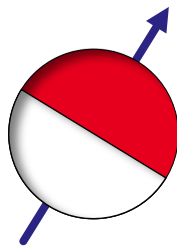


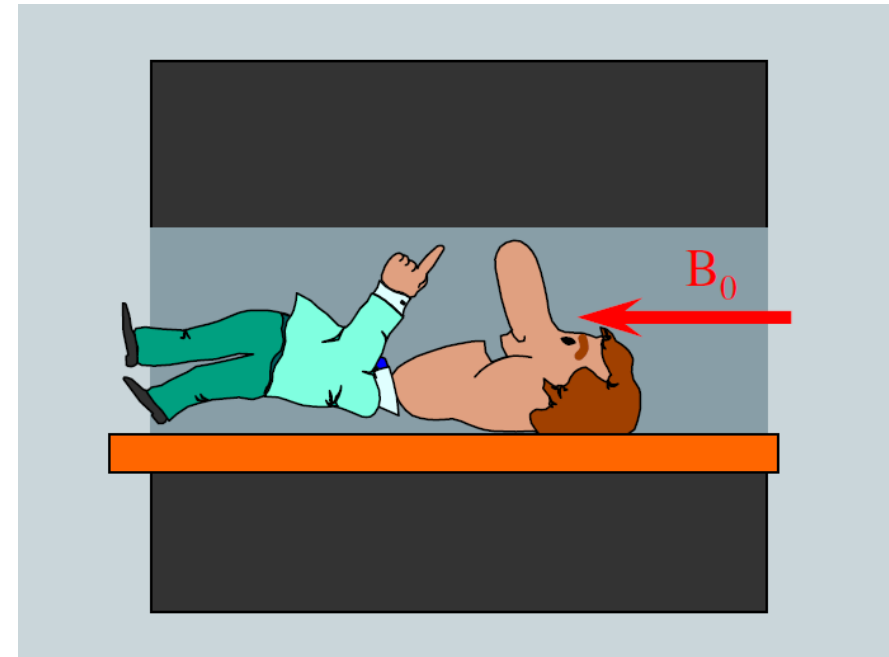
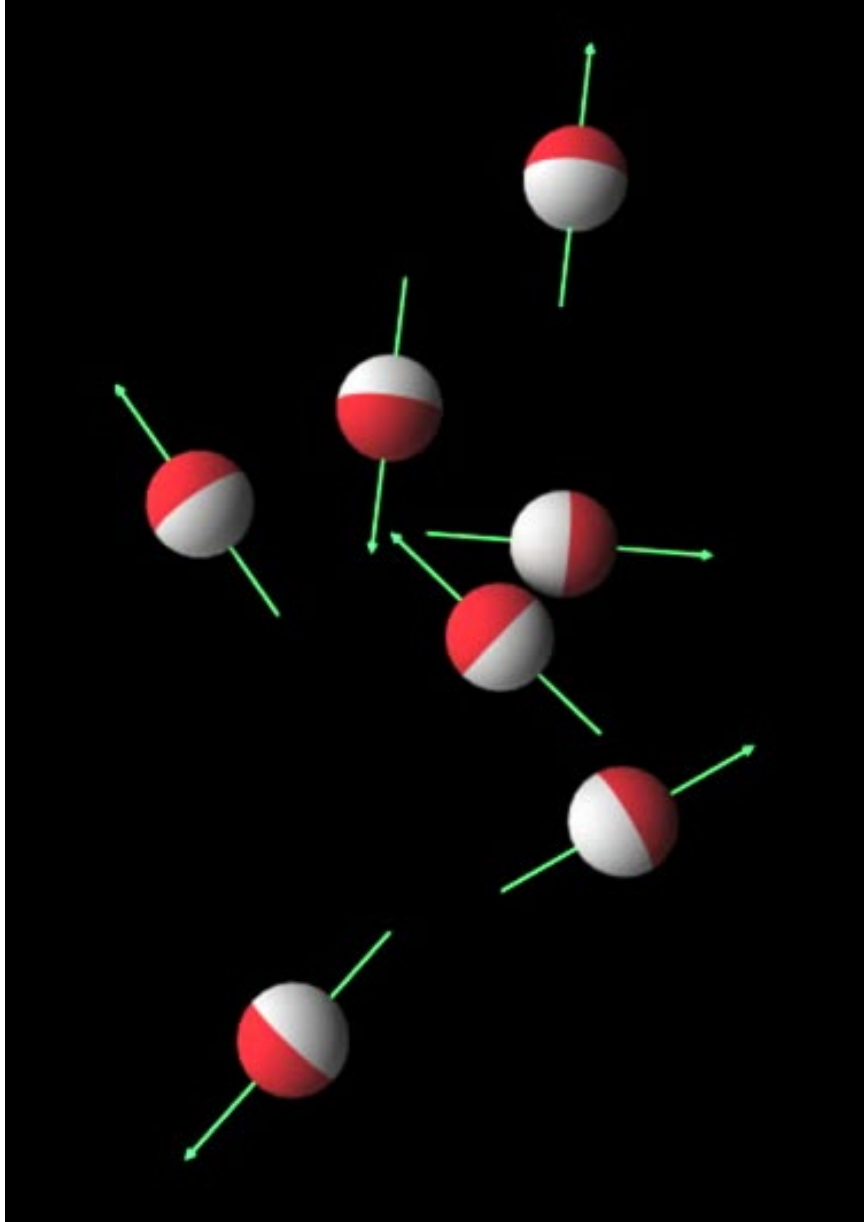
Introduction à l'IRM

I.R.M (N)

- Imagerie
- Résonance
- Magnétique
- Nucléaire (hydrogène)

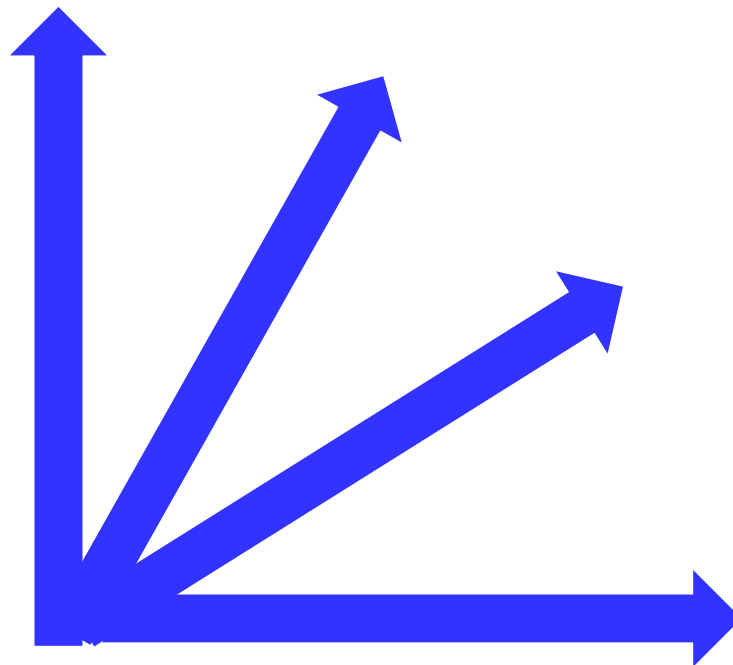
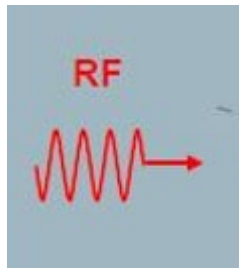
Le proton (hydrogène)





Magnetisation longitudinale

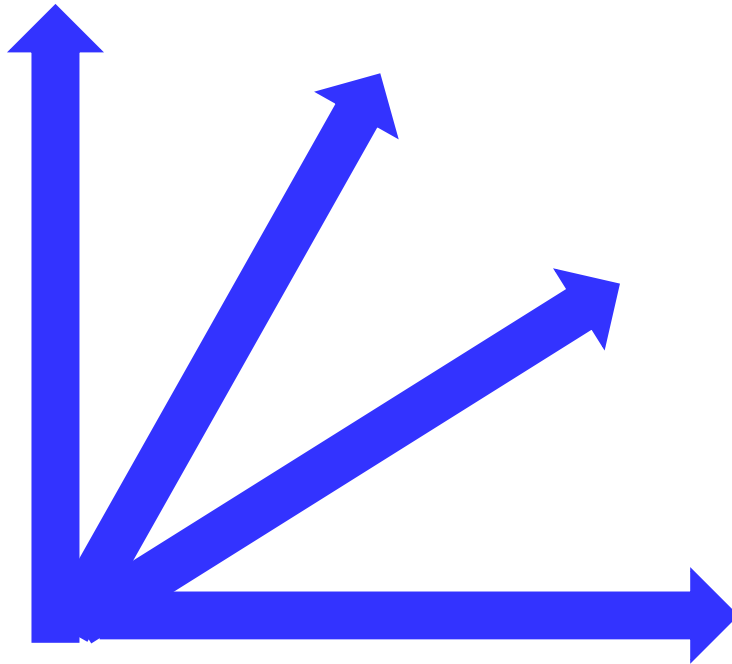
Magnétisation longitudinale



excitation

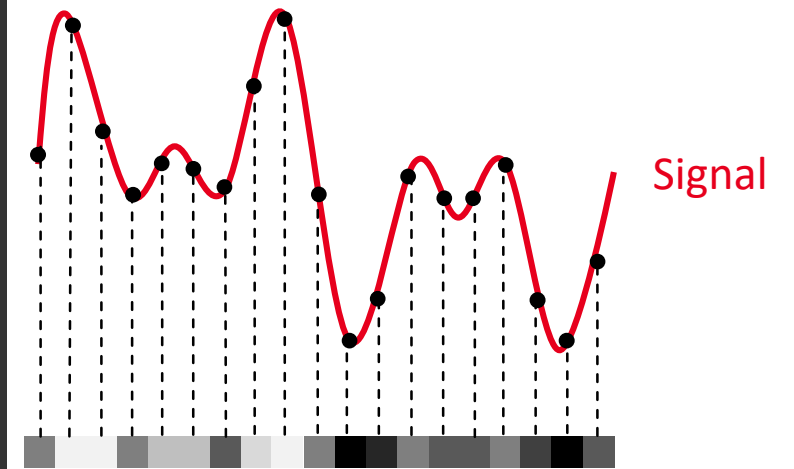
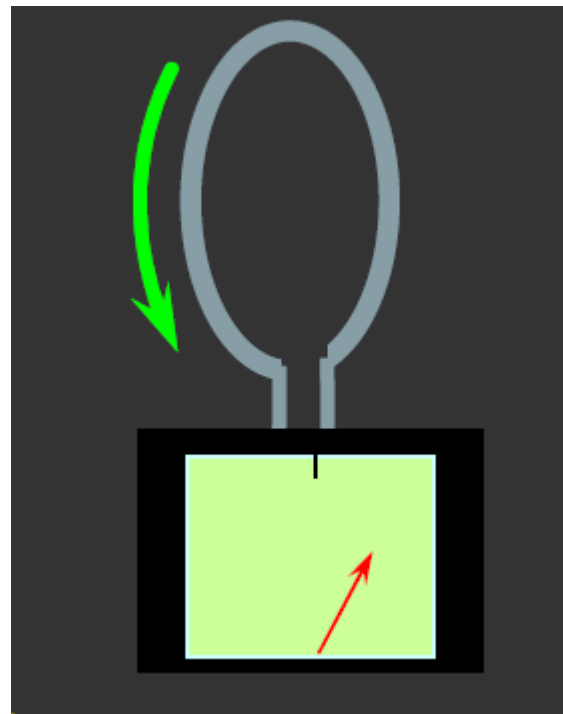
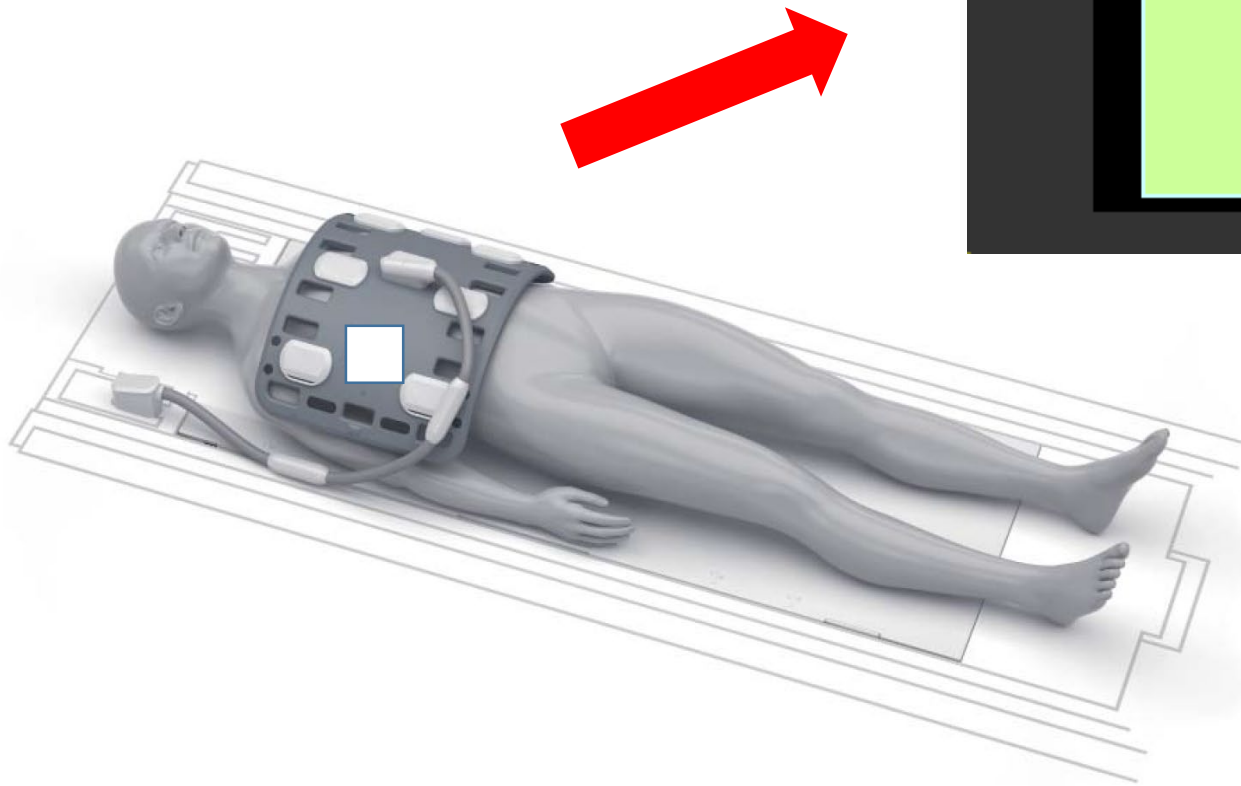
Magnétisation transversale

Magnetisation longitudinale

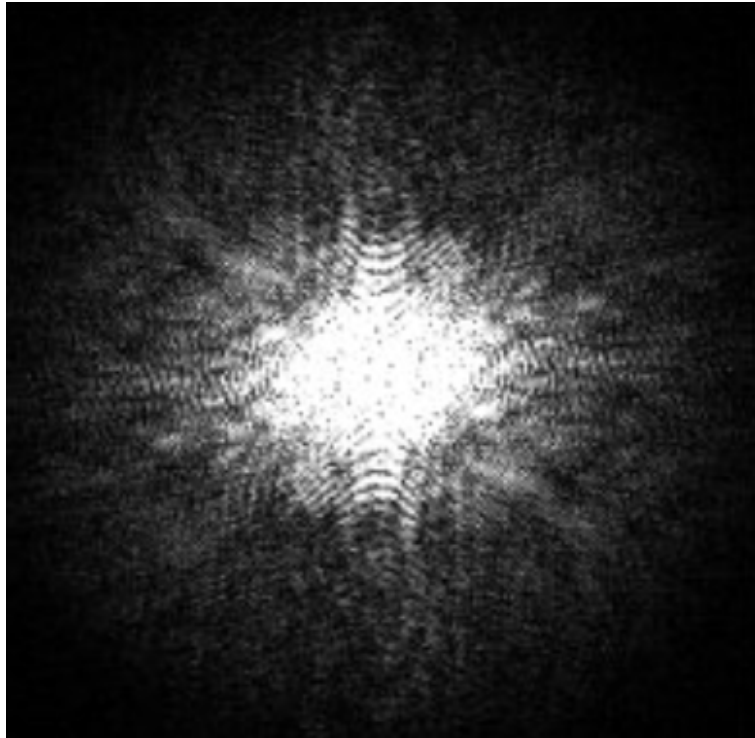


Récupération

Magnetisation transversale



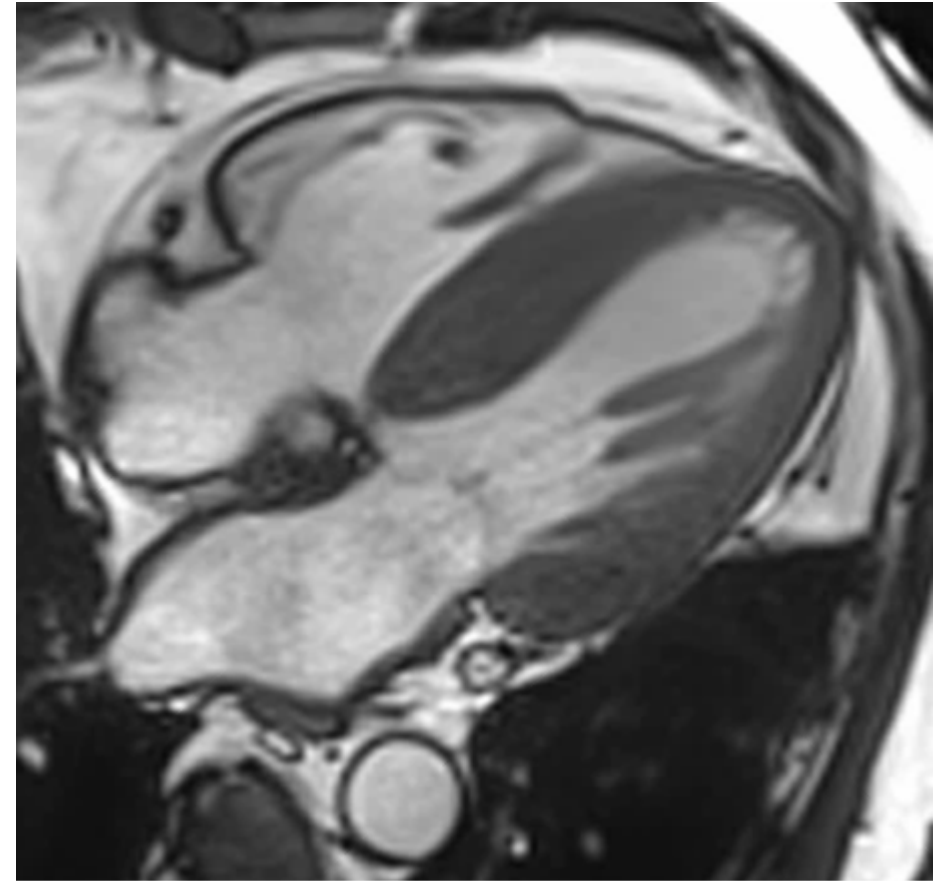
K space

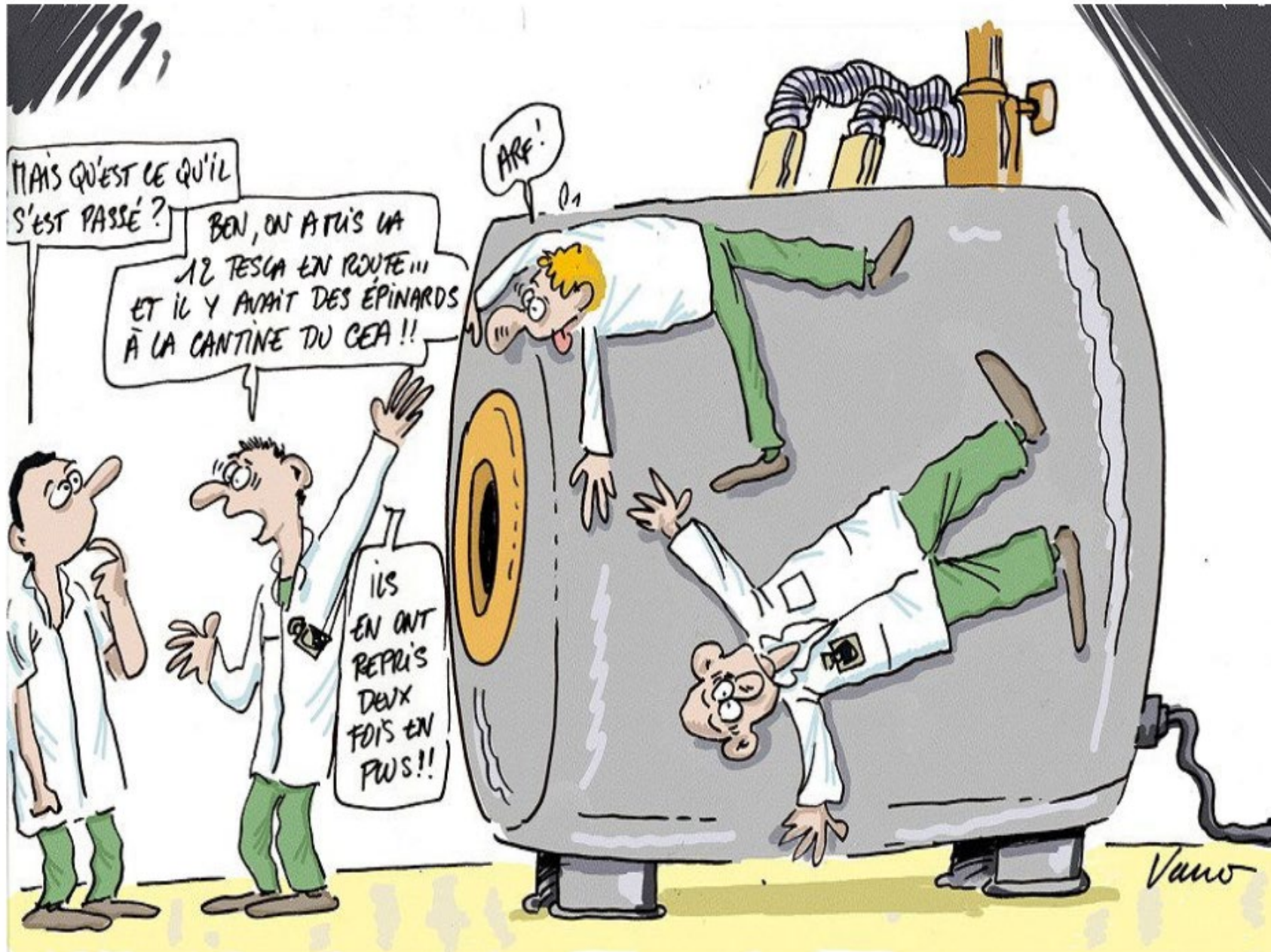


Transformé de Fourier



image





1,5 / 3 T

30,000 fois le champs
magnétique terrestre
(47 μ T en France)





DANGER - WARNING

LORS DE L'ENTRÉE EN SALLE IRM - FOR THOSE ENTERING THE MR ROOM



CHAMP MAGNÉTIQUE PUISSANT - STRONG MAGNET FIELD

DÉFENSE D'ENTRER - NO ADMITTANCE



Stimulateurs cardiaques
Pacemakers



Implants ferromagnétiques métalliques
Ferromagnetic metallic implants

Objets sensibles
Iron objects
Prothèses orthopédiques
Prosthetic artificial eyes

NON ACCEPTÉ - NOT PERMITTED



Objets métalliques
Sharp objects
Châssis métalliques
Metal frames
Prothèses dentaires
Dental prostheses



Objets ferromagnétiques en acier
Ferromagnetic objects
Prothèses dentaires
Dental prostheses



Prothèses dentaires en acier
Dental prostheses
Prothèses dentaires
Dental prostheses



Prothèses ferromagnétiques
Ferromagnetic prostheses



Appareils électroniques
Electronic devices
Prothèses électroniques
Electronic prostheses



Appareils de stockage
Storage devices
Prothèses de stockage
Storage prostheses



Objets ferromagnétiques en acier
Ferromagnetic objects
Prothèses dentaires
Dental prostheses



Prothèses ferromagnétiques
Ferromagnetic prostheses

Prothèses
L. 10110
Prothèses
L. 10110



Contraintes de l'IRM

- Temps d'acquisition vs qualité d'image
- Temps incompressible: il faut attendre le temps de relaxation du proton
- Mouvement: cœur + diaphragme
- Bruit: radiofréquences

CMR - Advantages

- Résolution spatiale and temporelle élevées
- Etude anatomique et fonctionnelle dans tous les plans de l'espace
Absence de limitations dû à la “fenêtre”
VG, VD, aorte, veines et artériels pulmonaires
- Evaluation “moins” observateur dépendant
- Non invasive and non irradiant: suivi de cardiopathie ischémique, aorte, cardiopathies congénitales de l'adulte (GUCH) !



Limitations

Contrindications à l'IRM

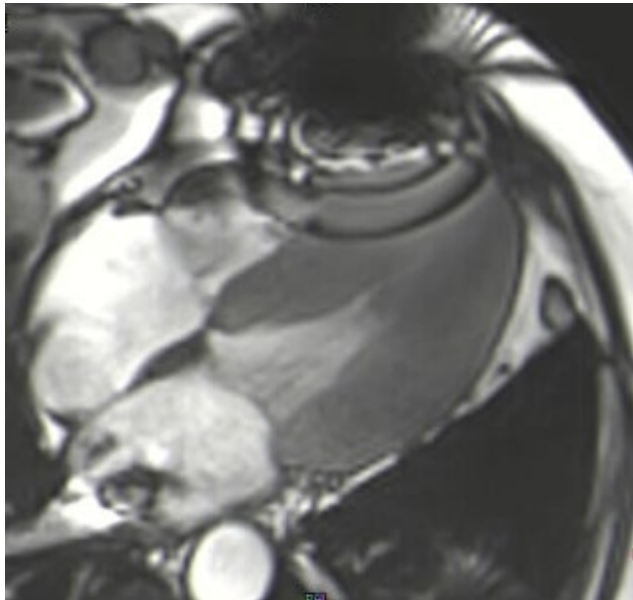
<http://www.mrisafety.com/>

- *éclat métallique oculaires*
- *clips cérébraux, implants cochléaires*
- *neurostimulateur, dérivation ventriculo-péritonéale, contrepulsion intraaortique*
- *Pacemaker / défibrillateur non MRI compatible*

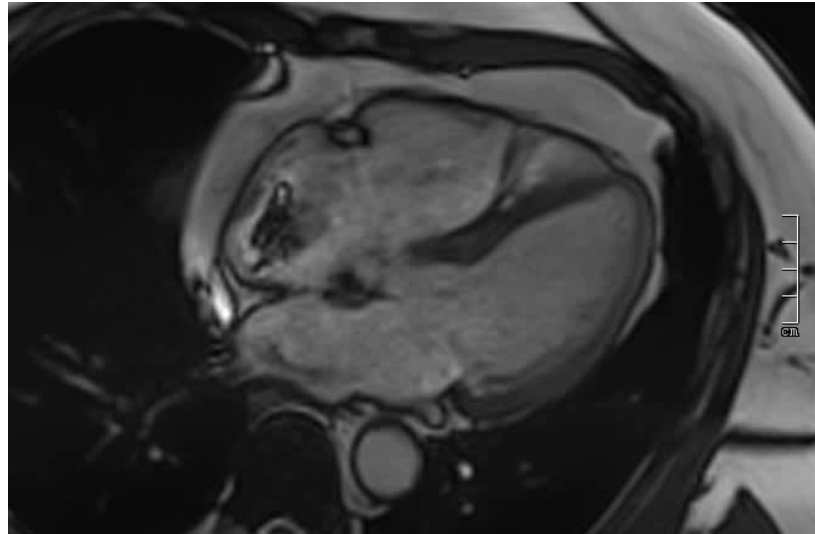
- Echauffement
- Déplacement
- Courants induits

Limitations

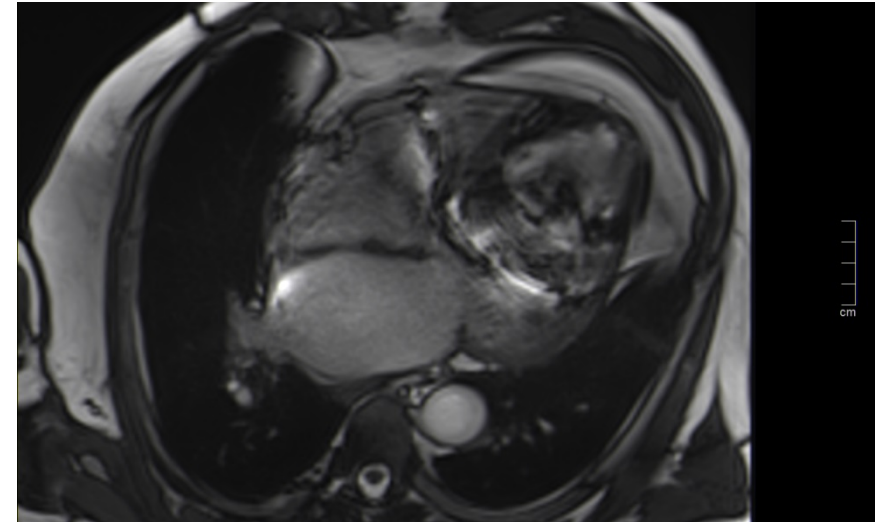
Reveal



PM



ICD



Limitations

➤ **Contrindications à l'IRM**

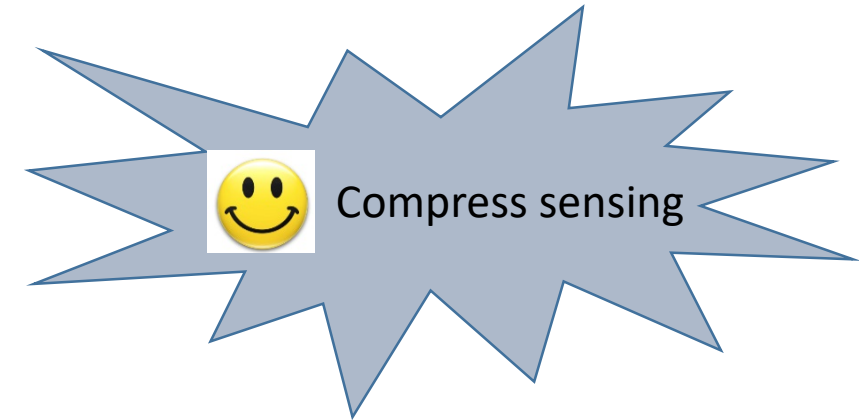
- *pacemaker/défibrillateur non MRI compatible*
- *éclat métallique oculaires*
- *clips cérébral, Neurostimulateur, implants cochléaires*

➤ **Limitations dû au patient**

- **arythmie:** FA, extrasystoles,
- **artefacts** dû au implants métalliques et stents
- **nécessité de la collaboration du patient:** examen long (45 min), apnée
- **claustrophobie**

➤ **Courbe apprentissage de l'opérateur**

- *Technique d'IRM*
- *Anatomie complexe (pathologie congénitale); pattern spécifique (ex sarcoïdose)*



accessibilité



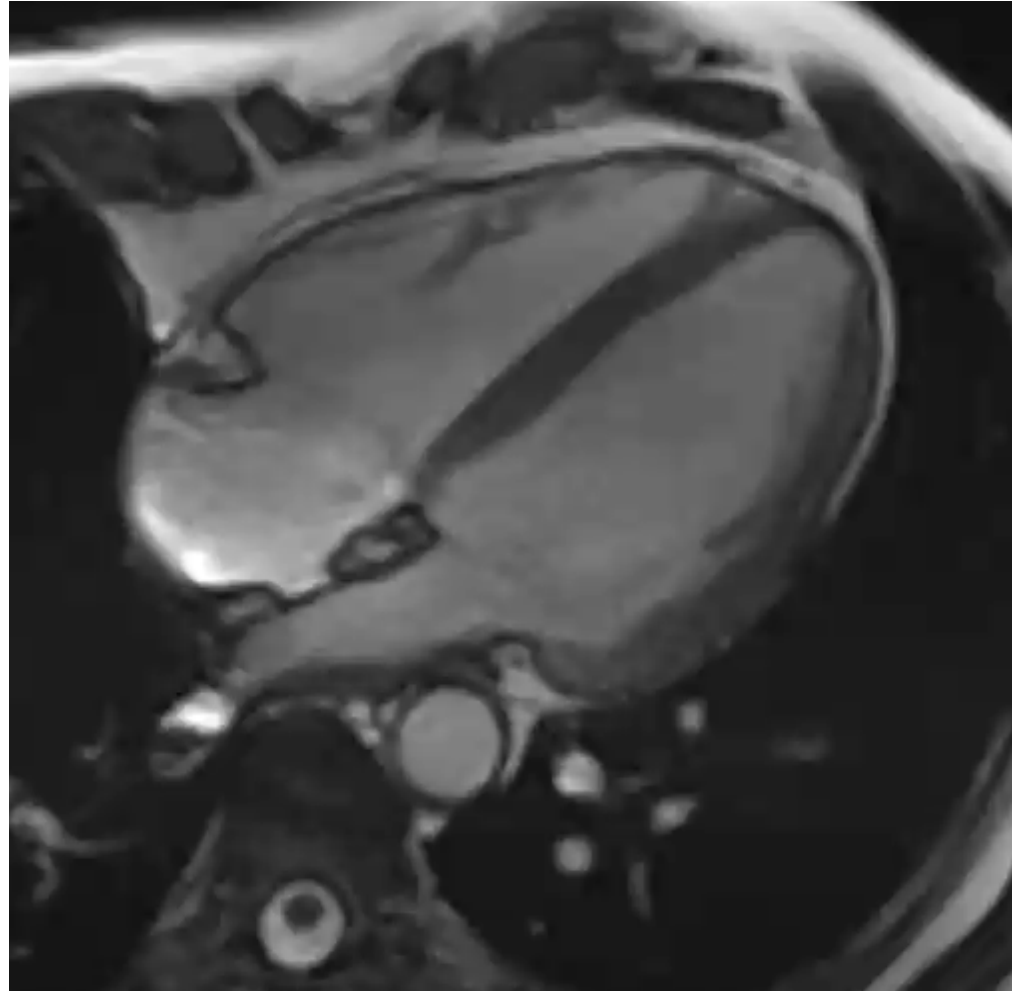
Coût !!!!

Menu des « séquences »



Séquences ciné *anatomie et fonction*

SSFP

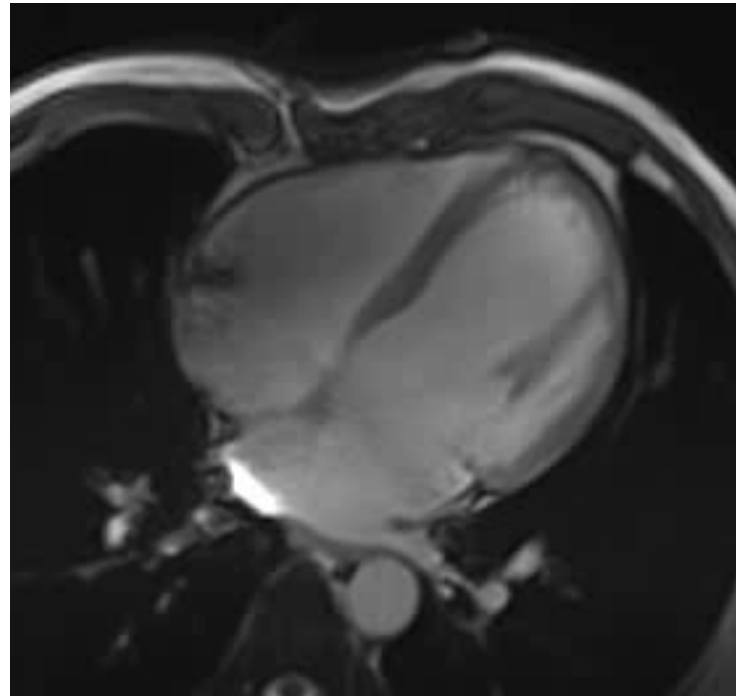


Pour chaque pathologie, une morphologie du VG

Péricardite constrictive



Barlow



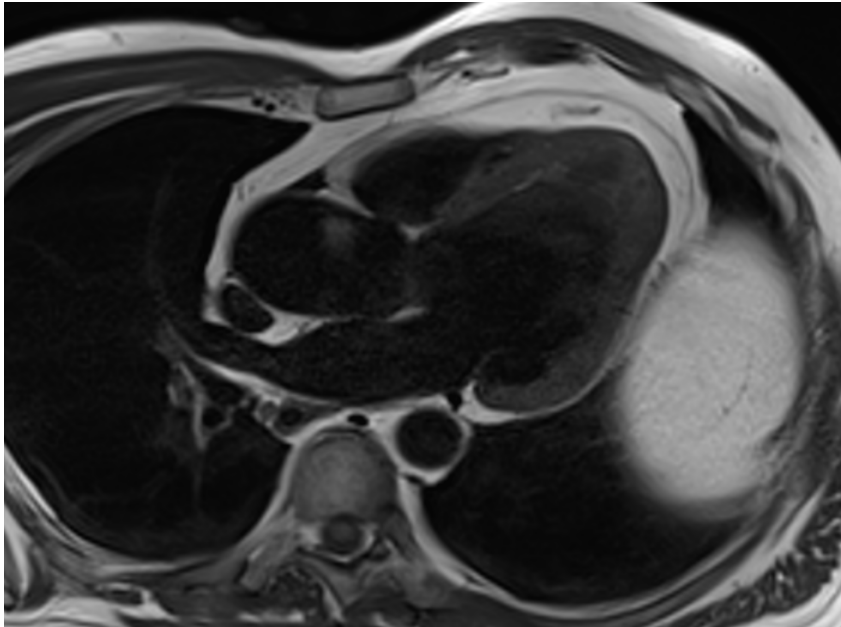
IDM



Séquences morphologiques: *anatomie et caractérisation tissulaire*

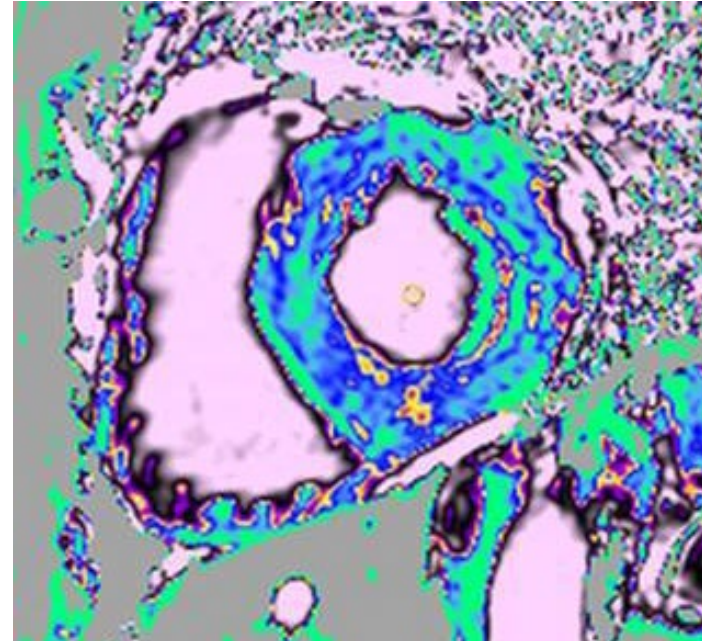


TSE T1



Analyse qualitative

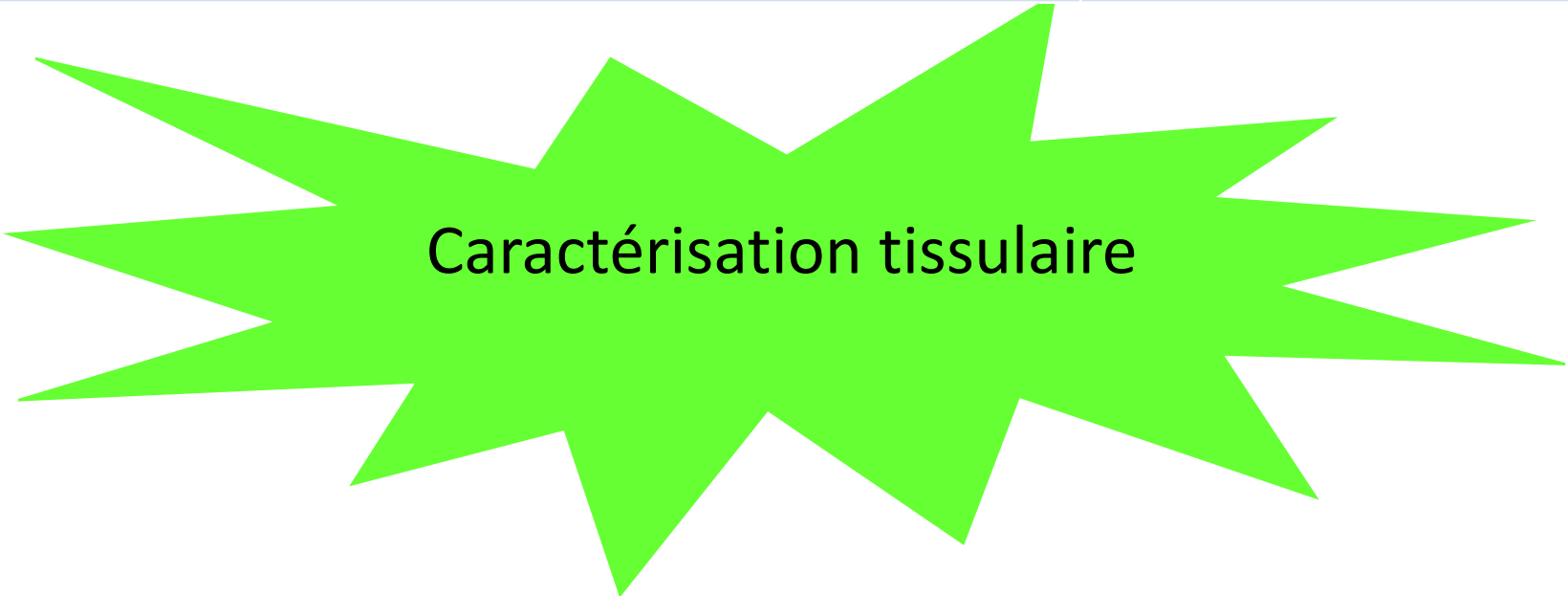
Mapping T1



Analyse quantitative



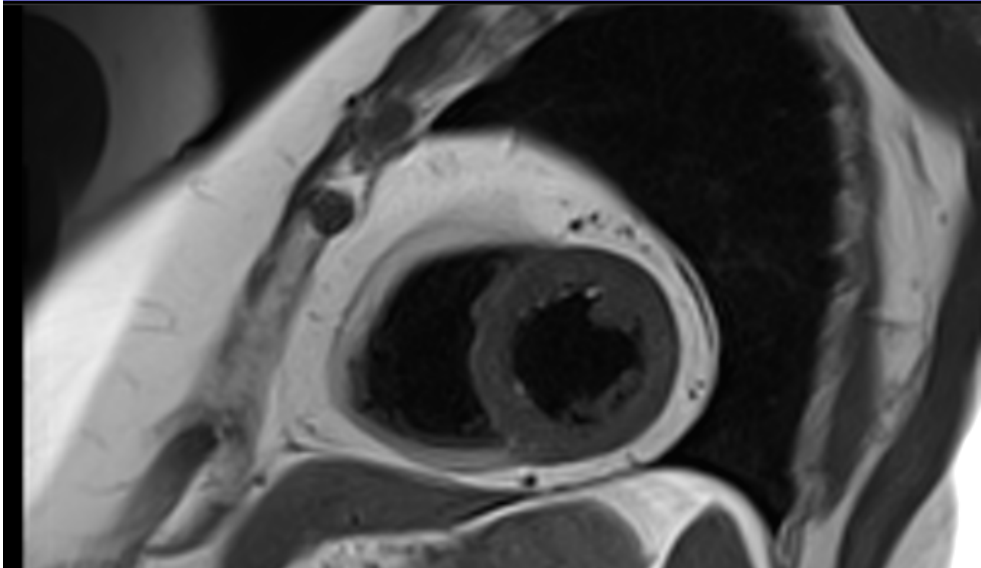
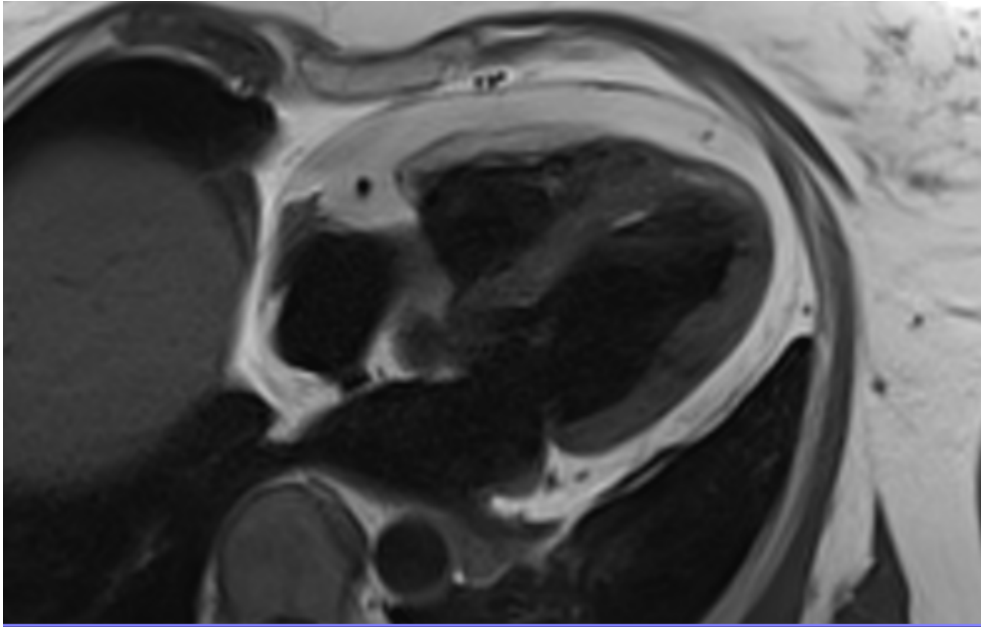
T1	vitesse récupération de la magnétisation longitudinale	Fibrose , graisse
T2	vitesse de diminution de la magnétisation transversale	œdème



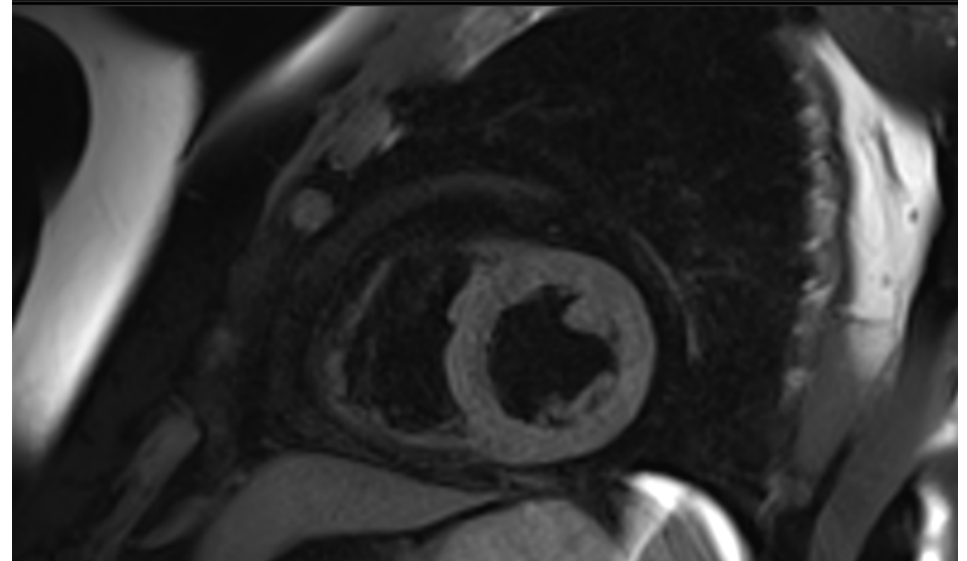
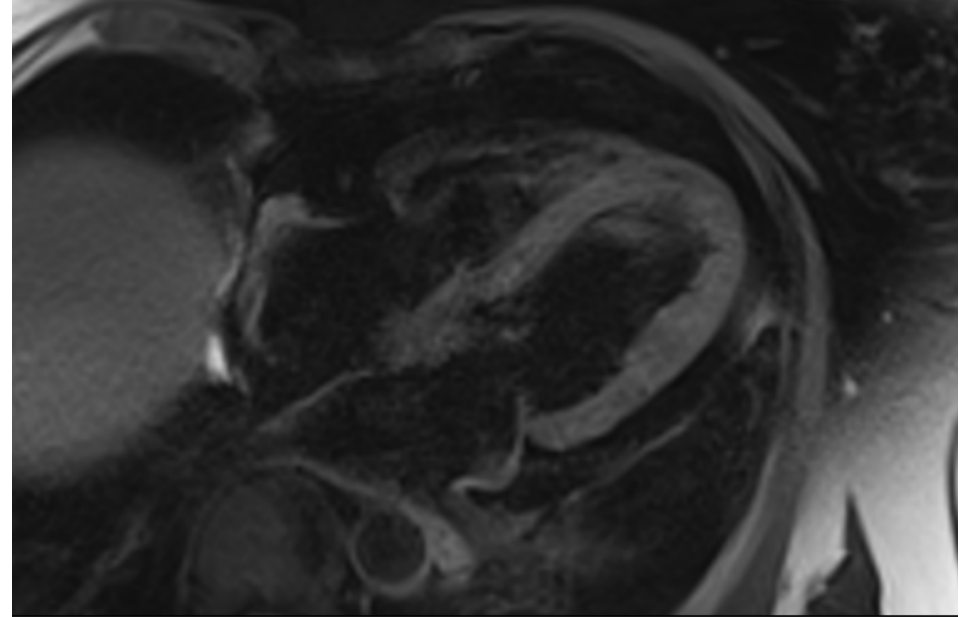
Caractérisation tissulaire

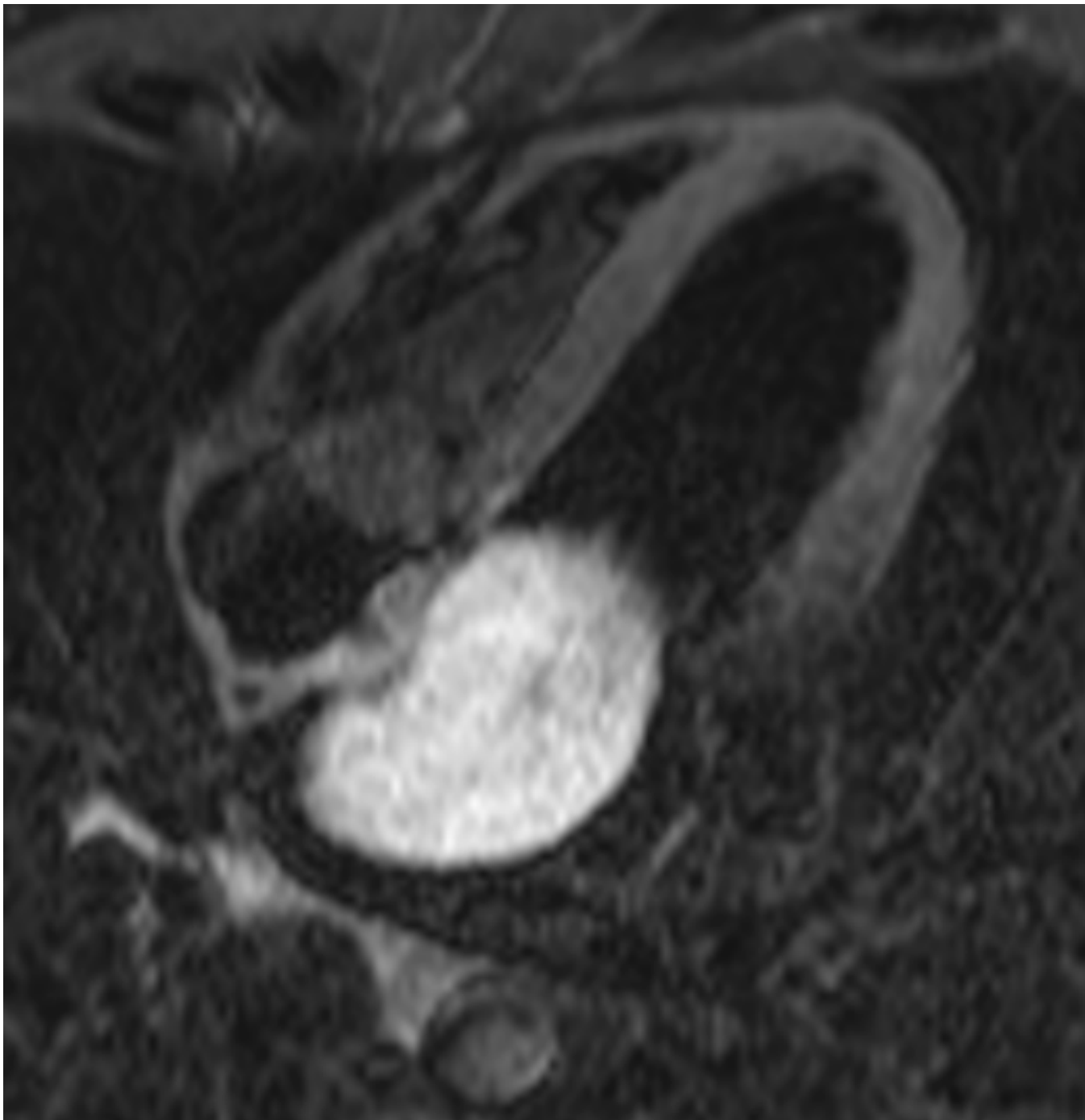
T1: graisse

TSE T1



Fat sat

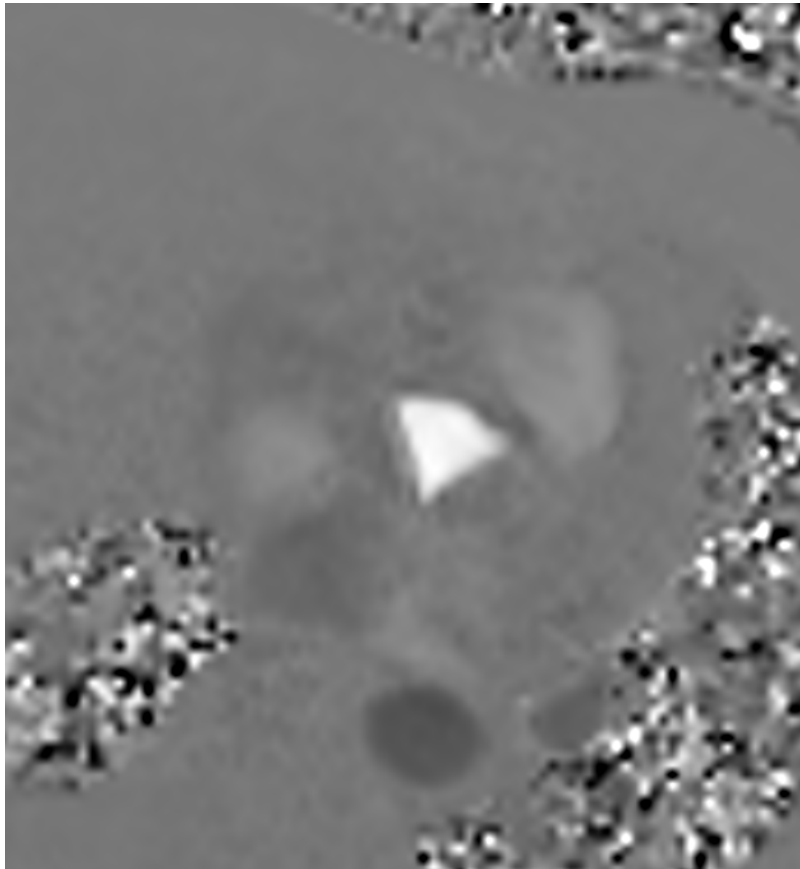




STIR

- séquence pondéré en T2
+ suppression de la graisse
- Seul H₂O est en hypersignal

Séquences en contraste de phase: *étude des flux*

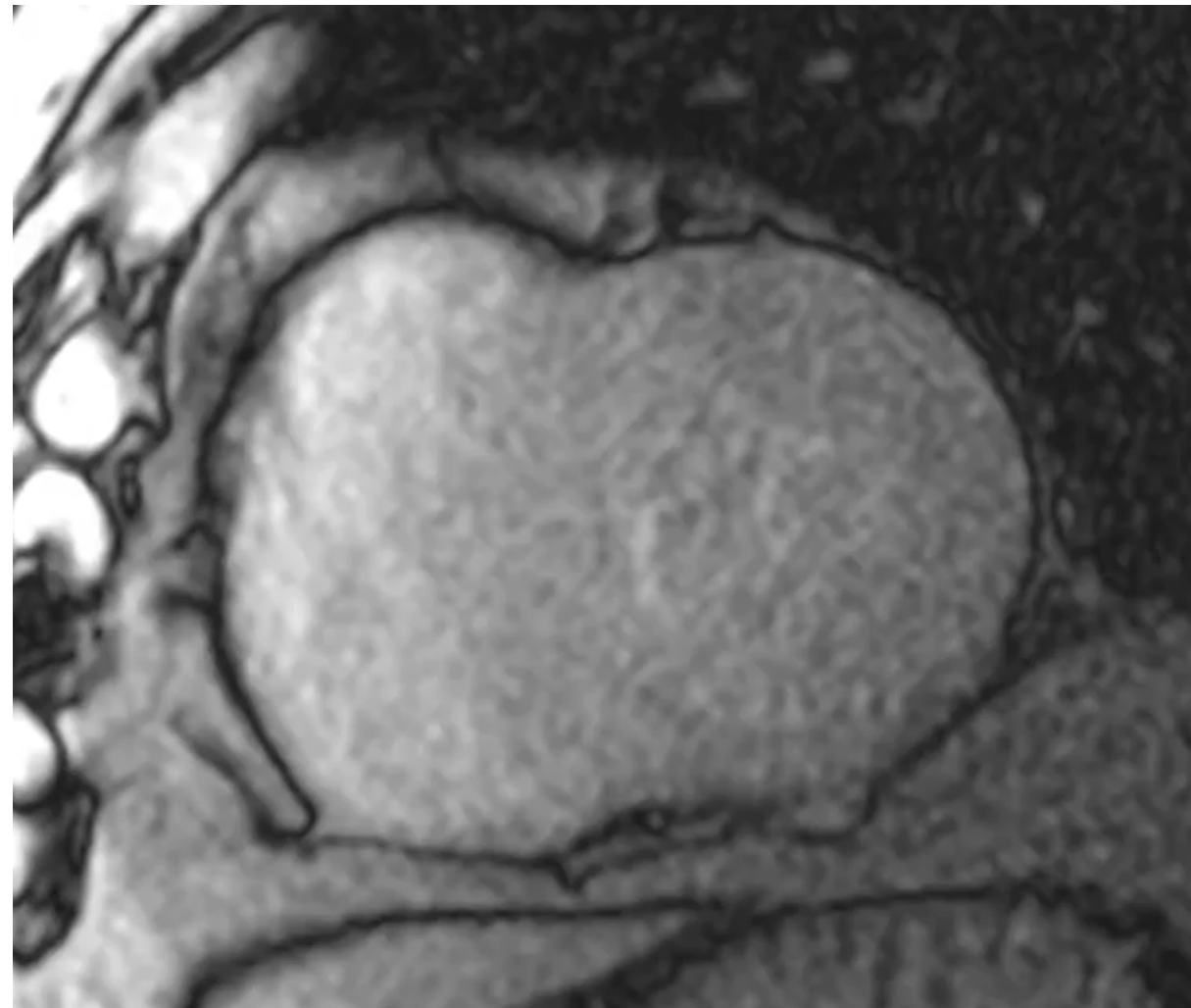
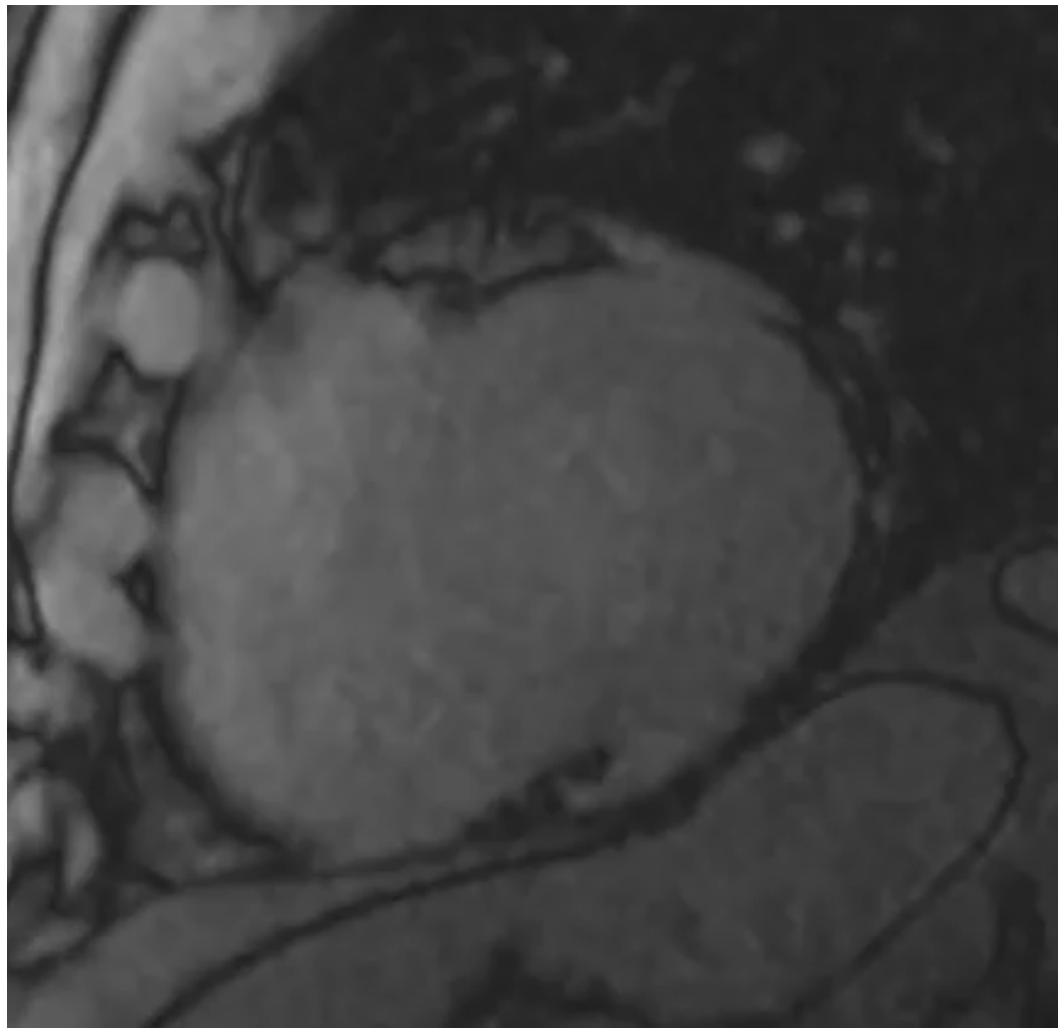


- chaque pixel représente une vitesse
- blanc / noir dépend de la direction du flux
- l'intensité du blanc/noir dépend de la vitesse

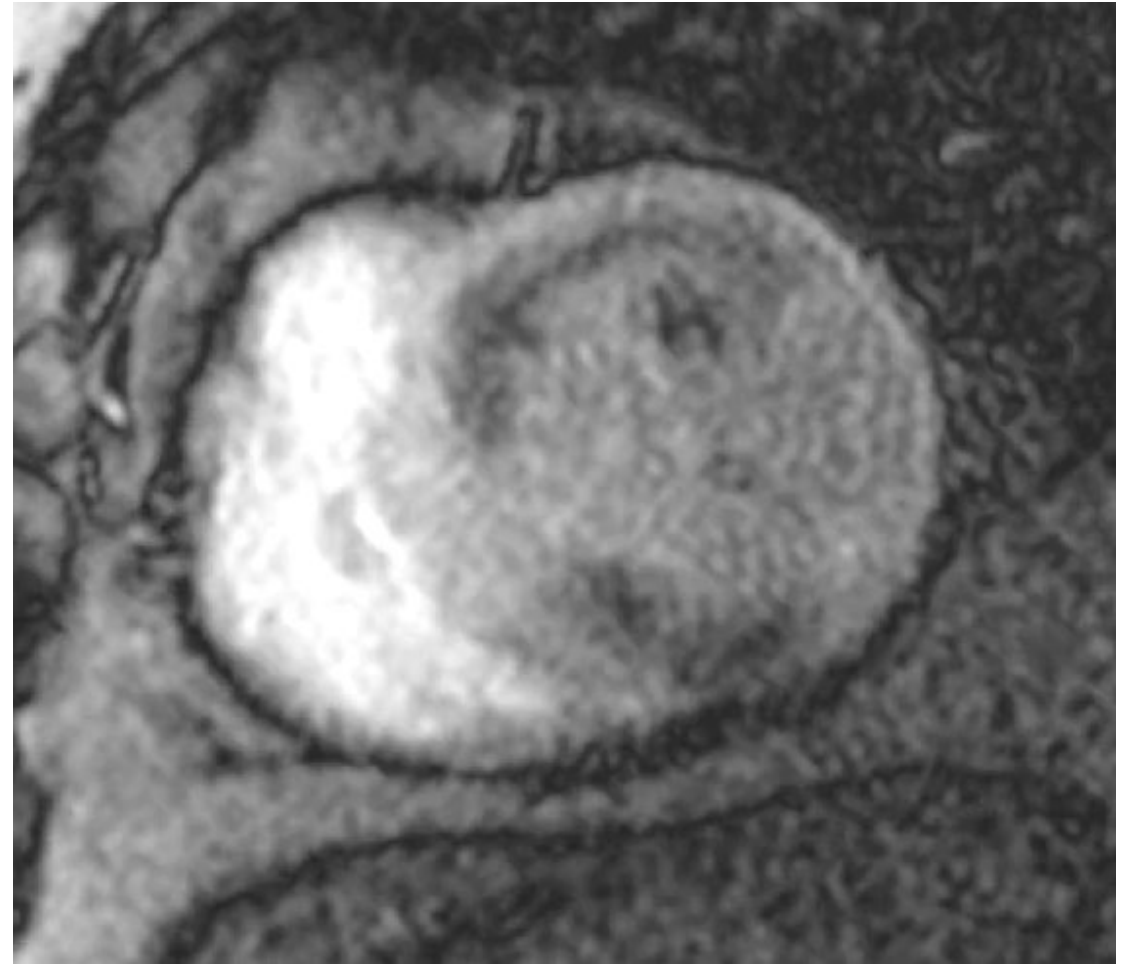
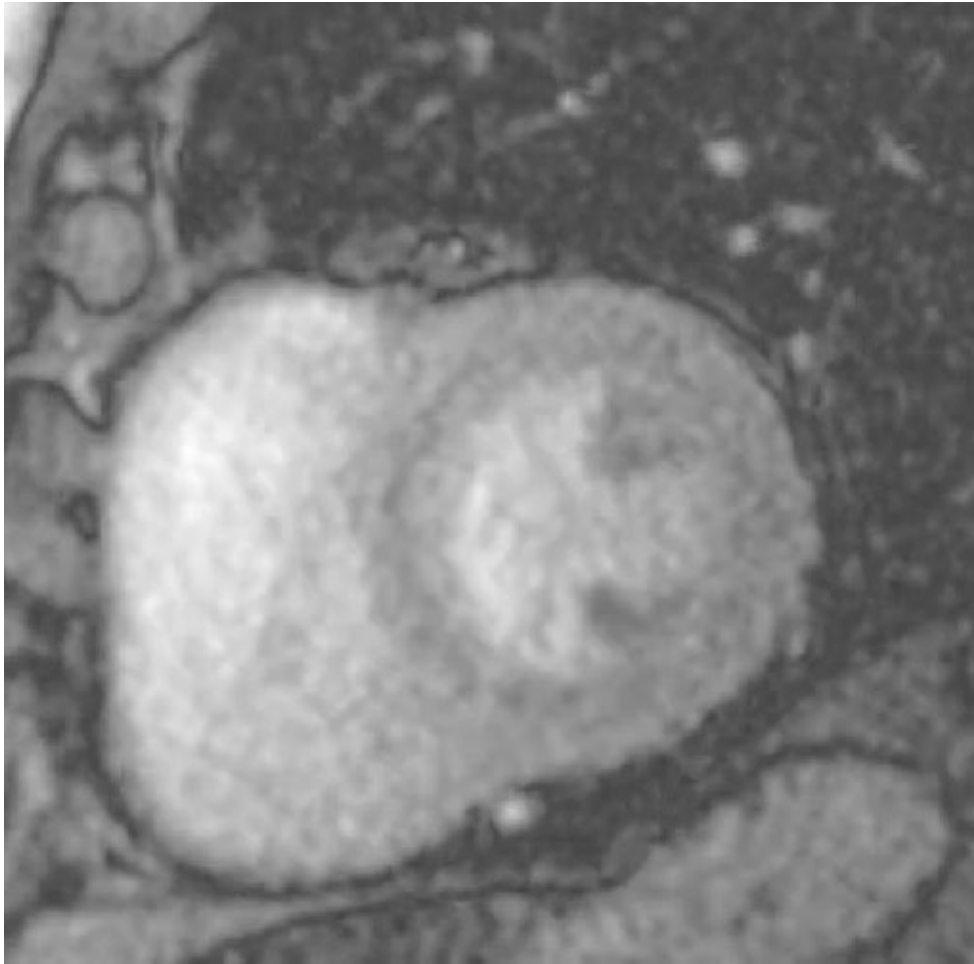
On mesure:

