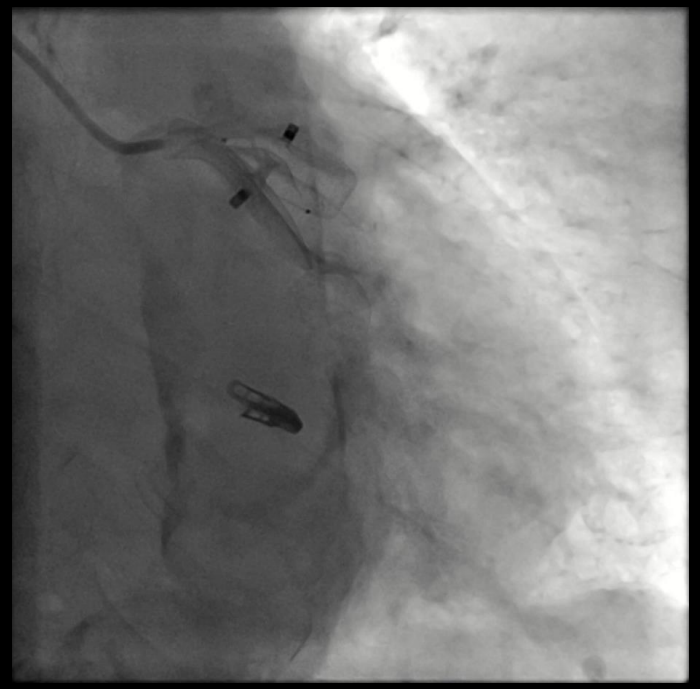


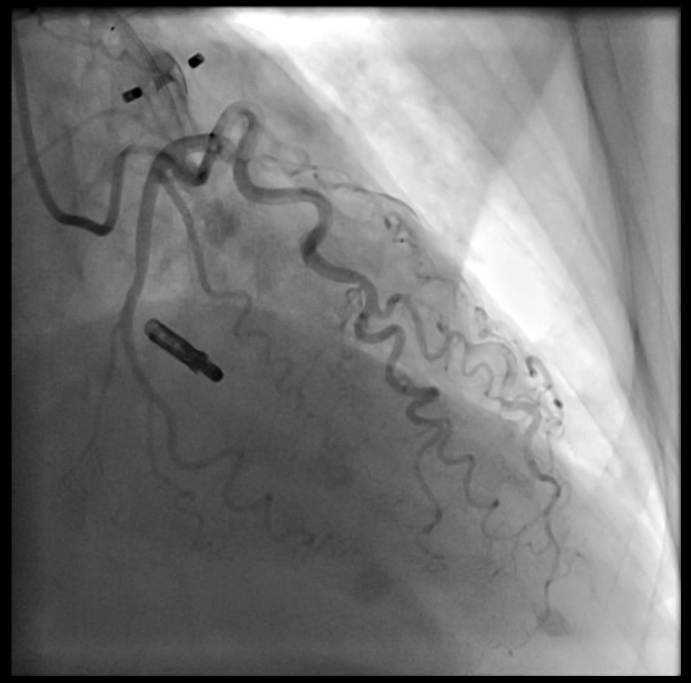


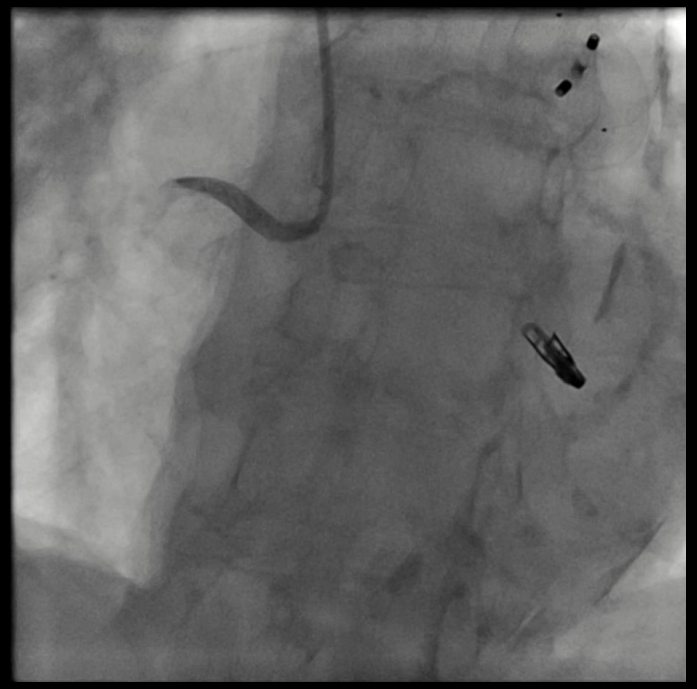


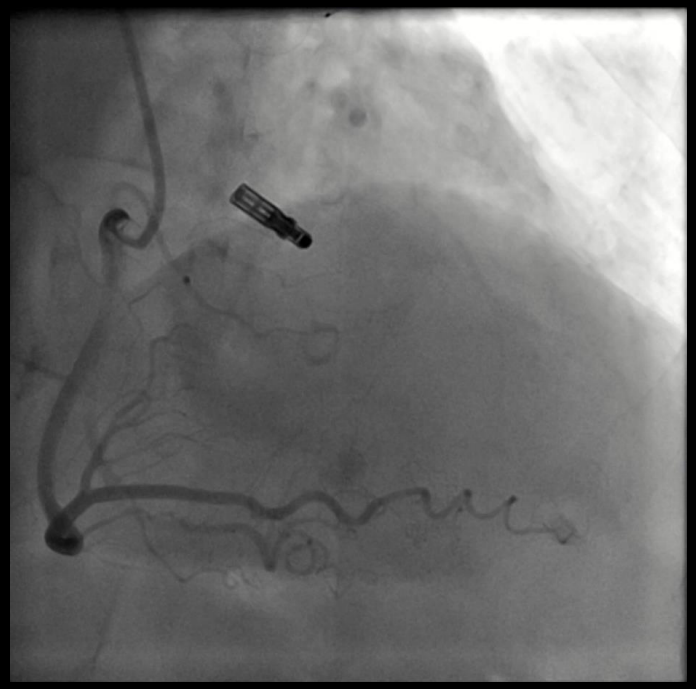


Management?







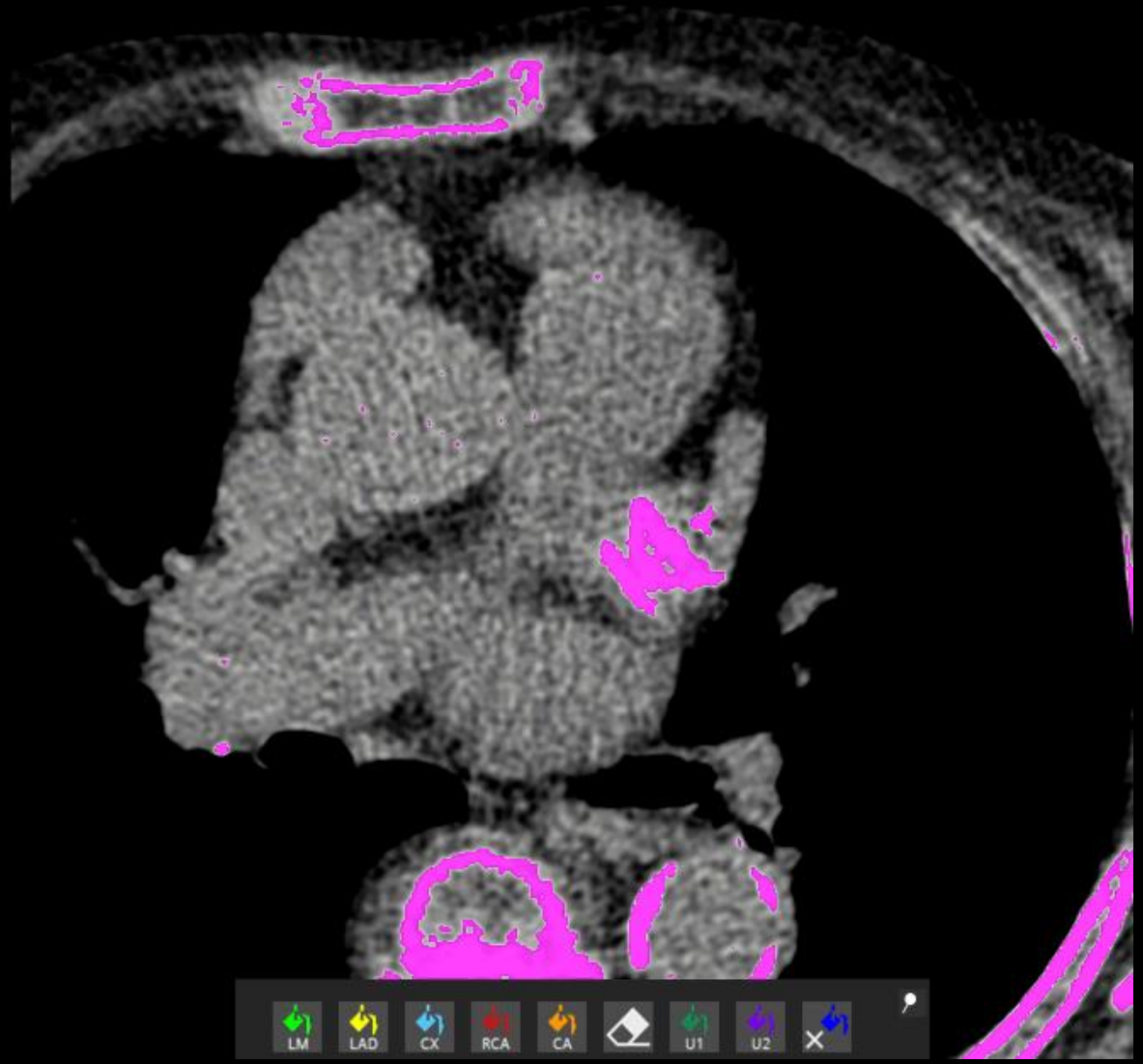


Koronartabelle:

Gefäss		Norm	< 50%	50 - 75%	75 - 95%	95 - 99%	100 %
ACD	Proximal	x					
	Mitte	x					
	Distal	x					
	RIVPO	x					
	Post. lat. d	x					
	RV-Ast	x					
linker Hauptstamm		x					
RIVA	Proximal	x					
	Mitte	x					
	Apikal	x					
	Diagonal 1	x					
	Diagonal 2						
RCX	Proximal	x					
	Marginalast 1	x					
	Marginalast 2	x					
	Distal	x					
Intermediärast							
LIMA							
RIMA							

