Société Française de Cardiologie

HIA LAVERAN

mort subite du jeune sportif pathologies à risque

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Service de cardiologie
HIA Laveran
Marseille

A. Puerta 1984-2007

T Simpson 1937-1967

P Sauer 1977-2012

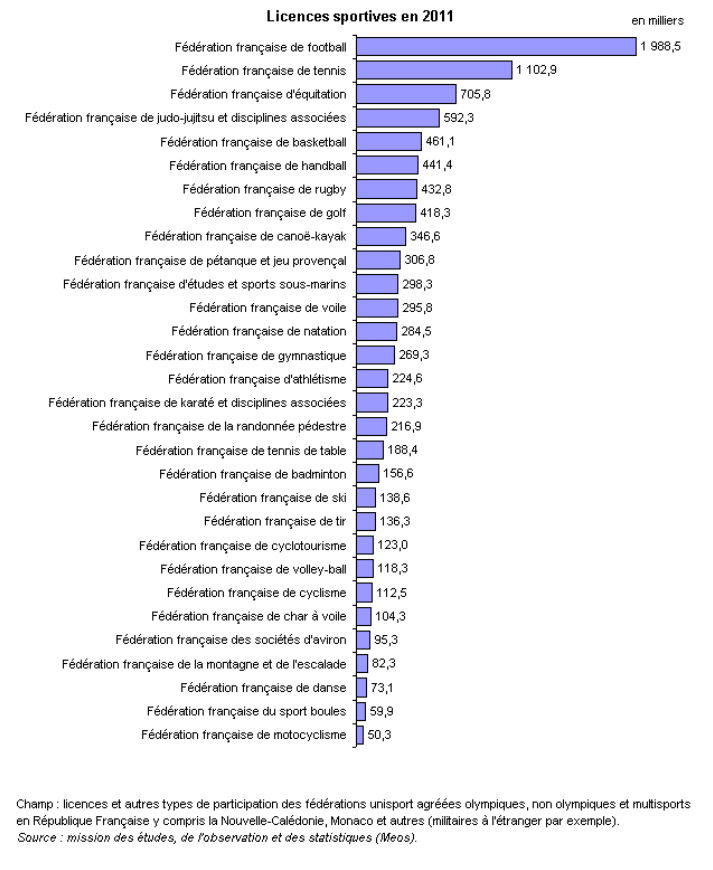
MV Foe 1975-2003

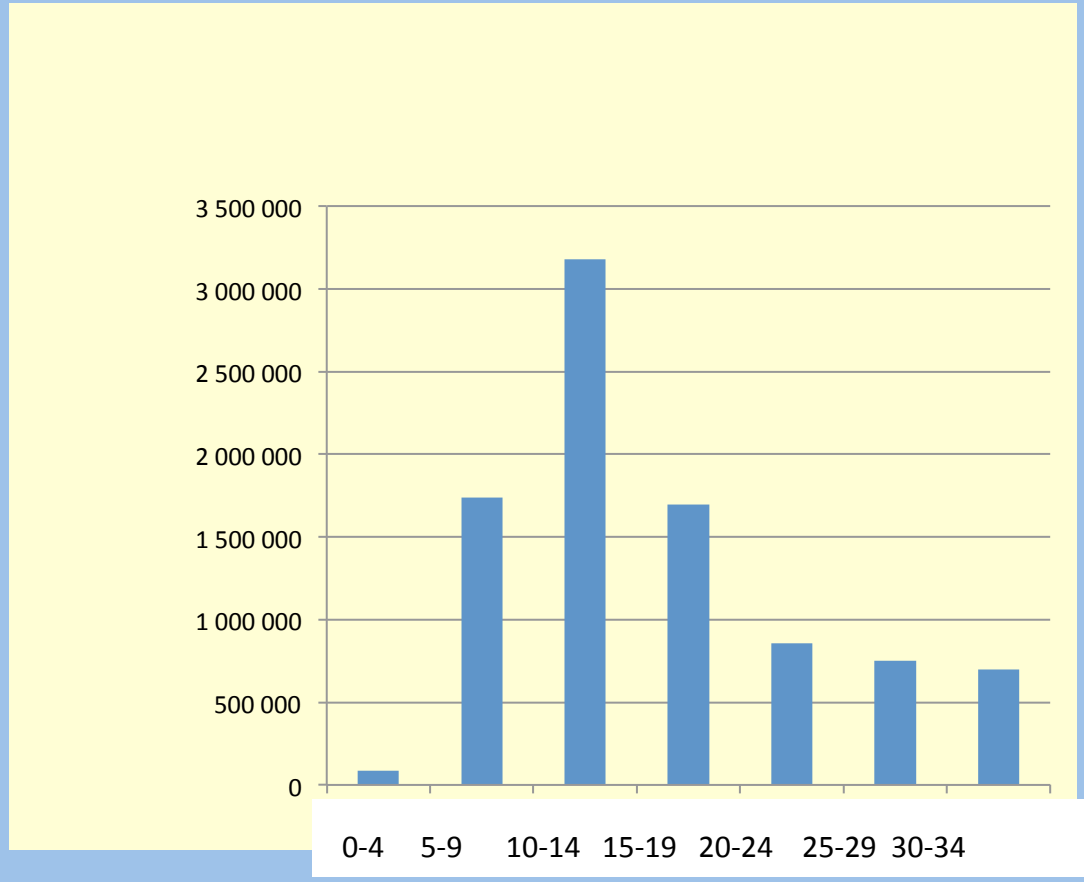
M Candau 1993-2009

F Latu 1980-2009

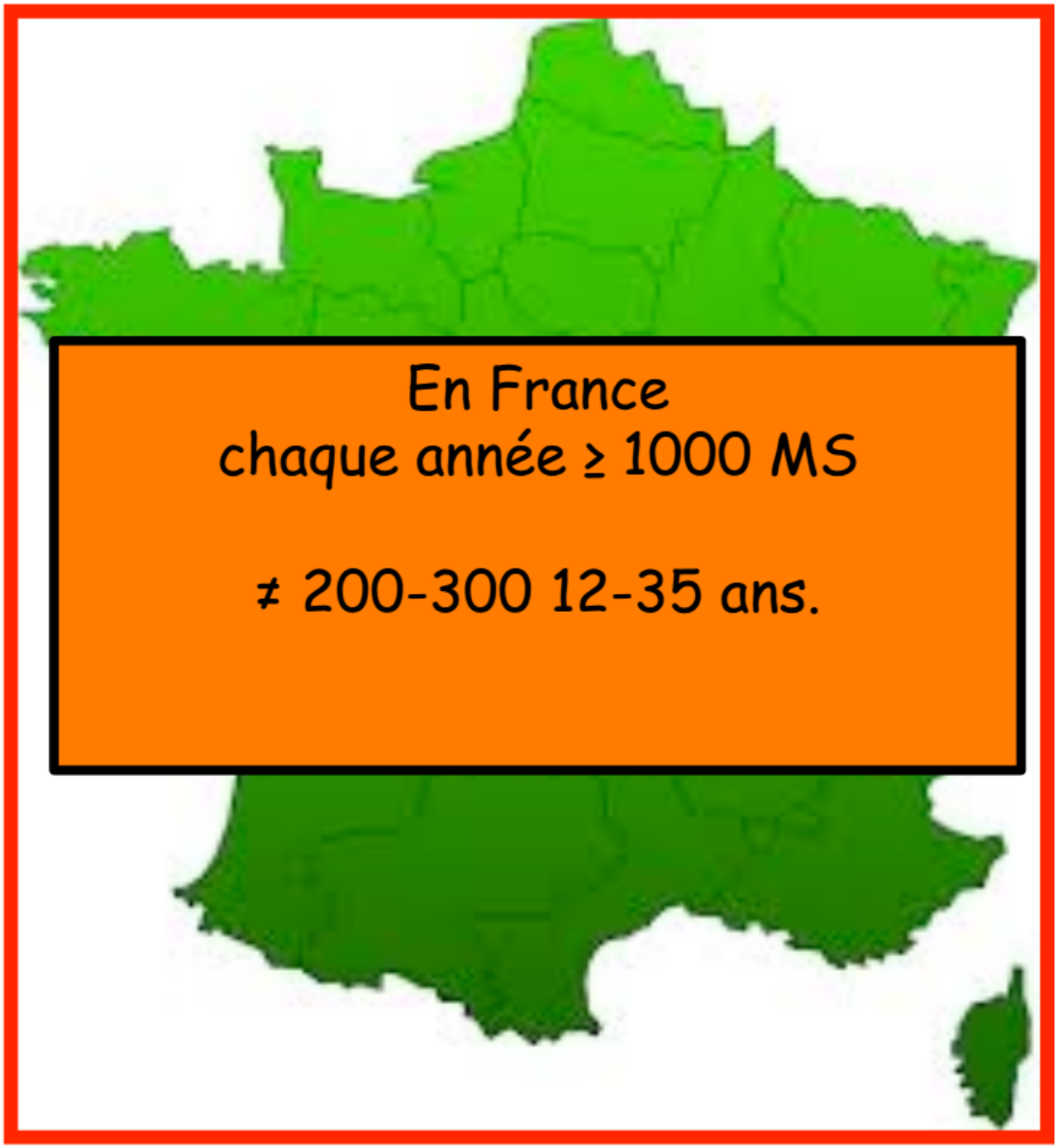
- **En France**
- 17.5 M
- 37% femmes
- 10 M competition

Une majorité de jeunes sportifs





INSEE 2011



En France
chaque année ≥ 1000 MS

≠ 200-300 12-35 ans.

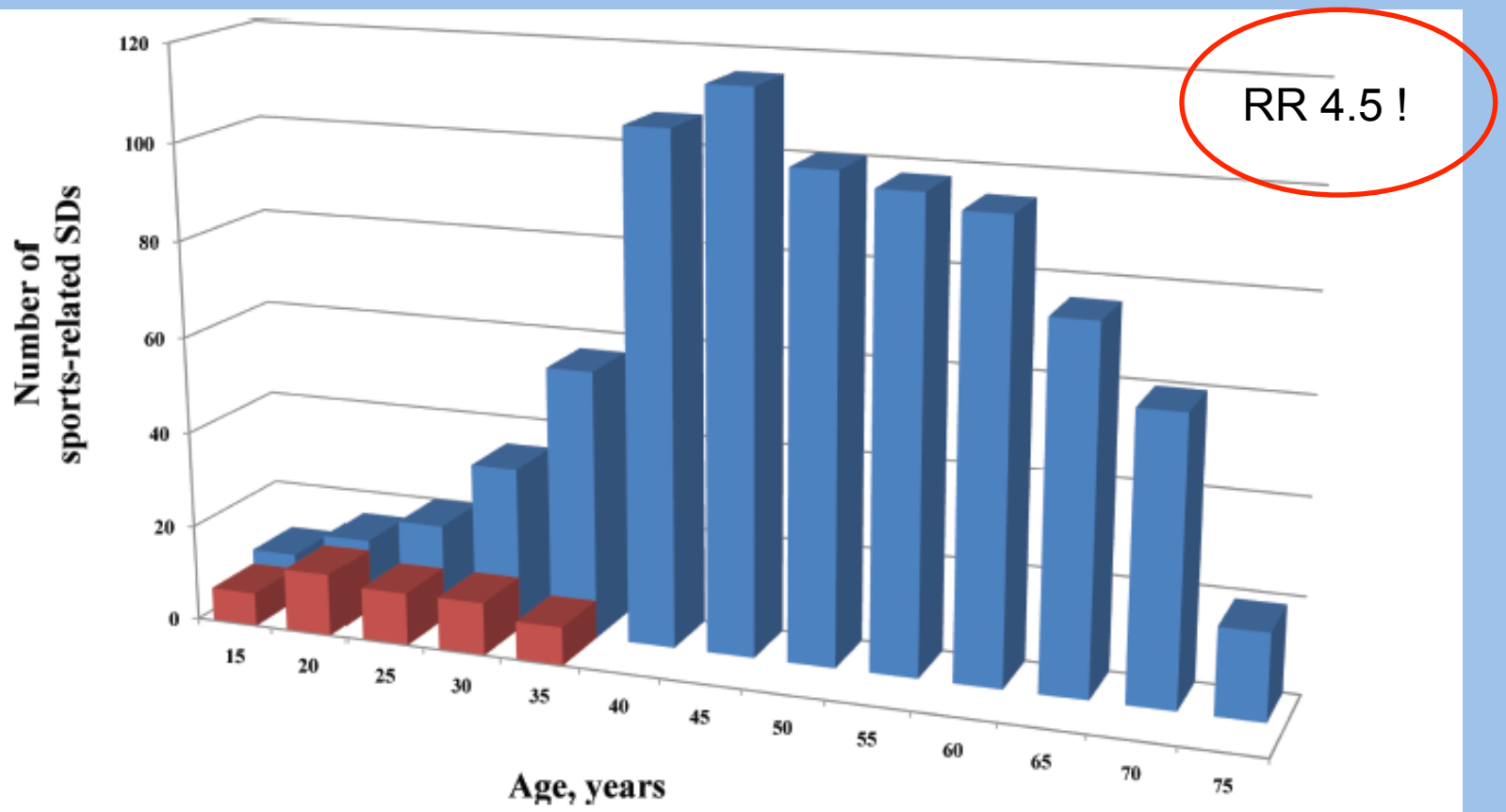
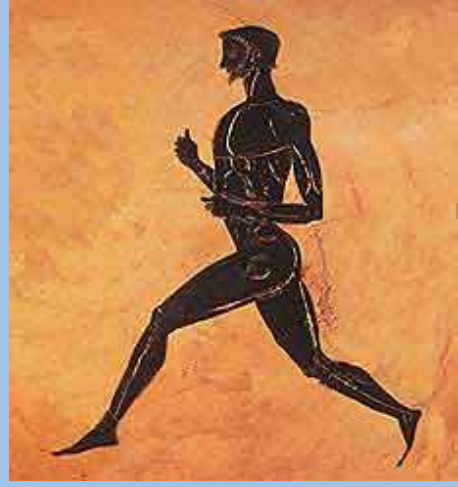


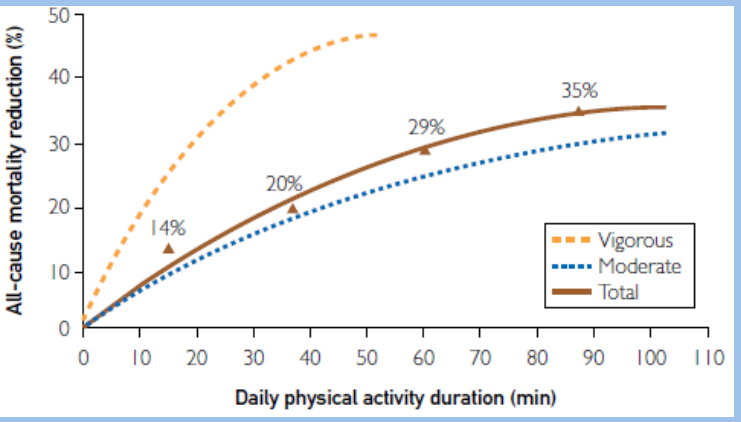
Figure 2. Distribution by age of sports-related sudden deaths (SDs) in the overall population (blue) and among young competitive athletes (red).

Marijon et al. Circulation 2011;124:672-81

Le paradoxe de l'effort sportif



- **Bénéfices**



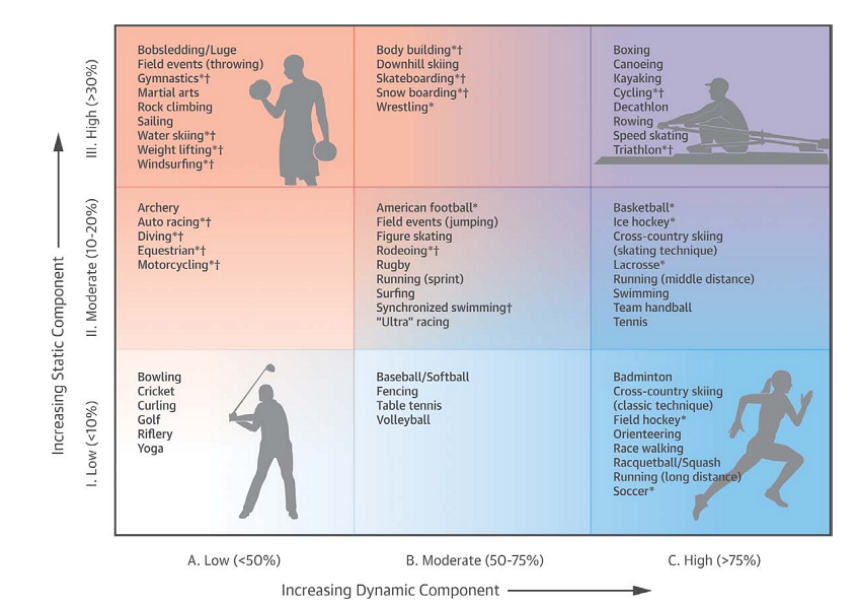
Wen et al. Lancet 2011;378:1244-53.

Corrado et al. Eur Heart J 2011;32:934-44.

Maron BJ. NEJM 2000; 1409-11

- **Risque accru de mort subite**
  - **1000-1500 MS /an en France**

Study population	Ref.	Study design and reporting system	Incidence (person-years)
US Military (age 18–35)	Eckart *et al.*	Retrospective, mandatory	1:9000
Italian Athletes (age 12–35)	Corrado *et al.*	Prospective, mandatory	1:25,000
US Adolescents (age 12–19)	Atkins *et al.*	Prospective	1:27,000
US Children (age 10–14)	Chugh *et al.*	Prospective, EMS/ Hospitals	1:58,000
US Athletes (age 12–35)	Maron *et al.*	Retrospective, public media reports	1:160,000



Mitchell J. J Am Coll Cardiol 1994;24:864–66.

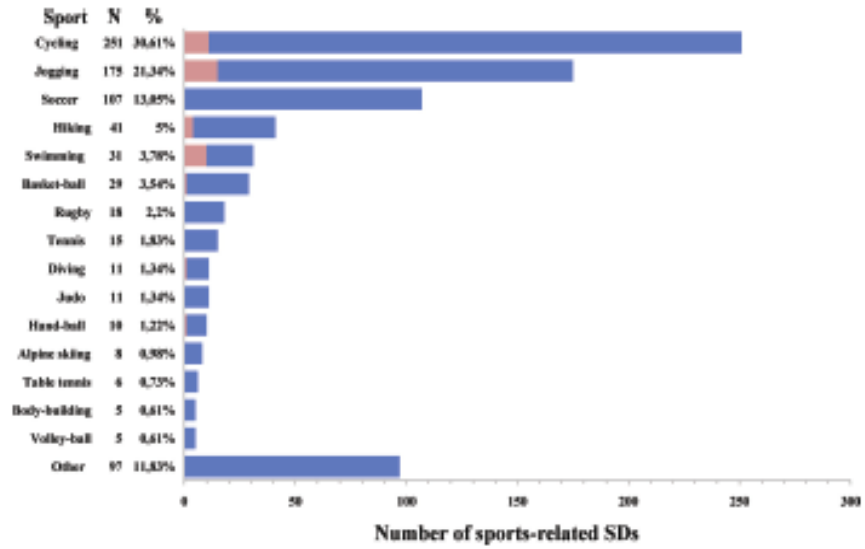
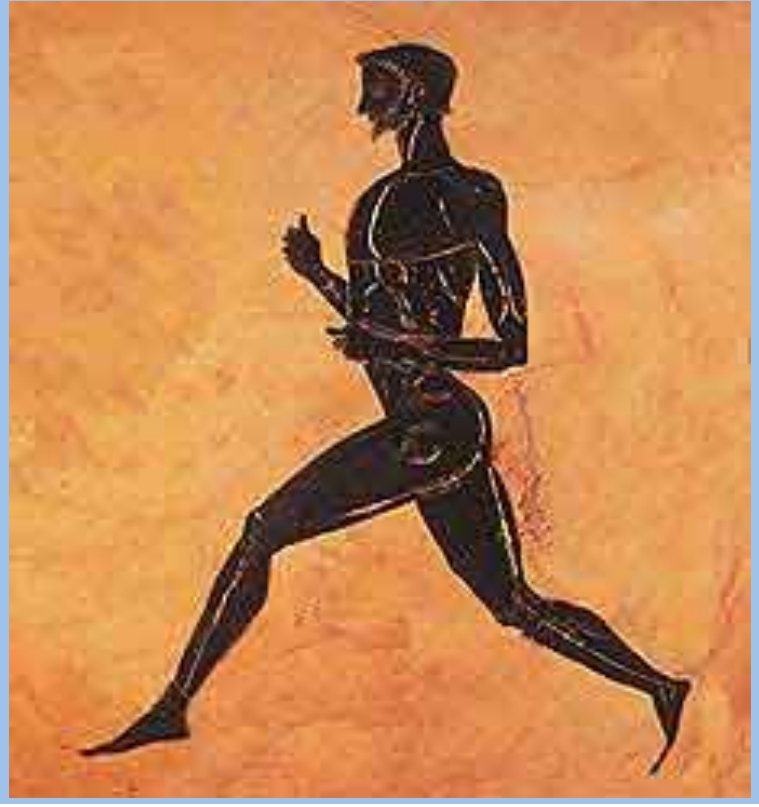


Figure 3. Sports engaged in at the time of sudden death (SD) in 820 sports participants. Shaded proportions of the bars represent the female ratio.

Marijon et al. Circulation 2011; 124:672-81 Marijon et al. Eur Heart J 2013;34:3632-40



MS:
- 80% pendant l'effort
- homme + exercice intense
- survie : 15 to 30%

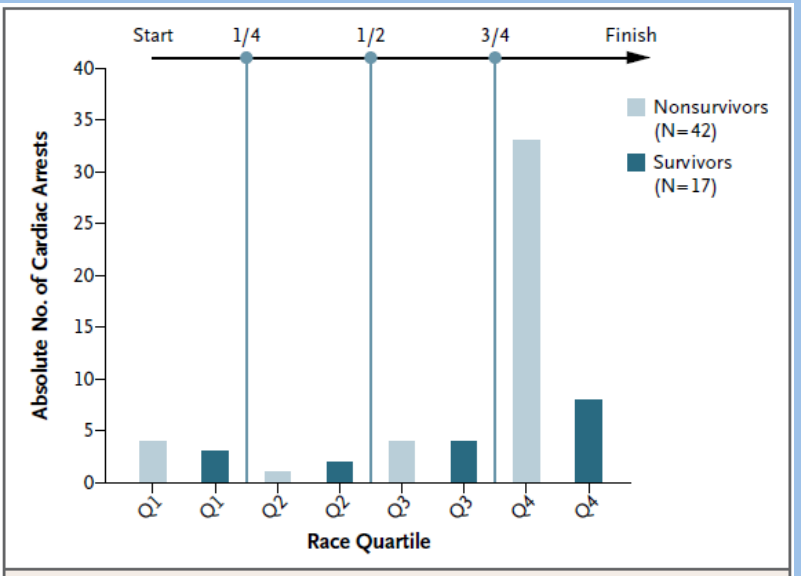
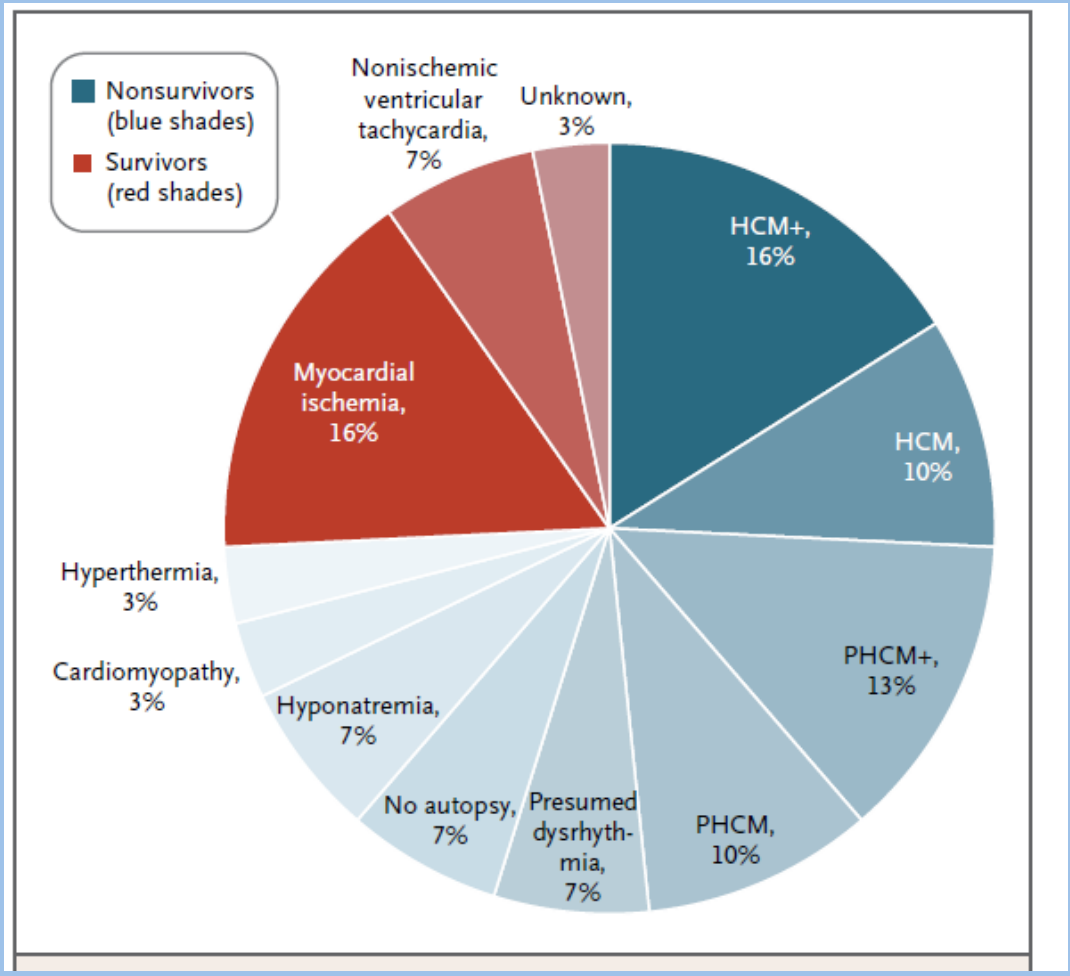
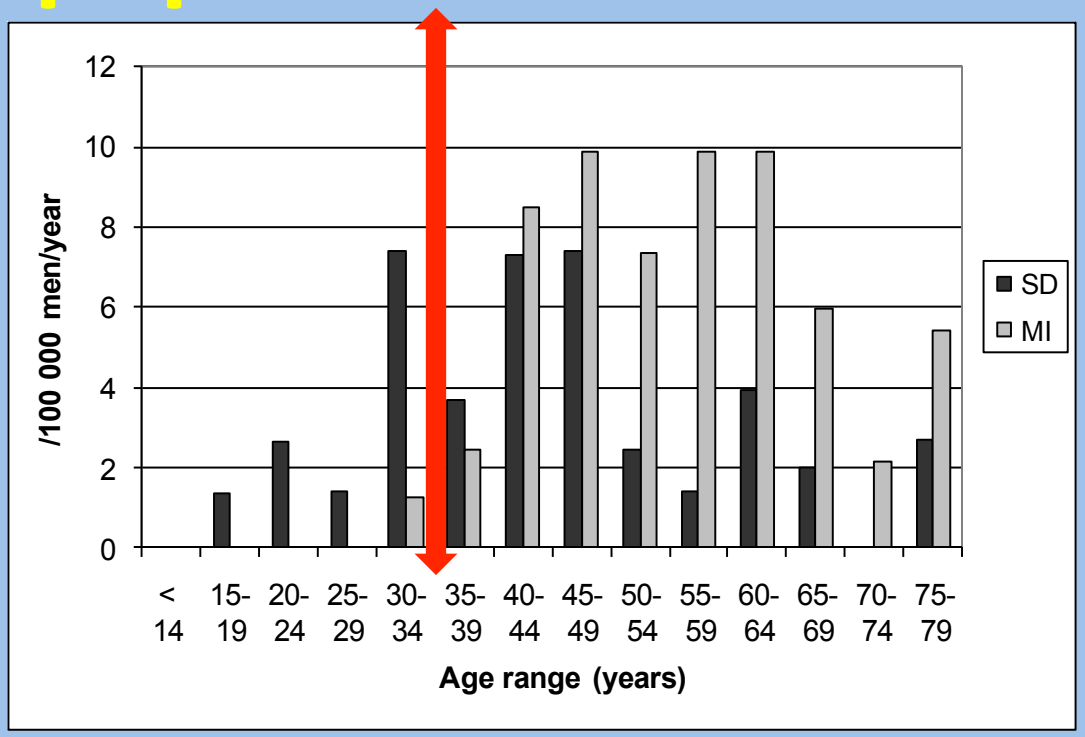


Figure 1. Location of Cardiac Arrest According to Race Quartile.