- V max
- Flux antérograde/rétrograde

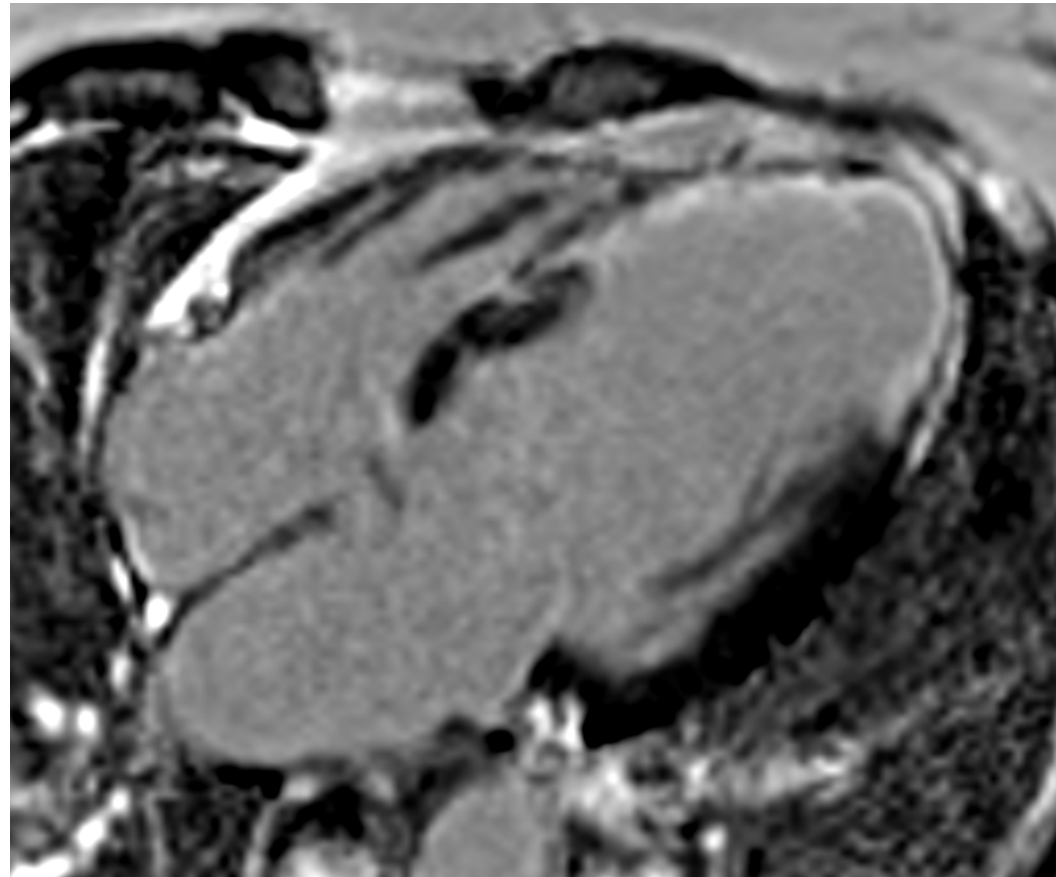
Séquences de perfusion



Séquences de perfusion

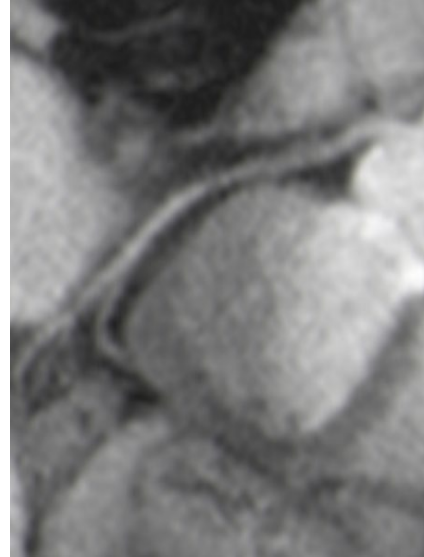
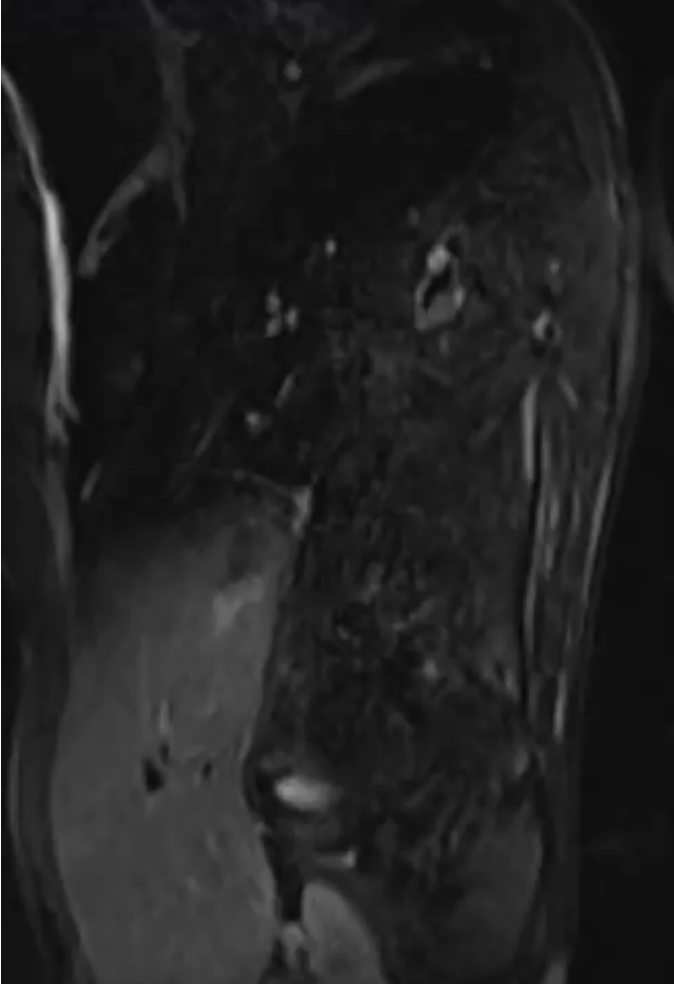


Séquences de rehaussement tardif: LGE late gadolinium enhancement

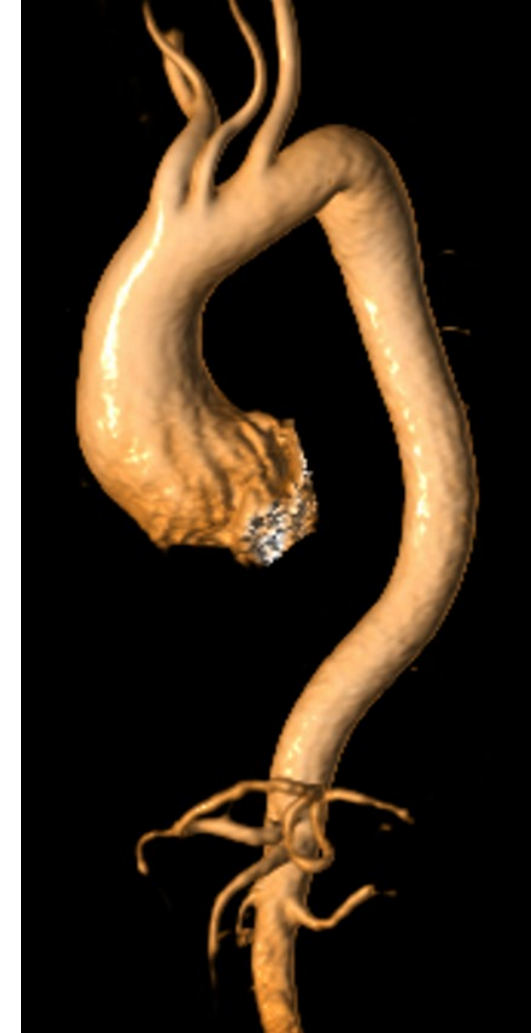


Séquences 3D

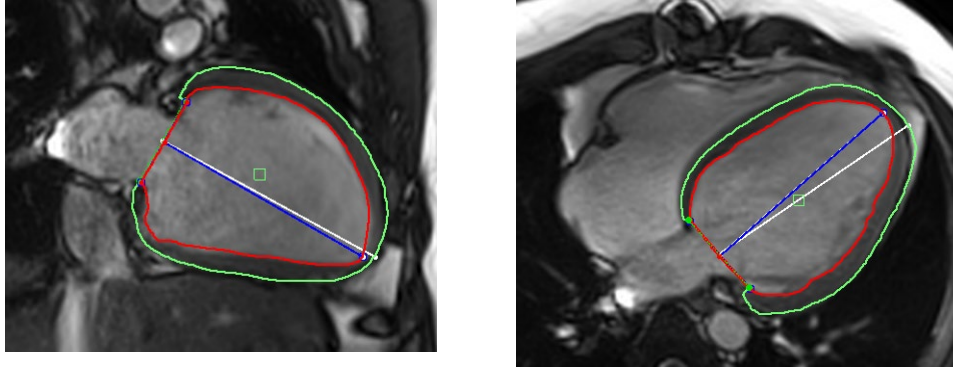
sans gadolinium: *coronaires*,
morphologie gros vaisseaux



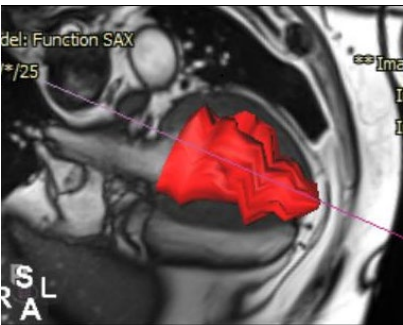
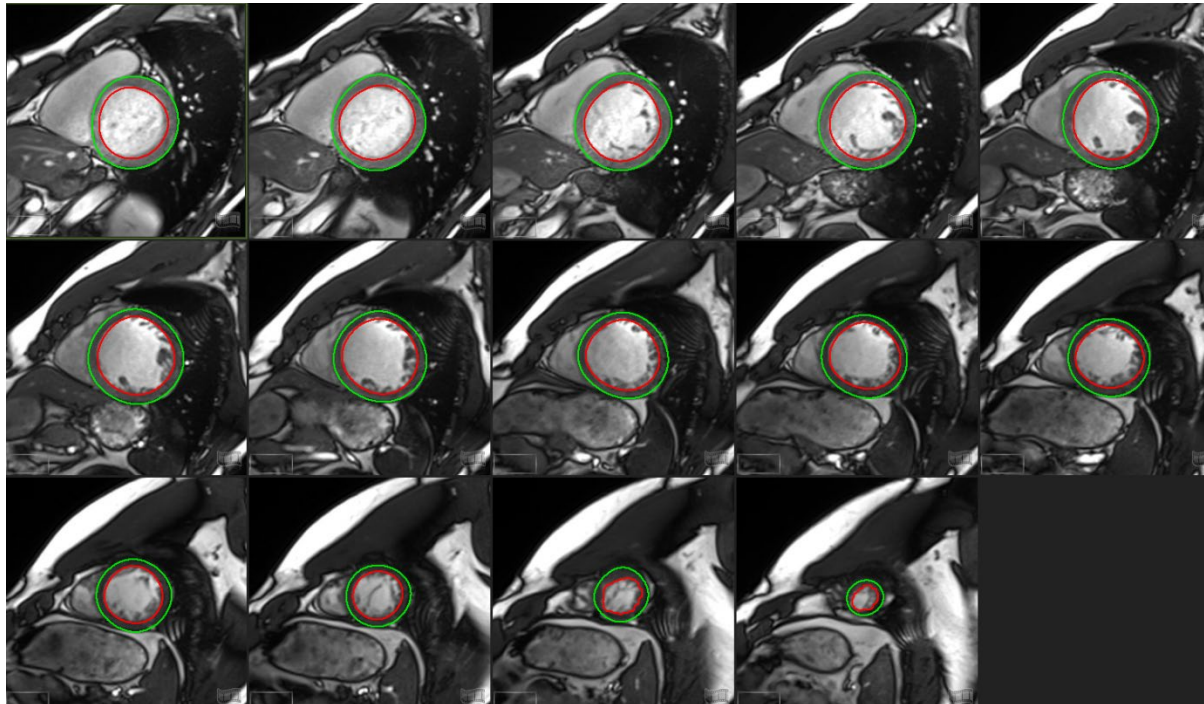
Angiographie avec gadolinium



1)



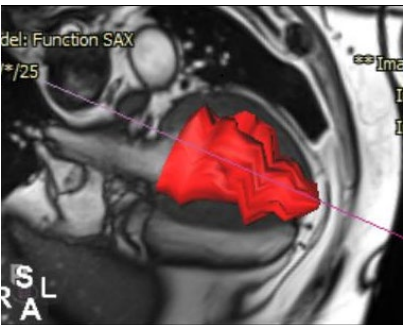
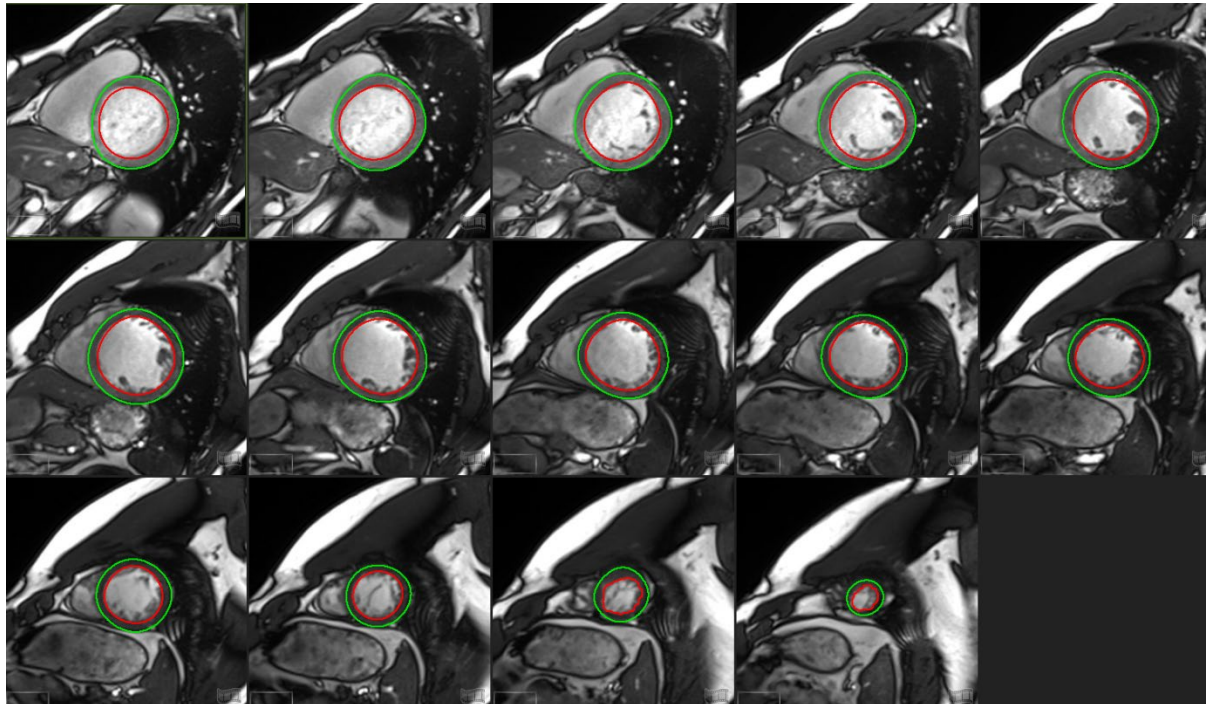
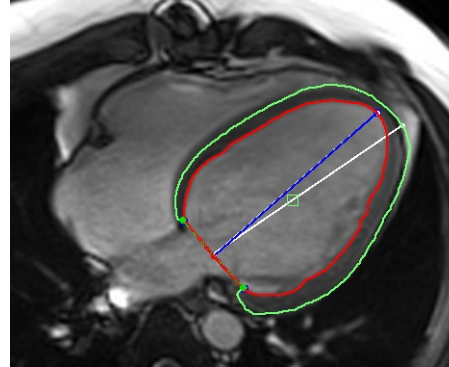
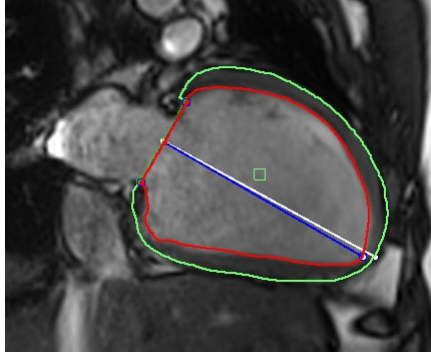
2)



~~IRM~~



VG/VD Volumes, FE, masse



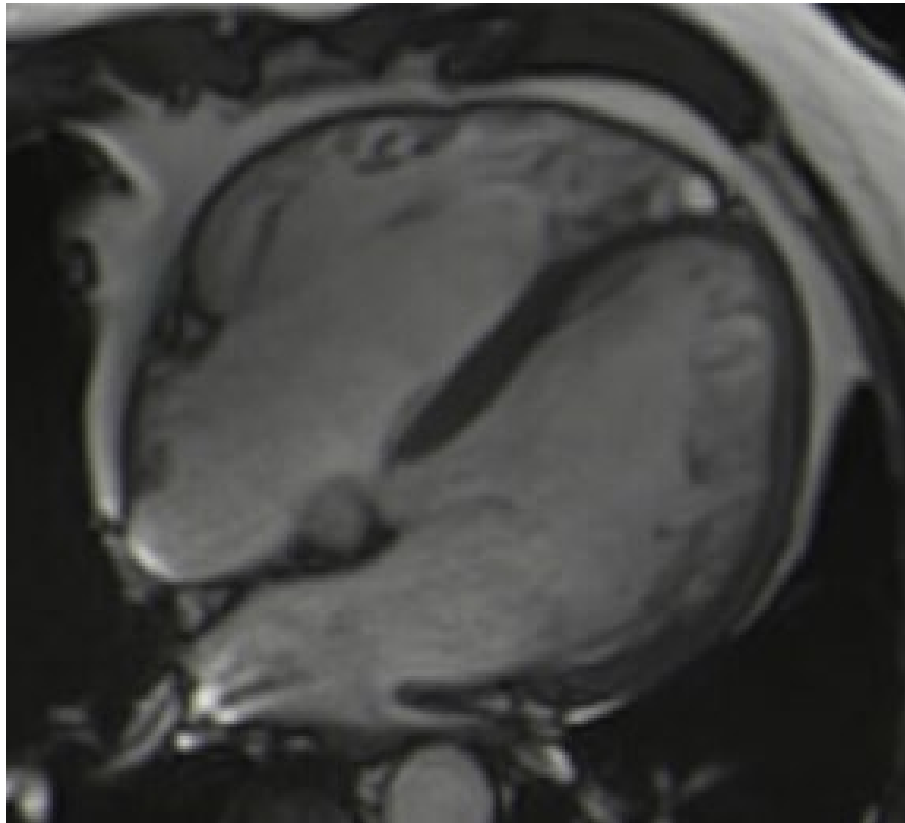
IRM

CT

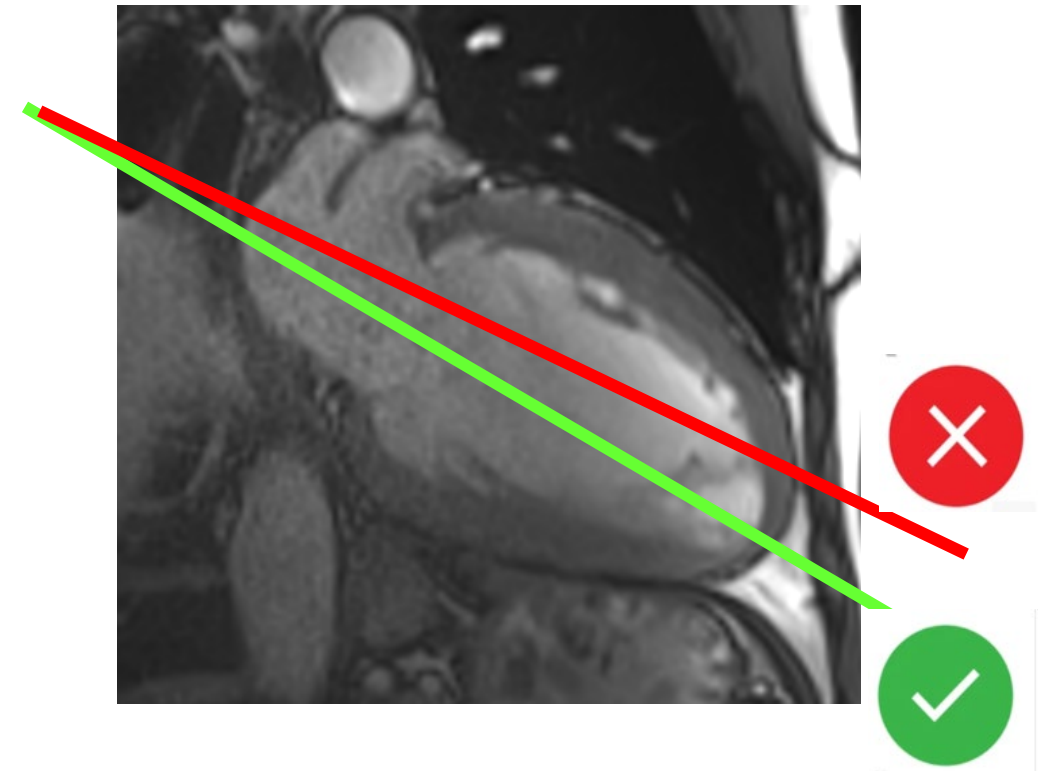


Pourquoi l'écho sous-estime les volumes

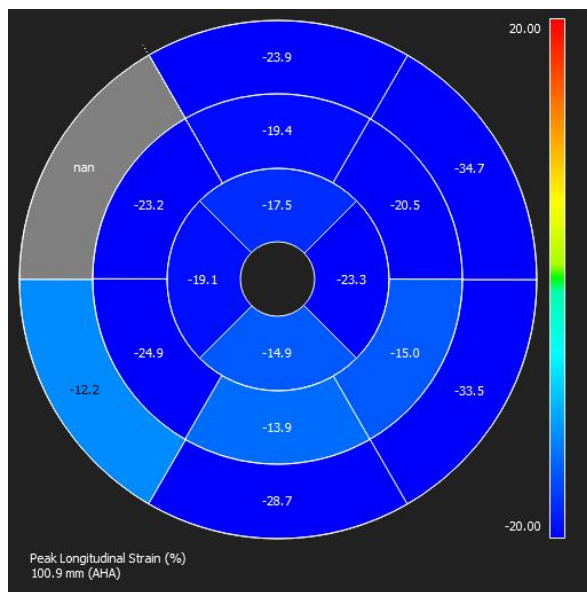
Visualisation endocarde, paroi latérale, apex



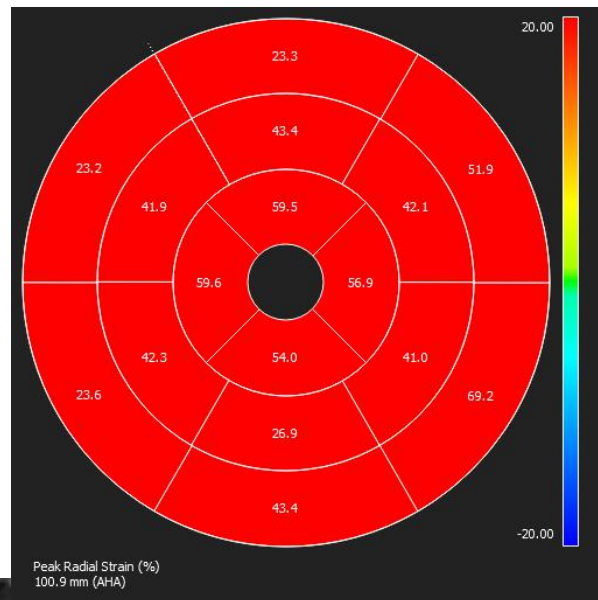
Planification de la coupe



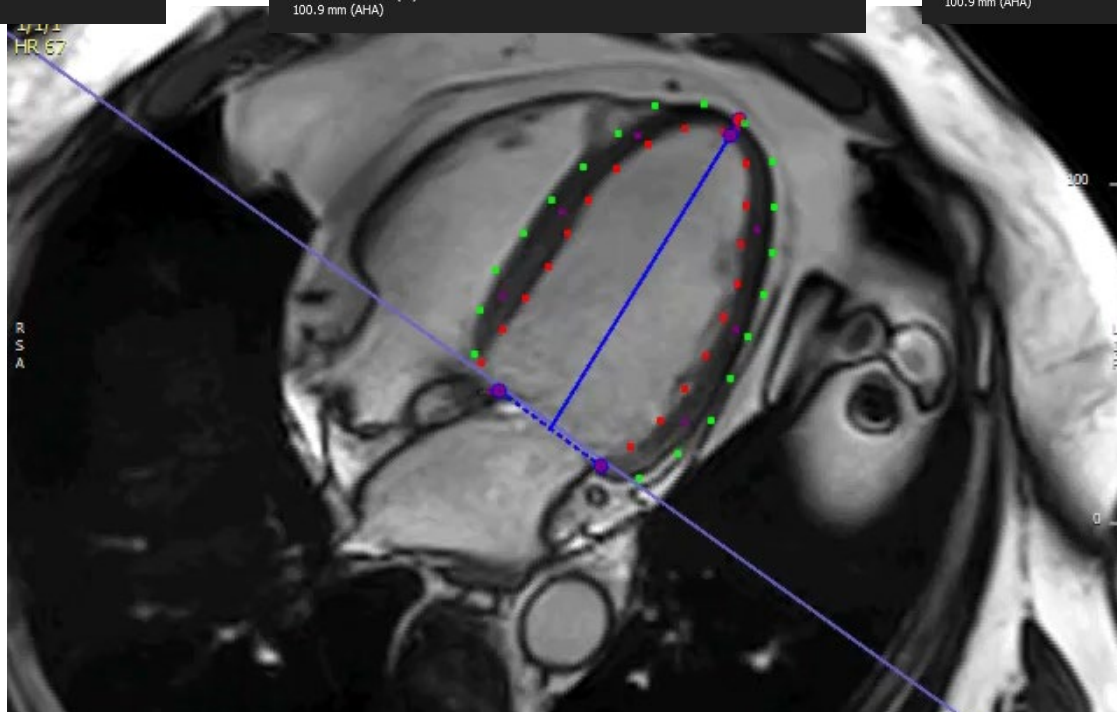
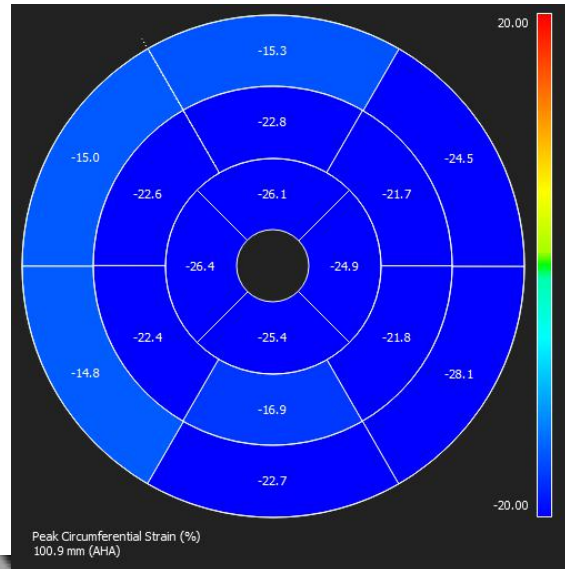
Longitudinal



Radial

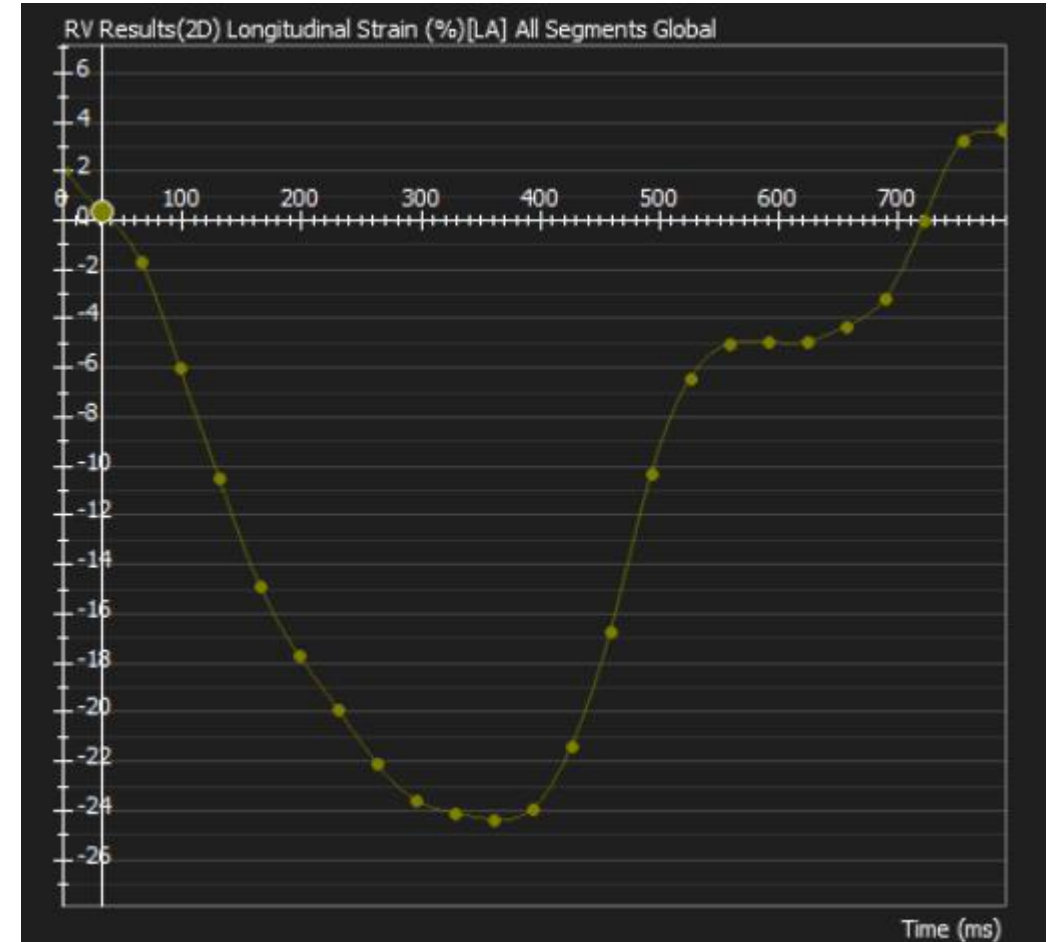
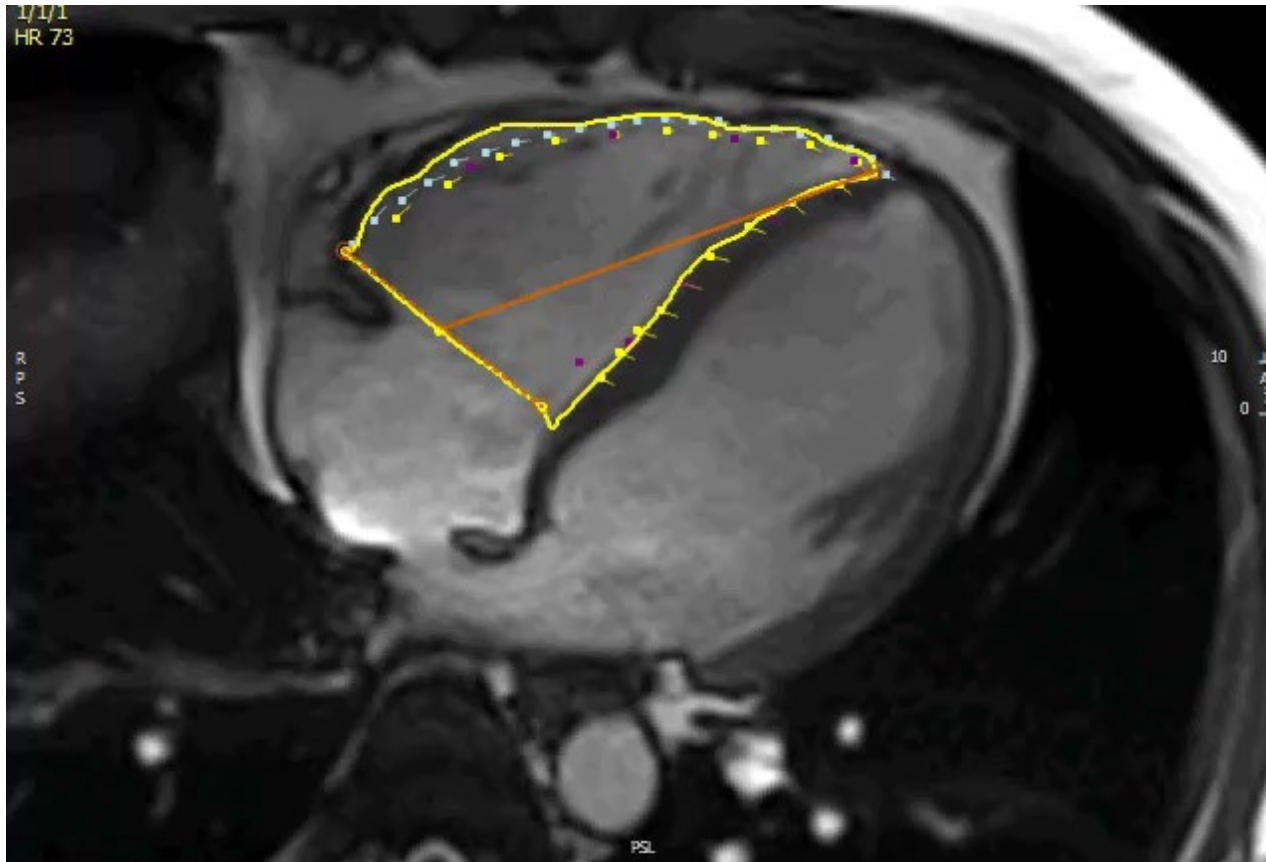


Circonférentiel



Strain
feature
tracking

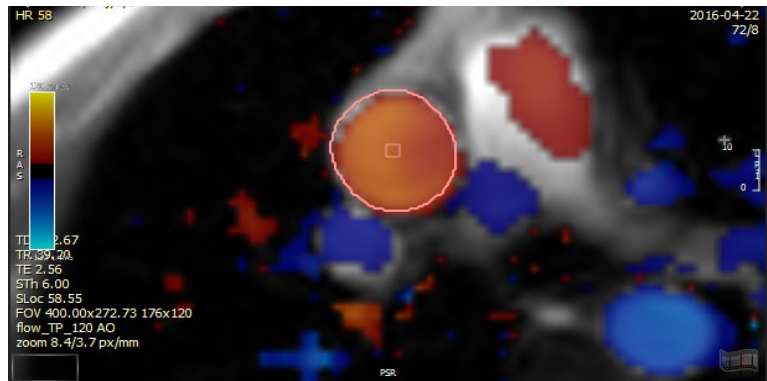
Strain VD



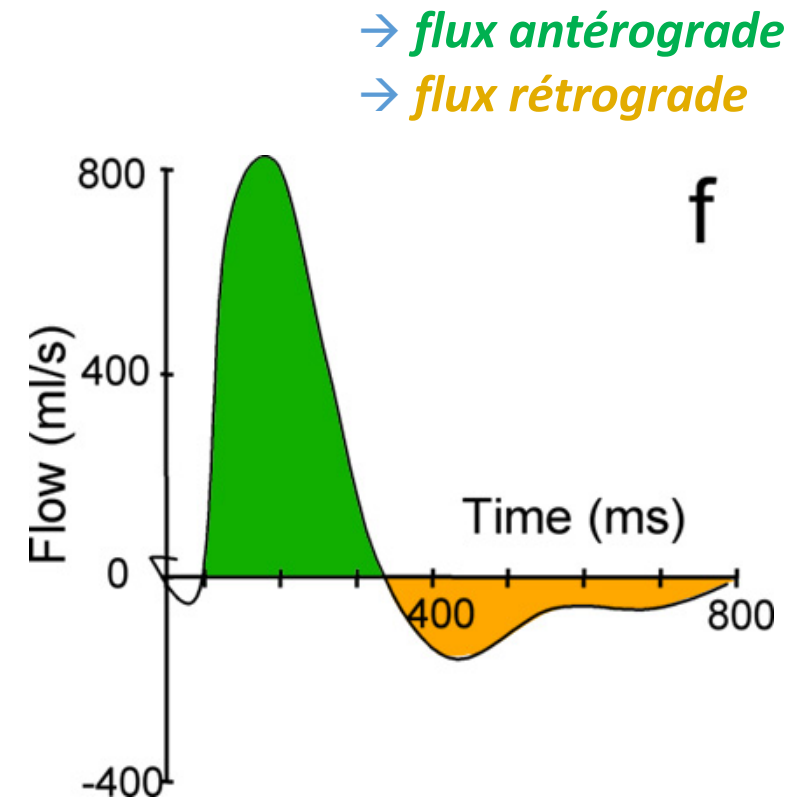
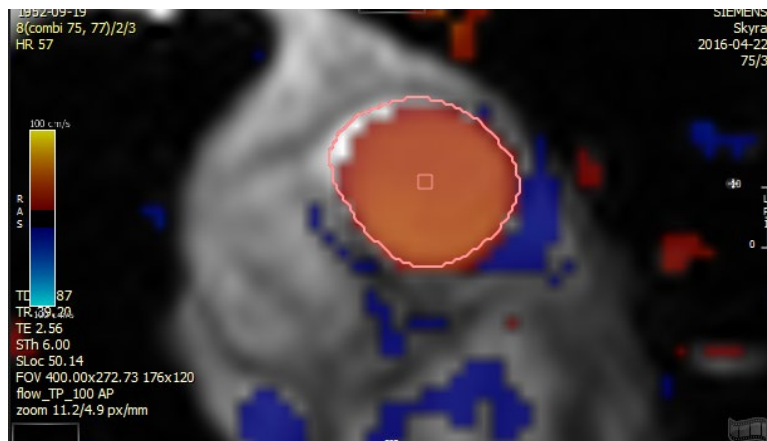
Flux: insuffisance valve aortique / pulmonaire

- Fraction de régurgitation : $\text{flux antérograde} - \text{rétrograde} / \text{antérograde} \%$
- Mesure directe fiable (TAVI 😊)

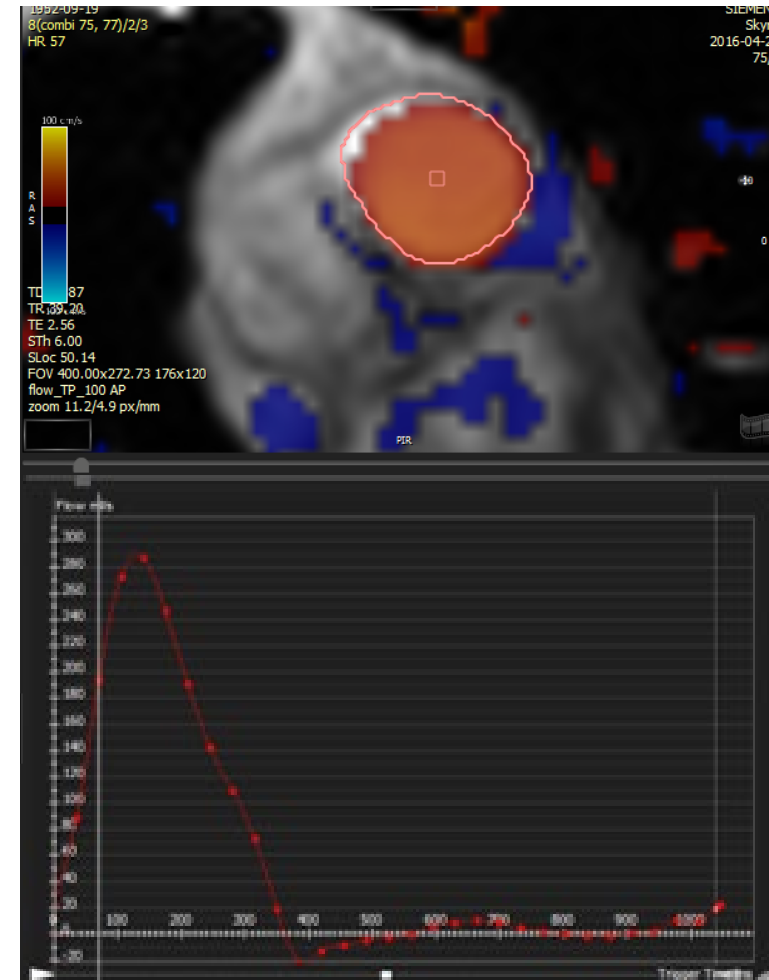
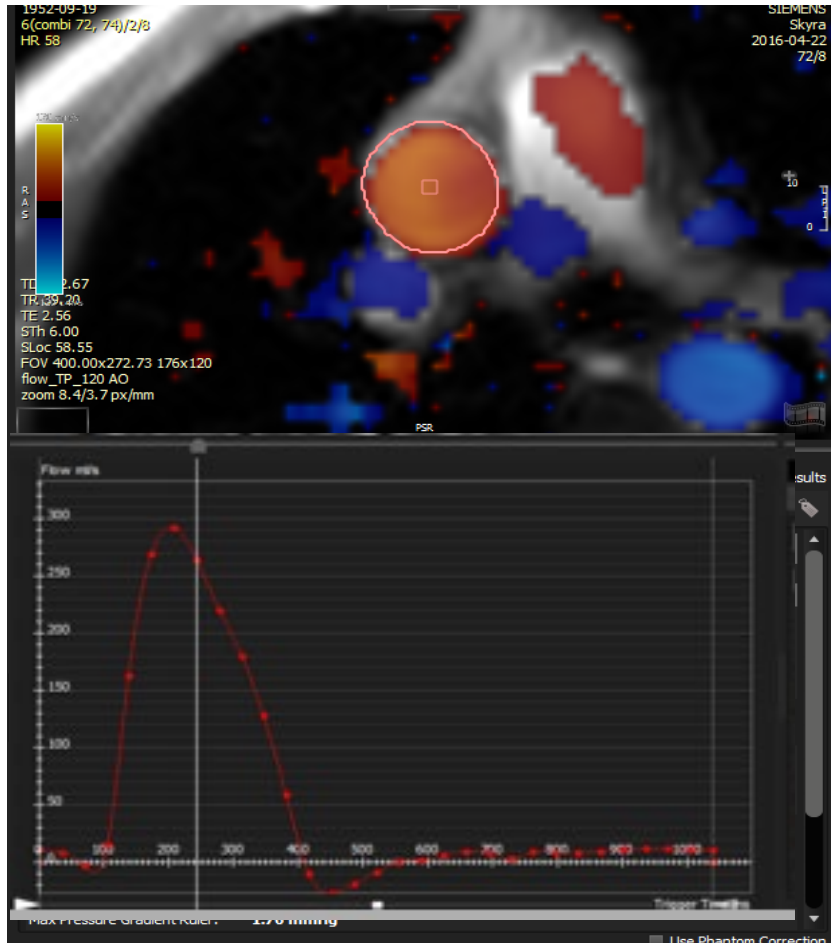
aorte
ascendante



Tronc AP



Qp / Qs

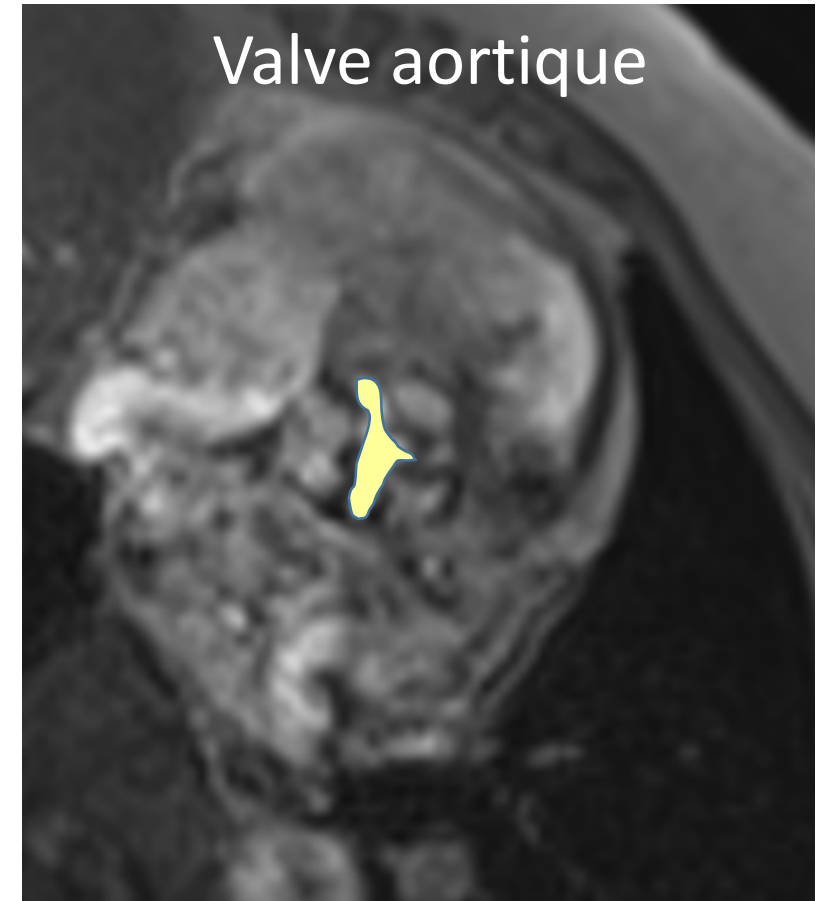
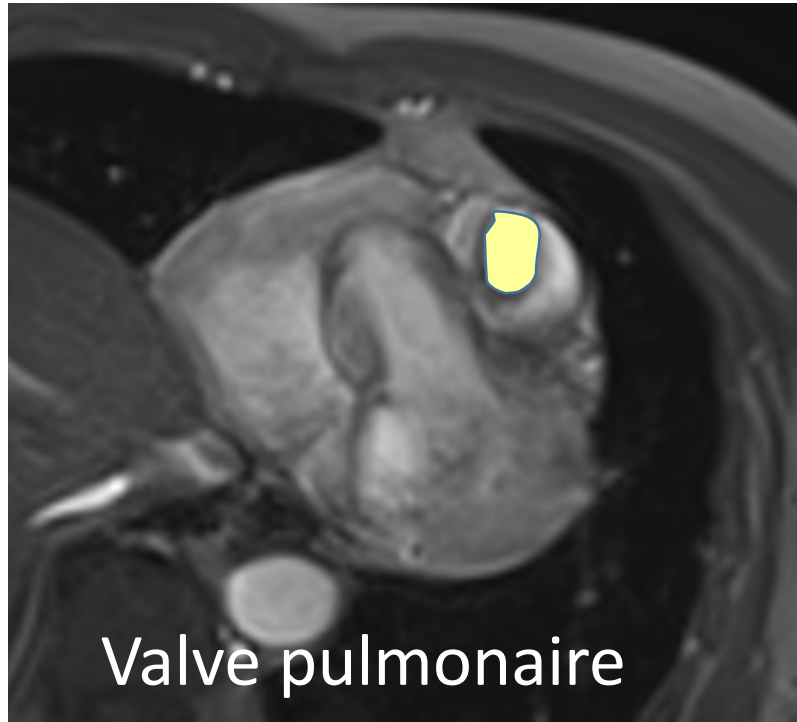


Sténose valve aortique / pulmonaire

- Planimétrie

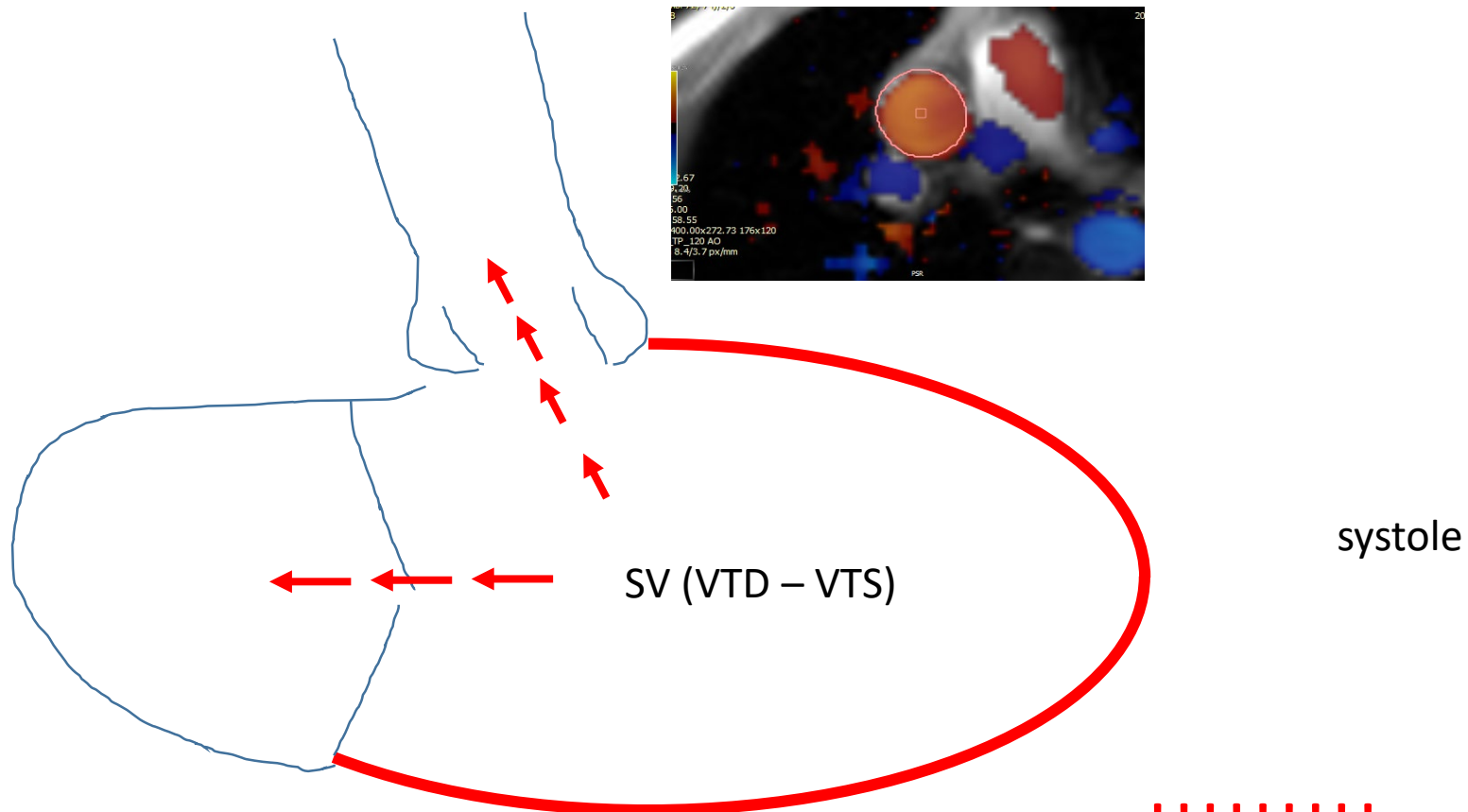


- Gradient



Insuffisance mitrale / tricuspide

- Volume régurgitant = SV (EDV-ESV) – flux antérograde (aorte/pulmonaire)
- Fraction régurgitation = VR/SV ventricule %



!!!!!!! Mesure indirecte : double source d'erreur

Sténose mitrale / tricuspideenne

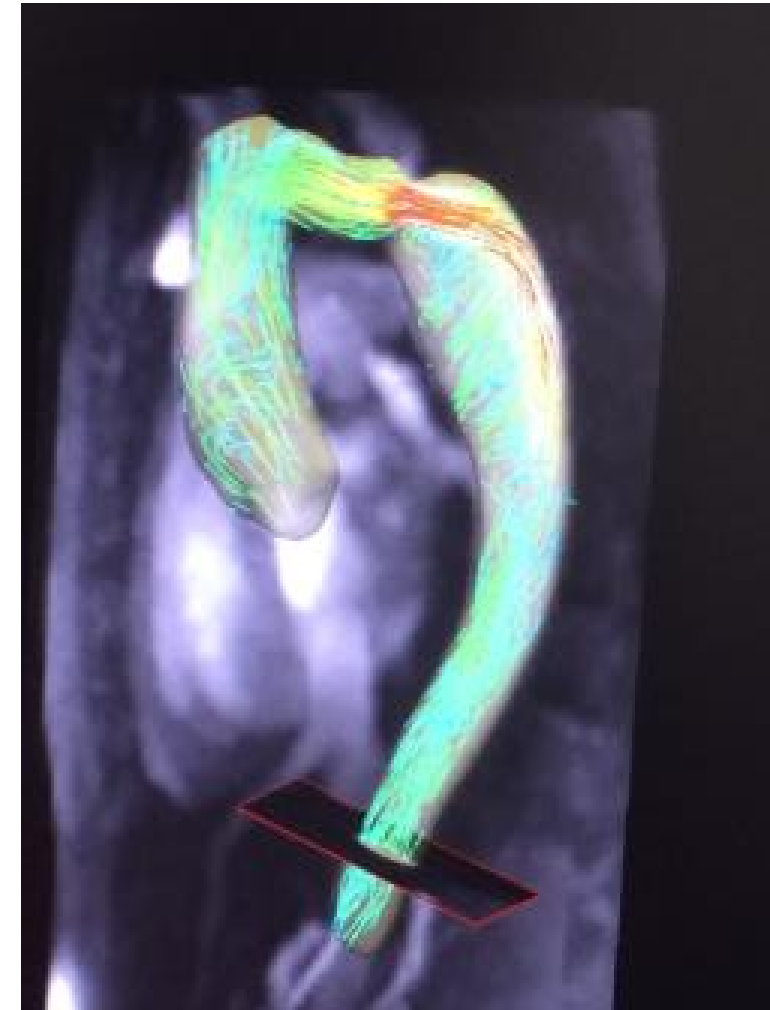
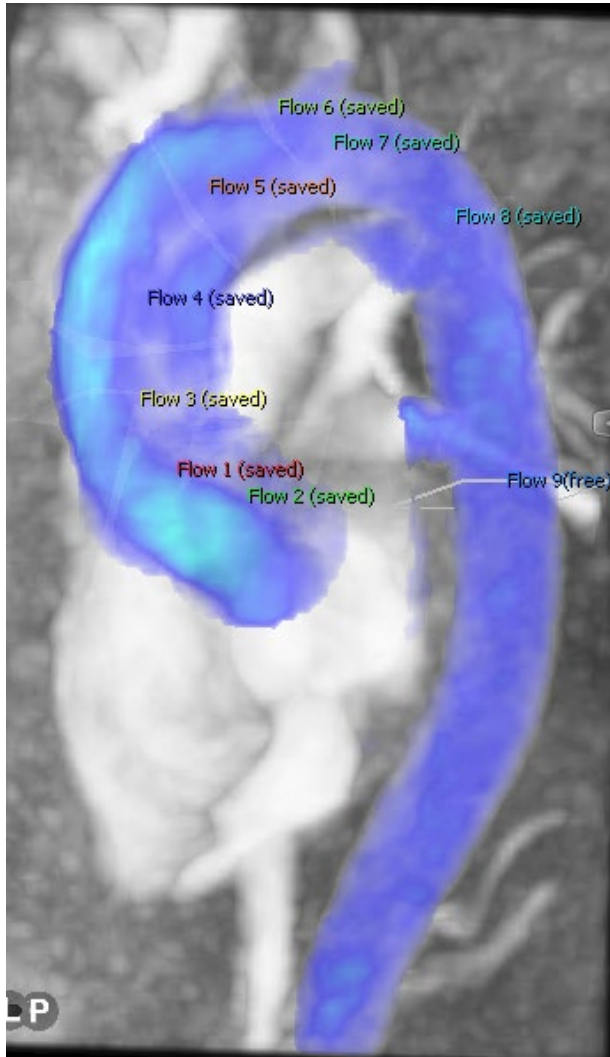
- Surface anatomique



- Gradient non fiable



Flux 4D



Wall shear stress

Intérêt de l'IRM dans les valvulopathie

	IM / IT	SM / ST	lao / IP	Rao / RP
sévérité	++	? (planimétrie)	+++ 	+++ (planimétrie) 
Retentissement ventriculaire	+++ (volumes) 	-	+++ (volumes) 	+++ (HVG, fibrose) 
aorte thoracique / Artère pulmonaire			+++ 	+++ 
Mécanisme	+	+	+	+++ 

Quantification de l'IM et Iao en IRM

Table 3 | Recommended grading of MR by CMR assessment

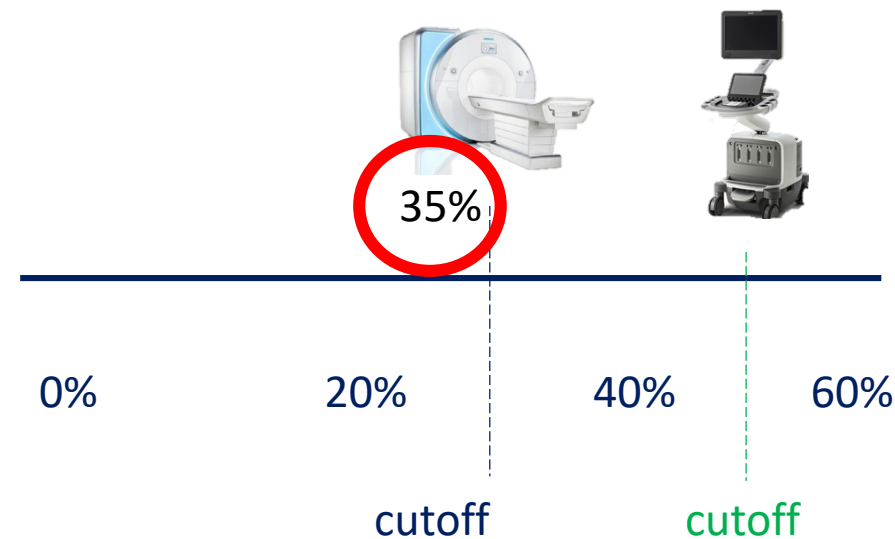
Type of MR	Grading of severity			
	Mild	Moderate	Severe	Very severe
Primary	$MR_{RF} < 20\%^a$	$MR_{RF} = 20-39\%^a$	$MR_{RF} 40-50\%$; $MR_{vol} > 55-60\text{ml}$	$MR_{RF} > 50\%$
Secondary	$MR_{vol} < 30\text{ml}$	$MR_{vol} = 30-60\text{ml}$	$MR_{vol} \geq 60\text{ml}$	—

From REFS^{12,13,19}. CMR, cardiovascular magnetic resonance imaging; MR, mitral regurgitation; MR_{RF} , mitral regurgitation fraction; MR_{vol} , mitral regurgitation volume. ^aNot study-based; mainly expert opinion.

Nat Rev Cardiol 2020 May;17(5):298-312

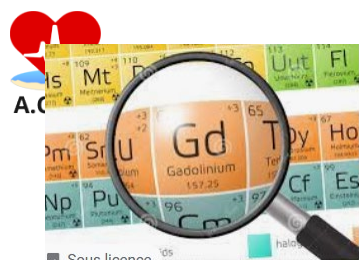
Regurgitant fraction

IAo



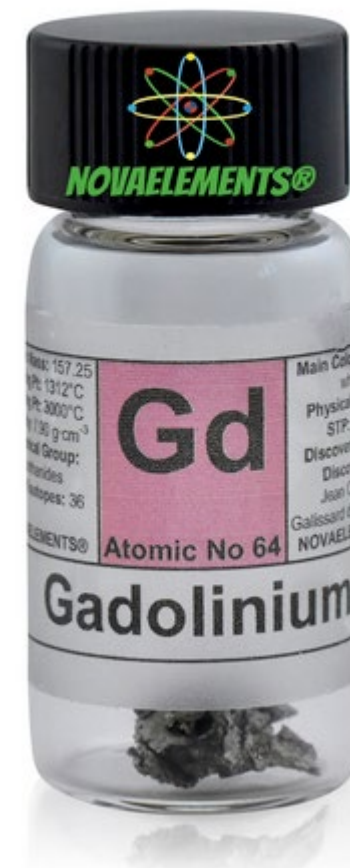
NON indication à l'IRM

- **Valve**: sténose mitrale/tricuspidienne, endocardite, thrombus valvulaire, évaluation sténose prothèse mécanique, fuite para valvulaire, abcès
- Recherche **thrombus** auricule gauche



Chélates de gadolinium

- Produit de contraste **extravasculaire** et **extracellulaire**
- Diffusion rapide dans l'interstitium



- **Contre-indications**

- Allergie 🤔
- Grossesse (contre-indication relative par principe de précaution, à apprécier en fonction du rapport bénéfice/risques)

- **Effets secondaires**

- Allergie
- Nausées
- Céphalées
- Altération du goût
- Paresthésies
- Allergie



- **Fibrose Systémique Néphrogénique (Dermopathie Fibrosante Néphrogénique)**

Atteinte fibrosante cutanée avec une extension viscérale possible dans les jours ou mois (2-9 mois) suivants une injection de chélates de Gadolinium chez les patients porteurs d'une **insuffisance rénale sévère** (ou hépatique).



Annexe

Recommandations de l'agence européenne visant à limiter les risques de fibrose néphrogénique systémique liée à l'administration de produits de contraste à base de sels de gadolinium

Produit de contraste à base de sel de gadolinium présentant un risque faible (Gadovist[®], Prohance[®], Dotarem[®])
et un risque modéré (Multihance[®]) de FNS

- Un bilan rénal complet est recommandé avant tout examen, en particulier chez les patients âgés de plus de 65 ans.
- La plus petite dose peut être administrée chez les patients présentant une insuffisance rénale sévère (clairance de la créatinine inférieure à 30 ml/min), chez les patients en attente ou ayant reçu récemment une transplantation hépatique, chez les nouveau-nés et chez les enfants. La réadministration devra être espacée d'au moins 7 jours.
- La décision de continuer ou d'arrêter l'allaitement pendant 24 heures doit être prise au cas par cas.
- L'utilisation pendant la grossesse n'est pas recommandée sauf si le tableau clinique l'exige.

Gadolinium storage condition

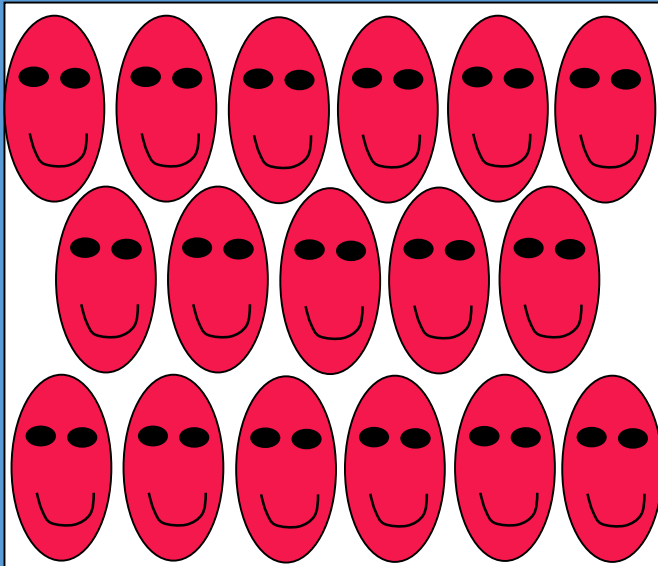
fibrosis. Findings have been reported regarding gadolinium deposition within the body and various reports of patients who report suffering from acute and chronic symptoms secondary to GBCA's exposure. At the present state of knowledge, it has been proved that gadolinium deposits also occur in the brain, irrespective of renal function and GBCAs stability class. To date, no definitive clinical findings are associated with gadolinium deposition in brain tissue. Gadolinium deposition disease is a newly described and probably infrequent entity. Patients presenting with gadolinium deposition disease may show signs and symptoms that somewhat follows a pattern similar but not identical, and also less severe, to those observed in nephrogenic systemic fibrosis. In this review, we will address

- Sensation de douleur brulante
- Picotement
- Epaississement de la peau et décoloration
- Douleur osseuse
- Rigidité articulations
- Spasme musculaire
- Gout métallique
- Sensation de « brain fog »

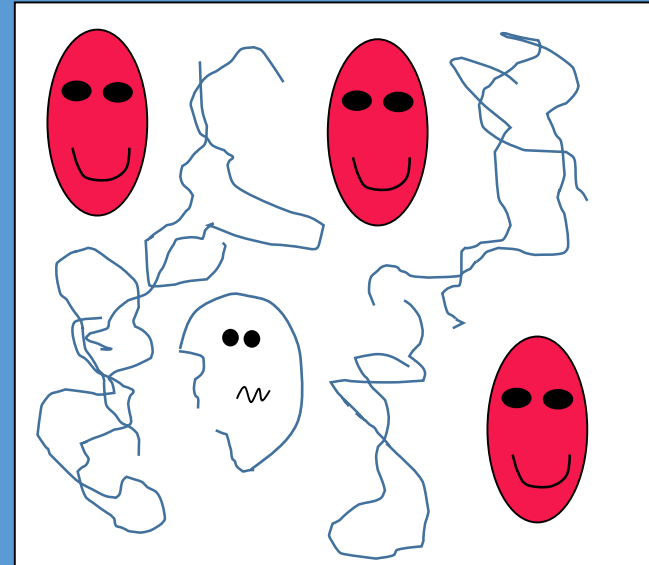
Gadolinium deposition disease

Pourquoi le processus pathologique (œdème, infiltration, fibrose) est blanc sur les images de rehaussement tardif (LGE) ?

myocarde normal

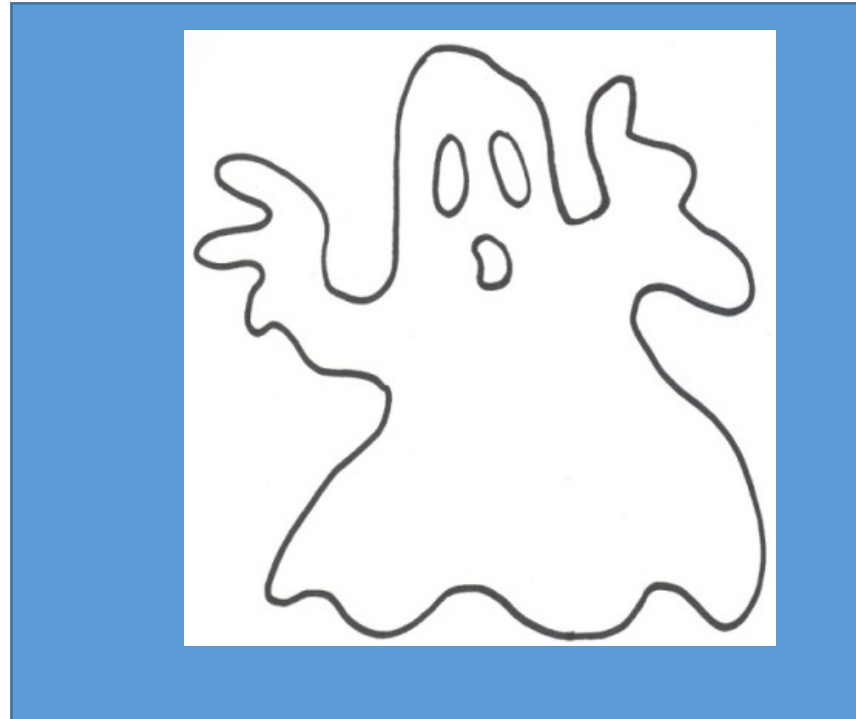


Infarctus, œdème,
infiltration (ex. amylose)



ECV 25%

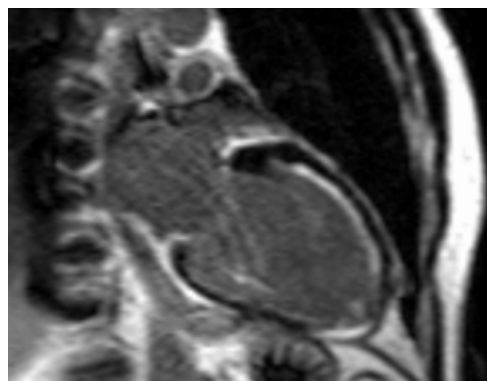
LGE “what is white is dead/bad”



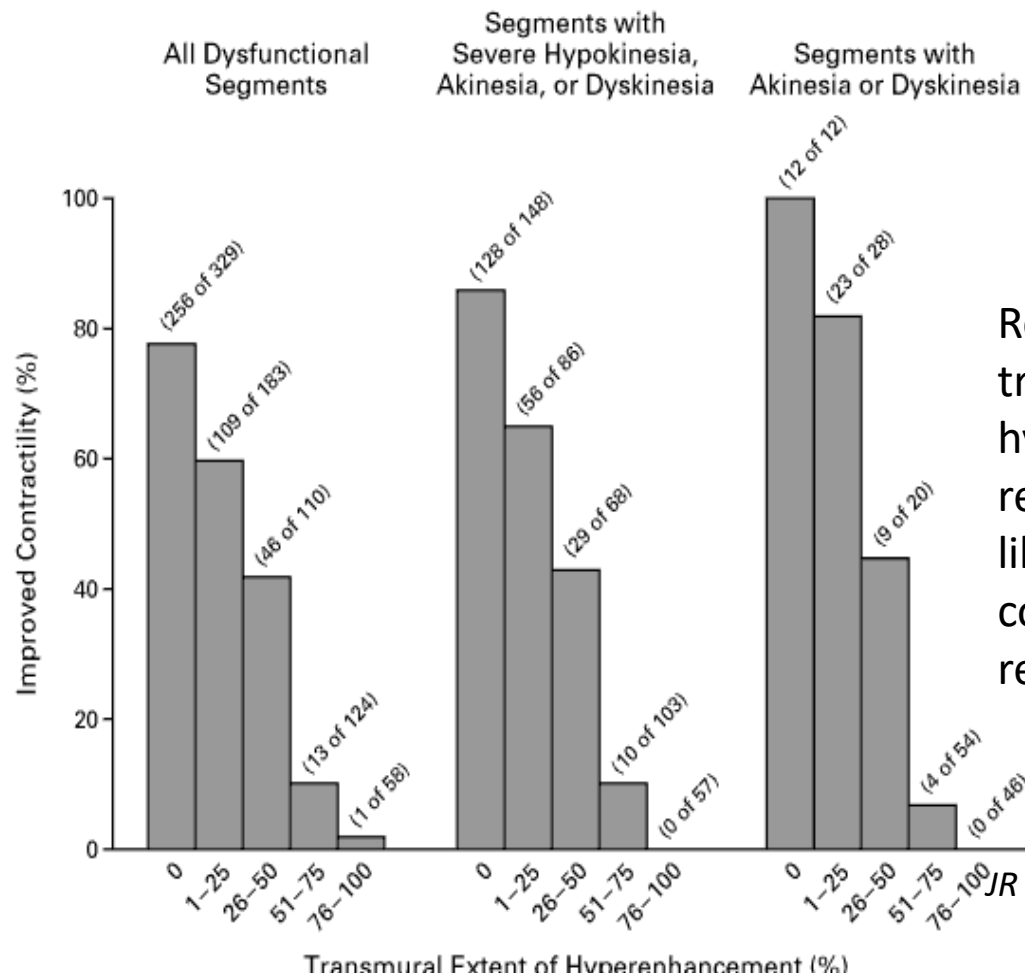
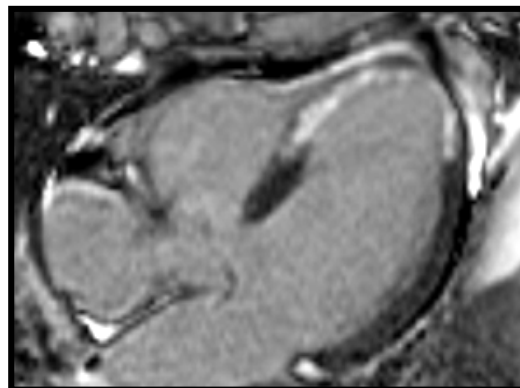
LGE distribution coronaires

LGE part tjrs de l'endocarde!