Ausschluss einer stenosierenden koronaren Herzkrankheit

Same patient

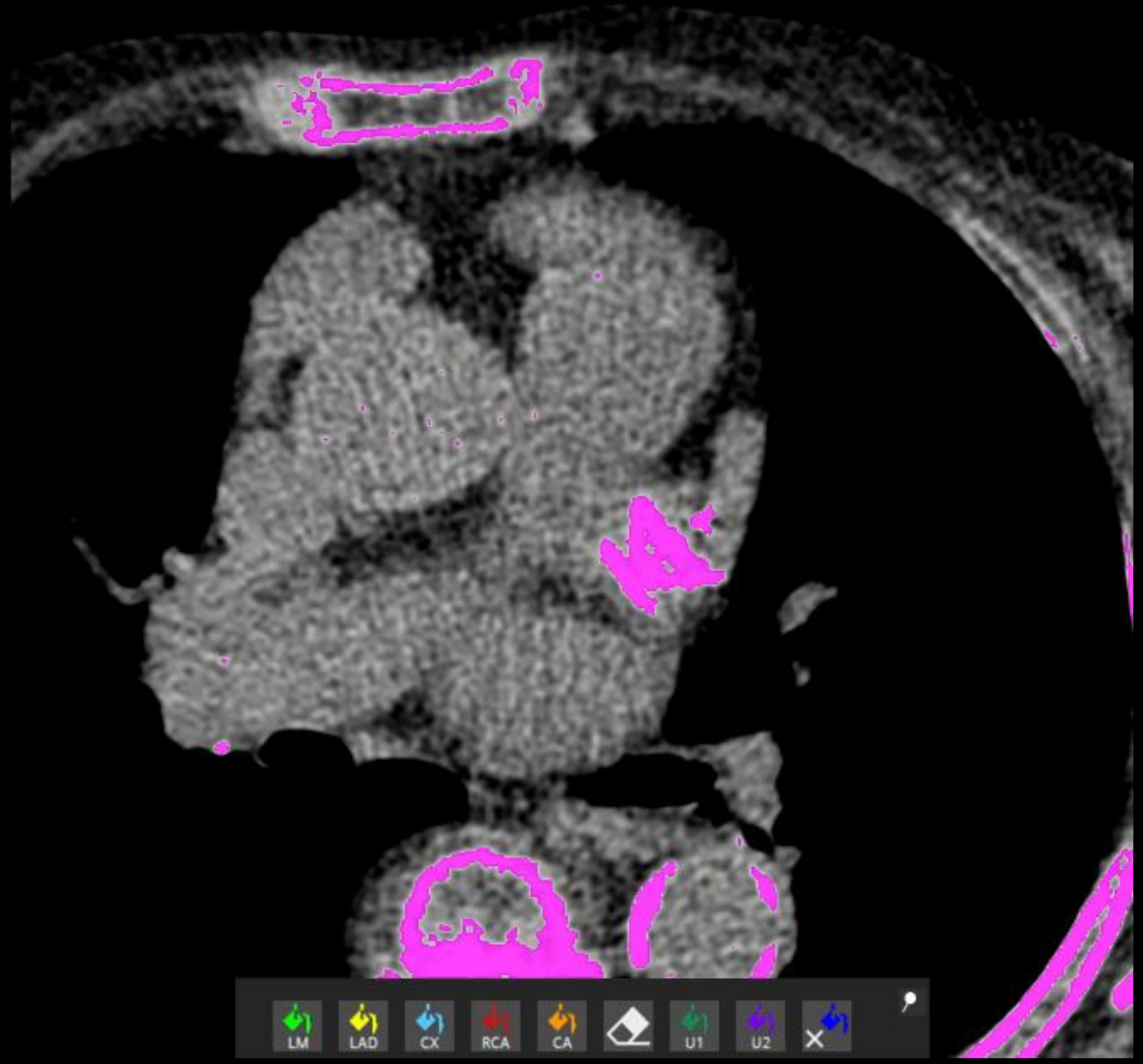


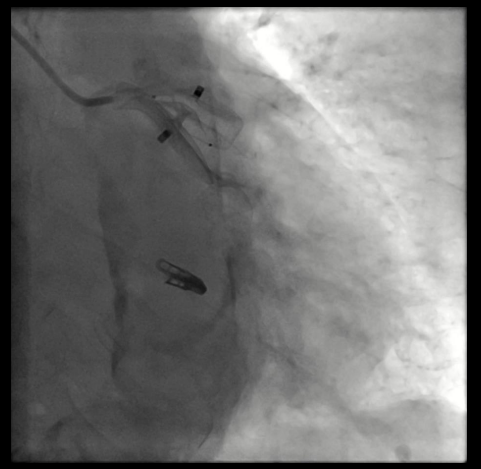
Artery	Score
LM	336.5
LAD	382.2
CX	528.1
RCA	226.6
Ca	0.0
Total	1473.4

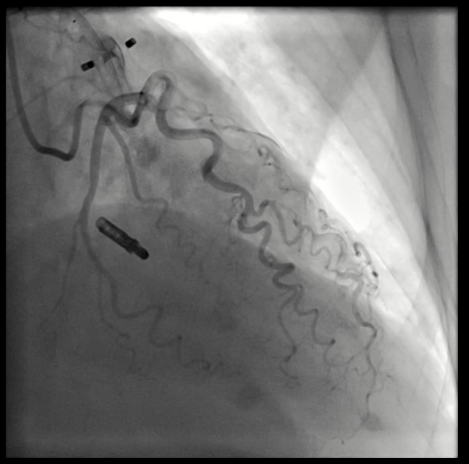
Calcium-Score: 1473
>90th Percentile

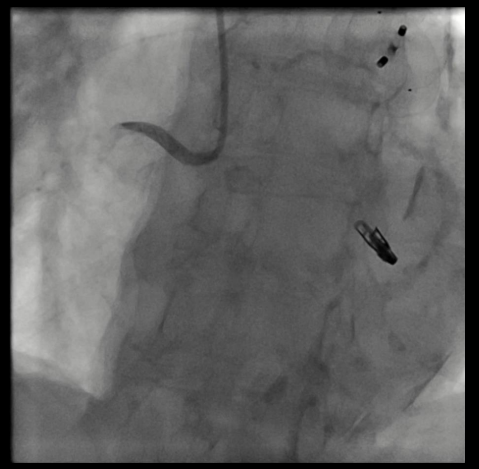
Avoidable??

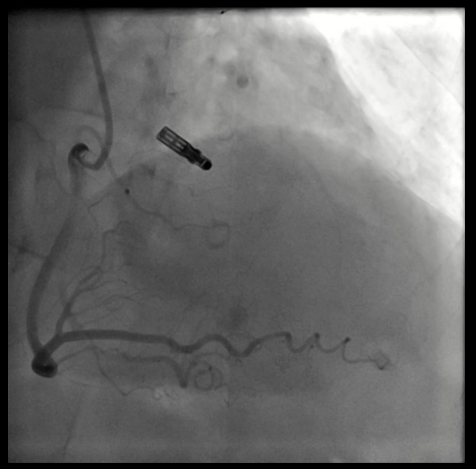
Same patient at same day



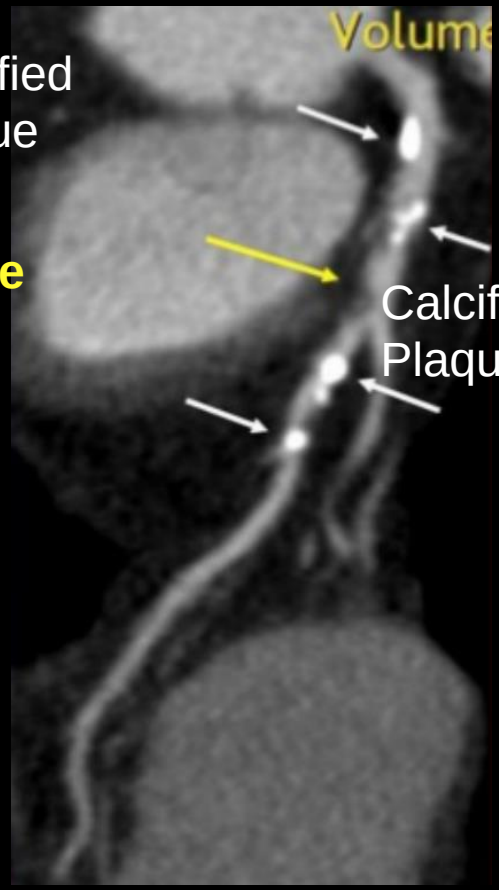




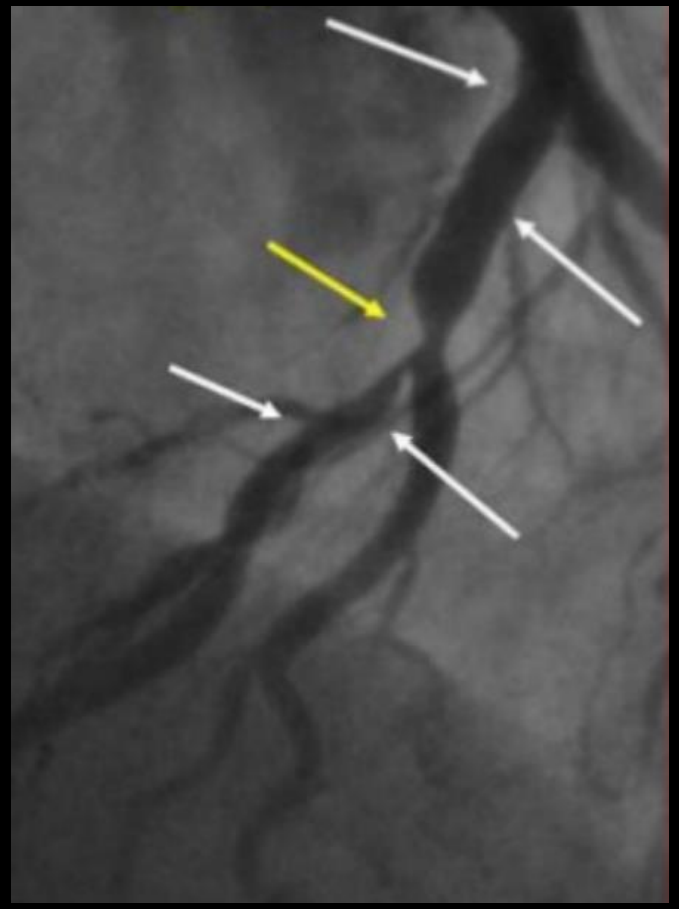




Calcifications → Blooming Artifacts in CTCA



Early detection of CAD
Challenge CT angiography:
Overestimation of stenosis with blooming artifacts

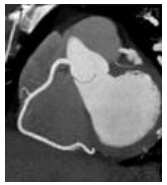


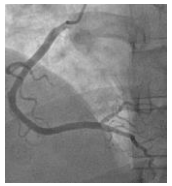
Optimal luminography
Challenge invasive angiography:
Early plaque detection

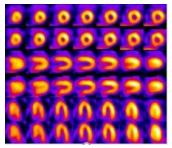
Various stages of coronary artery disease detectable by CTCA

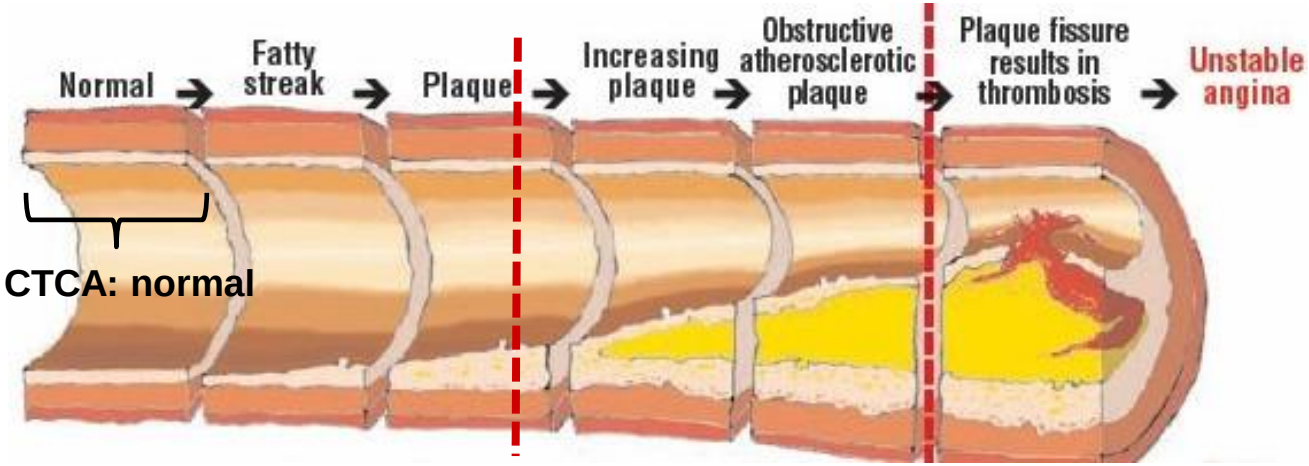
Rule Out of CAD NPV: 99%	Detection of early non-obstructive disease	Quantification of plaque burden (+Ca-Score)	Identification of high-risk plaques