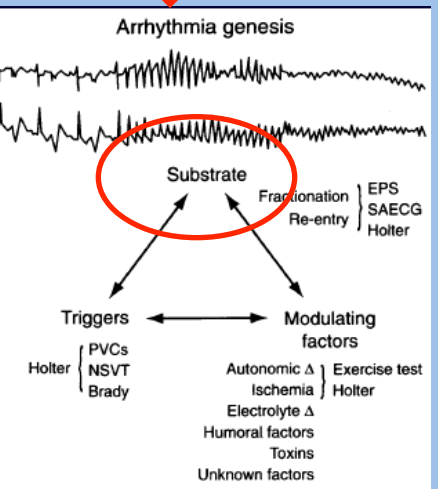
Kim et al. NEJM 2012;366:130-40.

2 populations différentes



< 35

0.5 to 2.5 / 100 000



> 35

1 to 4 / 100 000

Chevalier et al. Eur J Cardiovasc Prev Rehabil 2009;16:365-70.

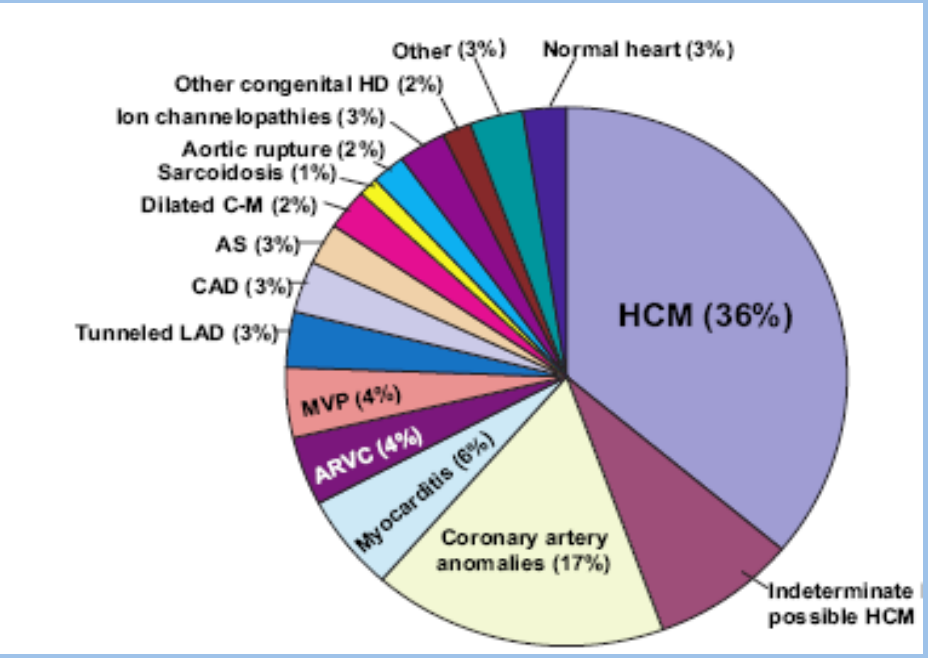
Corrado et al. Eur Heart J 2011;32:934-44.

Cardiopathies à risque chez le jeune athlète

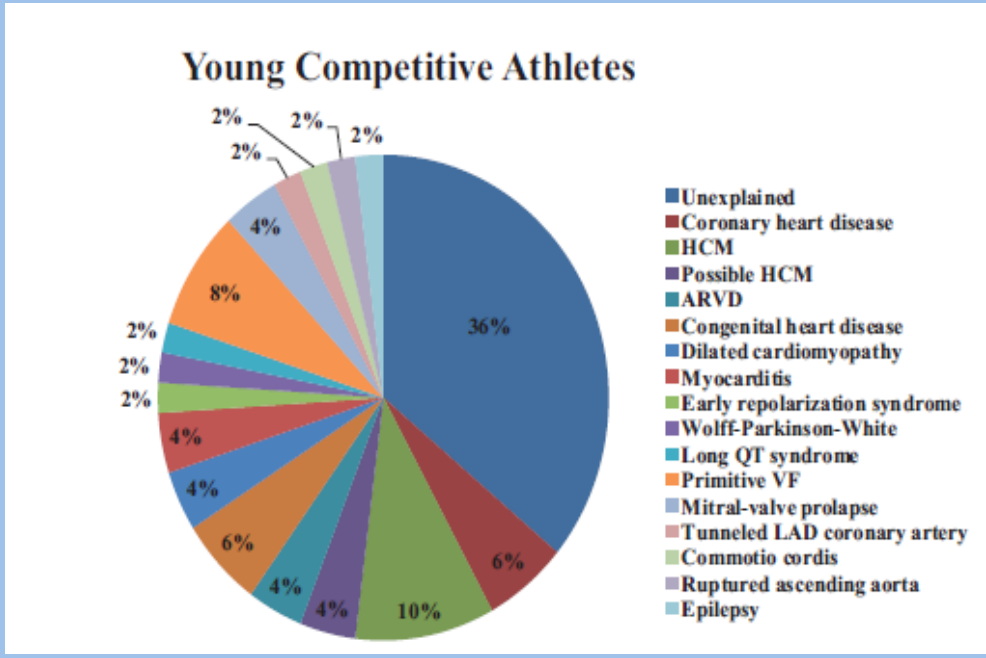
- Cardiopathie structurelle
  - congénitale
    - CMH: 10 à 40%
    - Anomalie de naissance coronaire: 10 à 30%
    - DAVD : 4 to 25%
    - Cardiopathie congénitale<5%
    - Valvulopathie (MVP) <5%
    - Marfan <5%
  - acquise
    - Myocardite aigue: 10 to 30%
    - Maladie coronaire prématurée: 5 to 35%
    - Sarcoïdose…
- Non structural cardiac disease
  - Inexpliquée: 5 to 40%
  - canalopathies
    - anomalies du QT (long/court),
    - TV polymorphes cathécholergiques,
    - Brugada
  - WPW
  - Repolarisation précoce

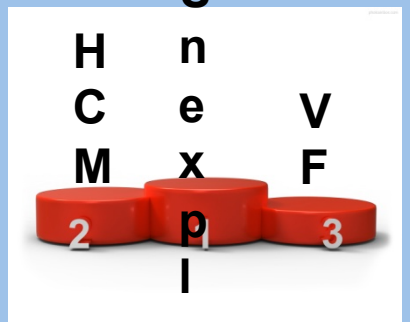
Causes de mort subite chez le jeune athlète (compétition)

Maron et al. Circulation 2006;114:1633-44



Marijon et al. Circulation 2011;124:672-81

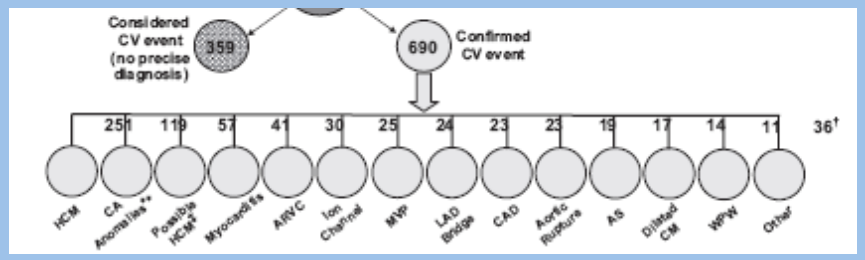


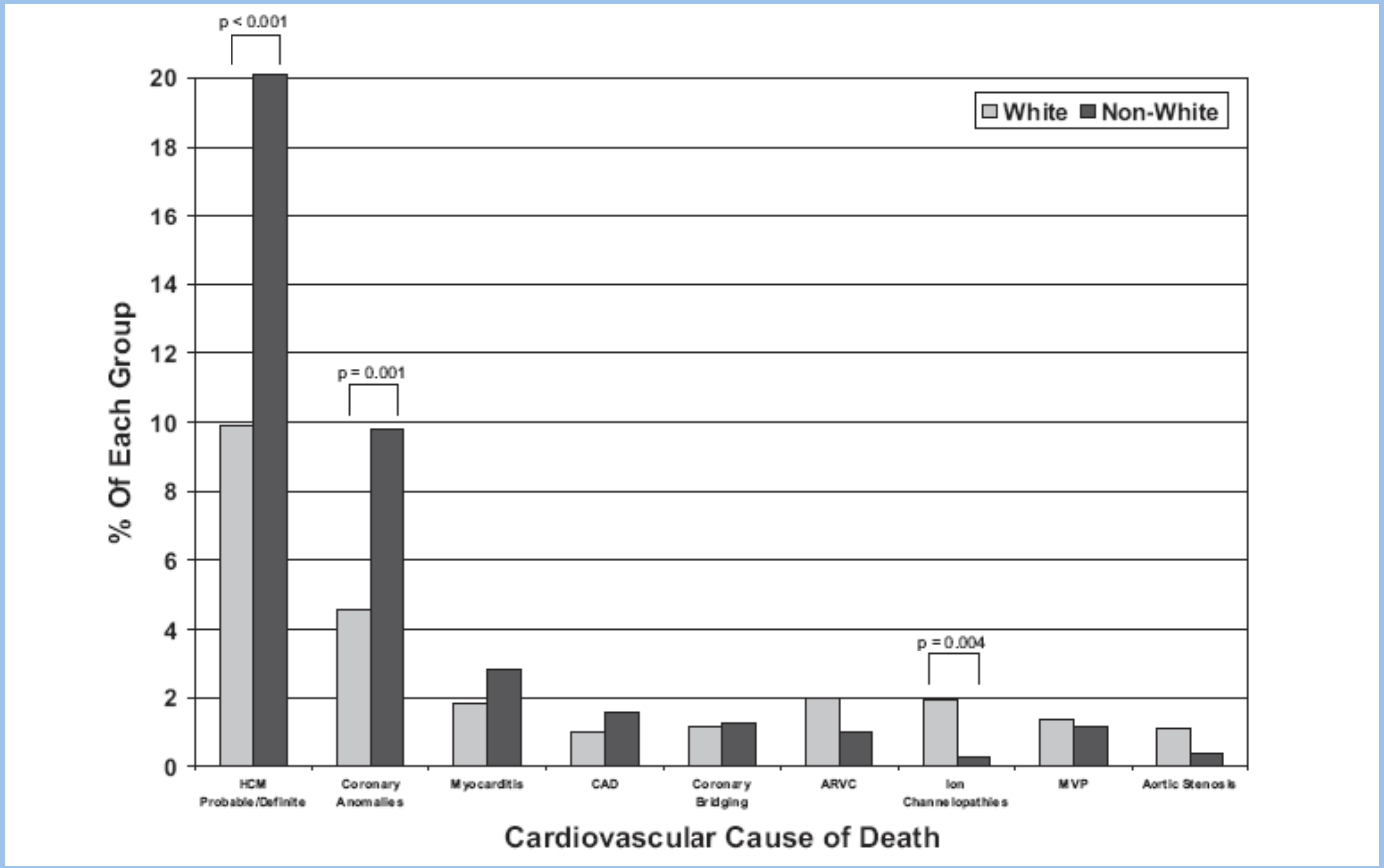


MS cardiaque à l' effort

Eckart Ann Intern Med. 2004	US (military)	108 SCD [18-35y]	**Unexplained (35%), coronary abn. (30%), Myocarditis (10%), HCM (6%)**
Sheppard M Br J sports Med 2012	UK	118 SCD [< 35Y]	**HCM / LVH (41%), ARVC (13%)**
Tabib A Eur Heart J 1999	France	27 SCD [<30y]	**HCM (29%), (ARVC 26%)**
Corrado NEJM 1998	Italy	49 SCD [11-35y]	**ARVC (23%) CAD 18% coronary abn (12%)**
Soldberg Eur J Cardiovasc Prev Rehabil. 2010	Norway	23 SCD [17-34y]	**M infarction (40%), Myocarditis (20%),**
Holst Heart Rhythm. 2010	Denmark	15 SCD [12-35y]	**ARVC (25%), unexplained (25%), CAD (13%)**
Suárez-Mier Rev Esp Cardiol. 2002.	Spain	61 SCD [11-65y]	**CAD (45%), unexplained (16%), ARVC (16%), HCM(10%)**
Raschka C Med Klin 1999.	Germany, Austria	47 SCD [<35y]	**CAD (36%), Myocarditis (30%)**

Différences ethniques





BJ Maron. Circulation 2009;119:1085-92.

Et les jeunes athlètes féminines?

- **Sex Ratio de MS cardiaque à l'effort:**
  - **1/5 à 1/30!**
  - **Moins de coronaropathie prématurée**

- **Cœur normal (MS rythmique…) > 50%**
  - **Afro-Americaines > recrues « blanches » (RR 10.2).**

- **Meilleur taux de survie à l'admission H (46.5 vs 30%)**

Kim et al. NEJM 2012;366:130-40.
Eckart RE. Am J Cardiol 2006; 97:1756-8
Marijon E et al. Circ Arrhythm Electrophysiol. 2013;6:1185-1191.

Visite de non contre-indication

Avez-vous déjà un dossier médical dans une autre structure, si oui laquelle :
Avez-vous eu connaissance dans votre famille des évènements suivants :
- Accident ou maladie cardiaque ou vasculaire survenue avant l'âge de 50 ans Oui Non
- Mort subite survenue avant 50 ans (y compris mort subite du nourrisson) Oui Non

Avez-vous déjà ressenti pendant ou après un effort les symptômes suivants :
- Malaise ou perte de connaissance Oui Non
- Douleur thoracique Oui Non
- Palpitations (cœur irrégulier) Oui Non
- Fatigue ou essoufflement inhabituel Oui Non

Avez-vous
- Une maladie cardiaque Oui Non
- Une maladie des vaisseaux Oui Non
- Été opéré du coeur ou des vaisseaux Oui Non
- Un souffle cardiaque ou un trouble du rythme connu Oui Non
- Une hypertension artérielle Oui Non
- Un diabète Oui Non
- Un cholestérol élevé Oui Non

Fumez vous
- Depuis combien de temps ?

- Suivi un traitement régulier ces deux dernières années (médicaments, compléments alimentaires ou autres) Oui Non
- Eu une infection sérieuse dans le mois précédent Oui Non
- Avez-vous déjà eu :
  - un électrocardiogramme Oui Non
  - un échocardiogramme Oui Non
  - une épreuve d'effort maximale Oui Non

- examen physique
  - Souffle
  - Pouls
  - Marfan? (basket, volley…)
  - FC et PA

- ECG (French (2009) – European guidelines)
  - > 12 ans: tous les 3 ans
  - >20 ans: tous les 5 ans (>>>35Y)

- Echocardiographie: athlètes elite.

Carre F, Brion R, Douard H et al. SFC guidelines 2009

Visite ne non contre-indication

- **…du jeune athlète**
  - **Asymptomatique**
  - **Examen normal**
  - **Non limité pour la performance**
  - **> 50% ne déclareront pas (sport ht niveau – jeunes - femmes)**

—>>> ECG?

Une visite cardiovasculaire sans ECG de repos ne détecte presque aucune maladie cardiovasculaire à risque lors de la pratique du sport

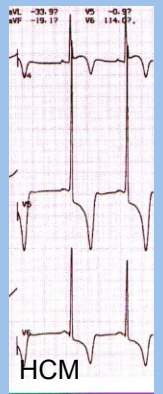
Corrado et al. Eur Heart J 2005; 26:516-24.

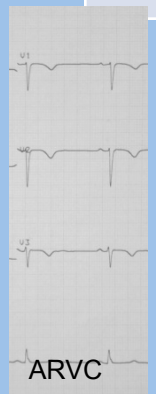
Chevalier L et al. Arch Mal Cœur Vaiss 2005;98:109-14

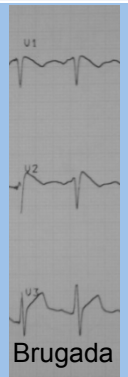
DETECTABLE

Cardiac disease	ECG value	Sensitivity (%)	Specificity
HCM	+++	73-98	low
ARVC	+++	25-94	low
Long QT	+++	83-100	moderate
WPW	+++	-	high
CMD	+	high?	low
Myocarditis	++	10-54	low
Brugada	++	>20	high
MVP	+/-	66	low
CAD	+/-	low	low
Coronary abn.	-	low	low
Marfan	-	low	low

NON DETECTABLE







…60% des causes de MS

Screening of young athletes for Cardiovascular diseases

(Center for Sports Medicine, Padua 1979-2004)

Athletes screened
42,386

Positive findings
3,914 (9%)

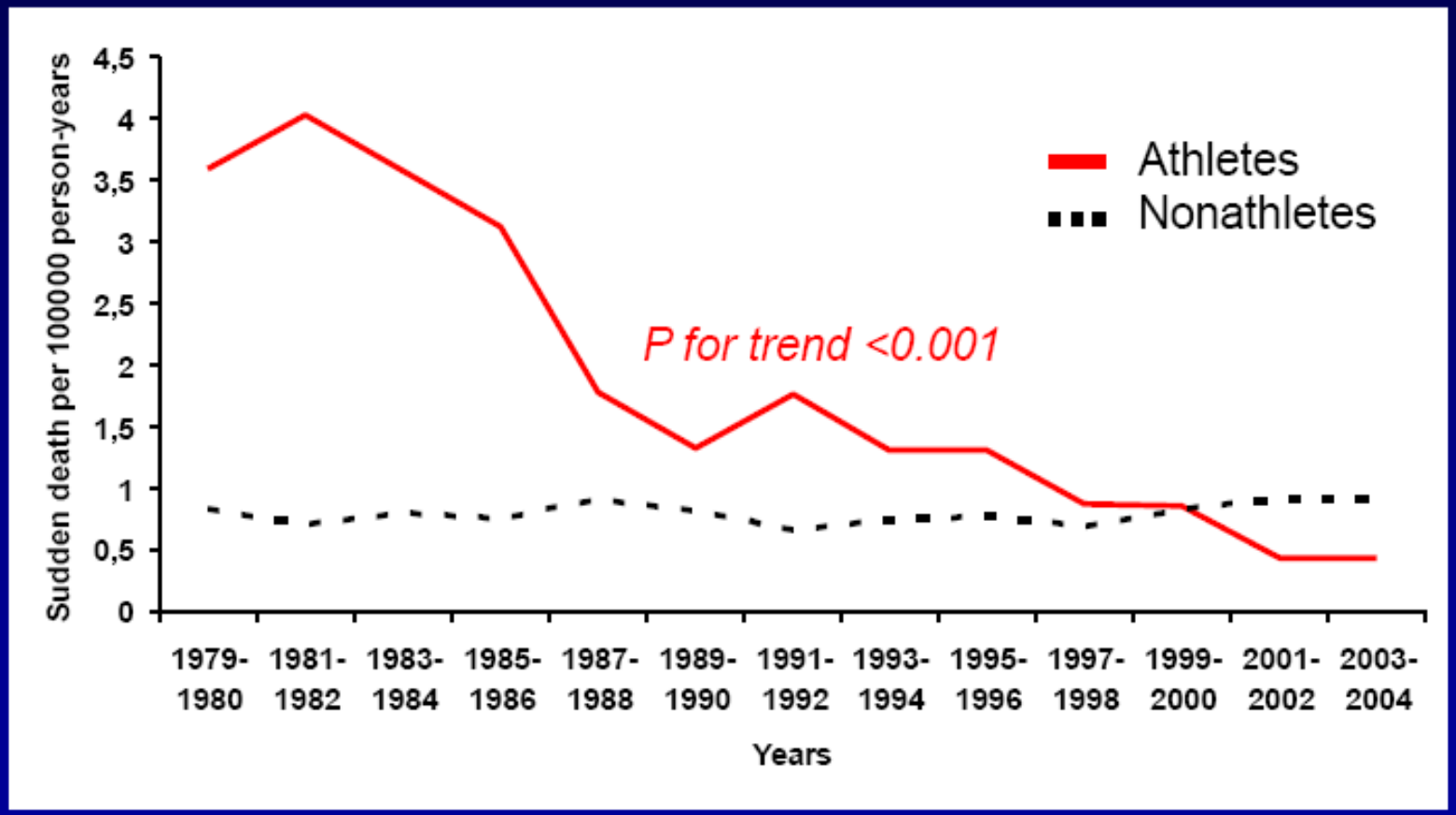
Heart diseases
879 (2%)

Potentially lethal heart diseases
91 (0.2%)

False positive ≈ 7%

Corrado et al JAMA 2006; 296: 1593-1601

Annual Incidence Rates of Sudden Cardiovascular Death in Screened Competitive Athletes and Unscreened Nonathletes Aged 12 to 35 Years in the Veneto Region of Italy (1979-2004)



Corrado et al JAMA 2006;296:1593-1601

The NEW ENGLAND JOURNAL of MEDICINE

CLINICAL DECISIONS
INTERACTIVE AT NEJM.ORG

Cardiac Screening before Participation in Sports

N ENGL J MED 369;21 NEJM.ORG NOVEMBER 21, 2013

Sanjay Sharma,

preparticipation cardiac screening should be required for all young athletes before they engage in organized competitive sports.

Domenico Corrado

On the basis of the available data, the students in the vignette should undergo a preparticipation cardiovascular evaluation including ECG — the only screening strategy that has been proved to be effective.

N.A. Mark Estes III,

versial. In addition, the premise that restriction of participation in sports improves outcomes remains unproven.

gram that included ECG.[6] The Italian, Israeli, and U.S. reports share the fundamental limitations of being retrospective, nonrandomized, observational, registry studies.[4,6-8]

Advancing potentially costly and harmful athletic screening without proven benefit would be premature.

Victoria L. Vetter,

family history.[12] However, the history and physical examination will result in fewer false positive findings than will ECG. Furthermore, a

Pre participation screening

Table 2. Preparticipation Athletic Screening and Athletic Restriction in Italy, the United States, and Israel

Country	Years	Screening	Initial	Examiners	Sudden Death
Italy[6]	1981–2008	Mandatory	History, PE, ECG, ETT	Sports medicine MD	Decrease
United States[30]	1985–2006	Recommended	History, PE	MD and non-MD	No decrease
Israel[26]	1985 to 2009	Mandatory	History, PE, ECG, ETT	Certified MD	No decrease

PE indicates physical examination; ETT, exercise tolerance test.

Link M, Estes N. Circulation 2012;125:2511-2516

- **Maron. Circulation 2009;119:1085-92**
  - 690 MS athlètes < 39 ans (1980 – 2006)
  - 70 % causes IDENTIFIABLES par PP screening…

- **Maron. Heart Rhythm. 2013;10:374-7.**
  - 2 M high school (12-18Y) athletes (1986-2011), 13 MS (autopsies).
  - Risque de MS cardiaque: 1:150,000 participants/an
  - 30% détectable (dont ECG)…

- **Rapport cout-efficacité:**

ECG: 43000 $ / année de vie sauvée

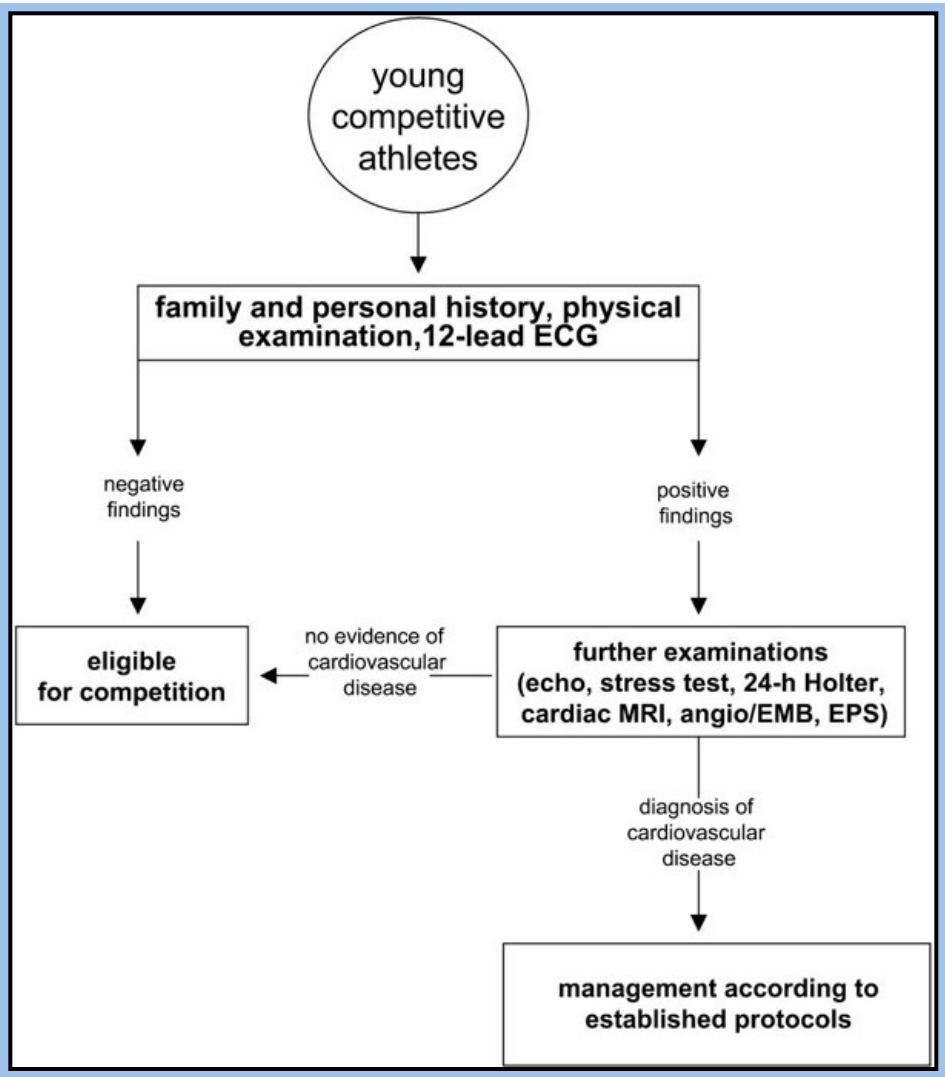
(= ICD ou accès public à la défibrillation).

- ETT: 200 000 $

MT Wheeler. Ann Intern Med 2010;152:276-86.