IDM sous-endocardique

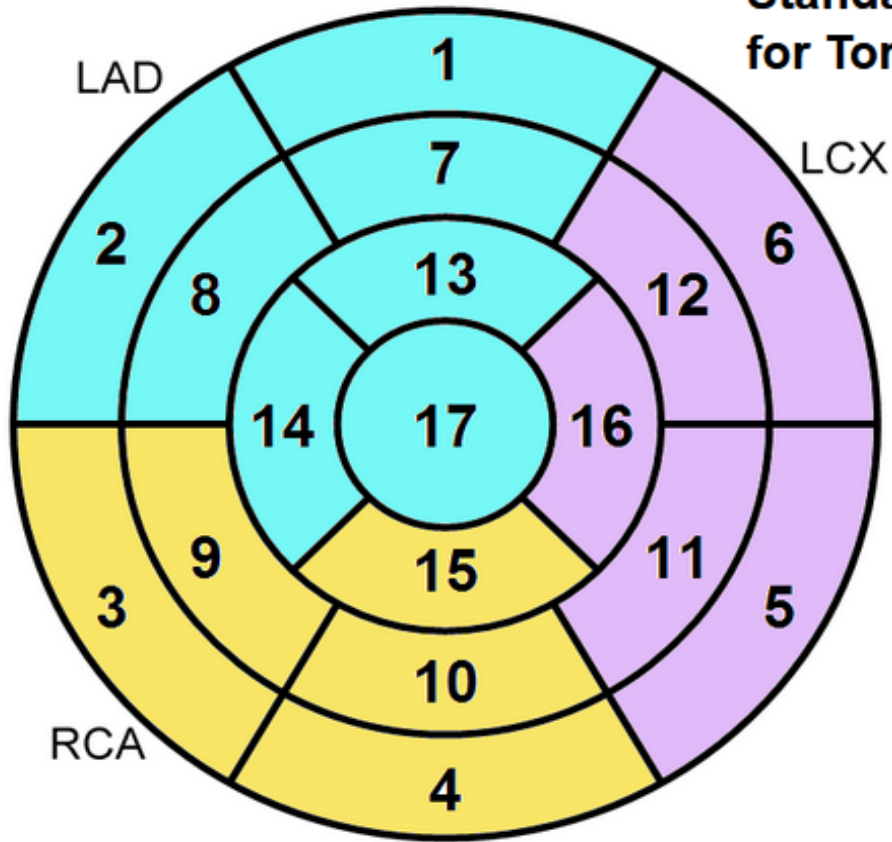


IDM transmural

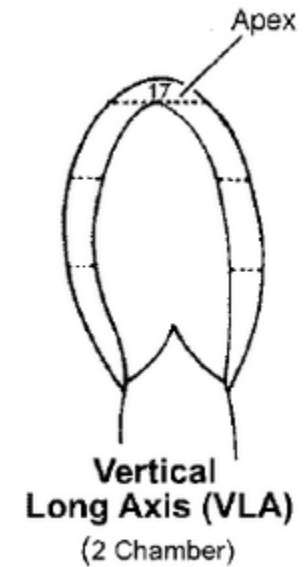
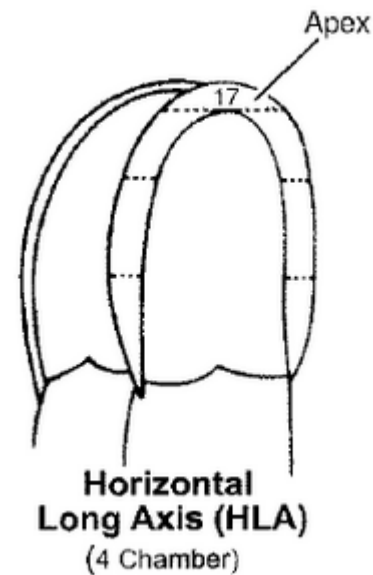


AHA SCIENTIFIC STATEMENT

Standardized Myocardial Segmentation and Nomenclature for Tomographic Imaging of the Heart



17-Segment Model (AHA)



Patterns LGE: non ischémique

Medio ventriculaire



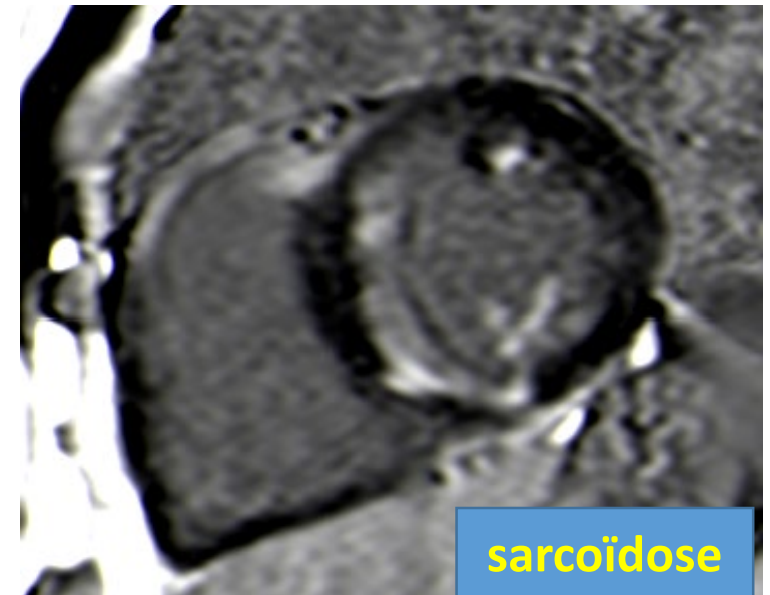
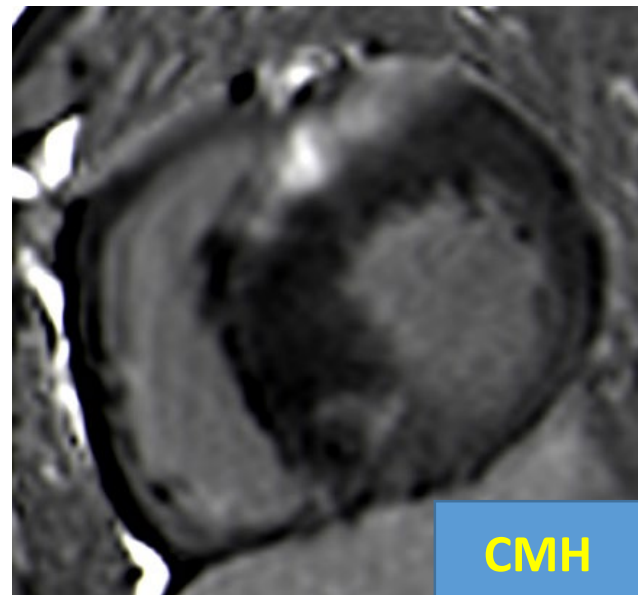
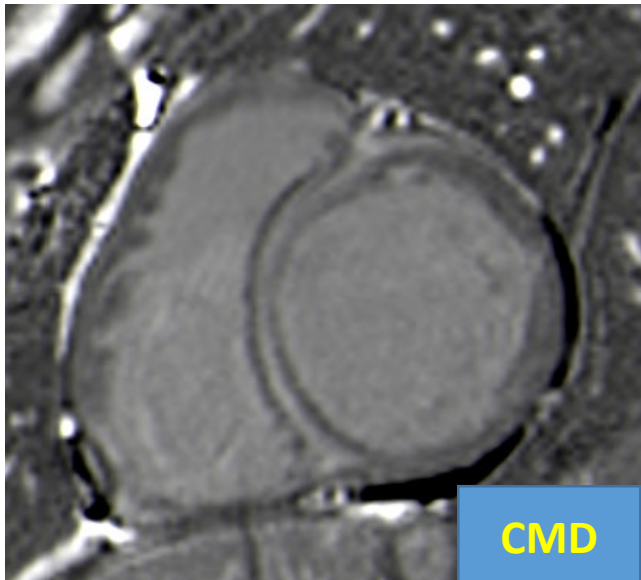
- Idiopathic dilated cardiomyopathy
- Myocarditis



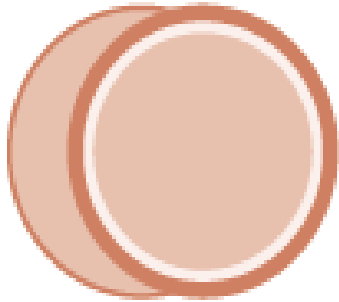
- Hypertrophic cardiomyopathy
- Right ventricular pressure overload (e.g. congenital heart disease, pulmonary HTN)



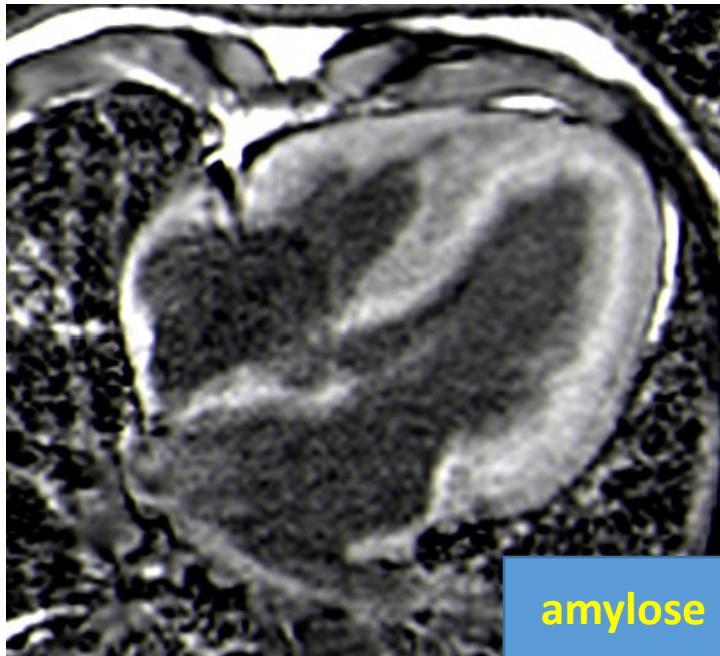
- Sarcoidosis
- Myocarditis
- Anderson-Fabry
- Chagas disease



Circonférentiel

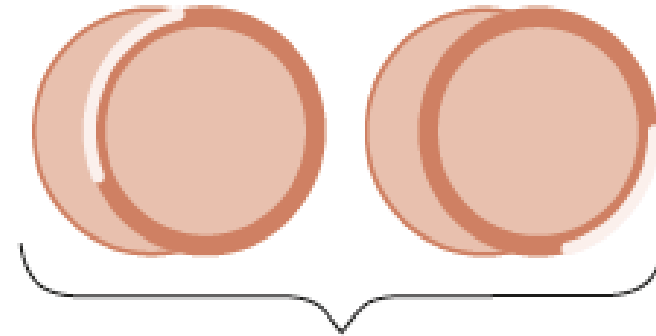


- Amyloidosis, systemic sclerosis, post cardiac transplantation

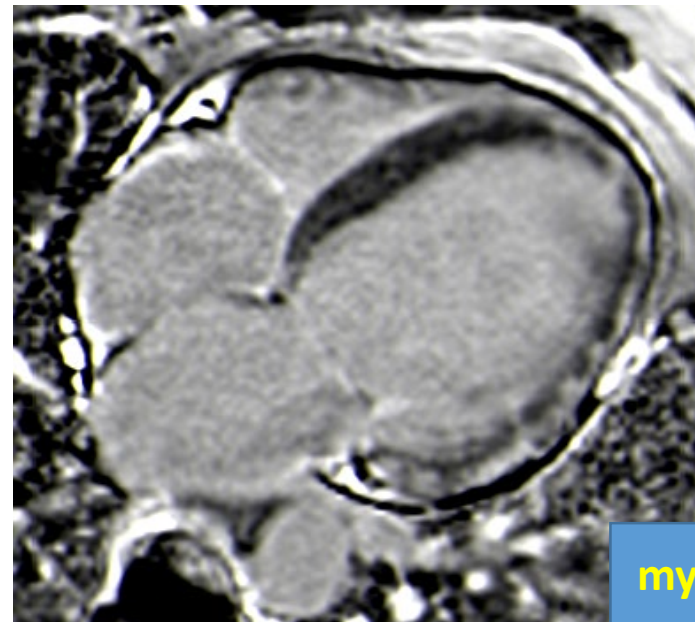


amylose

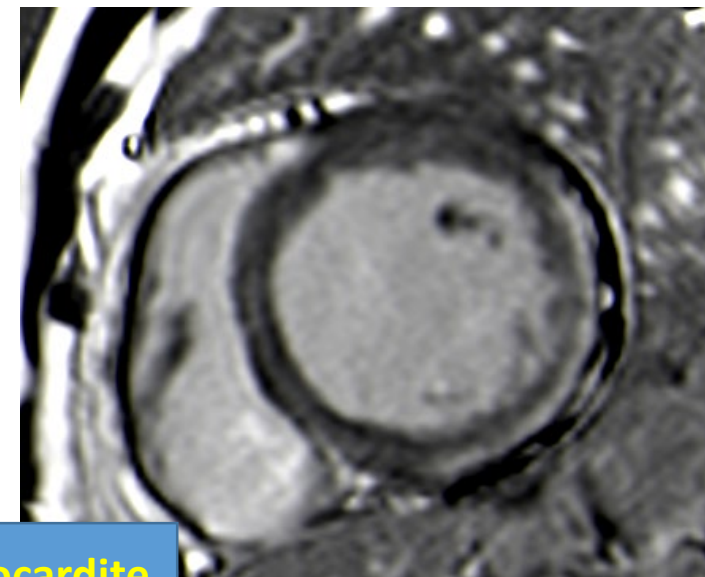
Sous-epicardique



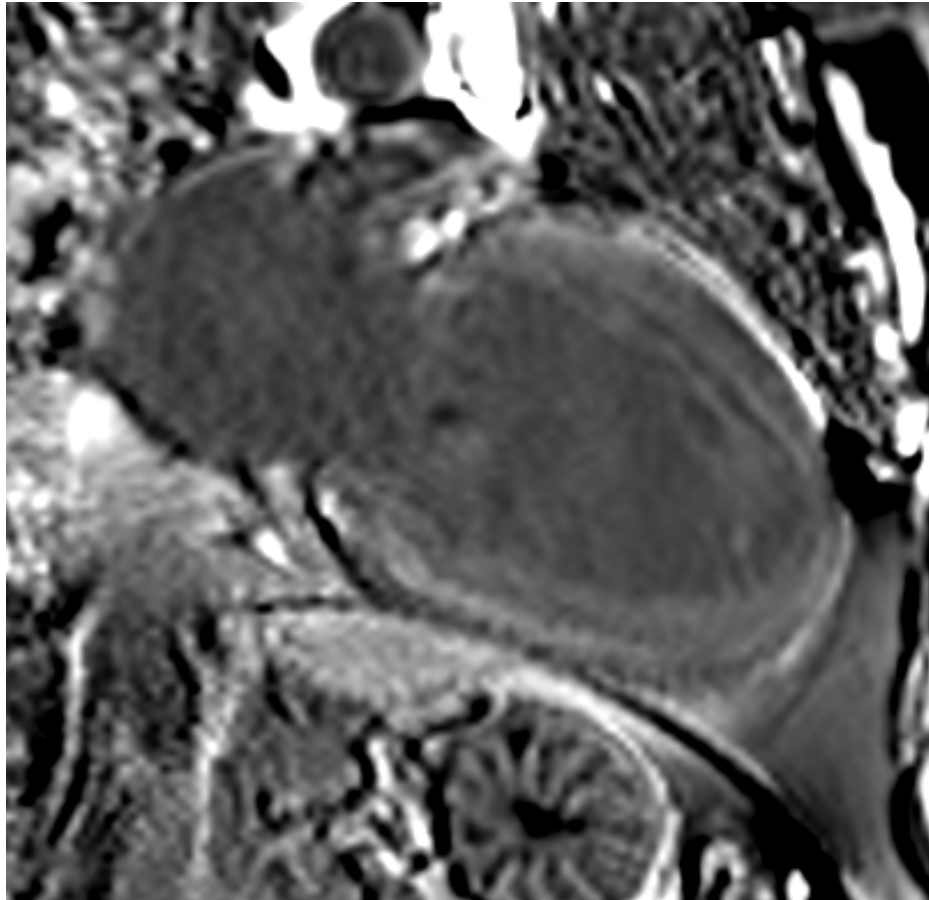
- Sarcoidosis, myocarditis, Anderson-Fabry, Chagas disease



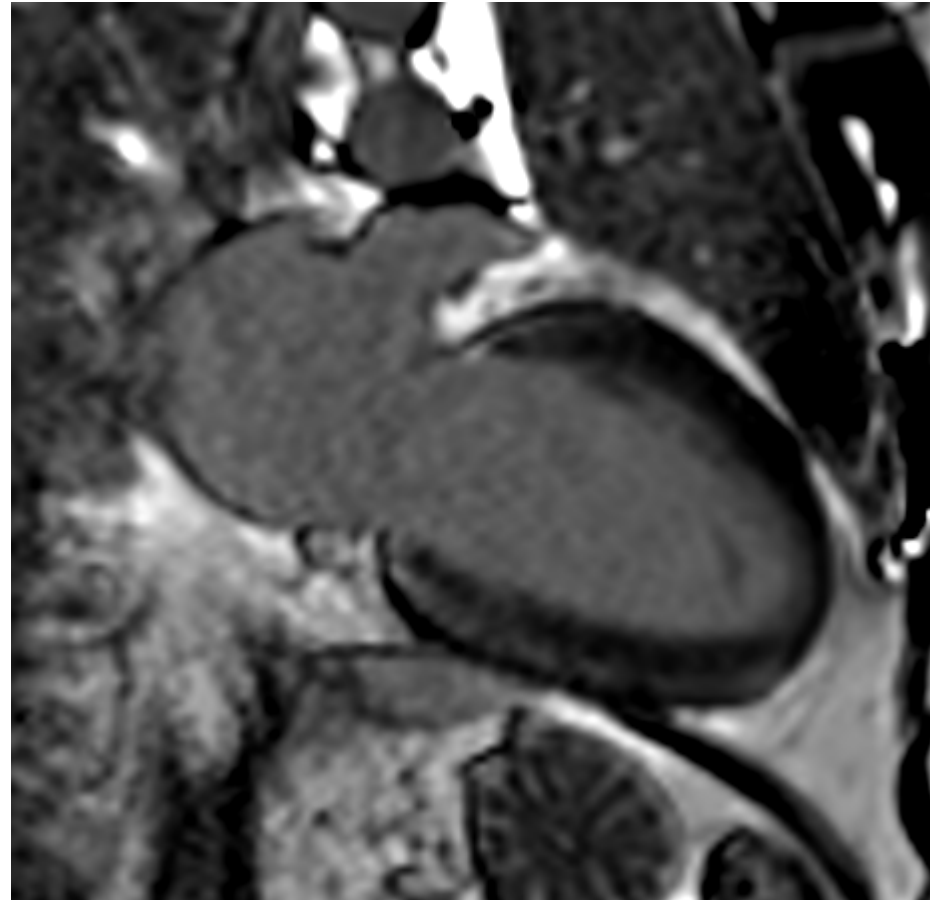
myocardite



Amylose?



Non, mauvais TI !!!!



Fibrose facteur pronostique indépendant dans nombreuses pathologies, au-delà de FE

The Prognostic Value of Late Gadolinium-Enhanced Cardiac Magnetic Resonance Imaging in Nonischemic Dilated Cardiomyopathy: A Review and Meta-Analysis

doi: 10.1016/j.jcmg.2018.03.006.

Prognostic Value of LGE-CMR in HCM: A Meta-Analysis

doi: 10.1016/j.jcmg.2016.02.031.

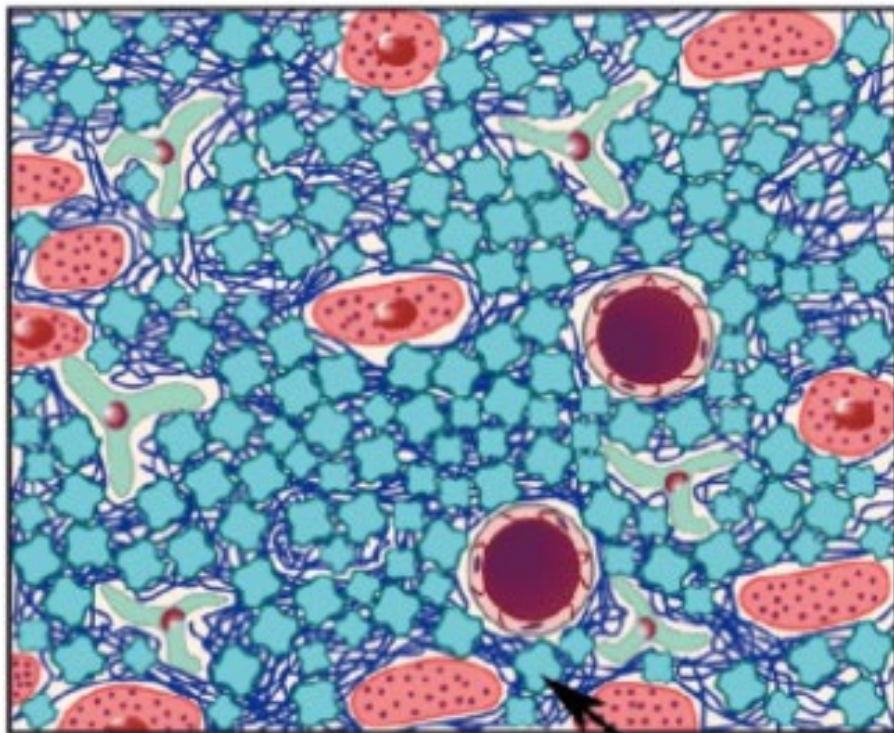
Irréversible



Réversible



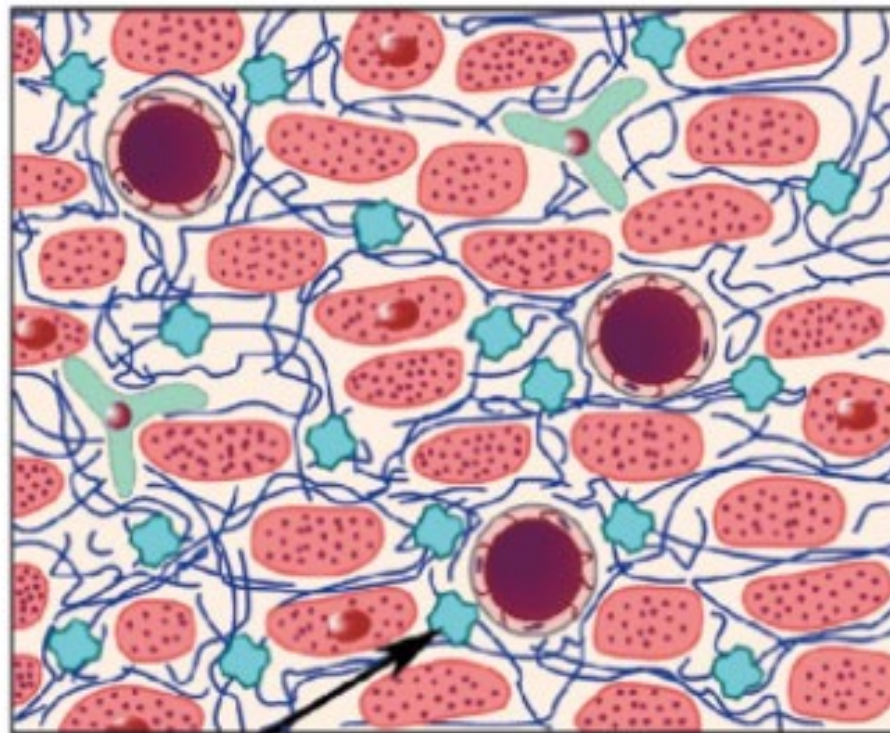
A. Replacement fibrosis



LGE

Gadolinium

B. Reactive interstitial fibrosis



Mapping T1

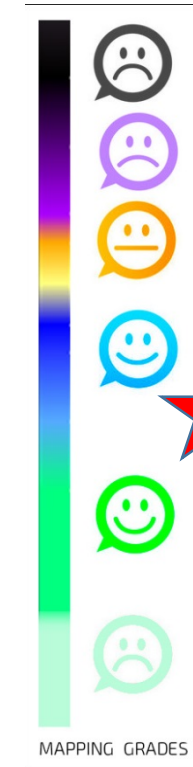
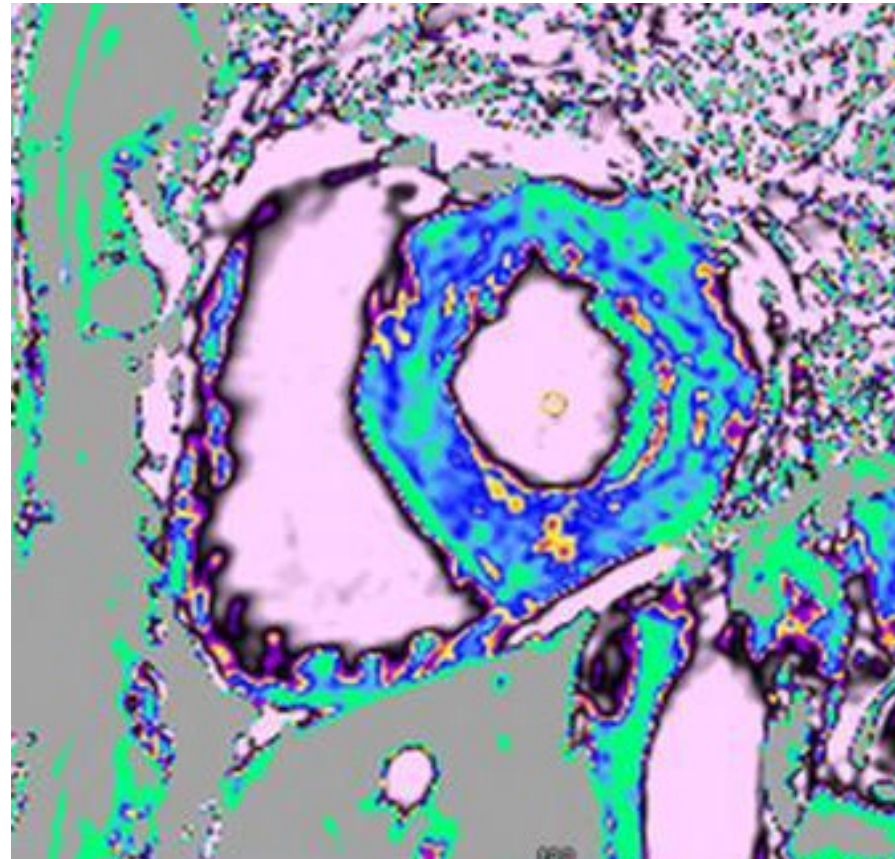
Différence de contraste entre le territoire sain et le territoire pathologique: LGE

Si tout le myocarde est pathologique????? → Mapping

mapping

- images paramétriques basées sur les temps de relaxation (T1, T2)

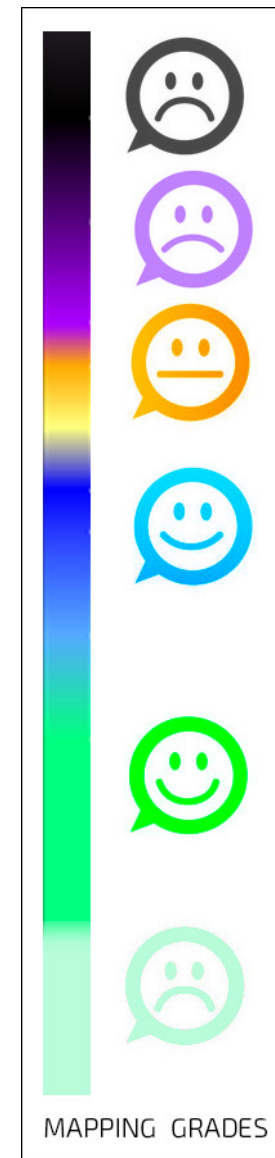
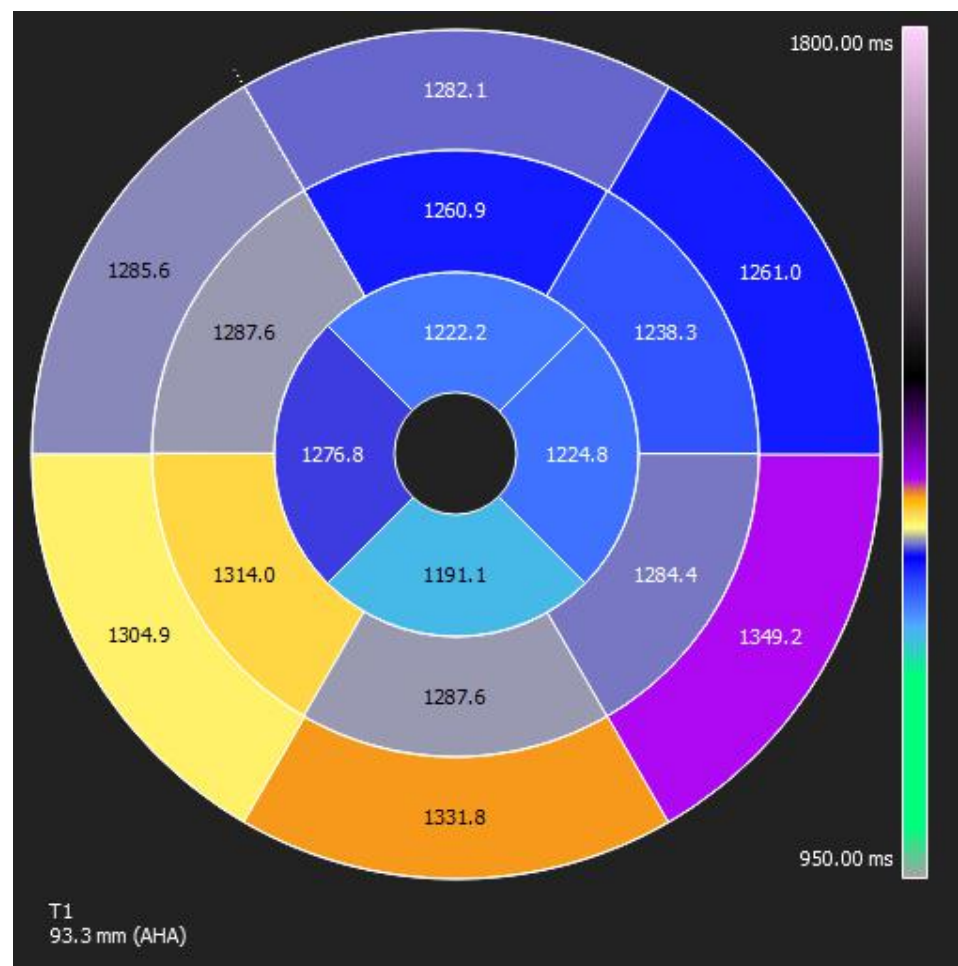
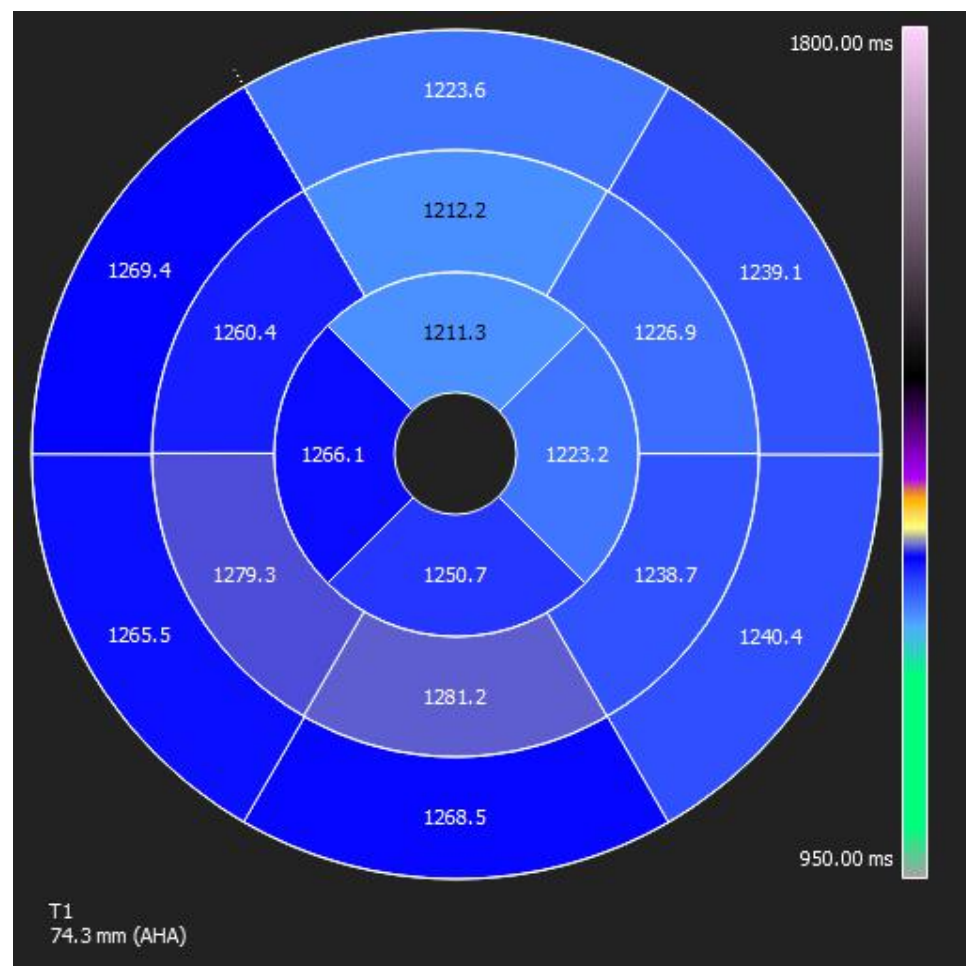
Mapping T1



Range normale (3T):
1150-1250 ms

mapping T1

Nle 1150-1250 ms à 3T



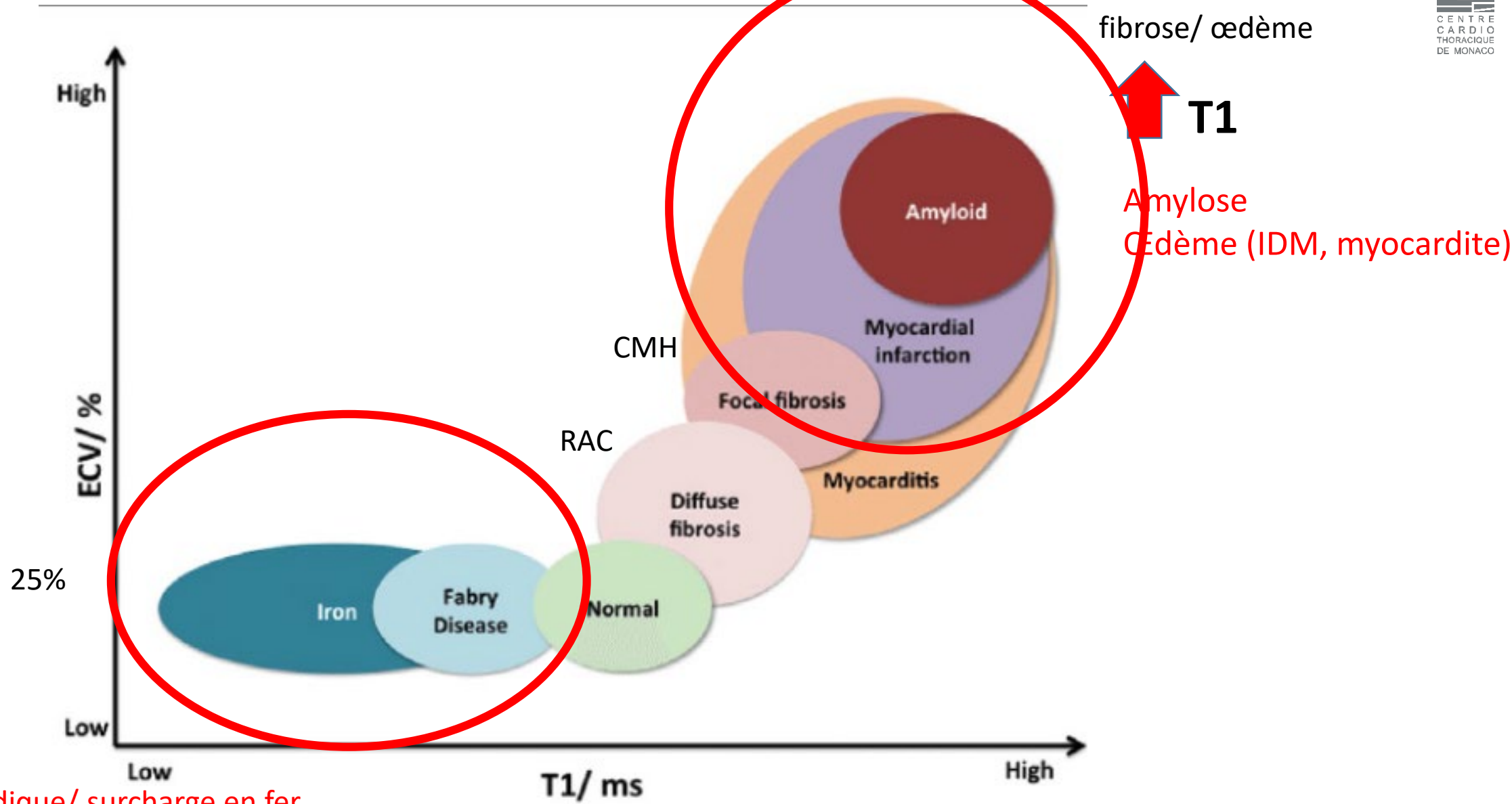
Extra cellular volume (ECV)

- volume extracellulaire (ECV) généré à partir des images t1 pré-et post-Gd

$$ECV = (1 - HCT) \frac{\frac{1}{T1 \text{ myo post}} - \frac{1}{T1 \text{ myo native}}}{\frac{1}{T1 \text{ blood post}} - \frac{1}{T1 \text{ blood native}}}$$

T1 volume intra et extracellulaire

ECV exclusivement volume extra-cellulaire (ne varie pas dans le cas d'accumulation intra-cellulaire *maladie de Fabry!!*).



infiltration lipidique/ surcharge en fer



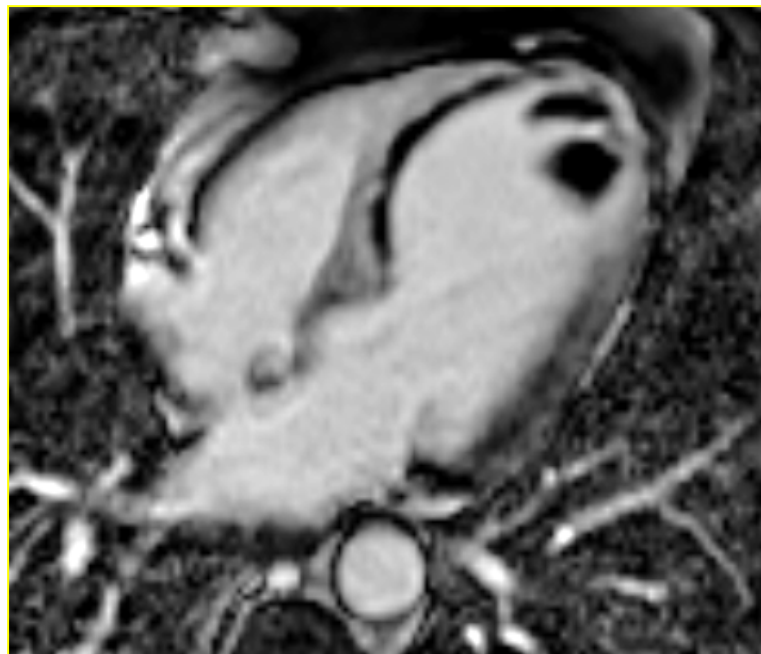
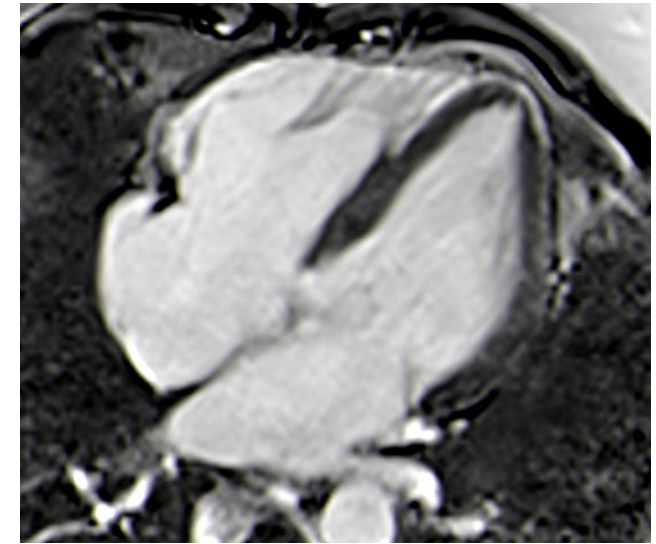
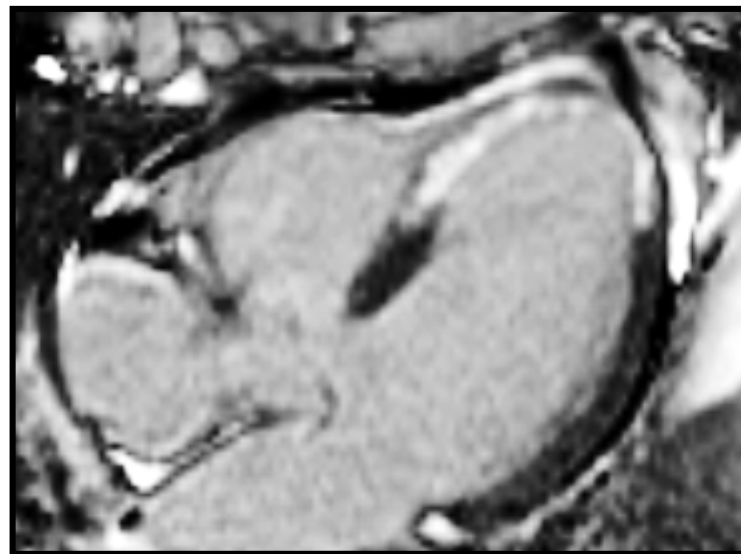
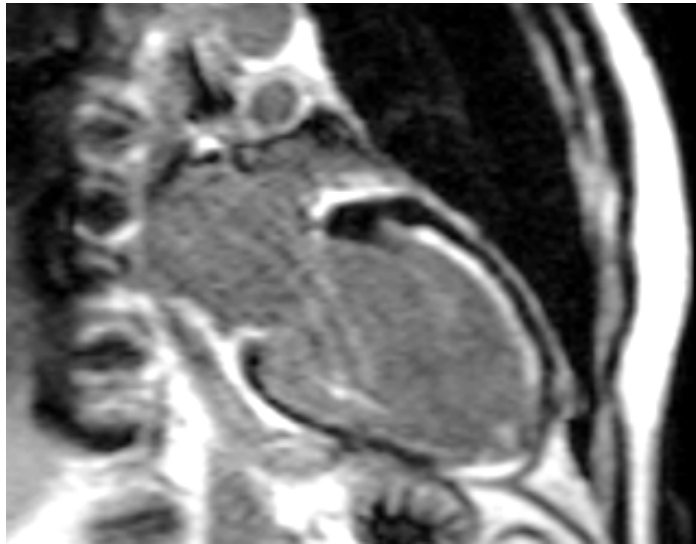
T1

Les bonnes indications de l'IRM cardiaque

- Pour quel patient l'IRM apportera des nouvelles informations qui changeront la PEC du patient?
- Registre EuroCMR:
 - 62% des cas, l'IRM change la PEC
 - 9% l'IRM change le diagnostic
- 11% IRM était normale, malgré un précédent diagnostique d'anomalies a été posé

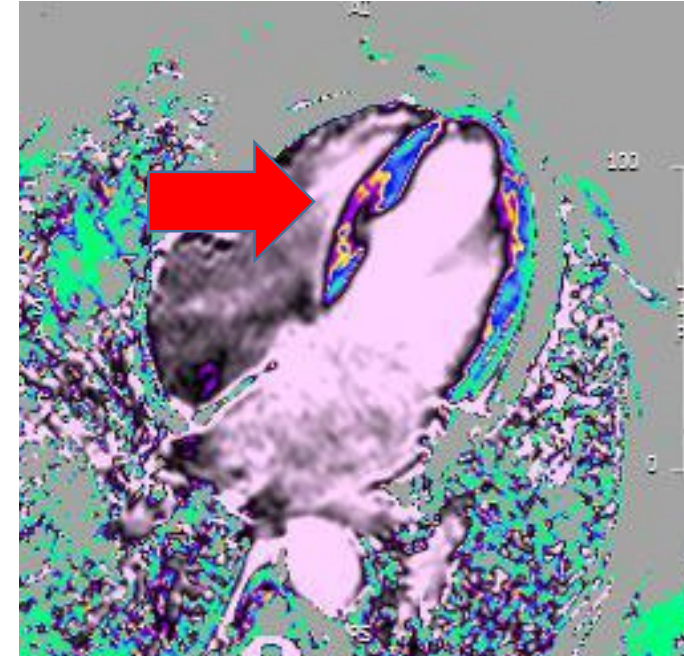
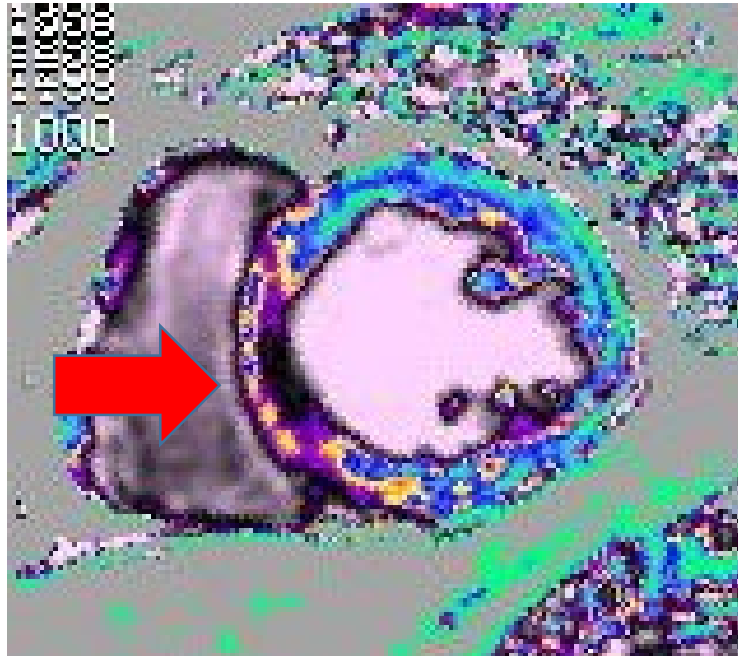
Apport de l'IRM dans le SCA tropo +

- **IDM**
 - Confirmation et étendu de la nécrose
 - IDM récente vs ancien (œdème, MVO)
 - recherche complication IDM (fissuration, IM, Dressler, CIV)
- **MINOCA** (myocardial infarction non-obstructive coronary arteries)
 - Rupture de plaque
 - Dissection coronaire
 - Spasme
 - Embolie coronaire
- **Takotsubo**
- **Myocardite**

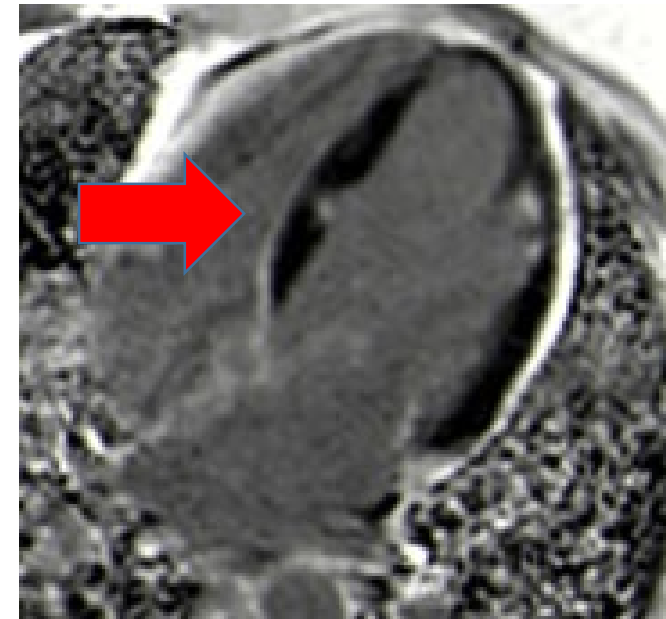
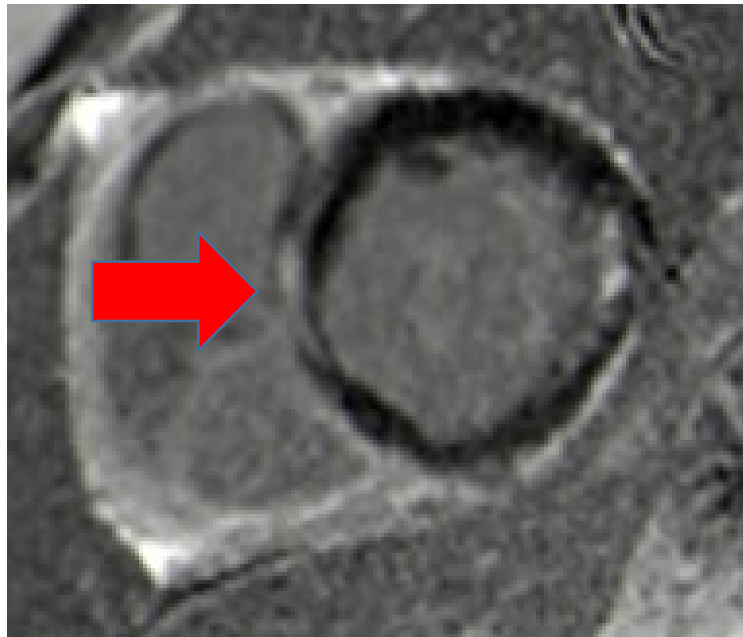


LGE

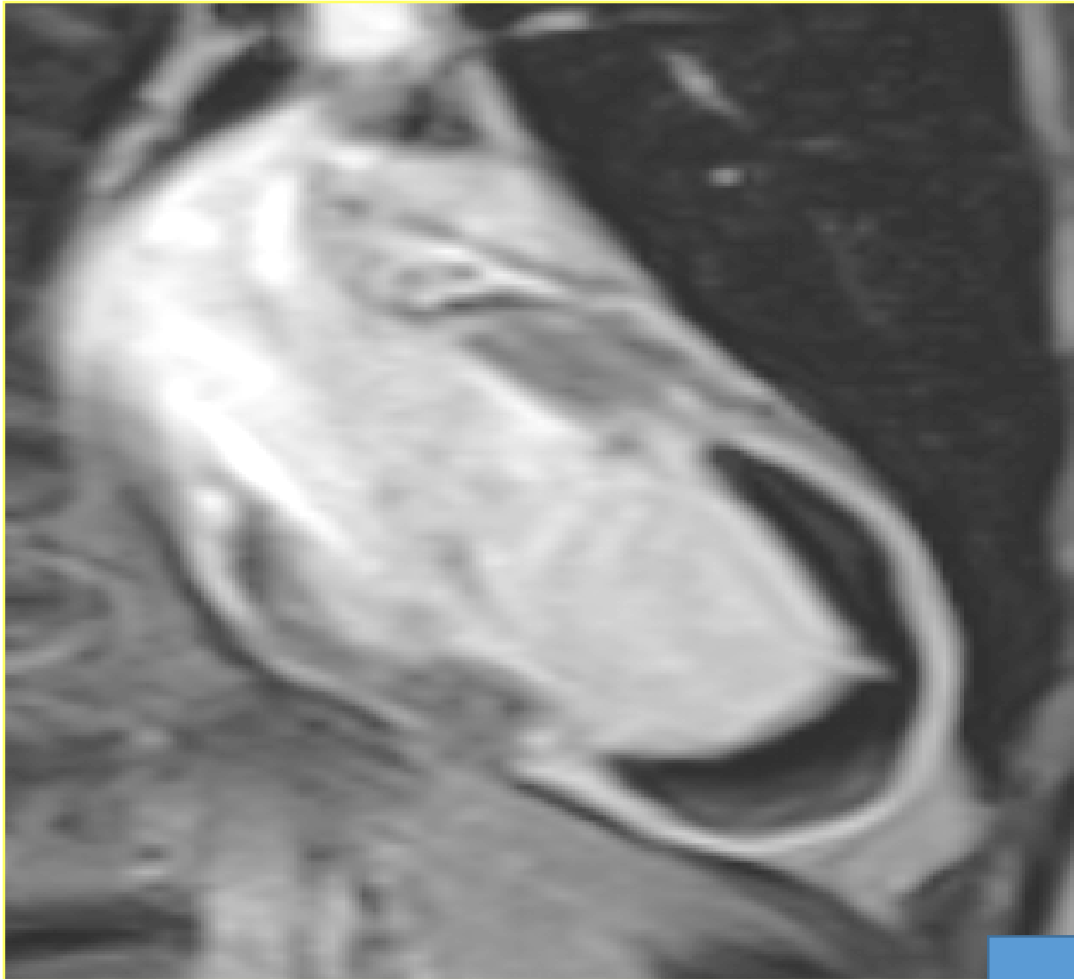
IDM récent vs ancien



femme 37 ans,
LES



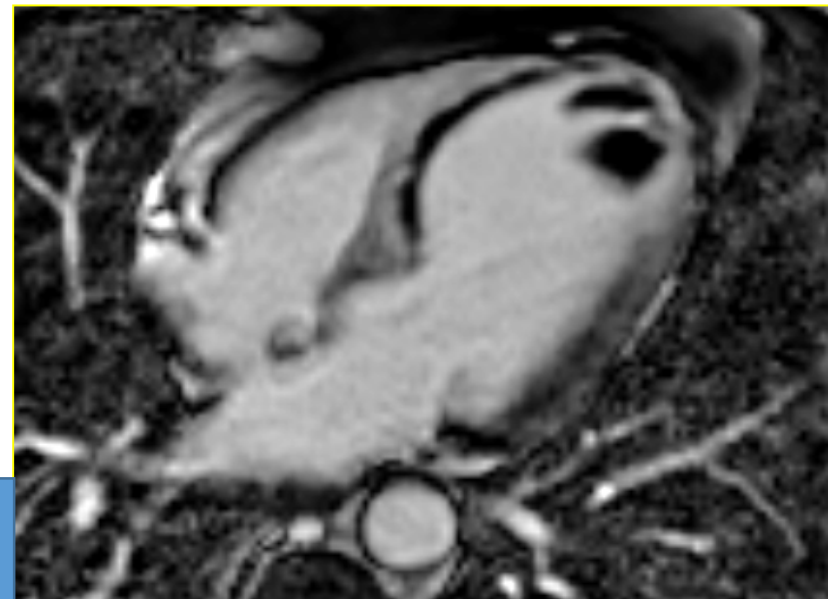
Complications IDM: thrombus MVO



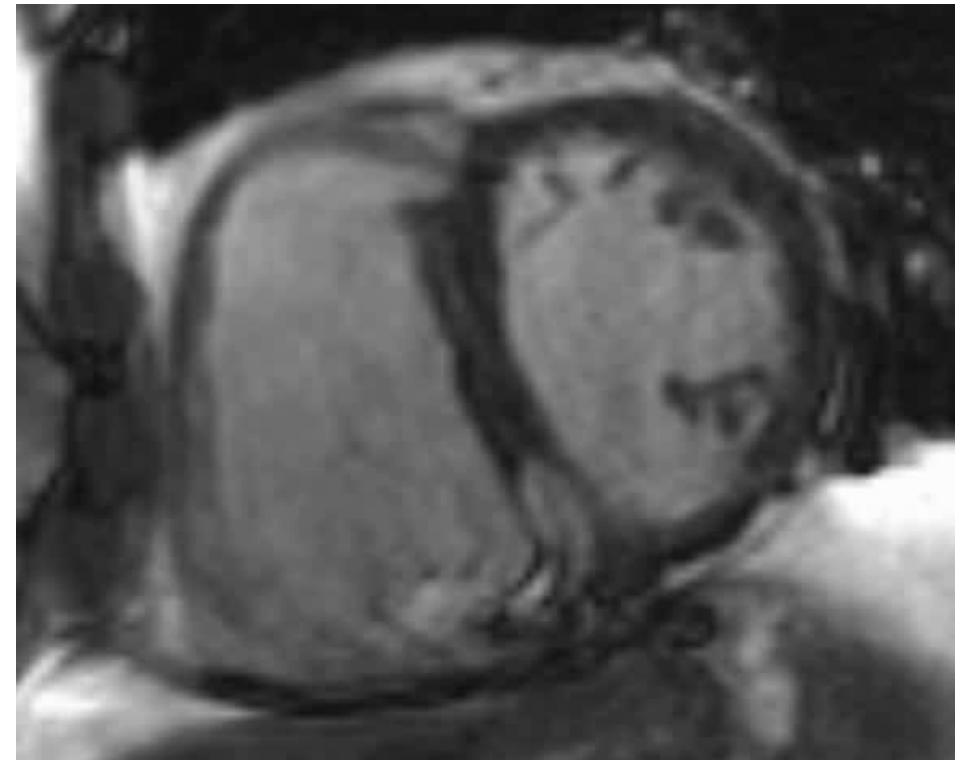
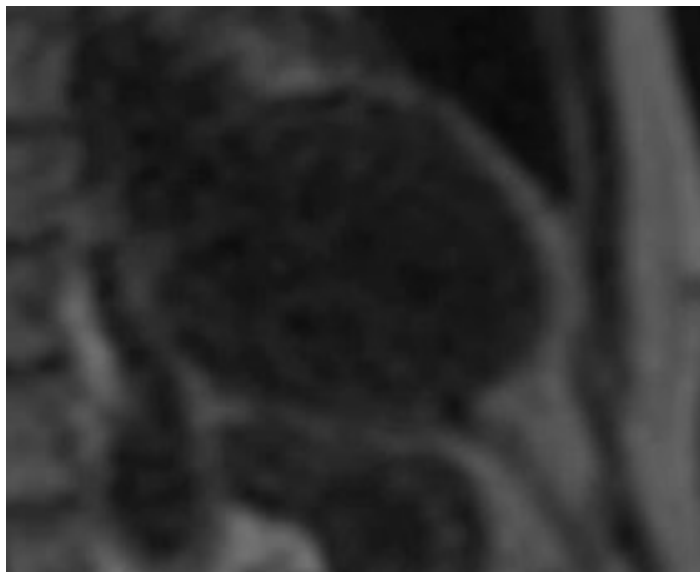
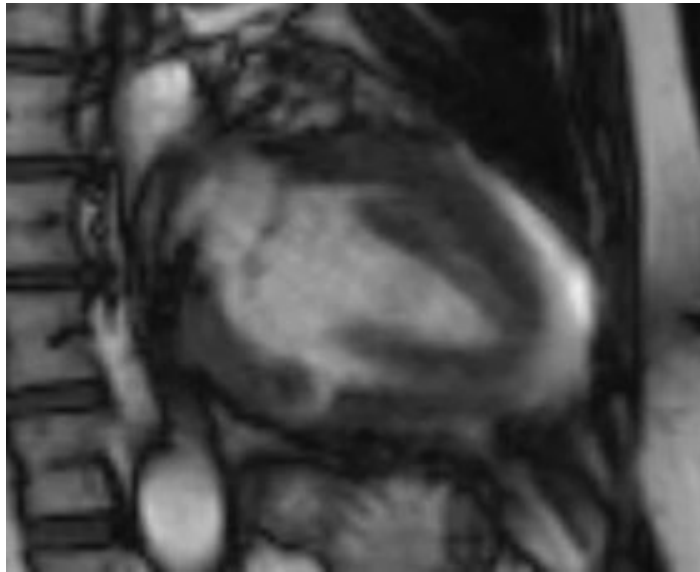
LGE



no reflow



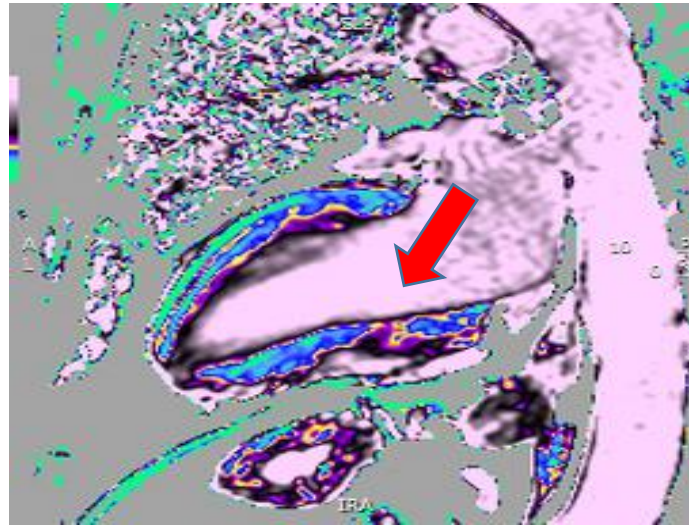
Complications IDM: fissuration



Apport de l'IRM dans le SCA tropo + coronaires normales

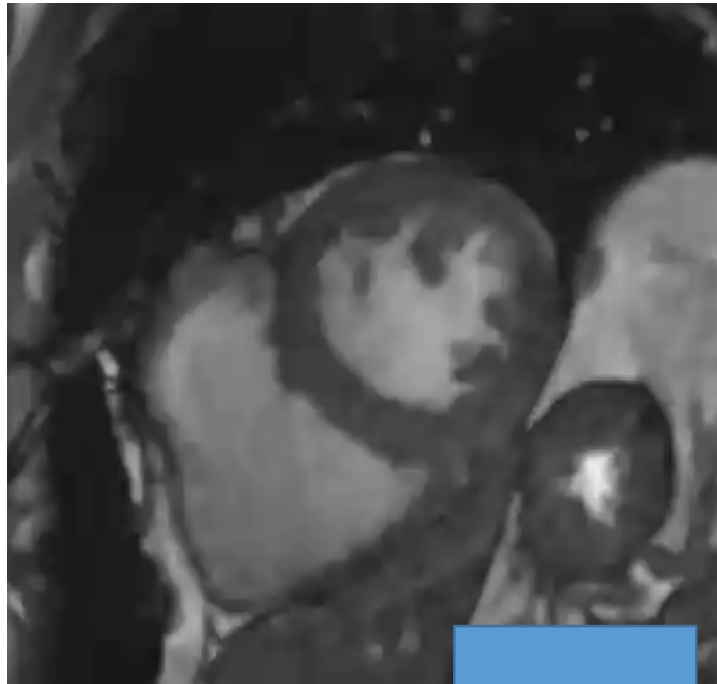
- **MINOCA** (myocardial infarction non-obstructive coronary arteries)
 - Rupture de plaque
 - Dissection coronaire
 - Spasme
 - Embolie coronaire
- **Takotsubo**
- **Myocardite**

Homme, 63 ans,
DT tropono 6 ng/ml
ECG et ETT dans la norme
Gastro-enterite

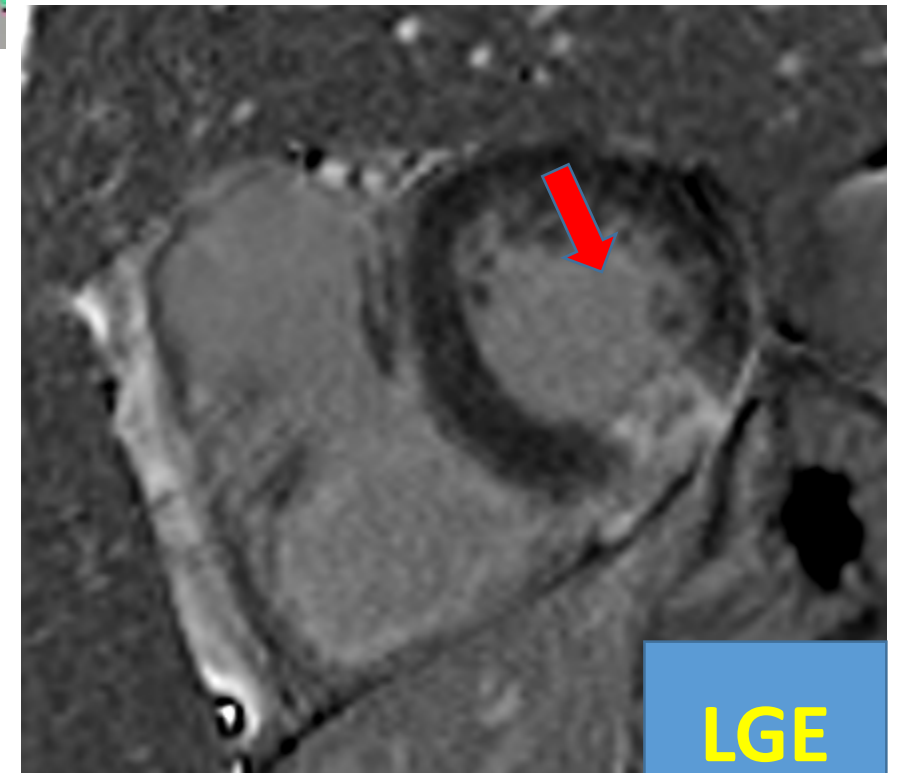
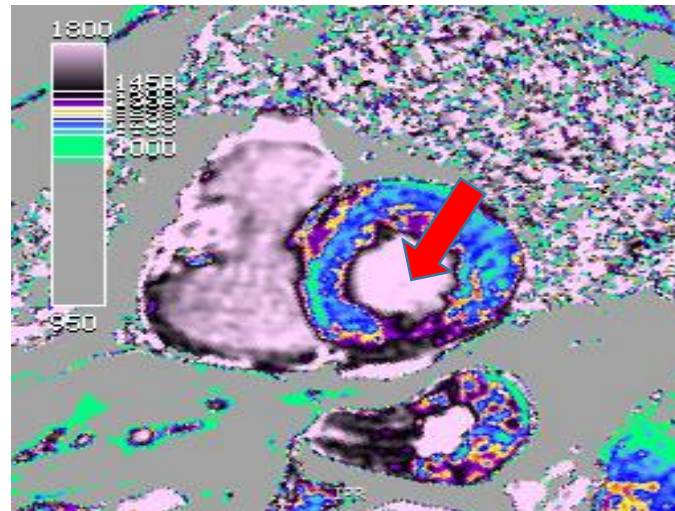


~~Myocardite?~~

IDM inféro-moyen récent

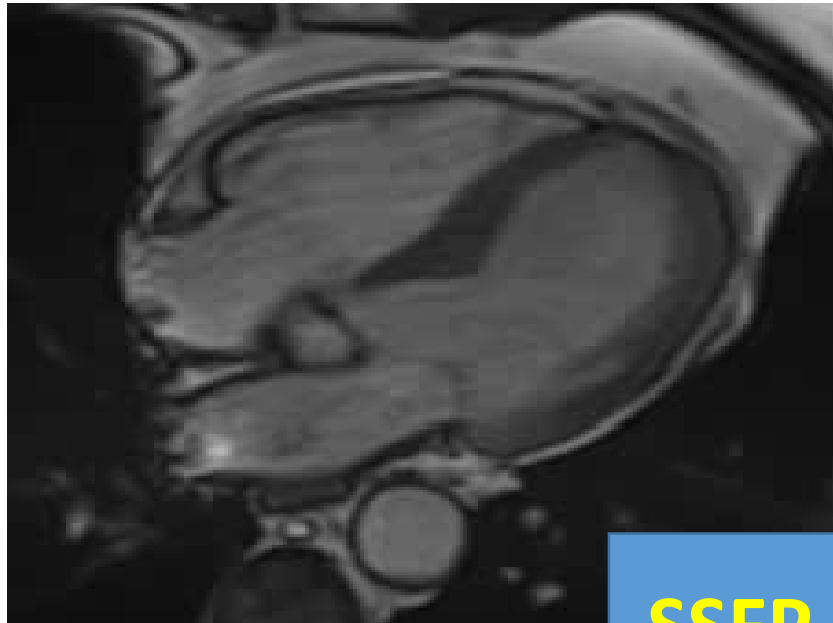


SSFP



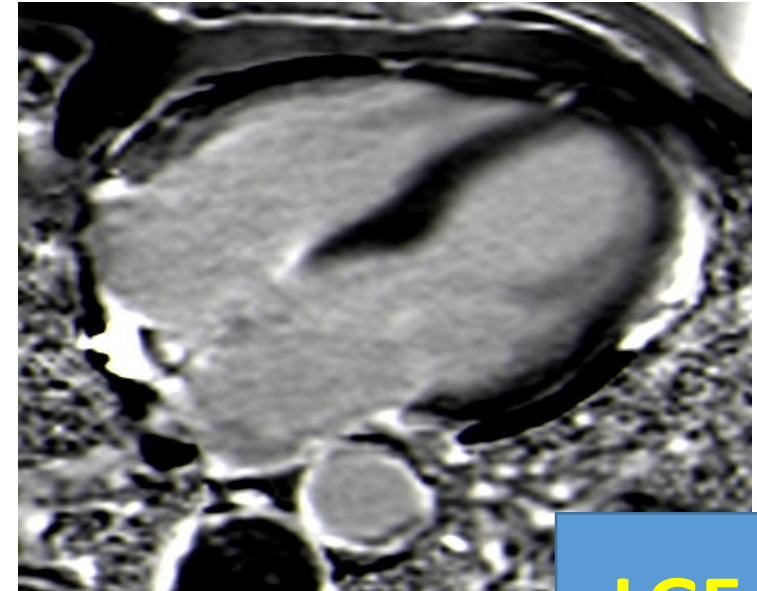
LGE

Homme, 63 ans
DT tropono 1.3 ng/ml
Coro: lésions < 50%

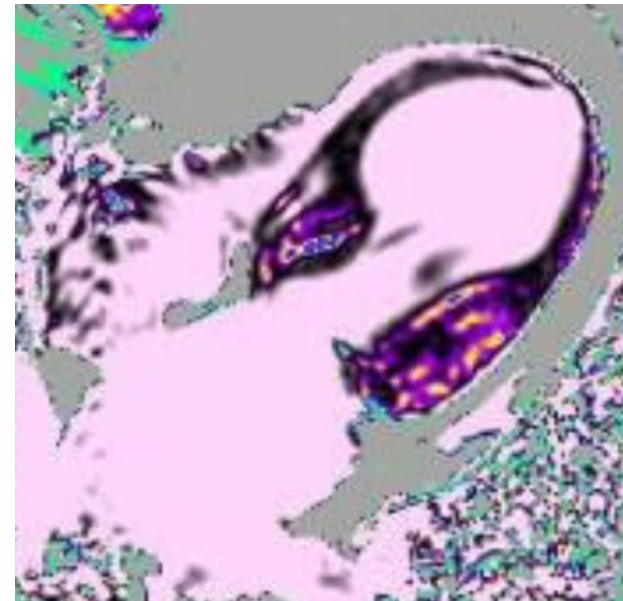


SSFP

Takotsubo



LGE



Takotsubo (cardiomyopathie de stress)