Early detection of CAD







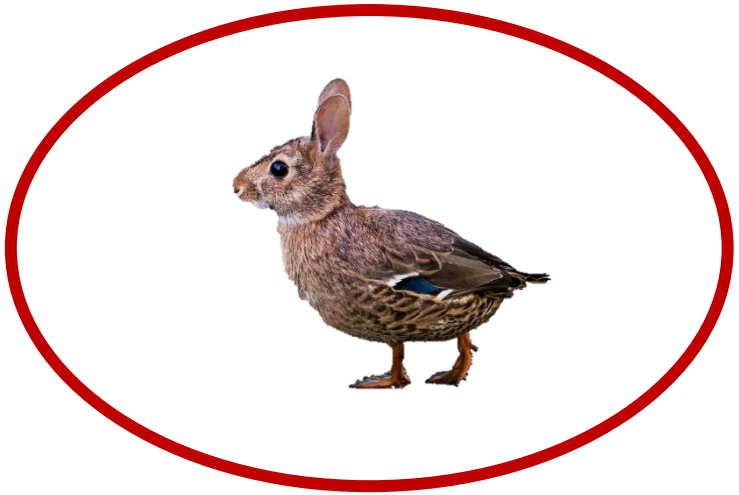


CTCA: normal

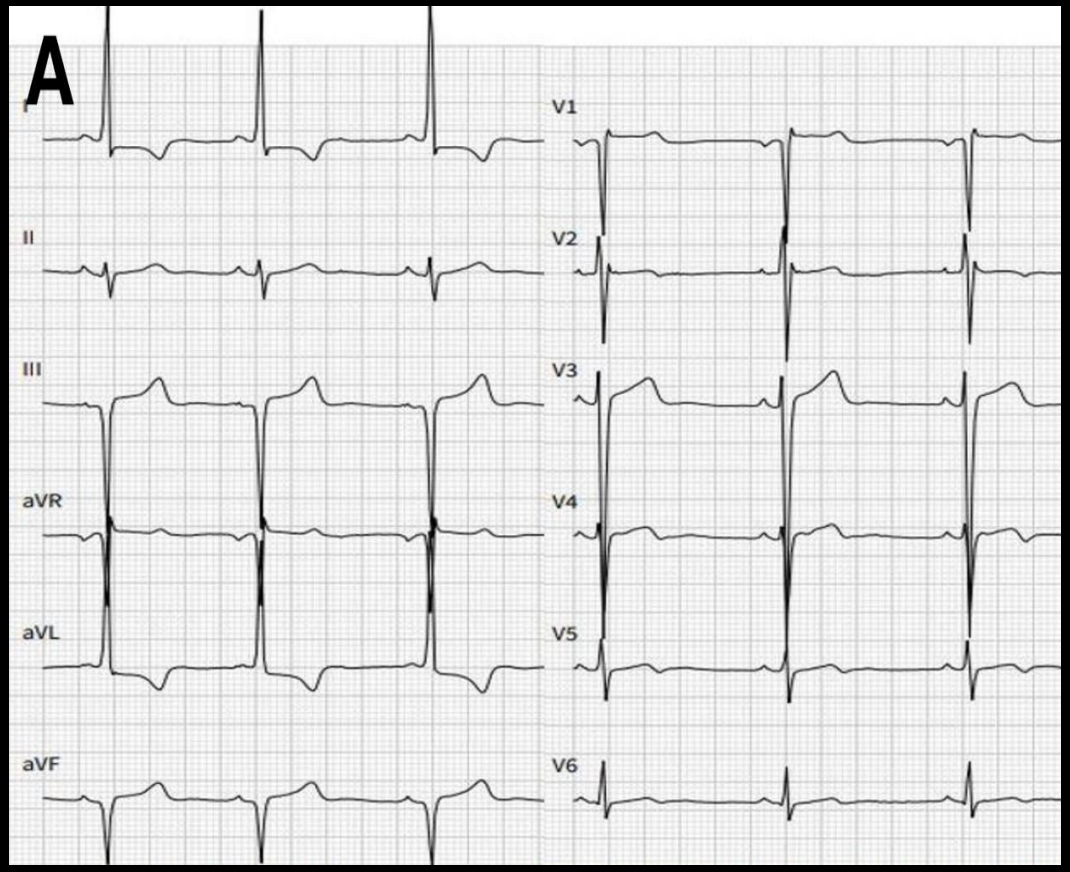
invasive angio: normal

non-invasive ischaemia imaging: normal

Shaw LJ Ann Intern Med. 2015 Hachamovitch R JACC 2003



Two in one!?



Sinus rhythm, left axis deviation, LV hypertrophy, repolarization abnormalities

- A 22-year-old asymptomatic Caucasian woman
- NT-pro-BNP of 1225 ng/L
- family history of unclear cardiomyopathy
- TTE: asymmetrical LV hypertrophy, no LVOT obstruction

CARDIOVASCULAR FLASHLIGHT

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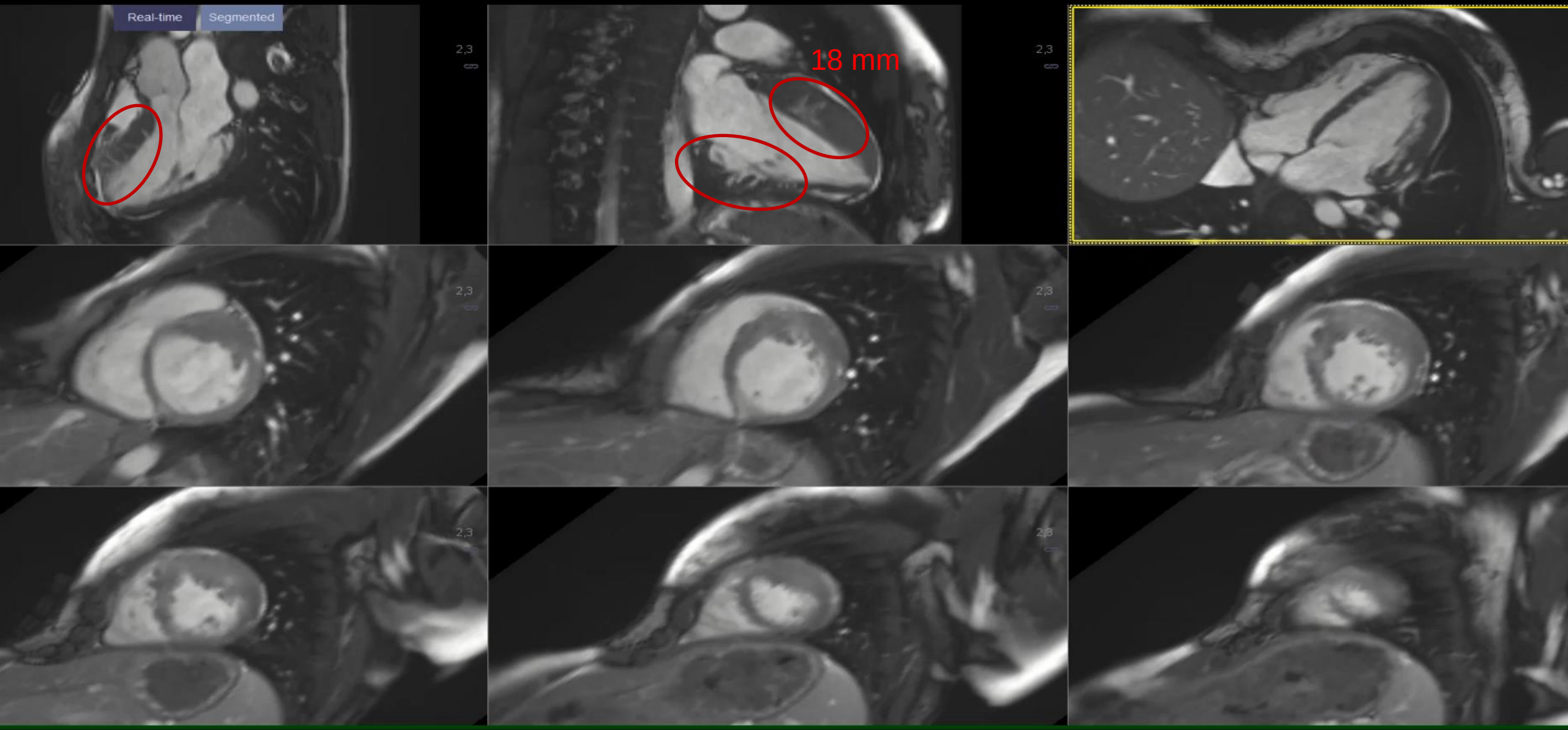
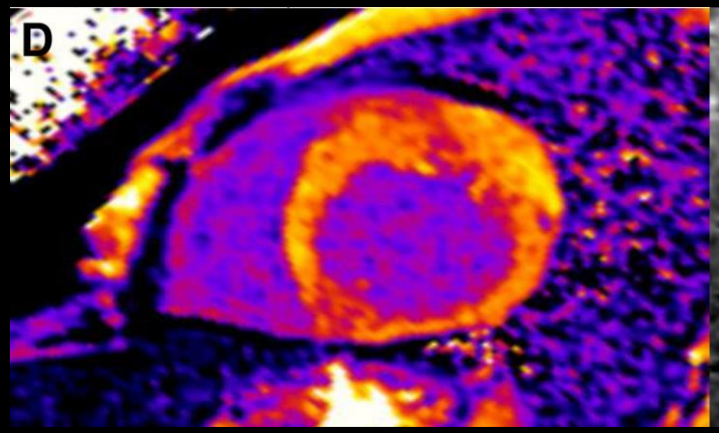
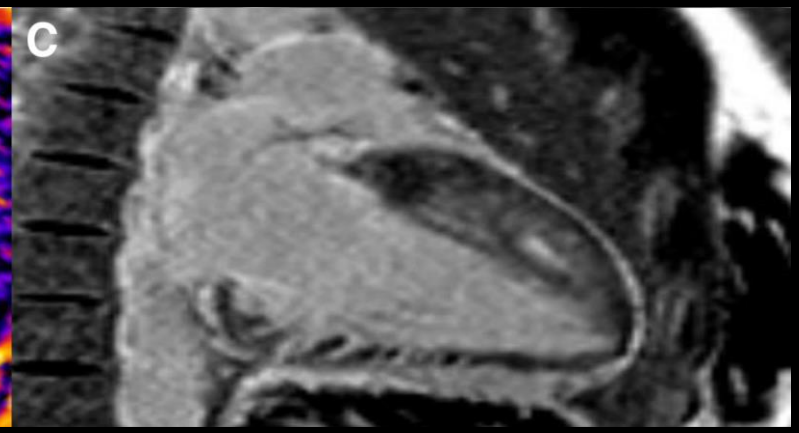


Multiple myocardial crypts/deep sinusoids
Asymmetrical LV hypertrophy
Hypertrabeculation

post-contrast T1 mapping



Late Gadolinium Enhancement



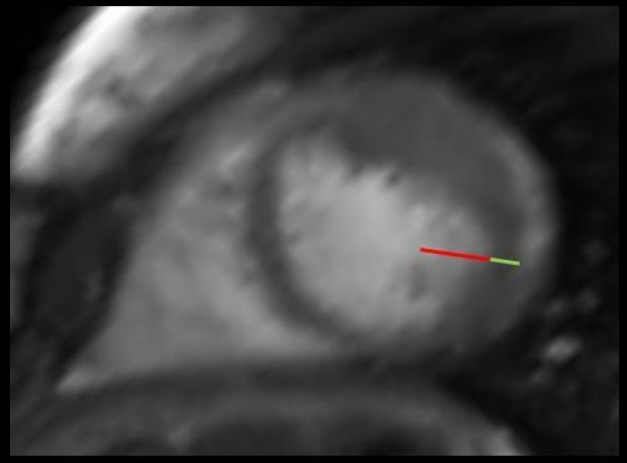
18 mm wall thickness

Focal patchy fibrosis:

- in the hypertrophied segments and
- at both right ventricular insertion points

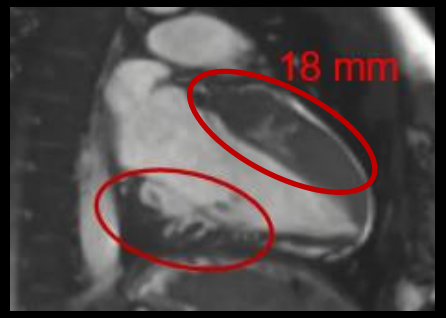
Typical HCM finding

NC:C Ratio



NC/C: 2.6

+ deep ant. + inf. sinusoids



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Hypertrophic cardiomyopathy and left ventricular non-compaction cardiomyopathy: two in one !?