Recommendations for competitive sports participation in athletes with cardiovascular disease

A consensus document from the Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology



European Heart Journal (2005) 26, 1422–1445

Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 2: Preparticipation Screening for Cardiovascular Disease in Competitive Athletes

A Scientific Statement From the American Heart Association and American College of Cardiology

15 Task forces….

1. Classification des sports
2. Pre participation screening
3. Cardiomyopathies
4. CP congénitales
5. Valves
6. HTA
7. Marfan aorte
8. Maladie coronaire
9. Arythmies
10. Canalopathies
11. Dopage
12. Prévention
13. Comotio cordis
14. Drépanocytose
15. Aspects médicaux légaux

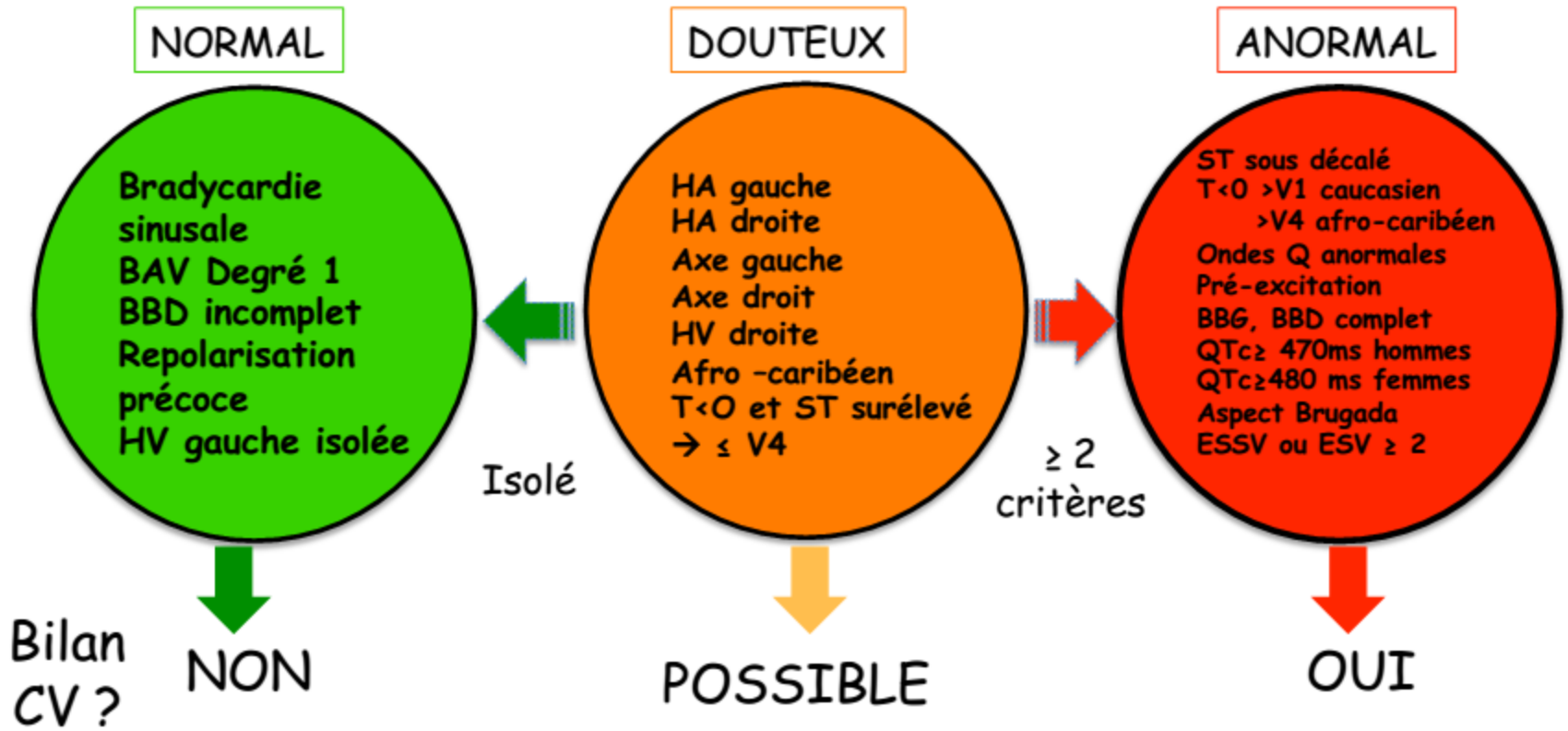
Circulation. 2015;132:e267–e272

Screening with 12-lead ECGs (or echocardiograms) in association with comprehensive history-taking and physical examination to identify or raise suspicion of genetic/congenital and other cardiovascular abnormalities may be considered in relatively small cohorts of young healthy people 12 to 25 years of age, not necessarily limited to competitive athletes (eg, in high schools, colleges/universities or local communities). Close physician involvement and sufficient quality control is mandatory. If undertaken, such initiatives should recognize the known and anticipated limitations of the 12-lead ECG as a population screening test, including the expected frequency of false-positive and false-negative test results, as well as the cost required to support these initiatives over time *(Class IIb; Level of Evidence C)*.

Mandatory and universal mass screening with 12-lead ECGs in large general populations of young healthy people 12 to 25 years of age (including on a national basis in the United States) to identify genetic/congenital and other cardiovascular abnormalities is not recommended for athletes and non-athletes alike *(Class III, no evidence of benefit; Level of Evidence C)*.

Circulation. 2015;132:e267–e272.

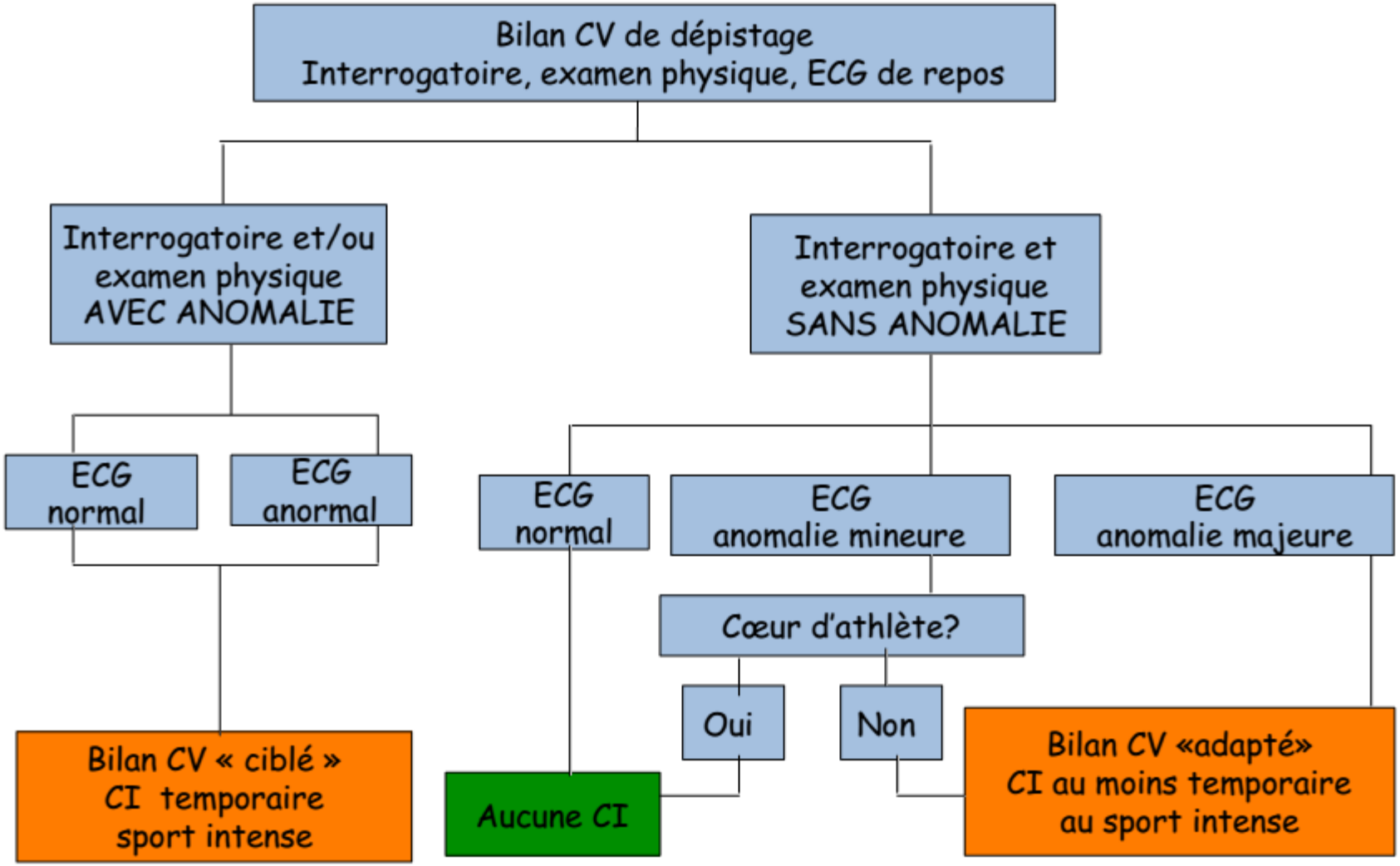
ECG

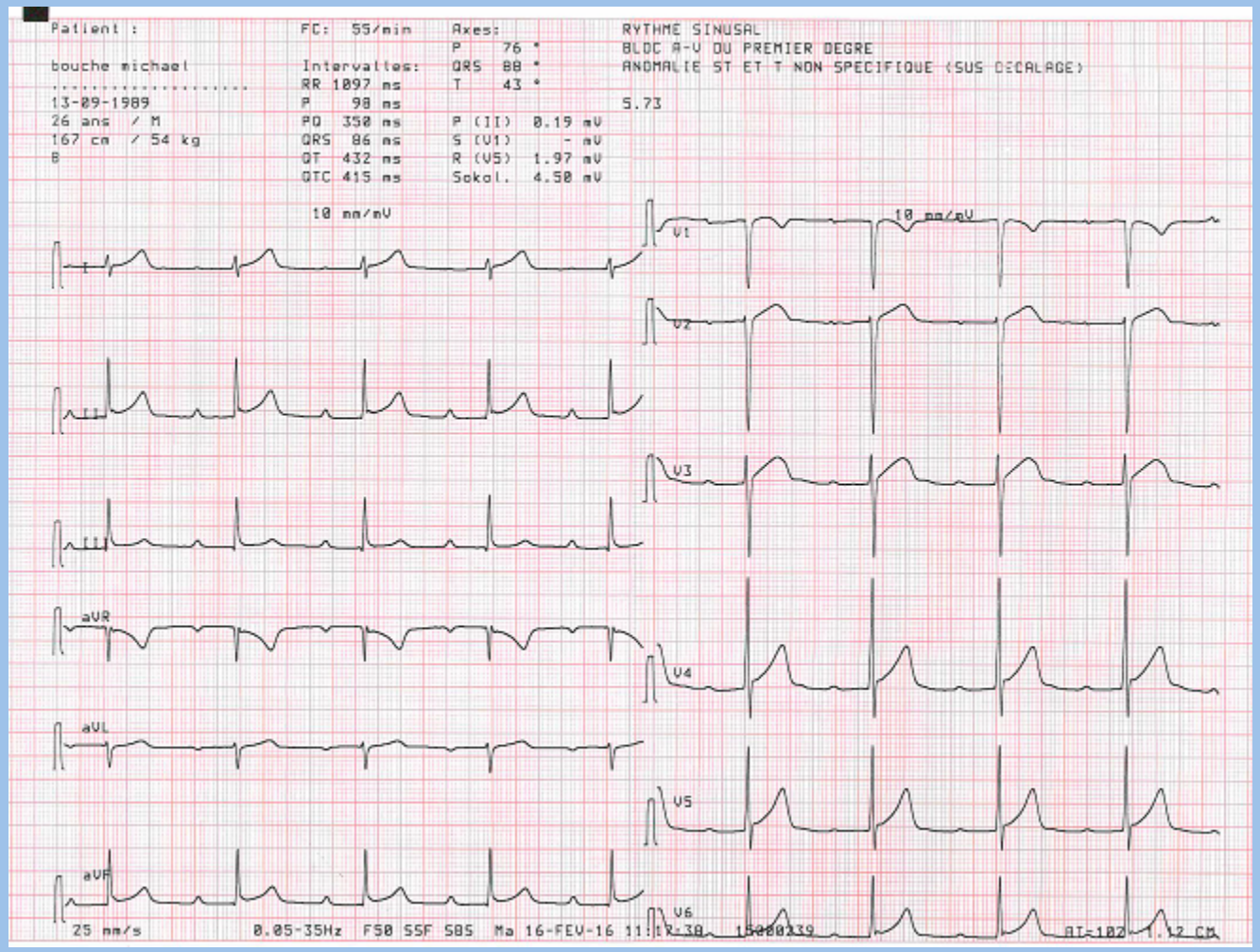


Sheikh N et al.
Circulation 2014;129:1637-49

HA hypertrophie atriale
BAV bloc atrio-ventriculaire
HV hypertrophie ventriculaire
ST segment ST

BBD,BBG bloc de branche droit, gauche
QT durée intervalle QT
ESSV, ESV extrasystole supra-ventriculaire, ventriculaire


Bilan CV de dépistage
Interrogatoire, examen physique, ECG de repos
Interrogatoire et/ou examen physique AVEC ANOMALIE
Interrogatoire et examen physique SANS ANOMALIE
ECG normal
ECG anormal
ECG normal
ECG anomalie mineure
ECG anomalie majeure
Cœur d'athlète?
Oui
Non
Bilan CV « ciblé » CI temporaire sport intense
Aucune CI
Bilan CV «adapté» CI au moins temporaire au sport intense



26 ans franco-togolais - 2h 30 de course/jour – championnat de France de Cross

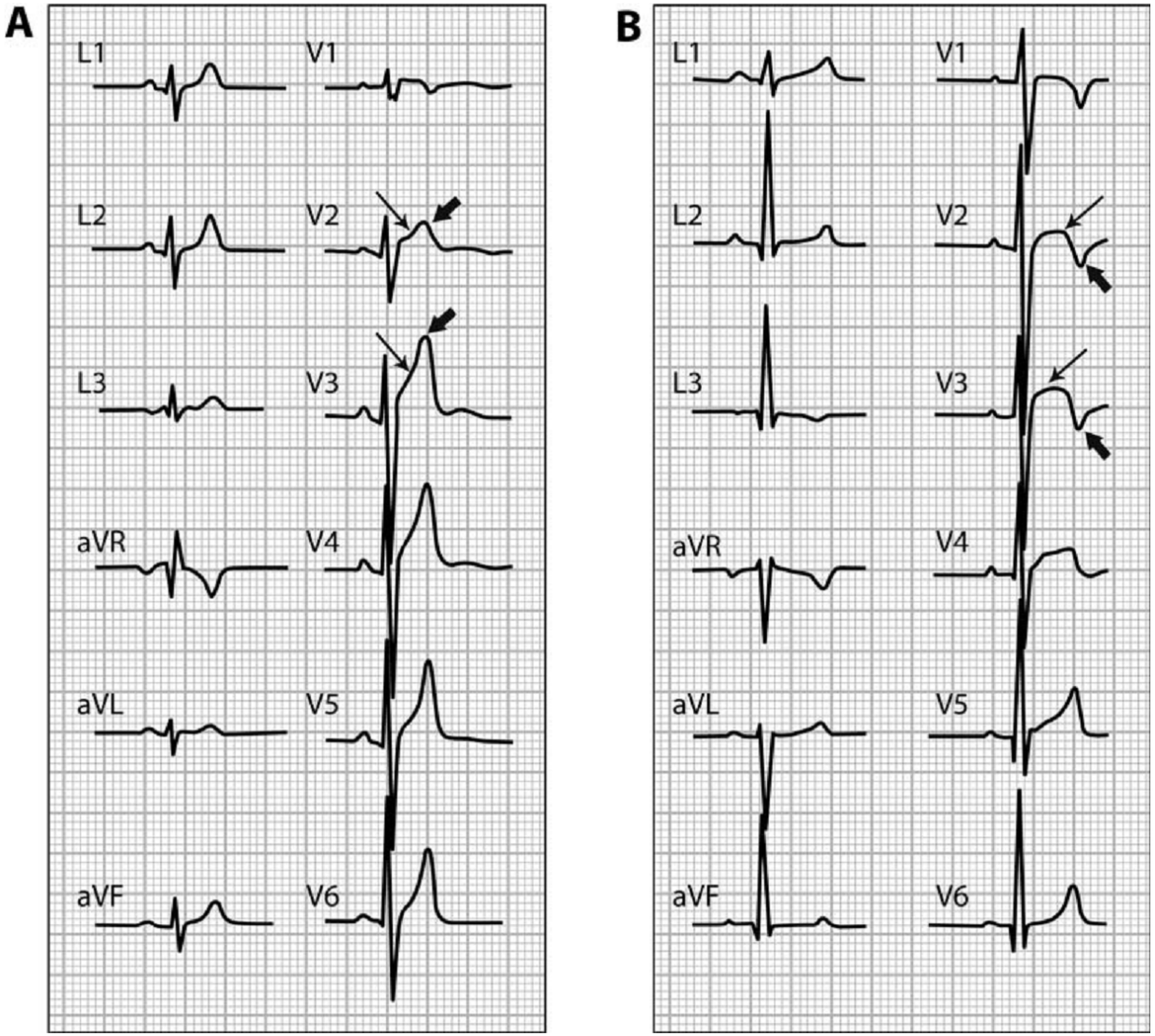
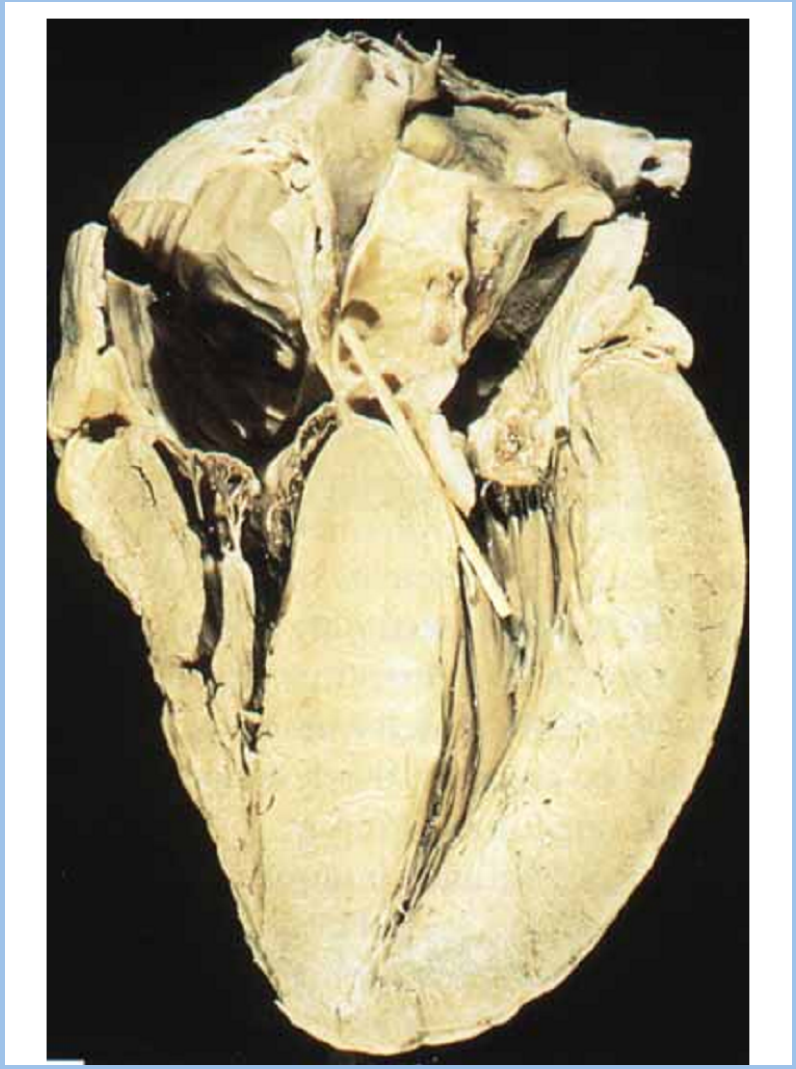
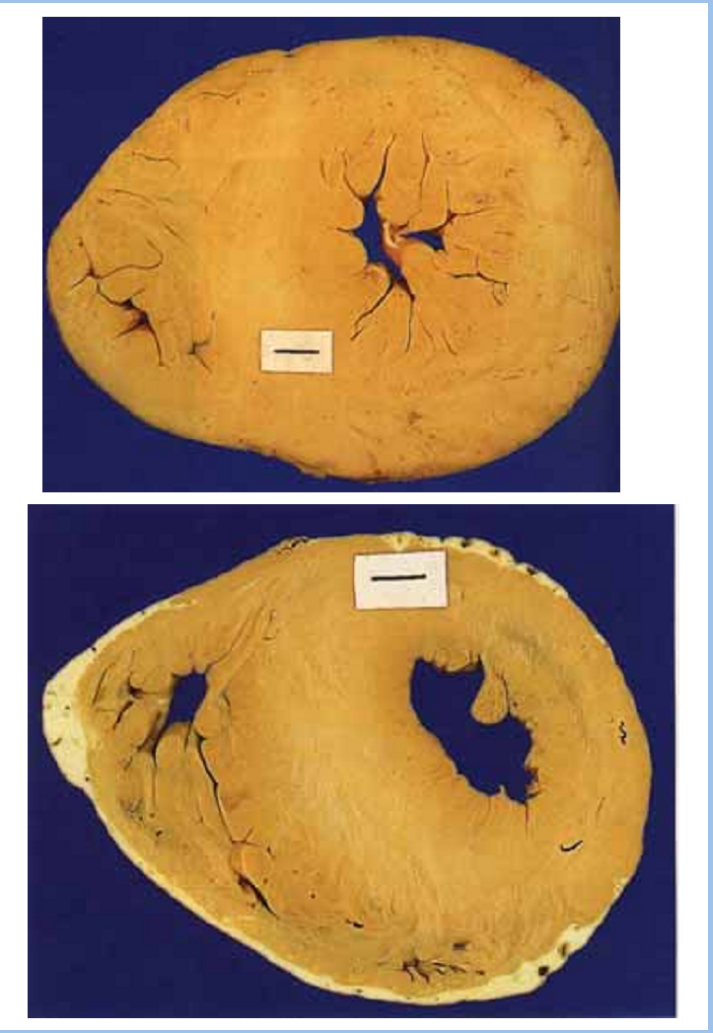


Figure 2 Different patterns of precordial early repolarization in two healthy athletes. (*A*) ST-segment elevation with upward concavity (arrows), followed by a positive T-wave (arrowheads). (*B*) ST-segment elevation with upward convexity (arrows), followed by a negative T-wave (arrowheads).

European Heart Journal
doi:10.1093/eurheartj/ehp473

CMH





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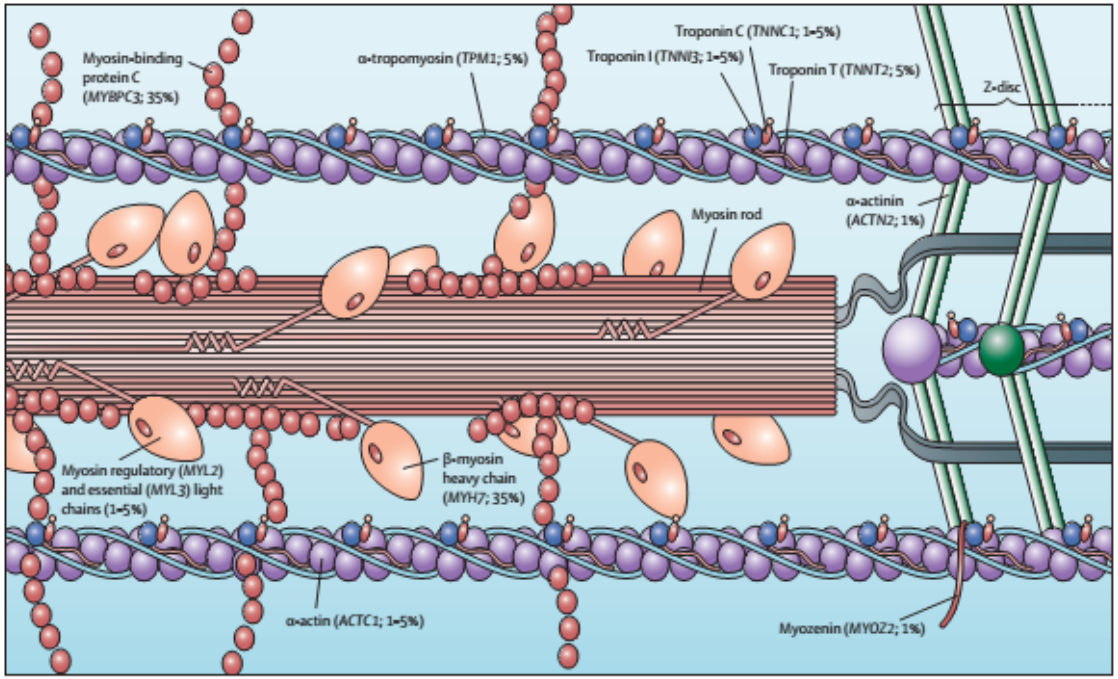
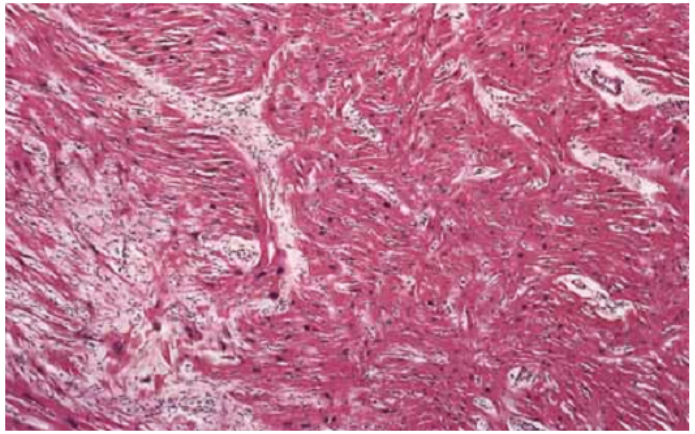
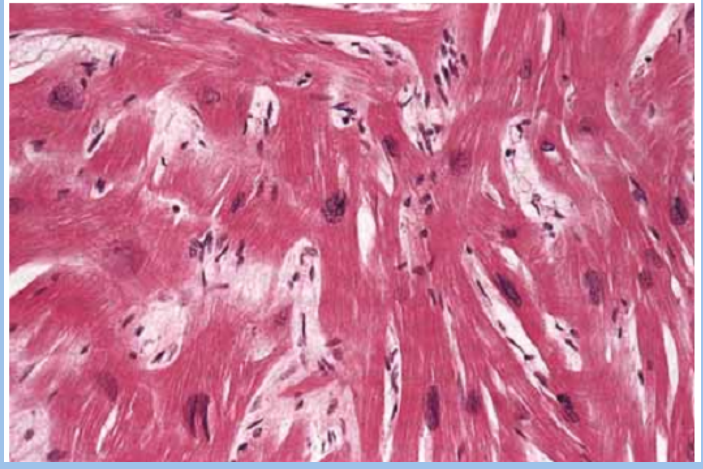


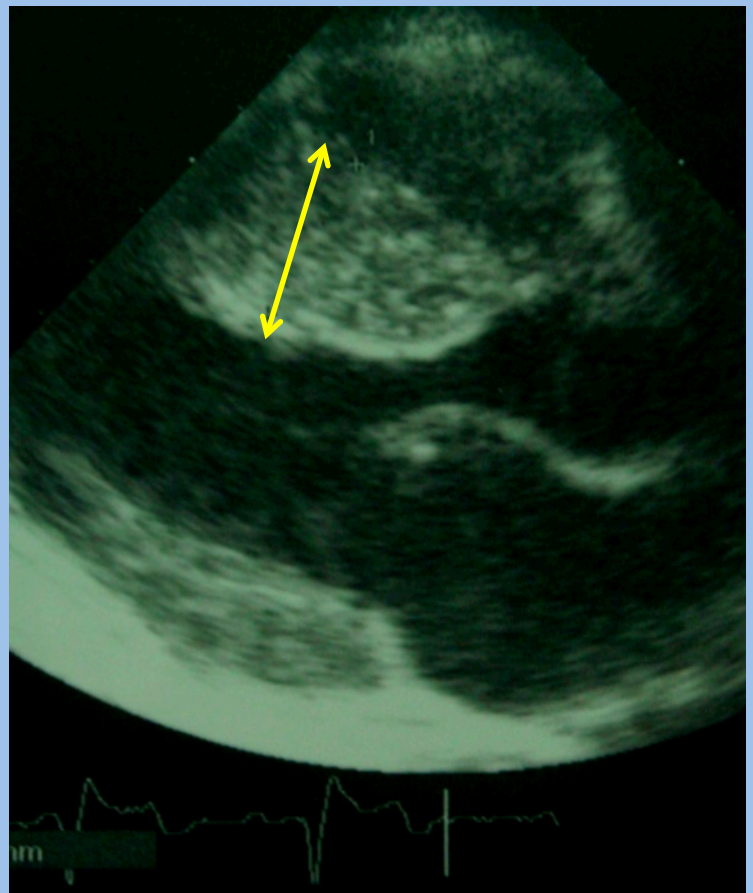
Figure 1: **Locations of genes within the cardiac sarcomere known to cause hypertrophic cardiomyopathy**
Prevalence of every gene (derived from data of unrelated hypertrophic cardiomyopathy probands with positive genotyping) is shown in parentheses.