- Femmes (60-75 ans)

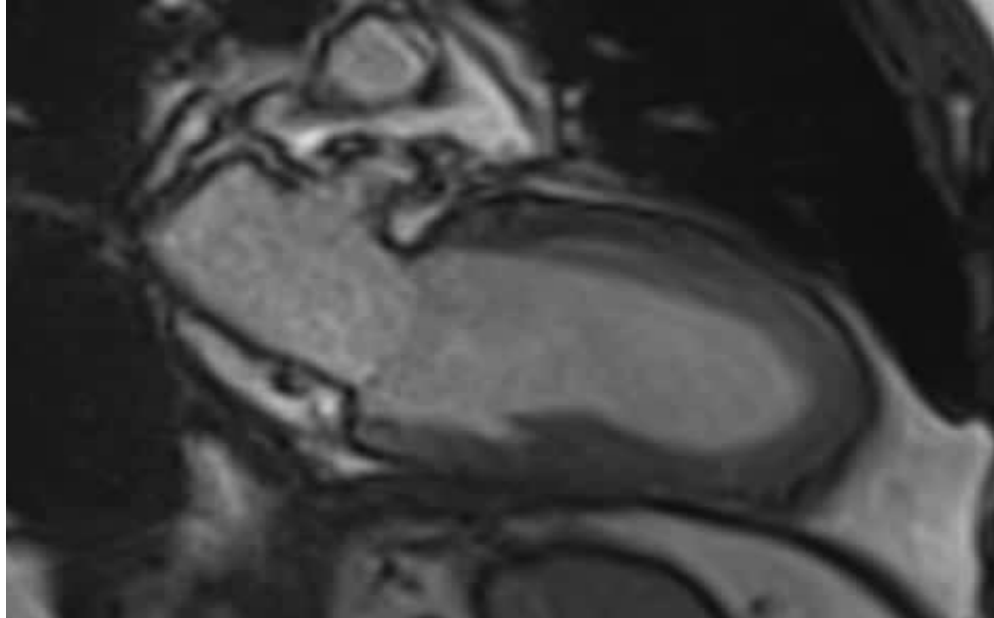


IRM:

- trouble de la **cinétique** (disproportionné au **faible** augmentation de la **troponine**)
- Œdème++++
- Absence de LGE !
- Recupération ad integrum

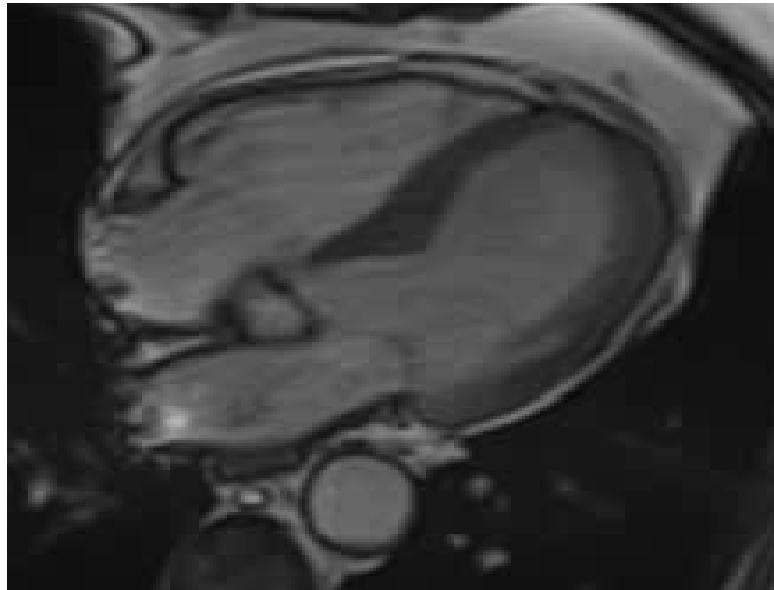


J 0 EF à 65 %

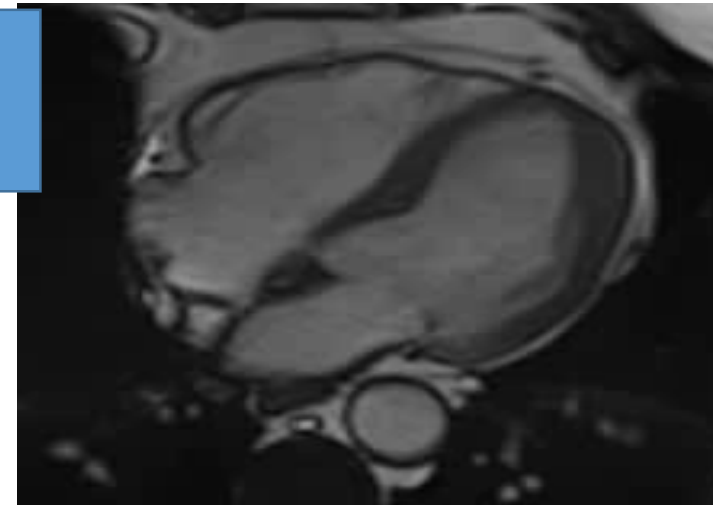


1 mois après

FE 73%

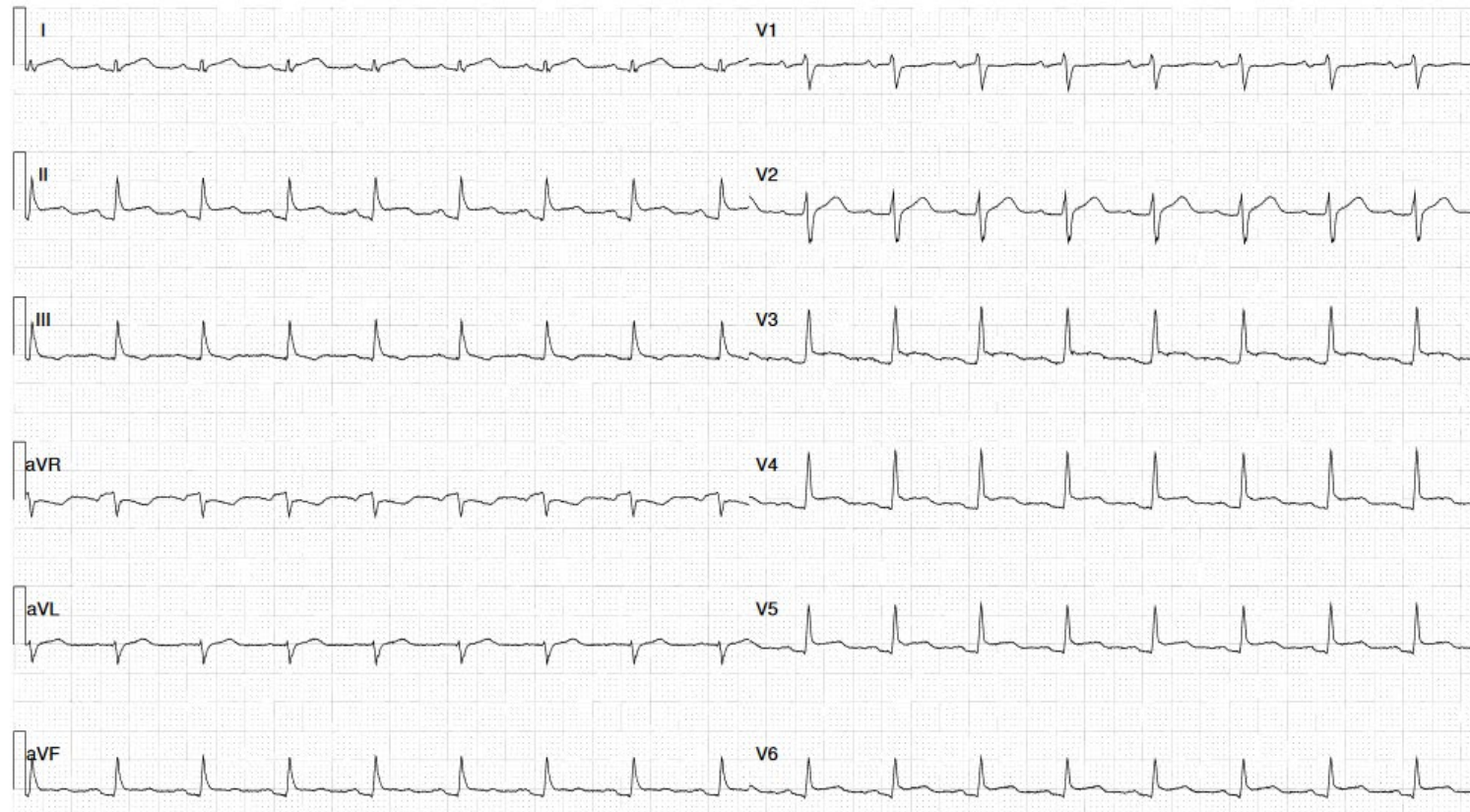


SSFP

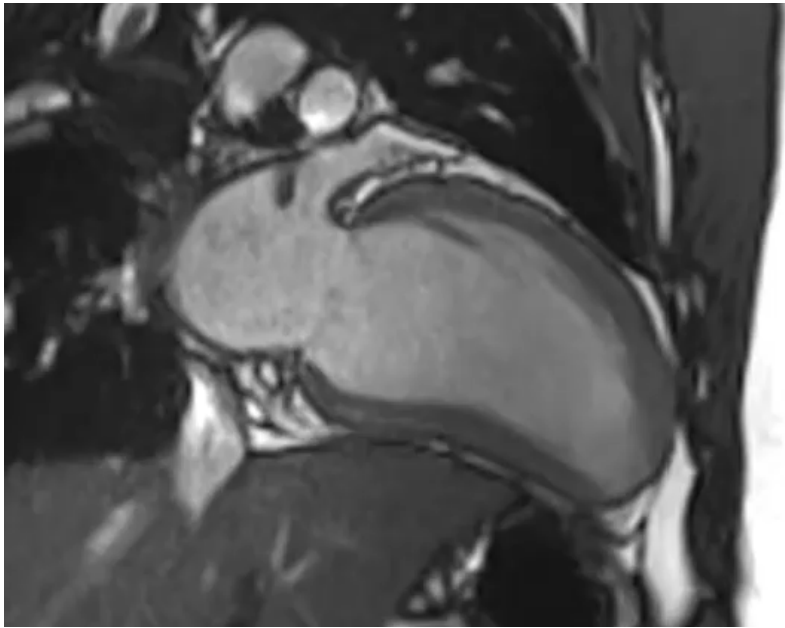


Homme, 32 ans

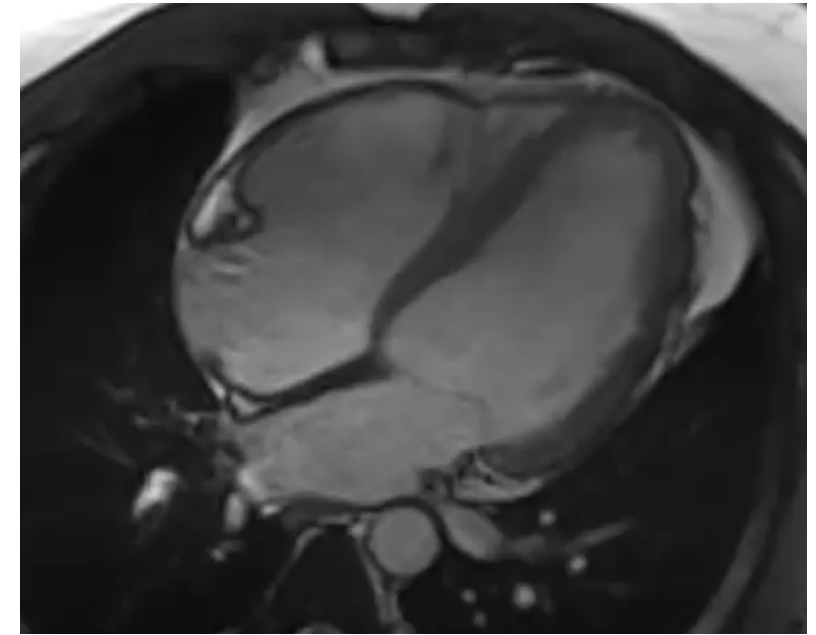
- Absence FDR
- hospitalisé pour SCA tropo + (30 ng/ml); syndrome inflammatoire biologique.
- ECG sus-décalage diffus et sous-décalage PR
- Coronarographie montrant l'absence de lésion coronaire.



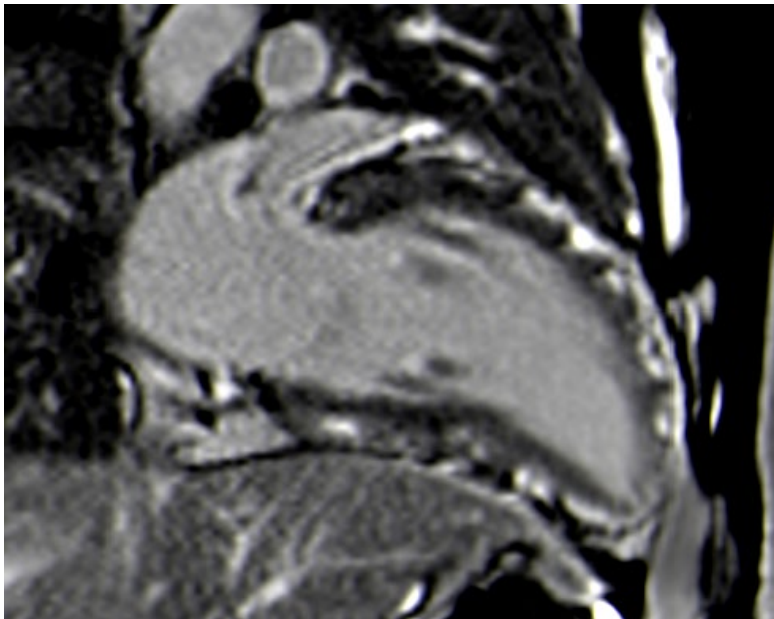
SSFP



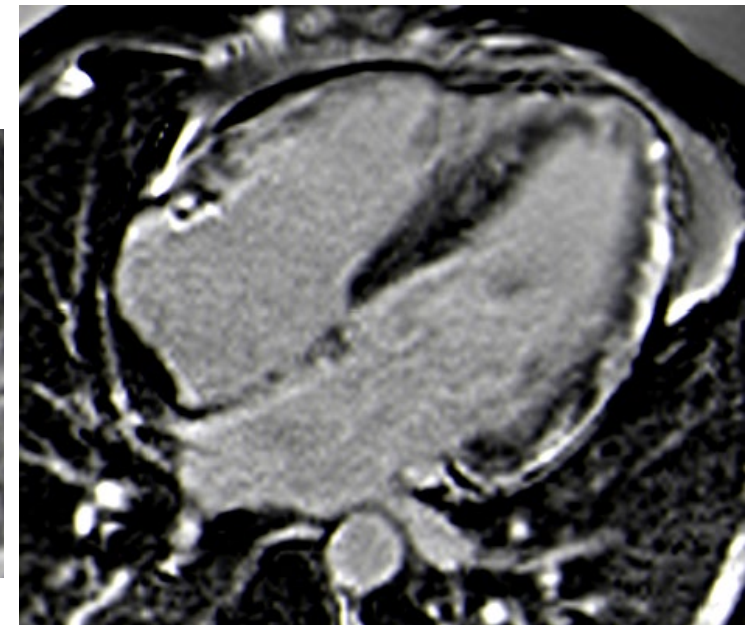
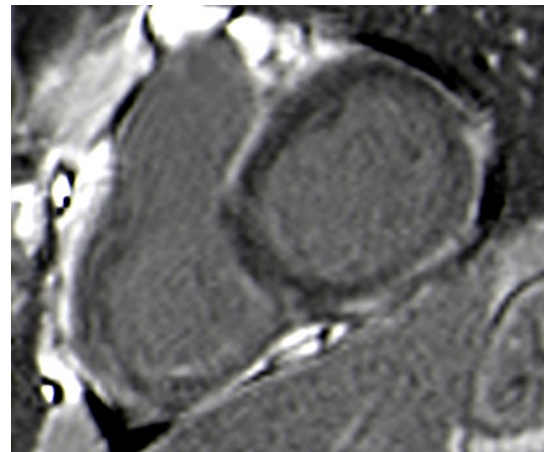
EF 45 %



LGE



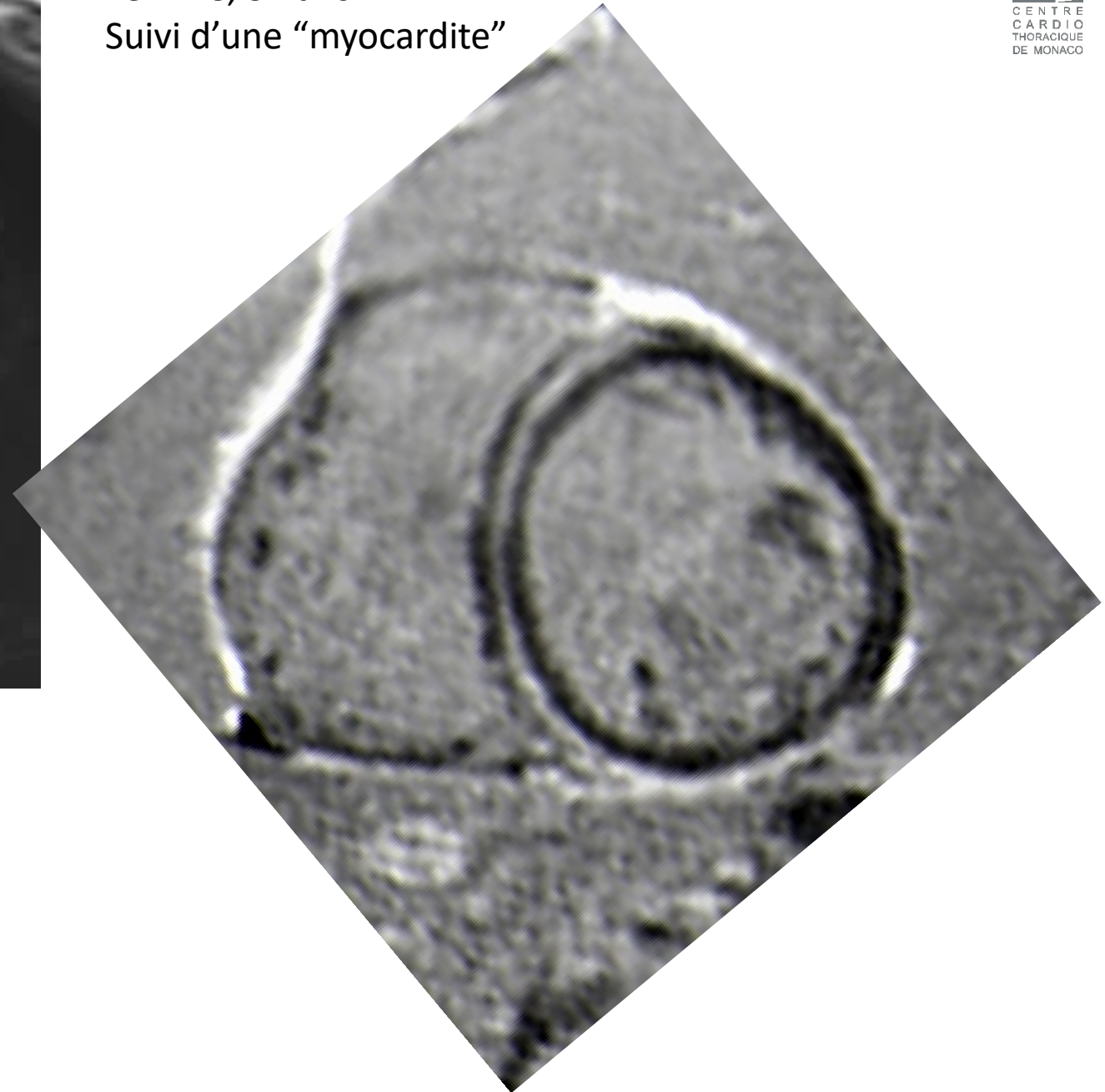
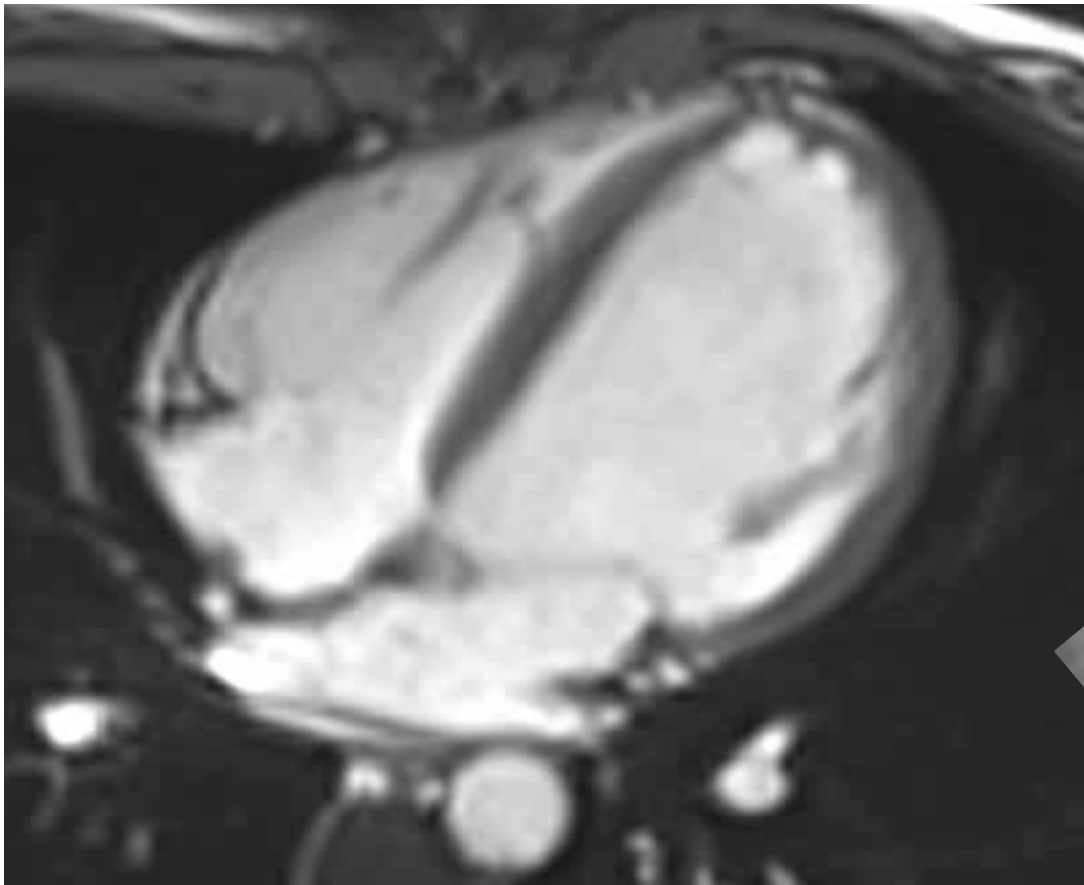
myocardite



IRM dans la myocardite

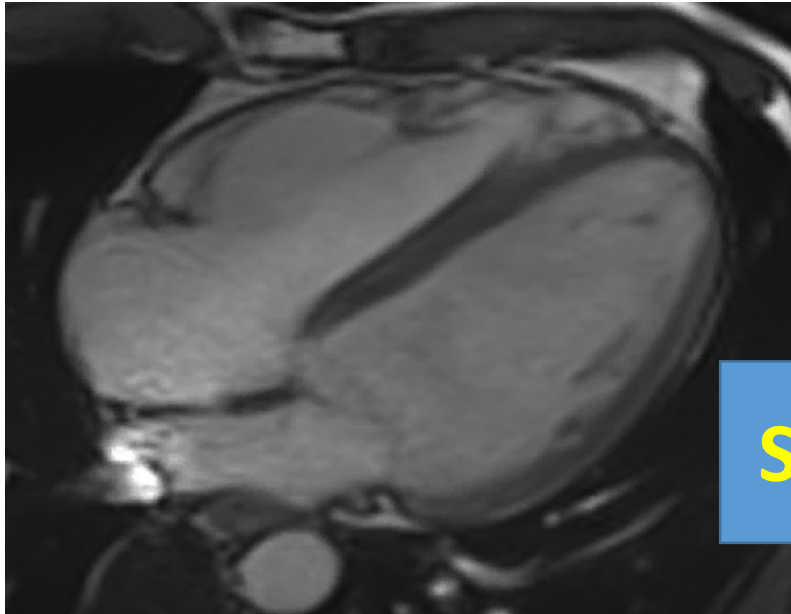
- Phase aiguë:
 - Confirmation du diagnostic (œdème, LGE pathognomonique)
 - Evaluation VG
- A distance (réabsorption de l'œdème)
 - Evaluation fibrose résiduelle (substrat arythmogène)

Femme, 37 ans
Suivi d'une "myocardite"

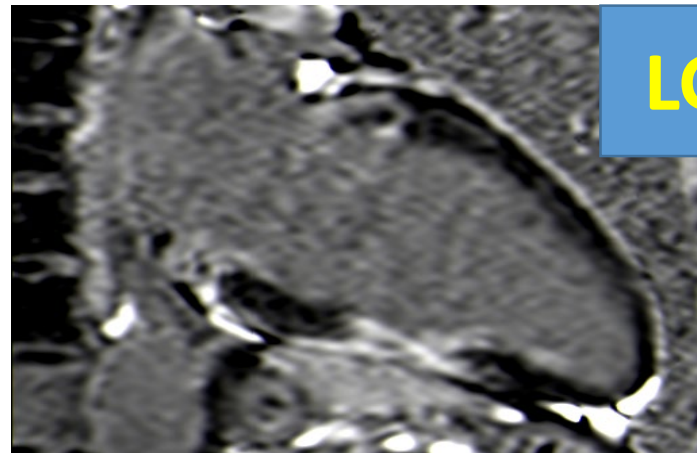
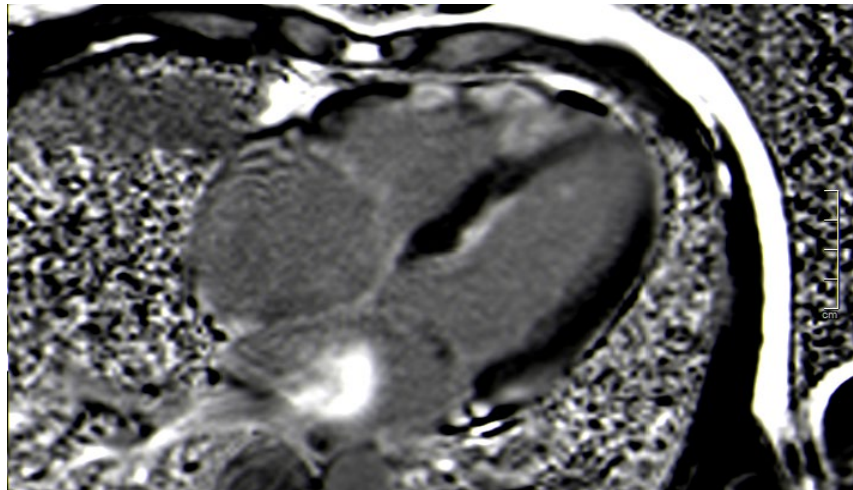
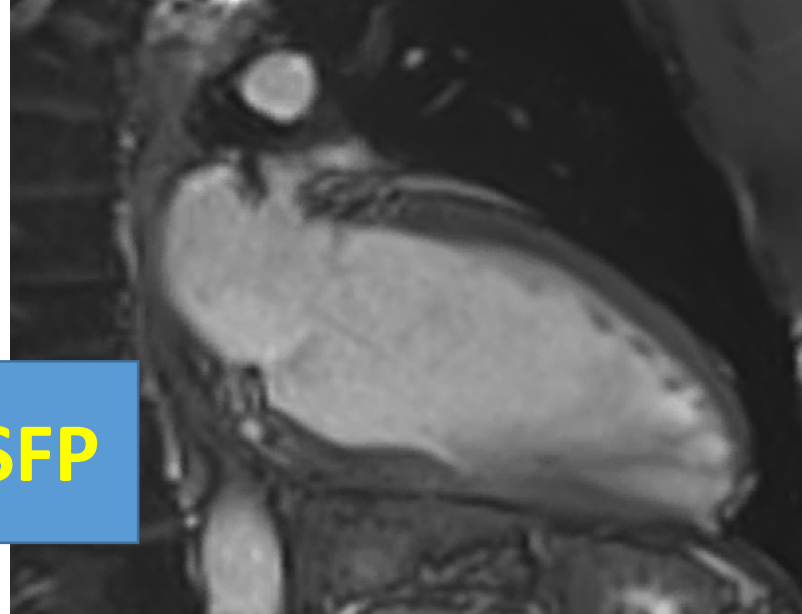


EF à 53 %
EDVI 87 ml/m²
SLG - 17 %.

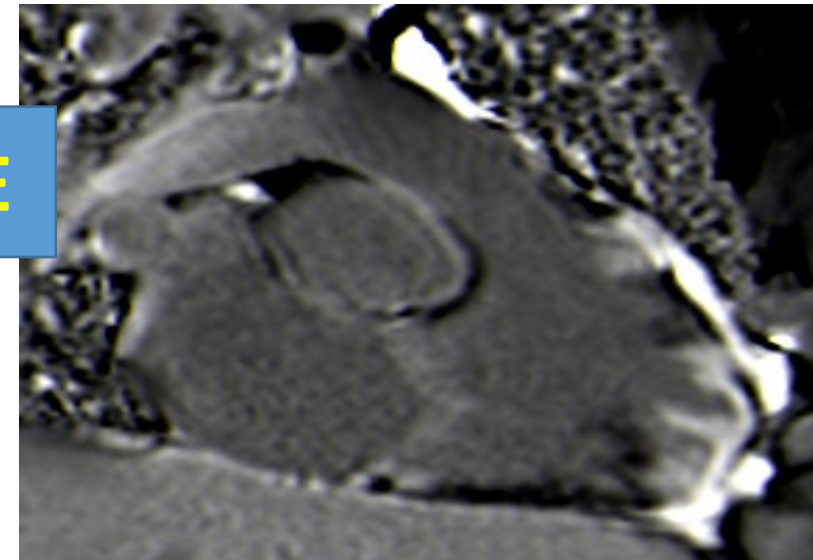
sarcoïdose



SSFP



LGE



Syndrome coronarien chronique

- fonction et volumes VG post IDM (indication à ICD)
- évaluation VD
- thrombus
- étude viabilité et ischémie (avant PTCA, CTO)
- arythmie ventriculaire (fibrose, ischémie)
- recherche ischémie
 - Suivi CCS
 - Dépistage coronarien avant chirurgie non cardiaque (AOMI, obésité, AAA, PTH...)

Homme, 52 ans
SCA antérieure tardive

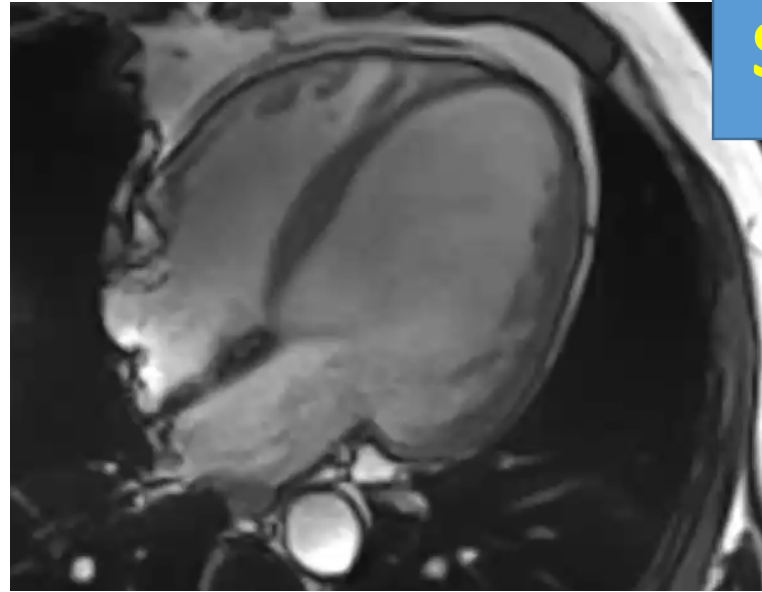
Coro:

- 2 stenting actifs de l'IVA I CTO
- sténose Mg 50%, Cx distale < 50%
- **CD indemne**

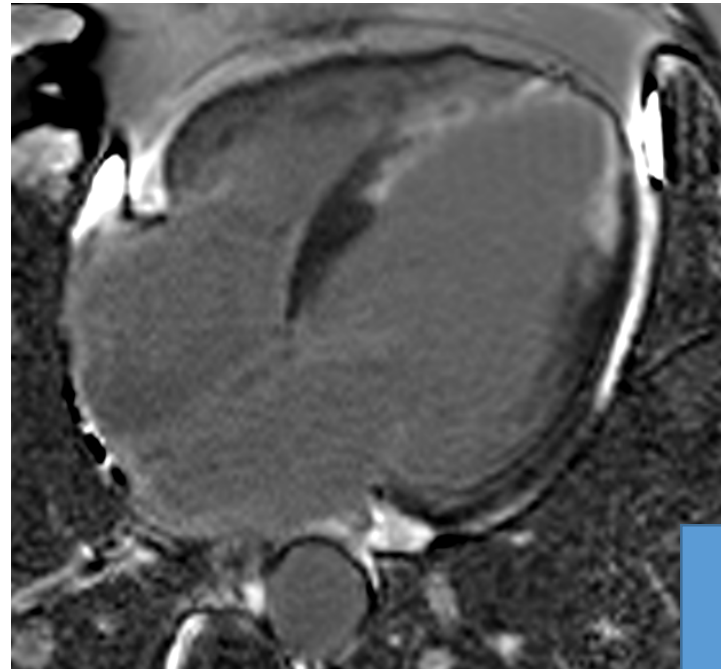
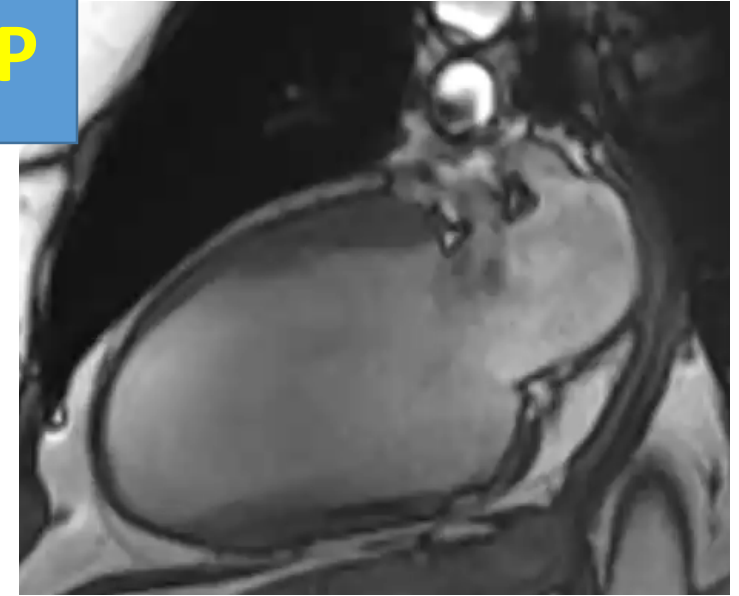
Bilan avant pose de ICD

IDM ASA + inférieur!

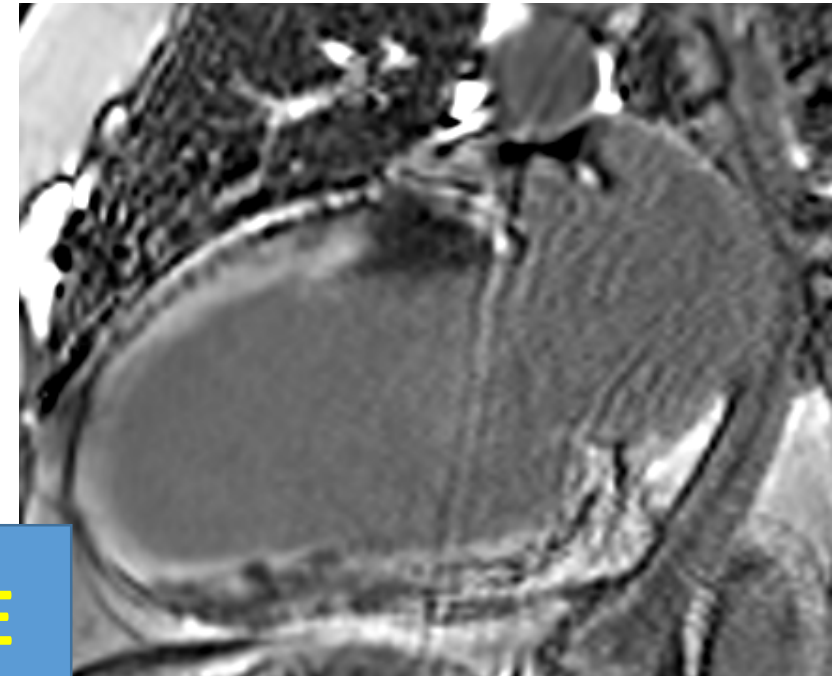
EF à 30%,
EDVI 155 ml/m²;



SSFP



LGE

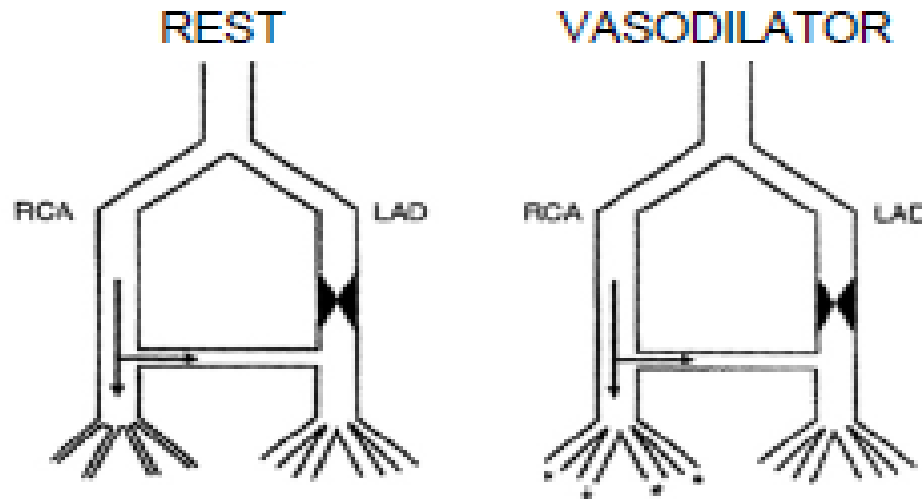
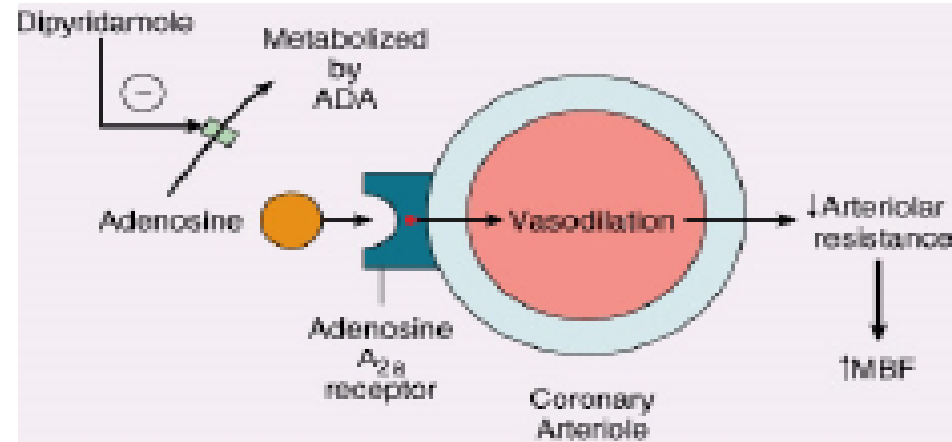


Recherche ischémie

test stress DOBUTAMINE

test perfusion ADENOSINE

Vasodilator 'Stress'



Effet de vol

Défaut de perfusion

Avant une IRM à l'adénosine

- Contrôle de la créatininémie < 3 mois
- Dérivées Xanthine : STOP 12 avant l'examen

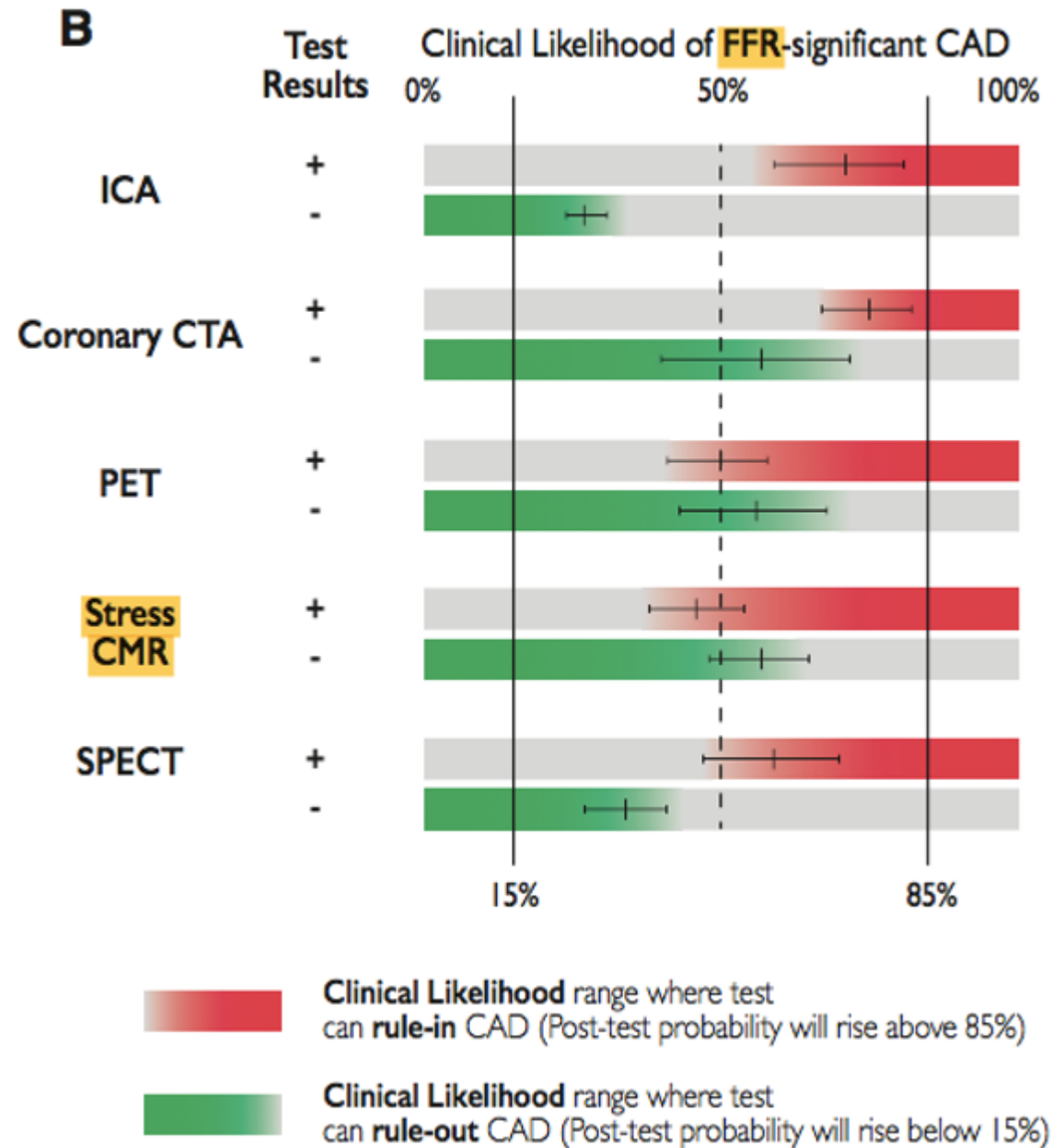


- B bloquant et médicament hypotenseur: la prise du matin de l'examen est décalée pour éviter une excessive bradycardie et hypotension



Adénosine CMR

- Safe
- fiable
- Valeur pronostique



CMR is safe

Table 2 Complications related to no stress vs. stress CMR

	All (n = 27396)		No stress (n = 17136)		Stress (n = 10228)	
Complications						
None	96.3%	(n = 26395)	98.6%	(n = 16893)	92.6%	(n = 9476)
Mild	3.6%	(n = 994)	1.4%	(n = 243)	7.3%	(n = 745)
Severe	0.0%	(n = 7)	0.0%	(n = 0)	0.1%	(n = 7)

Values are % (n).



No patient died for CMR

Bruder et al. *Journal of Cardiovascular Magnetic Resonance* 2013, 15:9
<http://www.jcmr-online.com/content/15/1/9>

 **Journal of Cardiovascular
Magnetic Resonance**

RESEARCH

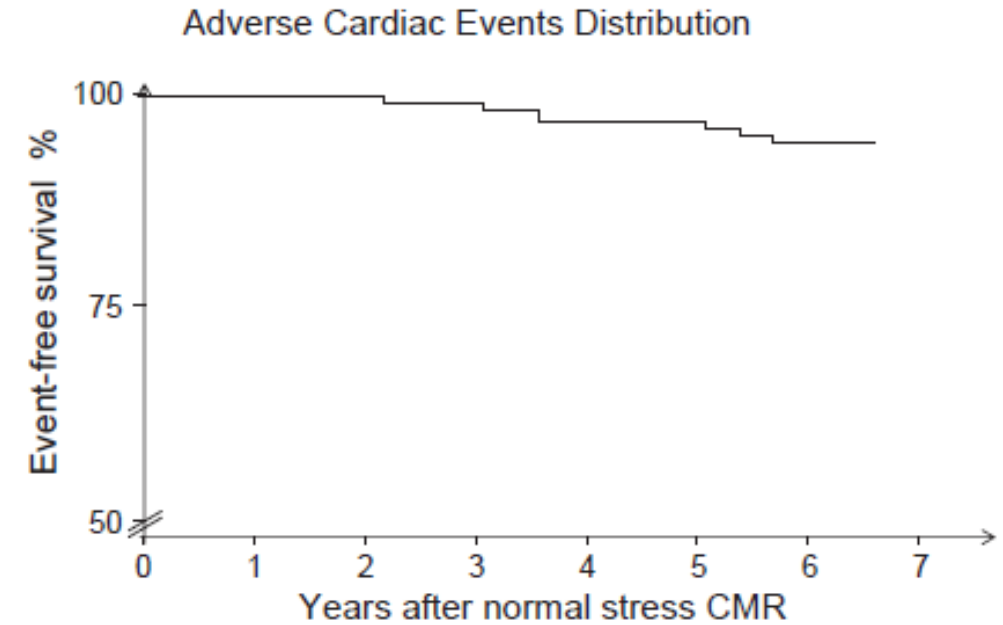
Open Access

European cardiovascular magnetic resonance (EuroCMR) registry – multi national results from 57 centers in 15 countries

Incremental value of normal adenosine perfusion CMR: Long-term outcome

- 300 patients
- Follow up 5,5 y
- The annual cardiac event rate was 1.3%
 - 0.78% in the first 3 y
 - 1.9% between the fourth and sixth year.

Ends point: global mortality and MACE

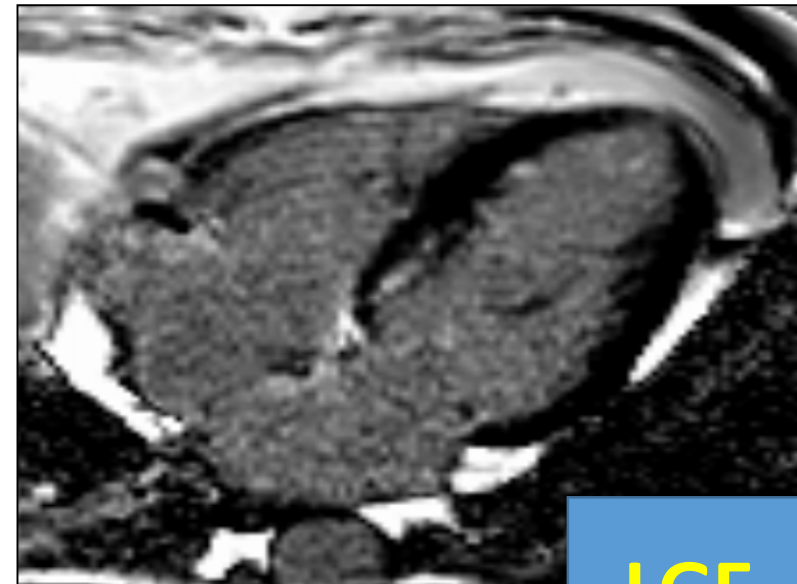
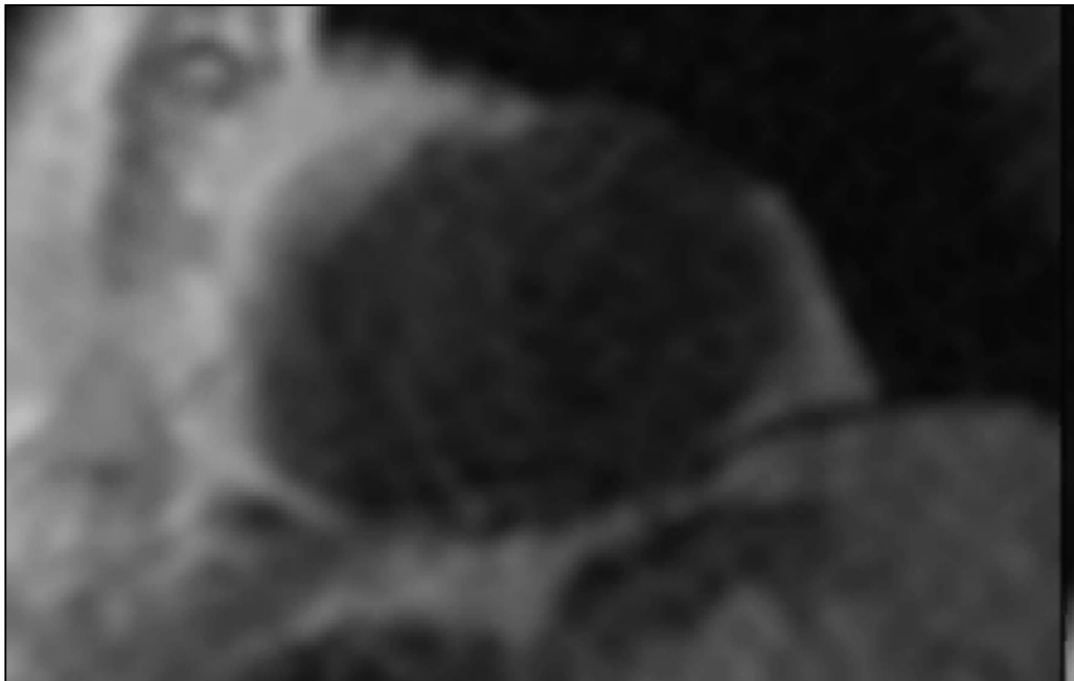


[Incremental value of normal adenosine perfusion cardiac magnetic resonance: Long-term outcome.](#)

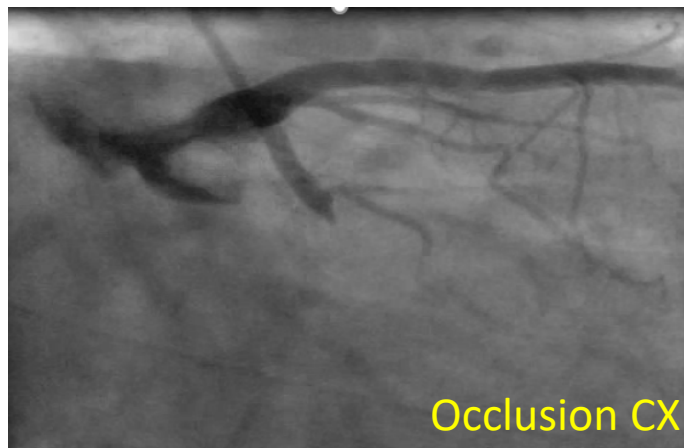
Sozzi FB, Iacuzio L,

doi: 10.1016/j.ahj.2015.03.001

Homme, 75 ans, bilan de TVNs



LGE

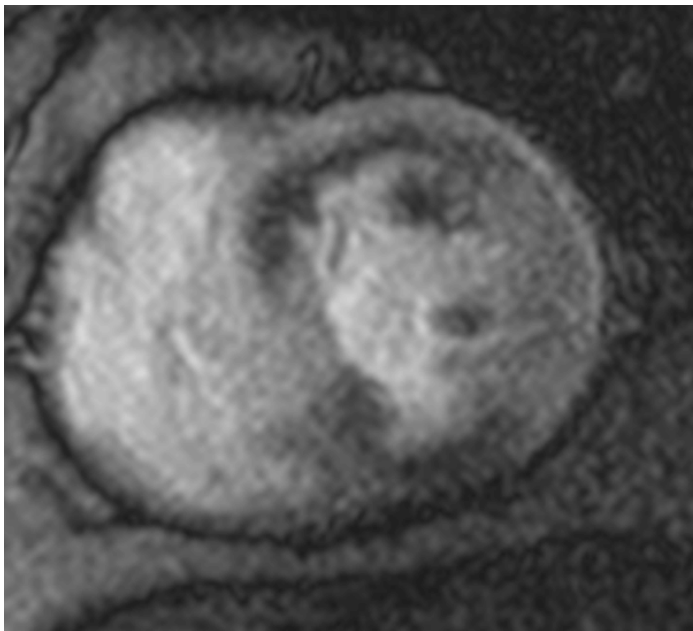


Occlusion CX

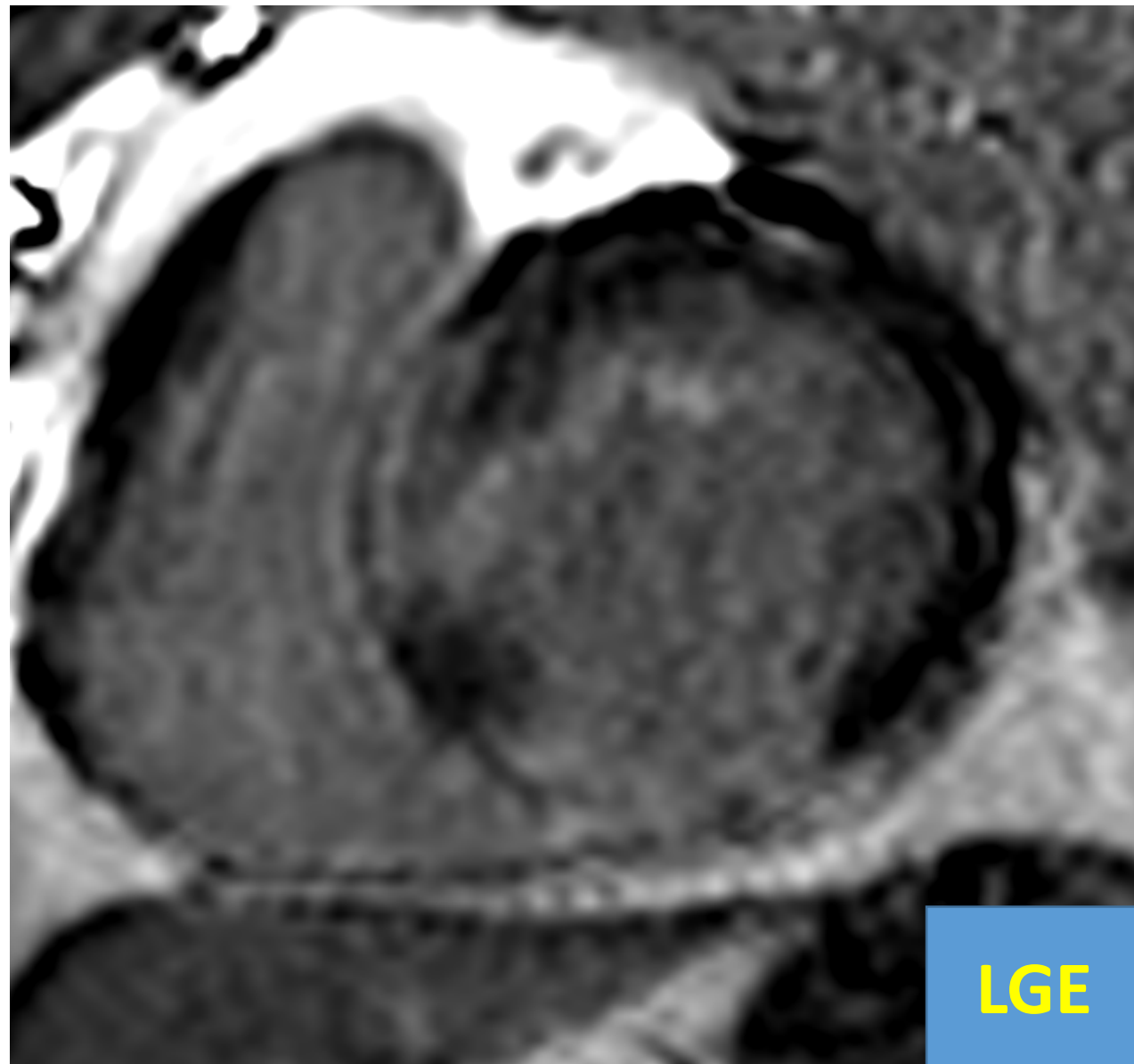
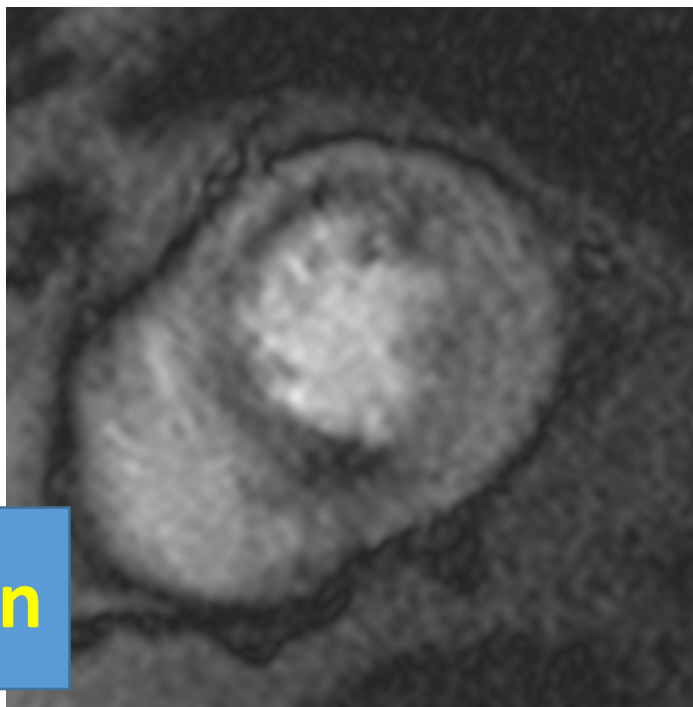
Hypoperfusion inféro-laterale,
plus étendue que la nécrose

Homme, 66 ans

- ATCD RTRA en 2002 (dissection chronique aorte ascendante et crosse) + RVAO en 2017
- stent actif redux CD 2011
- stent IVA en 2016.
- Actuellement patient **asymptomatique**.
- FDR : HTA, DNID, hérédité.

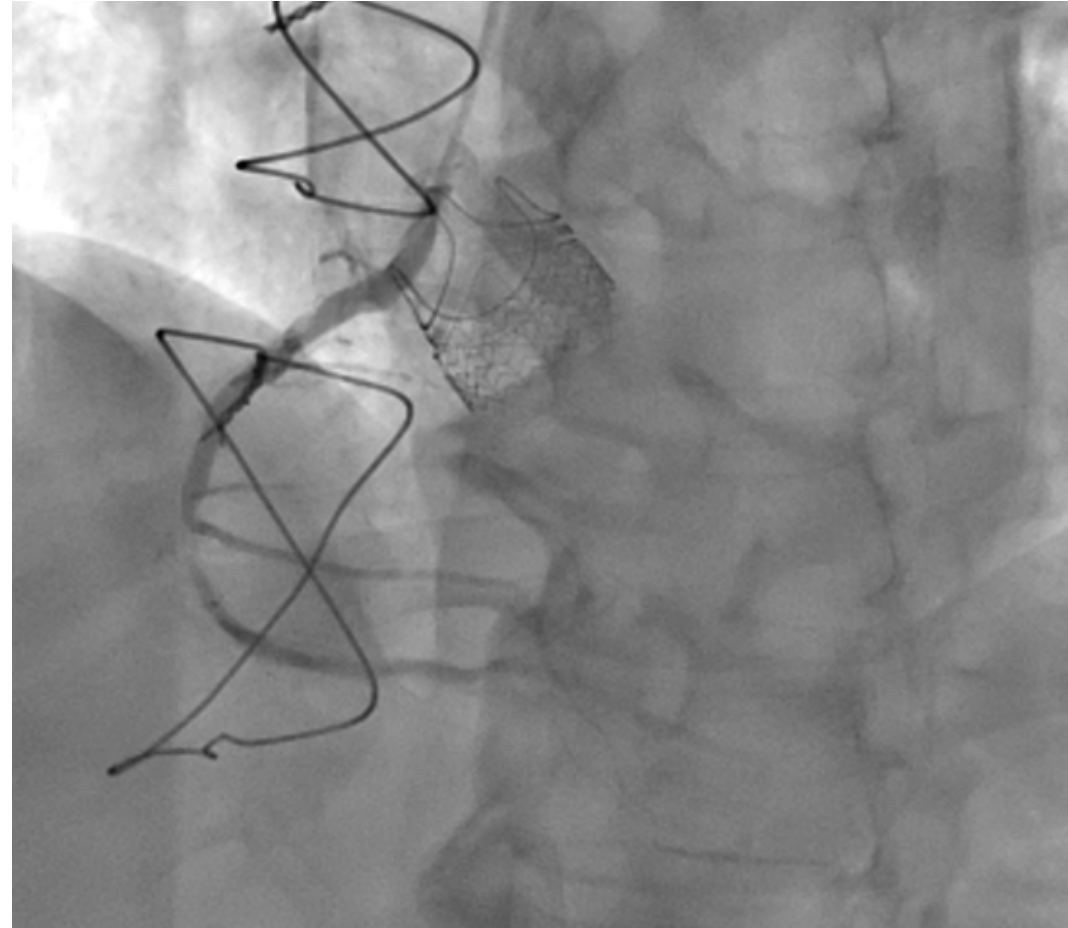
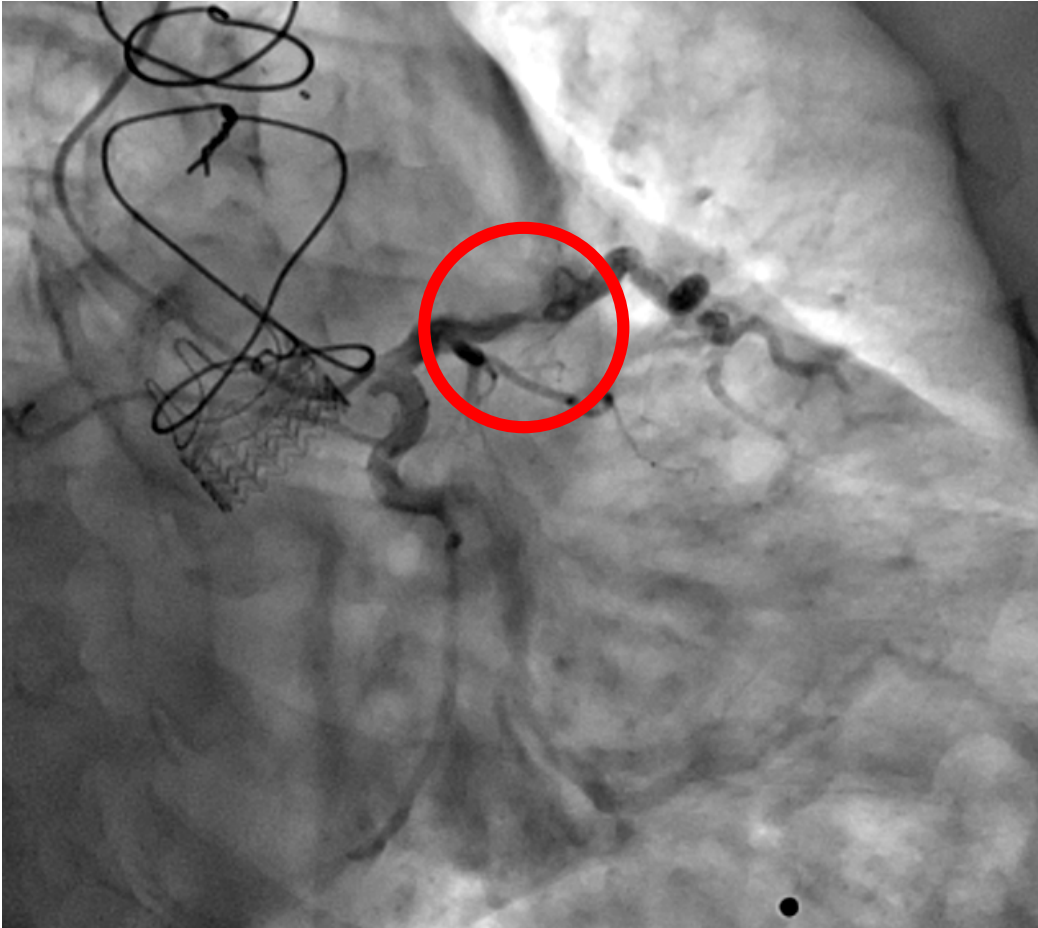


perfusion



LGE

- lésion sévère calcifiée menaçante de l'IVA II, bon résultat du stent de l'IVA II en aval
- occlusion chronique courte de la coronaire droite moyenne.