Beshoy Gabra [1], Nandan S. Anavekar[2,3], and Philip Haaf [1]*

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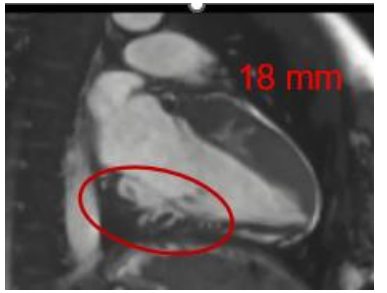
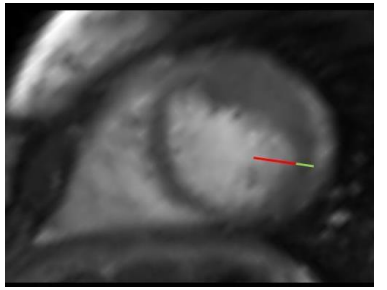
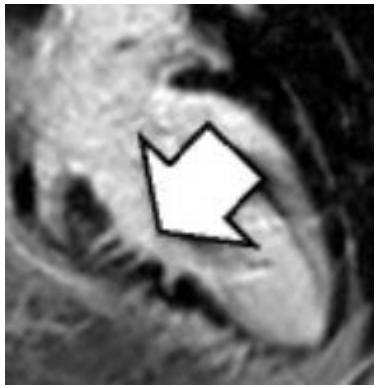
Eur Heart J 2022

Two in one?

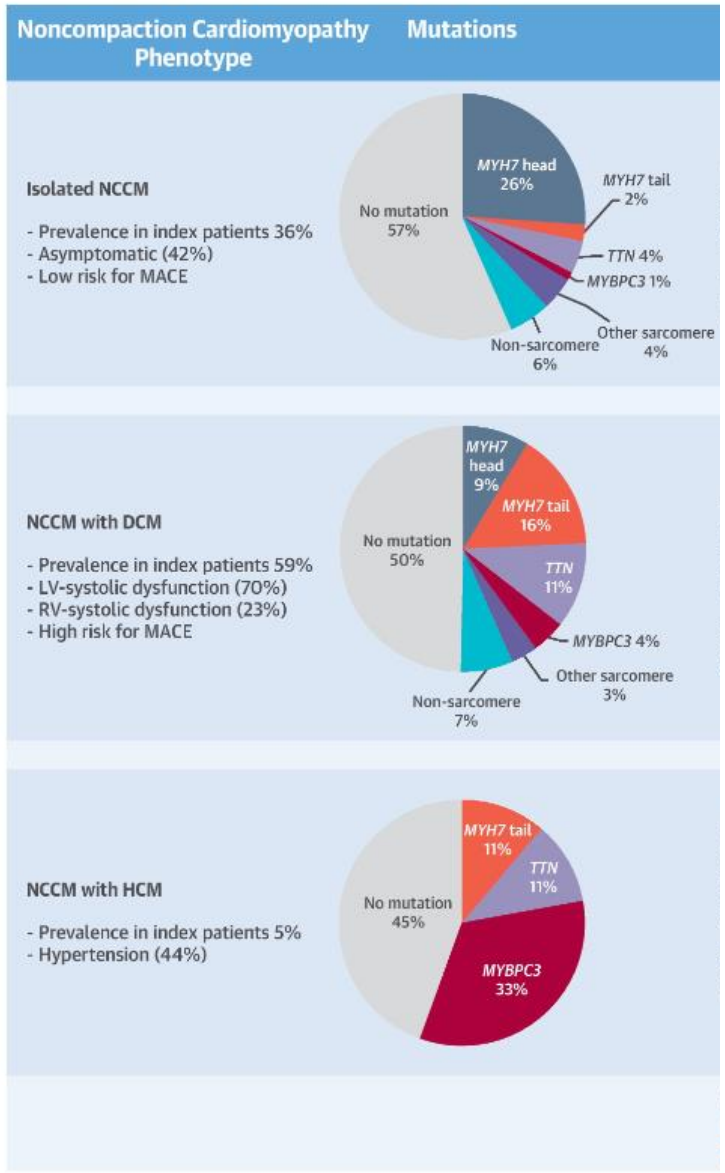
- 18 mm wall thickness + NC/C = 2.6
- Inferobasal myocardial crypts may be observed in phenotype-negative but genotype-positive patients with HCM
- Features of LVNC and HCM can co-exist in the same patient
- Sarcomere protein gene mutations can be observed in both HCM and LVNC, suggesting a possible common genetic origin of these cardiomyopathies.
- Are LVNC and HCM two different phenotypical aspects of the same underlying genetic aetiology?







CENTRAL ILLUSTRATION: Phenotypes in Familial Noncompaction Cardiomyopathy



van Waning, J.I. et al. J Am Coll Cardiol. 2019;73(13):1601-11.

Large genetic overlap of “LVNC” with HCM and DCM

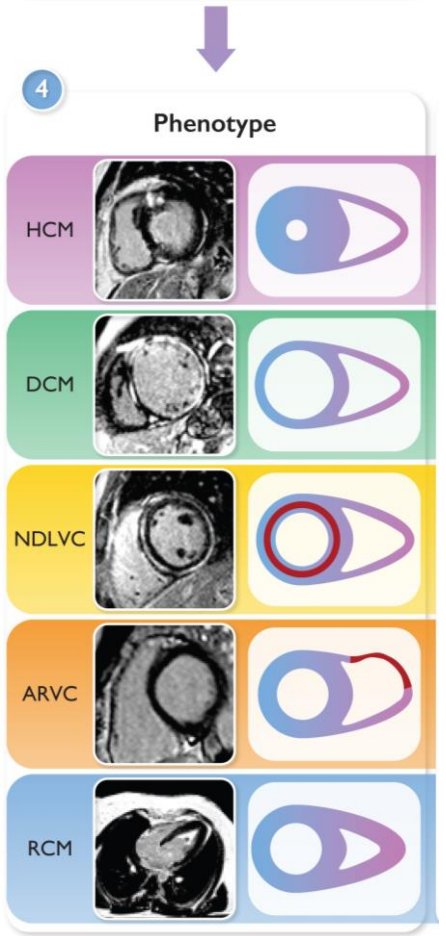
- MYH7

  Myosin heavy chain
- TTN

  Tittin
- MYBPC3

  Myosin-binding protein C

~~Left ventricular non-compaction (LVNC)~~



2023 ESC CMP Guidelines

Left Ventricular Non-Compaction Cardiomyopathy

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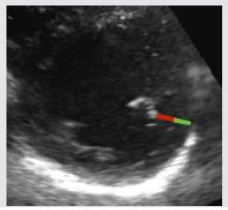
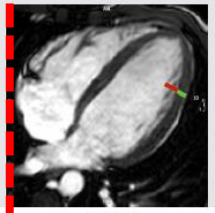
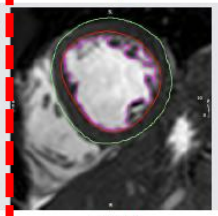
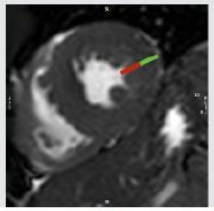
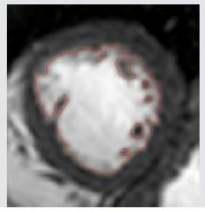
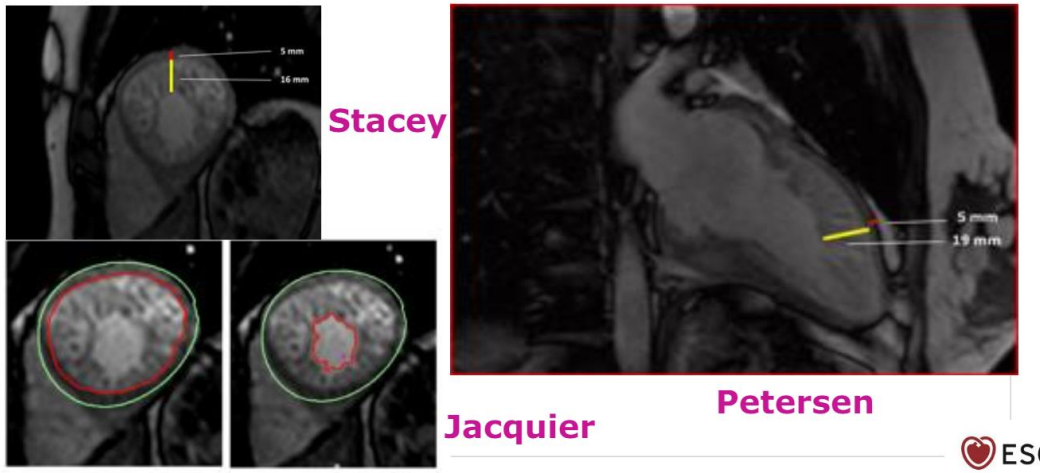
	Jenni	Petersen	Jacquier	Stacey	Captur
					
Modality	echocardiography	CMR	CMR	CMR	CMR
Criteria	- N/C in parasternal short-axis views - Colour Doppler evidence of deep perfused intertrabecular recesses - Decreased thickening and hypokinesia present within, but not limited to, the non-compacted segments	- two layered myocardium - long axis SSFP cine - measured at the most pronounced trabeculations - measurement perpendicular to compacted myocardium	- short axis SSFP fines to obtain total LV mass - same used to obtain compacted myocardial mass - difference between first and second provides the trabecular mass papilary muscles included in the myocardial mass	-apical short-axis views 16 to 24 mm from the true apical slice -region with the largest NC/C ratio	- short axis views - loss of base-to-apex FD gradient
Cardiac phase	End-systole	End-diastole	End-diastole	End-systole	End-diastole
Definition	NC/C ratio > 2	NC/C ratio > 2.3	Trabecular mass > 20%	NC/C ratio ≥ 2	FD ≥1.30
Studied subjects	34	7	16	122	30
Sensitivity and specificity	n/a	Sensitivity 86% Specificity 99%	Sensitivity 93.7% Specificity 93.7%	n/a	Sensitivity 83-100% Specificity 86-100%
Reproducibility (interobserver)	Agreement between observers 0-85%	ICC 0.82	ICC 0.71 for LVTM ICC 0.34 for LVTM%	not published	ICC 0.96