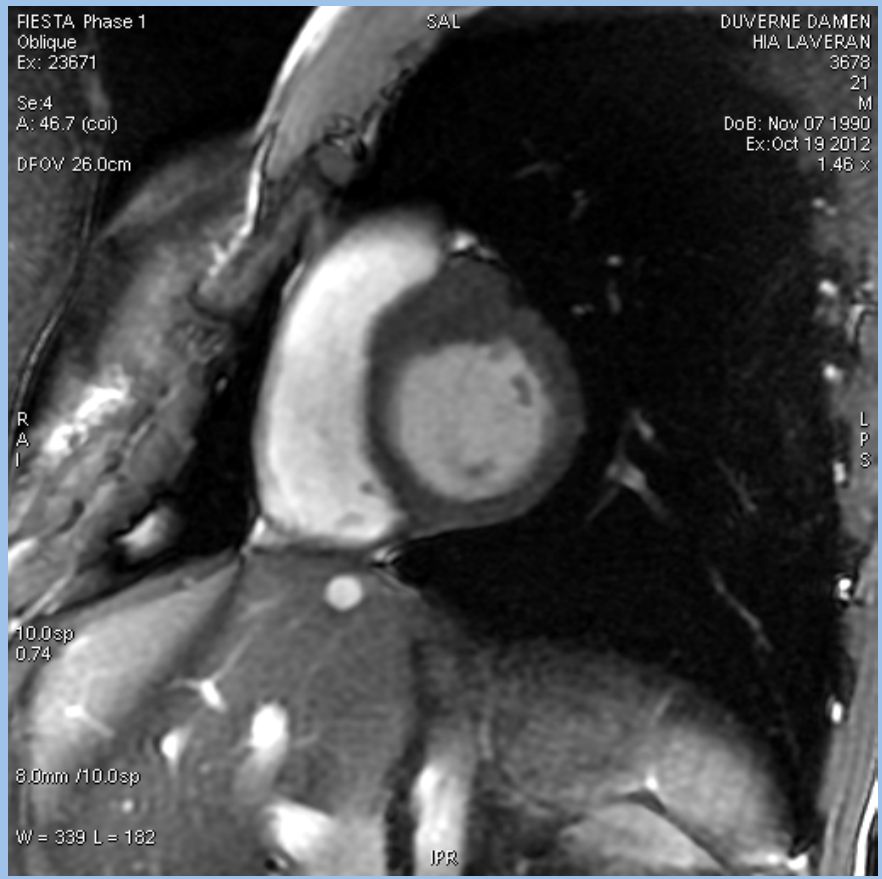




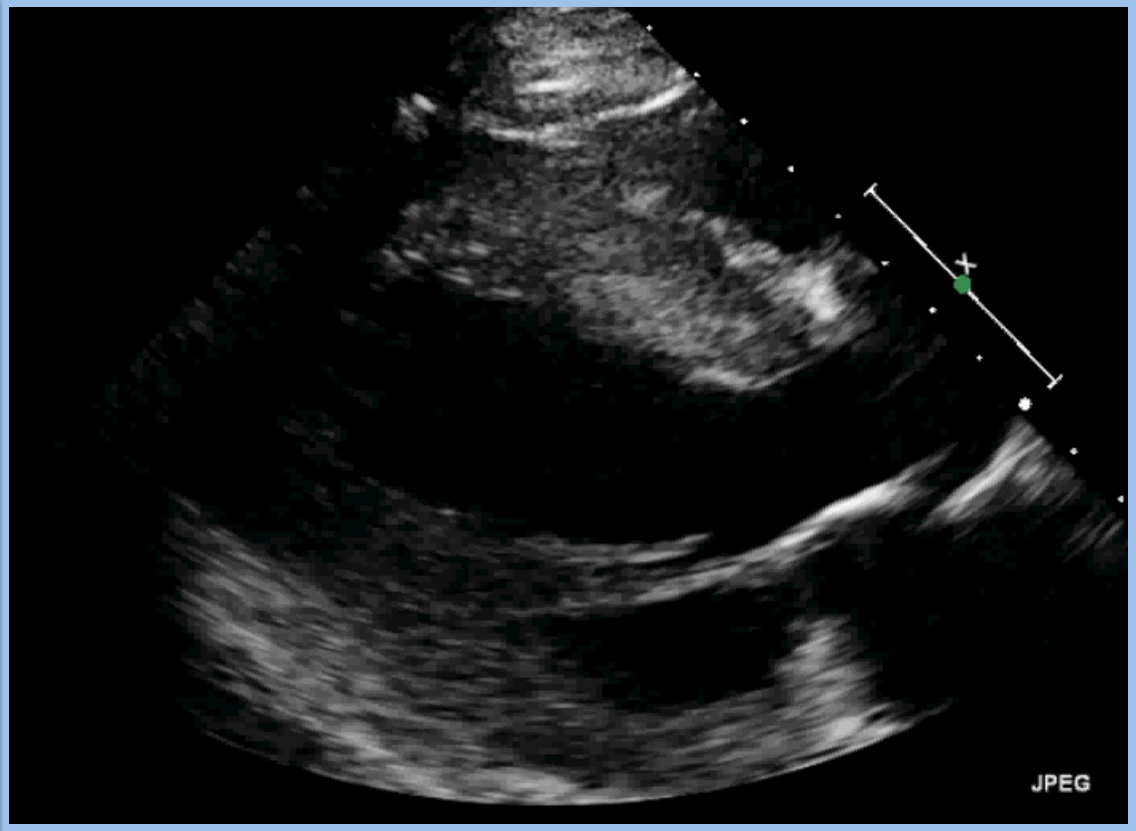
CMH

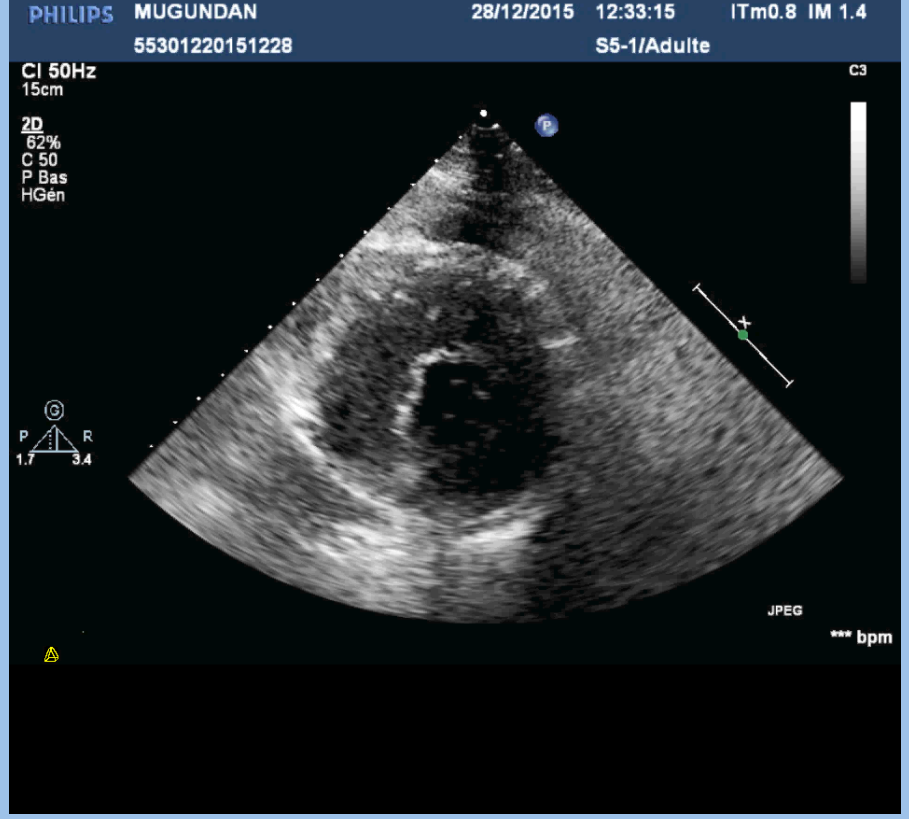
- 1/500
- Evaluation du risque
- Uncertain/ cœur d'athlète

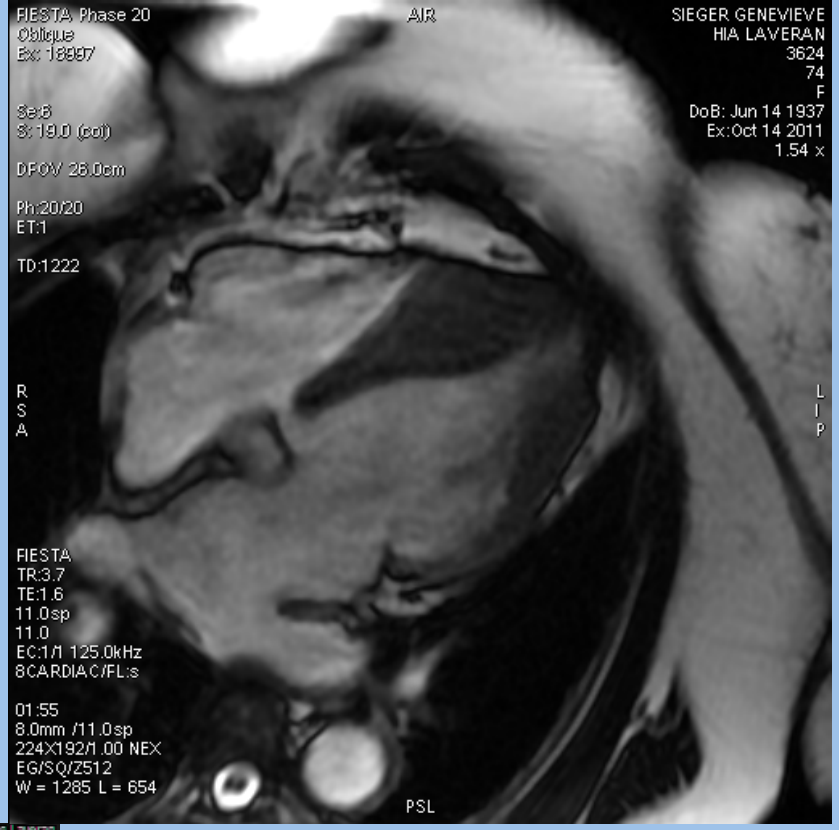


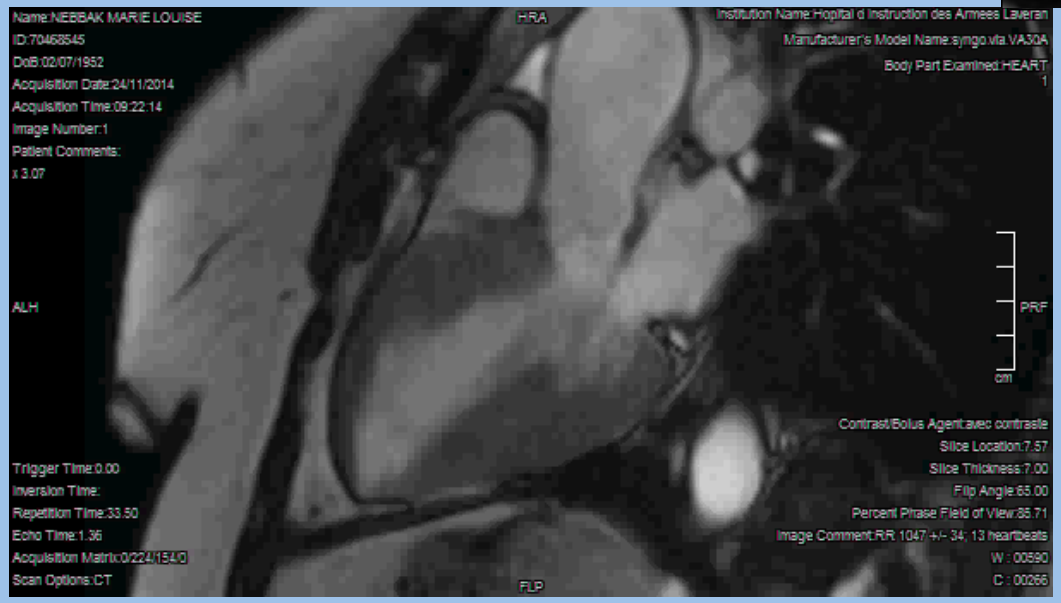


V1
V2
V3
V4
V5
aVF
V6


JPEG

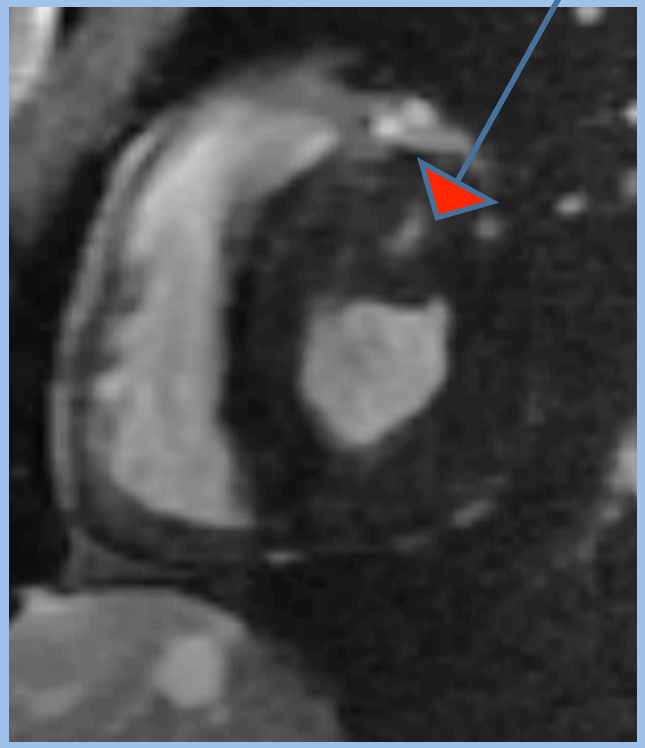


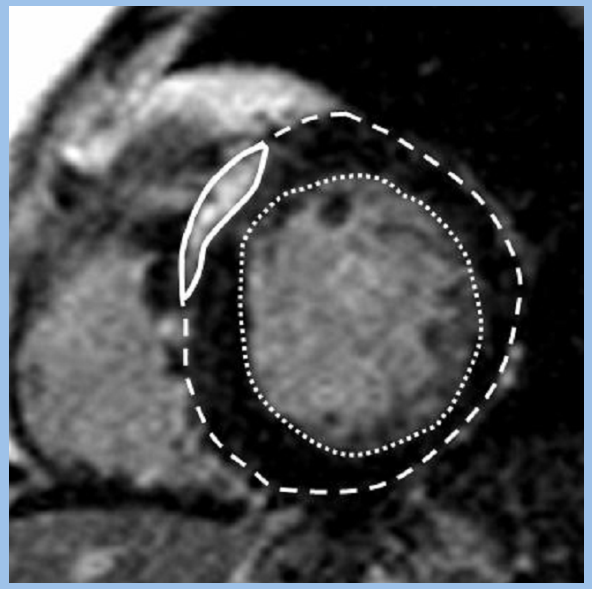


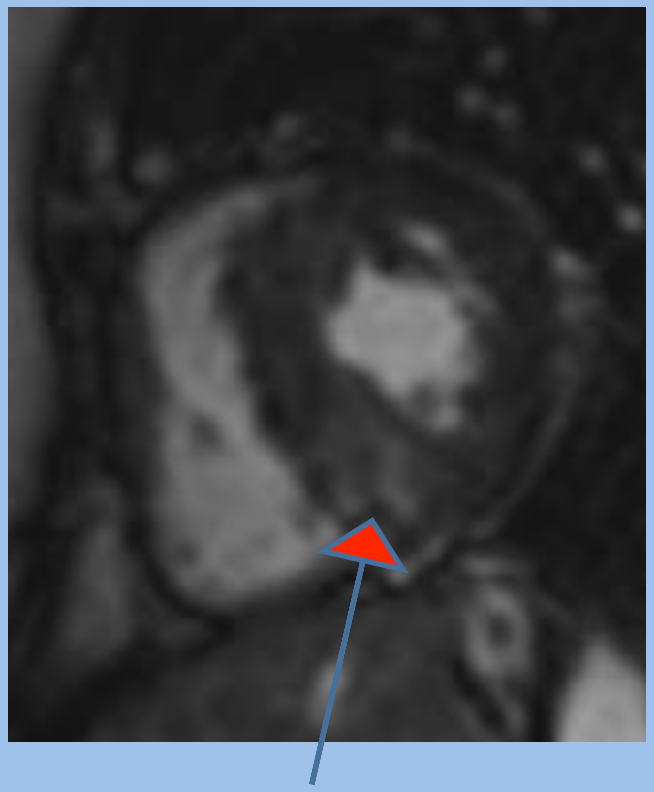


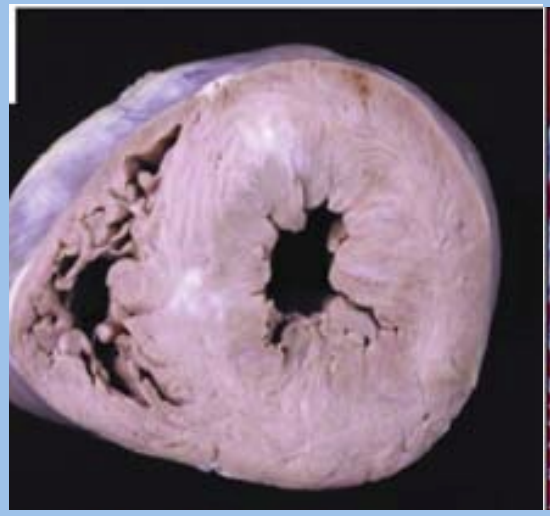
- **LOCALISATION**
- **OBSTRUCTION**

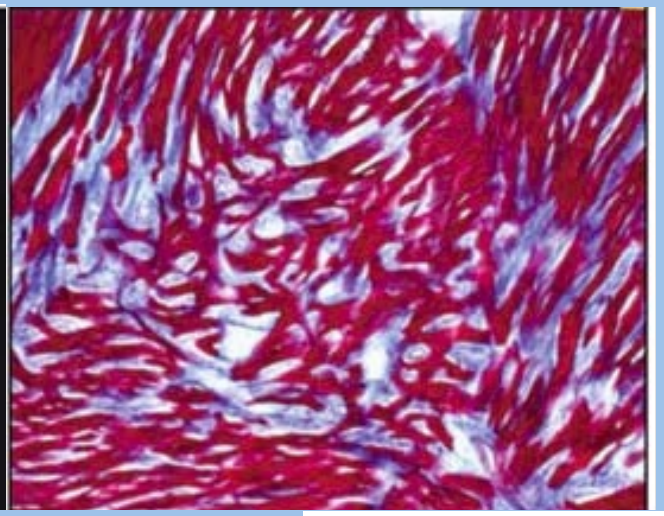
CMH











Ho CY et al. NEJM 2010;363:552-63

O'Hanlon R et al. JACC 2010;56:867-74.

Fluechter *et al. Journal of Cardiovascular Magnetic Resonance* 2010, **12**:30

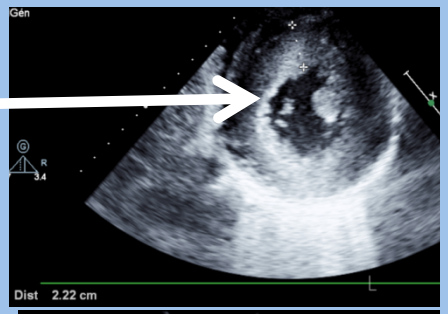
Calcul du risque

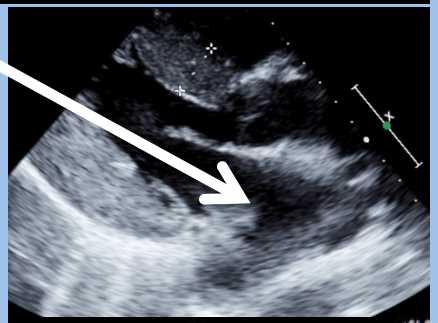
HCM Risk-SCD Calculator

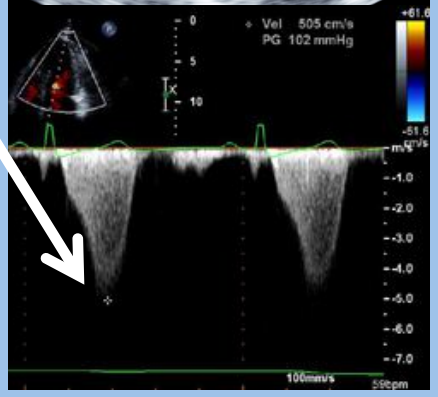
Age	Years	*Age at evaluation*
Maximum LV wall thickness	mm	*Transthoracic Echocardiographic measurement*
Left atrial size	mm	*Left atrial diameter determined by M-Mode or 2D echocardiography in the parasternal long axis plane at time of evaluation*
Max LVOT gradient	mmHg	*The maximum LV outflow gradient determined at rest and with Valsalva provocation (irrespective of concurrent medical treatment) using pulsed and continuous wave Doppler from the apical three and five chamber views. Peak outflow tract gradients should be determined using the modified Bernouilli equation: Gradient= $4V^2$, where V is the peak aortic outflow velocity*
Family History of SCD	No Yes	*History of sudden cardiac death in 1 or more first degree relatives under 40 years of age or SCD in a first degree relative with confirmed HCM at any age (post or ante-mortem diagnosis).*
Non-sustained VT	No Yes	*3 consecutive ventricular beats at a rate of 120 beats per minute and <30s in duration on Holter monitoring (minimum duration 24 hours) at or prior to evaluation.*
Unexplained syncope	No Yes	*History of unexplained syncope at or prior to evaluation.*

Risk of SCD at 5 years (%):	
ESC recommendation:	

EUROPEAN SOCIETY OF CARDIOLOGY®







HCM Risk-SCD should not be used in:

Paediatric patients (<16 years)

Elite/competitive athletes

HCM associated with metabolic diseases (e.g. Anderson-Fabry disease), and syndromes (e.g. Noonan syndrome).

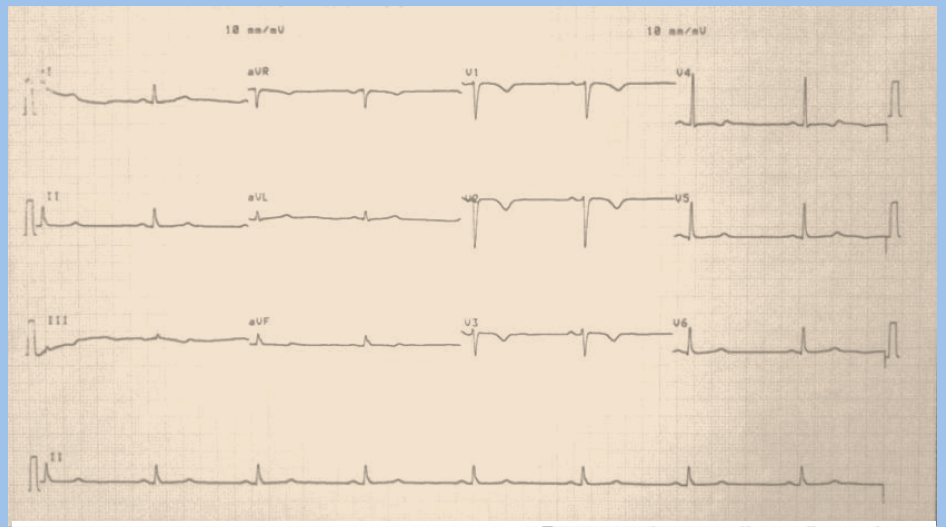
Patients with a previous history of aborted SCD or sustained ventricular arrhythmia who should be treated with an ICD for secondary prevention.

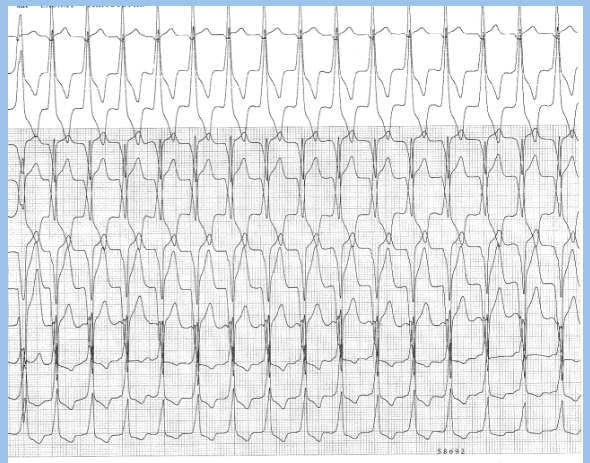
DAVD

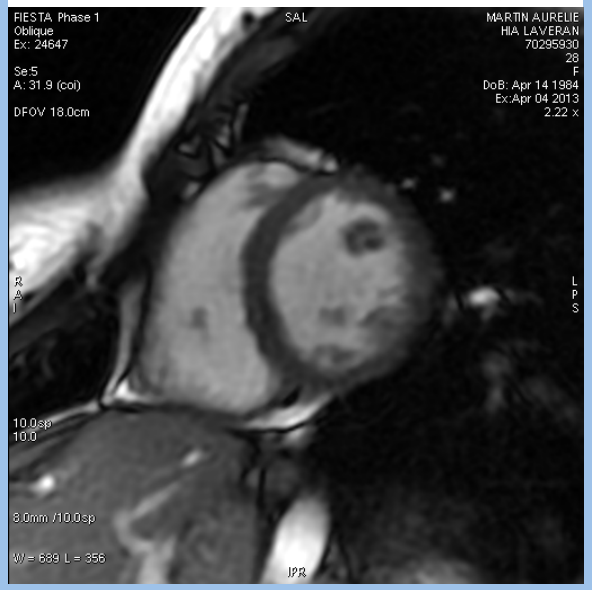
- **1/1000**

Sport: x 5 risque de MS!

CI compétition







Corrado D. J Am Coll Cardiol 2003;421959-63.

DAVD

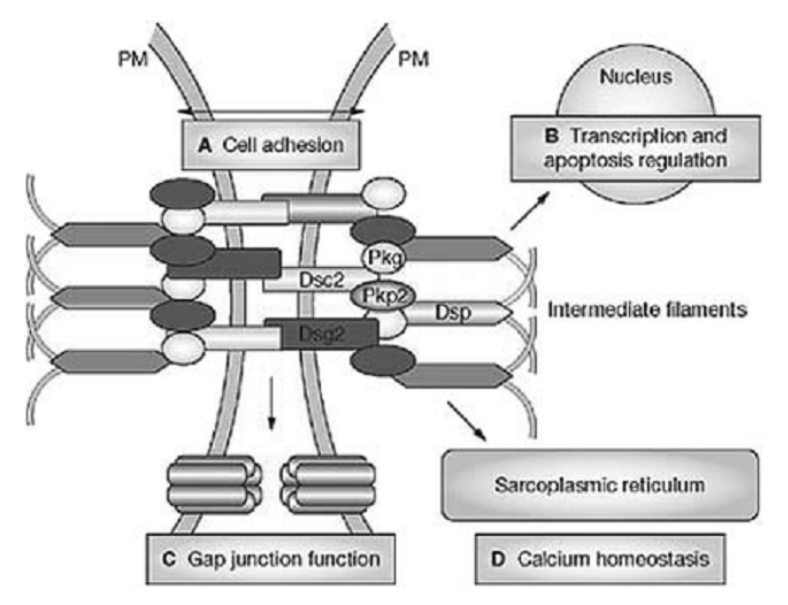
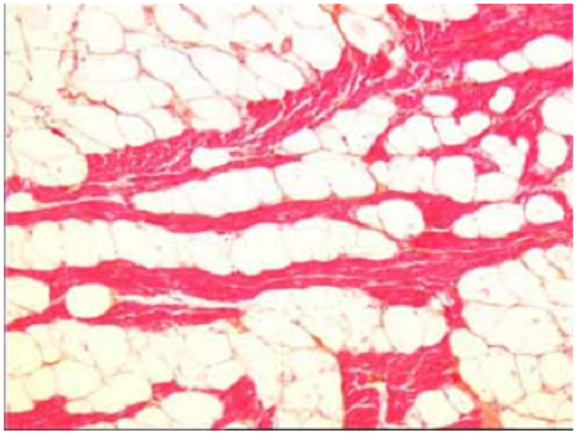


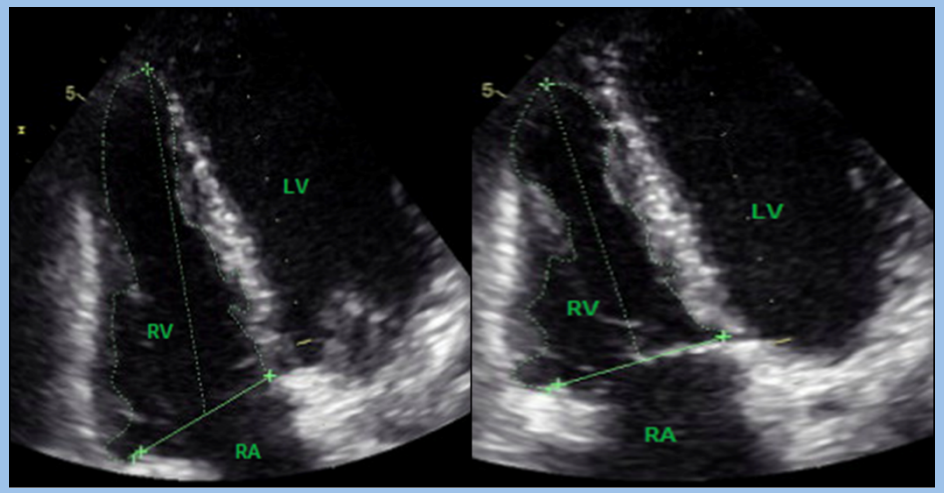
Figure 1 The cardiac desmosome and proposed roles of the desmosome in (*A*) supporting structural stability through cell–cell adhesion, (*B*) regulating transcription of genes involved in adipogenesis and apoptosis, and maintaining proper electrical conductivity through regulation of (*C*) gap junctions and (*D*) calcium homeostasis. Dsc2 indicates desmocollin-2; Dsg2, desmoglein-2; Dsp, desmoplakin; Pkg, plakoglobin; Pkp2, plakophilin-2; and PM, plasma membrane. Reprinted by permission from Macmillan Publishers Ltd: *Nat Clin Pract Cardiovasc Med*[11], © 2008.