Indication à l'IRM adenosine

- Patient coronarien connu, ATCD d'IDM (dd territoire nécrosé vs ischémique)
- Coronaropathie pluri-tronculaire avant angioplastie / CTO : quel vaisseau mérite d'être revascularisé
- Dépistage patient à bas – moyen risque qui n'est pas en mesure de faire un effort
- Contrindication à d'autre test d'ischémie

Non indication à l'IRM adenosine

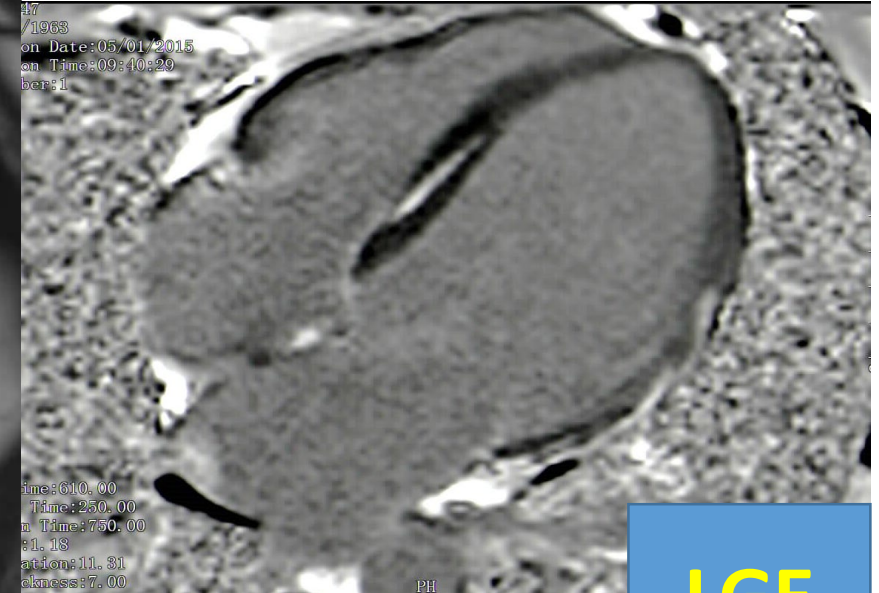
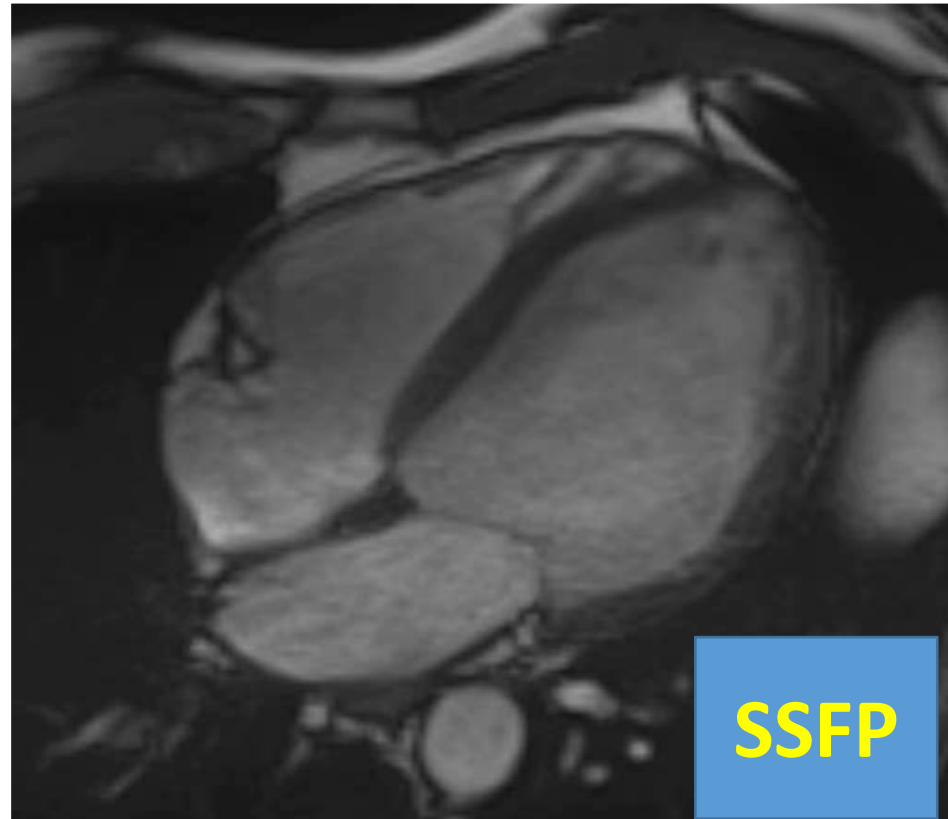
- anomalie d'origine de coronaires, angor spastique, pont musculaire
- chez le patient jeune

CMD

- Volumes / FE VG
- Volumes / FE VD ★
- Complication : thrombus
- Etiologie: ischémique? Non ischémique? Post myocardite?
- Pronostique: fibrose?
- Indication ICD, CRT (FE, viabilité de la paroi latérale)

DD dysfonction VG

- Homme, 52 ans
- Dyspnée
- FE 30%

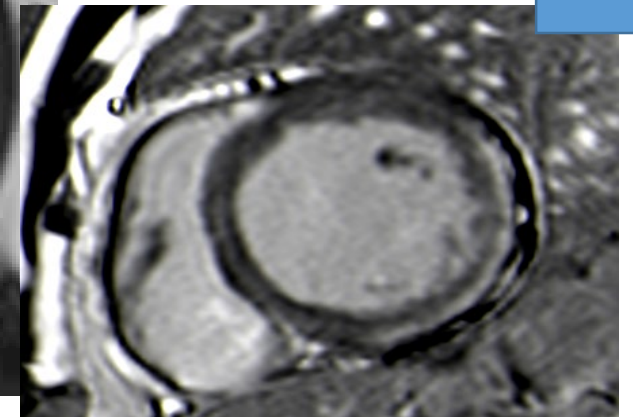
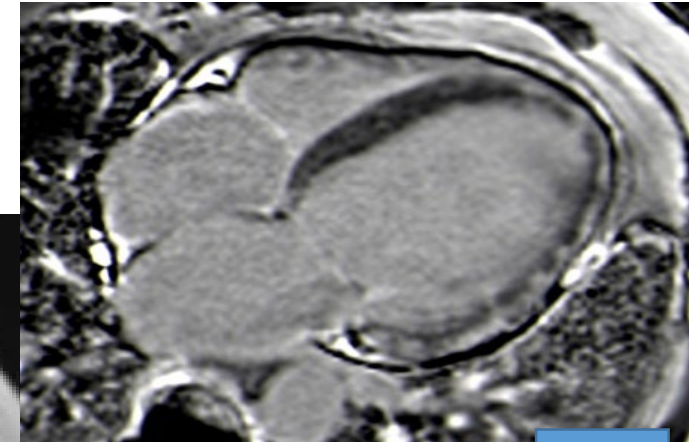
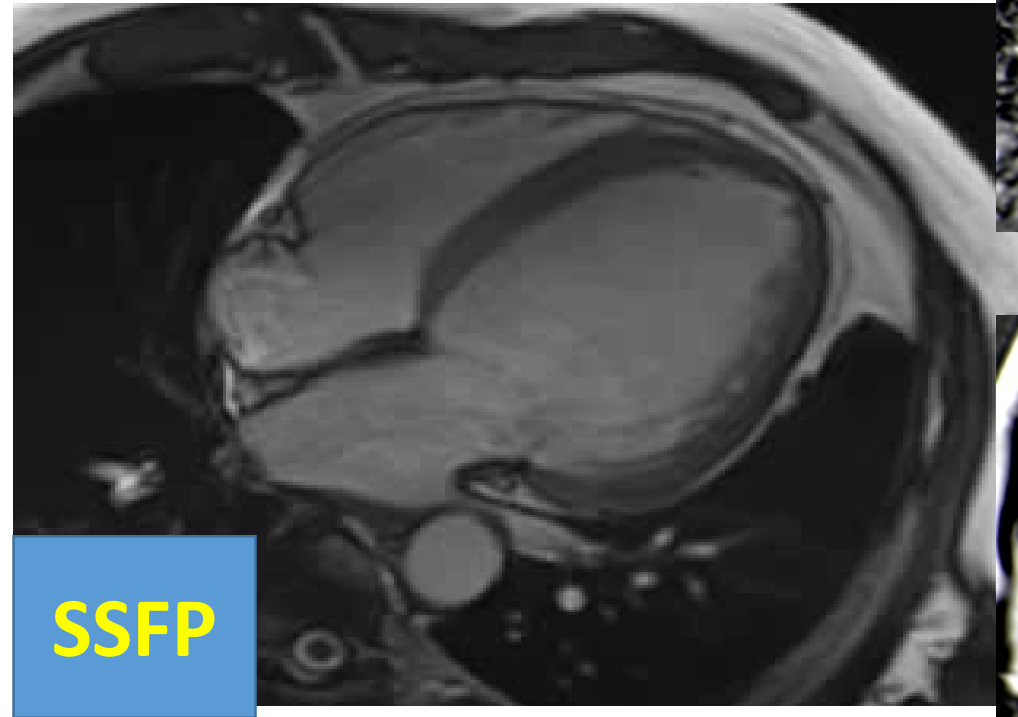


CMD vs ~~CAD~~

CMD post myocardite

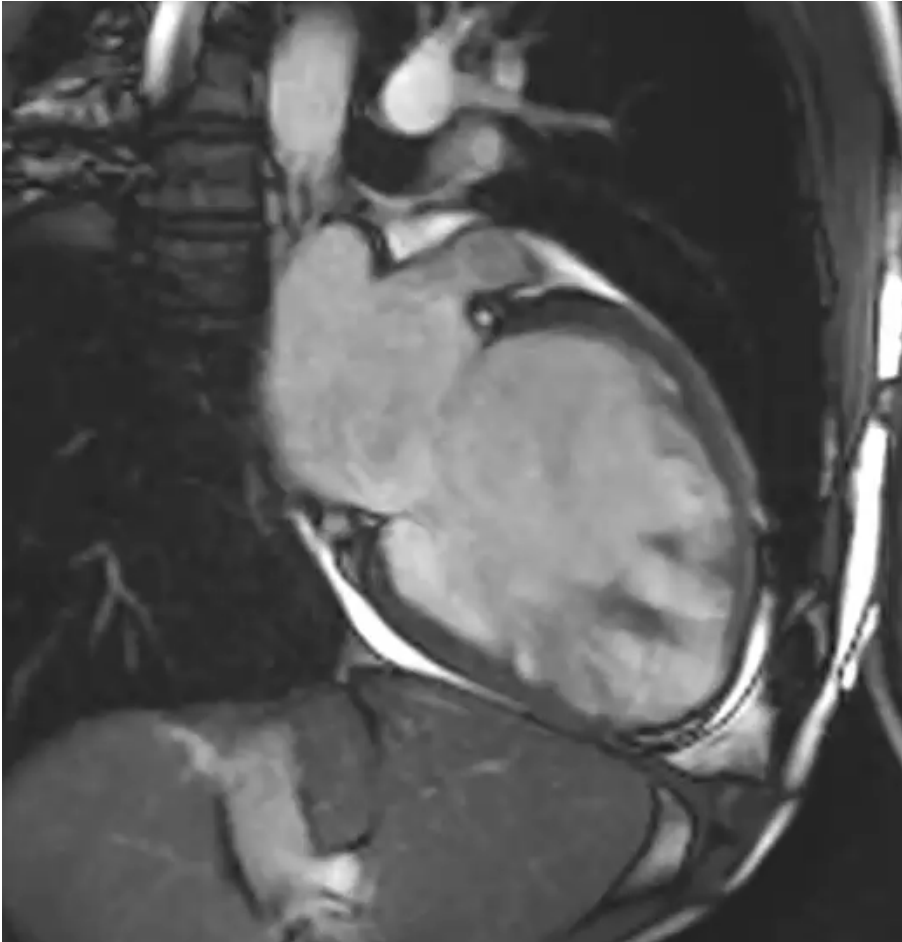
- Homme, 50 ans
- découverte d'une CMD FE 23 %.
- ECG : BBG incomplet
- Coronarographie ras

EF à 22 %
EDVI 157 ml/m²



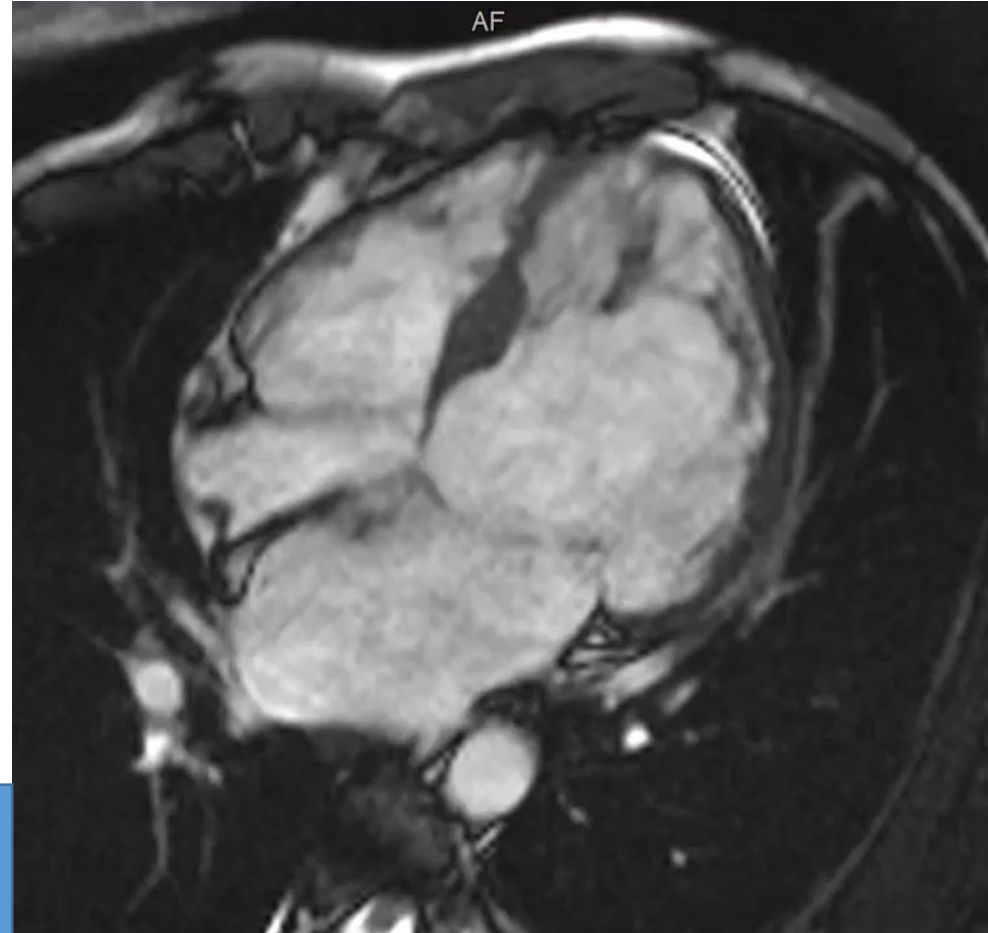
Non compaction

CMD + Iao II-III.

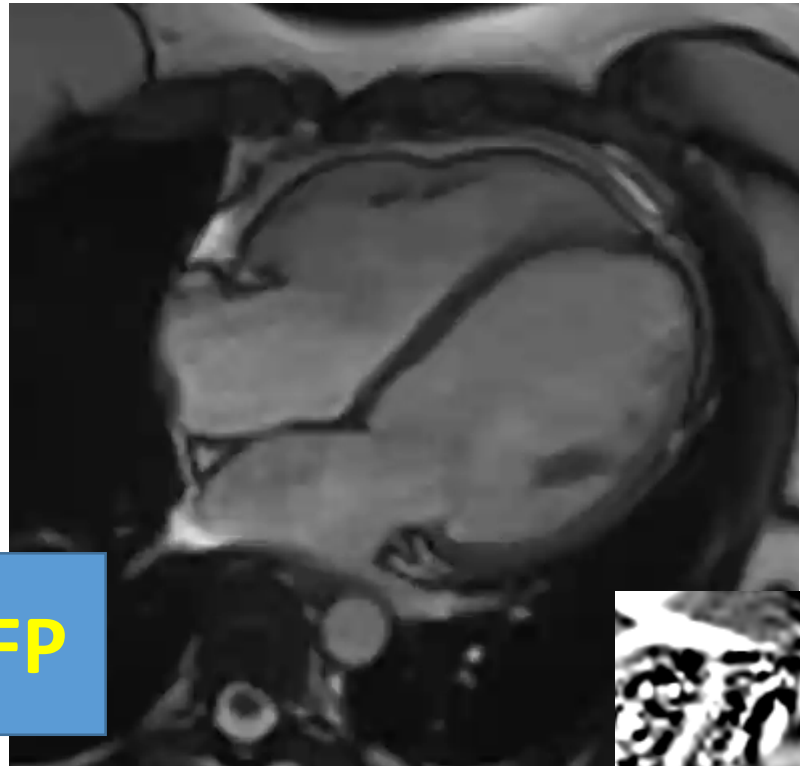


SSFP

FE 31 %, EDVI 182 ml/m²



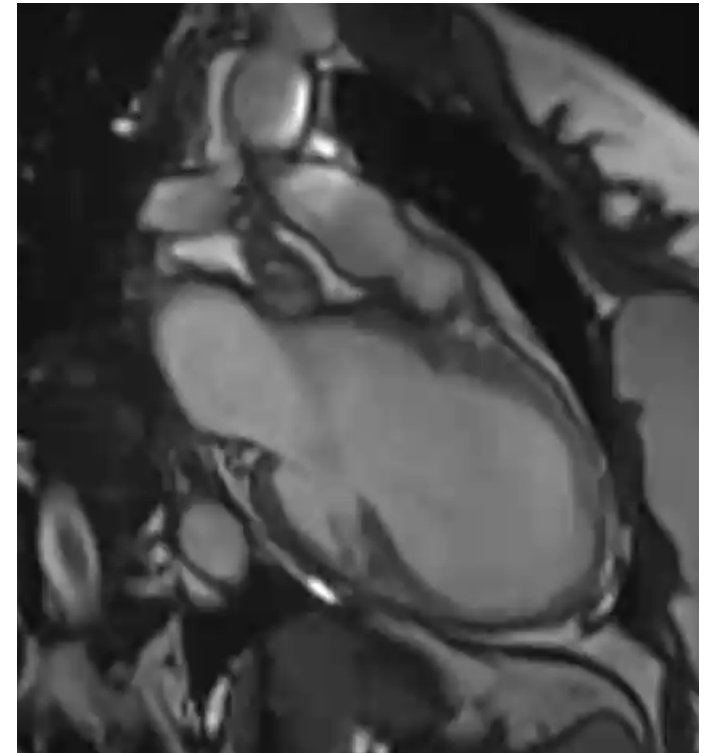
transplantation orthotopique l'année suivant



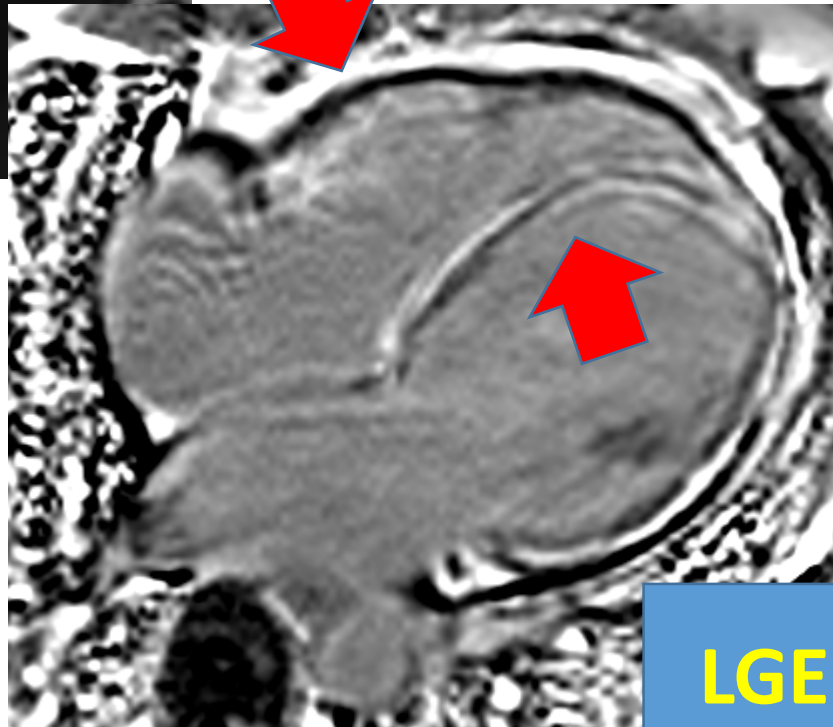
SSFP

Femme, 56 ans
Suivi CMD

Enquête génétique non concluants
(mère porteuse d'une CMD).



SIV paradoxal mais...
BBD incomplet



LGE

Cardiomyopathie
arythmogène
biventriculaire (ACM)

~~DAVD~~

2019 HRS expert consensus statement on evaluation, risk stratification, and management of arrhythmogenic cardiomyopathy

<https://doi.org/10.1016/j.hrthm.2019.05.007>

critère >

Regional RV akinesia or dyskinesia or dyssynchronous RV contraction and 1 of the following:

- a) Ratio RVEDV/BSA ≥ 110 mL/m² (male), ≥ 100 mL/m² (female)
- b) RVEF $\leq 40\%$

Infiltration ~~X~~ graisseuse

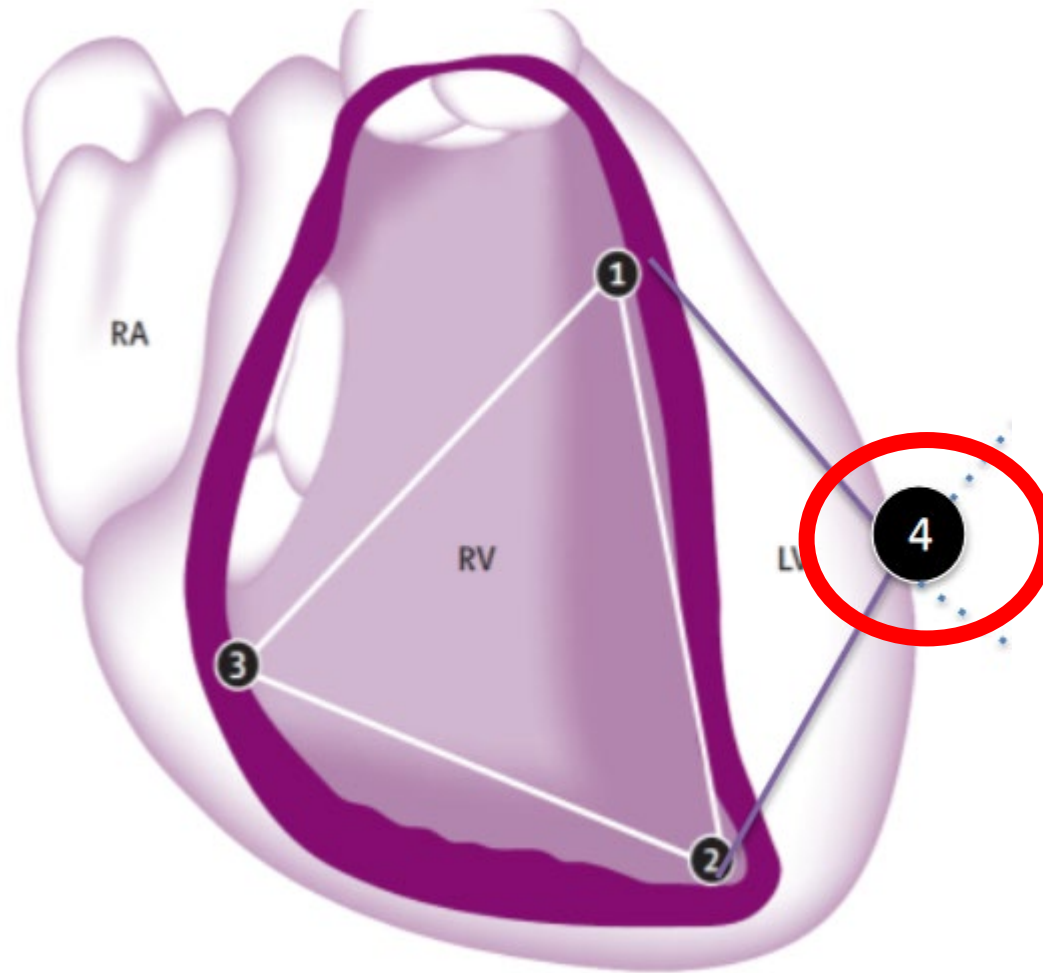
critère <

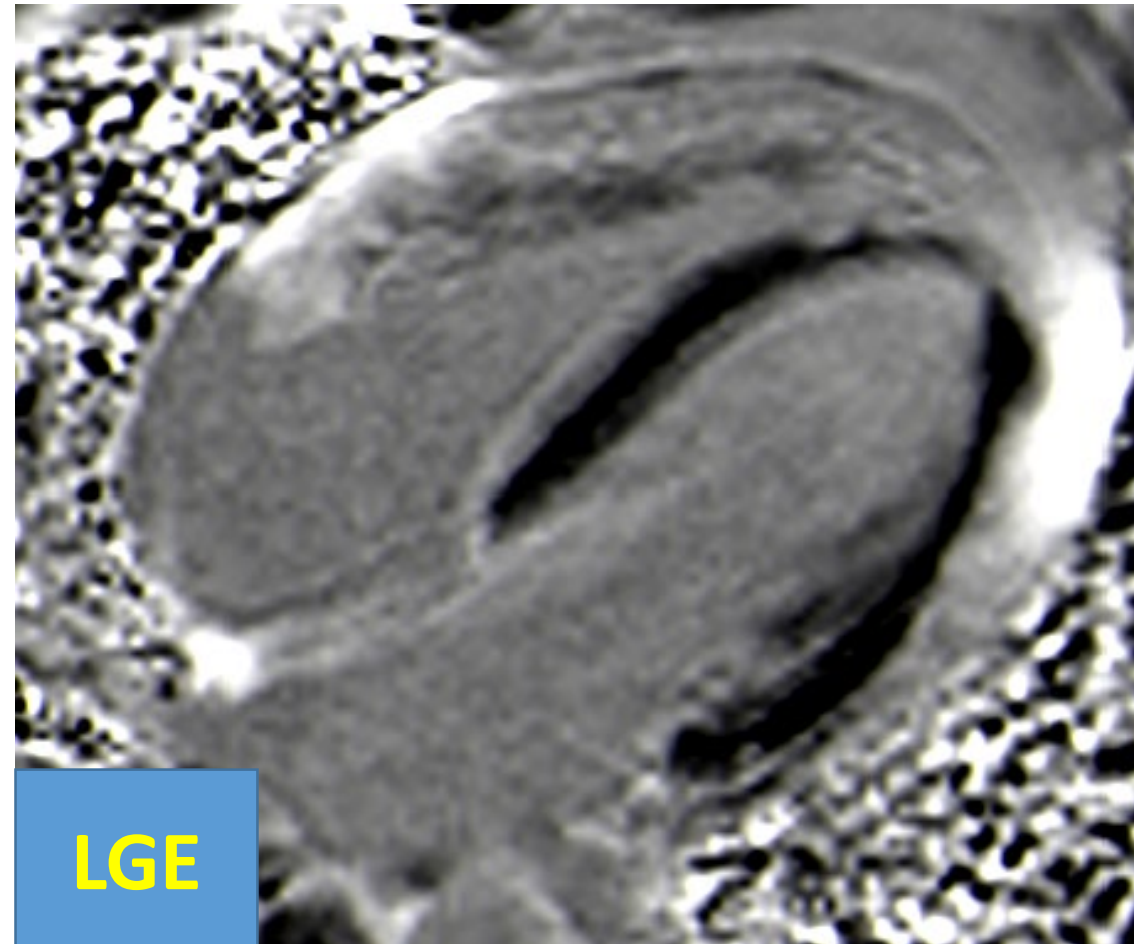
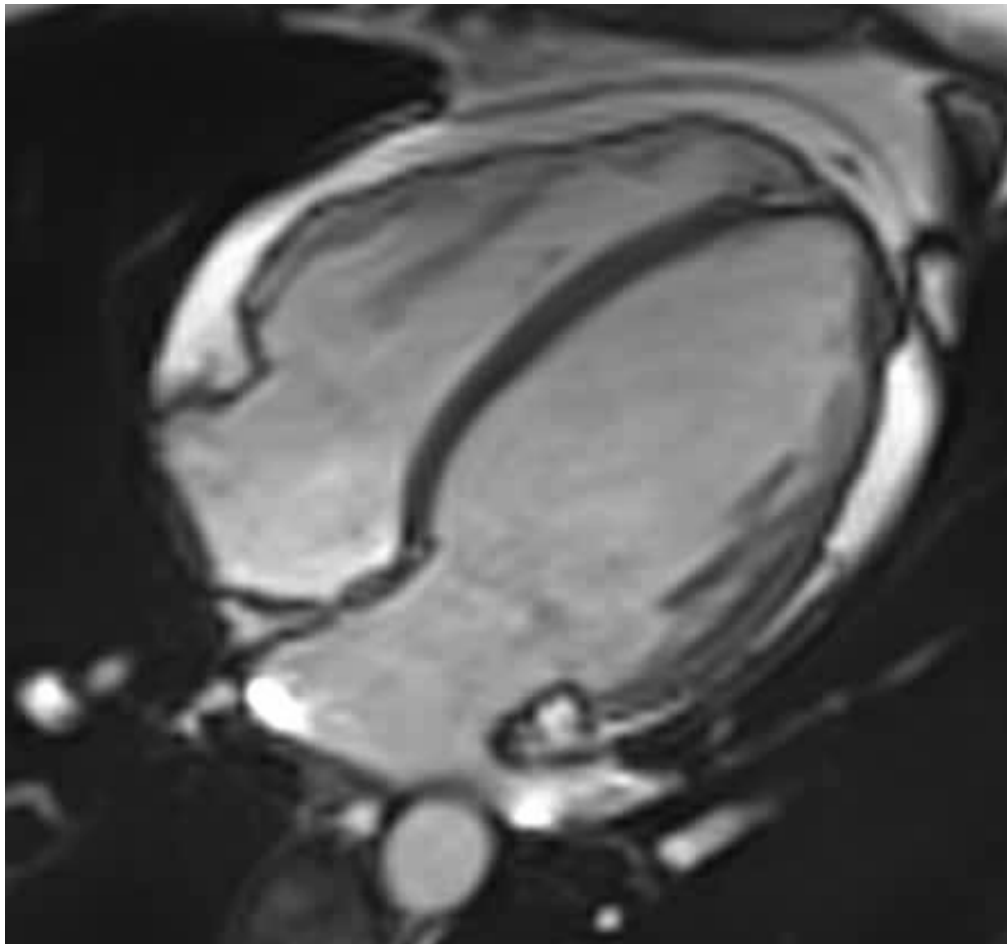
Regional RV akinesia or dyskinesia or dyssynchronous RV contraction and 1 of the following:

- a) Ratio RVEDV/BSA ≥ 100 to < 110 mL/m² (male), ≥ 90 to 100 mL/m² (female)
- b) RVEF > 40 to $\leq 45\%$

LX ~~E~~

Triangle Quadrangle de la dysplasie

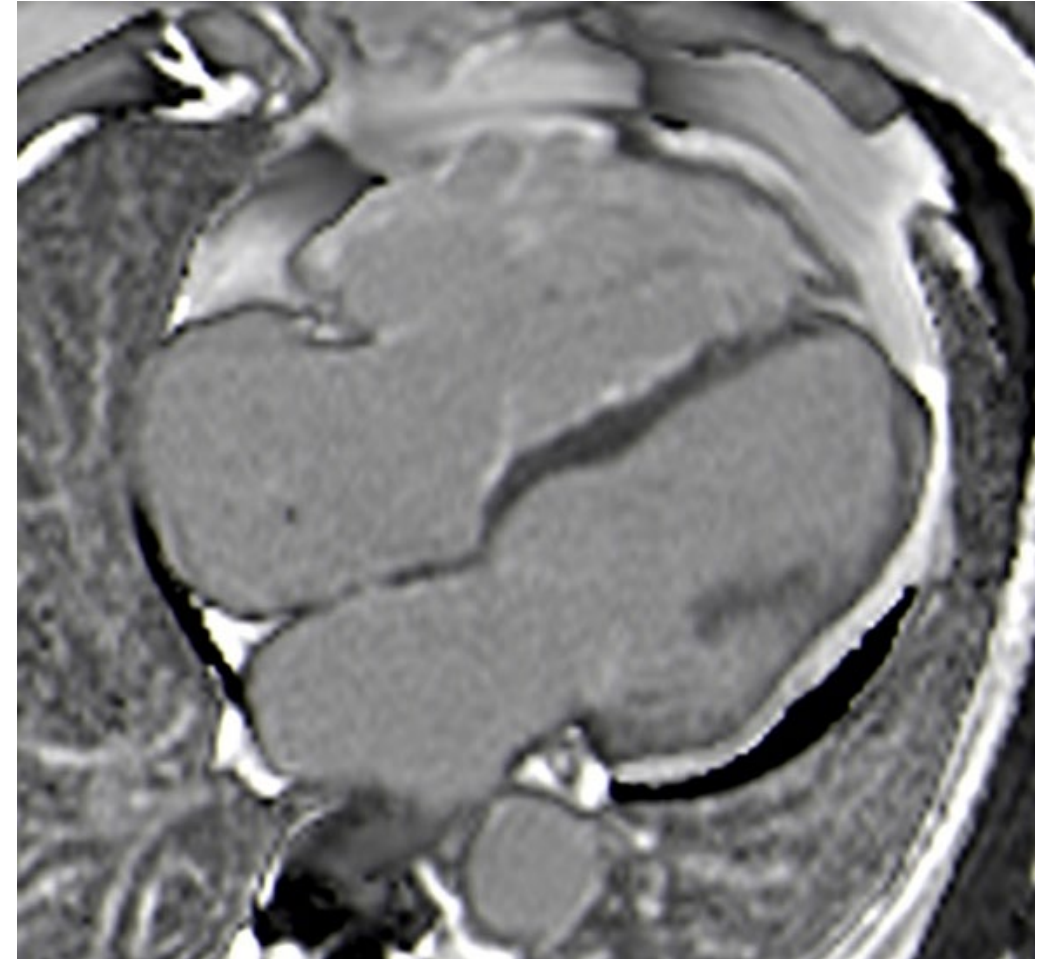
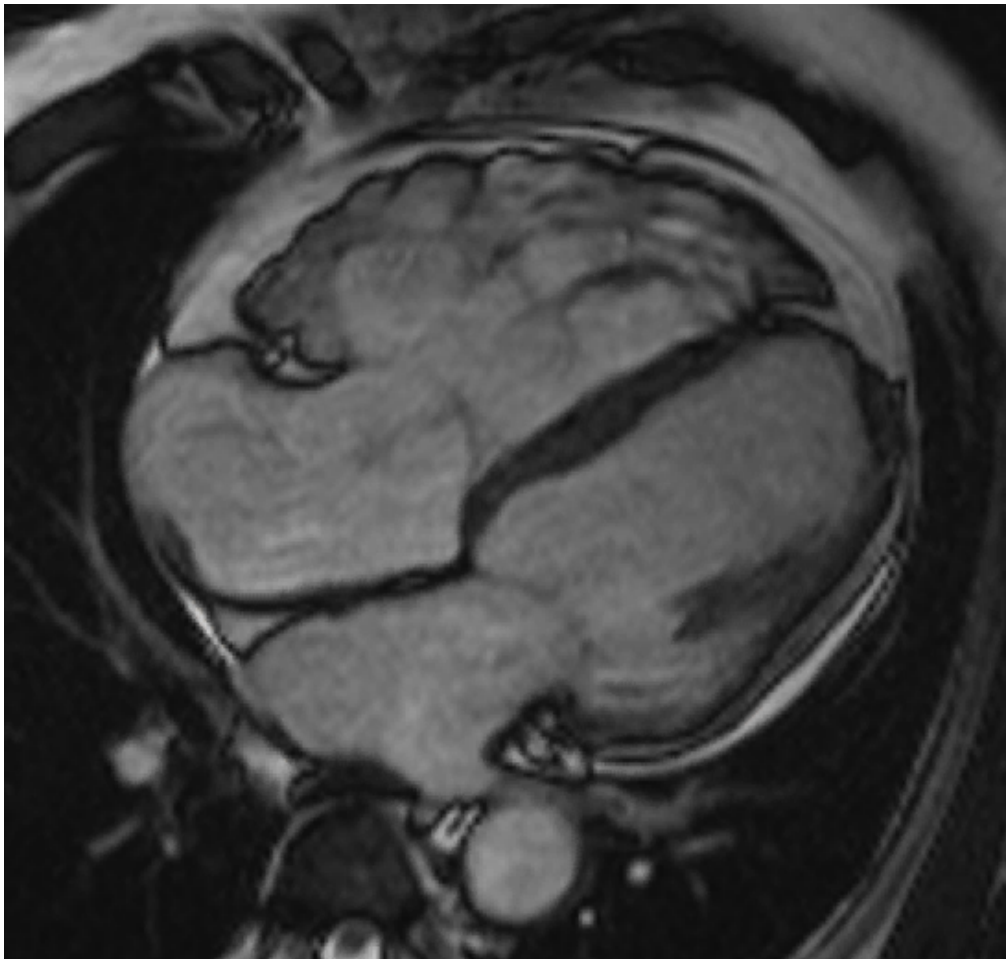




LGE

Homme, 59 ans....

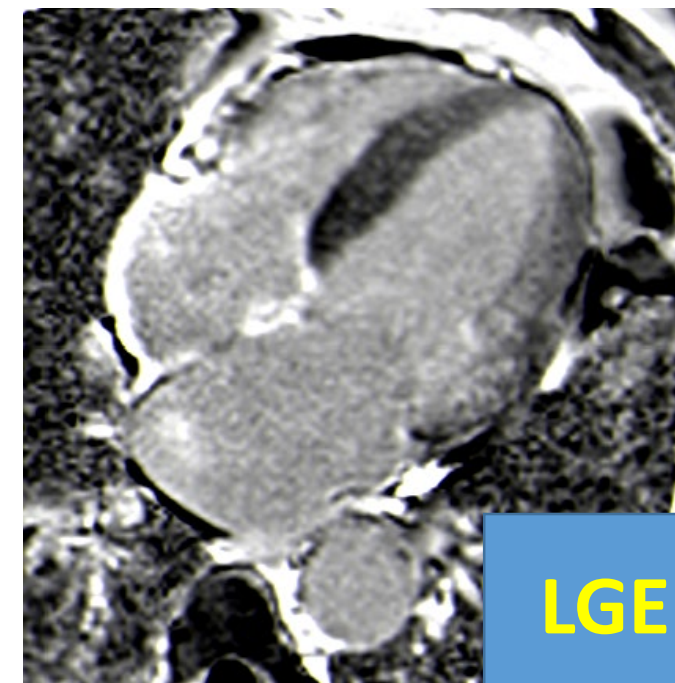
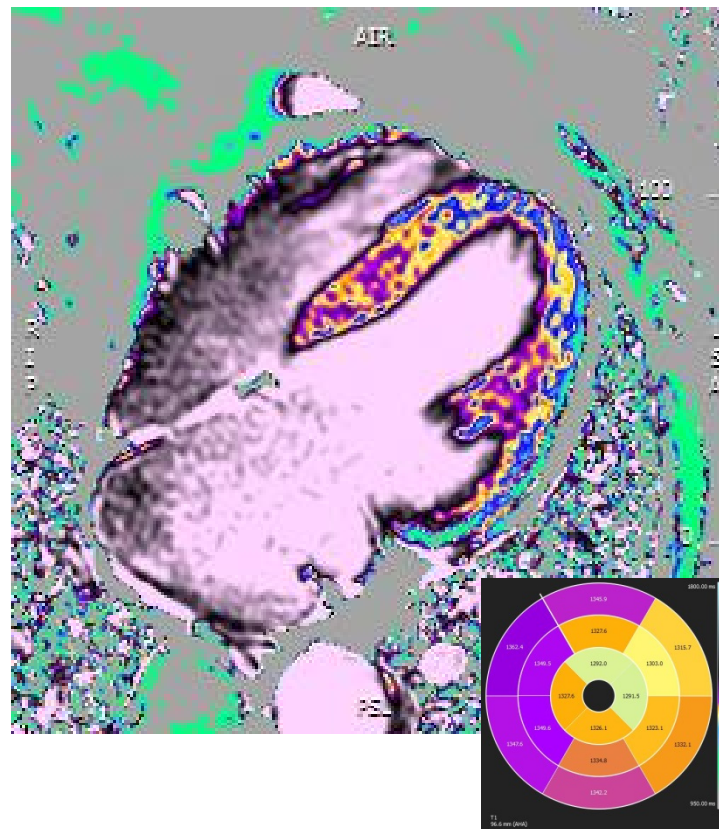
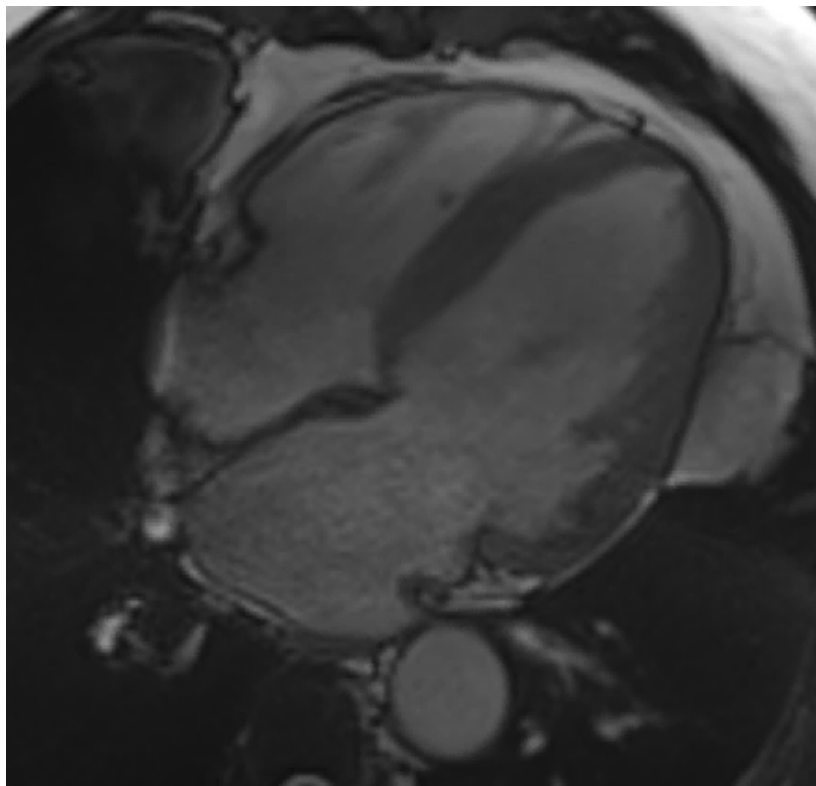
DAVD connue depuis 6 ans et stable au point de vue clinique et électrique sous bêtabloquants et Cordarone.



Diagnostic différentiel hypertrophie VG

- **Primitive** sarcomerique
- **Secondaire**
 - athlète
 - Rao
 - HTA
 - Coarctation
 - Infiltration (amylose, Fabry)

Home, 64 ans, HTA négligée

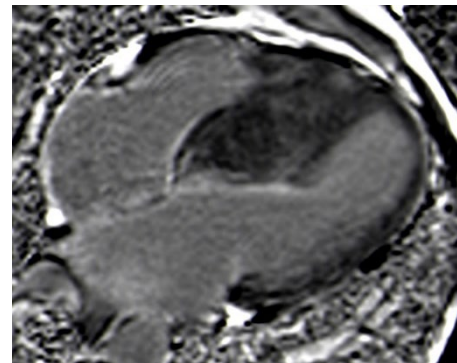
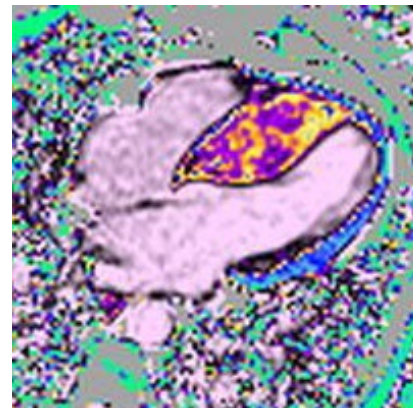
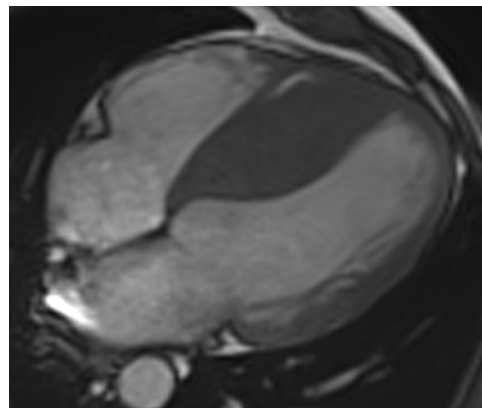
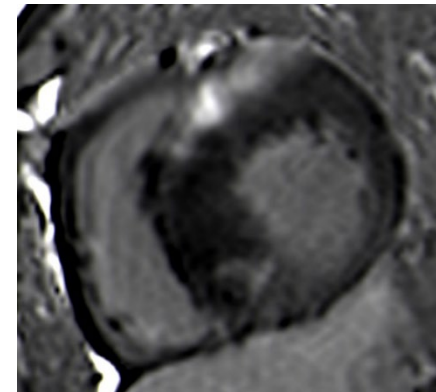
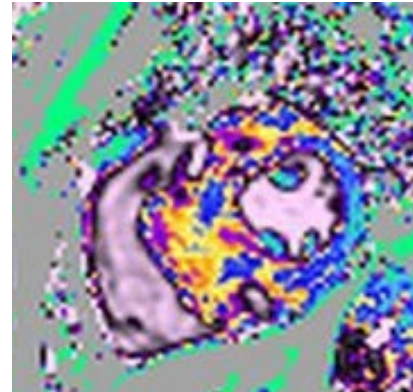
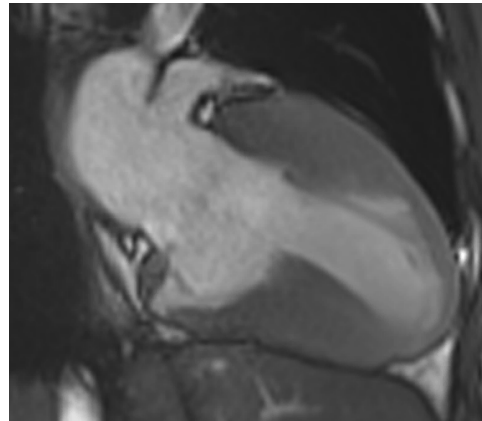
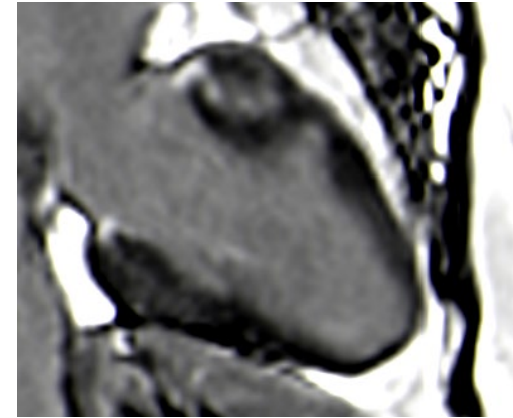
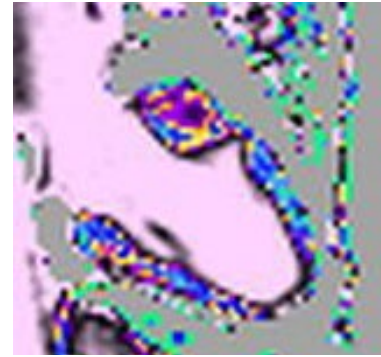
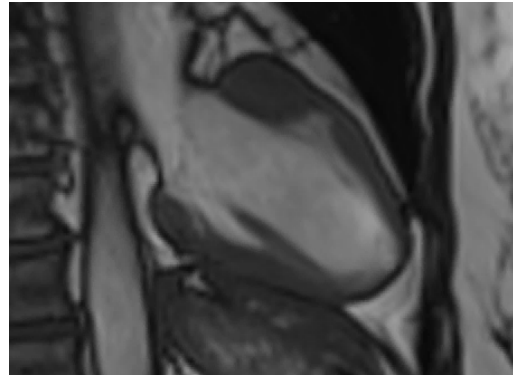


LGE

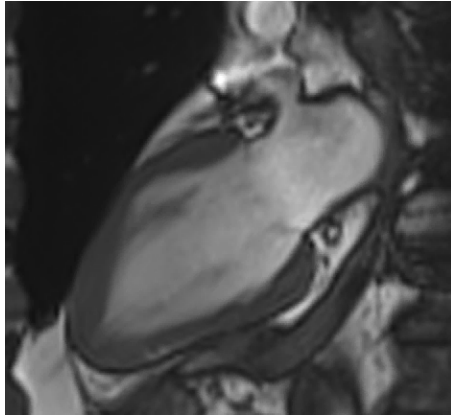
Mère, 63 ans

CMH sarcomerique

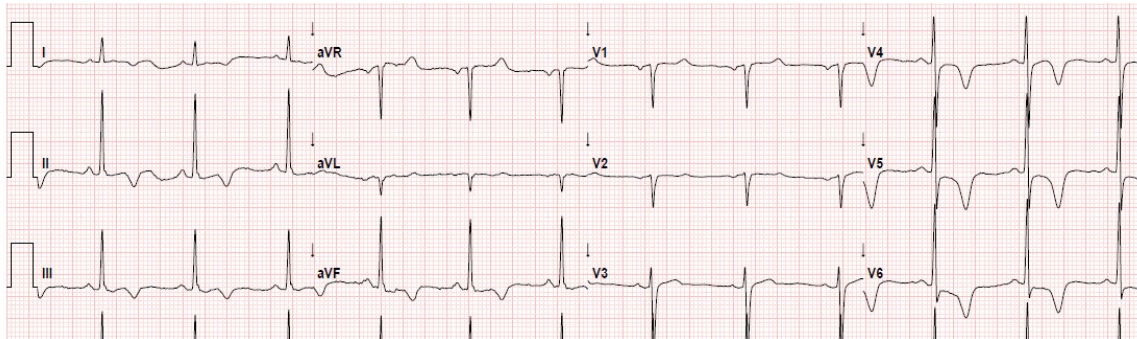
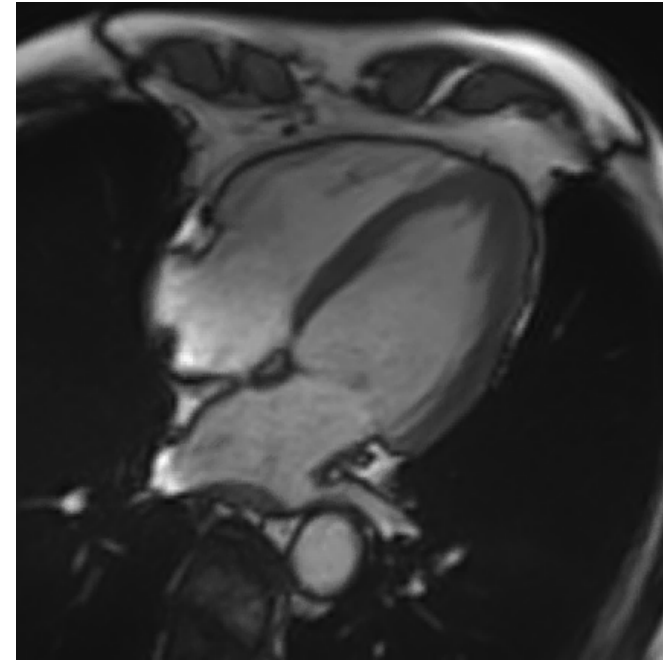
Fils, 39 ans



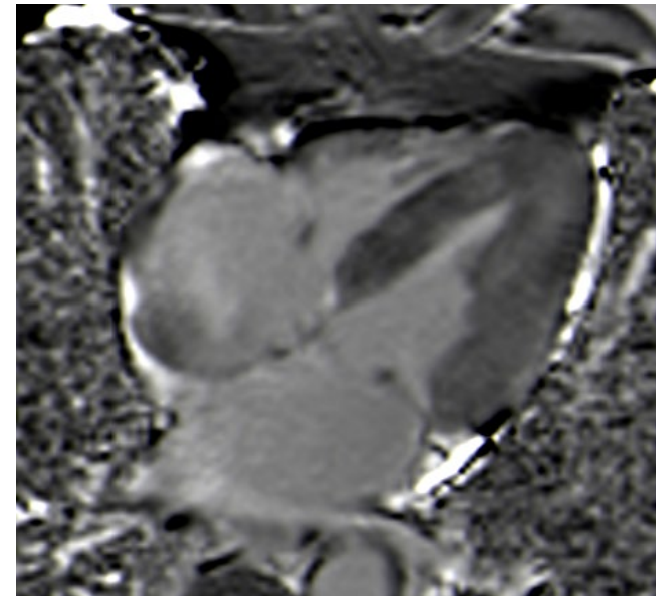
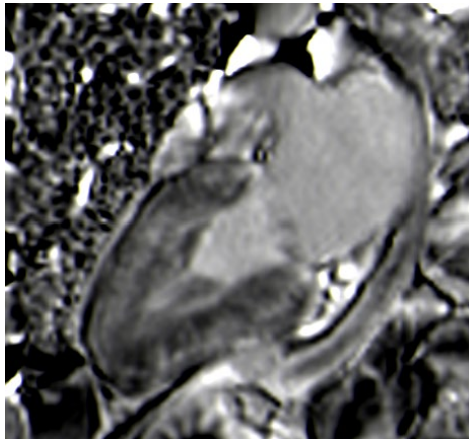
SSFP



Homme, 69 ans
Bilan HVG



LGE



RESEARCH

Open Access



Patients who do not fulfill criteria for hypertrophic cardiomyopathy but have unexplained giant T-wave inversion: a cardiovascular magnetic resonance mid-term follow-up study

Shuang Li^{1†}, Jian He^{1†}, Jing Xu¹, Baiyan Zhuang¹, Bailing Wu², Bingqi Wei³, Jinghan Huang⁴, Gang Yin^{1,6}, Xiuyu Chen^{1,6}, Zhenhui Zhu⁵, Hao Wang⁵, Shihua Zhao^{1*} and Minjie Lu^{1,6*} 

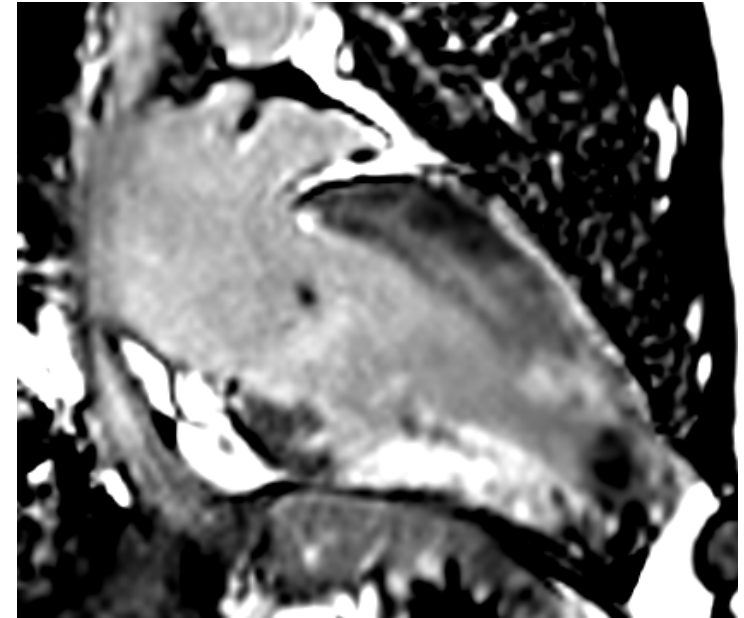
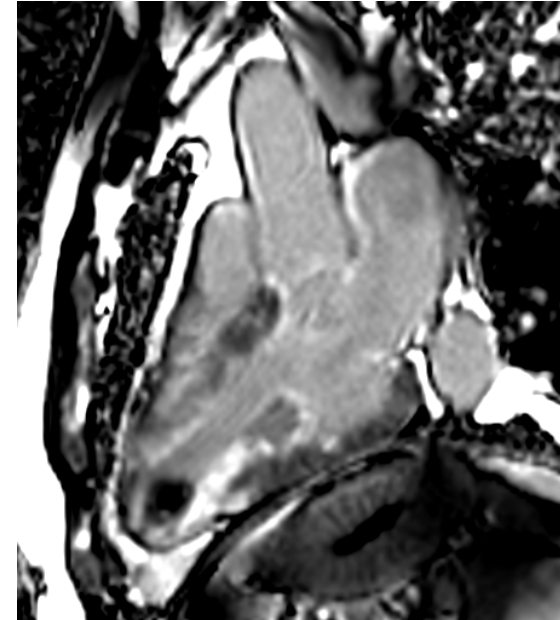
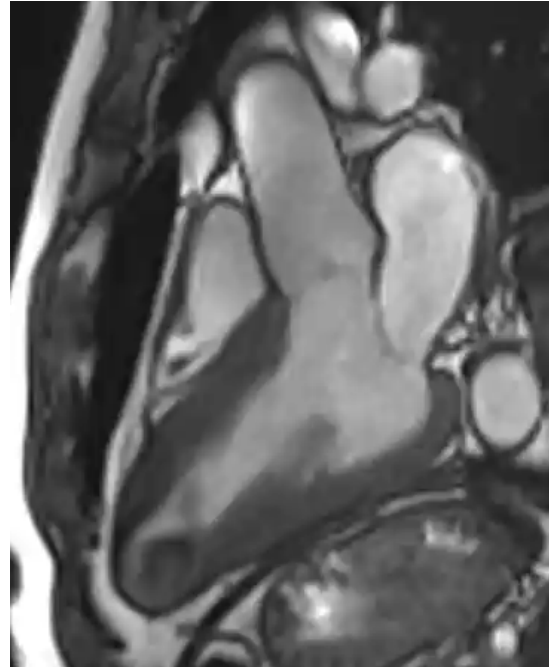
Conclusions: CMR metrics identify predictors for progression to HCM in patients with unexplained giant T-wave inversion.

63 patients en 20 ans..... 3 pat/an



CMH medio-ventriculaire: thrombus apical

Homme, 59 ans

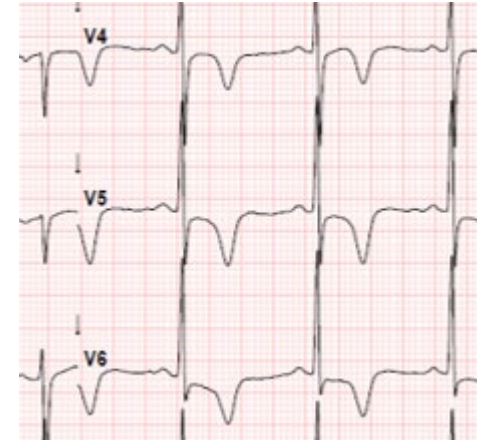


L'IRM dans la CMH

- Morphologie:

symétrique

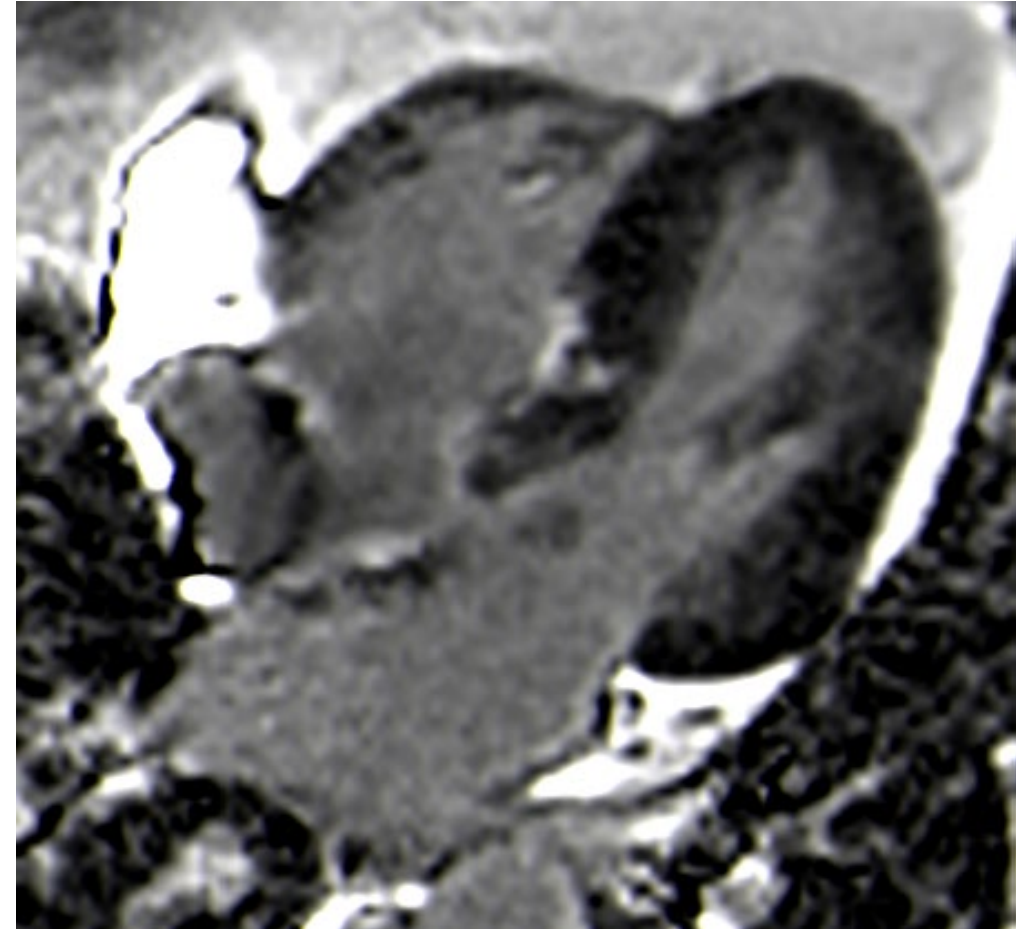
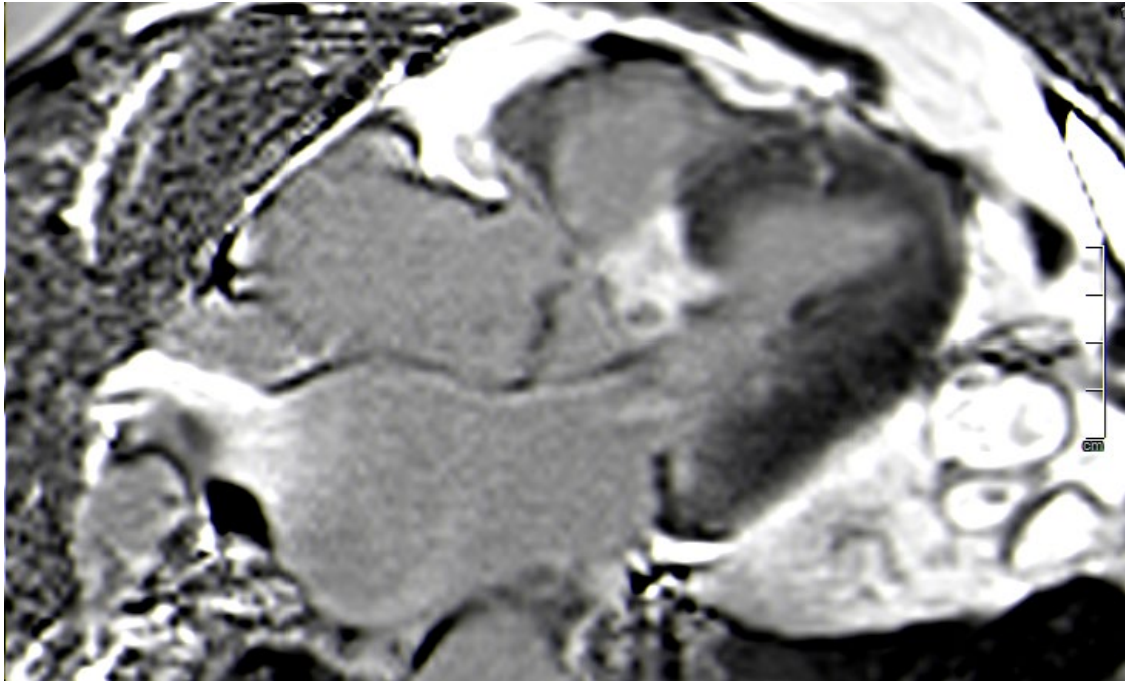
asymétrique (apicale!!!)



- Quantification de la masse, épaisseur SIV
- Fibrose (T1 mapping – LGE)
- Complication: (obstruction), thrombus apical
- DD HGV : HTA, sportif, maladie infiltrative (amylose, Fabry)

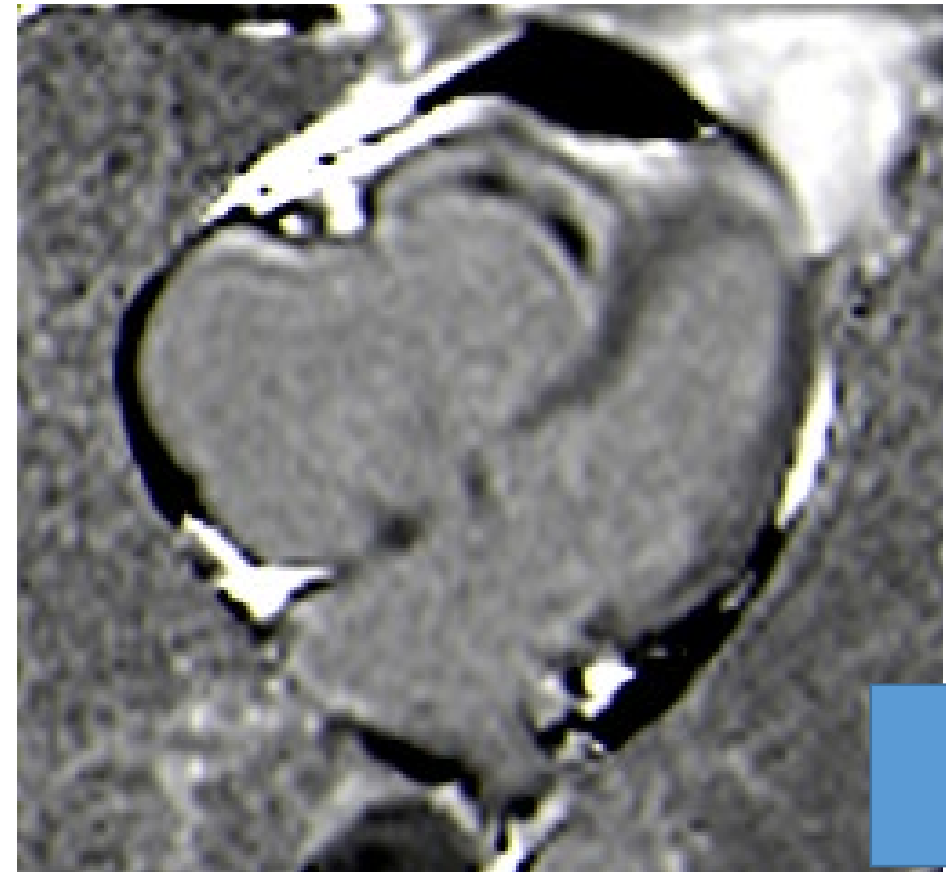
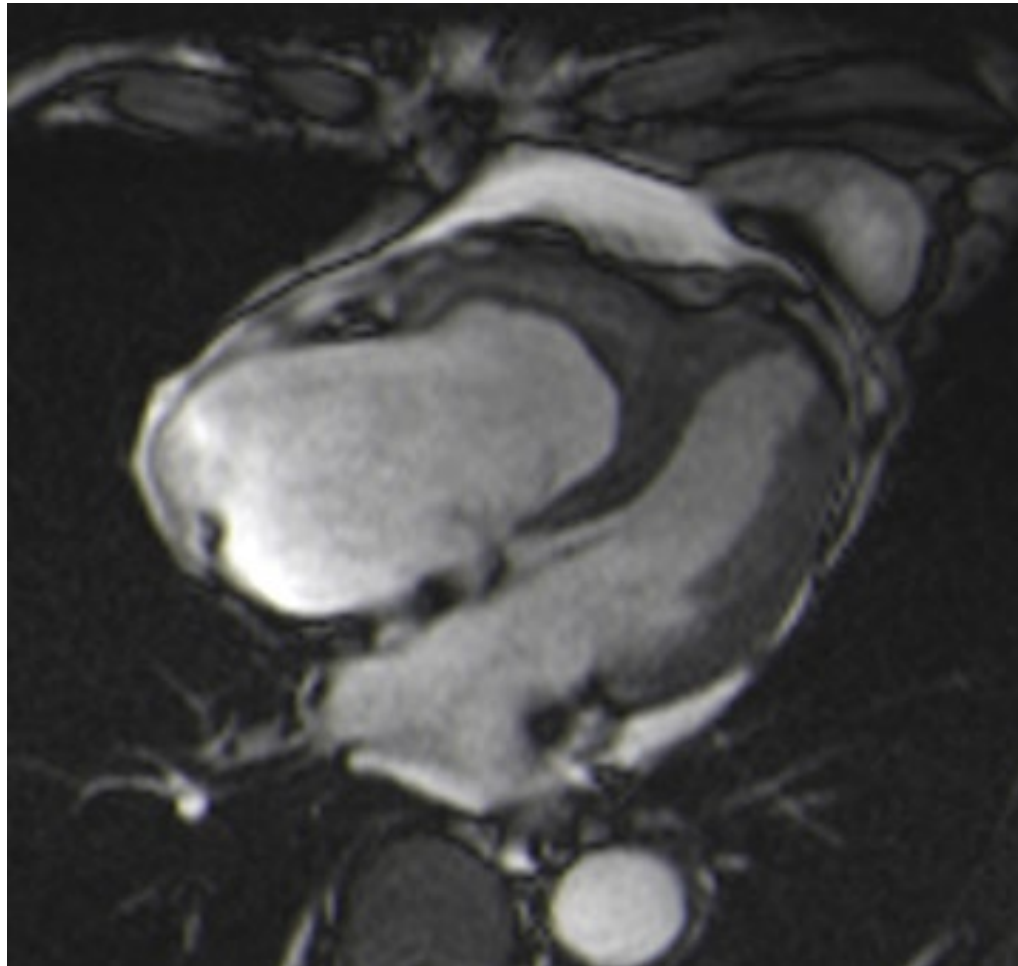


Après alcoolisation



CMP restrictive

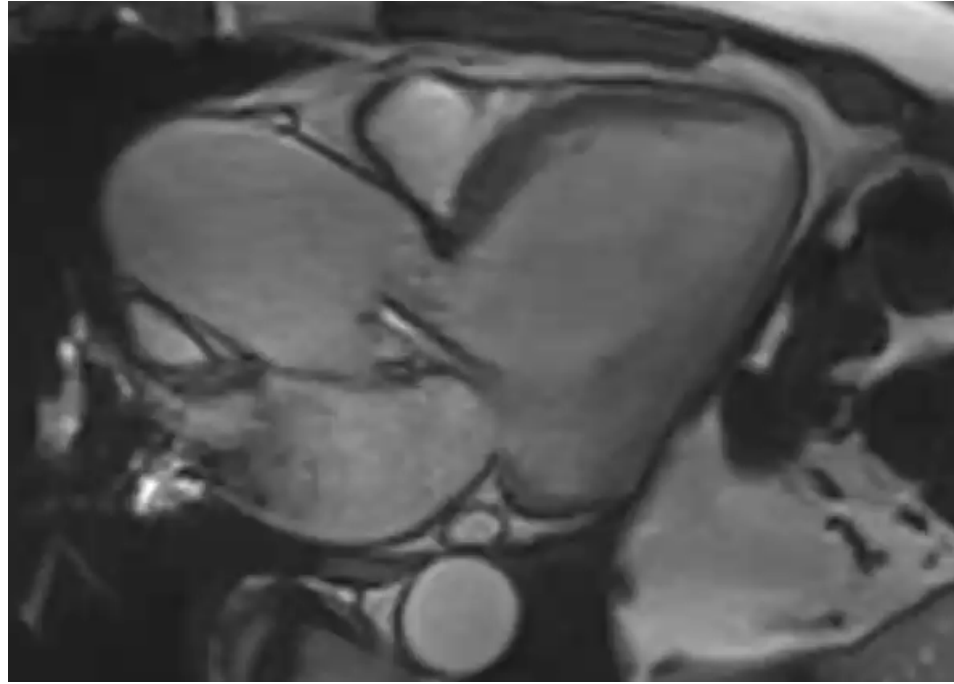
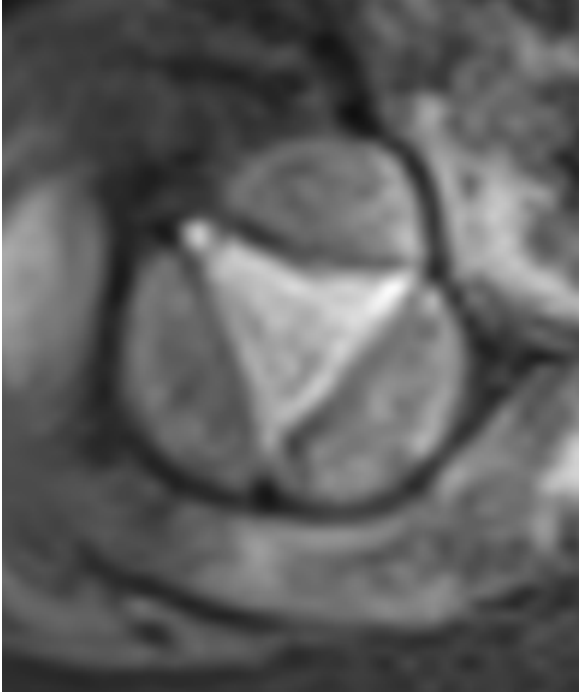
- Homme, 79 ans, maladie de Vaquez.
- **fibrose endomyocardique** du VD, avec signes d'insuffisance cardiaque droite.



LGE

Pathologie de la valve aortique et aorte thoracique

Femme, 76 ans; dilatation aorte ascendante, IAo



- Valve aortique tricuspide; IAo sévère (FR à 45%)
- EF à 48 %, EDVI et ESVI à 123 et 64 ml/m², masse à 98 g/m².
- Valsalva dilatée à 55 mm (Z score à 4.8).
- Aorte ascendante dilatée à 53 mm (Z score à 4.6).



Calcul du z-score du diamètre aortique

Plusieurs méthodes de calcul vont sont proposées ci-dessous

Calculs

Niveau Aortique*

Tubulair

Diamètre aortique (mm)*

46

Genre*

Feminin

Age (années)*

67

Taille (cm)*

173

Poids (Kg)*

58

Surface Corporelle (Dubois)

1.69 m2

RESULTATS

Selon Campens

Basé sur Campens, L. et al. 2014. Am J Cardiol 114: 914-920.