ESC CMR Pocket Guide

NC/C >2.3

Steffen Petersen



Steffen E Petersen

ESC

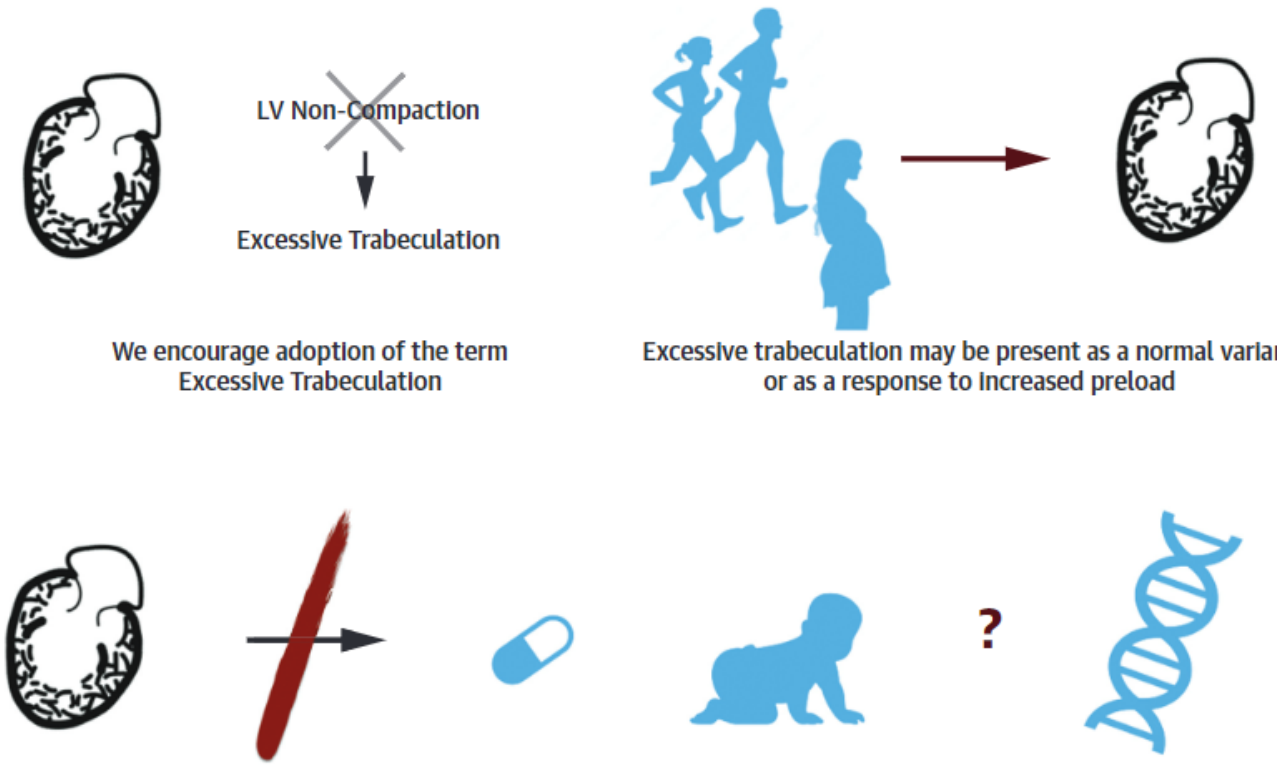
STATE-OF-THE-ART REVIEW

Excessive Trabeculation of the Left Ventricle

JACC: Cardiovascular Imaging Expert Panel Paper

Steffen E. Petersen, MD, DPhil,[a,b] Bjarke Jensen, MSc, PhD,[c] Nay Aung, MBBS, PhD,[a,b] Matthias G. Friedrich, MD,[d,e] Colin J. McMahon, MD,[f] Saidi A. Mohiddin, MBChB, MD,[a,b] Ricardo H. Pignatelli, MD,[g] Fabrizio Ricci, MD, PhD,[h] Robert H. Anderson, MD, PhD (Hon),[i] David A. Bluemke, MD, PhD[j]

CENTRAL ILLUSTRATION The Emerging Picture of (Excessive) Left Ventricular Trabeculation



Petersen SE, et al. J Am Coll Cardiol Img. 2023;16(3):408–425.

LV = left ventricular.

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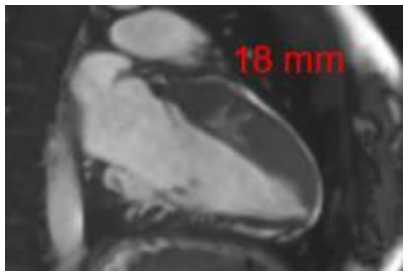

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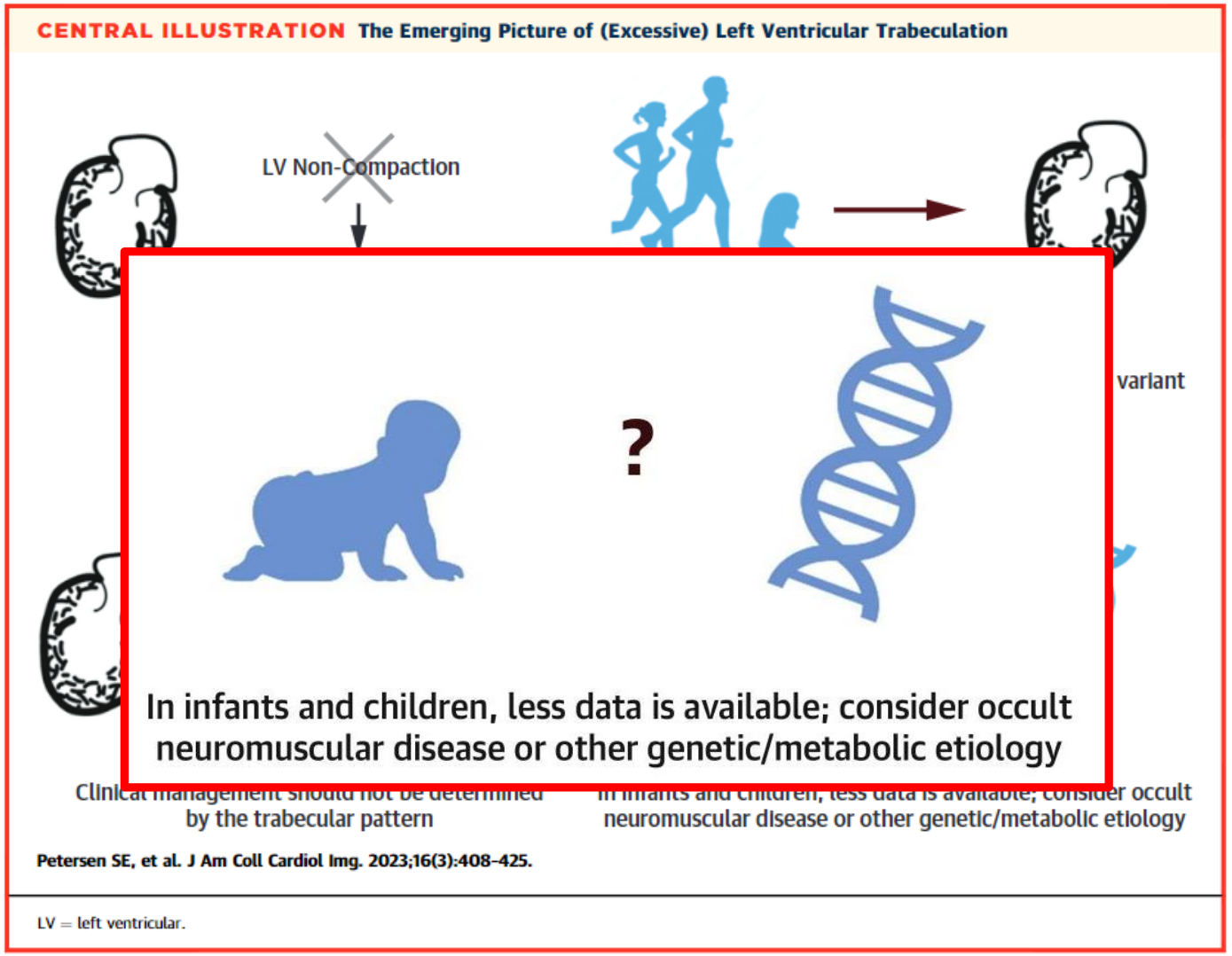

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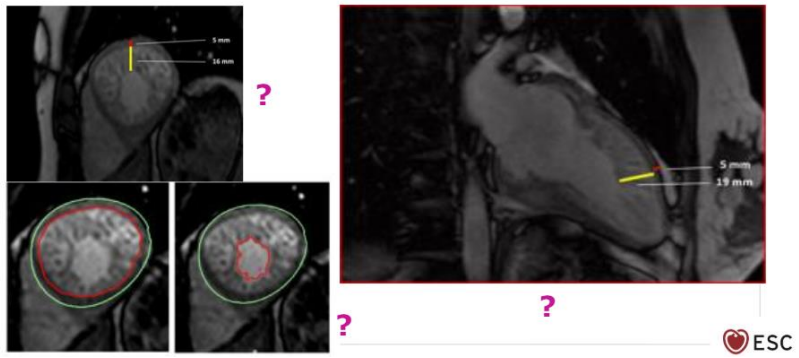
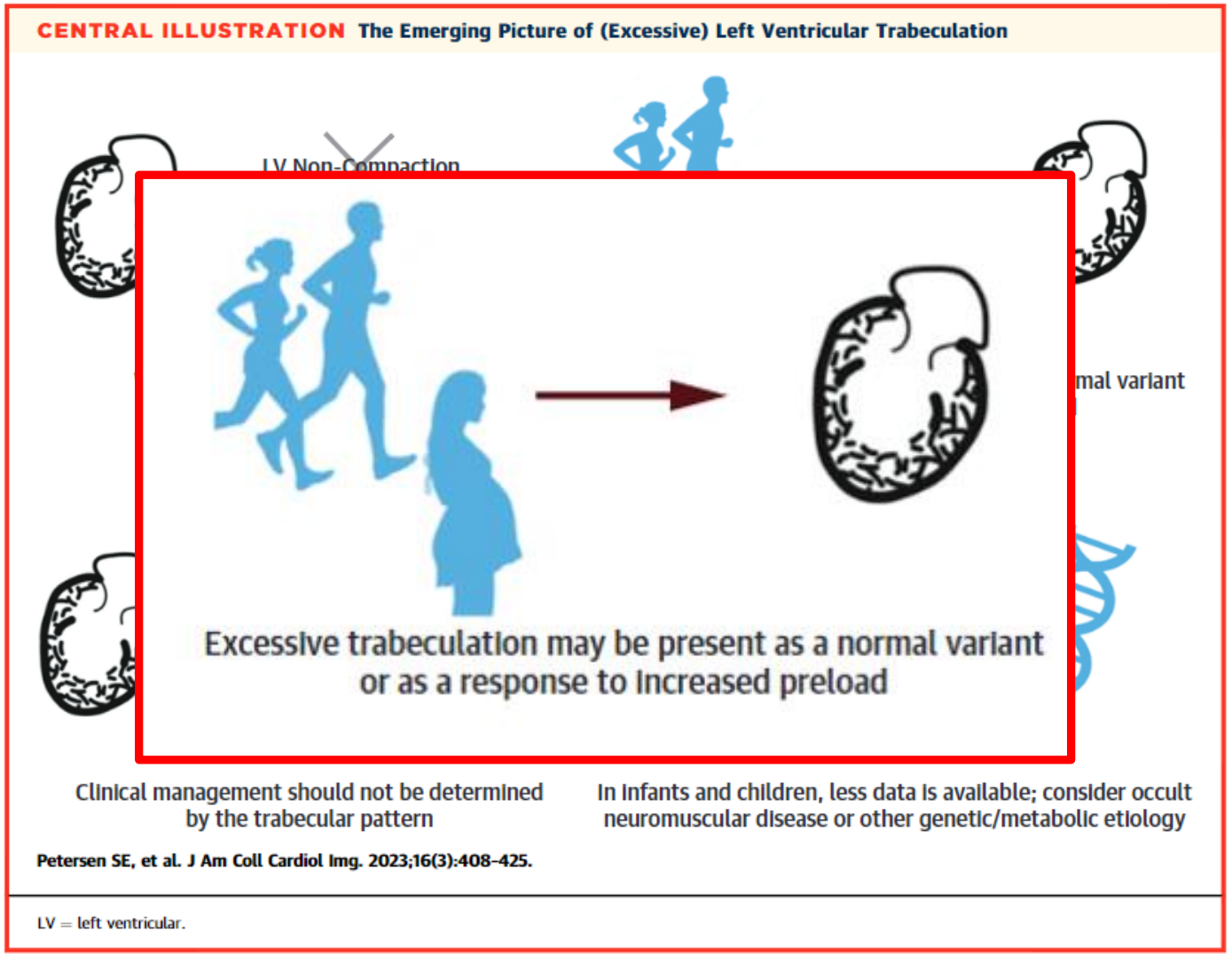


LVNC criteria fulfilled:

Very frequent MRI finding!
(up to 10% in athletes)