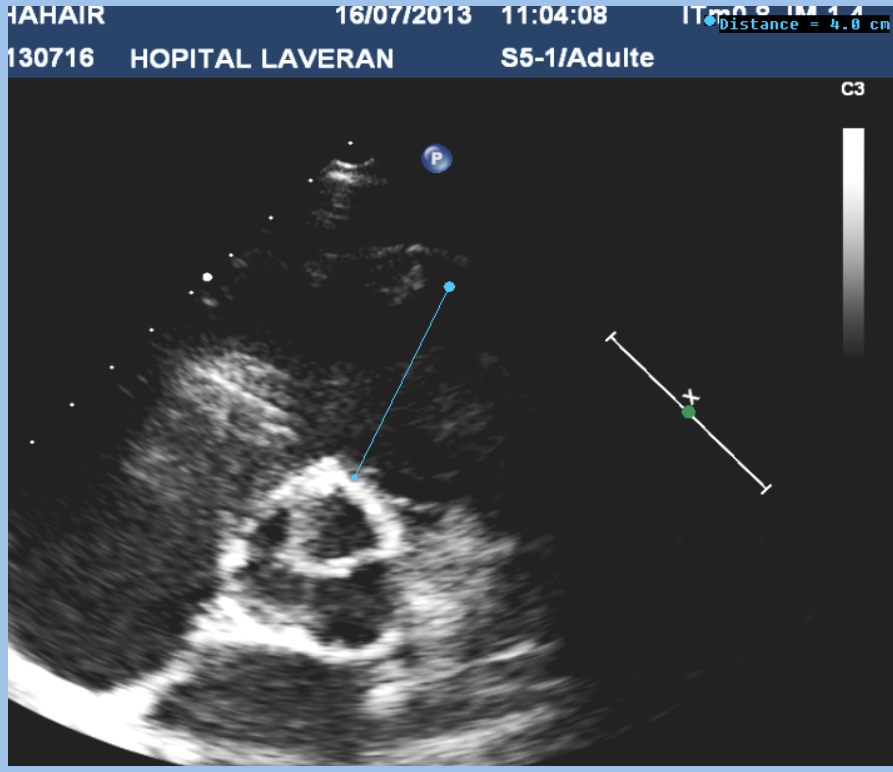


DAVD

By 2D echo:

- Regional RV akinesia, dyskinesia, or aneurysm
- *and* 1 of the following (end diastole):
  - — PLAX RVOT $\geq$32 mm (corrected for body size [PLAX/BSA] $\geq$19 mm/m^2)
  - — PSAX RVOT $\geq$36 mm (corrected for body size [PSAX/BSA] $\geq$21 mm/m^2)
  - — *or* fractional area change $\leq$33%





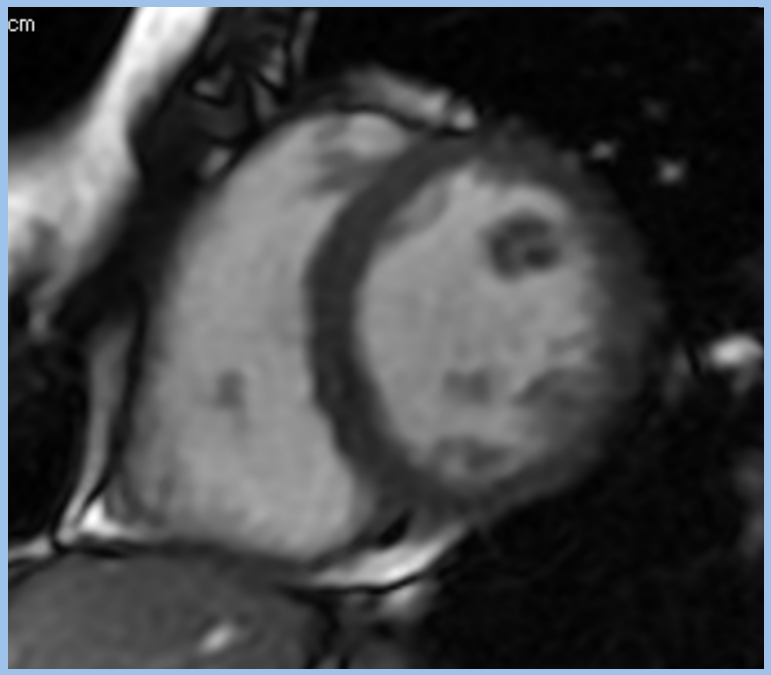
- Regional RV akinesia or dyskinesia
- *and* 1 of the following (end diastole):
  - — PLAX RVOT $\geq$29 to $<$32 mm (corrected for body size [PLAX/BSA] $\geq$16 to $<$19 mm/m^2)
  - — PSAX RVOT $\geq$32 to $<$36 mm (corrected for body size [PSAX/BSA] $\geq$18 to $<$21 mm/m^2)
  - — *or* fractional area change $>$33% to $\leq$40%

DAVD

Majeur

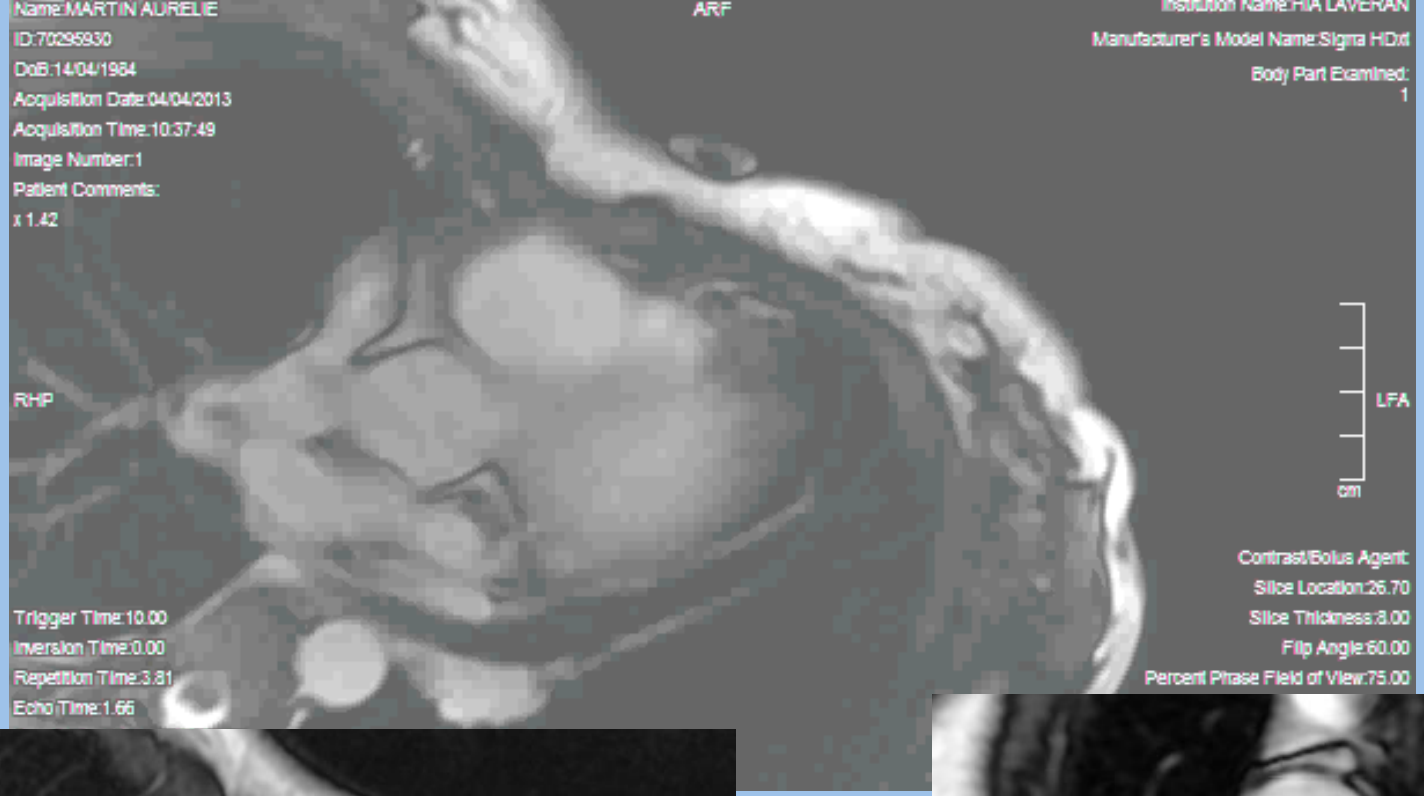
By MRI:

- Regional RV akinesia or dyskinesia or dyssynchronous RV contraction
- *and* 1 of the following:
  - — Ratio of RV end-diastolic volume to BSA $\geq$110 mL/m^2 (male) or $\geq$100 mL/m^2 (female)
  - — *or* RV ejection fraction $\leq$40%



Mineur

- Regional RV akinesia or dyskinesia or dyssynchronous RV contraction
- *and* 1 of the following:
  - — Ratio of RV end-diastolic volume to BSA $\geq$100 to $<$110 mL/m^2 (male) or $\geq$90 to $<$100 mL/m^2 (female)
  - — *or* RV ejection fraction $>$40% to $\leq$45%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Acquisition Date:04/04/2013
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Patient Comments:
x 1.42
ARF
Institution Name:RIA LAVERAN
Manufacturer's Model Name:Signa HDxt
Body Part Examined:
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RHP
LFA
cm
Contrast/Bolus Agent:
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Slice Thickness:8.00
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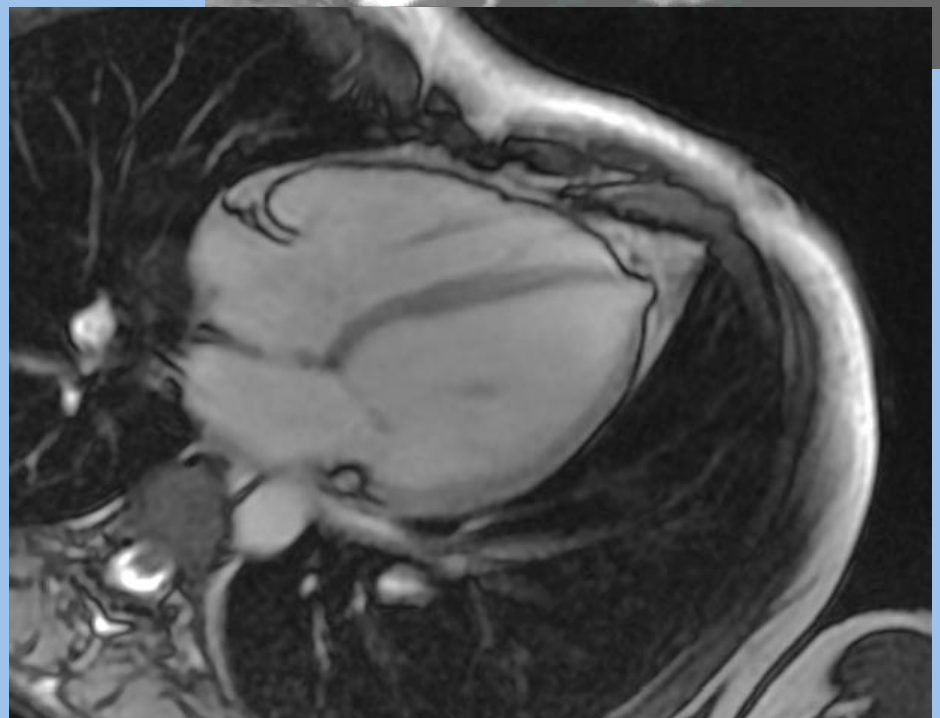
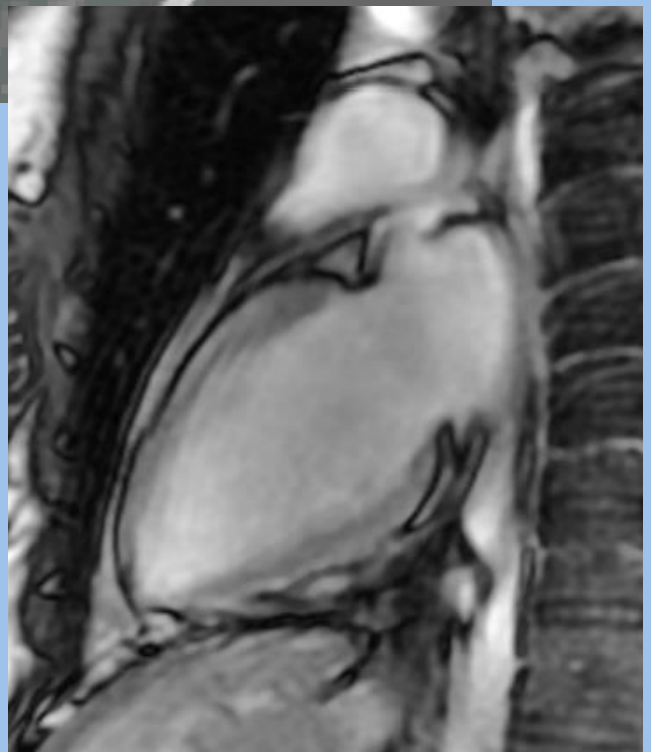
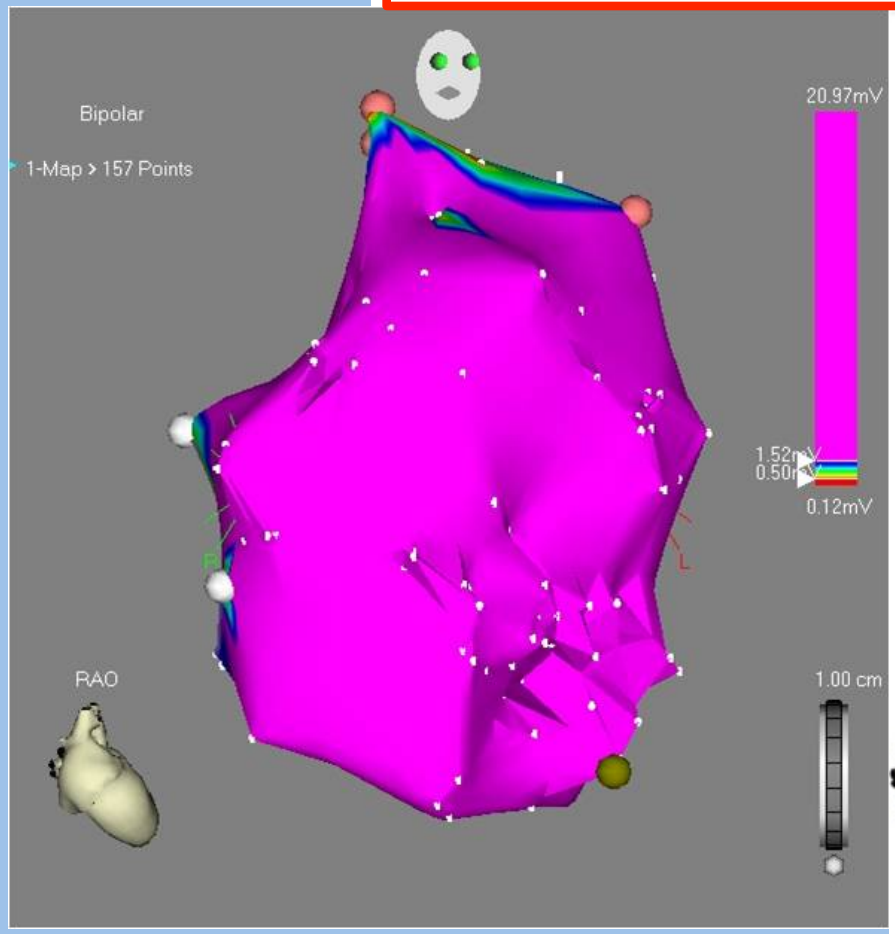
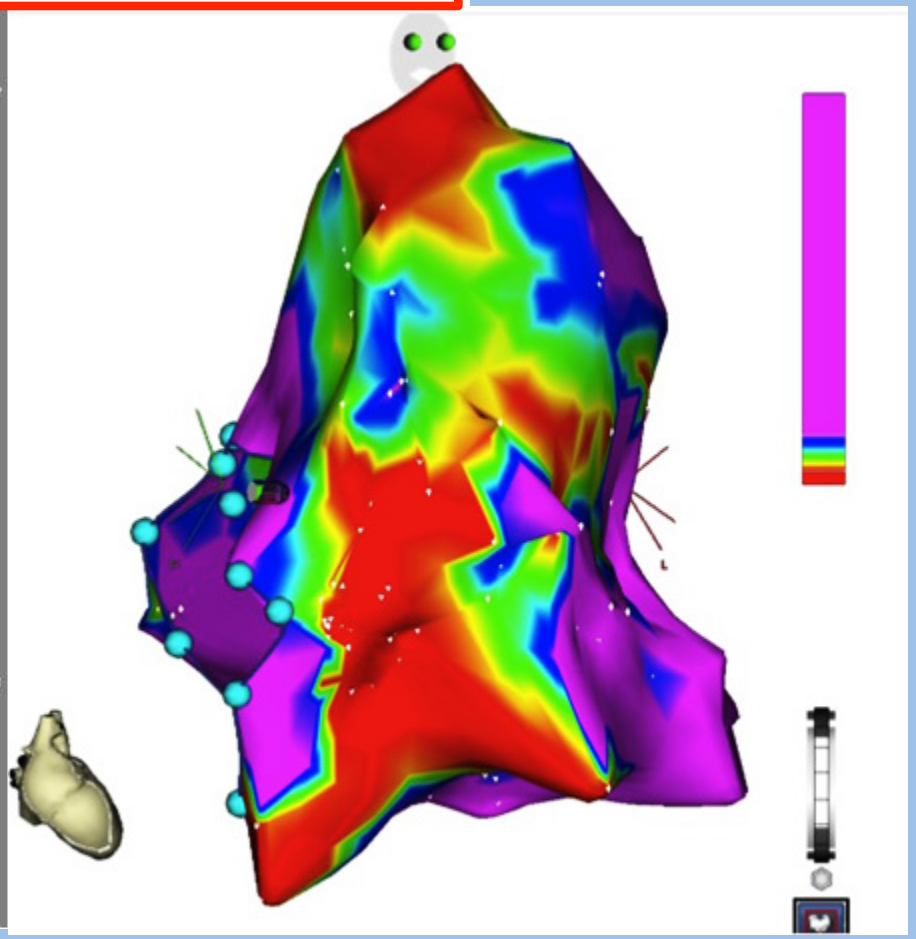
Echo Time:1.66





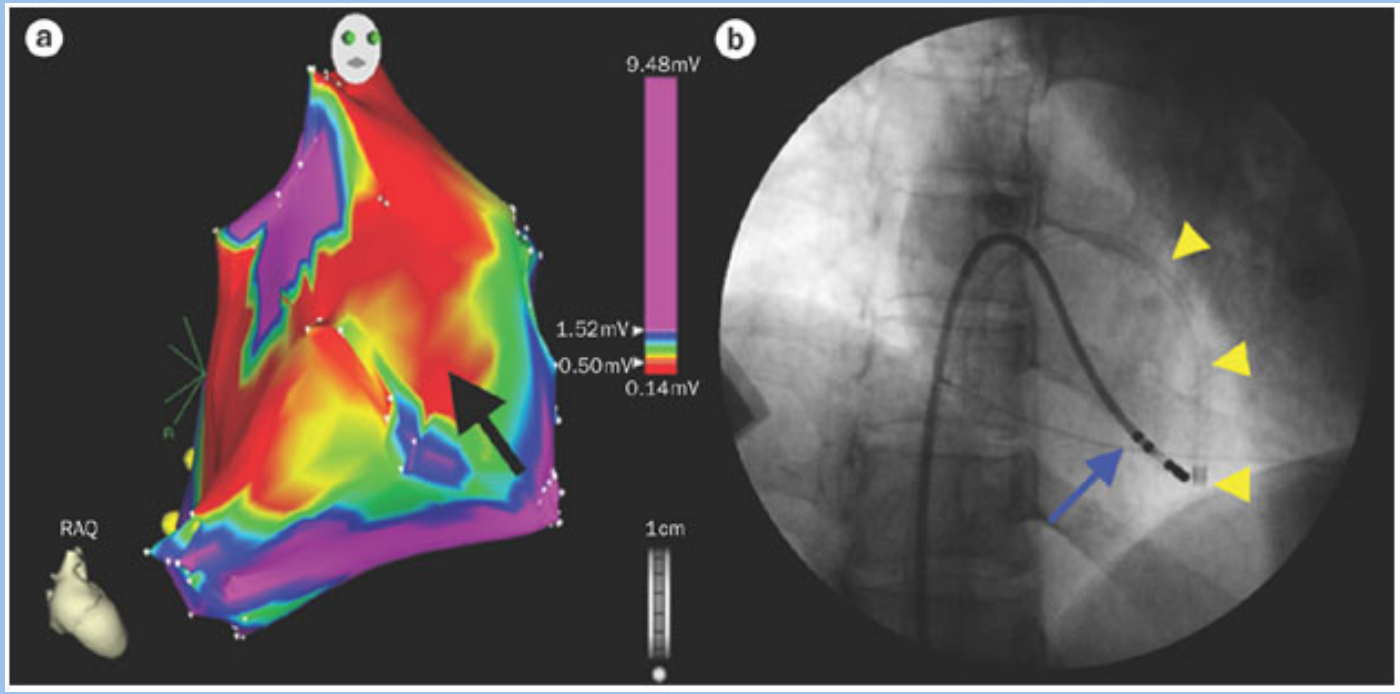
- EPS should be considered in the diagnosis and/or evaluation of patients with suspected ARVC/D (class IIa).
- Programmed ventricular stimulation may be considered for arrhythmic risk stratification of asymptomatic ARVC/D patients (class IIb).
- Endocardial voltage mapping may be considered in the diagnostic and prognostic evaluation of ARVC/D patients (class IIb).





European Heart Journal (2015) **36**, 3227–3237

Figure 1 Right anterior oblique view (30°) of electroanatomic map and EAM-guided endomyocardial biopsy in a patient with a noninvasive diagnosis of ARVC



King A (2009) FEAM-guided biopsy reveals cause of ventricular arrhythmias
Nat Rev Cardiol doi:10.1038/nrcardio.2009.42

nature REVIEWS CARDIOLOGY

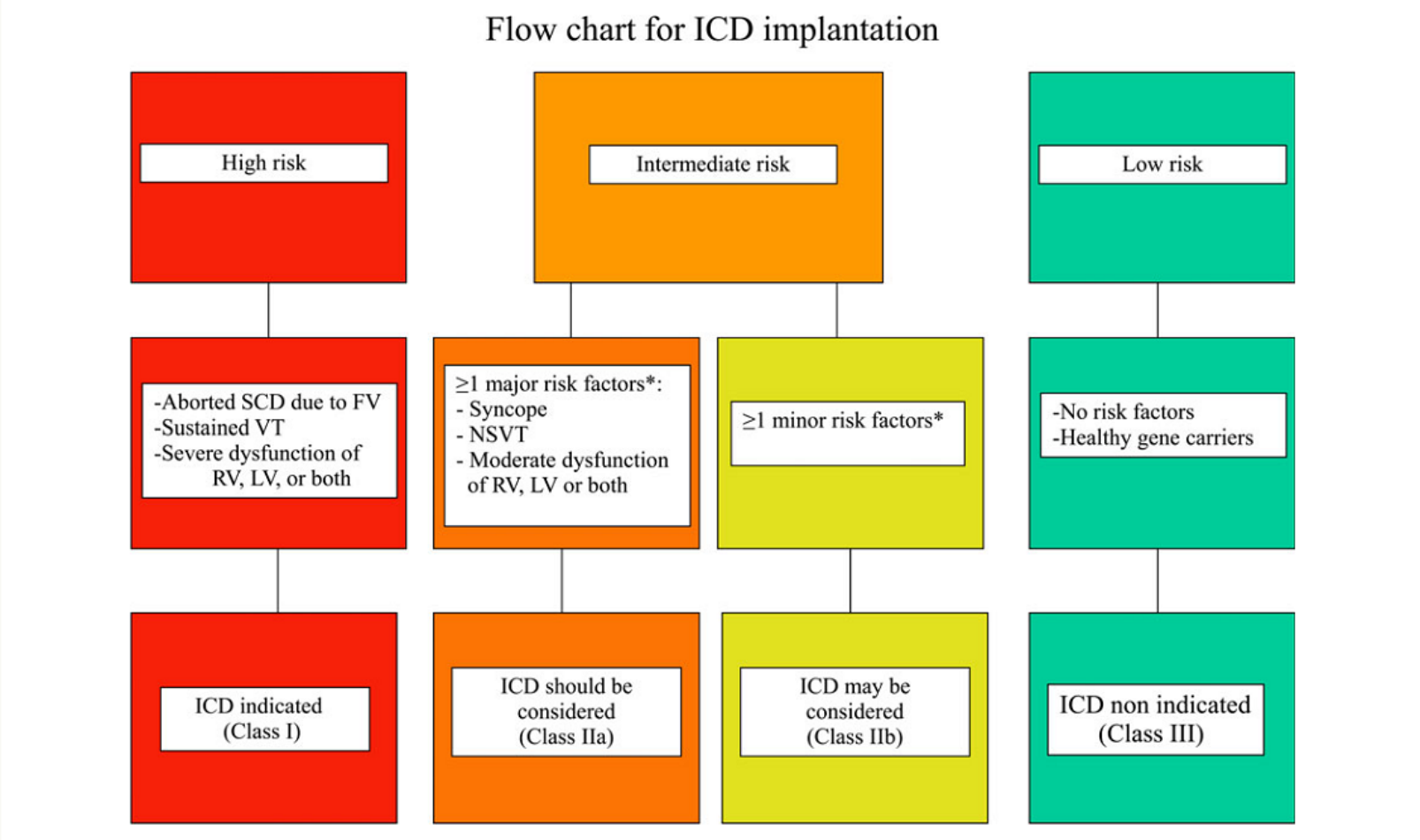
DAVD

- Haut risque
  - **MS / FV**
  - **TV soutenue**
  - **Dysfonction sévère VD,VG (FEVD ou VG < 36%)**
- FDR majeurs
  - **Syncope**
  - **TVNS**
  - **Dysfonction modérée VD (36 à 40%) et ou VG (36 à 45%)**

  – Autres FDR:

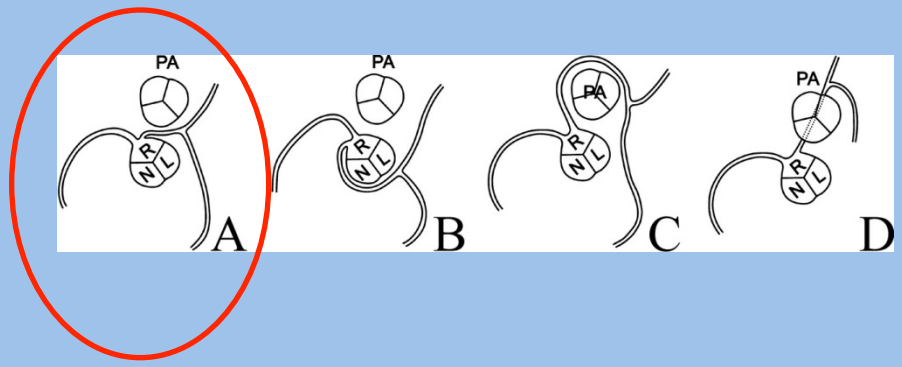
ATCD familial de MS, age jeune, sexe masculin, génotype complexe, TV – FV inductible, atteinte étendue au mapping EA, ondes T négatives ≥ 3 derivations; onde epsilon…