Limite supérieure diamètre normal
(Campens)

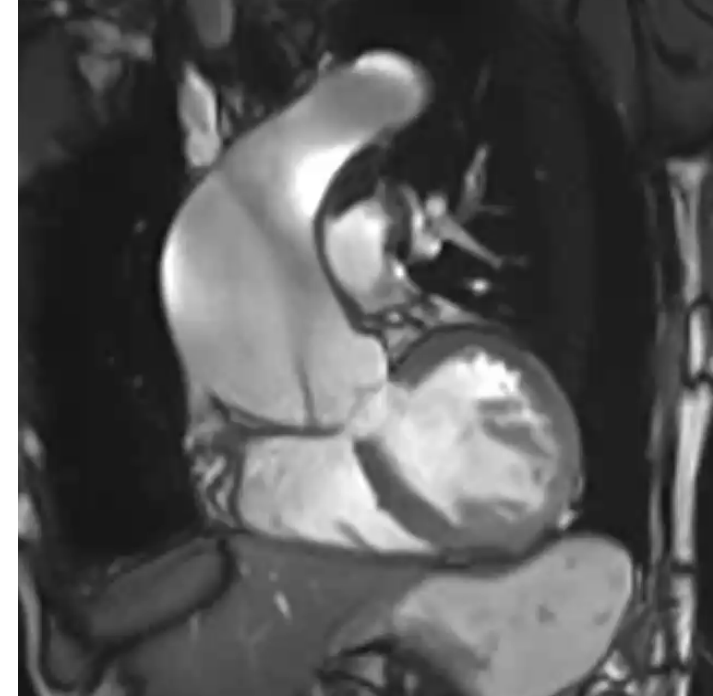
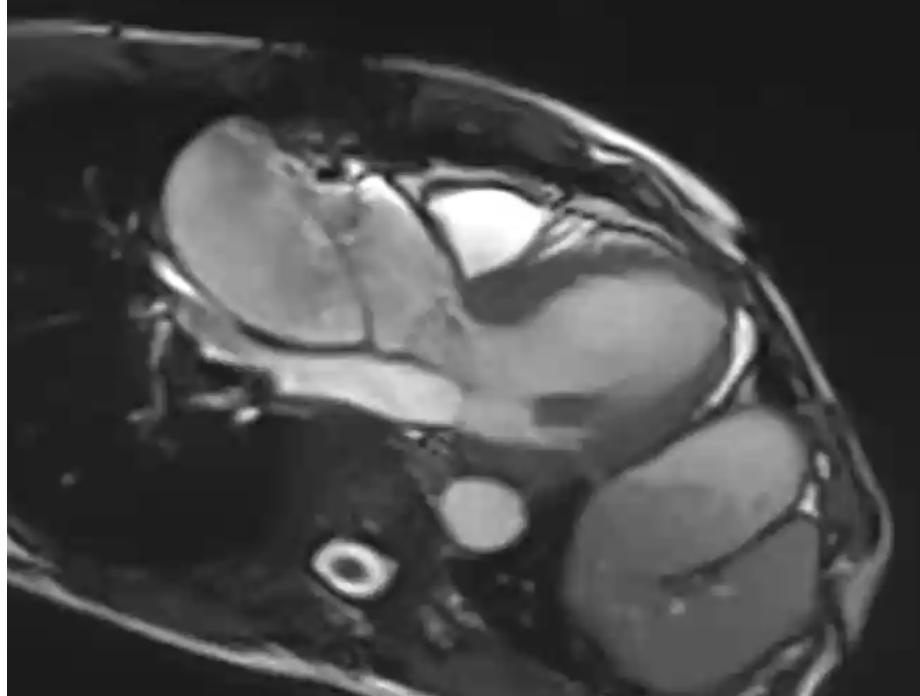
36.18406

Z-Score (Campens)

4.261114

Femme, 67 ans

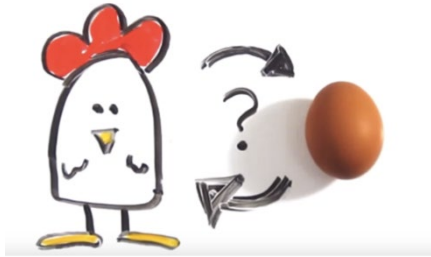
- Femme, 67 ans
- ATCD d'ablation d'un myxome OG + pontage coronaire (il y a 1 ans)
- Bilan Iao avec aorte ascendante à 48 mm.
- Absence de douleurs thoraciques.
- HTA mal équilibrée.



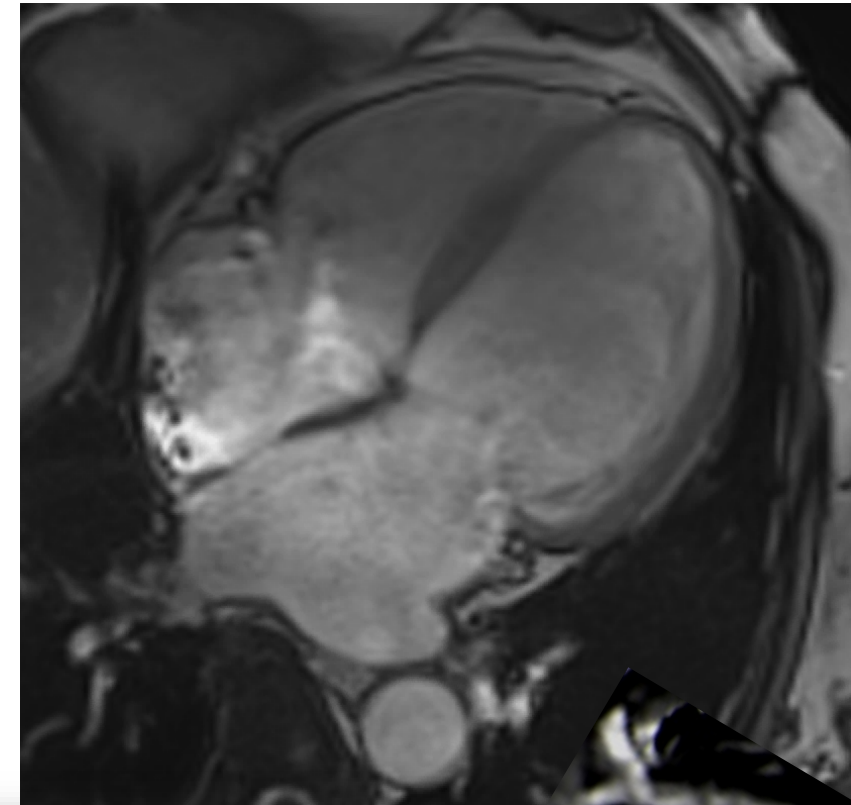
Aorte et valve aortique

- Etude de la valve aortique (bicuspidie???)
- Evaluation sténose (planimétrie)
- Quantification de l'insuffisance aortique (post TAVI !!!! plusieurs jets)
- Retentissement ventriculaire gauche
- Suivi des diamètres de l'aorte thoracique
 - Non irradiant 😊
 - Endoprothèse aorte descendante ☹️

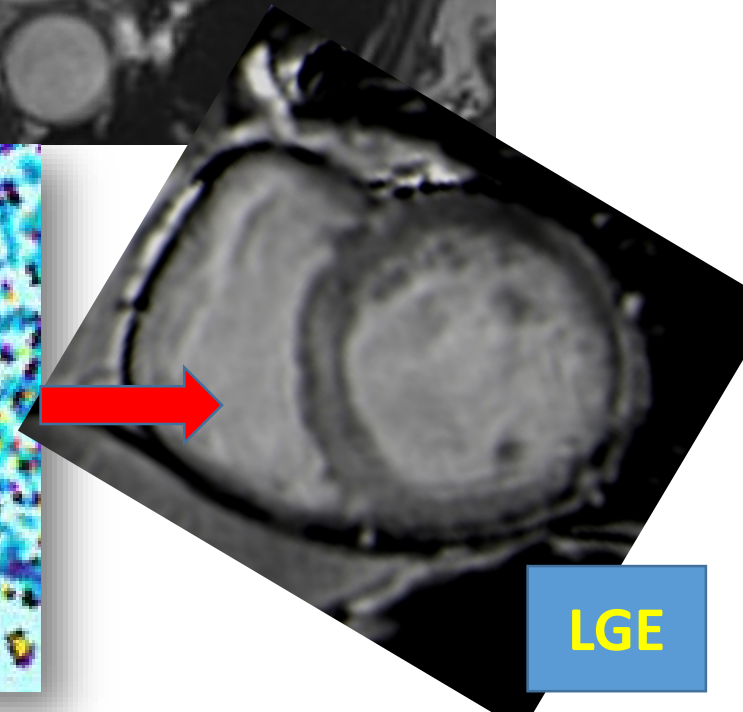
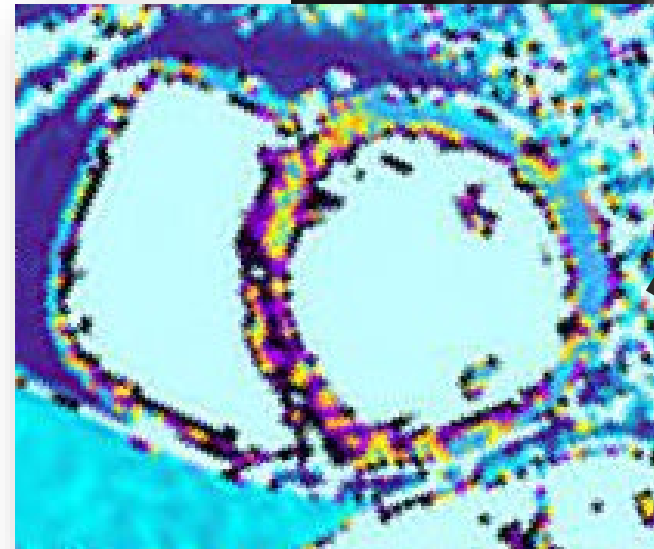
- Homme, 79 ans
- Dilatation VG + IAO moyenne



CMD
lao modérée



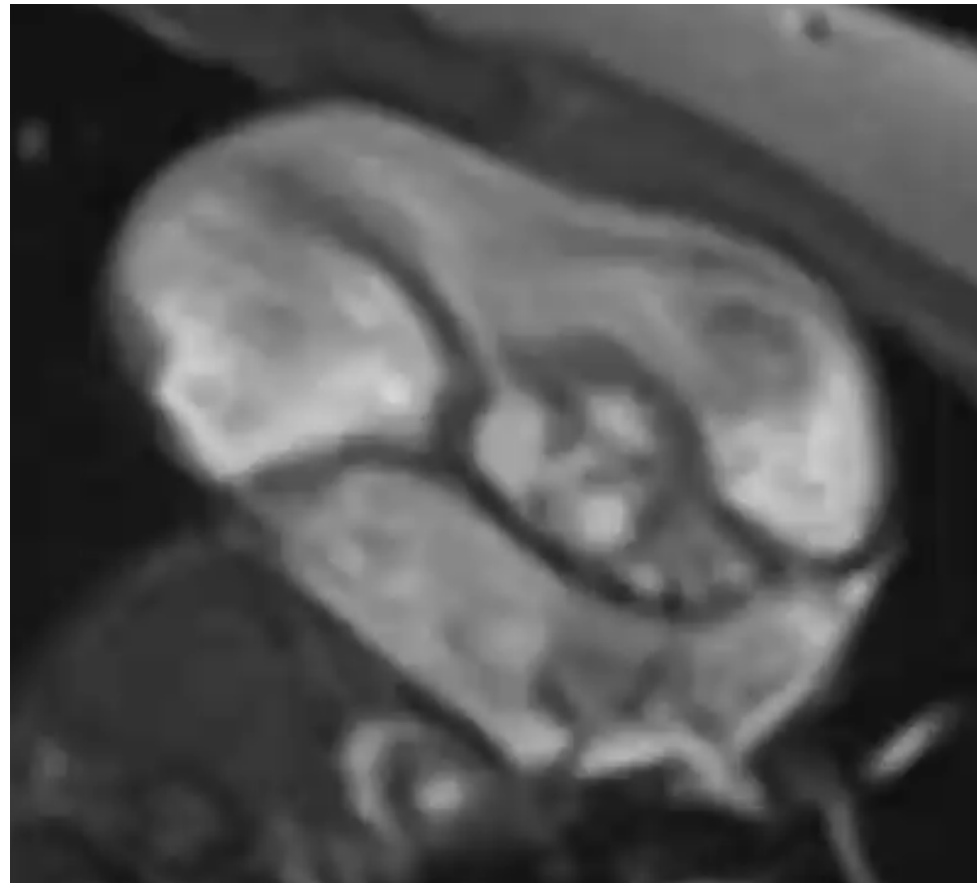
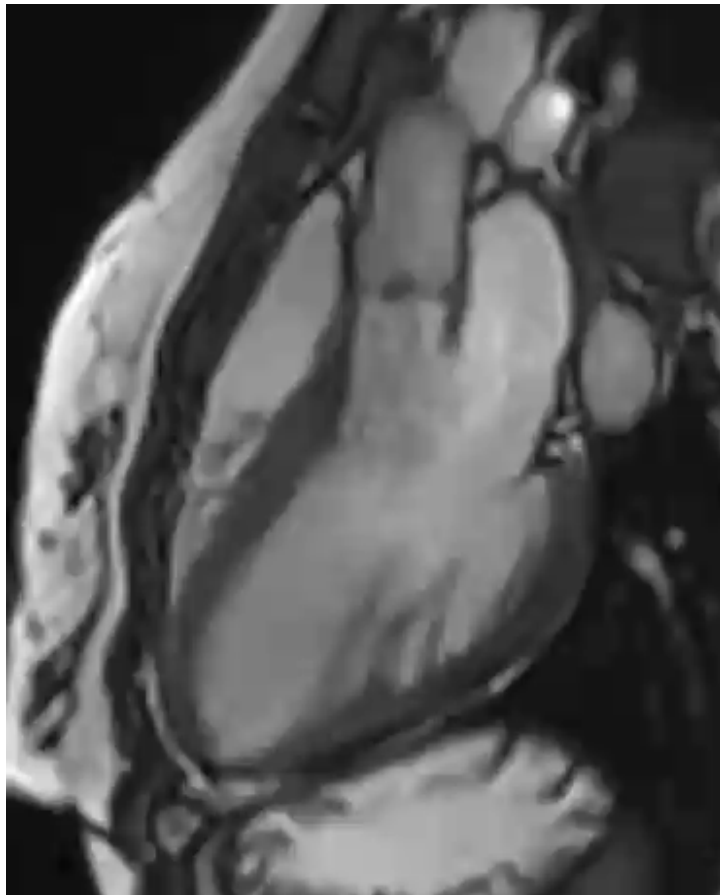
- EF à 40 %, EDVI à 143 ml/m²
- SLG à - 10 %.
- Aspect de non compaction à l'apex
- lao modérée, FR à 28 %.

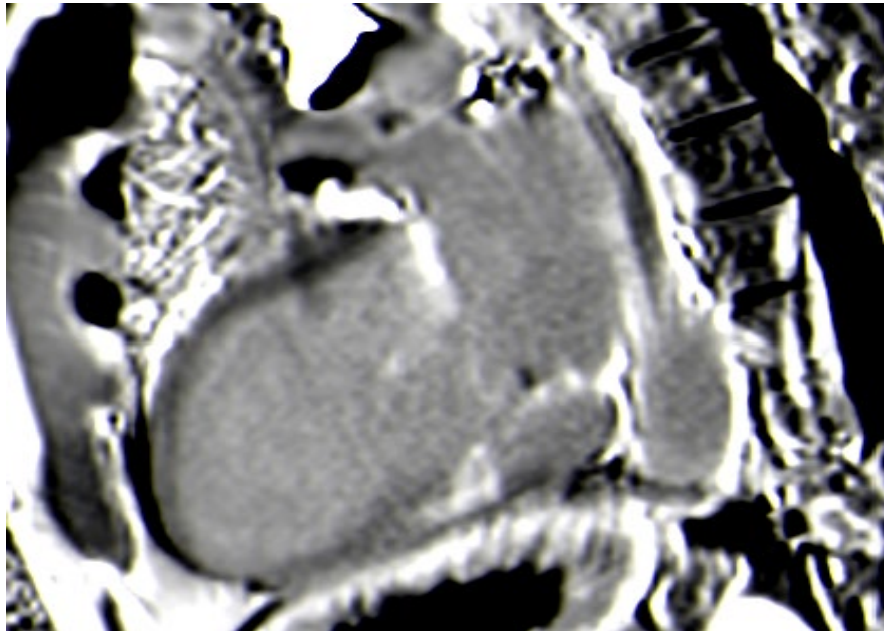


LGE

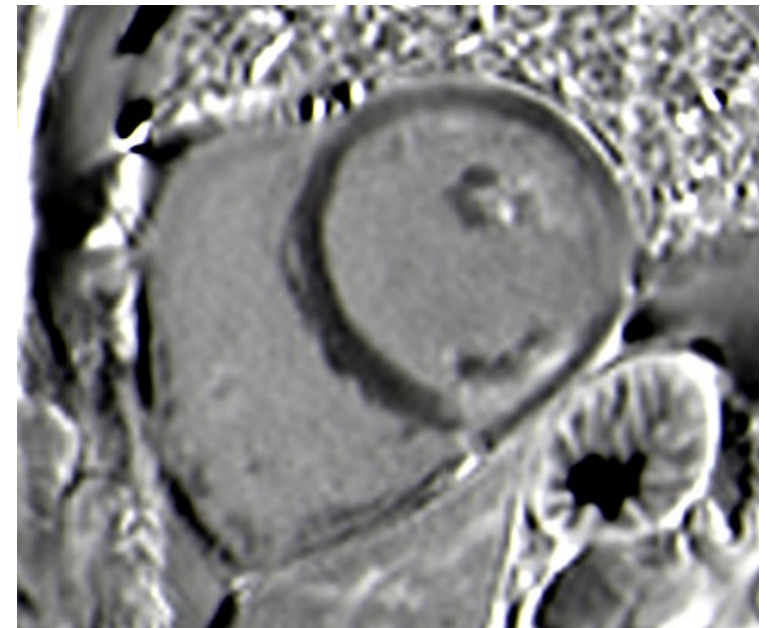
Valvulopathie lupique

- Femme, 28 ans, LES
- Maladie valvulaire aortique sévère.... Probable bicuspidie

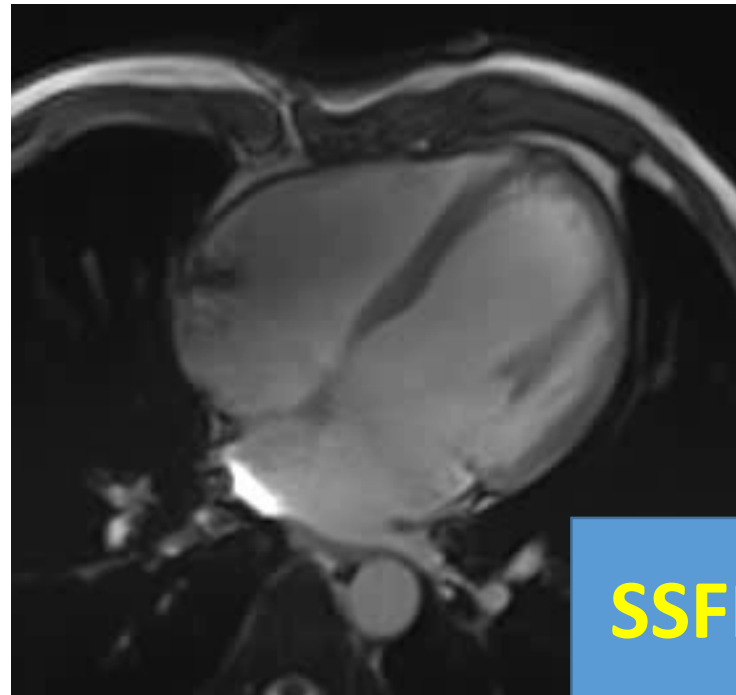




LGE



Prolapsus mitral



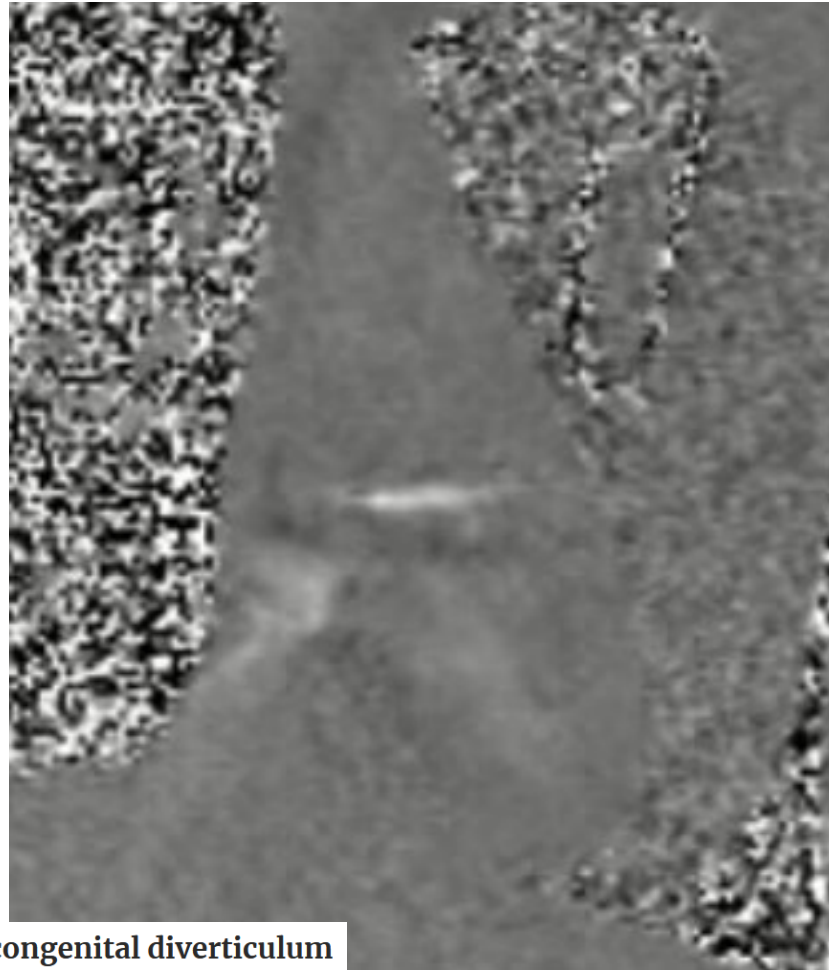
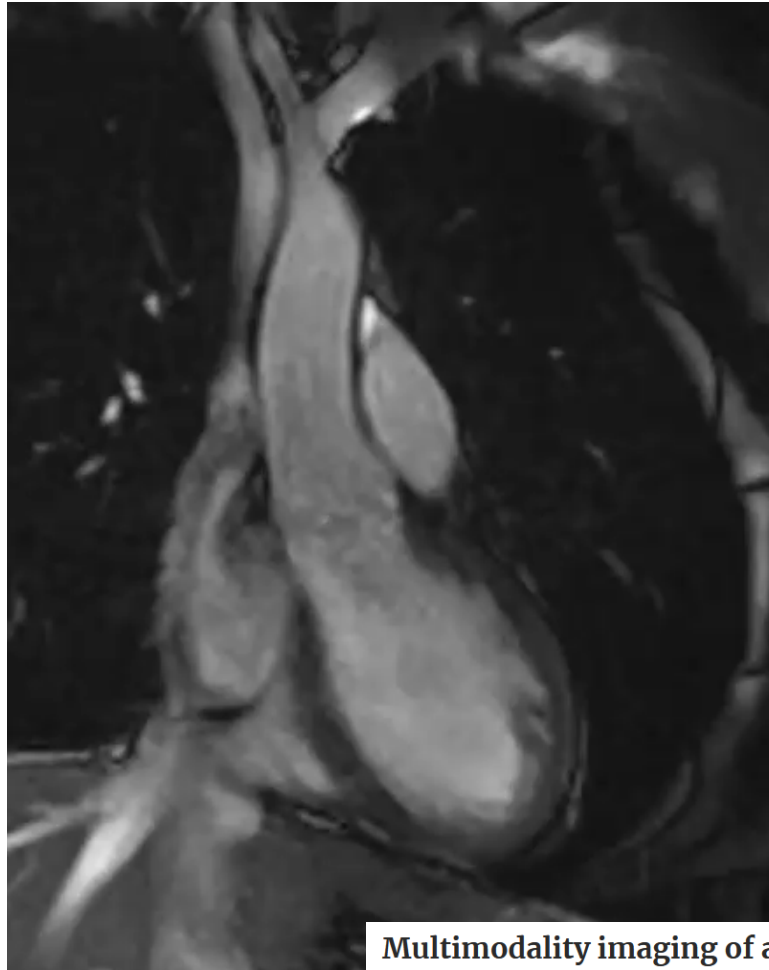
SSFP

**Arrhythmic Mitral Valve Prolapse and Sudden
Cardiac Death**

Circulation. 2015;132:556-566.
DOI: 10.1161/CIRCULATIONAHA.115.016291

Homme, 41 ans, DT suspect

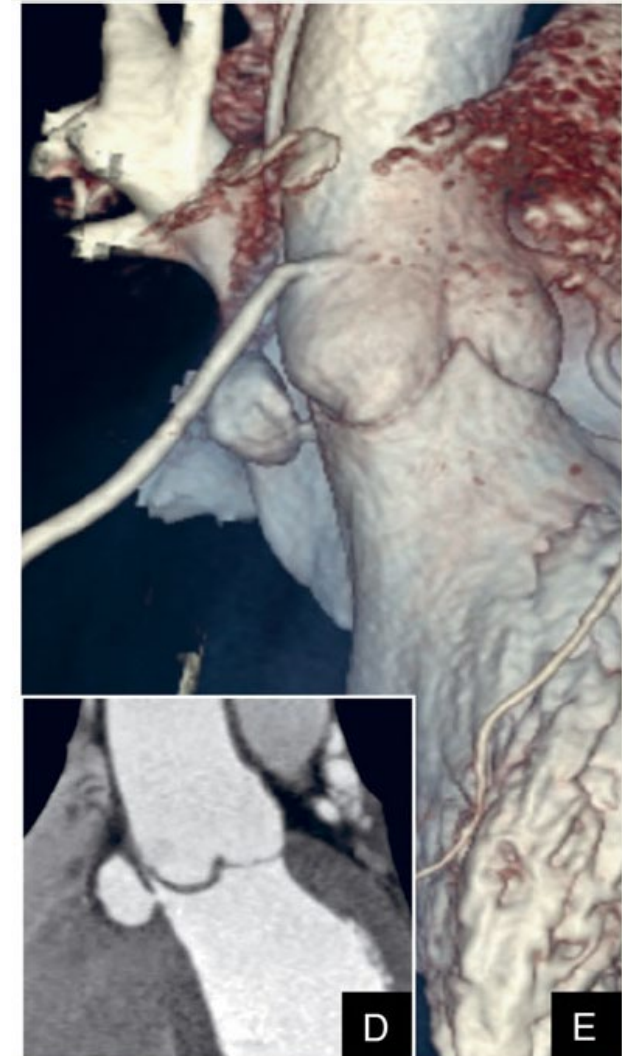
ETT: anévrisme du sinus de Valsalva non coronaire + IAo



Multimodality imaging of a congenital diverticulum of the left ventricular outflow tract 

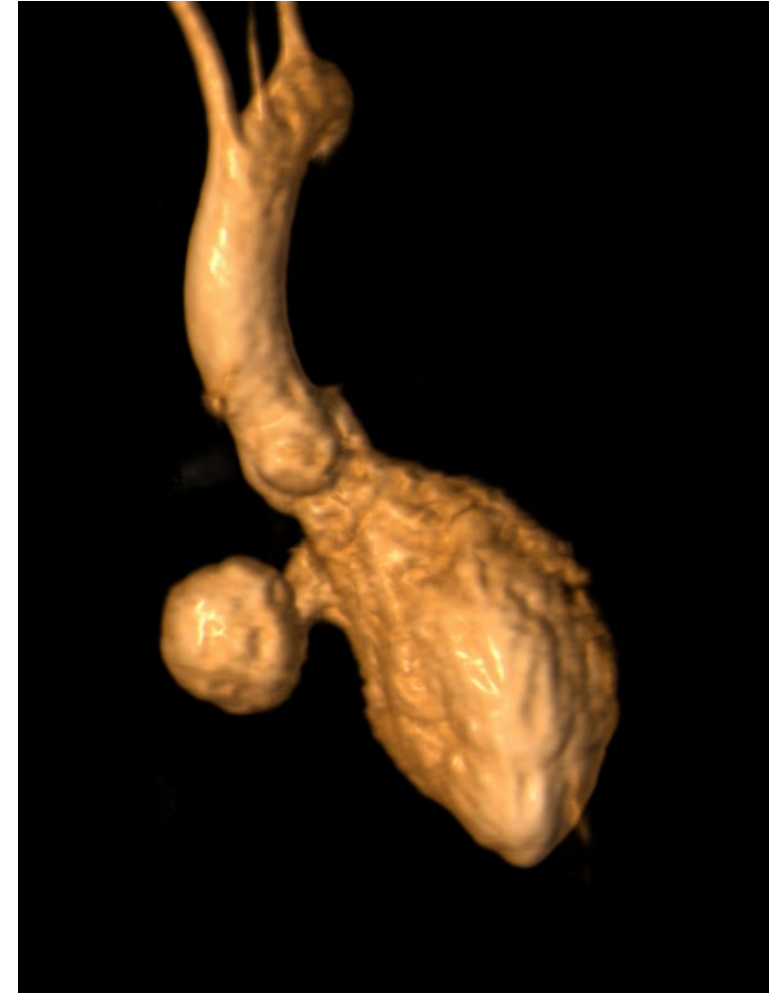
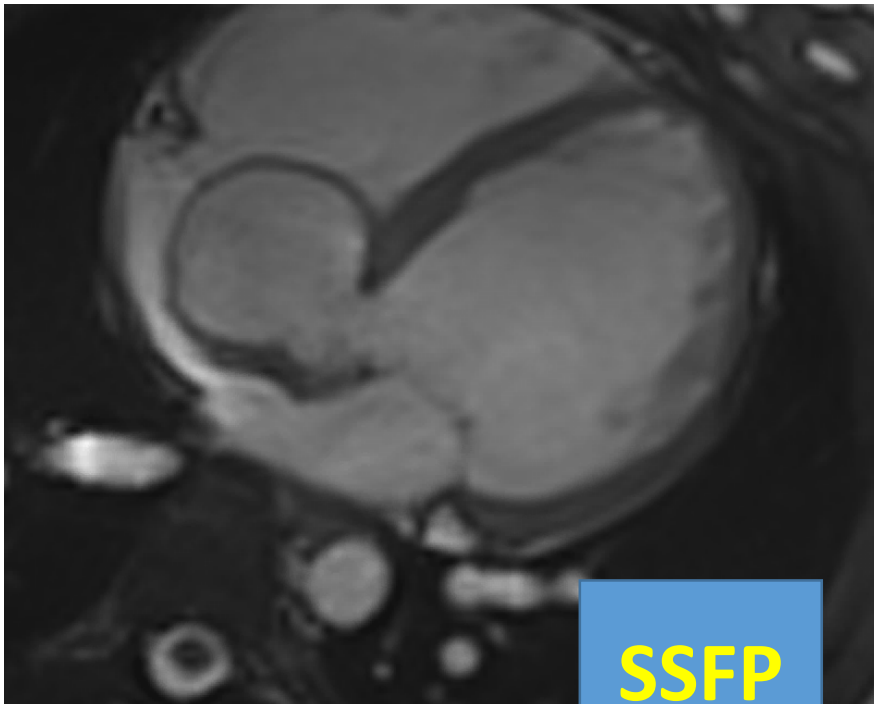
Fabiola B. Sozzi ✉, Nicolas Hugues, Filippo Civaia, Clara Alexandrescu, Laura Iacuzio

EJH CVI 2016



Femme, 28 ans

Asymptomatique
BAV I

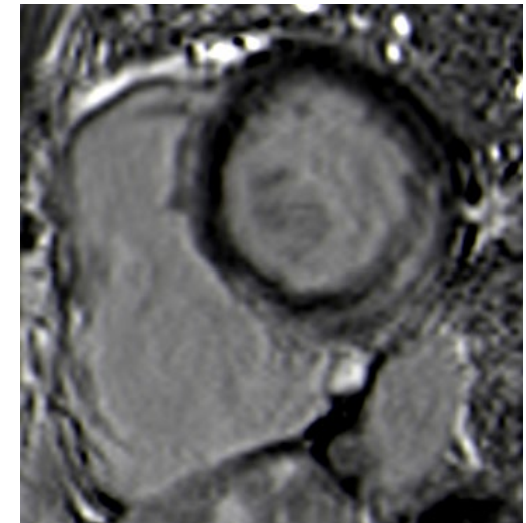
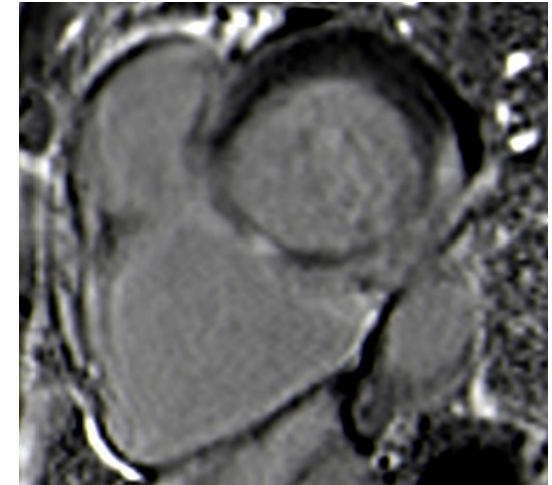
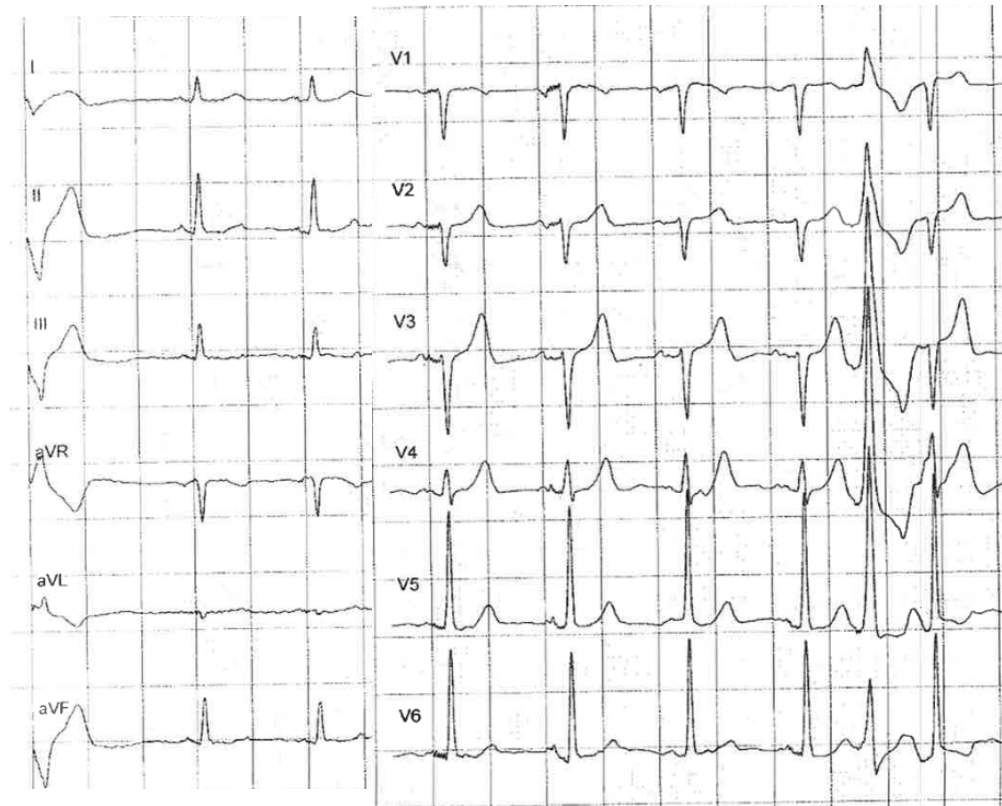


Recherche cardiopathie dans le bilan ESV

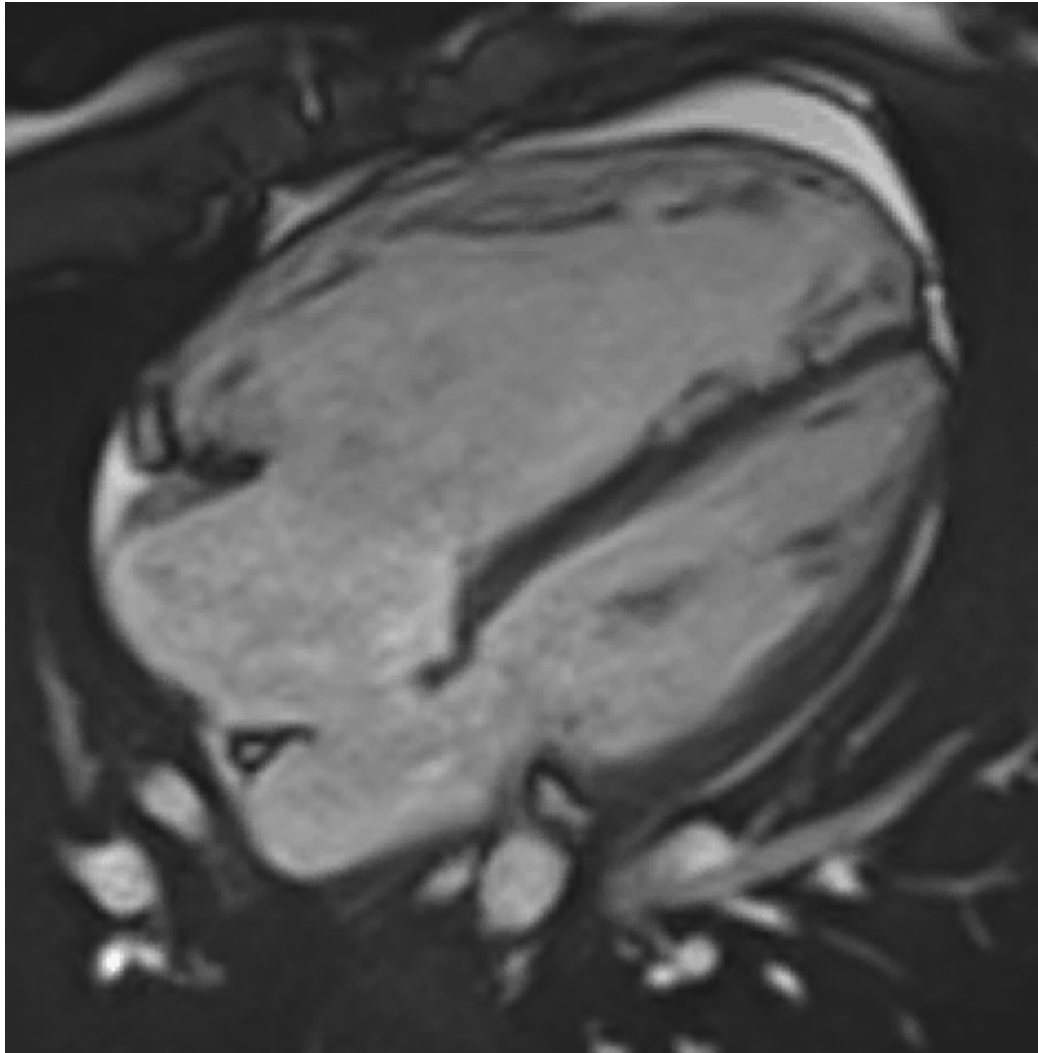
Femme, 68 ans

ESV recherche substrat arytmogène

Myocardite???



- Homme, 20 ans, athlète
- Dilatation VD, ESV



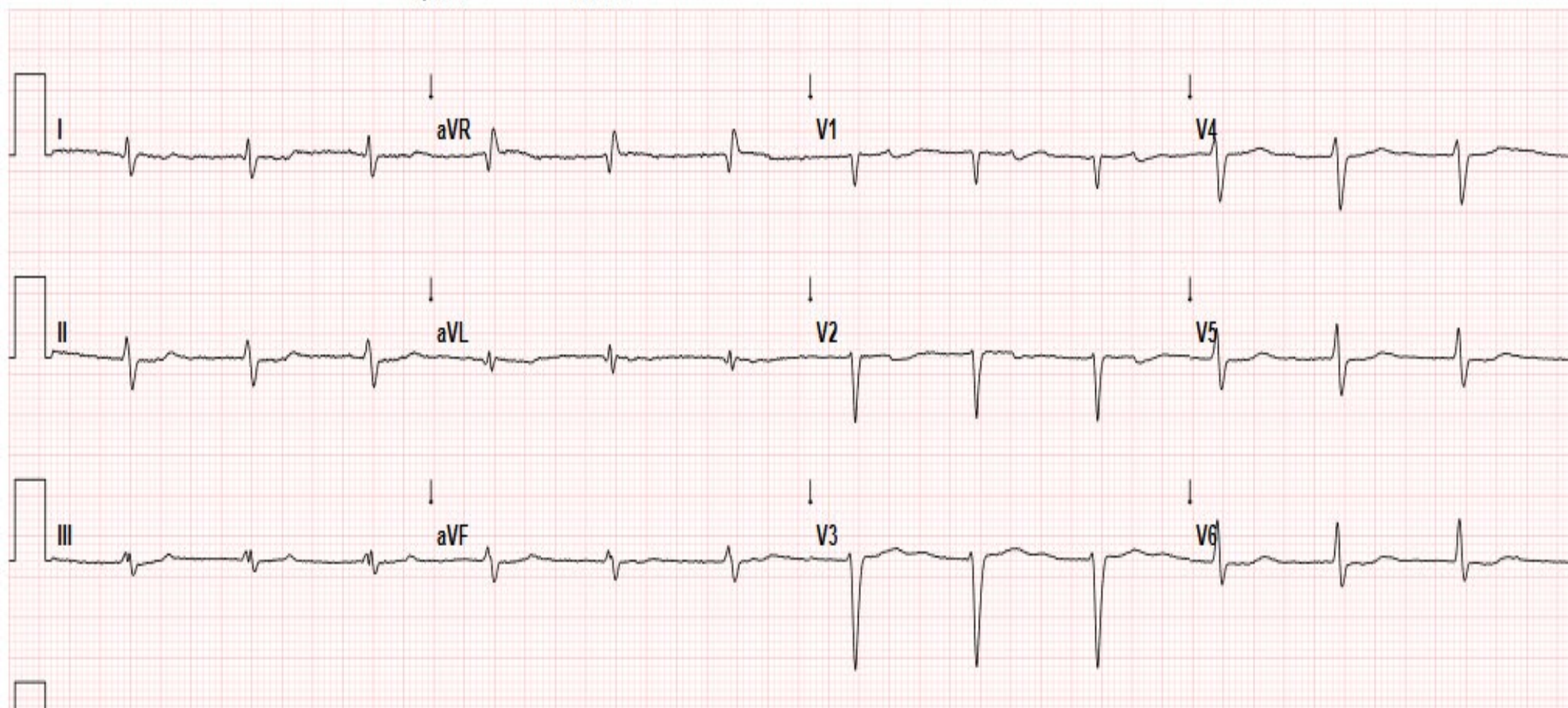
~~DAVD ????~~

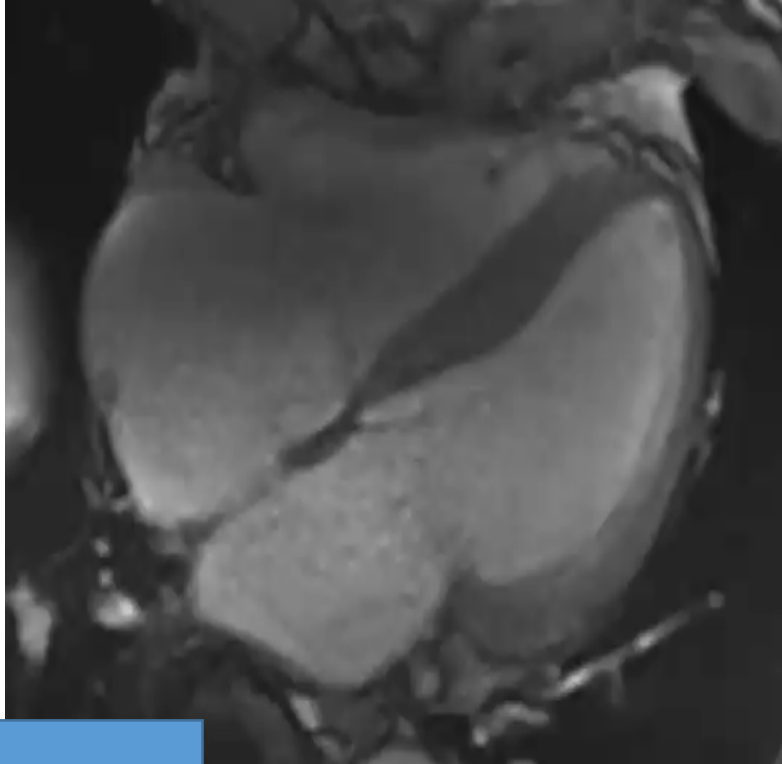
CIA type ostium secundum
Qp/Qs 2,7

Homme, 75 ans

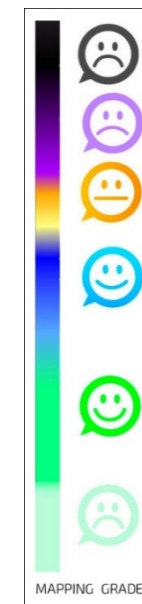
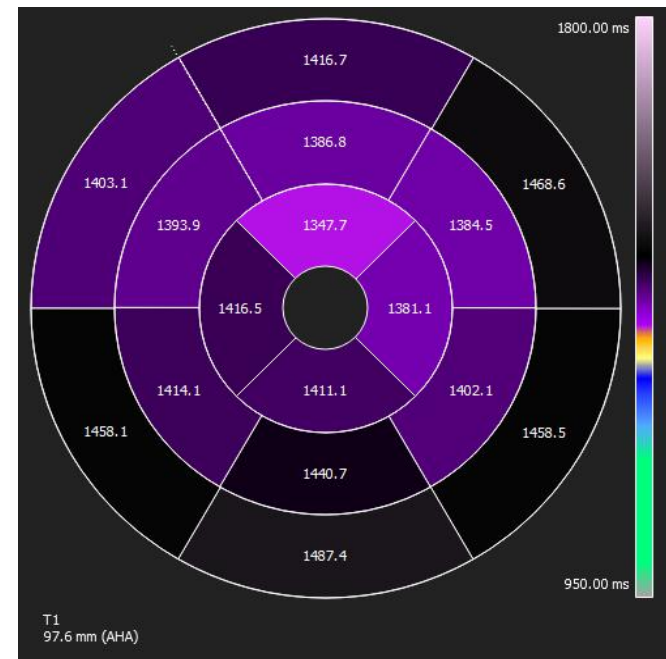
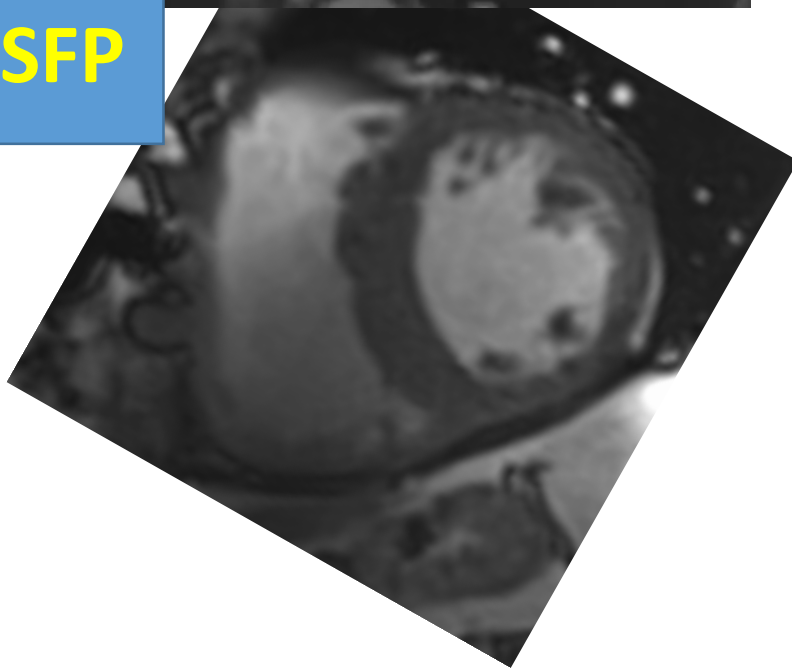
- Hospitalisé pour pec de IM + IT
- Ablation FA en 2015, 2018, 6/2021
- CEE 8/2021
- Ponction ascite 8/2021 (bilan nepro-epatique ras)
- insuffisance cardiaque à prédominance droite, réfractaire avec anasarque

Il présente actuellement une altération de l'état général avec amaigrissement, des signes d'insuffisance cardiaque avec dyspnée malgré la régularité de son rythme cardiaque, ascite (problème hépatique surajouté en cours d'exploration ?), élévation du BNP

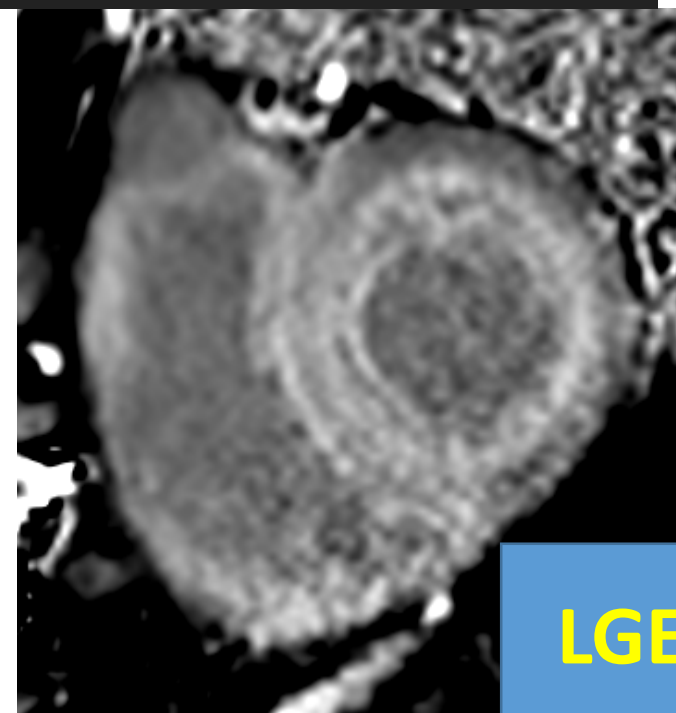




SSFP



AMYLOSE

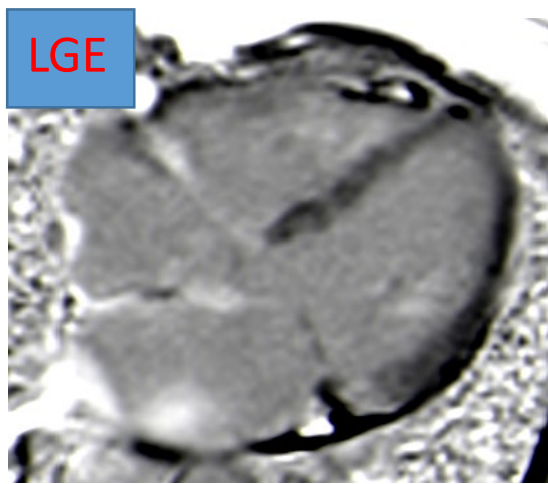
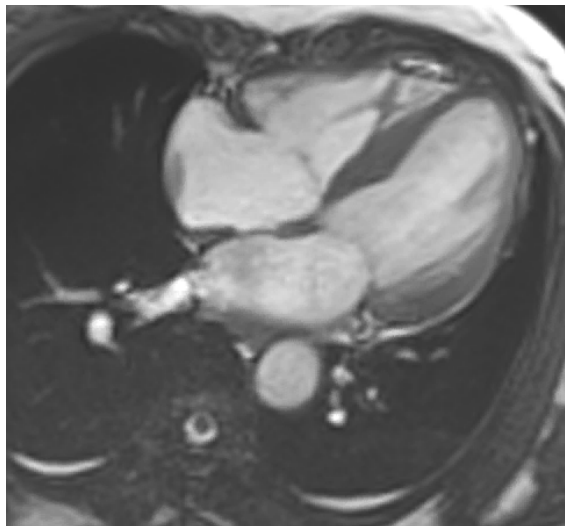


LGE

Femme, 76 ans

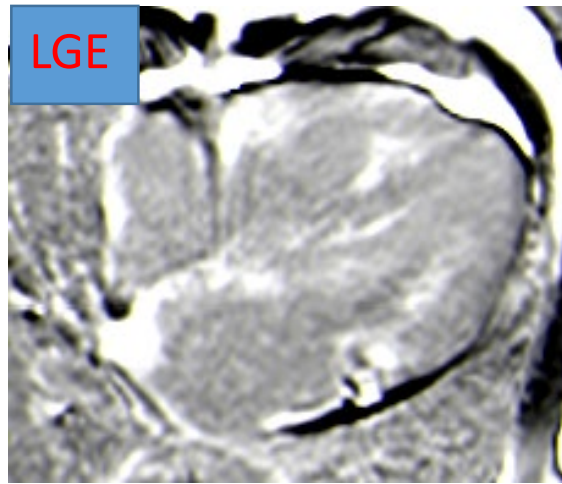
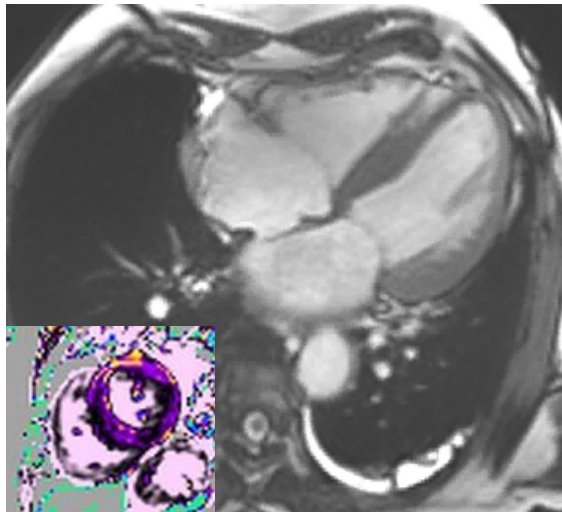
2016

SCA tr +; athérome coronaires



2018

décompensation cardiaque

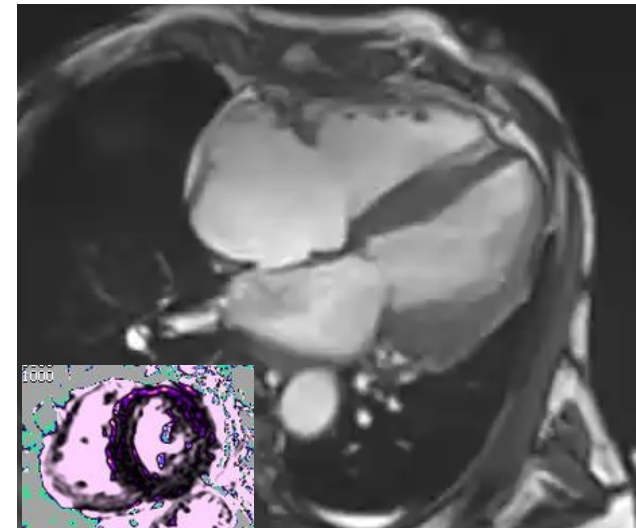


Scinti diphosphate 3+

canal carpien bilatéral opéré!!

2020

sous Tafamidis



FE 68% 60% 53%

SLG -15 -11% -8%

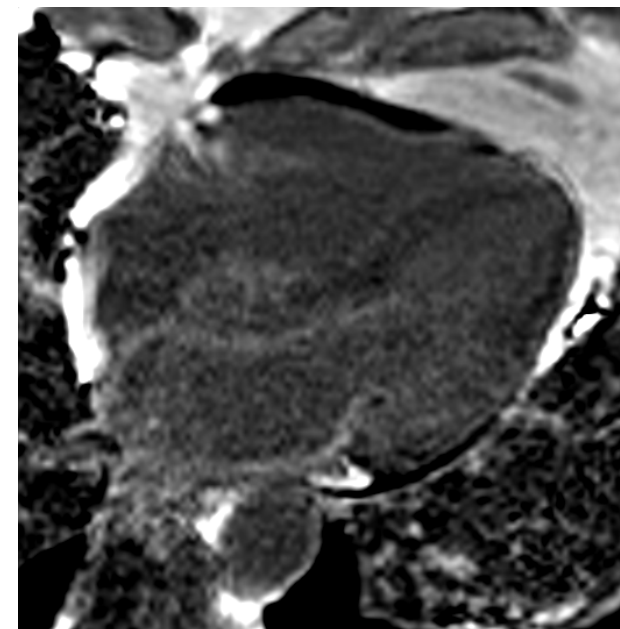
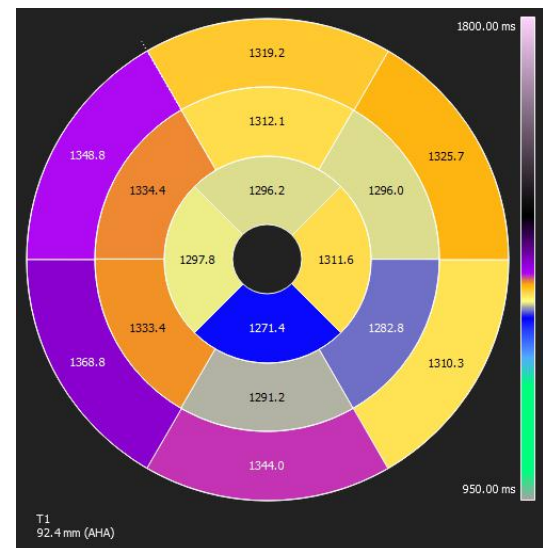
? 1432 ms 1468 ms

Amélioration Clinique !!

Homme, 67 ans Évolution à 1 ans amylose AL rénale + myélome multiple

- augmentation du T1 passant de **1315** à **1393** ms
- apparition d'un rehaussement tardif de gadolinium pathognomonique.

2020

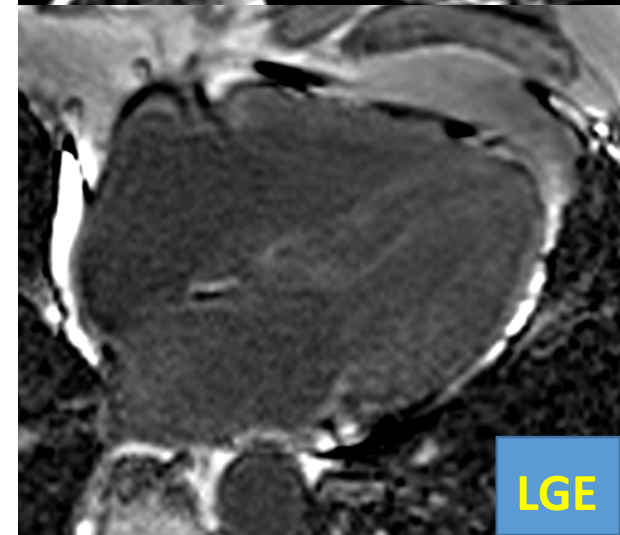
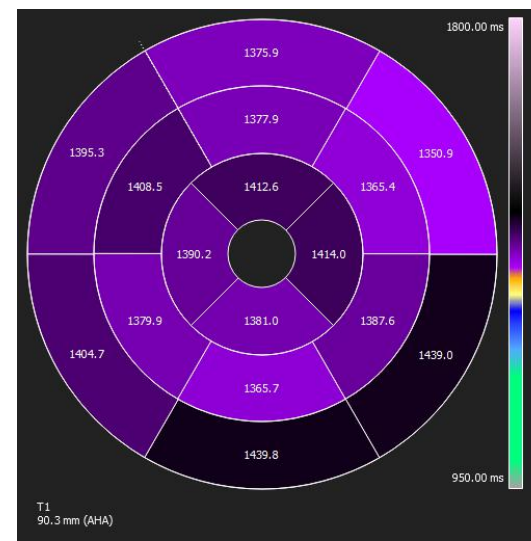


- masse à **67** -> **70** g/m²
- EF à **71** - **70** %,
- SLG - **12**% -14 %,

2021



SSFP



LGE

Eléments diagnostiques amylose



- **Hypertrophie**
 - VG / VD discordance avec bas voltage ECG
 - Hypertrophie des oreillettes et SIA
- Patient **dyspnéique** avec une fonction systolique VG limite inférieure de la norme. **SLG** très diminué !!
- **Epanchement** péricardique et pleural !!!!!
- HVG disproportionnée au Rao (bas débit – bas gradient)
 - 15% amylose pre TAVI

Amylose

- dépôt de fibrilles amyloïdes insolubles dans l'espace extracellulaire
 - AL: chaines légères (MGUS, myélome)
 - ATTR: transtiretine
 - ATTRm: héréditaire (aut.dom)
 - ATTRwt: sauvage – amylose systémique sénile
- Dépôt protéines trigger x myocytolyse et fibrose secondaire (AL!!)

AMYLOSE

	AL	ATTR
Réponse histologique	Effet toxique directe de chaines légères	Réponse hypertrophique des myocytes
HVG	+/-	+++
HVG	Symétrique	Symétrique/asymétrique
T1 native	↑ ↑ ↑	↑ ↑
T2 (œdème)	(prognose) ↑	↔
ECV	↑ ↑	↑ ↑ ↑

APPORT DE L'IRM

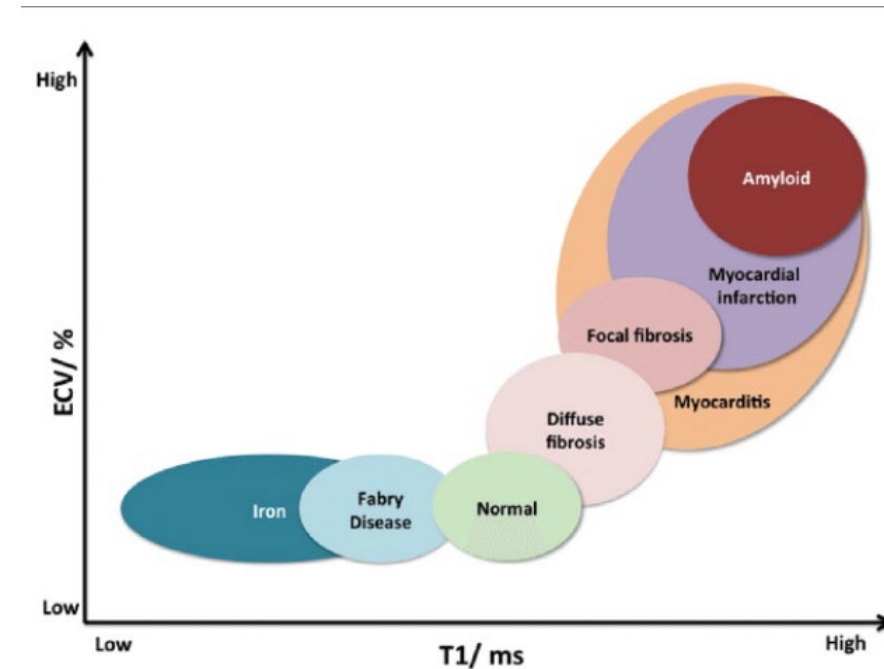
- **MAPPING:**

- T1 augmente en AL et ATTR
- marquer précoce et sensible « spécifique »
- Evaluation quantitative Follow up ☺
- ECV: facteur pronostique

- **LGE:**

- Rehaussement diffus (endocarde -> transmurale)
- Pattern LGE pathognomonique, mais:
 - Evaluation Non quantitative (Suivi....)
 - Attention : Insuffisance rénale

- HVG
- FE SLG
- Epanchement péricardique et pleural



Quand demander une IRM

- dd HVG
- Retentissement cardiaque dans l'amylose avérée

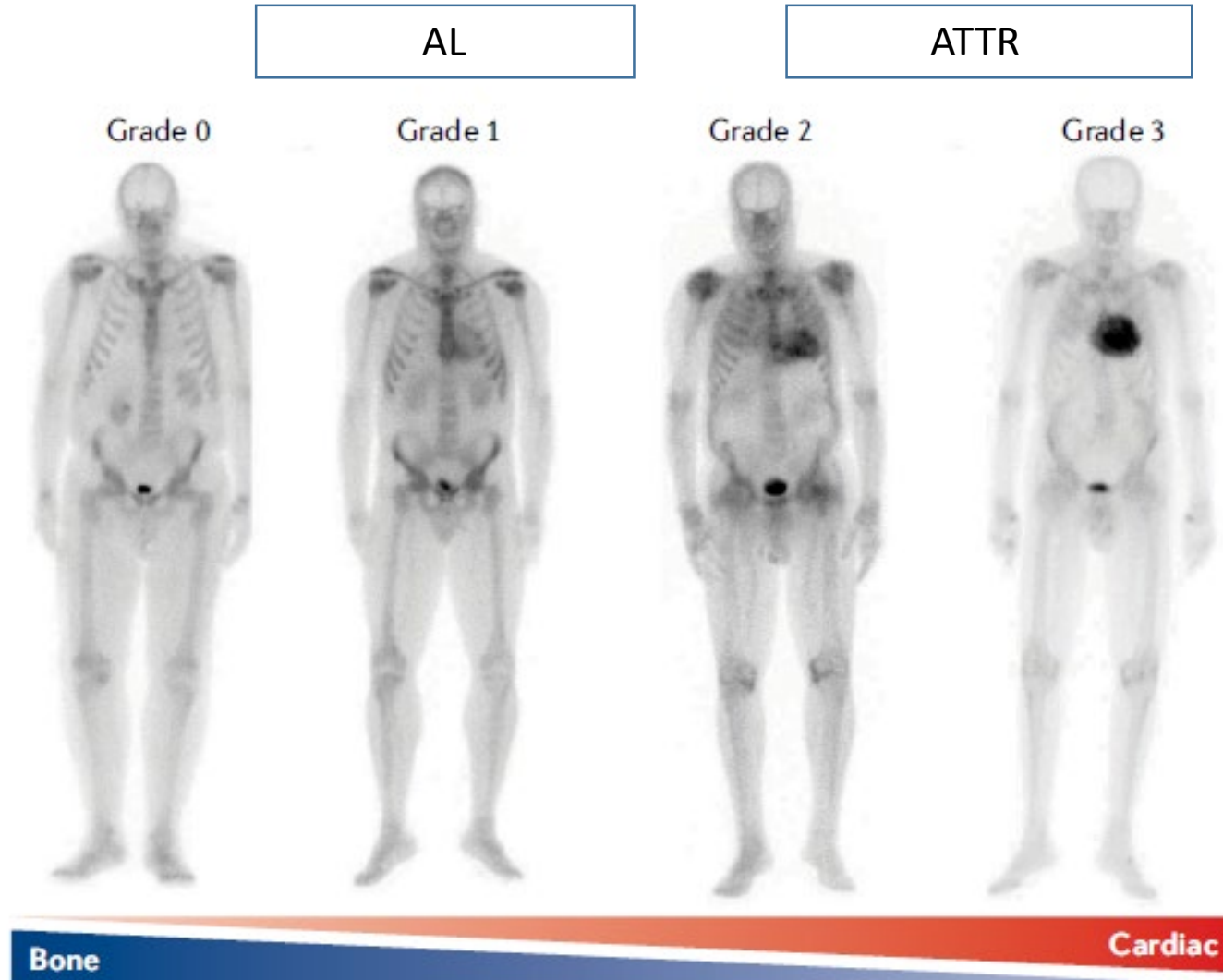


Quel test pour quel scenario ?

- DD **HVG**:
 - **Echo**: suspicion
 - **IRM**
 - dd HVG : CMH, CP hypertensive, AFD
 - Manifestation atypique : CMP dilatée
 - Dosage **chaines légères** + **scintigraphie** au **difosphonates**: dd AL vs ATTR
- NB: scinti grade2/3 + absence de chaines légères: ATTR!!!

Scintigraphie osseuse bisphosphonates

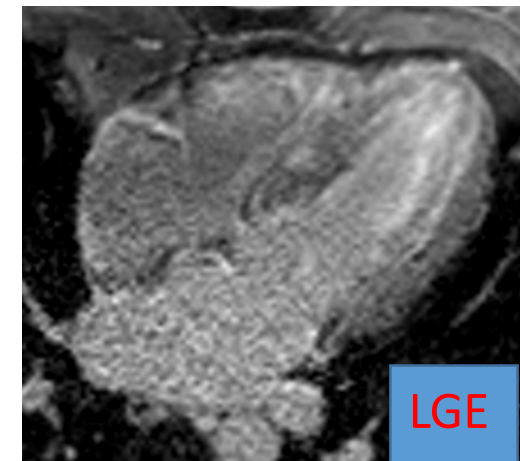
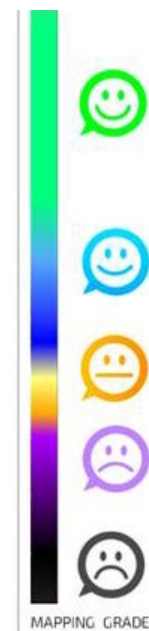
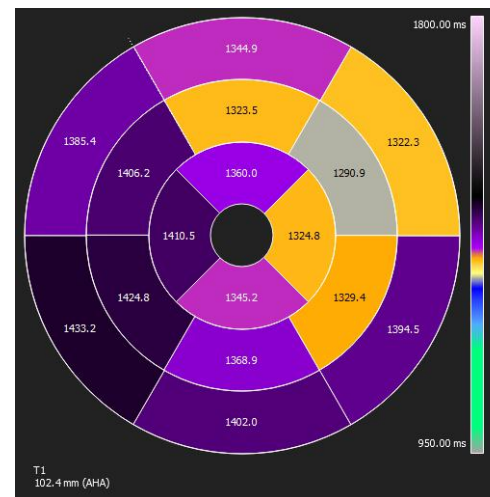
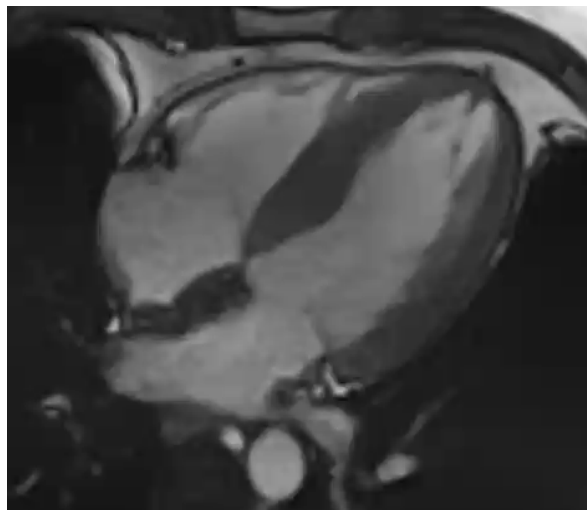
Classification Perugini de la fixation myocardique



- ATTRm (glu 89 GLN),
- avec mère décédée à 56 ans pour IC

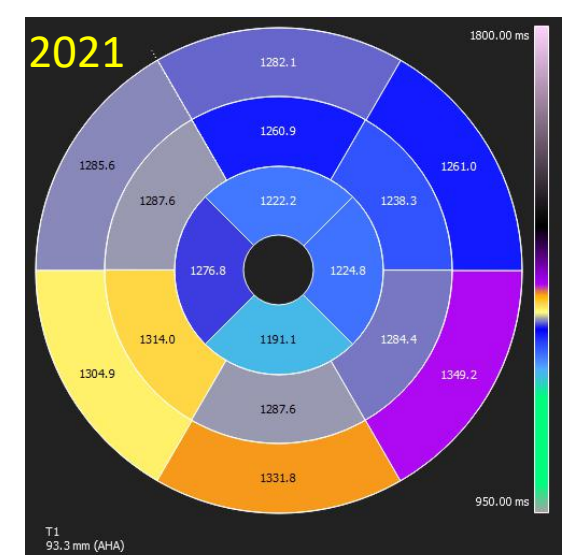
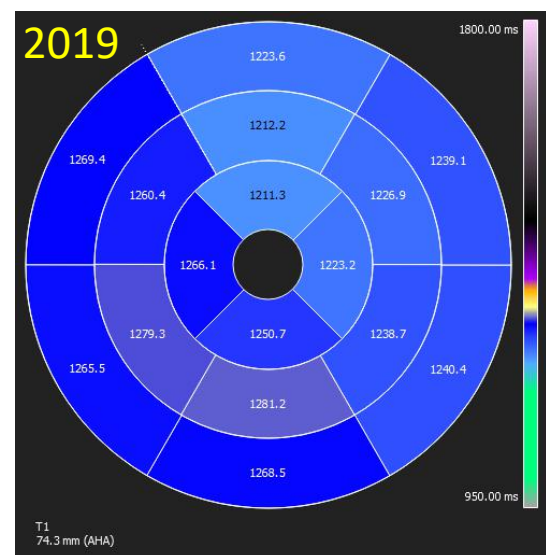
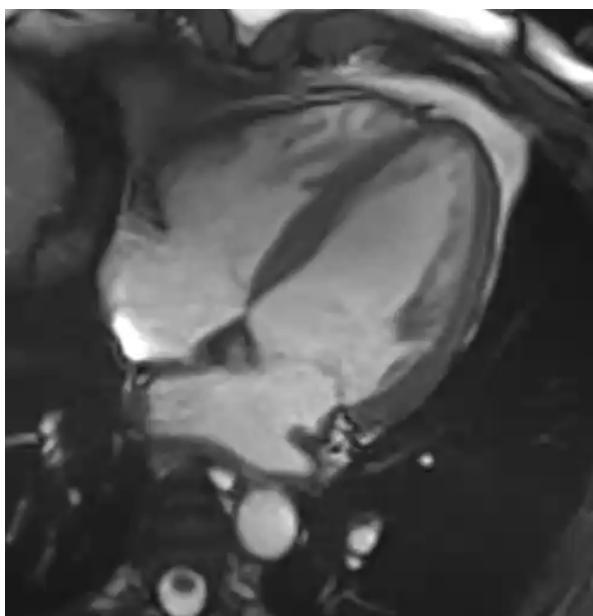
Frère S, 48 ans

- neuropathie



Frère C, 48 ans

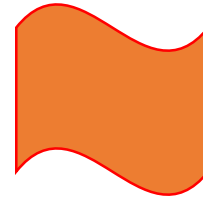
- asymptomatique



Conclusion: amylose

Think about !!!