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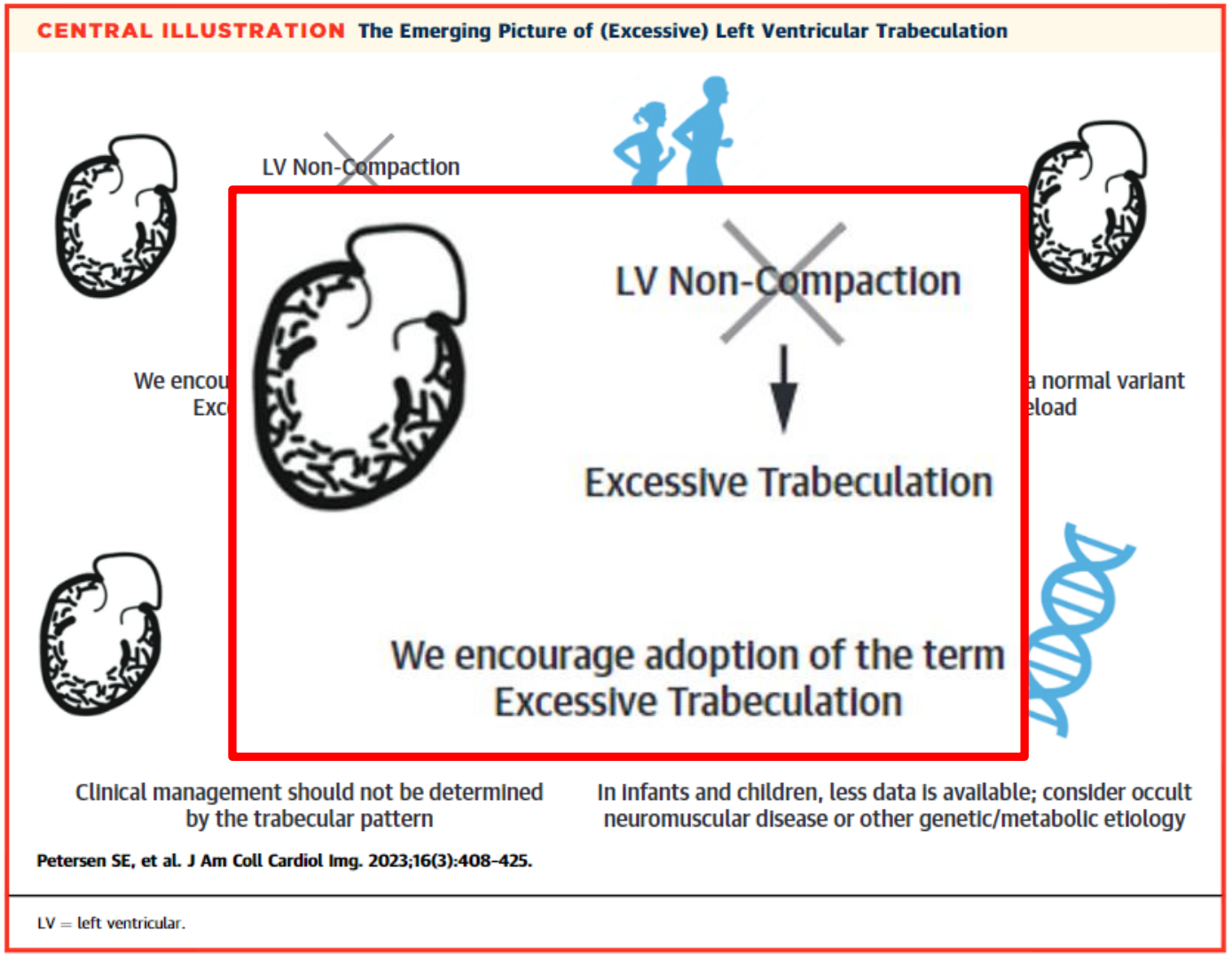
Misnomer:

Noncompaction likely results from *abnormal growth of the compact wall* and not from failed compaction of the embryonic trabeculations.



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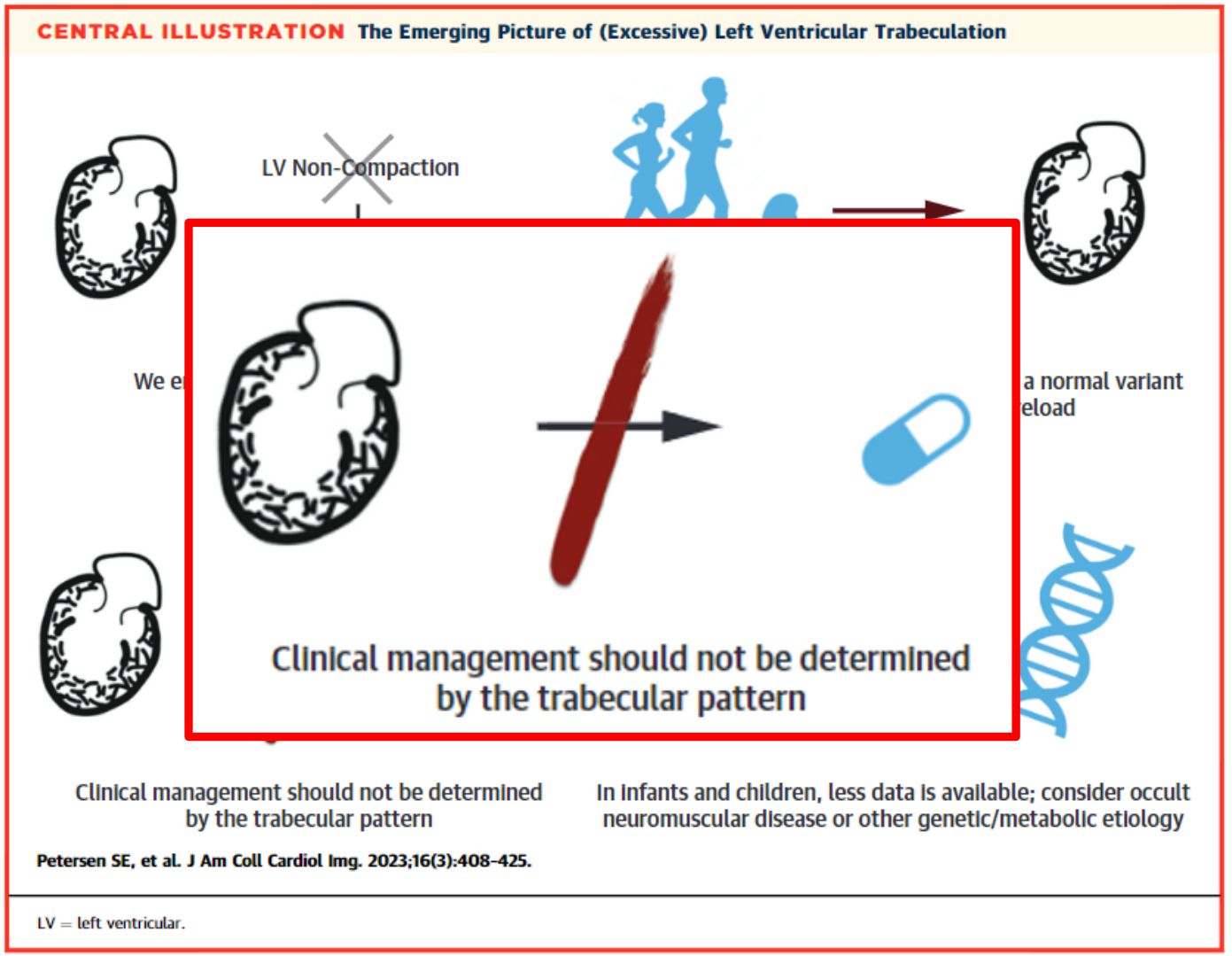
"phenotypic trait that can occur either in isolation or in association with other cardiac abnormalities "

Triad of LVNC: A myth !?

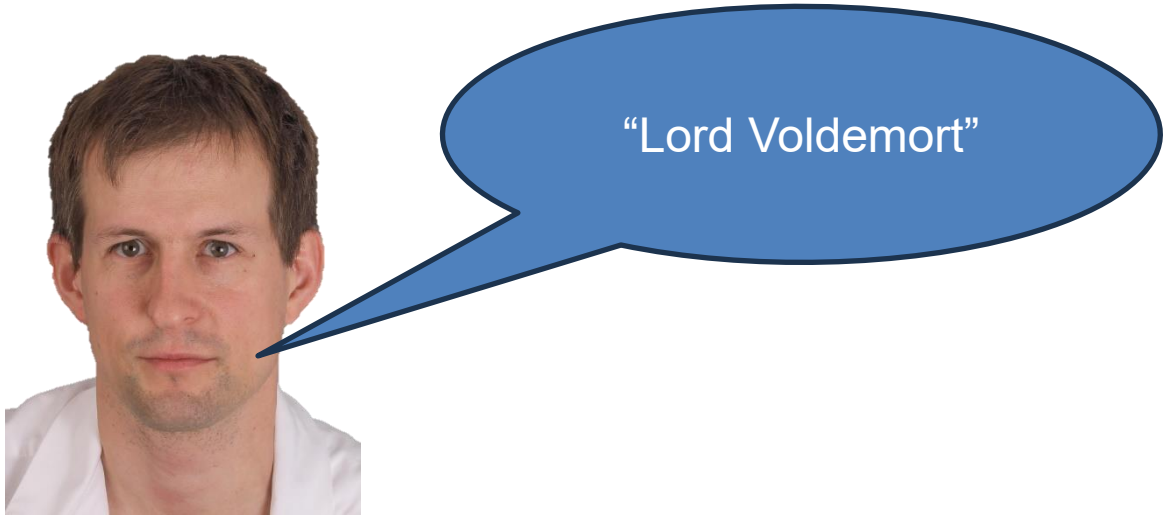
1. **Arrhythmia**: after correction of confounders (EDV, EF, LGE) no additional arrhythmic risk of trabeculations
   [Andreini D. JACC 2016]
2. **LV thrombus**: after correction of confounders (LVEF, heart failure) no elevated risk of trabeculations
   [Sigvardsen Eur Heart J CV Im 2021]
3. **LV dysfunction**: no or only very weak correlations of trabeculations and LVEF
   [Meyer HV Nature 2020]

➔ **LV trabeculations: no independent prognostic role.**

~~Left ventricular non-compaction (LVNC)~~



Soweit zur Statistik…



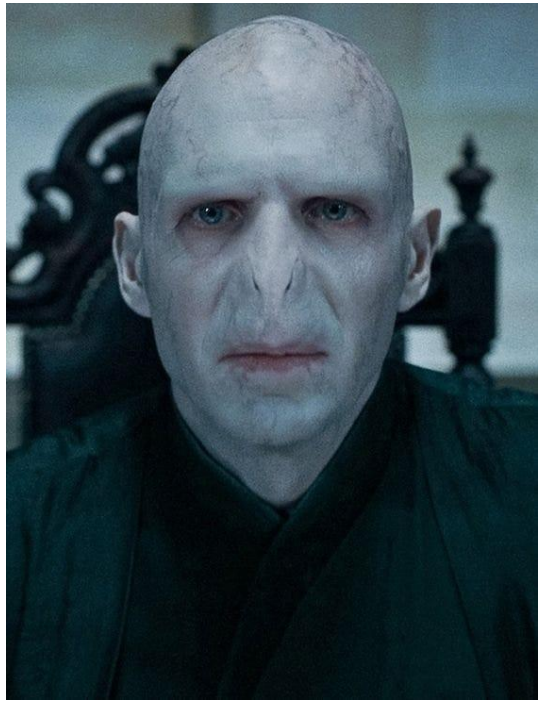
LV-Noncomp…

Schaden

- unnötige jahrelange OAK (!?)
- viele unnötige ICDs (!?)