Treatment of arrhythmogenic right ventricular cardiomyopathy/dysplasia: an international task force consensus statement

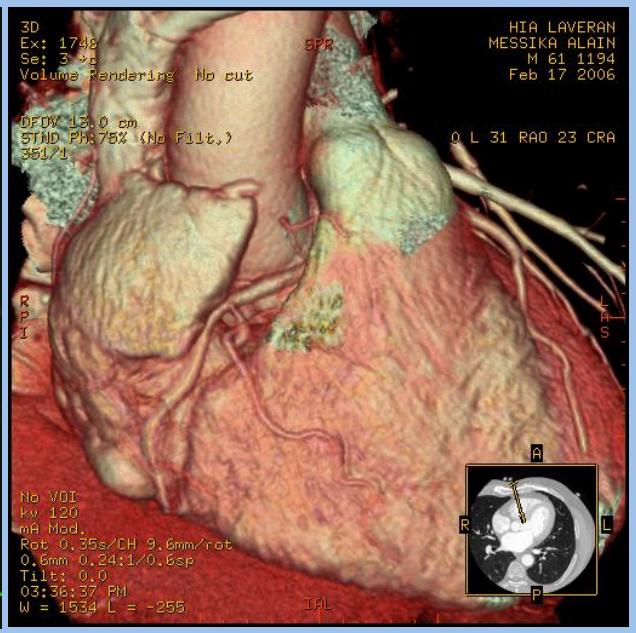


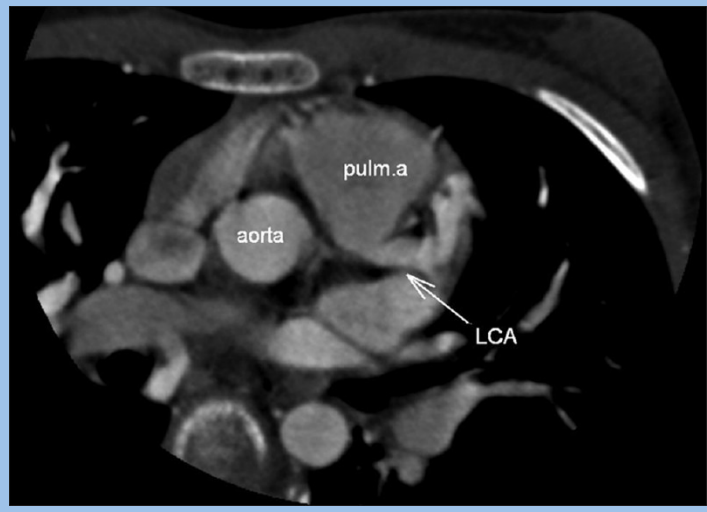
European Heart Journal (2015) **36**, 3227–3237

Anomalies de naissance des coronaires

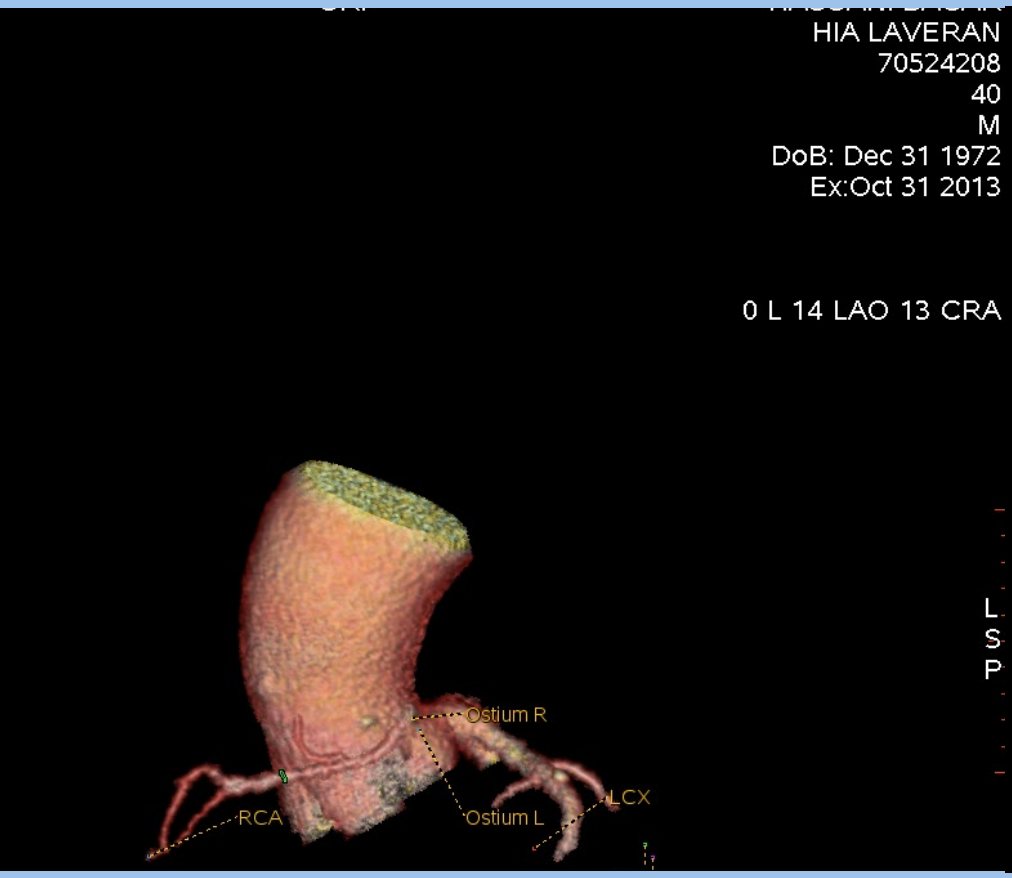


- Prevalence: 0.1 - 0.2%
- Risque de MS x 3 to 6 in athletes
- Symptomes : 30% to 40%
  - Syncope
  - Douleur thoracique
- Sensibilité de l'épreuve d'effort: 22%....





Peñalver et al. BMC Cardiovascular Disorders 2012, 12:83-93.
Basso C et al. JACC;2000:35:1493-501.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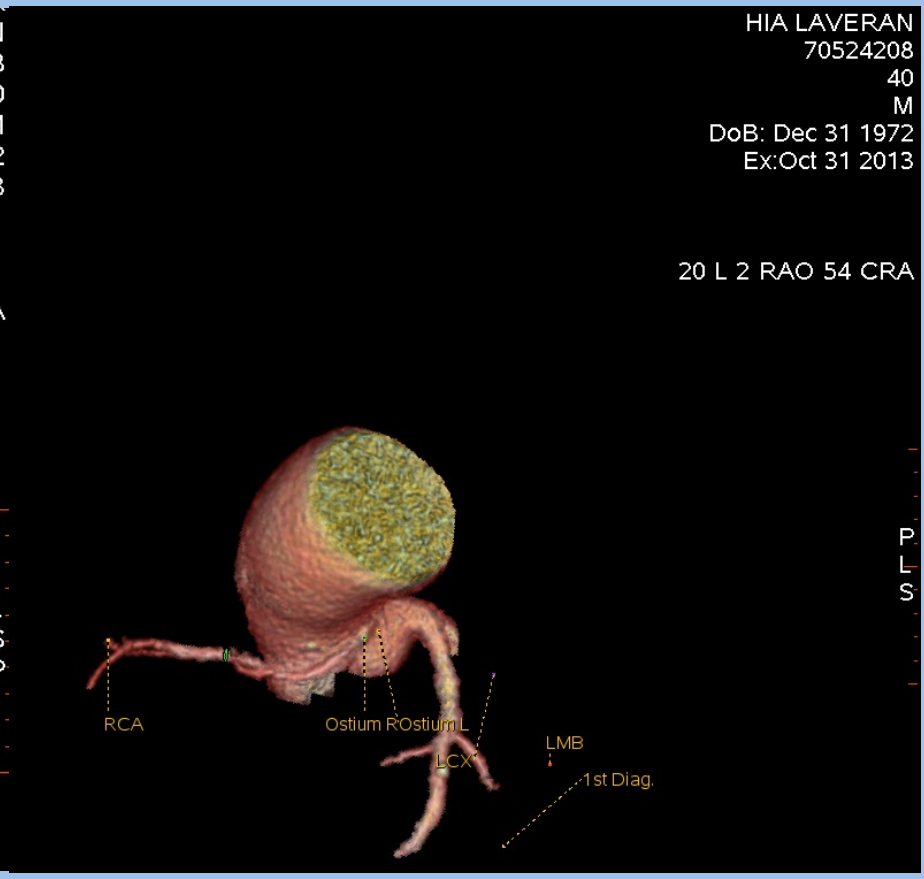
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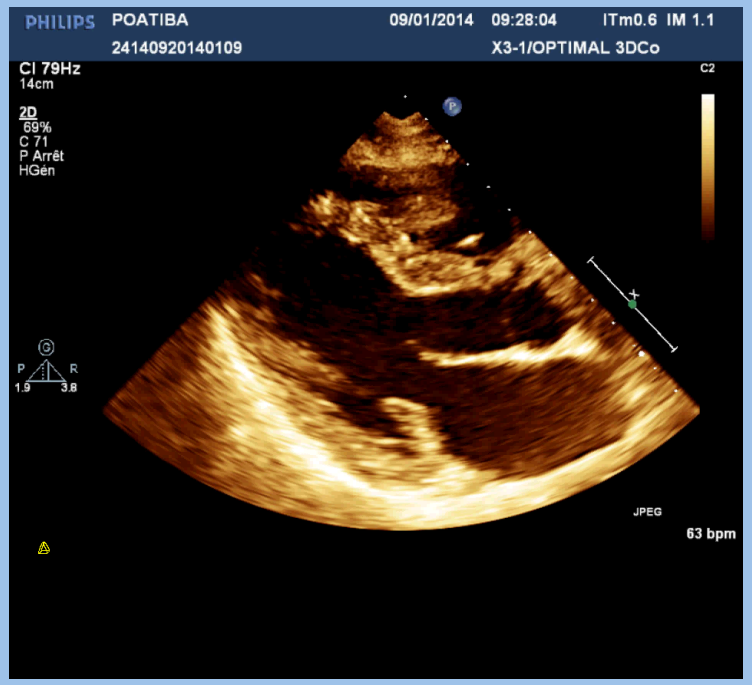
Ostium L
LCX
LMB
1st Diag.

Valvulopathies

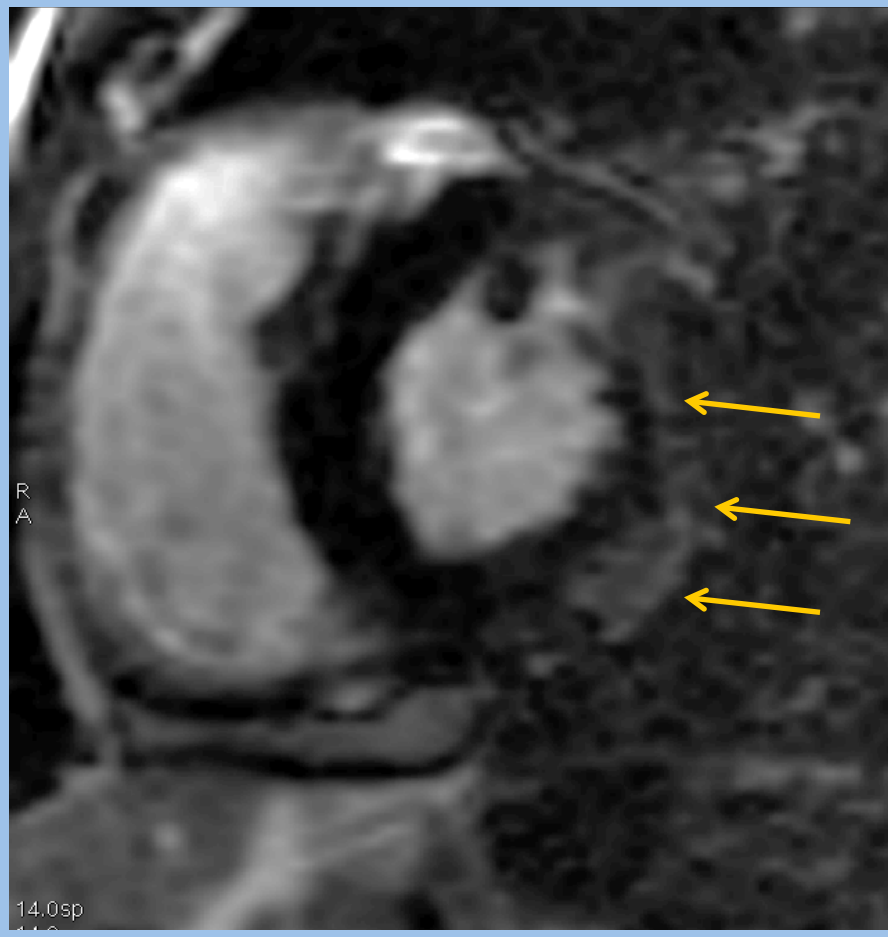
- **Mineure**
  - tous sports
- **Modérée à sévère (+ dysfonction VG)**
  - Pas de compétition
- **Prolapsus mitral…**
  - Syncope, histoire familiale de MS, arythmies
- **Prothèse:**
  - I,II - A, B / risque de collision (AVK)



Pelliccia et al. Eur Heart J 2005;26:1422-45

Myocardite

- **Active…**
  - Pas de compétition
- **6 premiers mois:**
  - Symptômes
  - Fonction VG
  - arythmies
- **suivi**
  - >6 mois
- **Prévention**
  - … 58% poursuivent un sport malgré une fièvre…



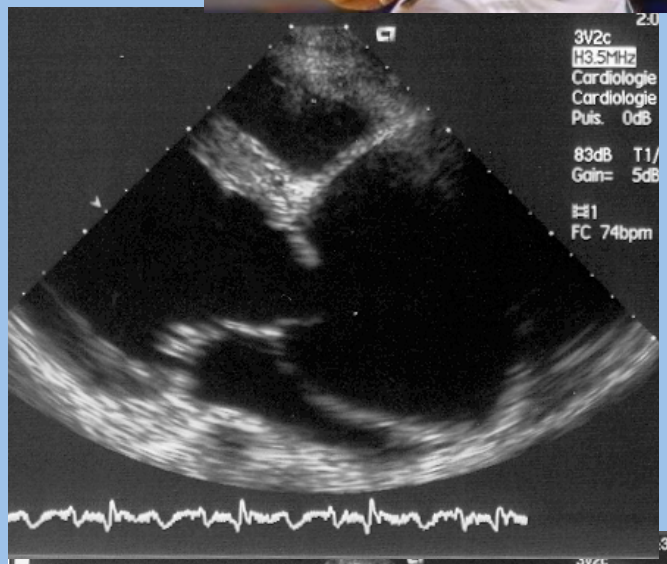
Basso C. Clin cardiol 2007;25:423-9.
Pelliccia et al. Eur Heart J 2005;26:1422-45.
Chevalier L et al. Arch Mal Cœur Vaiss 2005;98:109-14.

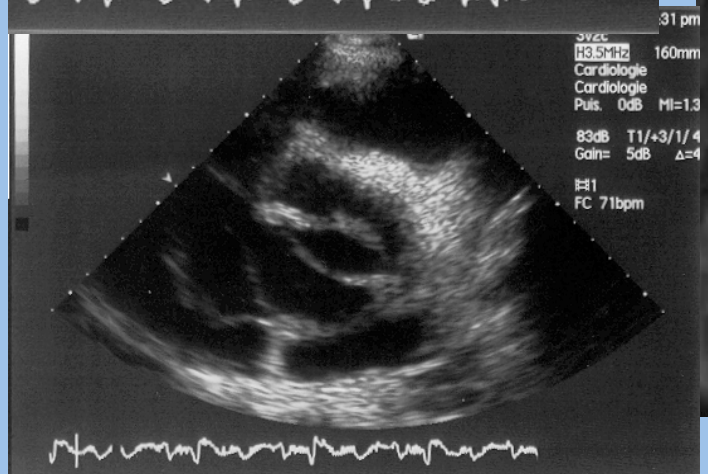
Marfan

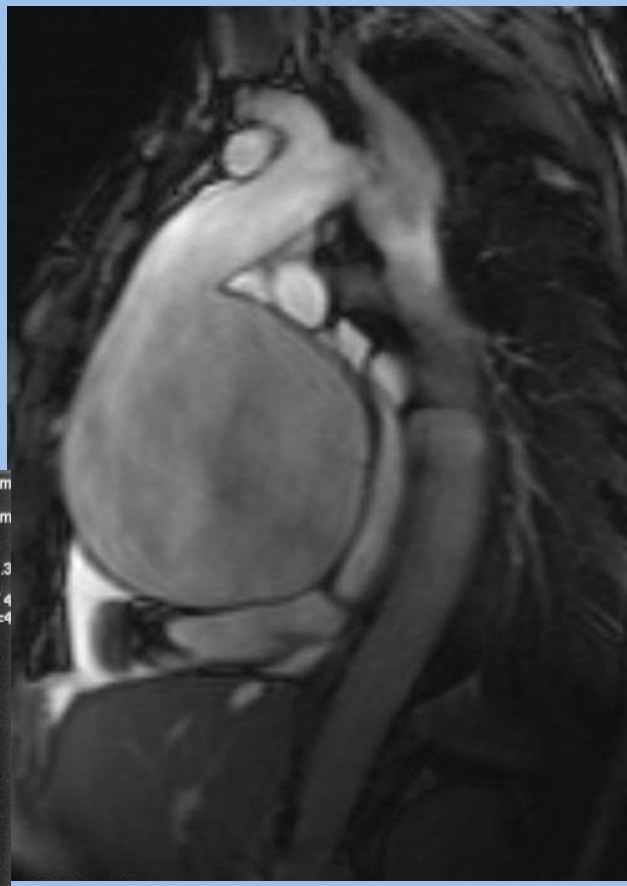
- 1/5000
- Rupture aortique / dissection
- <u>Malgré des dimensions normales ou subnormales</u>
- Phenotype
- Genotype +
- ATCD familial:

>>>pas de compétition

- suivi+++
  - Echo 6 à 12 mois







Yetman AT. J Am Coll Cardion 2003;41:329-32.

Arythmies

- <u>Supraventriculaire</u>
  - Flutter, TRIN….: ablation
  - FA symptomatique: ablation
  - FA permanente (en l'absence de cardiopathie, WPW) : cas par cas

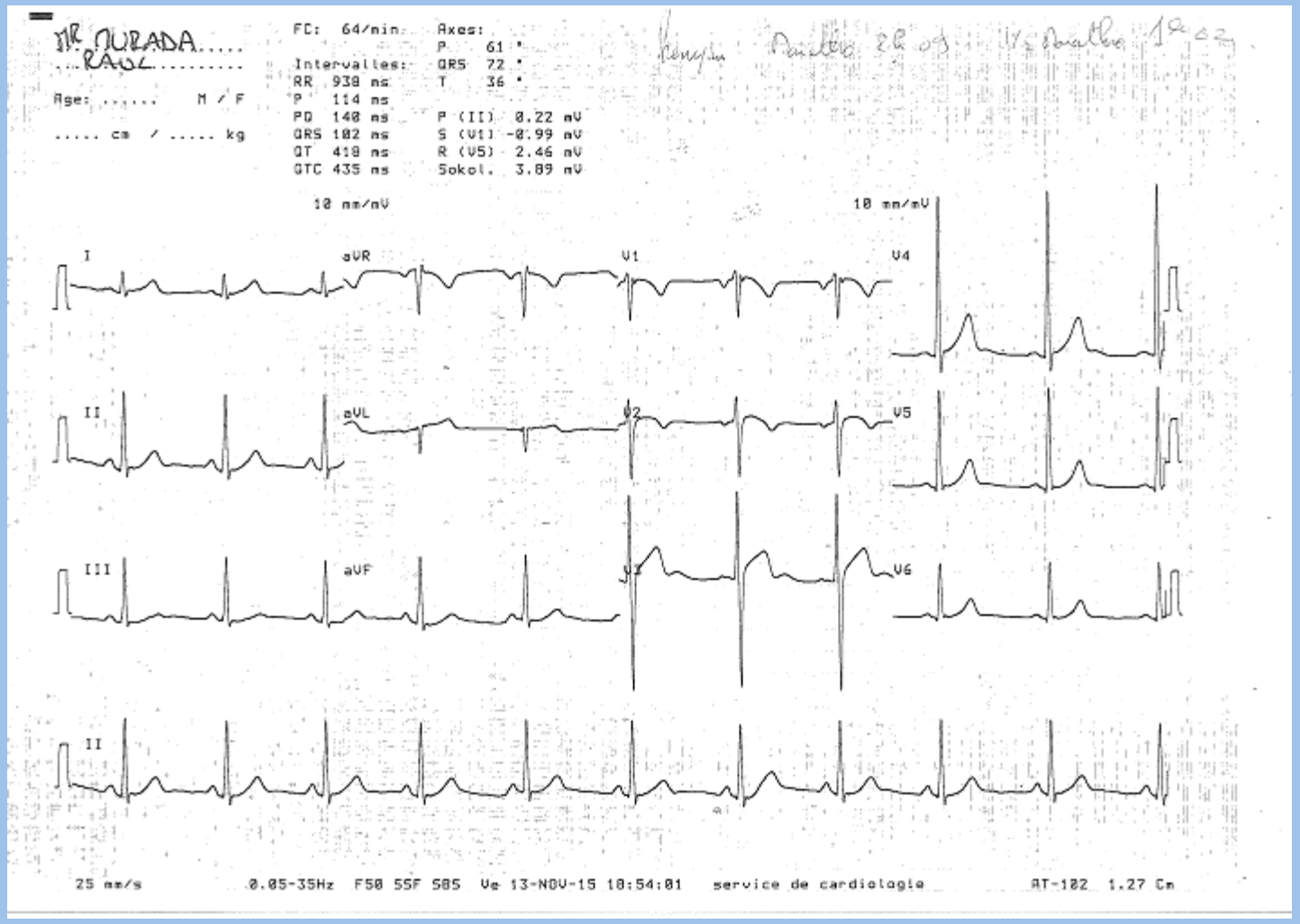
- <u>WPW</u>
  - Ablation (FA, symptômes…)
  - Non ablaté: ep effort / EEP (PRA >

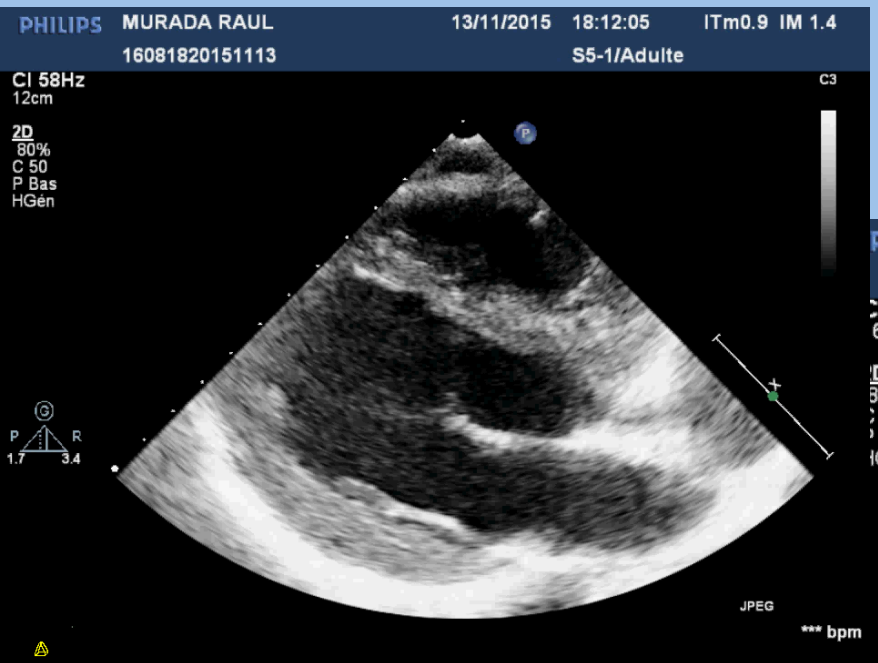
Arythmies

- Ventriculaire
  - ESV, TVNS… chercher une cardiopathie
  - ablation
- Long QT, Brugada: pas de compétition (liste)
- Repolarisation précoce: fréquente chez l'athlète (40%?)
- Pacemaker: IA IB excepté sport de contact et plongée (> 30 m).
- ICD: IA IB (+6 mois après implantation).

Tikkanen et al. Circulation 2011;123:2666-73.

Le cœur d'athlète?

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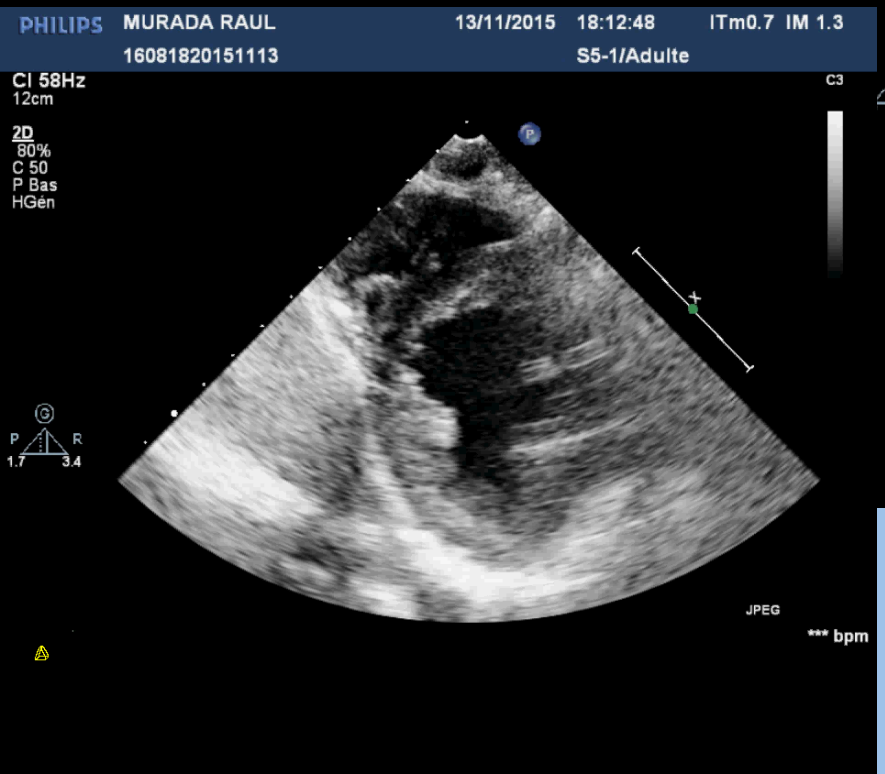
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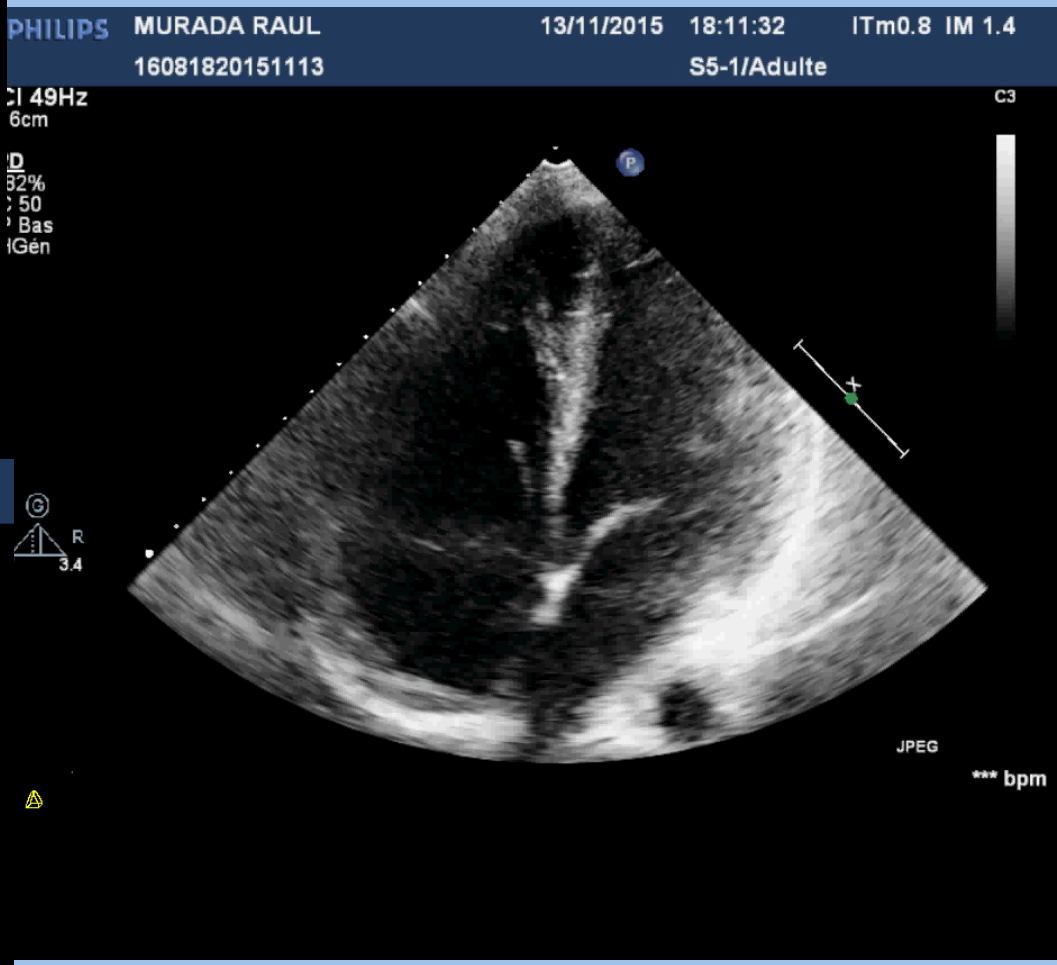
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Le cœur d'athlète?