- HFpEF (résistant au tx traditionnel)
- Tunnel carpal bilatéral (5-10 ans avant)
- Neuropathie, dysautonomie
- Troponine  sur plusieurs prélèvements; NT pro BNP
- HVG + bas voltages
- HVG + sténose valvulaire aortique (15% ATTR pre TAVI)
- Apical sparing (SLG)



Diagnostic précoce pour -> traitement



Review > JACC Cardiovasc Imaging. 2019 Nov;12(11 Pt 2):2345-2356.

doi: 10.1016/j.jcmg.2019.06.023. Epub 2019 Aug 14.

Myocardial Amyloidosis: The Exemplar Interstitial Disease

Marianna Fontana¹, Andrej Ćorović², Paul Scully³, James C Moon³

Review > Nat Rev Cardiol. 2020 Jul;17(7):413-426. doi: 10.1038/s41569-020-0334-7.

Epub 2020 Feb 10.

Diagnostic imaging of cardiac amyloidosis

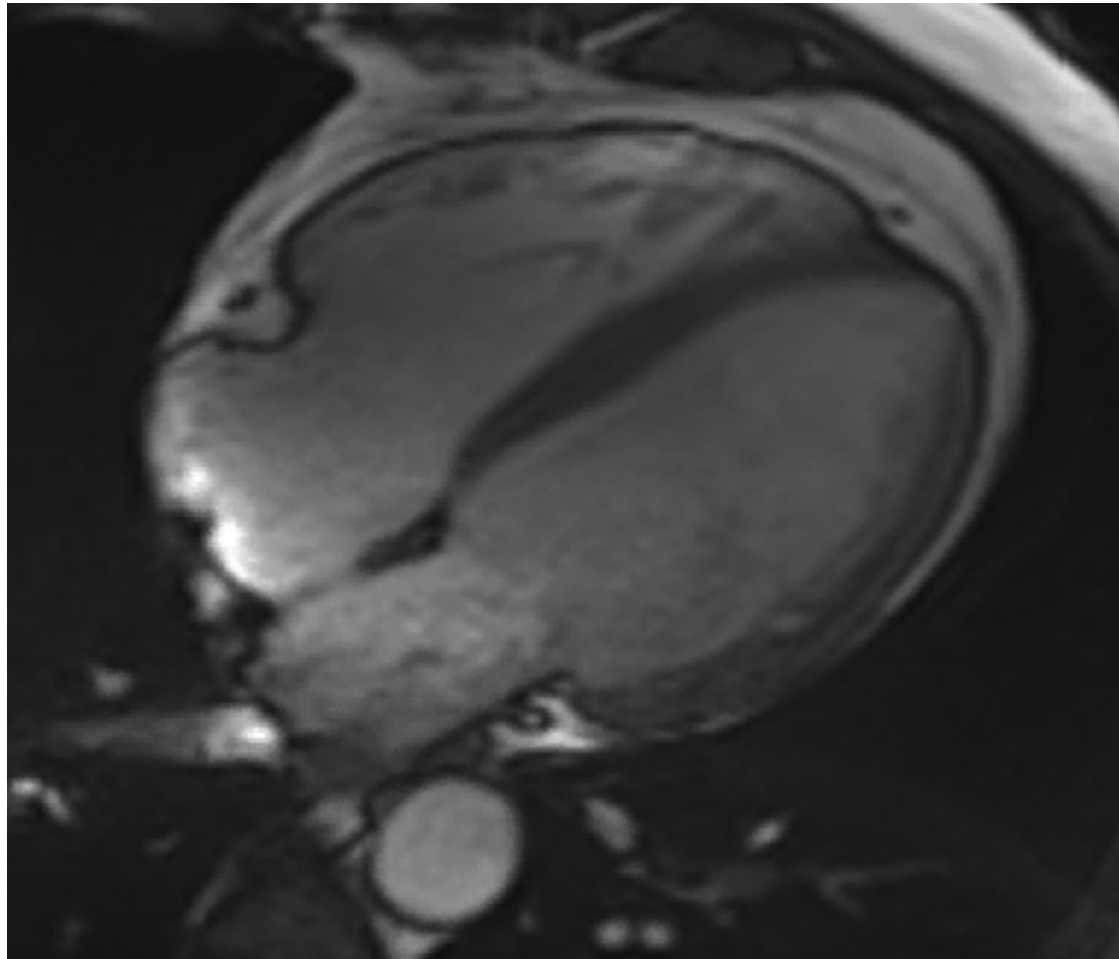
Ana Martinez-Naharro¹, A John Baks^{2 3}, Philip N Hawkins¹, Marianna Fontana⁴

Dépistage familial maladie héréditaire atteinte cardiaque dans maladie systémique

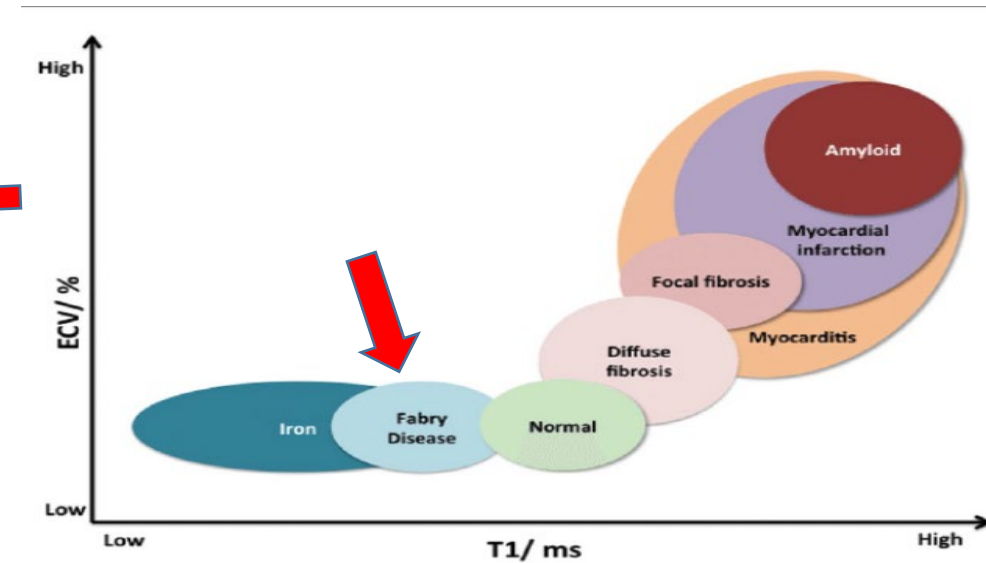
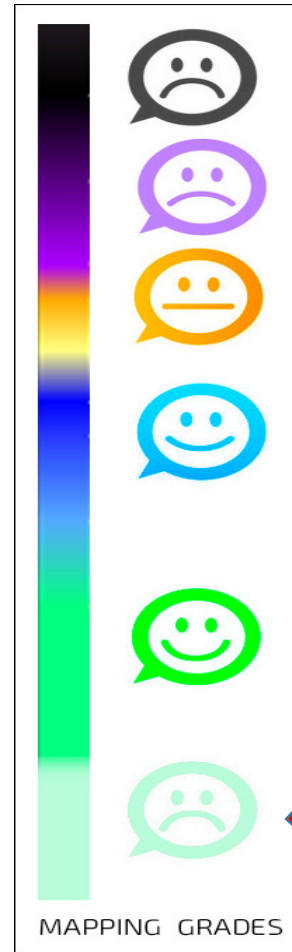
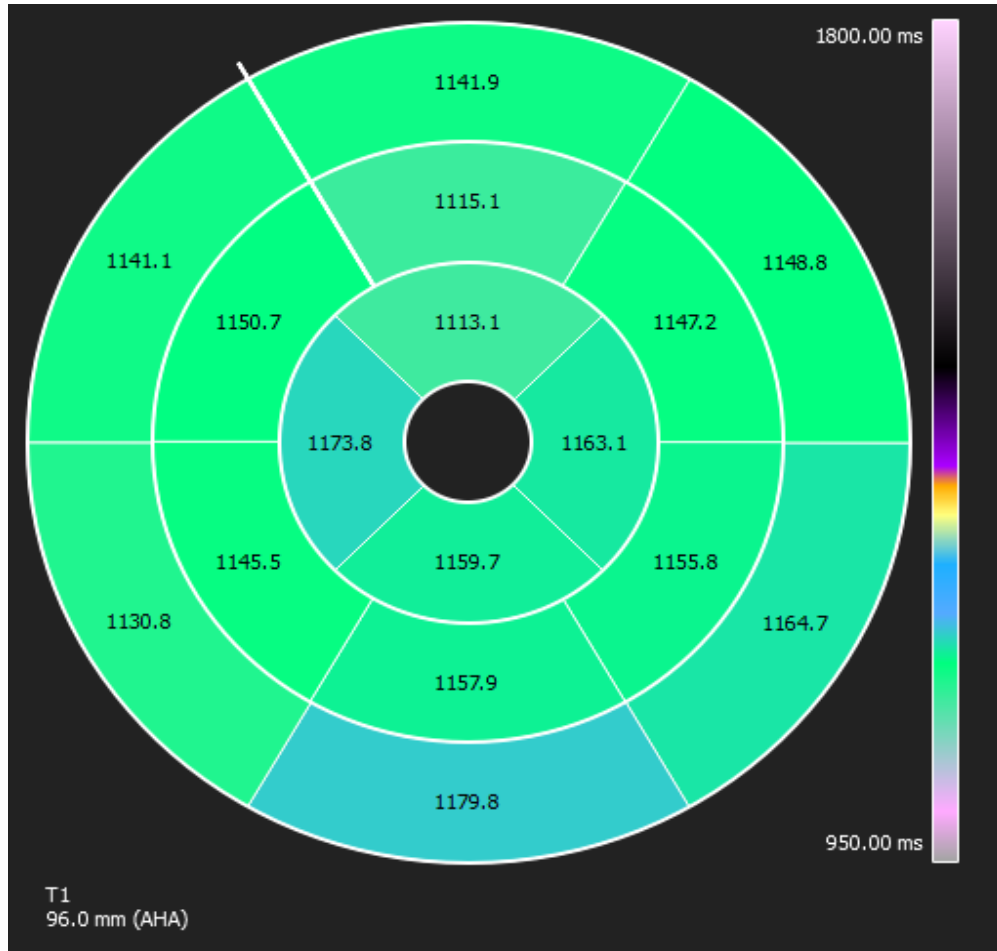
- CMH
- ATTRh
- Maladie de Fabry
- ACM (DAVD)
- Bicuspidie
- Marfan
- LES (cardiopathie ischémique, valvulopathie)

Homme, 53 ans

- porteur d'une **maladie de Fabry**.
- ETT, Holter ECG, ergométrie RAS



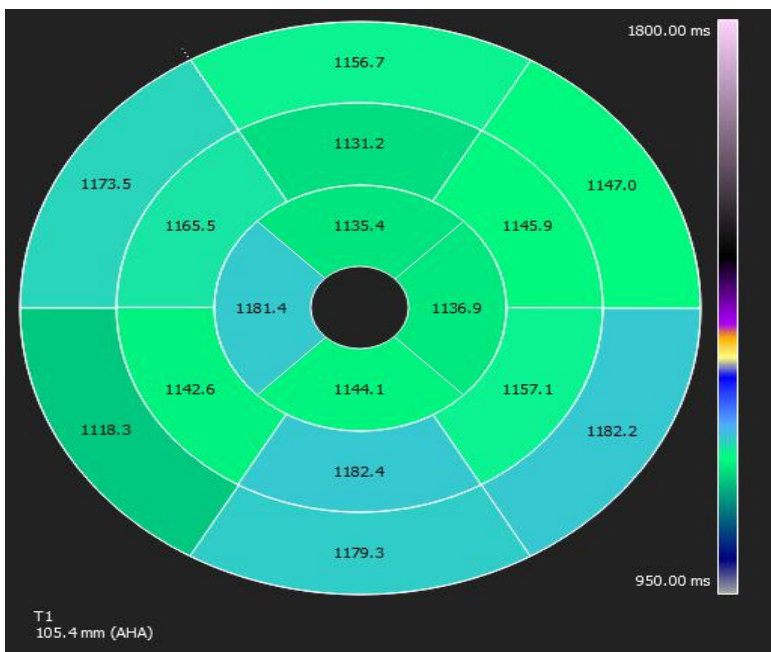
T1 diminué



Pseudo-normalisation du T1 dans le segment inféro-latérale à 1178 ms.

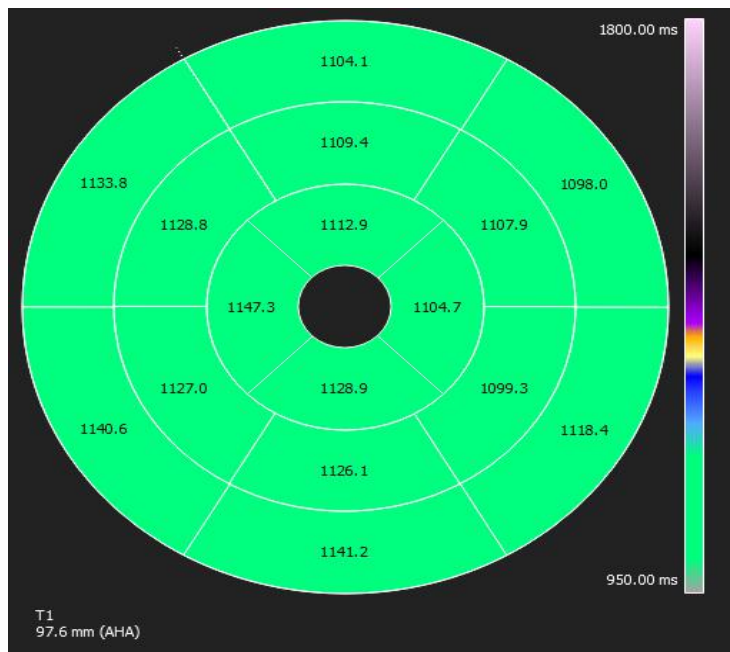
2019

masse à 110 g/m²,
SLG à -17%,
T1 à 1155 ms



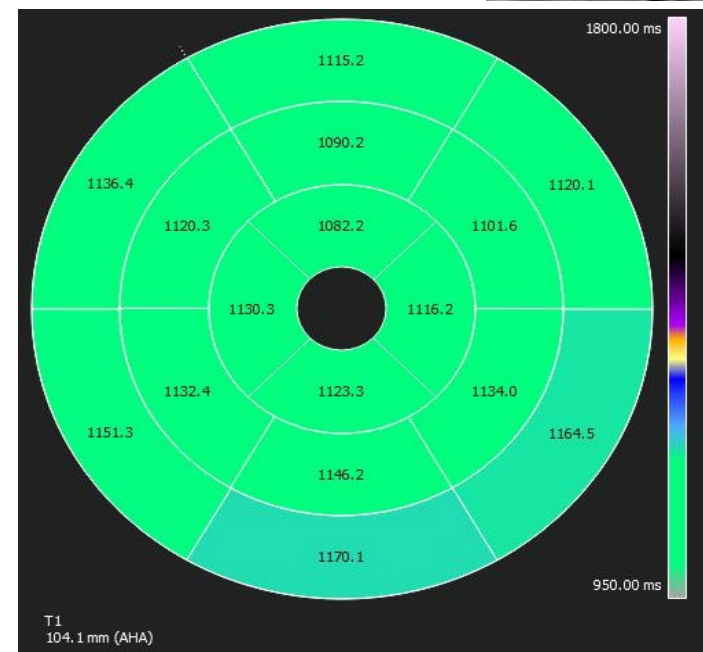
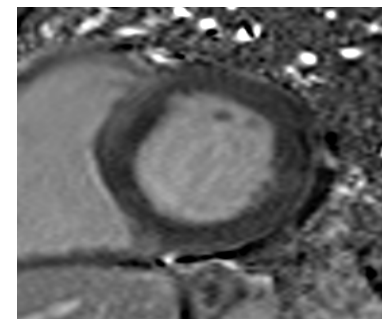
2020

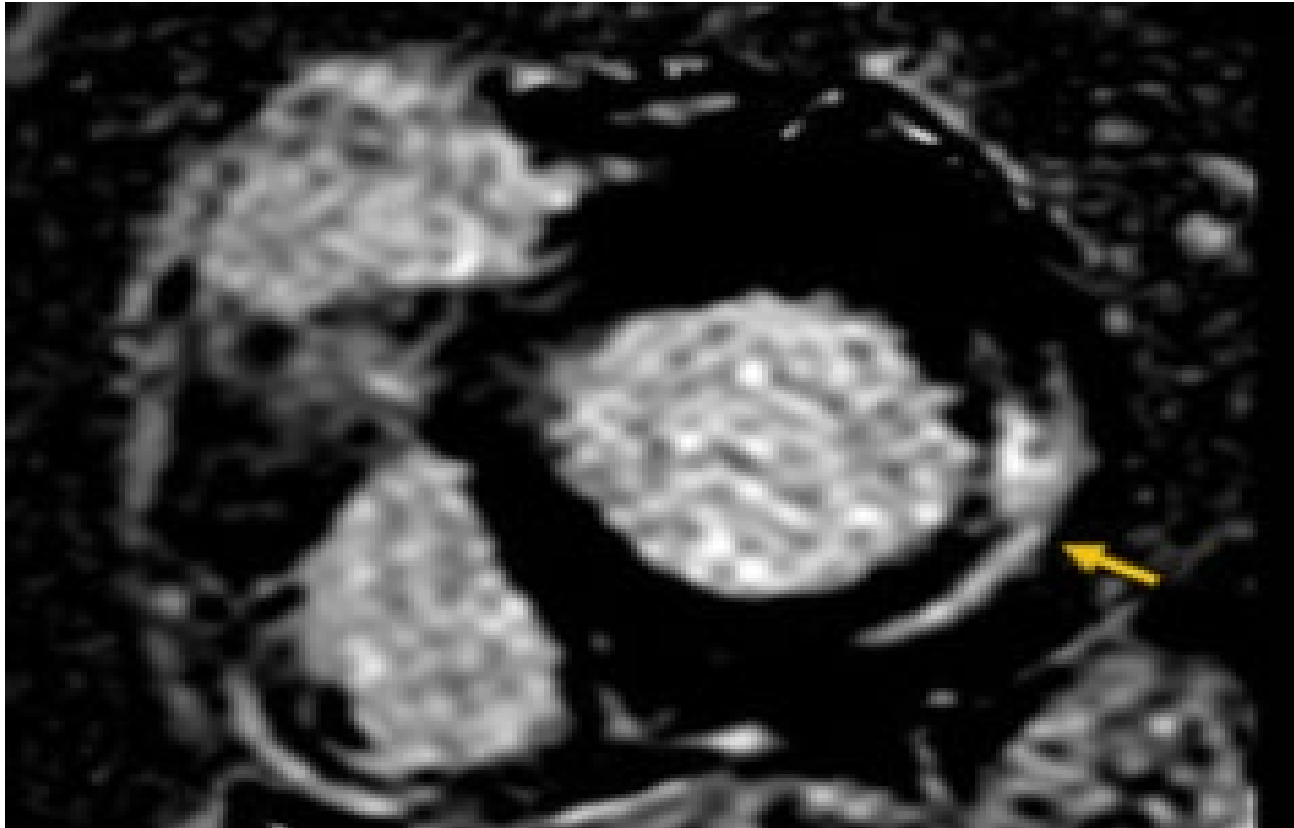
masse à 108 g/m²
SLG -16%,
↓ T1 à 1120 ms



2021

masse à 107 g/m²
SLG -16%,
T1 à 1126 ms





- <https://doi.org/10.1161/CIRCIMAGING.119.009013>

Maladie de Fabry:

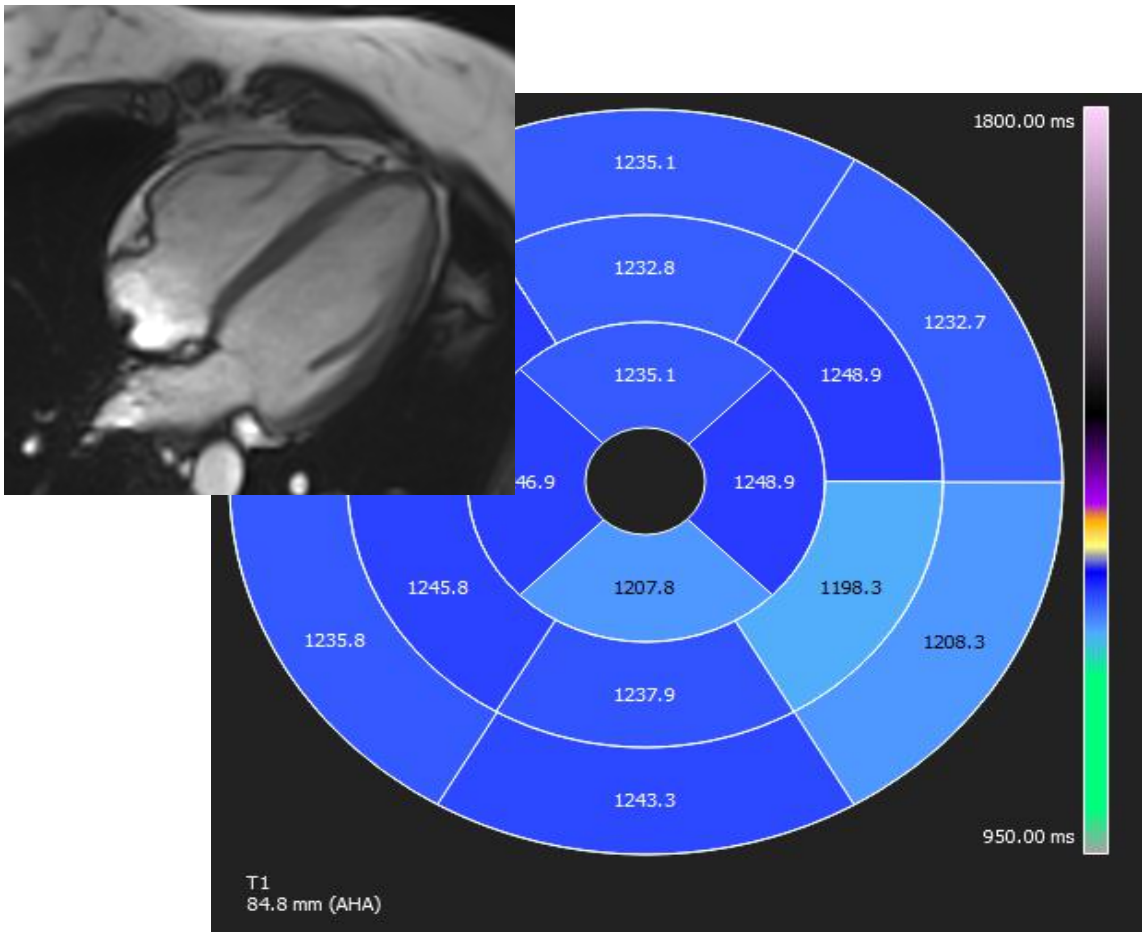
accumulation intra lysosomal glycosphingolipides pour déficit α -galattosidase

- HVG concentrique (plus rarement apicale ou septale asymétrique)
 - HVG homme >> femme
- T1 mapping diminué
 - Homme T1 < femme
 - Pseudo-normalization ou augmenté dans la paroi infero-lateral (LGE+)
- LGE intra myocardique infero-lateral
 - Femme : LGE en l'absence de HVG

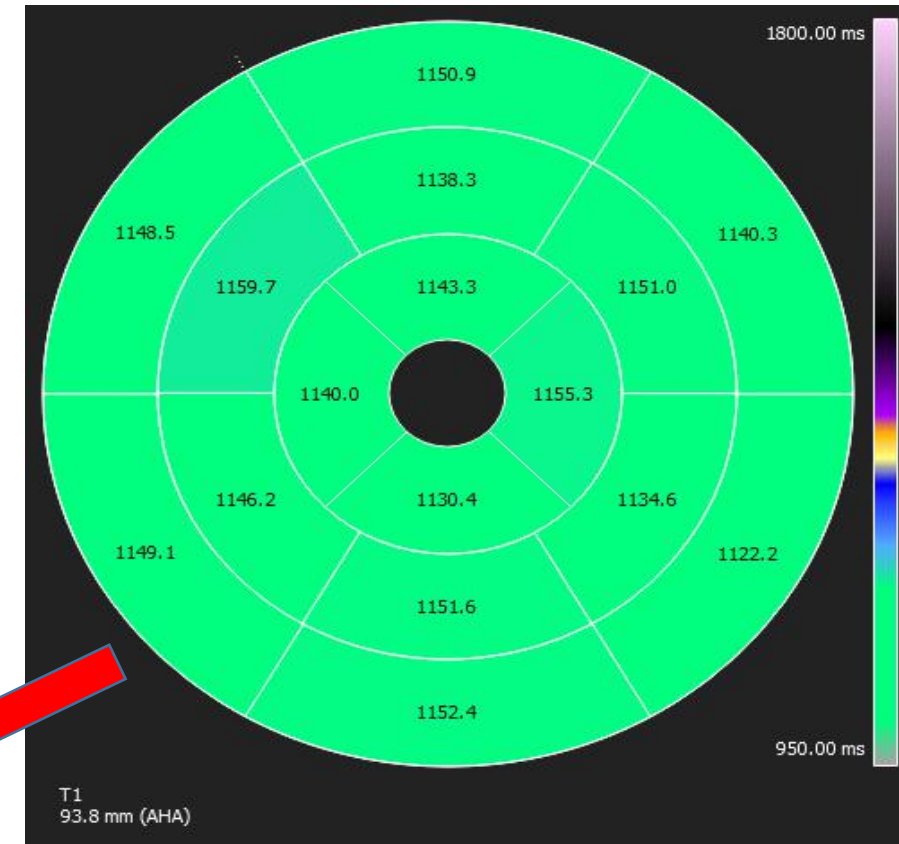
Femme 48 ans

Recherche d'attente cardiaque chez une patiente porteuse d'une mutation de l'alpha-galactosidase (frère atteint de Maladie de Fabry).

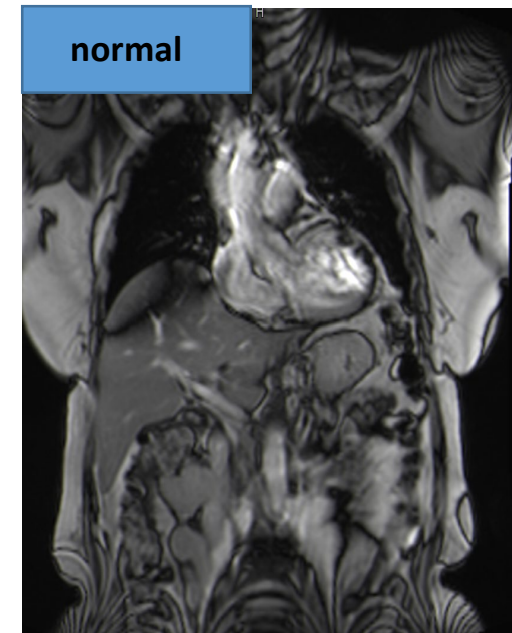
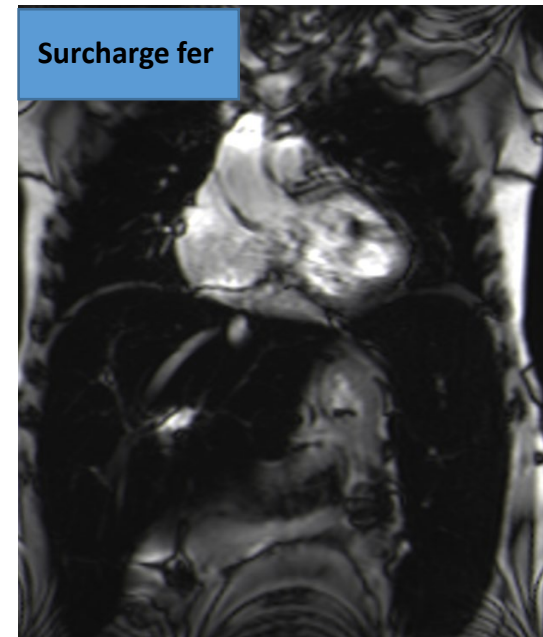
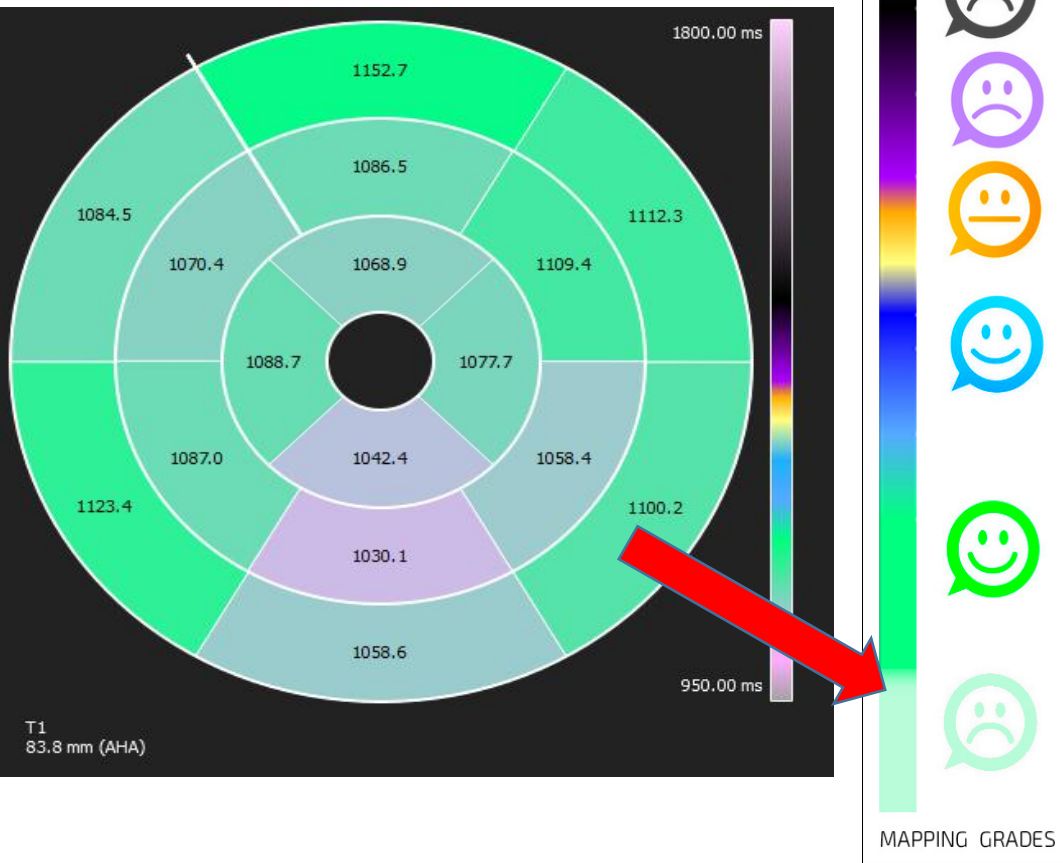
T1 1228 ms



Frère , 51 ans
T1 1145 ms



Femme, 30 ans thalassémie



Congenital heart disease

2020 ESC Guidelines for the management of adult congenital heart disease

The Task Force for the management of adult congenital heart disease of the European Society of Cardiology (ESC)

small, highly mobile structures such as vegetations. CMR is ideal for accurate quantification of ventricular volumes, ejection fraction (EF), valvular regurgitation,¹⁵ calculation of pulmonary and systemic blood flow, and myocardial fibrosis assessment. Cardiovascular computed

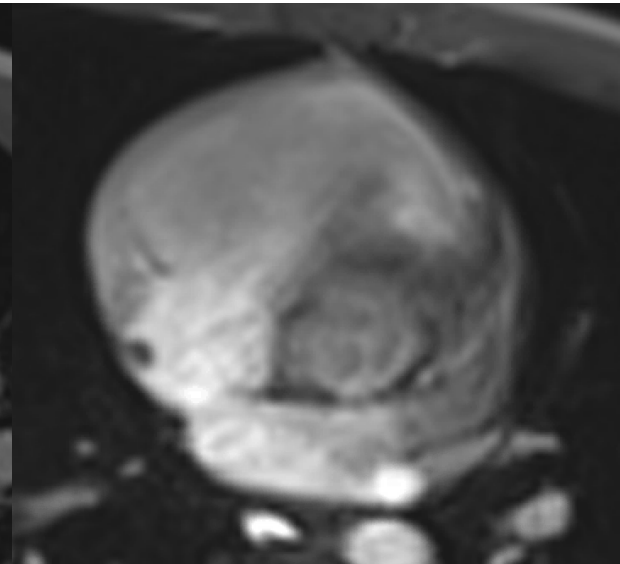
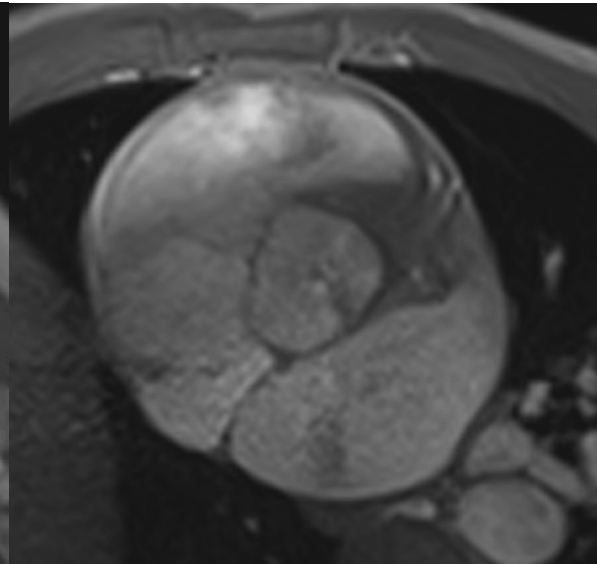
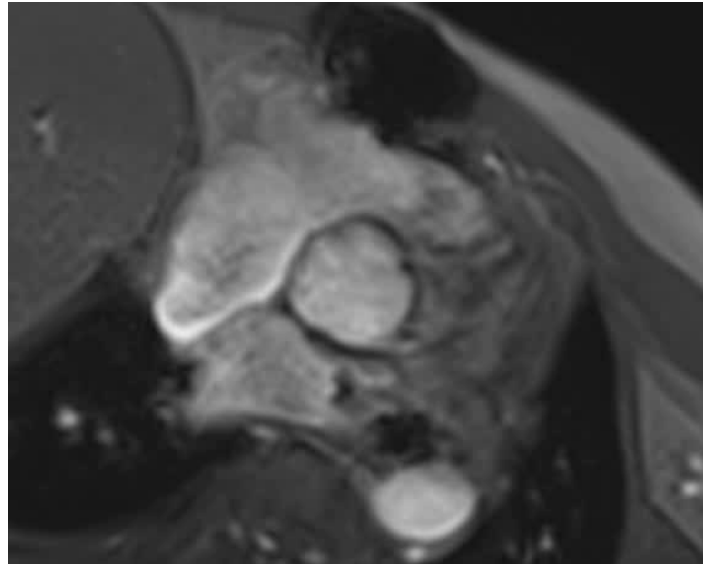
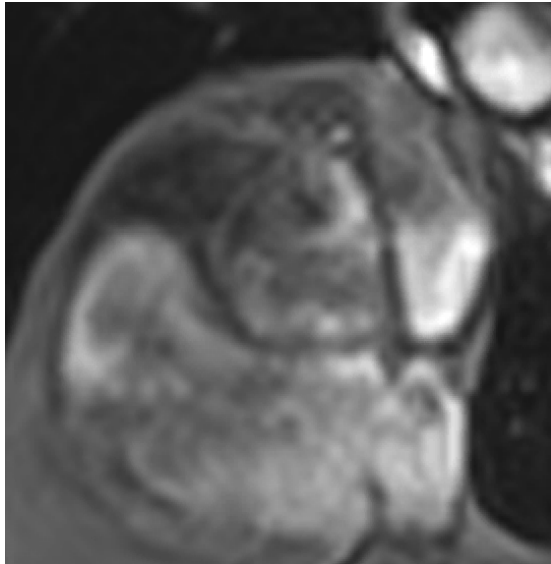
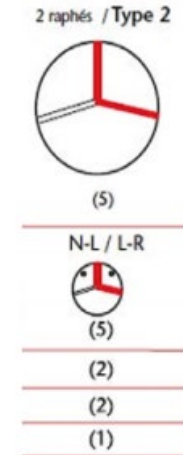
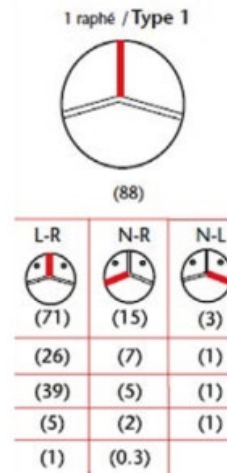
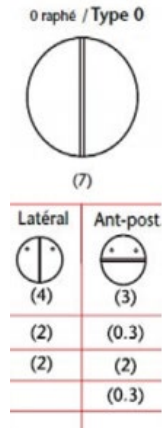
Diagnosis – follow up

Indications for CMR in ACHD patients

- Quantification of RV volumes, EF (including subpulmonary RV, systemic RV, and single ventricle)
- Evaluation of RVOTO and RV–PA conduits
- Quantification of PR
- Evaluation of PAs (stenoses, aneurysms) and the aorta [aneurysm, dissection, coarctation (CCT may be superior)]
- Evaluation of systemic and pulmonary veins (anomalous connection, obstruction, coronary venous anatomy pre-procedure, etc.)
- Collaterals and arteriovenous malformations (CCT may be superior)
- Coronary anomalies and CAD (CCT is superior for intramural course, slit-like course, acute angle take-off, myocardial bridging, and plaque assessment)
- Detection and quantification of myocardial ischaemia by CMR stress perfusion
- Evaluation of intra- and extracardiac masses
- Quantification of myocardial mass (LV and RV)
- Detection and quantification of myocardial fibrosis/scar (late gadolinium enhancement, T1 mapping) tissue characterization (fibrosis, fat, iron, etc.)
- Quantification of systemic and pulmonary blood flow to calculate Qp:Qs
- Quantification of perfusion distribution to the right/left lung
- Measurement of pulmonary blood flow in patients with multiple sources of blood supply (i.e. with major aorto-pulmonary collateral arteries)

Bicuspidie (type 0, 1, 2)

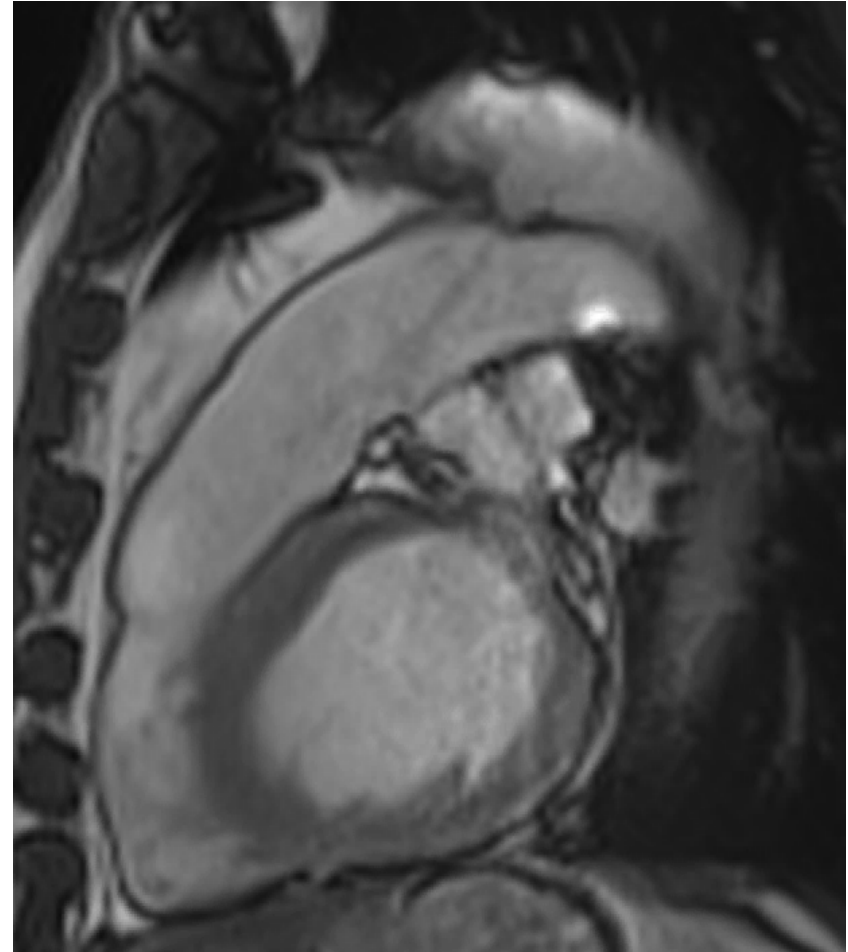
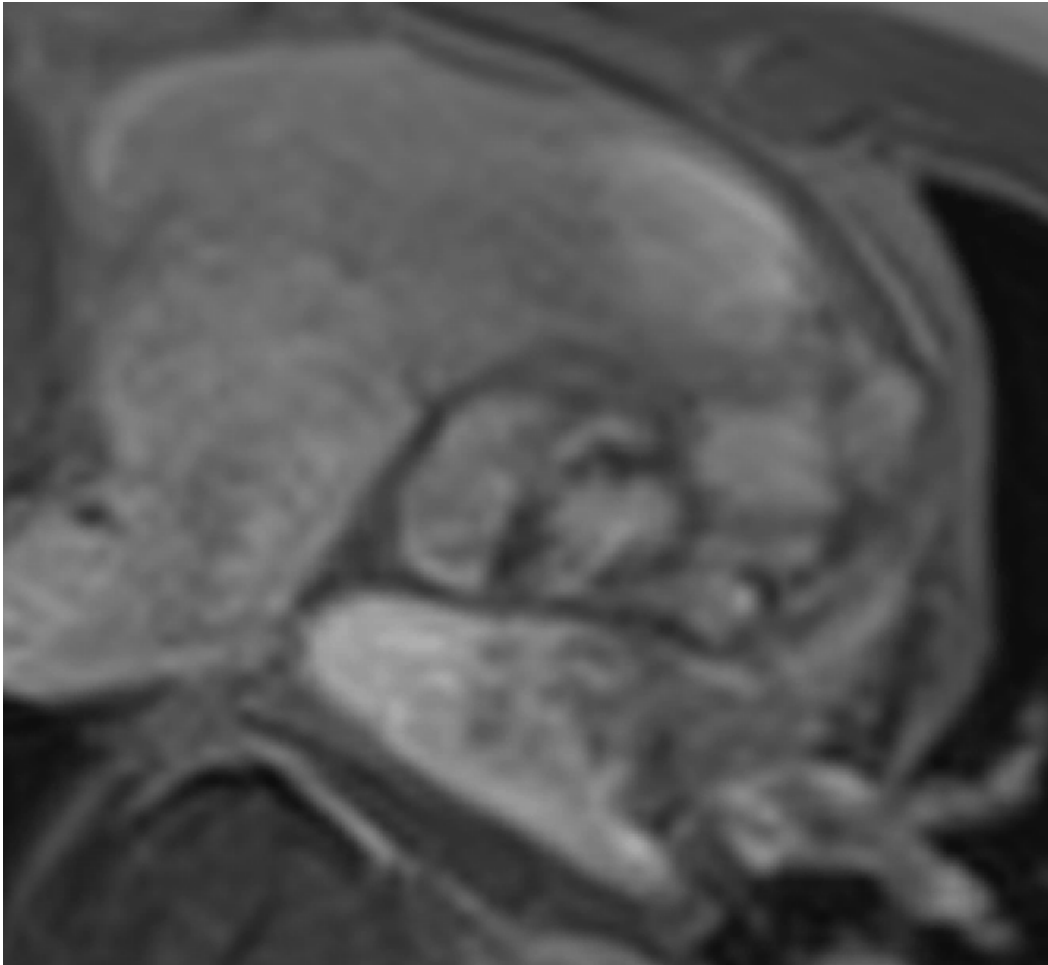
1-2% population générale



Quadricuspide



Homme, 65 ans – stenose aortique



Coarctation aortique

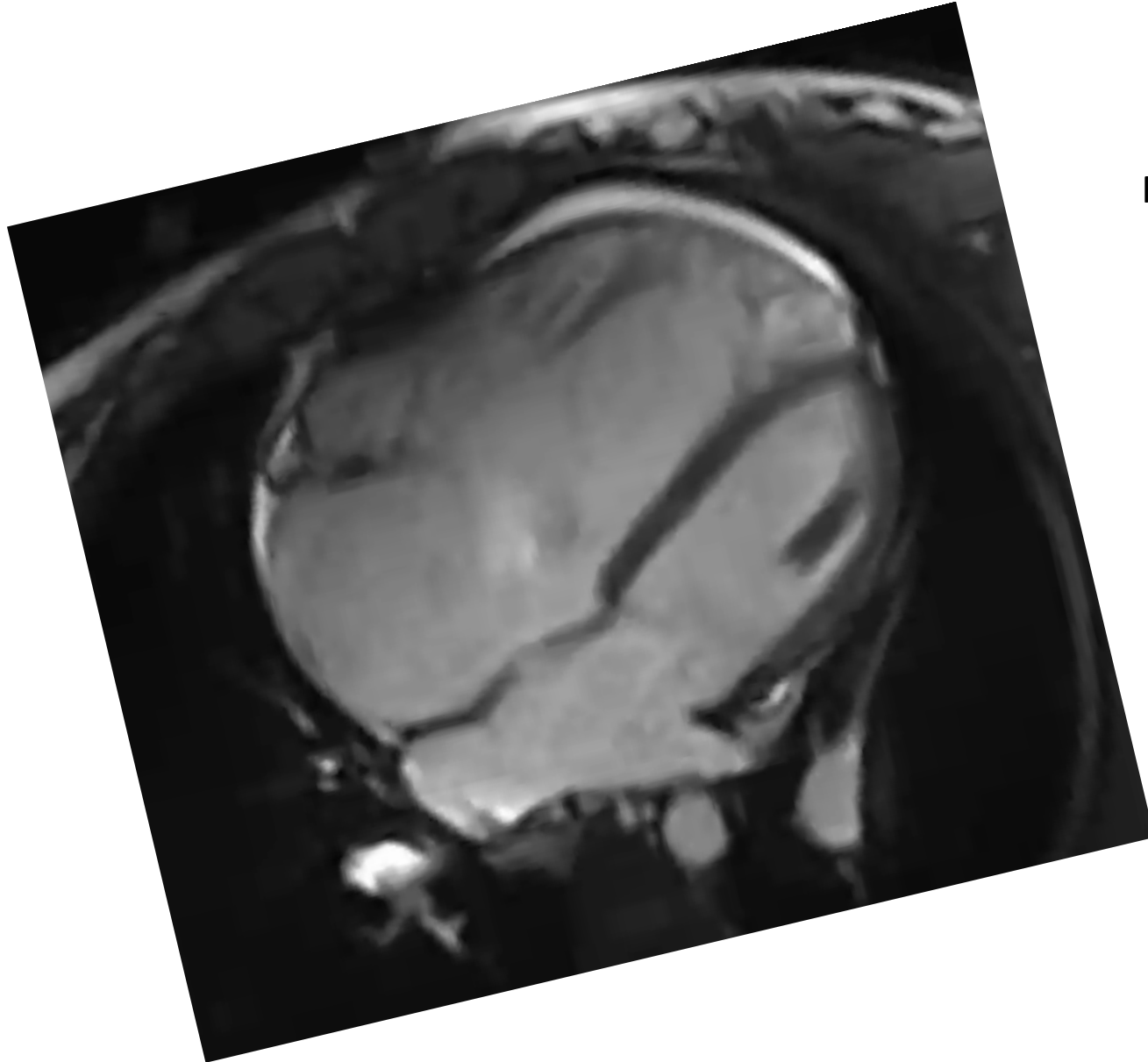


Pre



Post

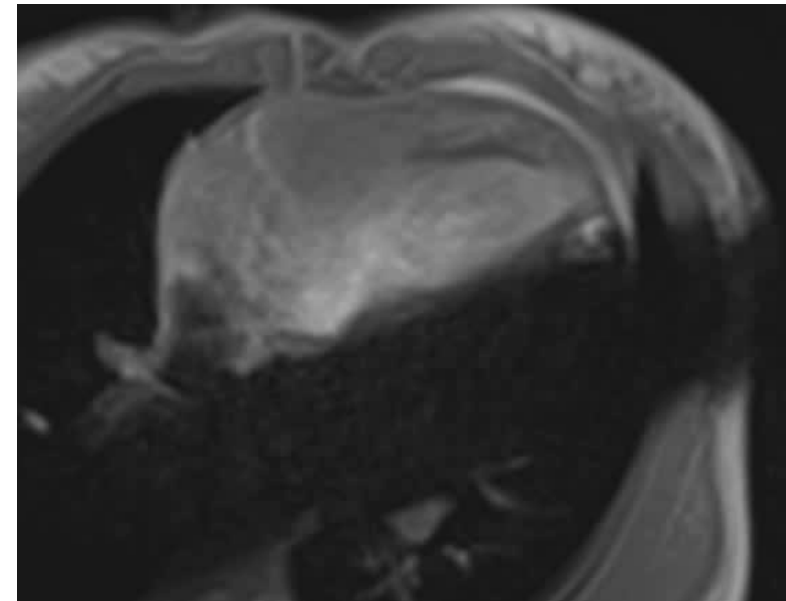
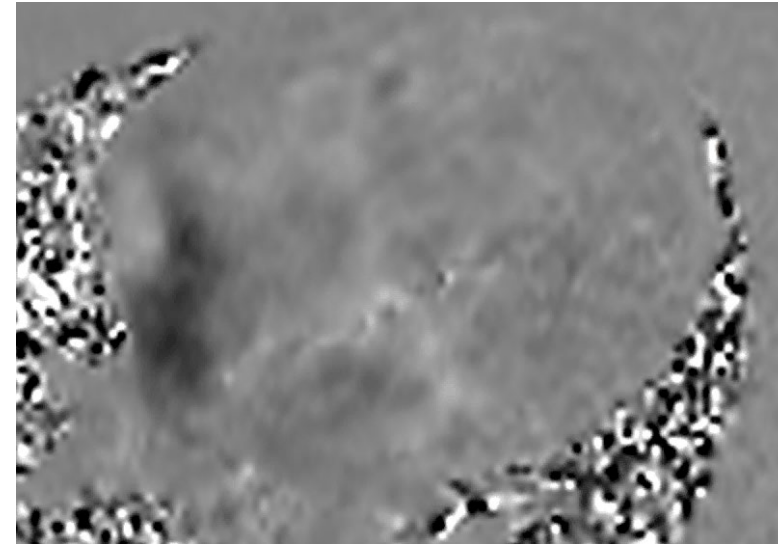
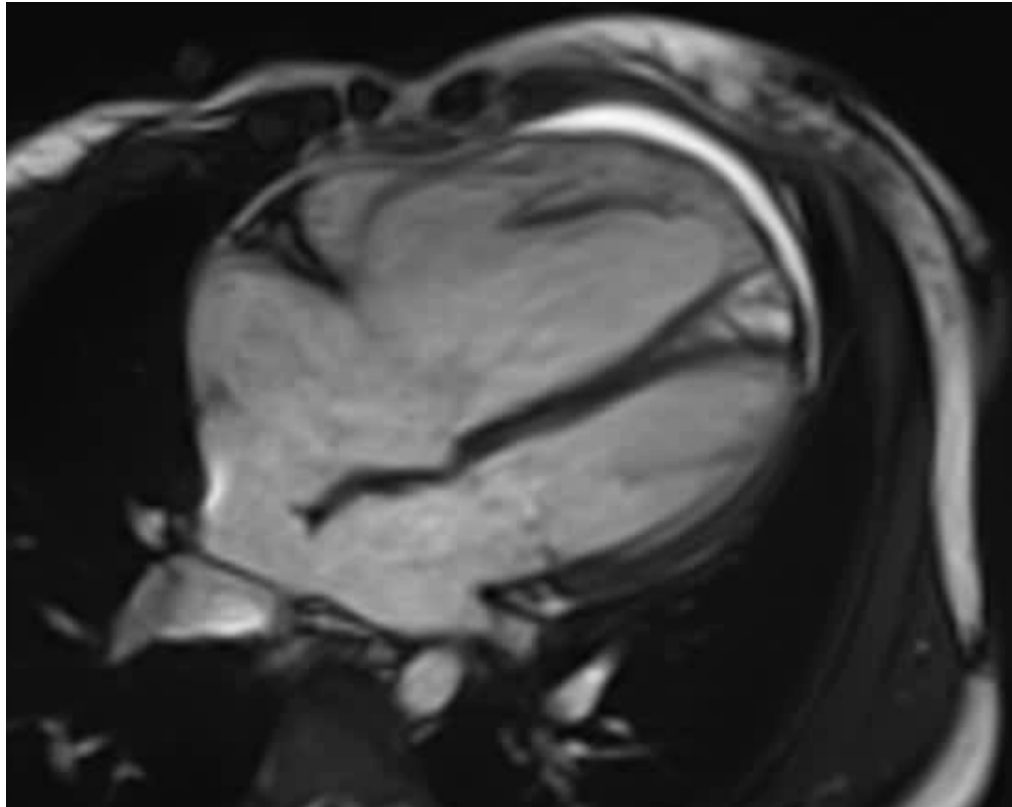
Femme, 35 ans ; dyspnée



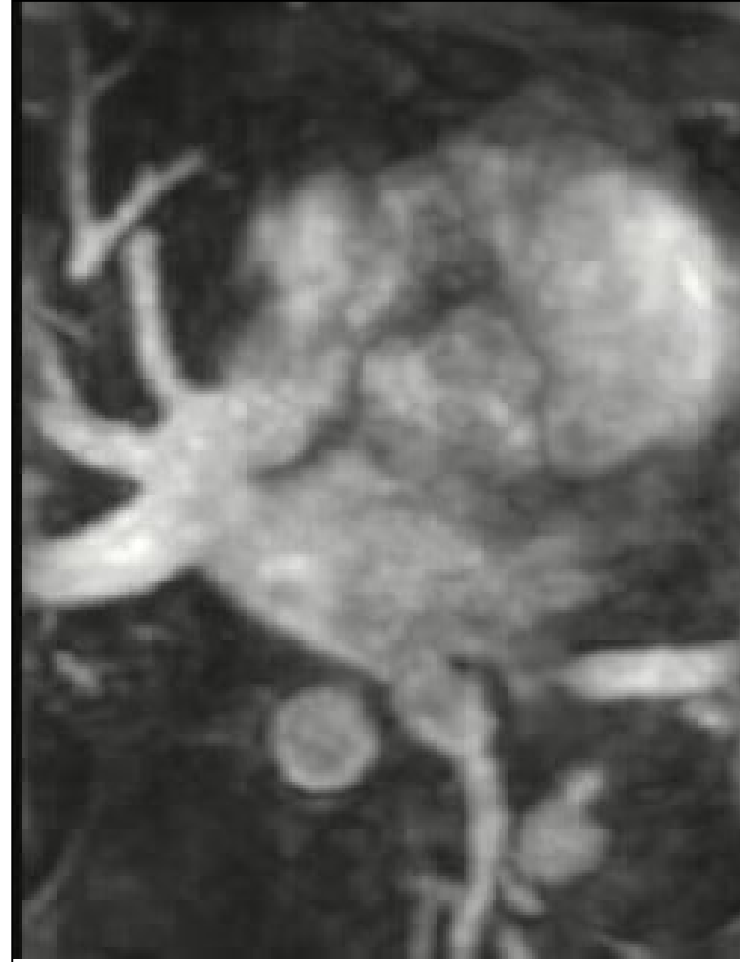
RV volumes: EDVI 240 ml/m²

CIA type sinus venosus

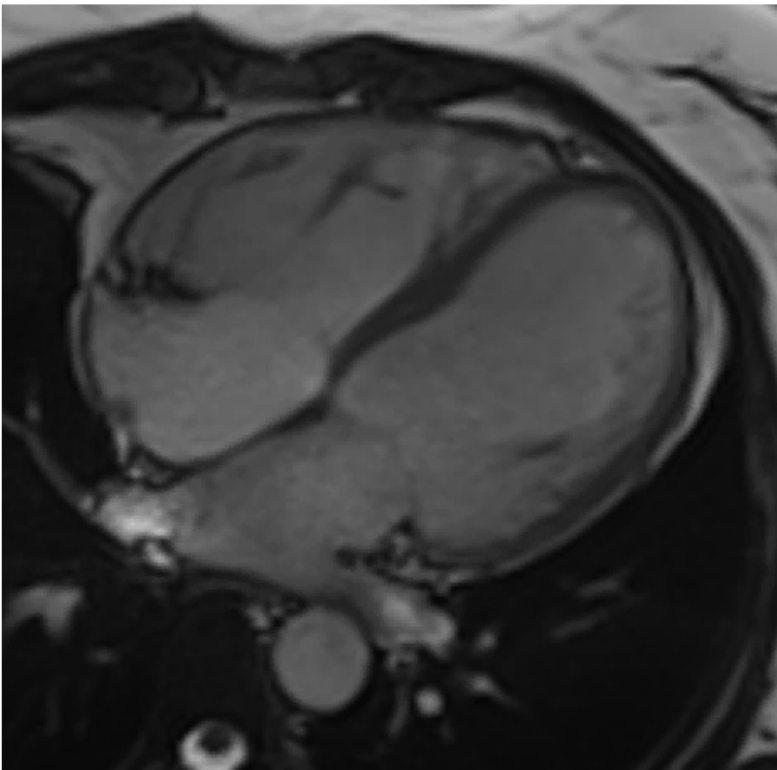
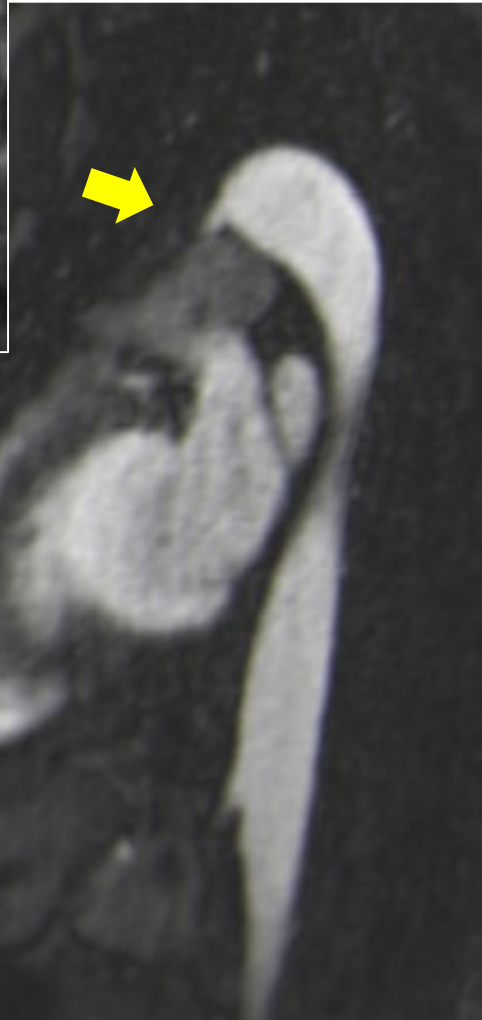
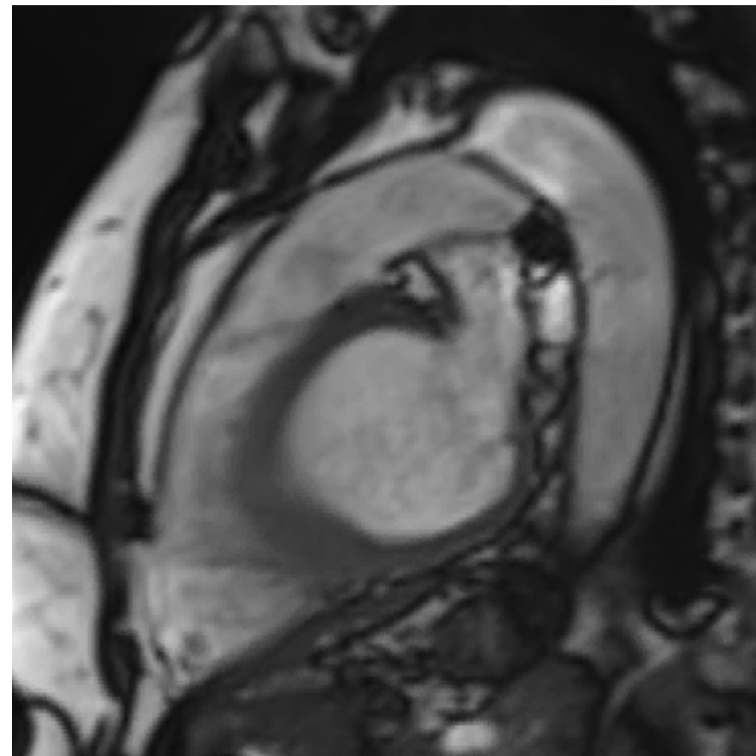
- Visualization direction shunt: gauche-droite
- $Q_p/Q_s = 4$



CIA type sinus venosus – Retour veineux pulmonaire anormal



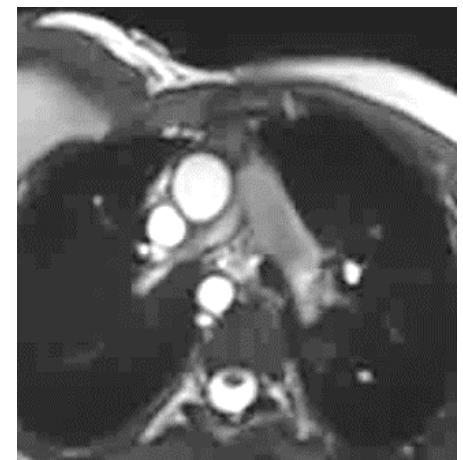
- Femme, 37 ans
- **Asthénia**
- ETT PAPs **35** mmHg
- IRM
 - VG 64 %, EDVI **104** ml/m², SV **66** ml/m²
 - VD 43 %, EDVI 79 ml/m², SV **33** ml/m².