Leid

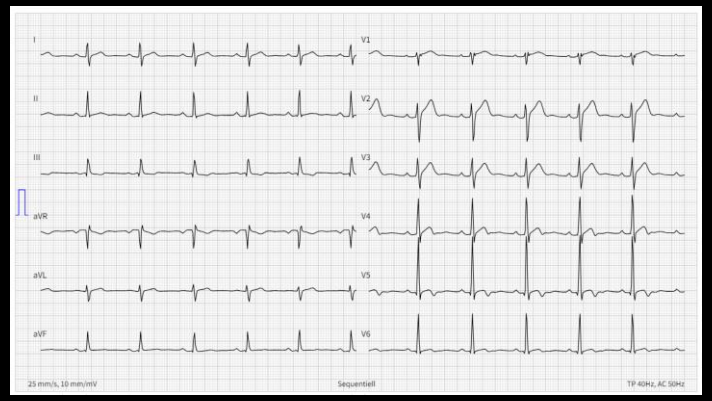
- Viele Sorgen über Herzkrankheit
- (Lebens-)Versicherungen…



Lord Voldemort

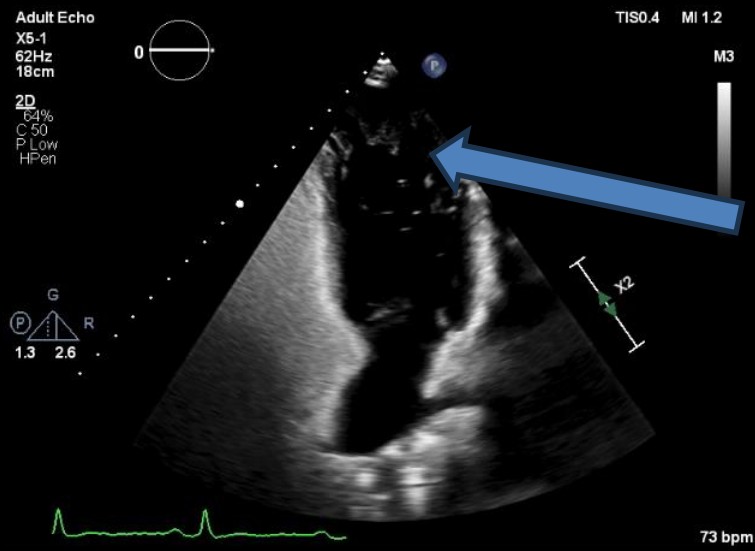
dessen Namen man nicht aussprechen darf, da er so viel Schaden und Leid verursacht hat

“Stroke-Echo”

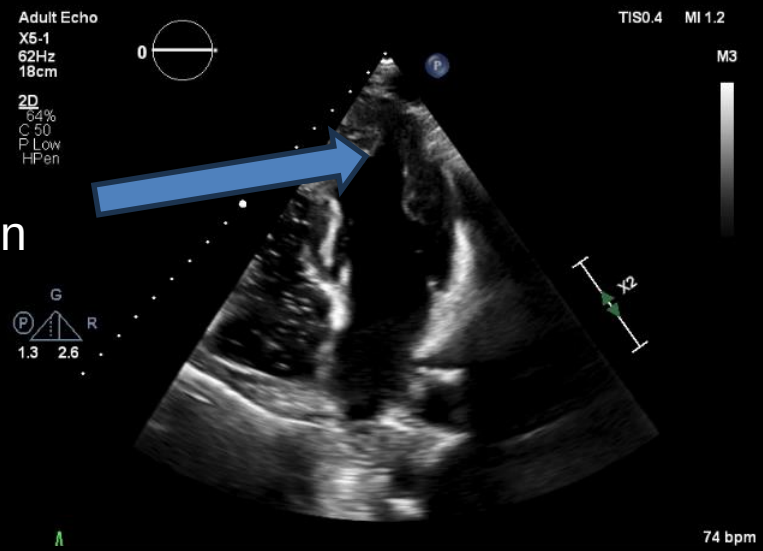


L.M. männlich *2000

Akuter ischämischer Hirninfarkt rechten Centrum semiovale
JL/Klinik: passagere motorische Schwäche Bein links
danach Kribbelparästhesien Arm links



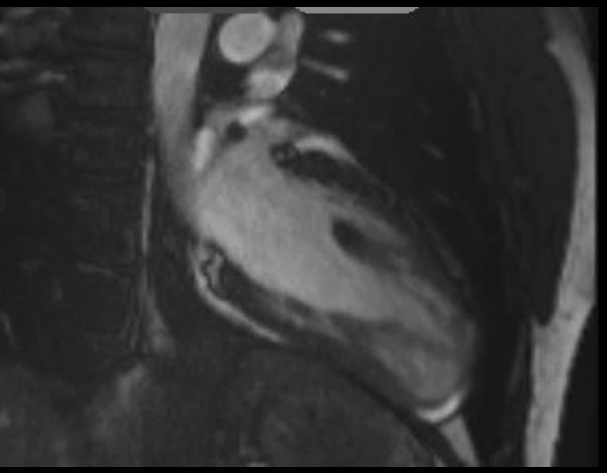
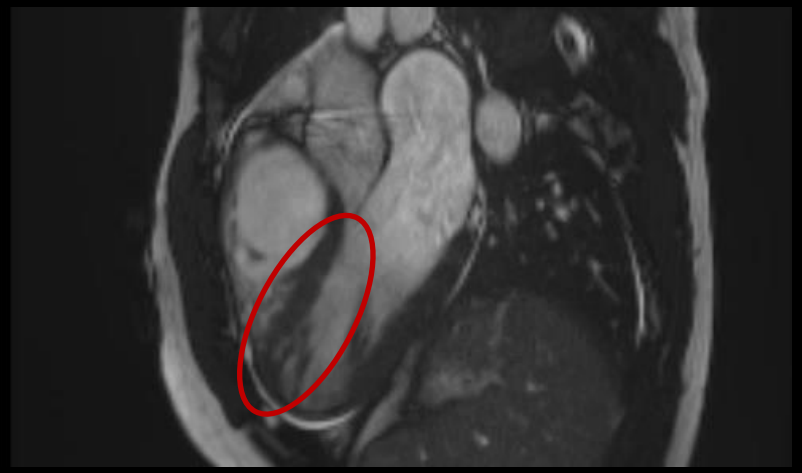
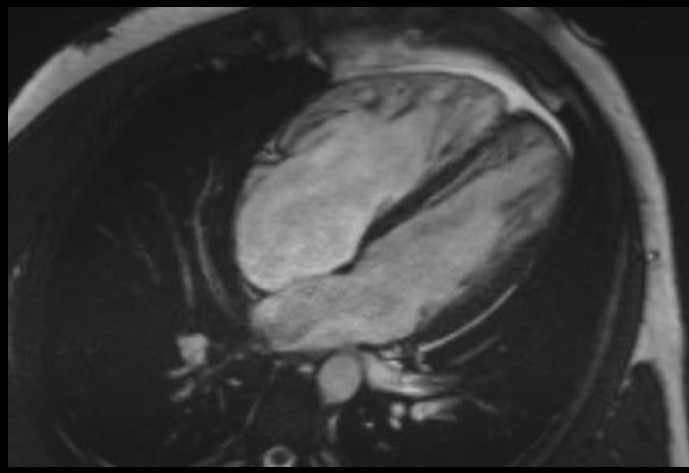
Hypertrabeculation



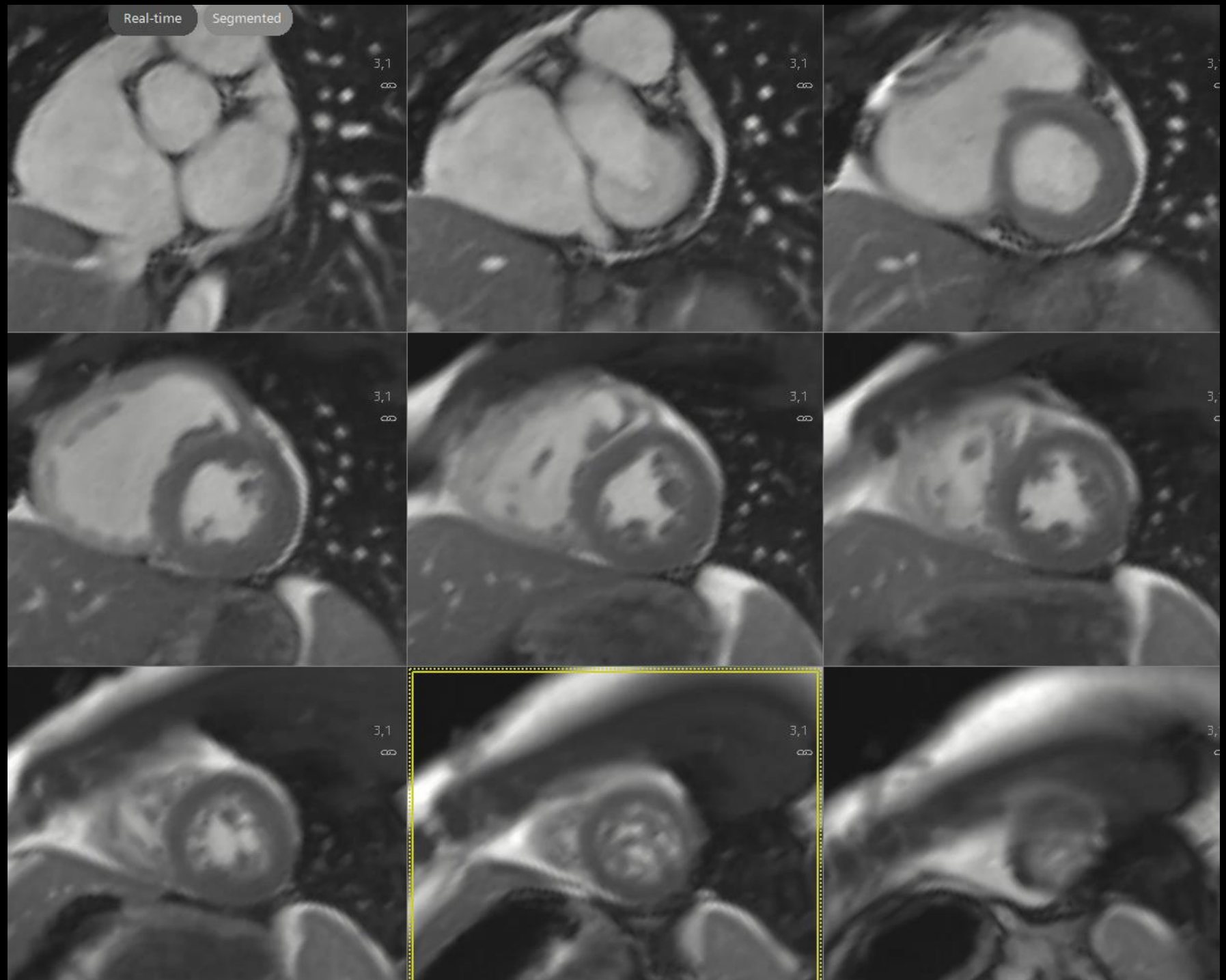
Rest

Valsalva

Schwallartiger Übertritt von “bubbles” unter Valsalva → V.a. PFO

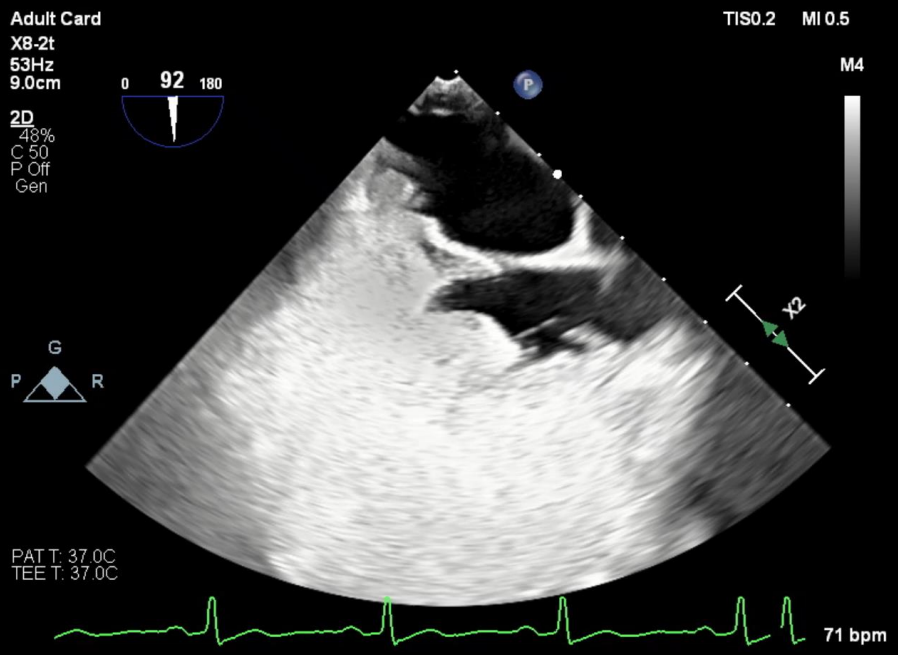
- Hypertrabeculation with multiple deep myocardials sinusoids of the septum
- Step-like thinning of the compacted myocardium in the apex
- LVEF 59%; LV-EDVi 101 ml/m^2 [47-107 ml/m^2]
- RVEF 57%, RV-EDVi 142 ml/m^2 [53-123 ml/m^2], RA ↑↑
- RV-SV : LV-SV = 150 / 108 ml (~Qp:Qs=1.4)
- Atrial septum aneurysm (TTE with suspected large PFO)
- No intraventricular thrombus
- Exclusion of an extracardiac shunt