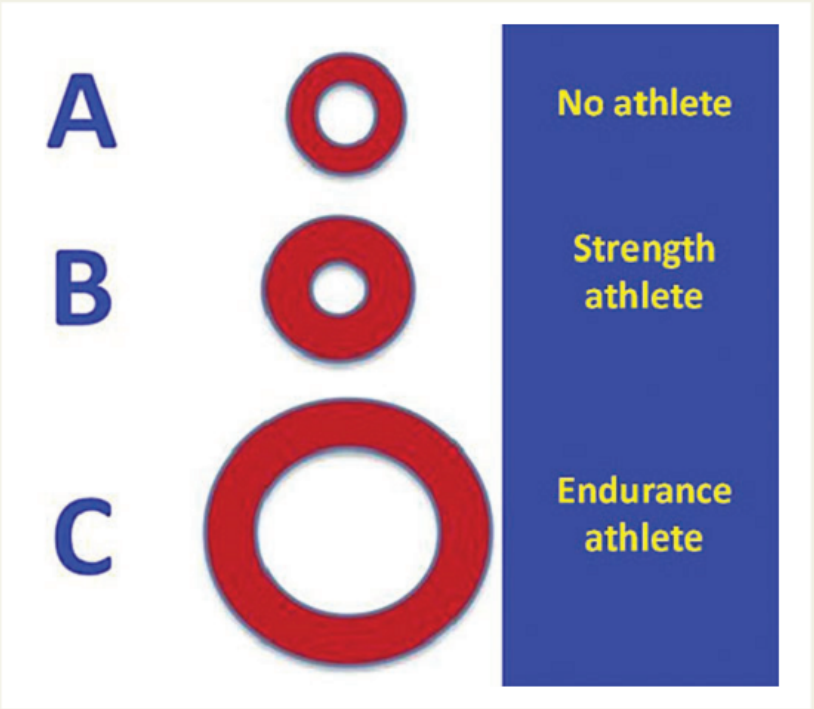
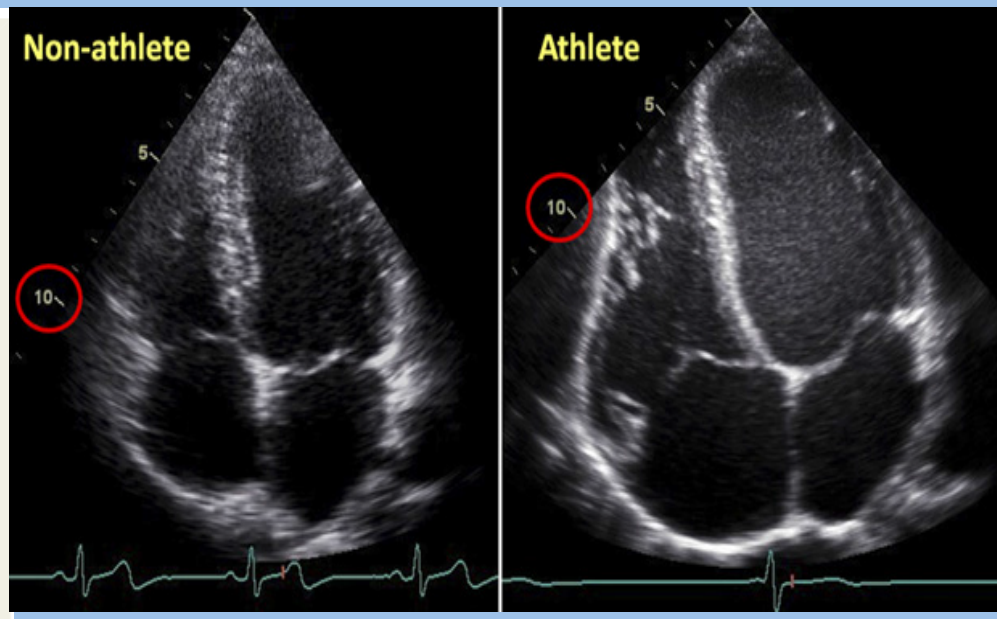


Figure 1: The 'Morganroth hypothesis'. A = no athlete, B = Strength training athlete: concentric LV hypertrophy, C = Endurance athlete: eccentric LV hypertrophy.



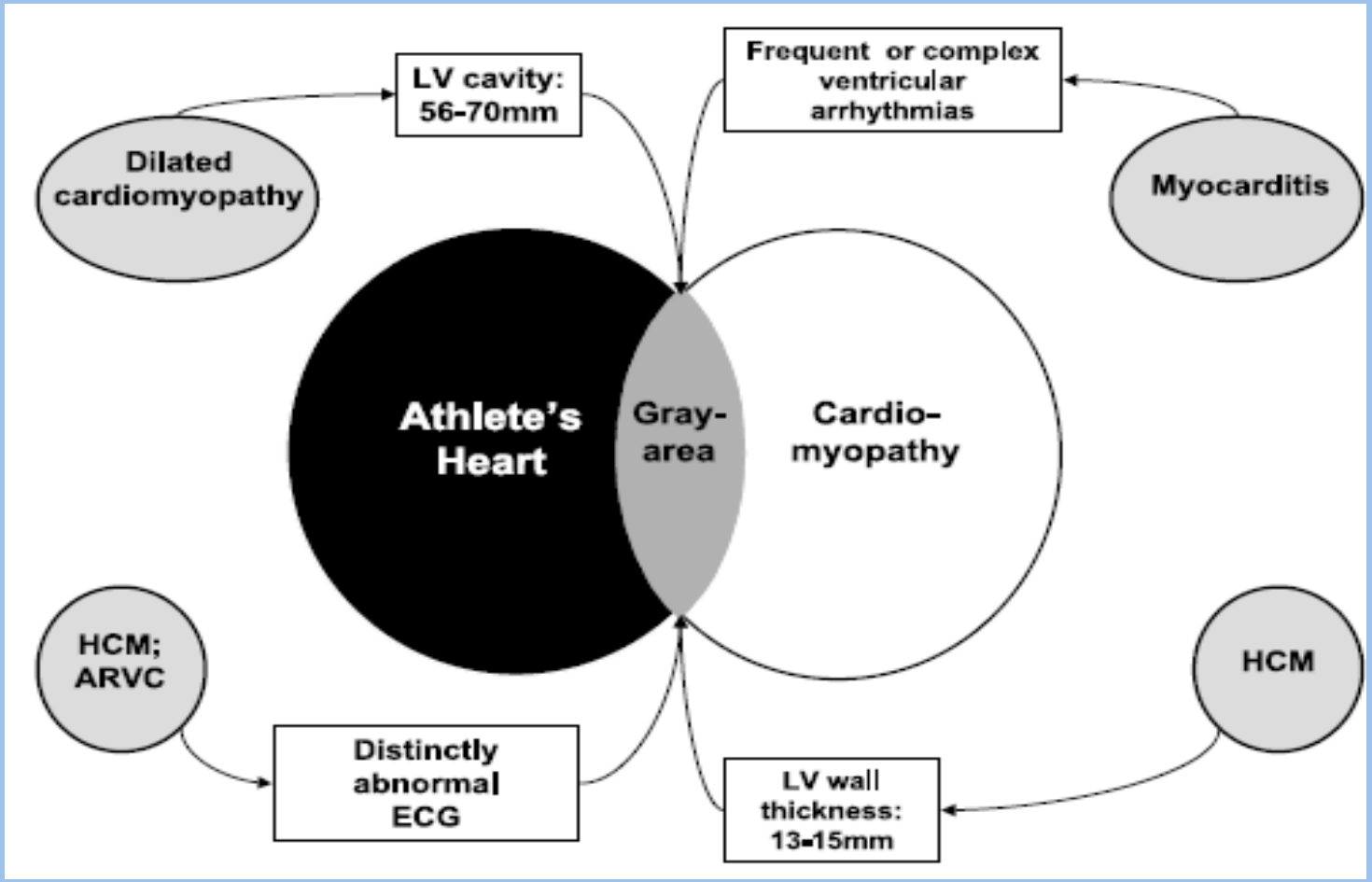
- **Remodelage structurel + fonctionnel + électrique**
- **Ces adaptations physiologiques permettent les hautes performances!**
- **Quel est l'effet pro arythmogène de ce remodelage?**

European Heart Journal – Cardiovascular Imaging (2015) **16**, 353

Le cœur d'athlète?

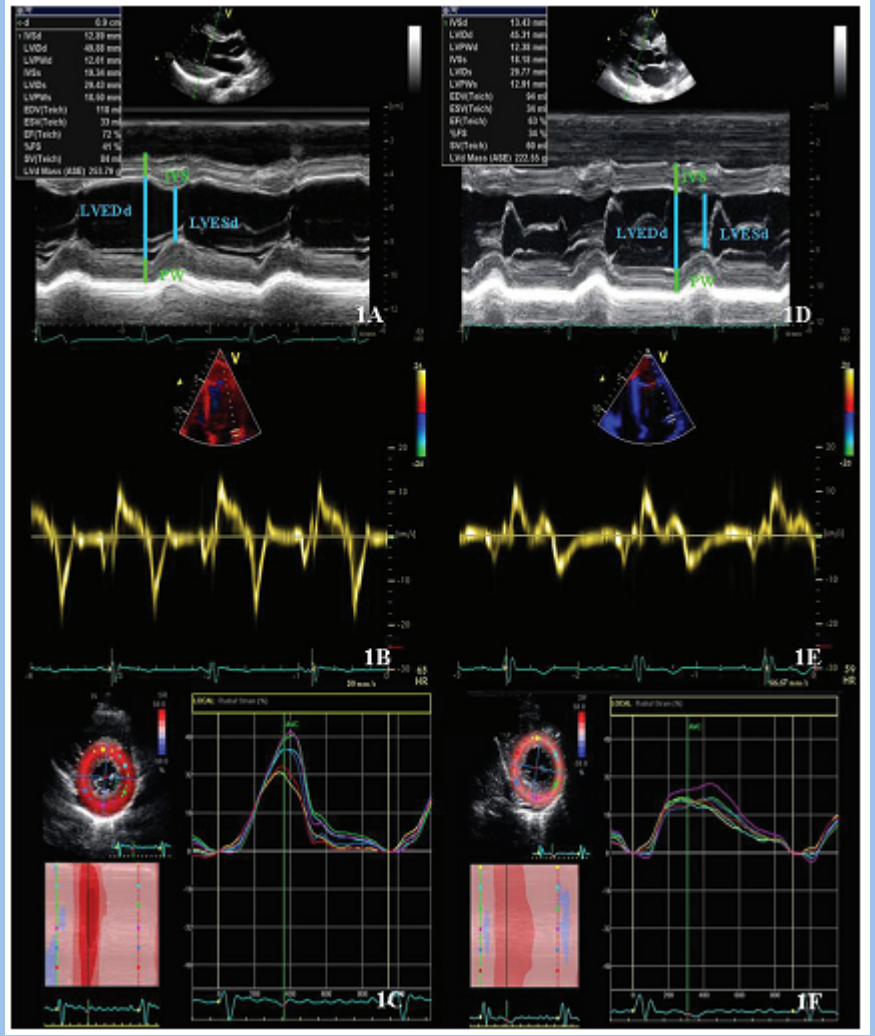
Reference	Year	Athletes, n	Athlete Type	Exercise Exposure	Primary Findings*
Training studies					
Ehsani et al[51]	1978	8	Swimmers (7 male, 1 female)	Swimming for 9 wk	Increased LVMI (104 vs 109 g/m²)
Wieling et al[52]	1981	9	Freshman rowers (all male)	Rowing for 7 mo	Increased LVIDd (51.5 vs 53.3 mm)
		14	Senior rowers (all male)	Rowing for 7 mo	Increased LVIDd (56.1 vs 57.9 mm)
Abergel et al[42]	2004	37	Cyclists (all male)	Professional cycling for 3 y	Increased LVID (58.3±4.8 to 60.3±4.2 mm); decreased LVWT (11.8±1.2 to 10.8±1.2 mm)
Naylor et al[45]	2005	22	Rowers (17 male, 5 female)	Rowing for 6 mo	Increased LVM (236±7 vs 249±9 g; $P<0.05$); increased early diastolic function
duManoir et al[53]	2007	10	Rowers (all male)	Rowing training for 10 wk	Increased LVMI (88±20 vs 103±22; $P<0.05$) associated with increased LVEDV and LVWT
Baggish et al[35]	2008	40	Rowers (20 male, 20 female)	Rowing for 90 d	Increased LVM (116±18 vs 130±19 g/m²) with increased LVEDV
		24	Football (24 male)	Football for 90 d	Increased LVM (115±14 vs 132±11 g/m²) with increased LVWT
Baggish et al[44]	2008	20	Rowers (10 male, 10 female)	Rowing for 90 d	Increased LV radial and longitudinal strain; decreased septal circumferential strain owing to concomitant RV dilation
Weiner et al[54]	2010	15	Rowers (all male)	Rowing for 90 d	Increased LV torsion and LV early diastolic untwisting rate
Detraining studies					
Fagard et al[55]	1983	12	Cyclists (all male)	Detraining for 12 wk	Decreased IVS (12.3±0.4 vs 11.5±0.4)
					Decreased PWT (13.0±0.5 vs 11.8±0.6); LVIDd unchanged
Pellicia et al[56]	2002	40	Mixed, elite (all male)	Detraining for 12 wk	Decreased LVWT by 15% (12.0±1.3 vs 10.1±0.8); decreased LVIDd by 7% (61.2±2.9 vs 57.2±3.1)

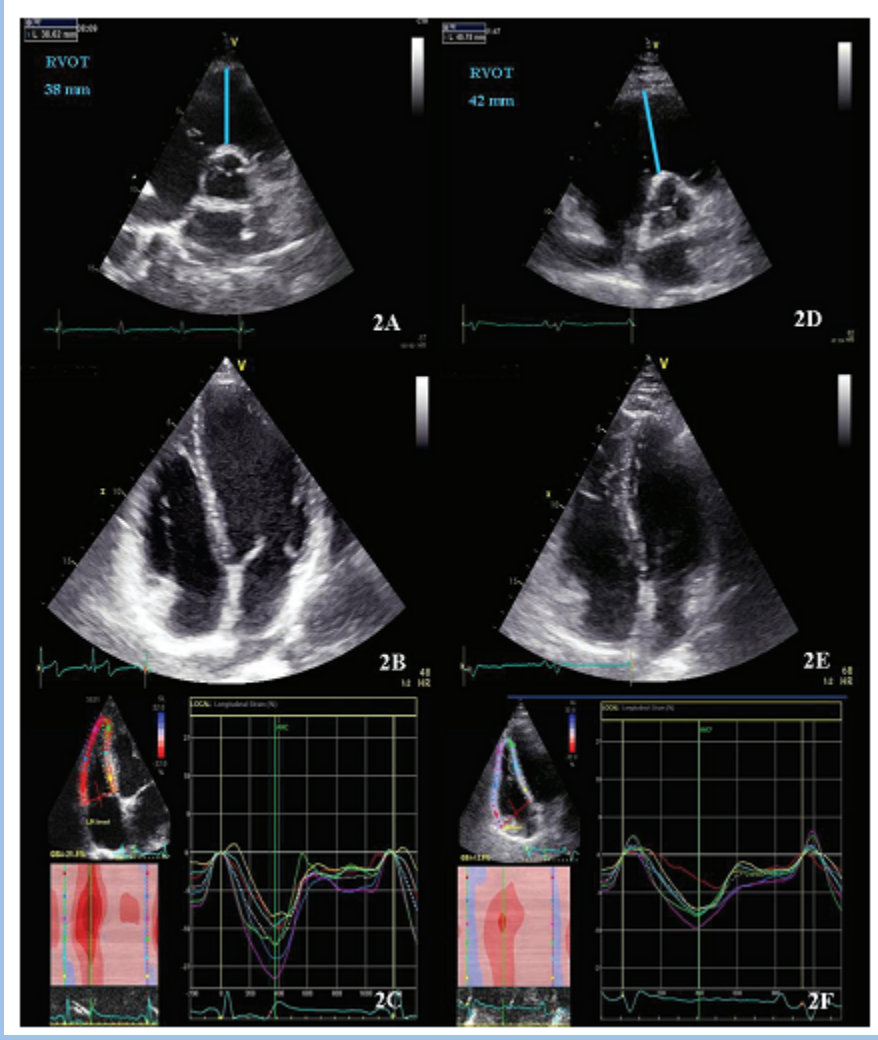
Baggish AL. Circulation 2011;123:2723-35
Breuckmann et al. Radiology. 2009;25:50-57.
La Gerche. Eur Heart J 2012; 33:998-1006



Baggish AL. Circulation 2011;123:2723-35

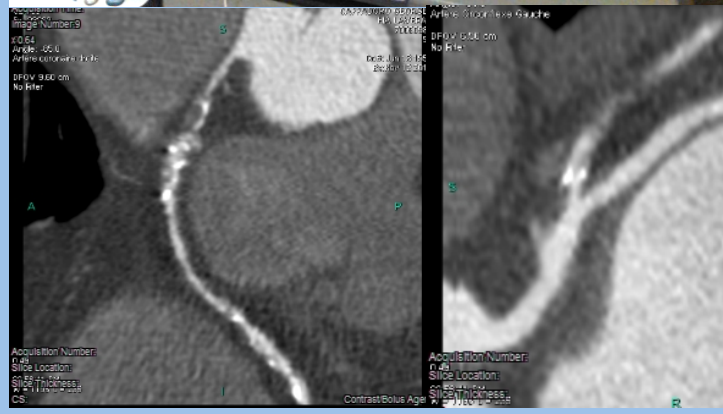
The multi-modality cardiac imaging approach to the Athlete's heart: an expert consensus of the European Association of Cardiovascular Imaging





European Heart Journal – Cardiovascular Imaging (2015) **16**, 353

Gold standard : volumes, masse, fonction
Cardiomyopathies
Fibrose…



Coronaires
Aorte
… poumons, médiastin…

Réserve contractile
Ischémie?
Valves,
Arythmie d’effort…

Le dopage

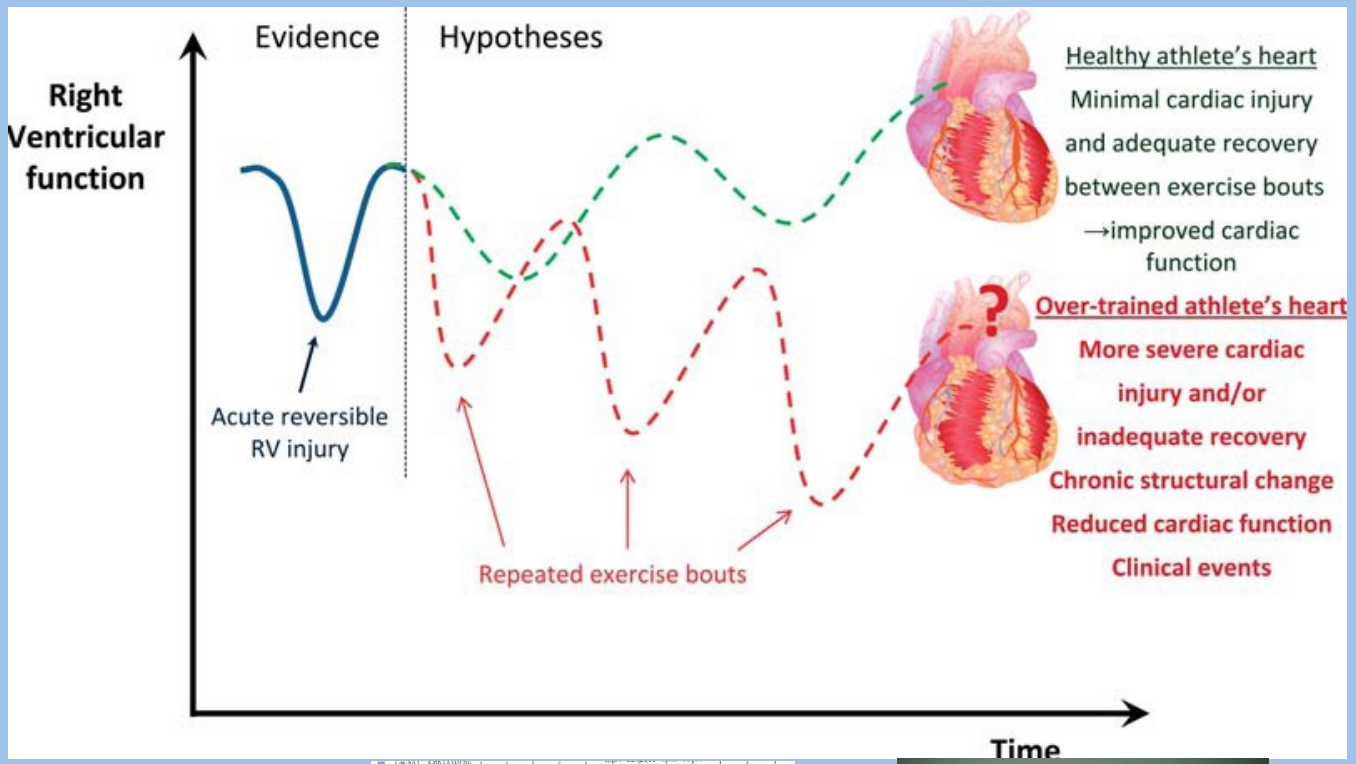
Table 2 **Power of cardiac imaging for suspected diagnosis performance-enhancing substances involvement**

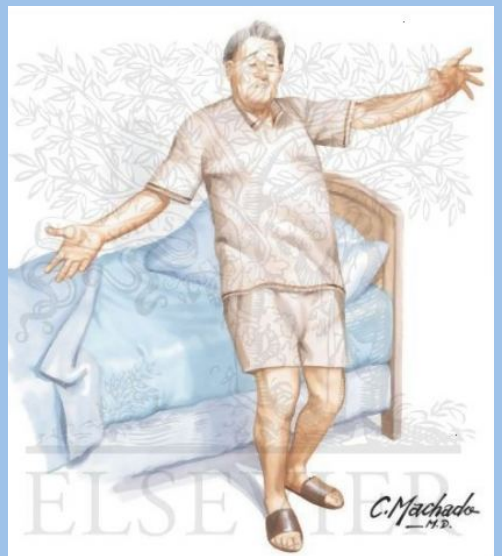
Prohibited substances	Cardiac effect	Demonstration by cardiac imaging
Anabolic androgenic substances	LVH, LV diastolic dysfunction, reduction of EF, reduction of longitudinal strain	+++
Non-steroids anabolic agents (B2 agonists, peptide hormones, growth factors, recombinant human erythropoietin)	LVH, LV diastolic dysfunction	+
Stimulants (amphetamines, methamphetamine and cocaine)	Acute and chronic CAD, Aortic dissection	++

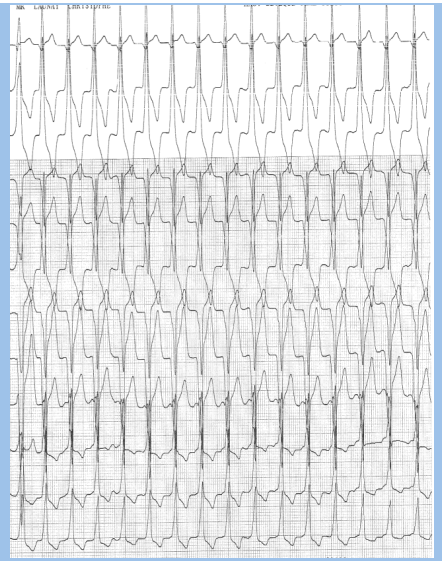
+++, largely demonstrated; ++, well demonstrated; +, poorly demonstrated.