Persistence
ductus arteriosus

Tetralogie de Fallot : follow up

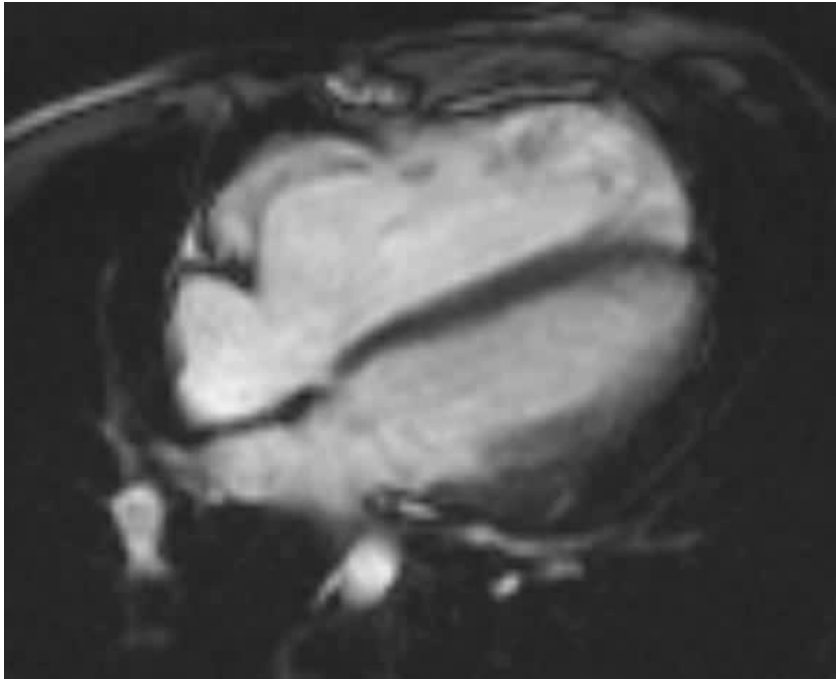
- Volumes VD (timing valvulation pulmonaire)
- Quantification de la régurgitation pulmonaire
- Sténose infundibulaire résiduelle
- Sténose des artères pulmonaires / répartition des flux



Remplacement valvulaire pulmonaire

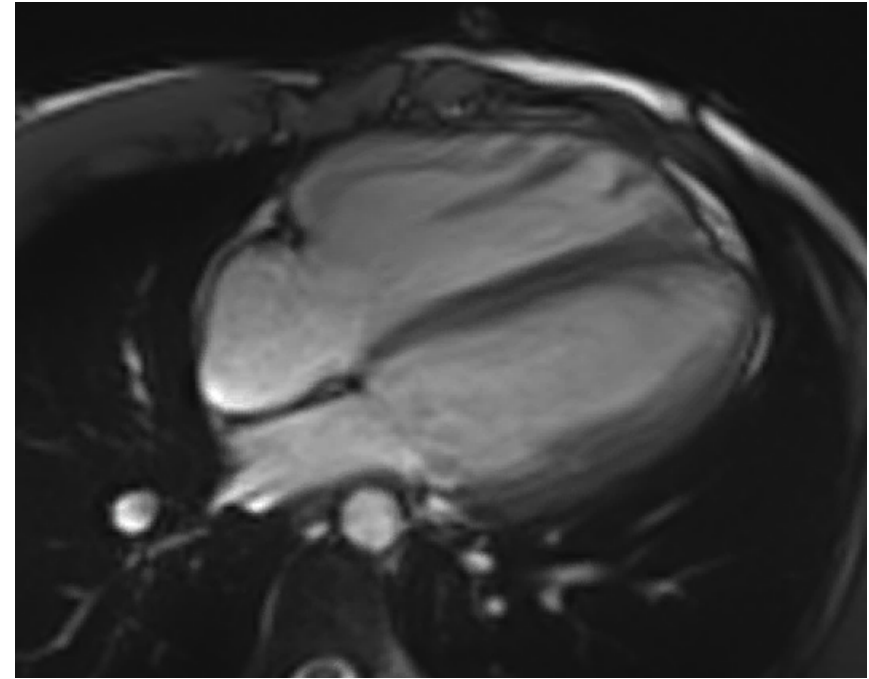
15y, boy

Pre



RVEF 51 %,
EDVI 192 ml/m²
ESVI 95 ml/m²

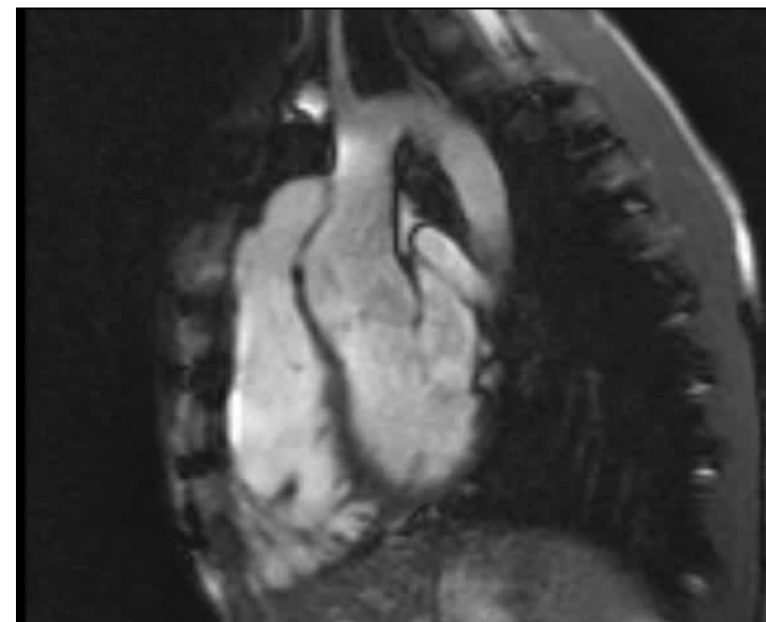
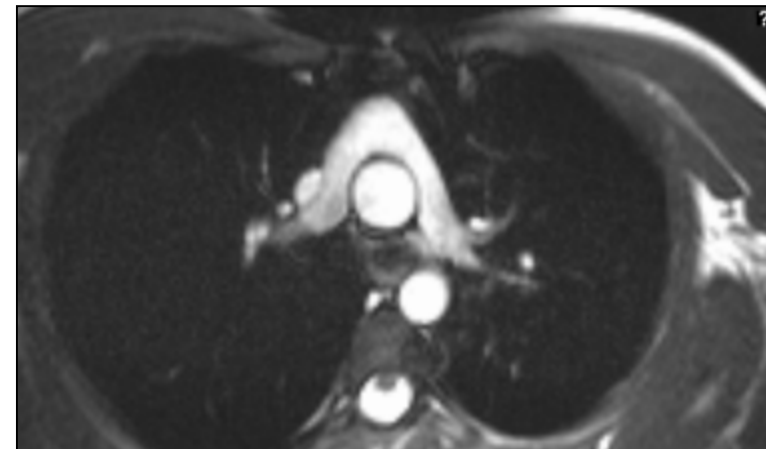
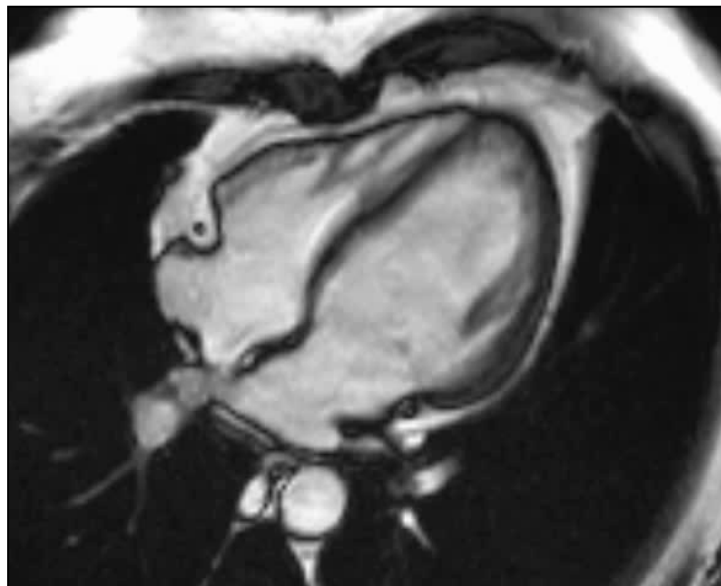
Post



EF 52 %
↓ EDVI 112 ml/m²
ESVI 53 ml/m²

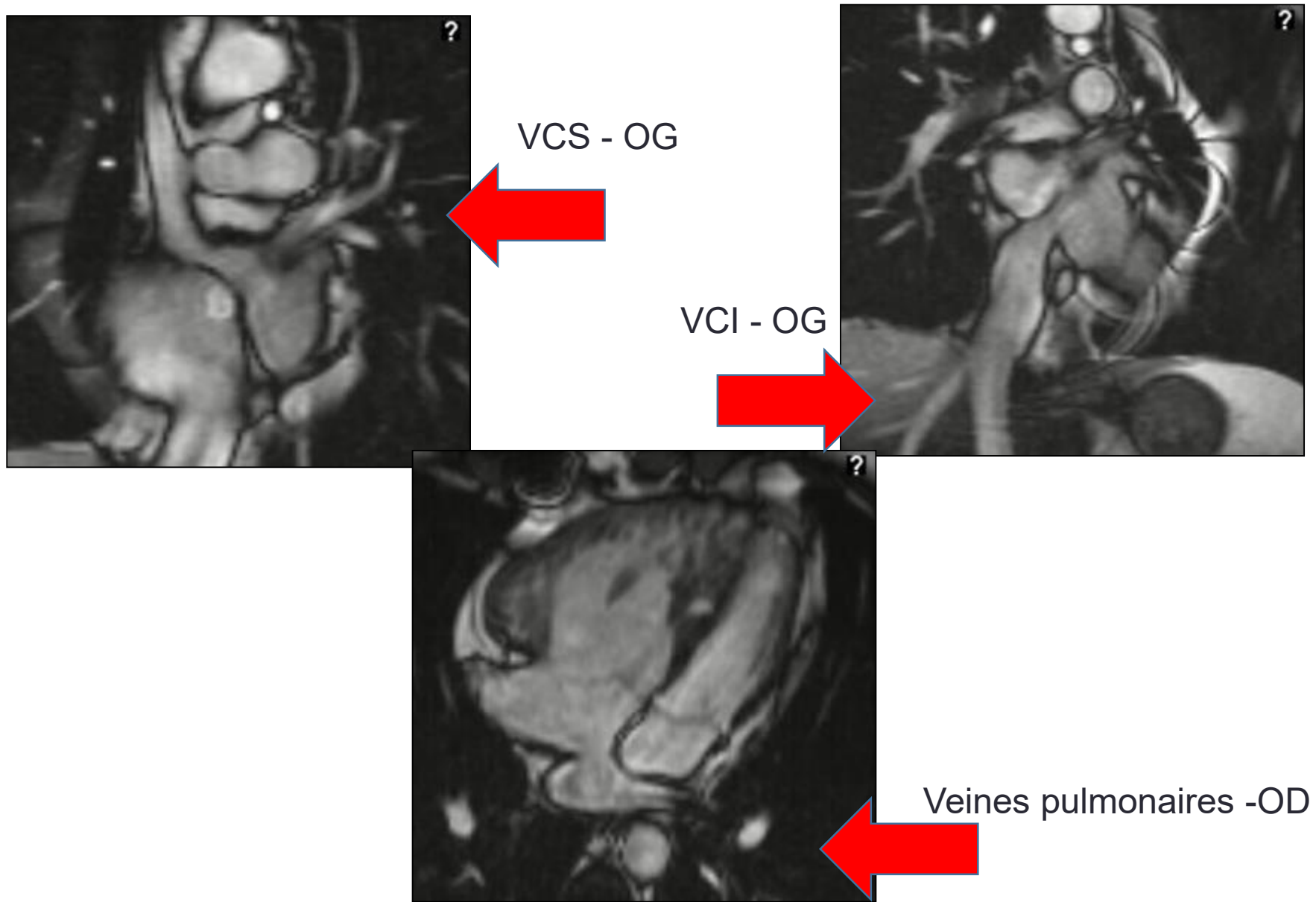
TGA : arterial Switch

manoeuvre de lecompte



15y boy

TGA : venous Switch (Mustard /Senning correction)



Double Discordance (CCTGA)

Homme 30 ans, asymptomatique

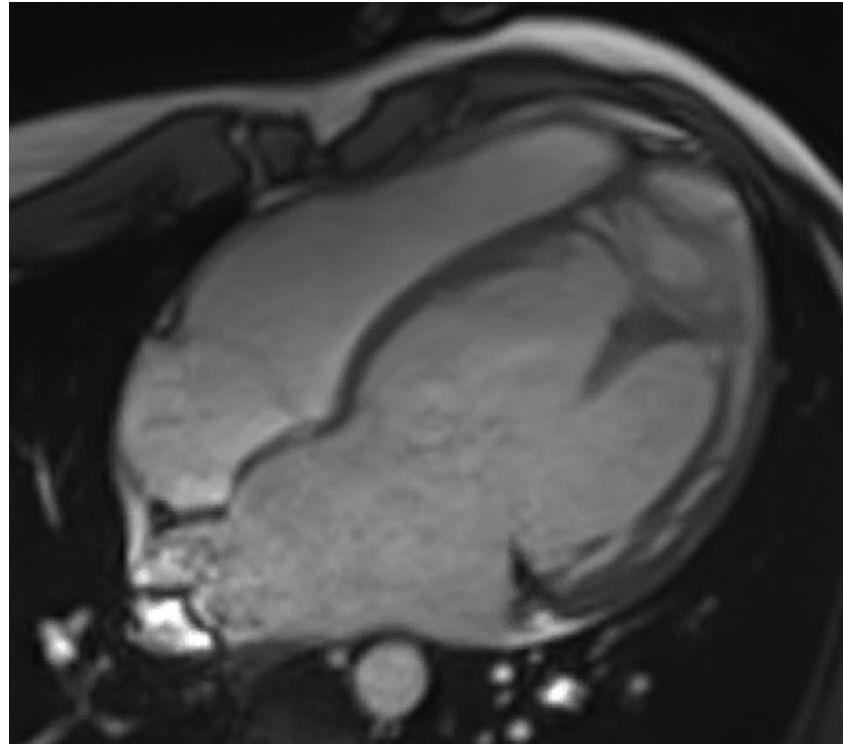
«ventricule systémique EF 50 %

EDVI 217 ml/m²

LV or RV?



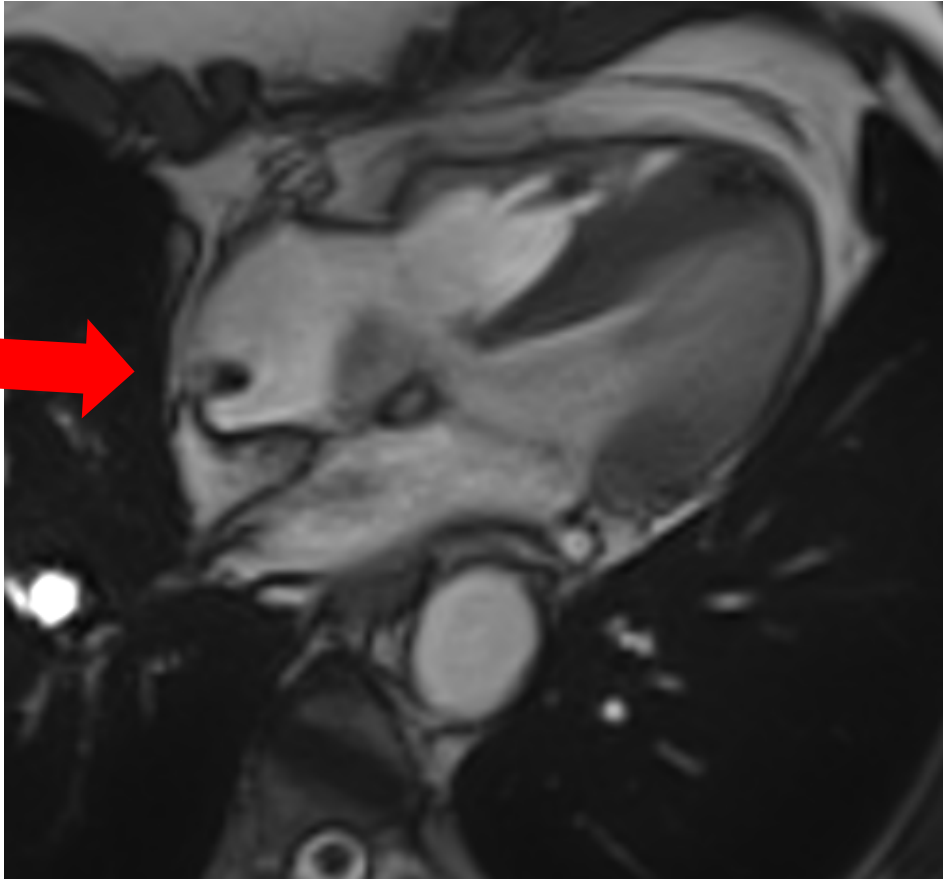
- Bande modératrice
- Valve AV suis le ventricule
- dislocation tricuspide vs apex



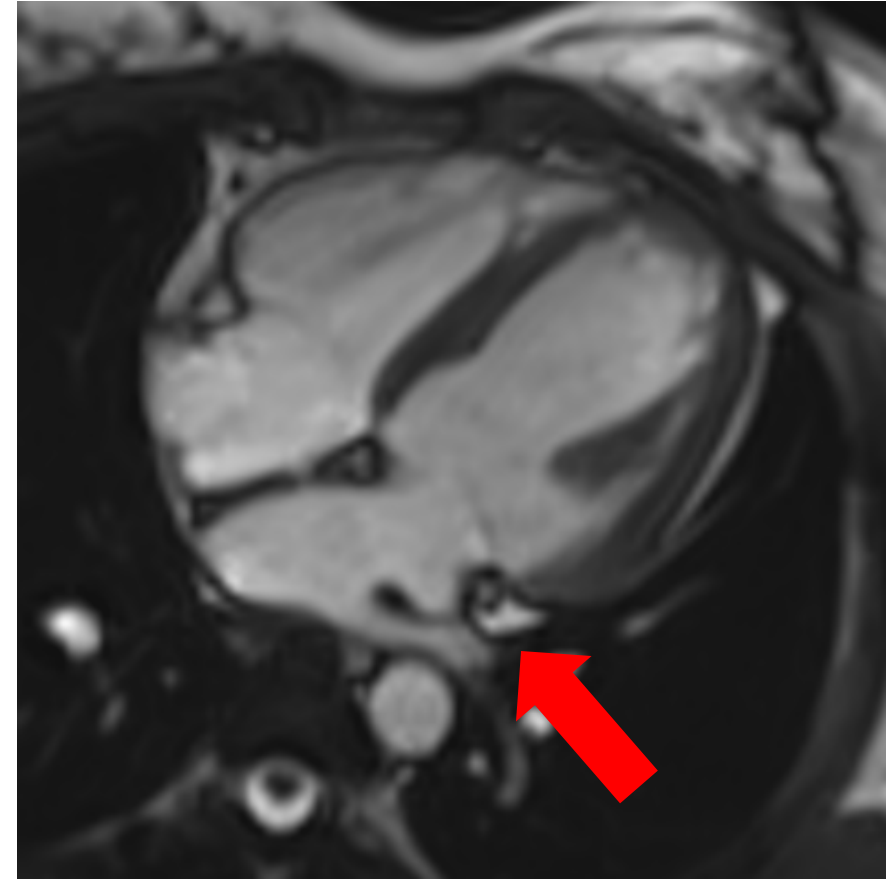
Masses cardiaques

- Variante anatomique normale
- Masses non tumorales (thrombus, calcifications, kyste)
- Tumeur bénigne
- Tumeur maligne
 - Localisation
 - Forme
 - Rapport avec structures cardiaque et extra cardiaque
 - Signale T1, T2 , perfusion, LGE

Crista terminalis



coumadin ridge

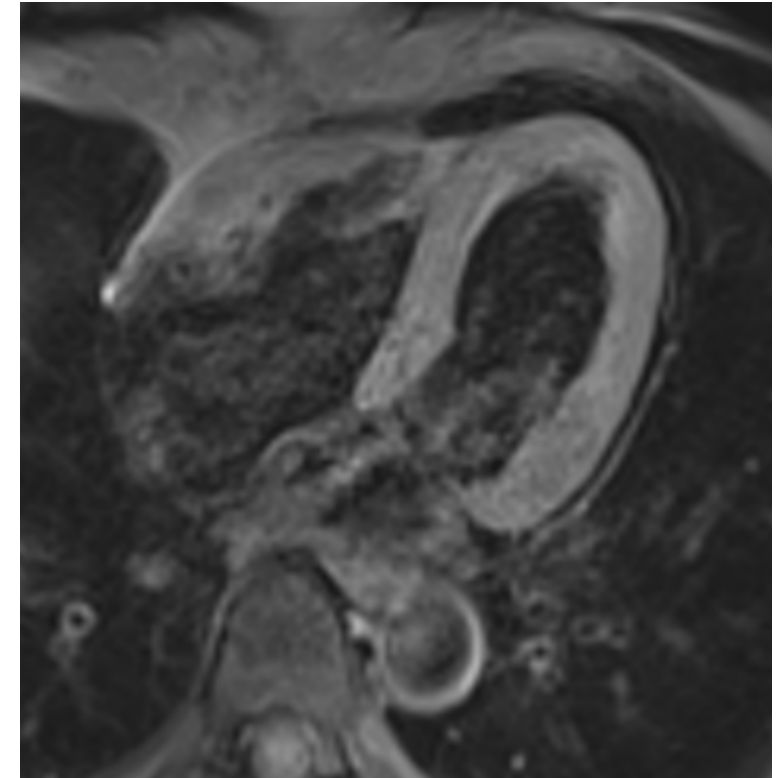
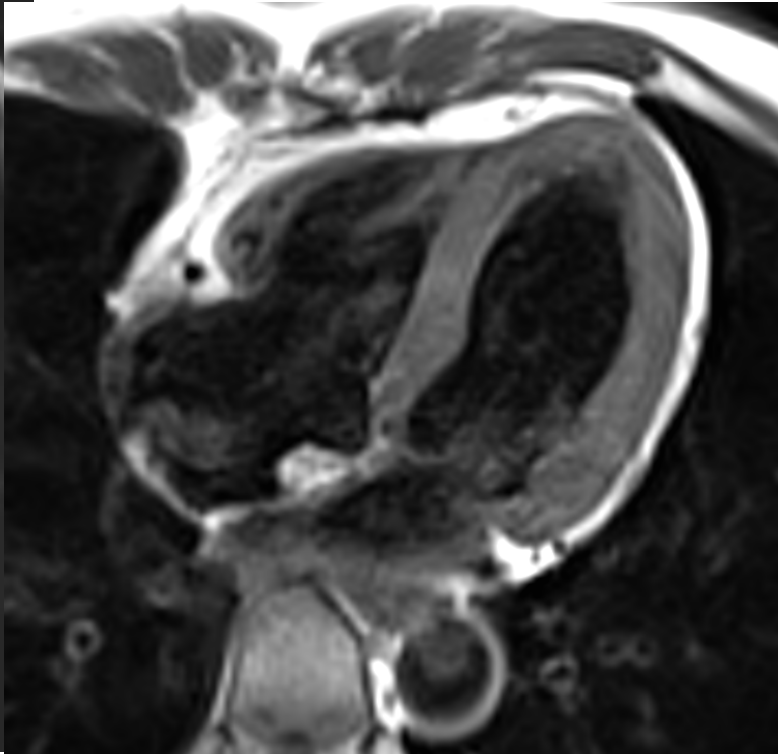
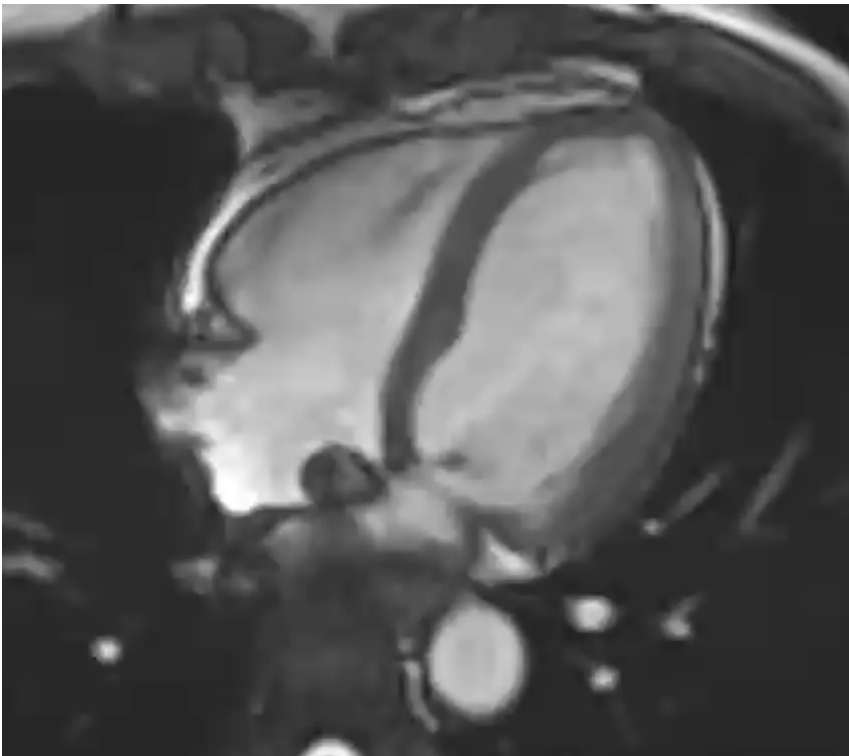


hypertrophie lipomateuse du SIA

SSFP

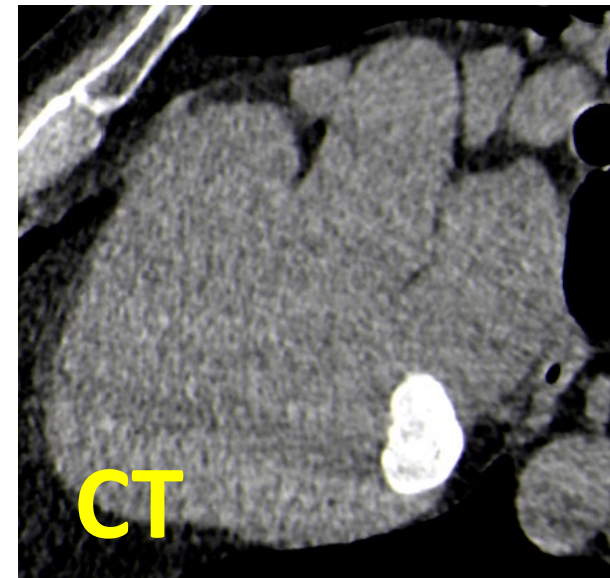
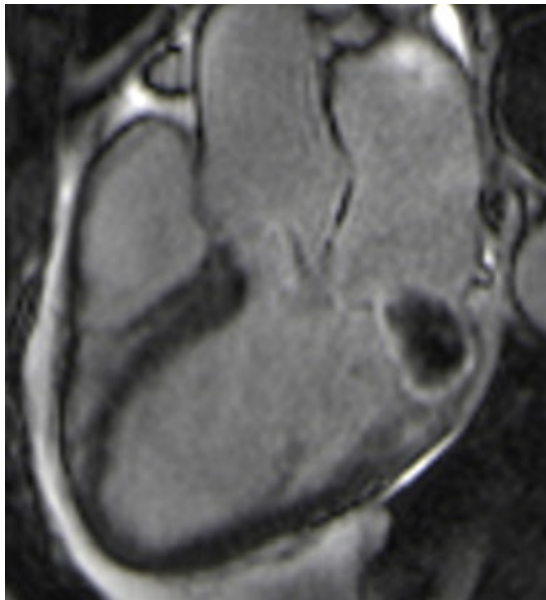
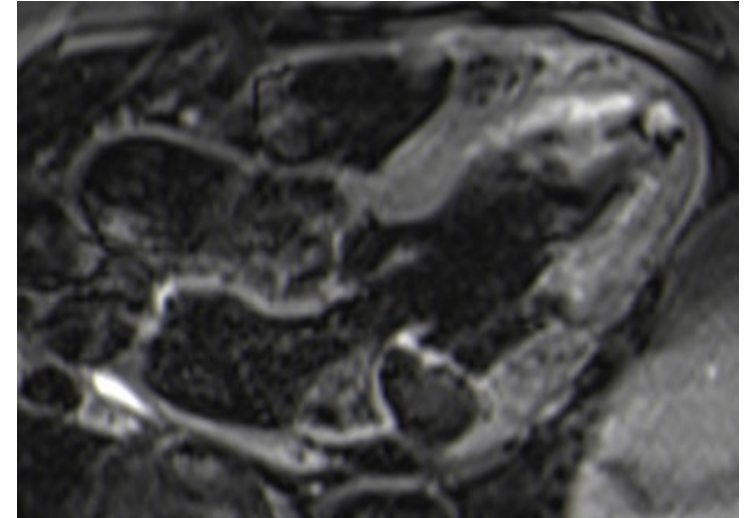
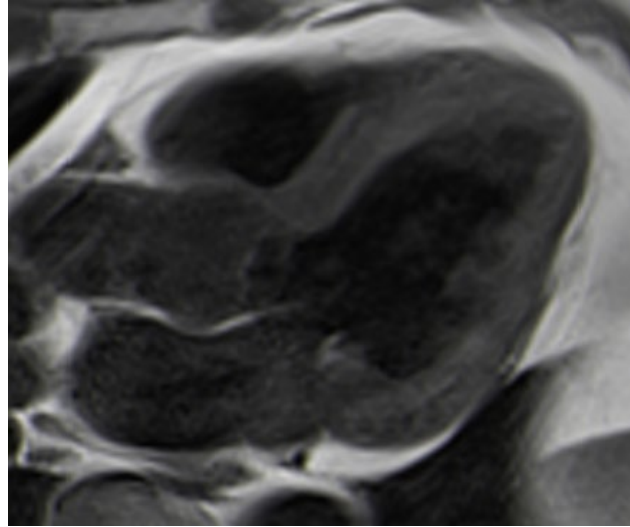
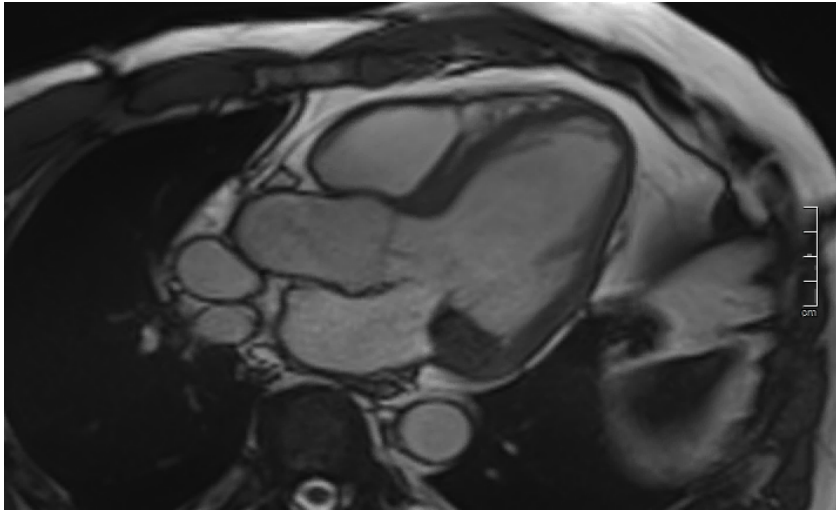
TSE T1

fat sat



Homme , 62 ans, découverte fortuite

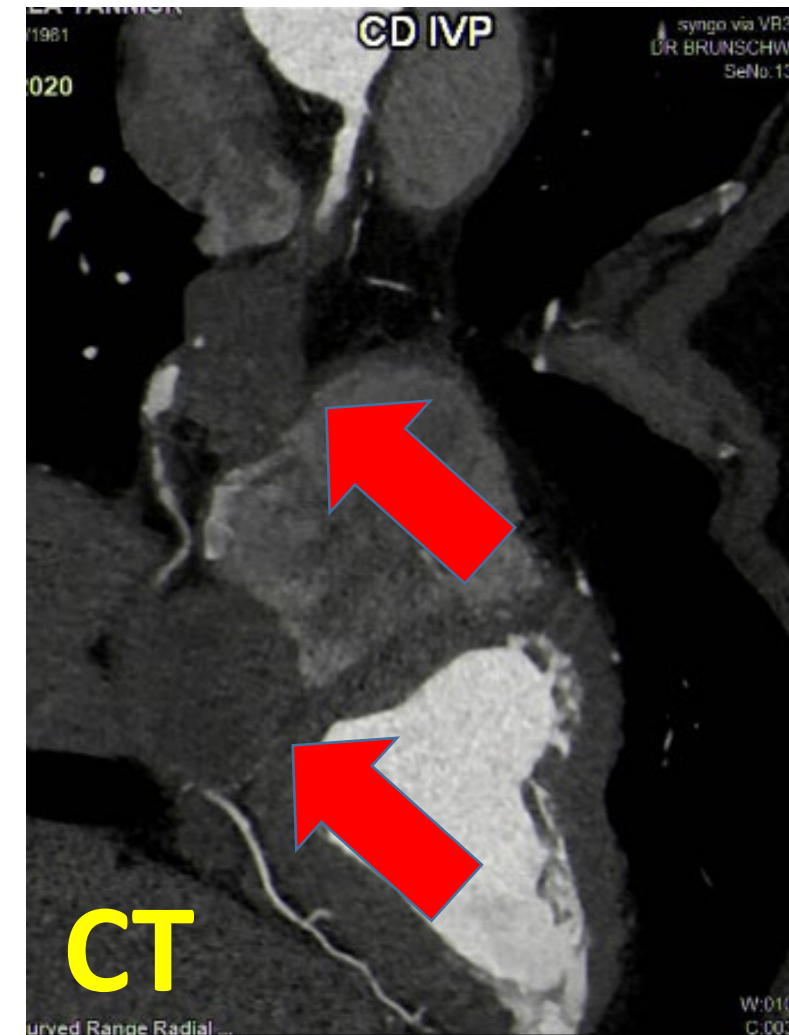
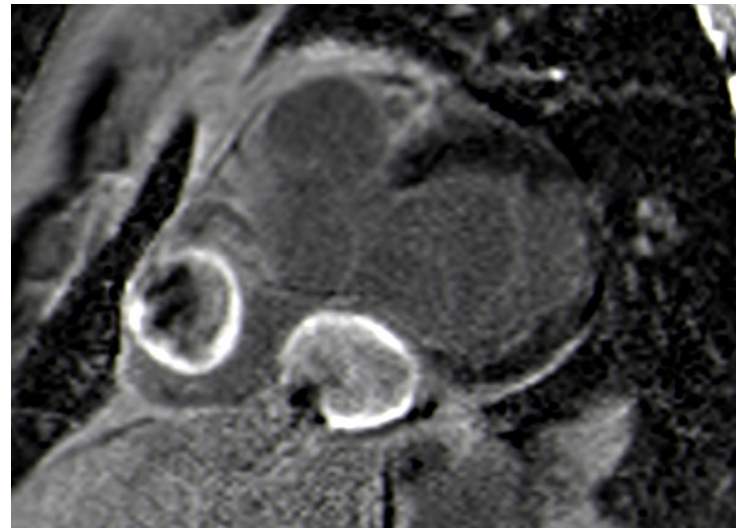
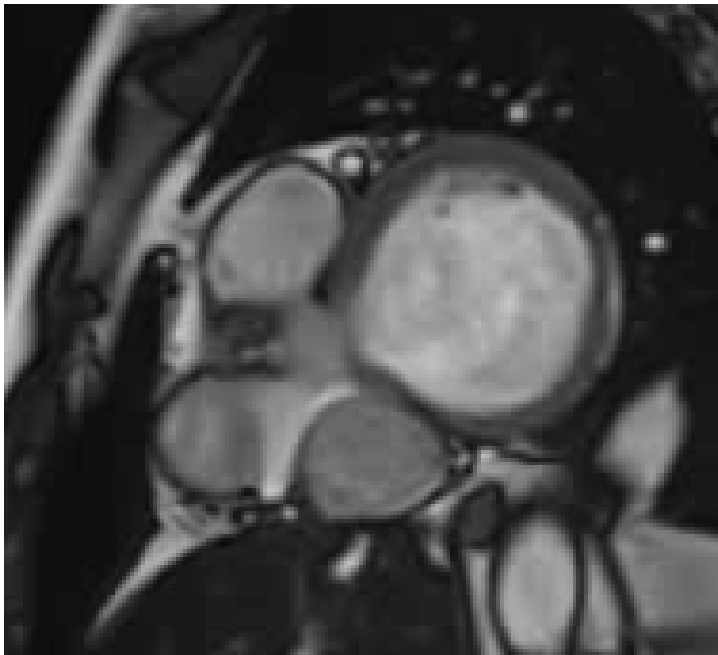
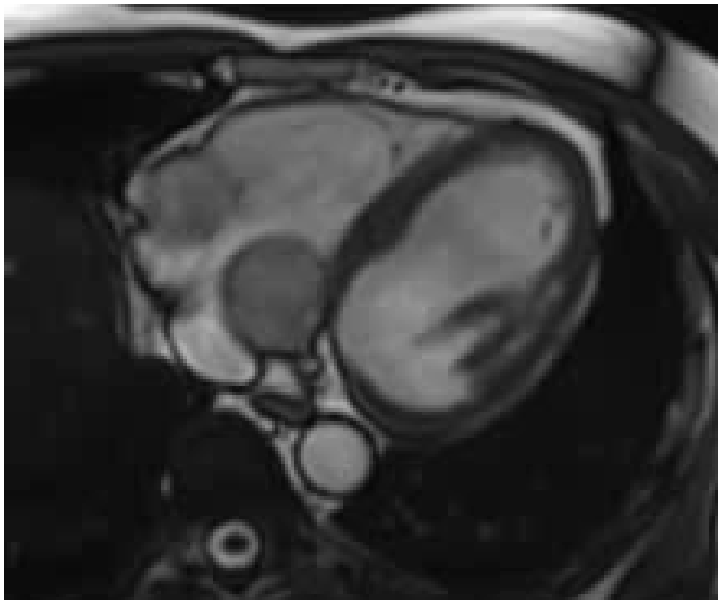
Calcification caséuse de l'anneau mitrale



Homme, 59 ans

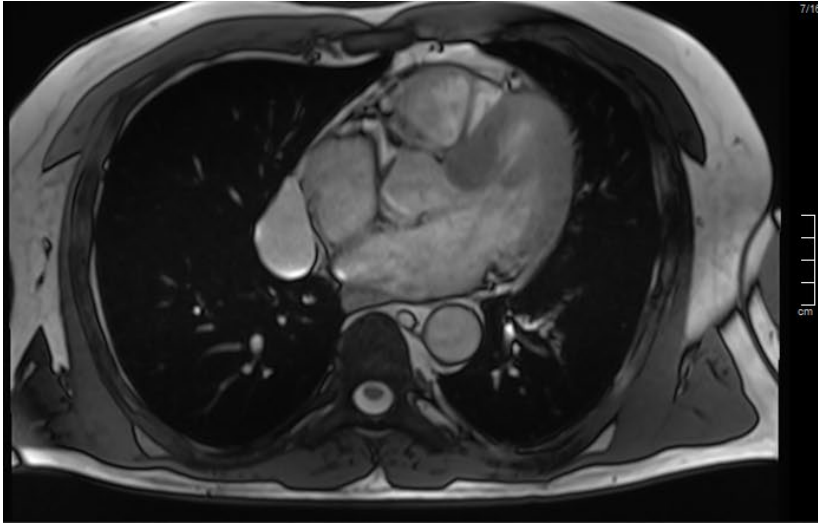
- ATCD infarctus en 2009 (« ectasie des coronaires »)
- leucémie aigüe en Mars 2019
- 6 mois apres à l'ETT présence de deux « tumeurs para-cardiaques » devant l'OD

Double anévrisme thrombosé de la CD

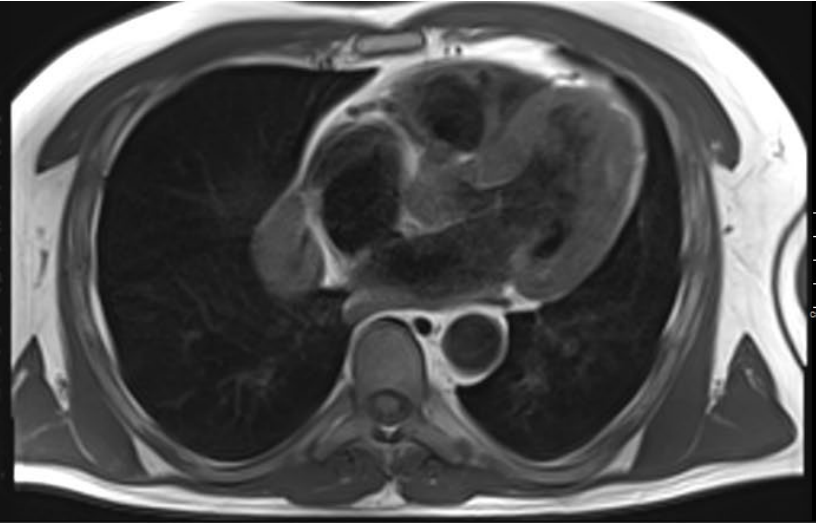


Kyste pleuro-pericardique

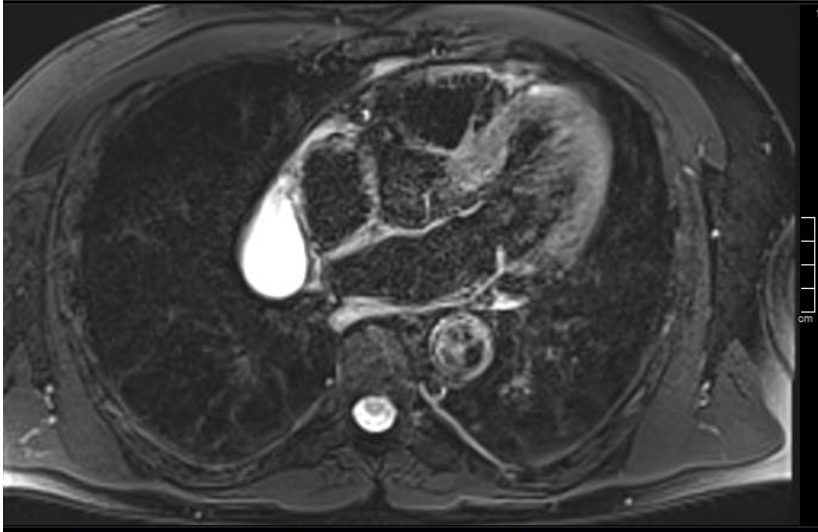
SSFP



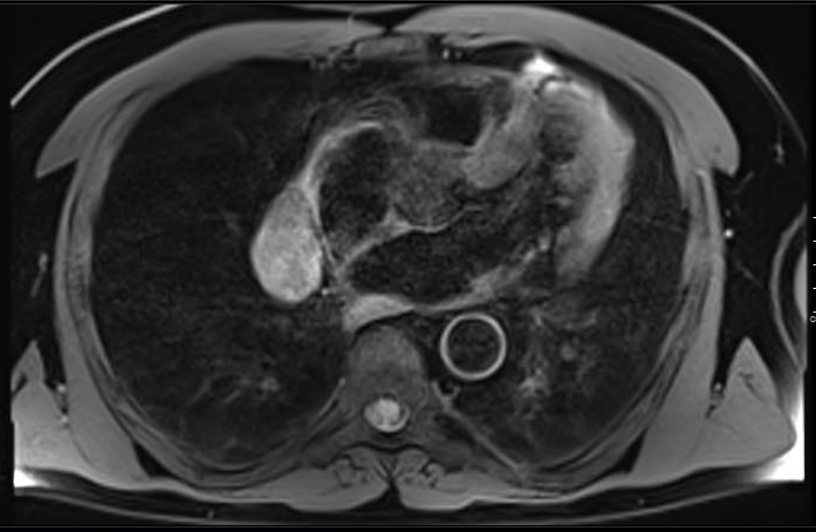
TSE T1



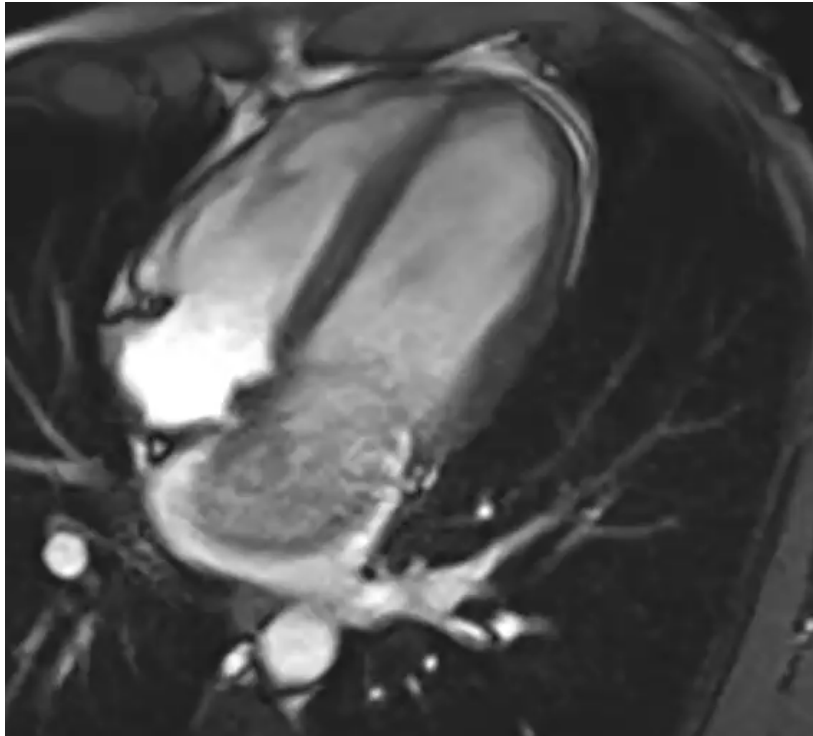
STIR T2



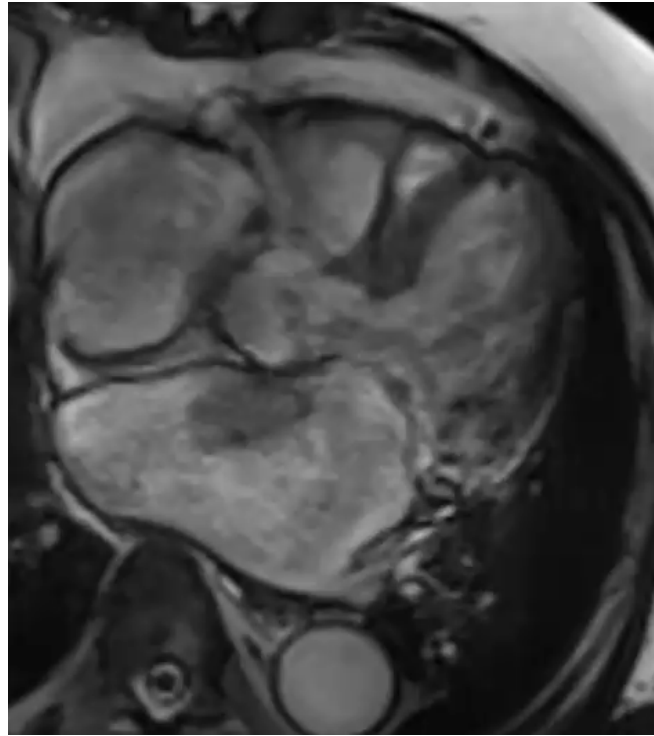
TSE T Fat Sat



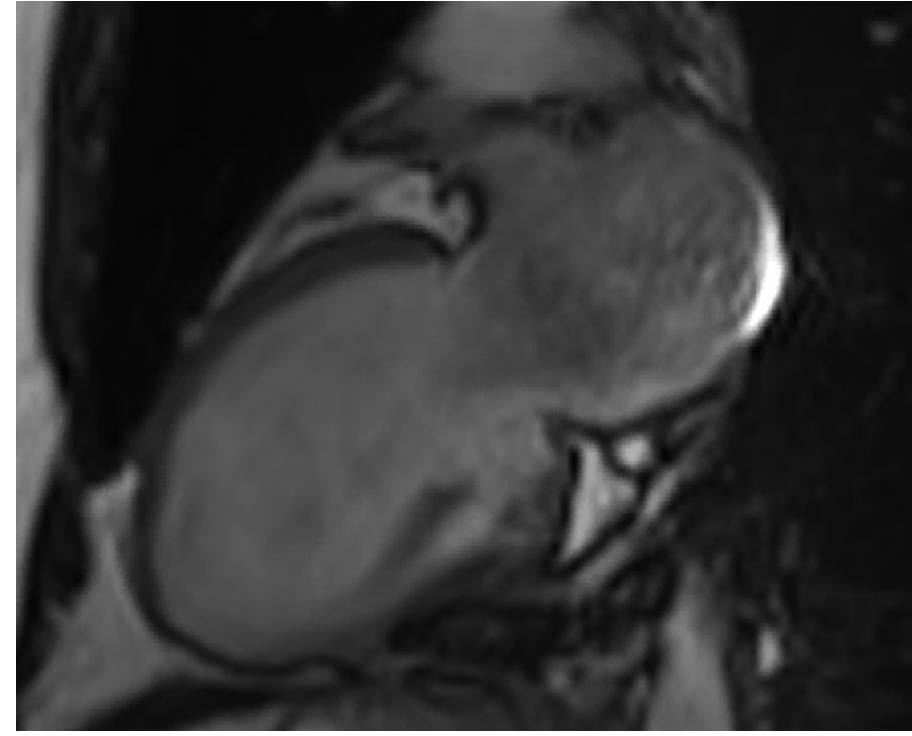
Myxoma



thrombus



sarcome



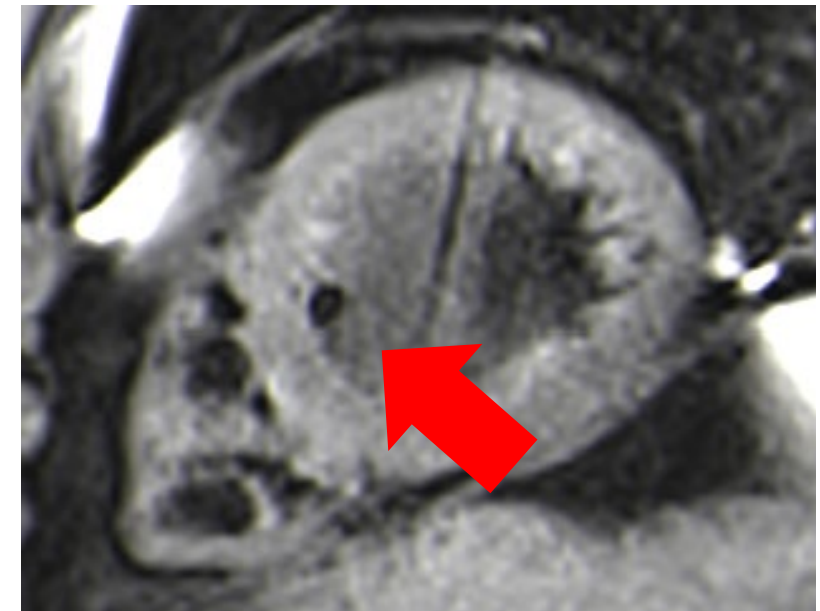
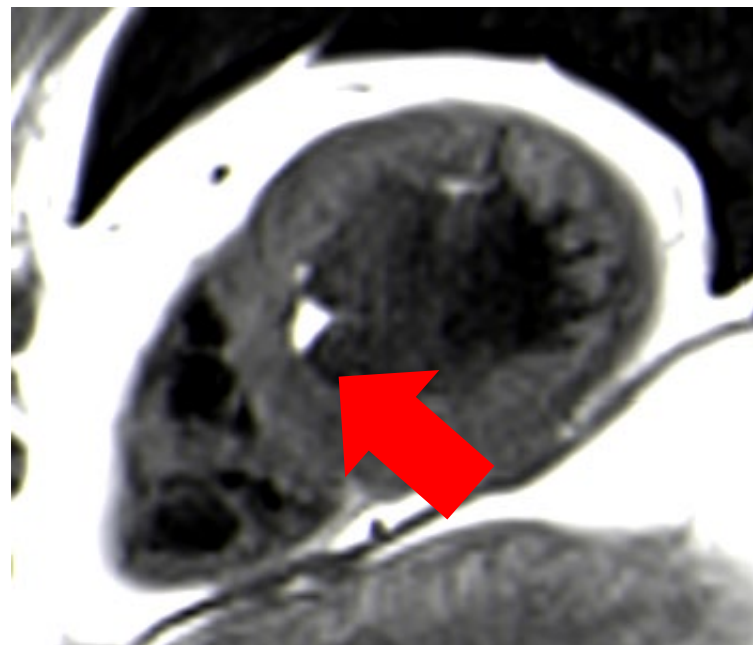
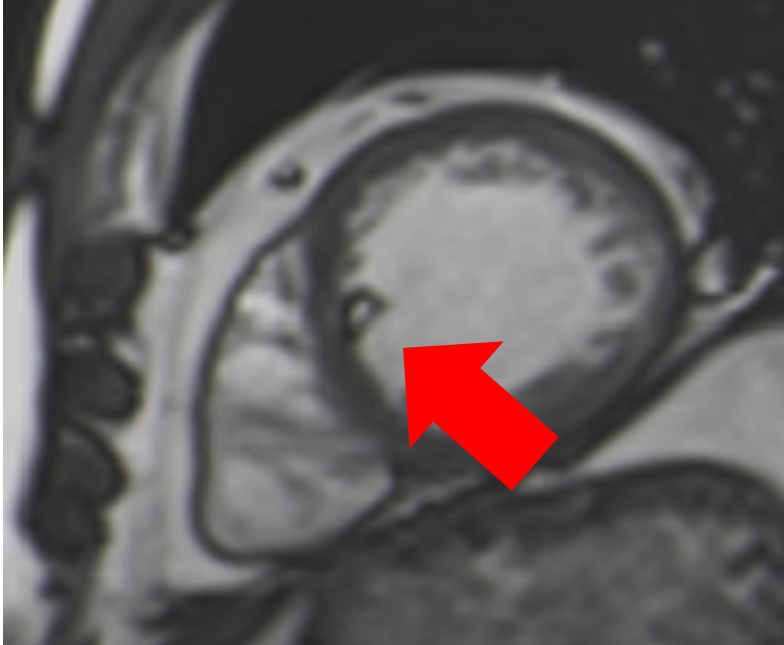
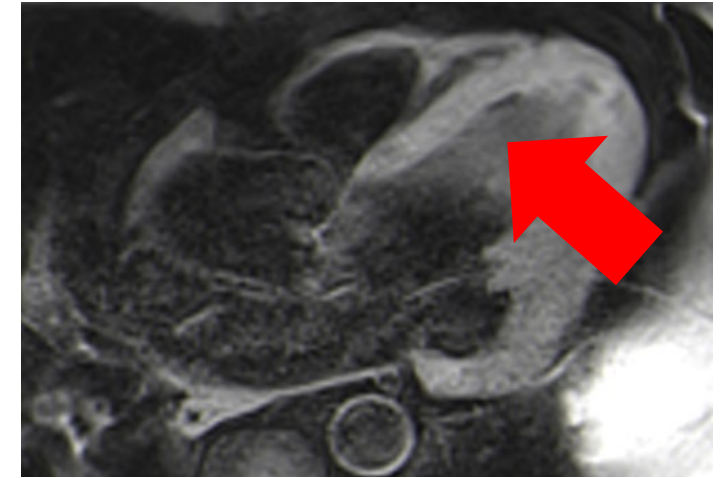
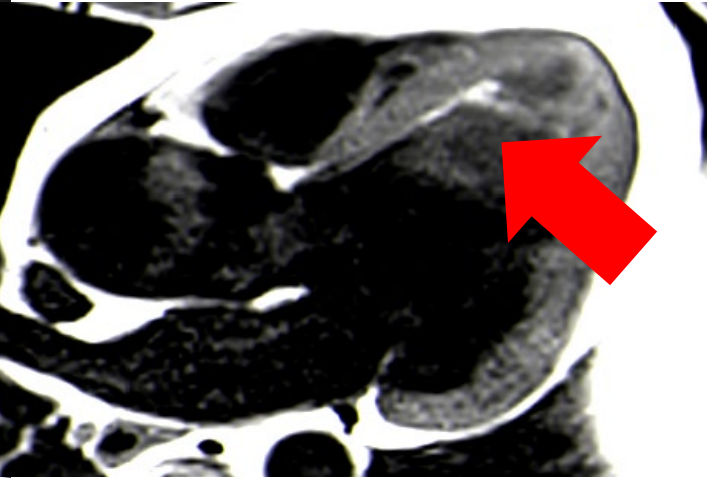
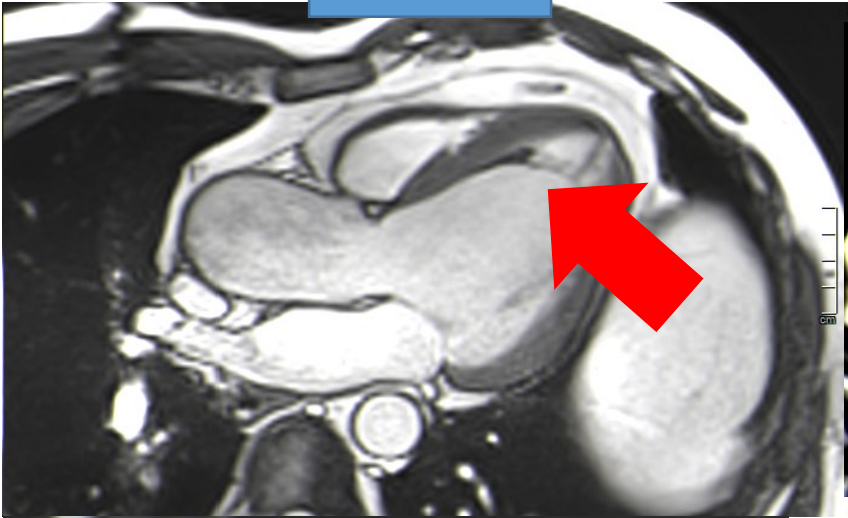
Homme 53 ans

- CMD depuis 2004 FE 39%
- IRM (16/6/21) présence d'une image de 8mm à l'apex, avec rehaussement de gadolinium (thrombus ? fibrome ?).

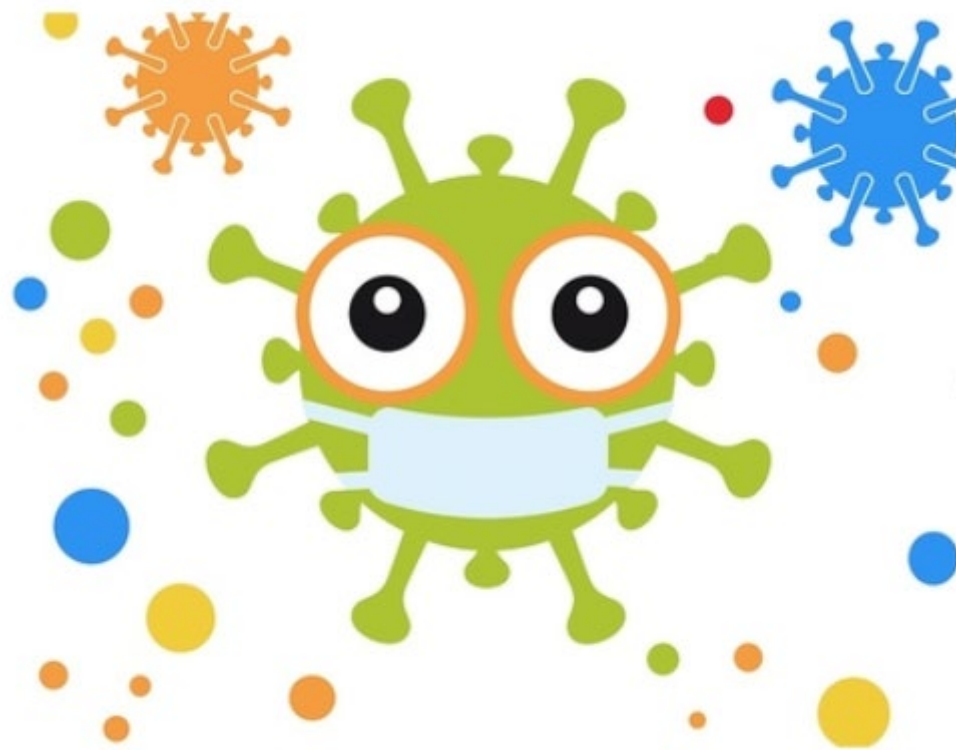
SSFP

TSE T1

Fat sat



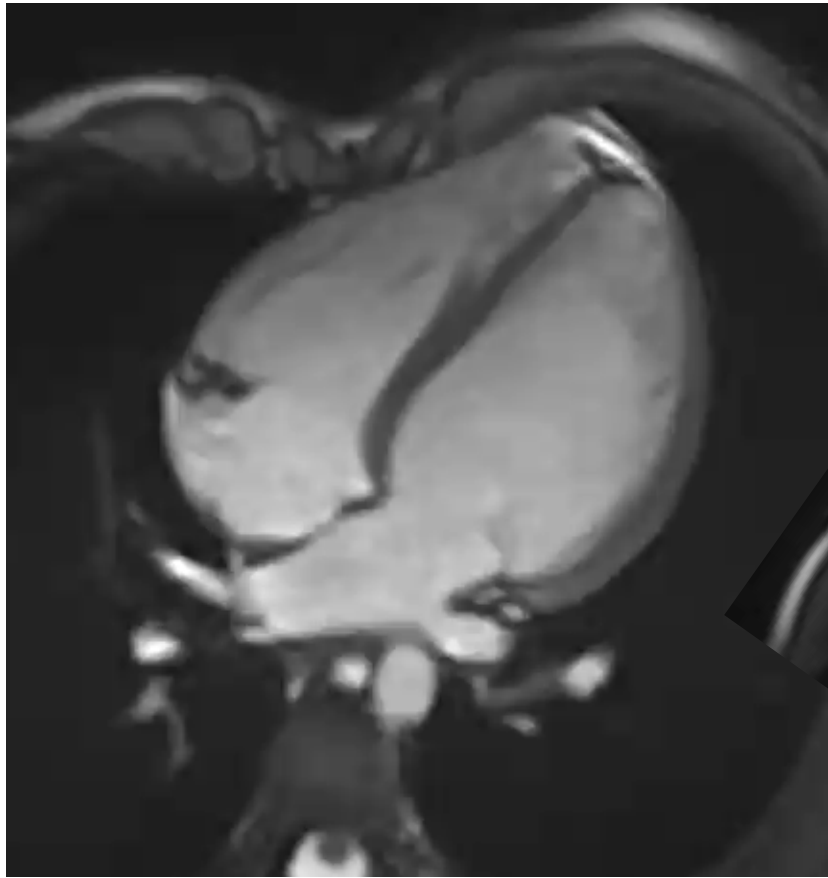
EF à 50 %, EDVI 110 ml/m²



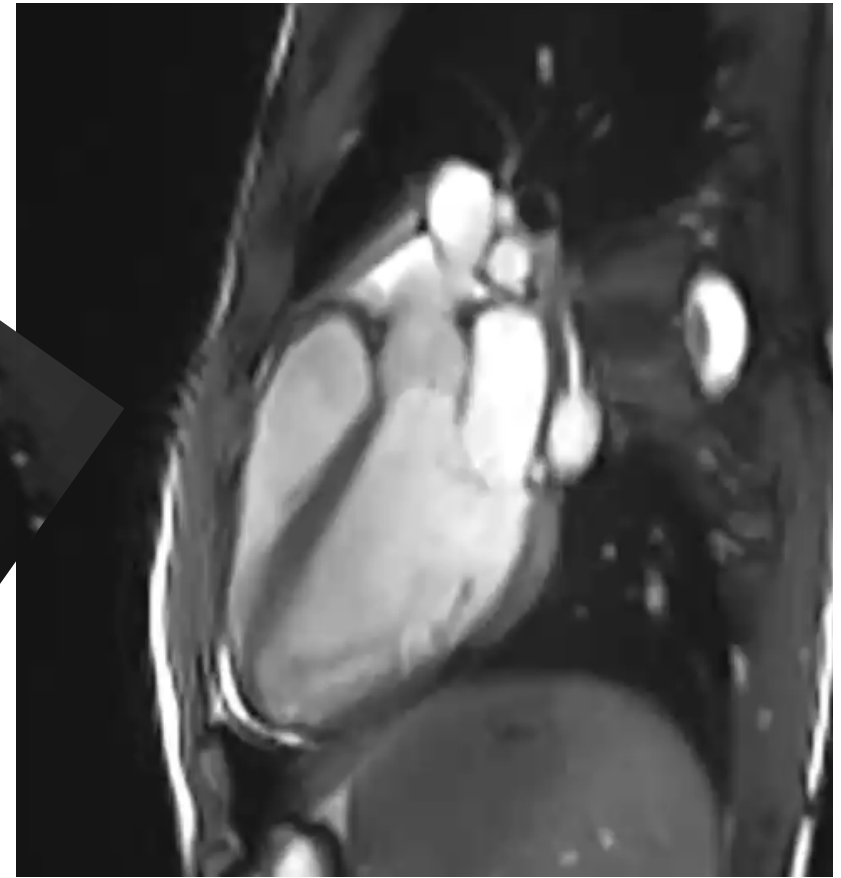
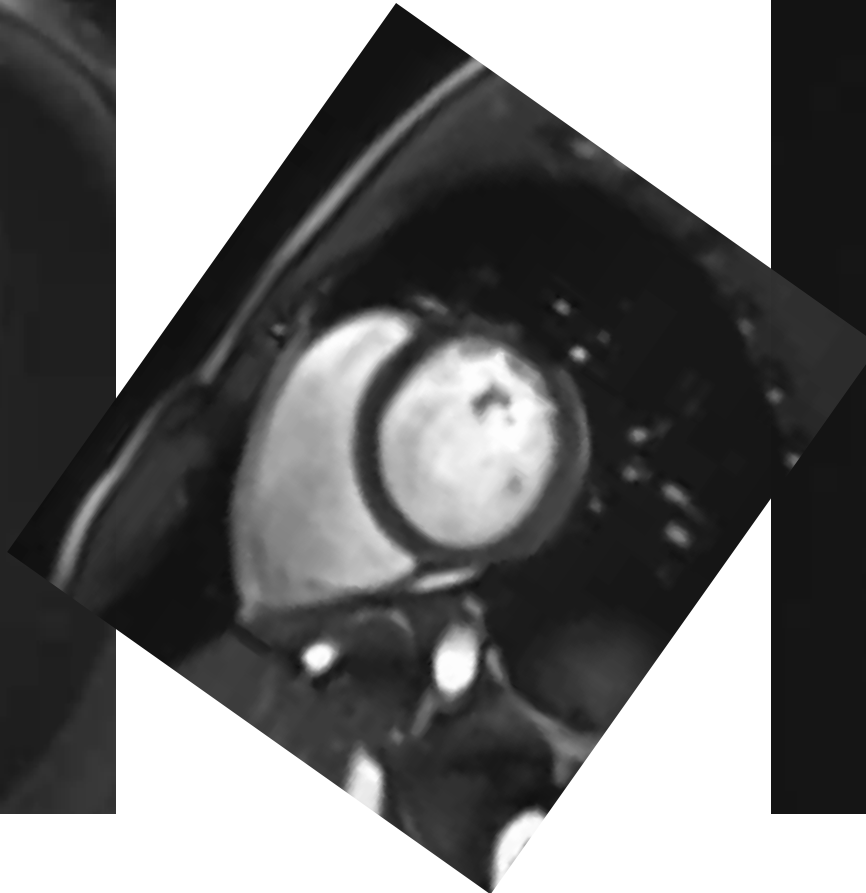
- Jeune patient de **13 ans**, sans ATCD, ayant présenté une **douleur thoracique** 48h après la deuxième injection du Vaccin anti COVID Pfizer
- **troponine** élevé à 354 ng/l (norme < 14)
- NT-pro-BNP à 197 pg/l.
- ETT dans la norme.



Adressé en IRM pour recherche de myocardite.

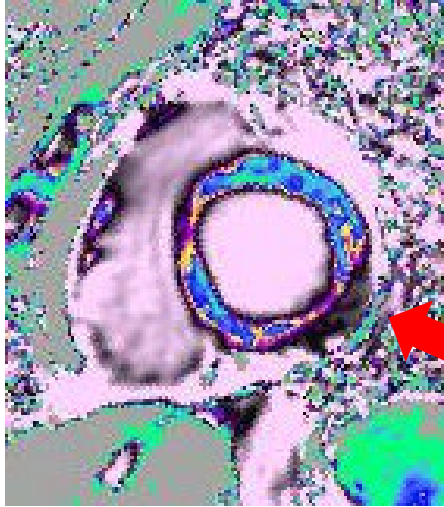


EF à 69 %

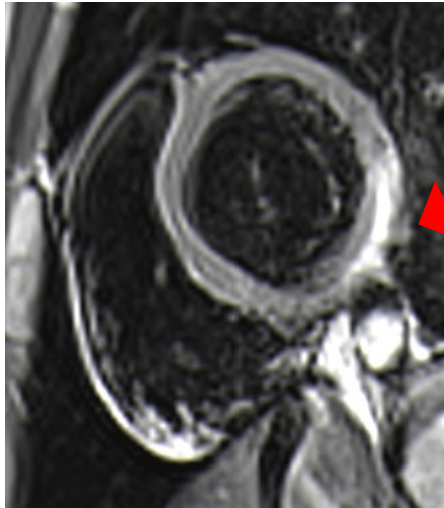
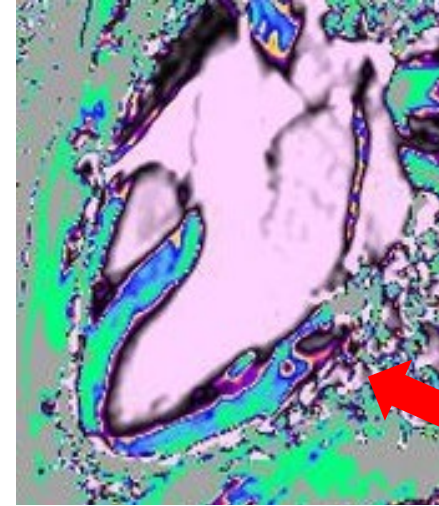


SLG à - 19%.

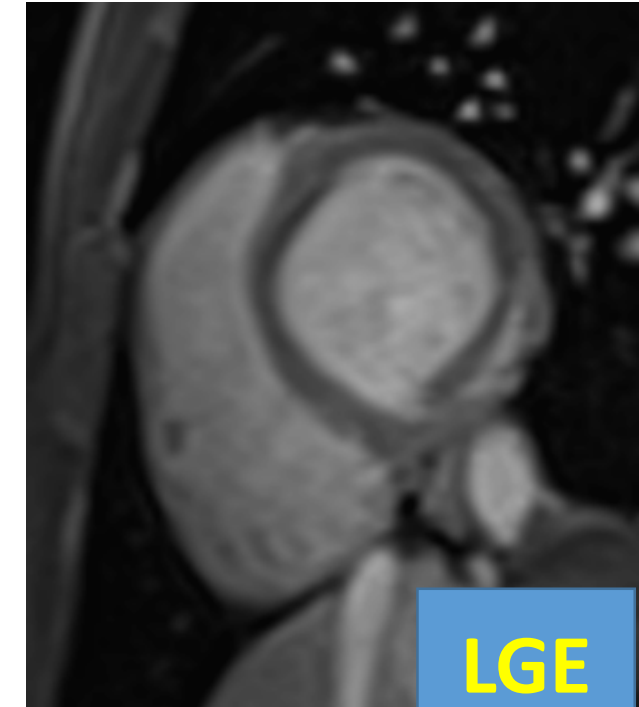
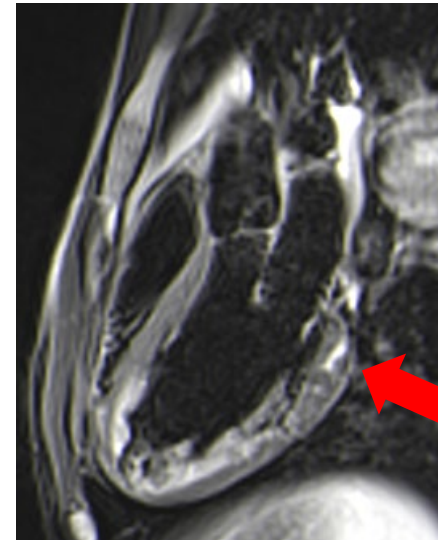
Myocardite post vaccine anti COVID



T1 mapping

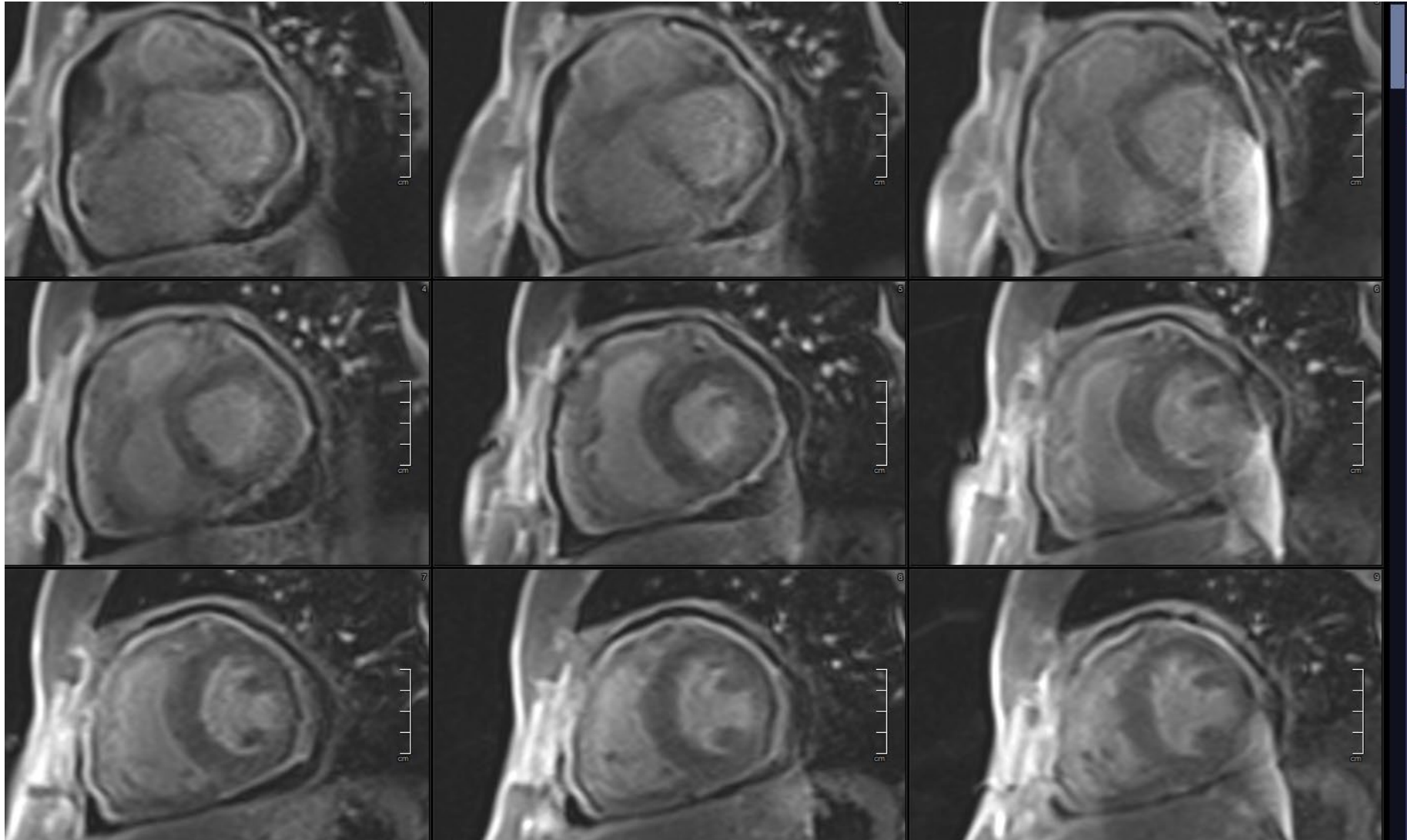


STIR (séquence T2)



LGE

- Homme, 29 ans
- **anasarque** avec syndrome biologique inflammatoire
- **Drainage péricardique** de 1.1 l
- Vaccin anti-COVID 19 **Pfizer** 3 mois avant



Valeur ajoutée de l'IRM cardiaque

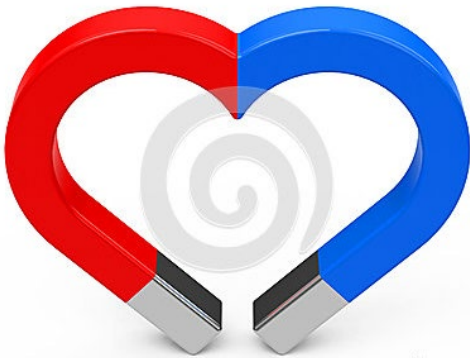
Diagnostic

- Caractérisation tissulaire
- Visualisation aisée de VD, apex VG, péricarde, aorte, pulmonaire



Facteurs pronostiques:

- FE
- Volumes
- Strain
- VD
- fibrose
- ischémie

I 
CMR

Merci !!!