- NC/C Ratio up to 7.7
- Subtle diastolic D-Shaping of septum

- Right heart dilatation → TOE for «PFO assessment»

TOE

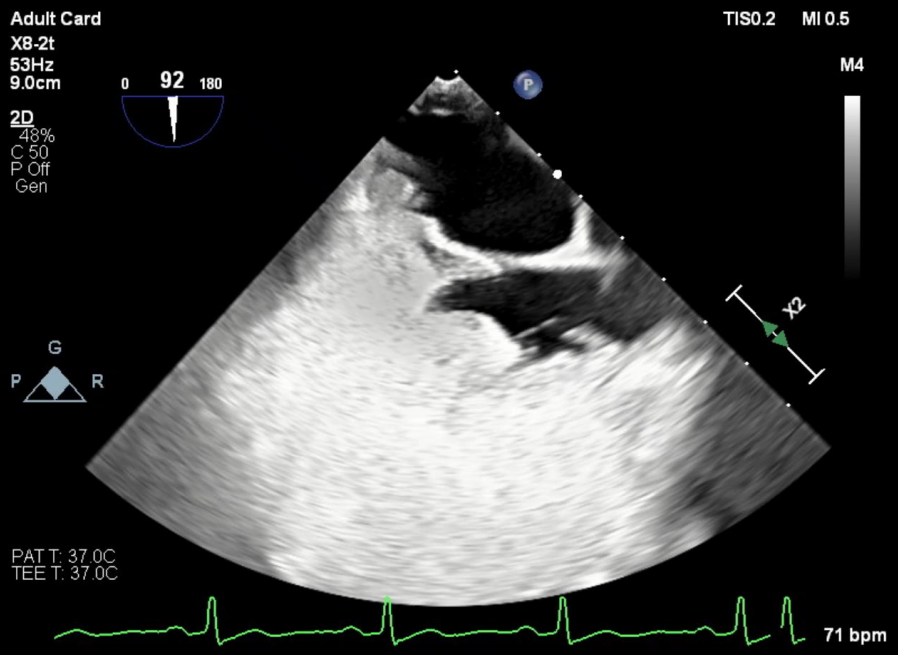


Pathology??

Shunt??

Venous access??

TOE



Pathology

ASD Type 2 with two orifices

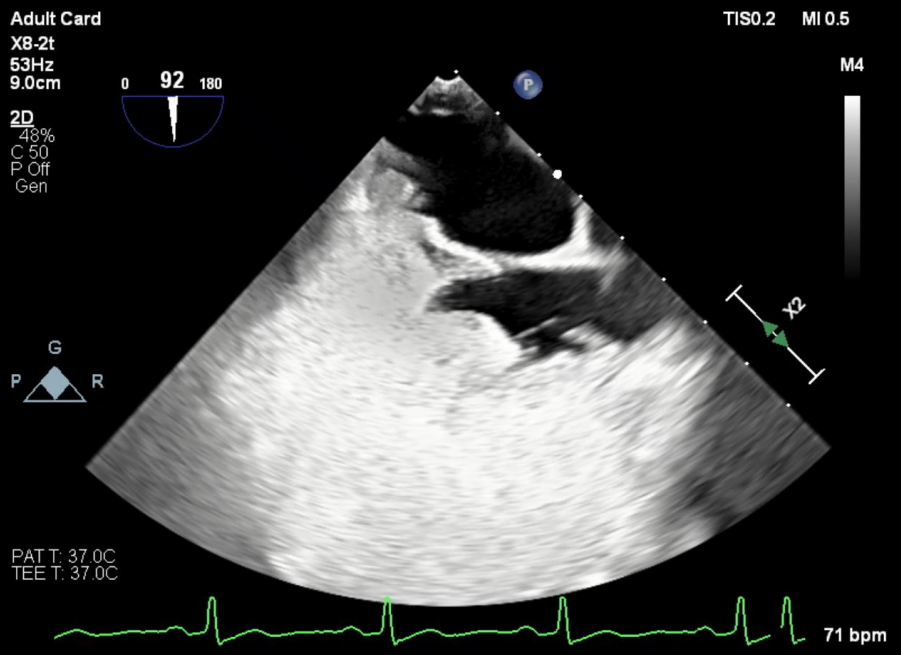
Shunt

bi-directional (predominantly L-R-shunt)

Venous access

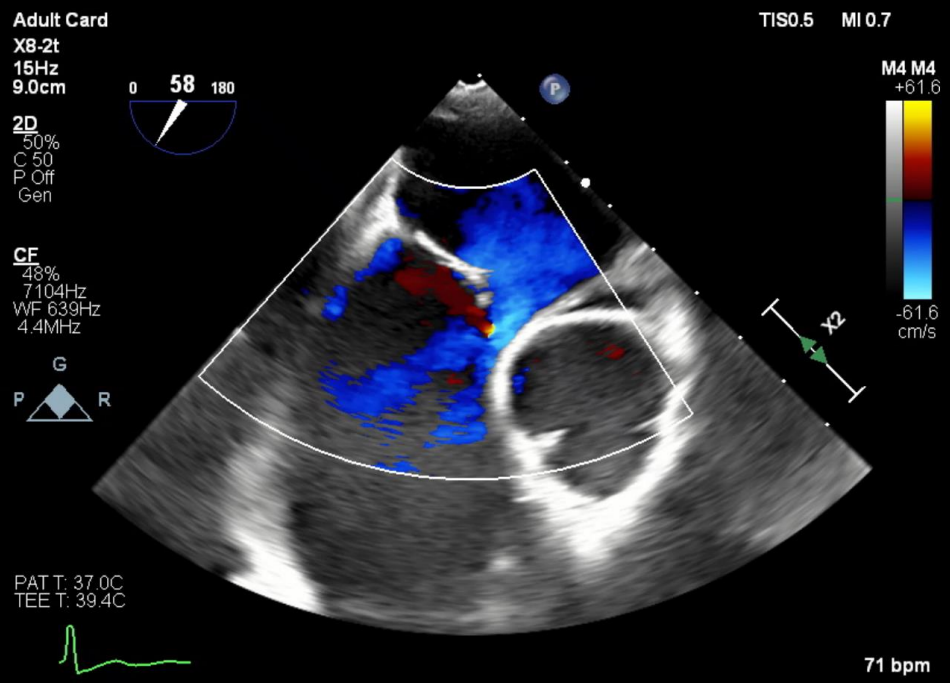
leg → IVC for exclusion of inferior sinus venosus defect

TOE



ASD with 2 orifices

Bidirectional shunt



Right heart catheter: no PAH, Qp:Qs = 1.5

Closure of ASD? OAK? Follow-Up?...

«LV non-compaction CMP» + complex ASD

or

Complex/genetic cardiomyopathy with ASD, hypertrabeculation and sinusoids ?

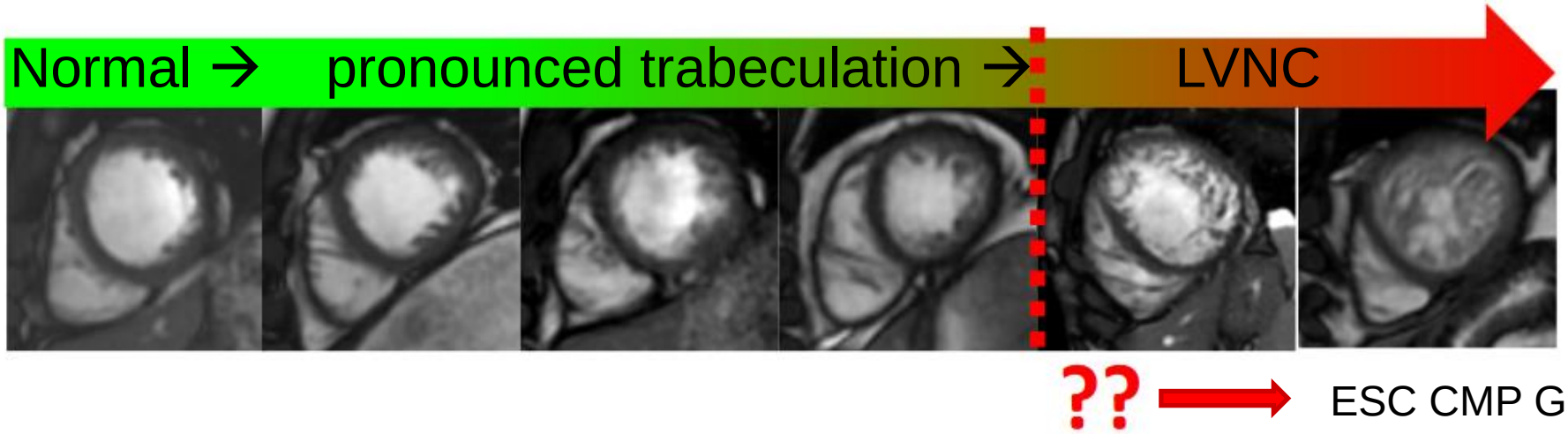
Case

- 24y old patient with ischaemic stroke
- excessive trabeculations (NC/C ratio 7.7), multiple sinusoids
- No LV thrombus, no LGE
- ASD Type 2 (2 orifices) with right heart dilatation

Heart Team recommendation:

- Surgical closure of ASD (Right heart dilatation, no PAH)
- «LV non-compaction Cardiomyopathy» → hereditäre Sprechstunde
- NOAK until closure of ASD, re-evaluation during follow-up

LV non-compaction CMP



ESC CMP Guidelines 2023

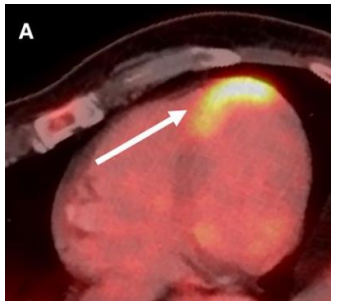
- **Be very cautious about making a diagnosis…**
- In the vast majority LV hypertrabeculation in the context of another (genetic) CMP (DCM, HCM, ARVC, IHD) or acquired
- Often over-diagnosed in the past, particular in
  - *DCM* (thin compacted myocardium)
  - *Afro-Americans*

Excessive trabeculation + no other feature = benign

Take Home Messages

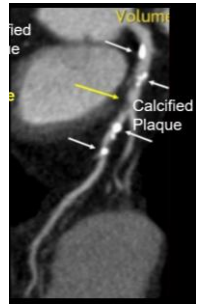
Cardiometabolics

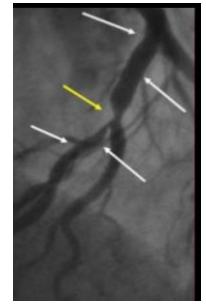
Lipid Switch in FDG-PET for detection of ischaemia/hibernation



Coronary artery disease

Extraluminal vs. Intraluminal disease





Cardiomyopathy

Genetic overlap of Cardiomyopathies and LV-Nonc…