European Heart Journal – Cardiovascular Imaging (2015) **16**, 353

Le surentrainement

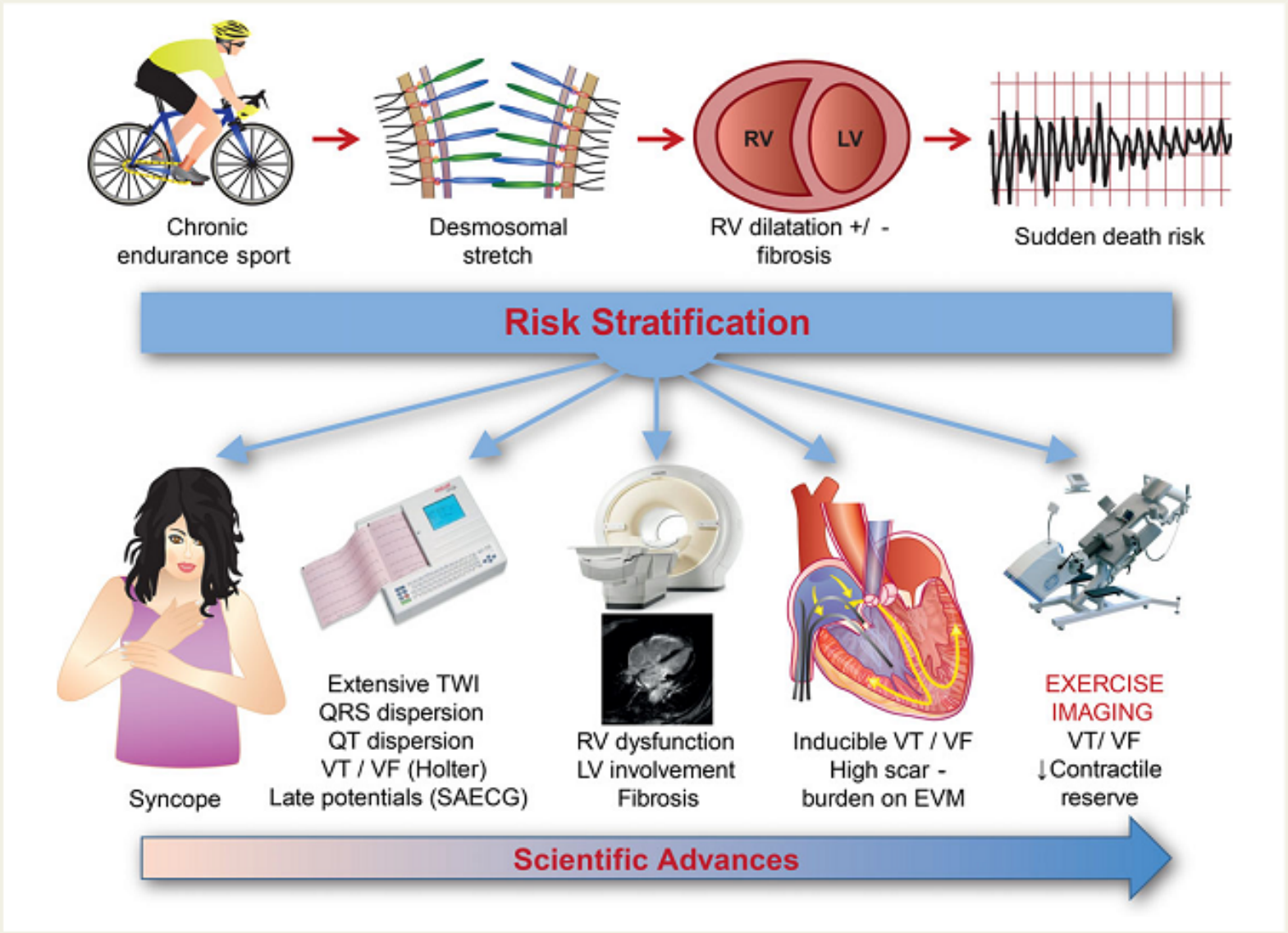




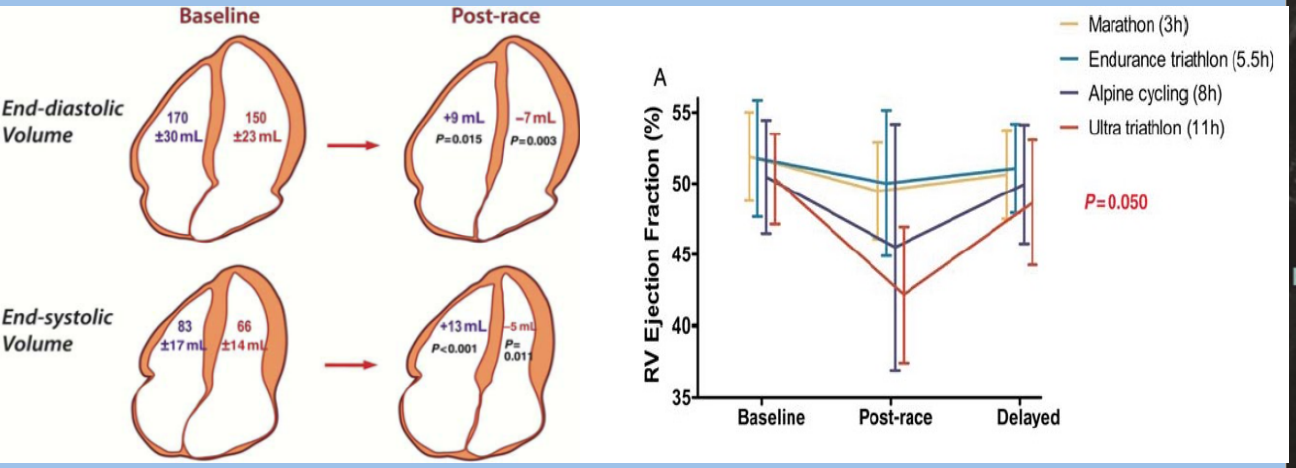


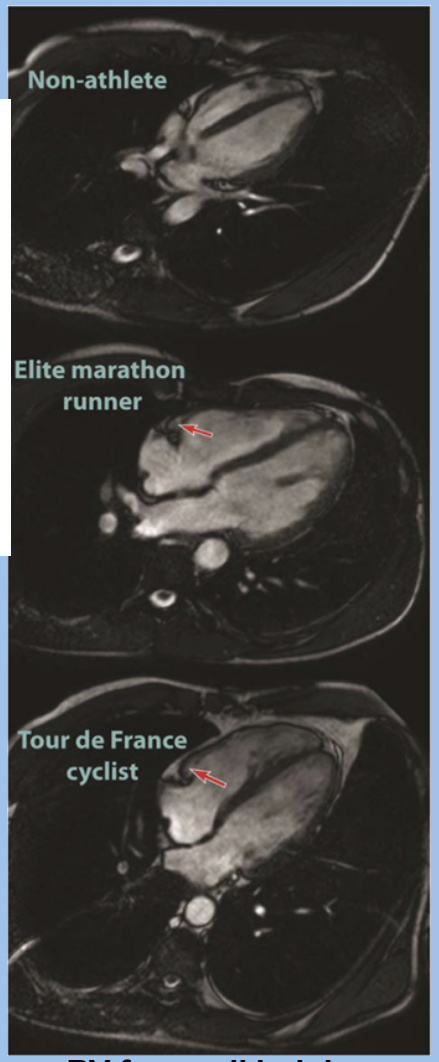
Arrhythmogenic right ventricular remodelling in endurance athletes: Pandora's box or Achilles' heel?

European Heart Journal (2015) **36**, 1955–1957



DAVD induite par l'effort: mythe ou réalité?





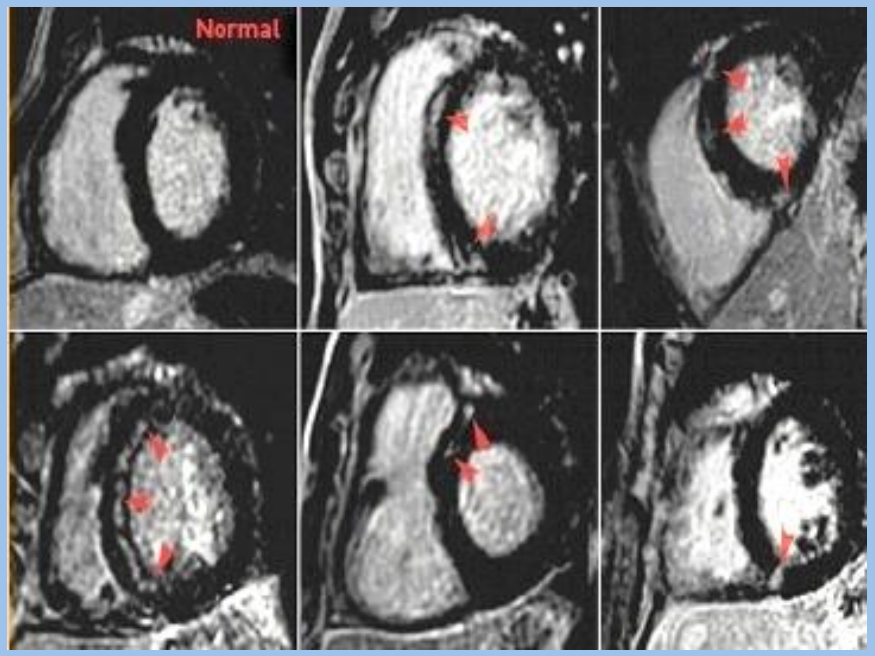
RV free wall bulging

- **27% des athlètes endurants avec des arythmies ventriculaires avaient les critères d'une DAVD**

- **12% ont des mutations des gènes du desmosome (athlète endurant avec arythmies ventriculaires complexes originaires du VD)**

Scherr et al. Med Sci Sports Exerc 2011;43:1819-27.
Prior. Heart 2012;98:947-55.
Ector J et al Eur Heart J 2007;28:345-53.
La Gerche. Eur Heart J 2012; 33:998-1006.
La Gerche A. Heart 2010;96:1268-74.

Fibrose myocardique induite par l'effort?



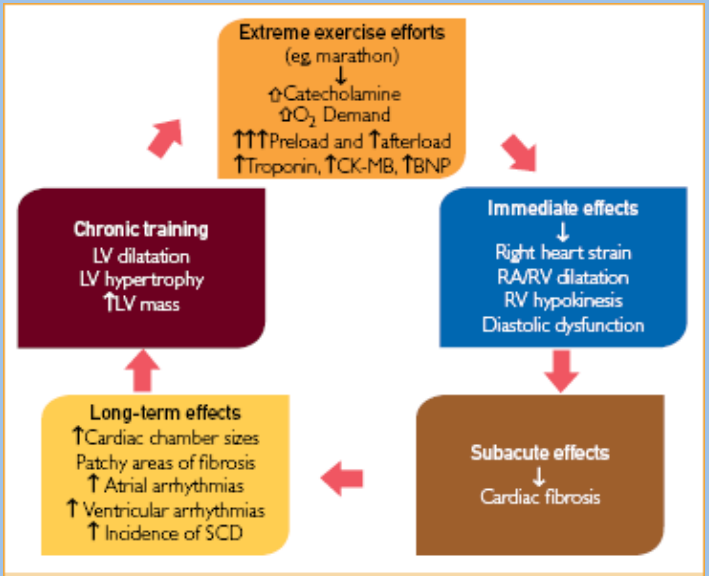
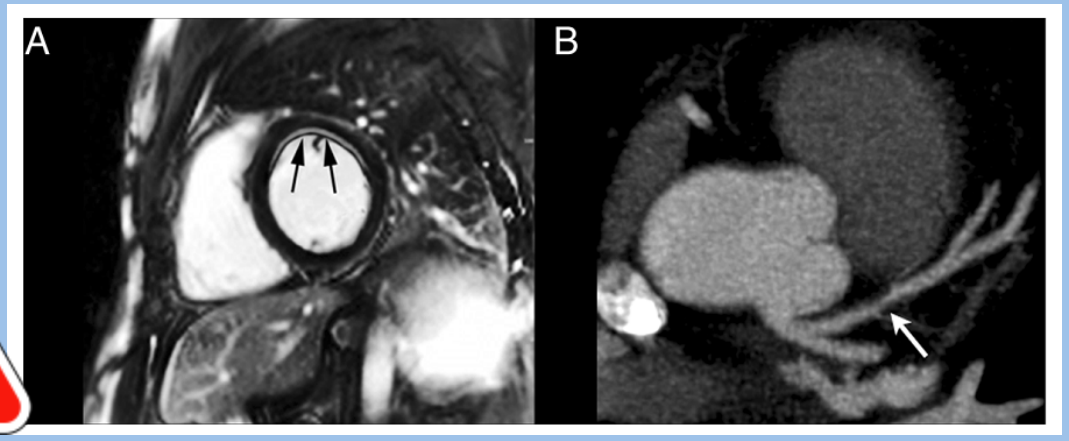


FIGURE 7. Proposed pathogenesis of cardiomyopathy in endurance athletes. BNP = B-type natriuretic peptide; CK-MB = creatine kinase MB; LV = left ventricle; RA = right atrium; RV = right ventricle; SCD = sudden cardiac death.



La Gerche. Eur Heart J 2012; 33:998-1006.
O Keefe et al. Mayo Clin Proc 2012;87:587-95.

Conclusion

- Un examen attentif incluant l'ECG
- Il y aura toujours des accidents imprévisibles (anomalies de naissance coronaires…)
- Large accès public à la défibrillation

Les situations à risque…

ECG Screening

Normal athlete's heart	Abnormal finding
Sinus bradycardia	T wave inversion
First degree AV block	ST segment depression
Incomplete RBBB	Pathological Q waves
Early repolarisation	Left atrial enlargement
Isolated QRS voltage criteria for LVH	Left axis deviation/left anterior fascicular block
	Right axis deviation/left posterior fascicular block
	Right ventricular hypertrophy
	Ventricular pre-excitation
	Complete LBBB or RBBB
	Long or short QT interval
	Brugada-like early repolarisation

…60% of cardiac diseases

Critères ECG d'HVG (Se 10 à 50%...)

SV1 + RV5 ou V6 > 35 mm : c'est **l'index de Sokolow-Lyon** qui est le plus utilisé chez les sujets de plus de 35 ans. Chez les sujets plus jeunes, en dessous de 35 ans, on exige une amplitude > 45 mm. Certains utilisent V2 : c'est le critère de Sokolow modifié qui donne plus de faux positifs;
(RI - RIII) + (SIII - SI) > 17 mm **: indice de Lewis;**
RI + SIII > 25 mm : **indice de Gubner**;
SV2 + RV7 >35 mm : **indice de Blondeau et Heller**;
Onde R maximale + onde S maximale dans les précordiales > 45 mm;
Amplitude des ondes R ou S prises isolément (sensibilité médiocre) :RV5 ou V6 >26 mm, SV1 >24 mm, SV2 >25 mm, RaVL >11 mm, RaVF >20 mm.

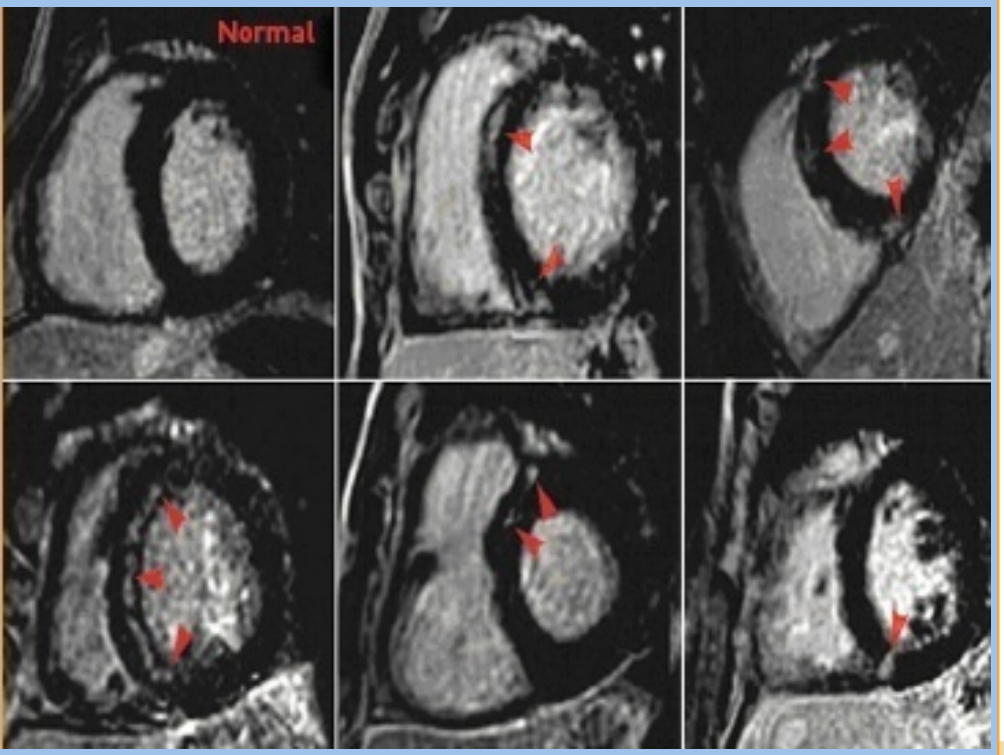
Parmi les critères plus récents, on trouve :
RaVL + SV3 >28 mm chez l'homme, > 20 mm chez la femme : **indice de Casale ou de Cornell;**
Somme des voltages QRS dans les 12 dérivations(61);
Durée de QRS x indice de Cornell(62).

Romhilt et Estes ont proposé un "Point score system" où les diverses modifications possibles produites par une HVG sont regroupées dans un score global :

Une HVG certaine est diagnostiquée lorsque le score est > 5 points; 4 points indiquent une HVG probable.

Voltage a) en périphériques, R ou S max > 20 mm, ou b) en précordiales SV1 ou V2 max > 30 mm , ou RV5 ou V6 max > 30 mm	3 points
Anomalies secondaires de repolarisation de type surcharge a) sans digitale b) avec digitale.	3 points 1 point
Hypertrophies auriculaire gauche (à l'exception de la sténose mitrale) : Indice de Morris positif	3 points
Déviation axiale gauche d'au moins -30º	2 points (sauf si bloc fascciculaire antérieur gauche)
Durée de QRS > 0,09 seconde	1 point
Déflexion intrinsécoïde en V5,V6 > 0,05 seconde	1 point

Exercise induced cardiomyopathy?



10 to 50% of healthy marathon runners… > 50Y.

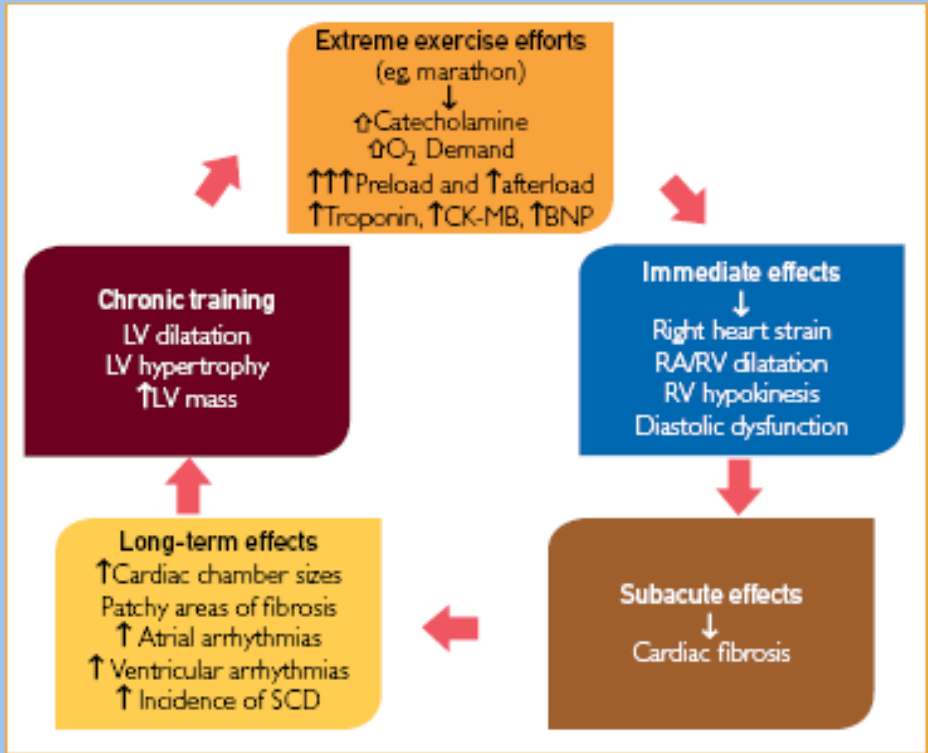


FIGURE 7. Proposed pathogenesis of cardiomyopathy in endurance athletes. BNP = B-type natriuretic peptide; CK-MB = creatine kinase MB; LV = left ventricle; RA = right atrium; RV = right ventricle; SCD = sudden cardiac death.

Breuckmann et al. Radiology. 2009;25:50-57.

La Gerche. Eur Heart J 2012; 33:998-1006.

O Keefe et al. Mayo Clin Proc 2012;87:587-95.

What about young women athletes?

- Sex Ratio of SCD during exercise :
  - 1/5 to 1/30!
  - Less premature CAD
- Normal heart (arrhythmic SCD…) > 50%
  - Afro-American > White recruits (RR 10.2).
  - Impact of exercise in regular and elite athletes (MRI) :
    - LV EDV > 105 ml/m², RV EDV > 110 ml/m².
    - RV EDD> 50 mm, LV EDD > 60 mm in 16% (short-axis)
- Higher Survival at hospital admission (46.5 vs 30%)

Eckart RE. Am J Cardiol 2006; 97:1756-8

Marijon E et al. Circ Arrhythm Electrophysiol. 2013;6:1185-1191.

Kim et al. NEJM 2012;366:130-40.

Prakken NH. Eur J Cardiovasc Prev Rehabil. 2010

Safety of Sports for Athletes With Implantable Cardioverter-Defibrillators

Results of a Prospective, Multinational Registry

Table 4. Bivariate Association of Clinical Characteristics and Appropriate Shocks During Sports

	Total, n	VT/VF Shock During Competition/ Practice (n= 21), n (%)	*P* Value
Age, 10–19 y	89	1 (1)	0.03
20–60 y	283	20 (7)	
Competitive subgroup	60	1 (2)	0.22
Others	312	20 (6)	
Indication			
Secondary prevention	155	10 (6)	0.65
Primary prevention	217	11 (5)	
Cardiac diagnosis			
Idiopathic VT/VF (normal heart)	33	4 (12)	<0.01
CAD	39	3 (8)	
ARVD	53	8 (15)	
HCM	65	1 (2)	
LQTS	73	2 (3)	
Others	109	3 (3)	
Sex			
Female	123	10 (8)	0.16
Male	249	11 (4)	
β-Blocker			
Yes	229	14 (6)	1.00
No	115	7 (6)	

ARVD indicates arrhythmogenic right ventricular dysplasia; CAD, coronary artery disease; HCM, hypertrophic cardiomyopathy; LQTS, long-QT syndrome; VF, ventricular fibrillation; and VT, ventricular tachycardia.

77 individuals/315: 121 Shocks

48 (13%): appropriate

40 (11%) : inappropriate

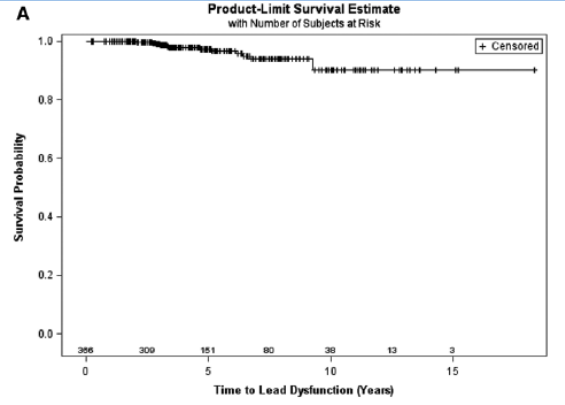


Table 6. Number of Shock Events and of Individuals Receiving Shocks, Competitive Subgroup (Varsity/Junior Varsity/Traveling Team)

Rhythm	Competition Related, n (%)*	Physical Activity Related, n (%)†	Other, n (%)	Total, n (%)
Ventricular tachycardia	0/0	4/2 (3)	2/1 (2)	6/3 (5)
Ventricular fibrillation	2/1 (2)	2/2 (3)	2/1 (2)	6/4 (7)
Sinus tachycardia	0/0	0/0	0/0	0/0
Atrial fibrillation	1/1 (2)	2/2 (3)	0/0	3/3 (5)
Other supraventricular tachycardia	1/1 (2)	1/1 (2)	0/0	2/2 (3)
Noise	0/0	1/1 (2)	2/2 (3)	3/3 (5)
T-wave oversensing	1/1 (2)	1/1 (2)	1/1 (2)	3/3 (5)
Other	1/1 (2)	1/1 (2)	0/0	2/2 (3)
Total	6/5 (8)	12/10 (17)	7/5 (8)	25/17 (28)

Lampert et al. Circulation 2013;2021-30.

Congenital heart diseases

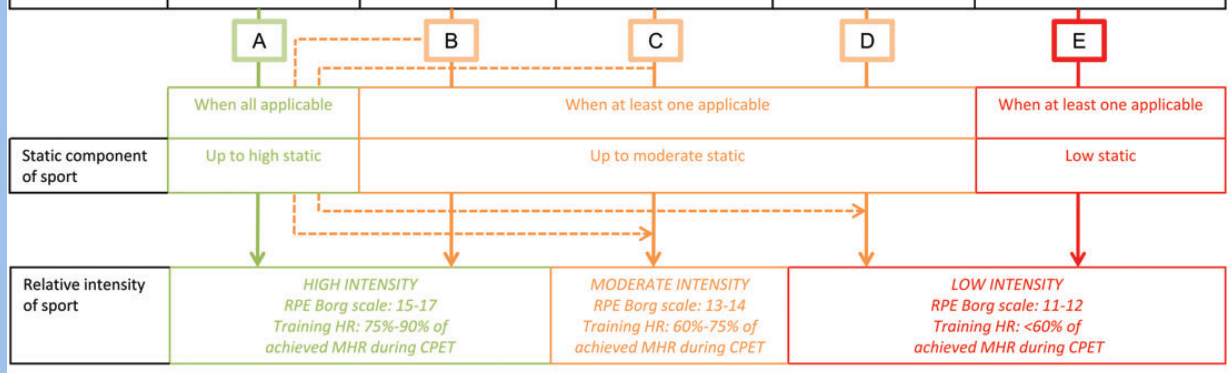
- Small or repaired septal defect
- Operated PDA
- Repaired coarctation
- Treated pulmonary stenosis
- Mild moderate aortic stenosis

TOF

TGA (switch)

- Eisenmenger Sd
- Secondary Pulmonary Hypertension
- Univentricular hearts
- Congenitally, or Mustard, Senning, Rastelli Corrected TGA

1. Ventricles	No systolic dysfunction No hypertrophy No pressure load No volume load	No systolic dysfunction No hypertrophy Mild pressure load Mild volume load	Mild systolic dysfunction Mild hypertrophy Single ventricle physiology Systemic right ventricle	Moderate systolic dysfunction Moderate hypertrophy Moderate pressure load	Severe systolic dysfunction Severe hypertrophy Severe pressure load Moderate/severe volume load
2. Pulmonary artery pressure	Low pulmonary artery pressure	Low pulmonary artery pressure	Mildly elevated pulmonary artery pressure		Moderately/severely elevated pulmonary artery pressure
3. Aorta	No/mild dilatation	Moderate dilatation	Severe dilatation	Dilatation approaching indication for repair	
4. Arrhythmia	No arrhythmia	No arrhythmia	Mild arrhythmic burden Non-malignant arrhythmia		Significant arrhythmic burden Malignant arrhythmia
5. Saturation at rest/during exercise	No central cyanosis	No central cyanosis	No central cyanosis	Central cyanosis	



Solid lines indicate recommendation ; if option for sports with high static component, reduce intensity (dotted lines)

- Guidelines cover < 1% CHD population
- …Leisure time activities

Pelliccia et al. Eur Heart J 2005;26:1422-45

Budts et al. Eur Heart J 2013; 34:3669-74

ECG habituel

Bradycardie sinusale (≥30/min)

Arythmie sinusale

Rythme atrial ectopique

Rythme jonctionnel

Bloc Atrio Ventriculaire de premier degré (espace PR> 200 ms)

Bloc Atrio Ventriculaire de deuxième degré type Mobitz I (Wenckebach)

Bloc de branche incomplet droit

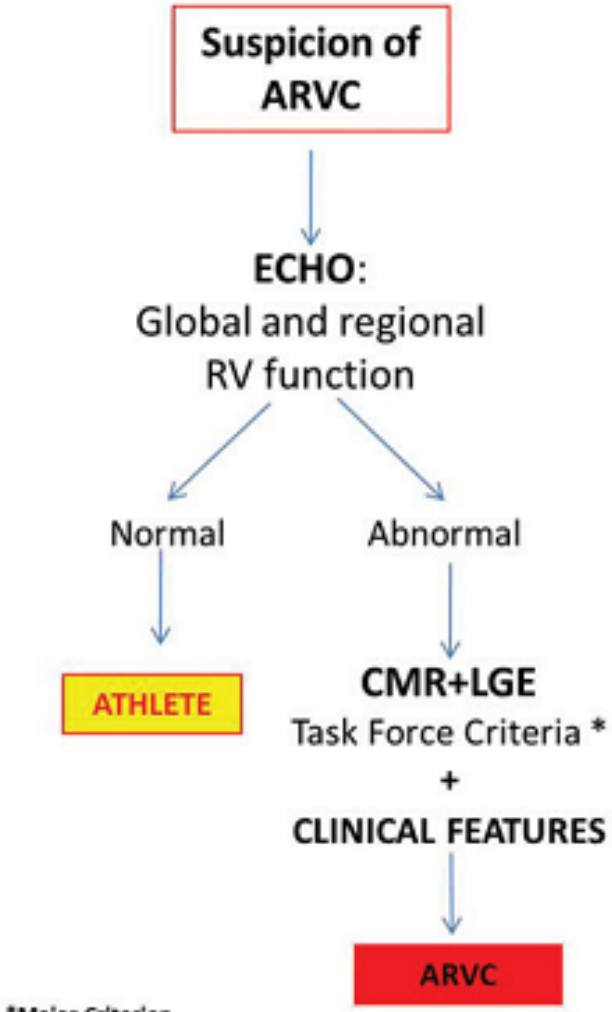
Critère isolé d'hypertrophie ventriculaire gauche (exclue les critères suivants: onde Q, sous décalage du segment ST, ondes T négatives ou pathologiques, déviation axiale gauche, élargissement des ondes P en faveur d'une hypertrophie atriale gauche)

Repolarisation précoce (élévation du ST, élévation du point J, ondes J, crochetage de la partie terminale du QRS).

Segment ST en dôme avec une inversion des ondes T en V1-V4 chez l'athlète noir/Africain.

ECG inhabituel

Inversion des ondes T	>1 mm de profondeur dans au moins 2 dérivations V2-V6, II et vF ou I et vL (exclue V1, vR et III)
Sous décalage de ST	≥0,5 mm de profondeur dans au moins 2 dérivations
Ondes Q pathologiques	>3mm de profondeur ou >40 ms de durée dans au moins 2 dérivations (vR et III exceptés)
Bloc de branche gauche complet	QRS≥120 ms, QRS majoritairement négatif en V1 (QS ou rS) et onde R monophasique en I et V6.
Délai de conduction intra ventriculaire	QRS≥140 ms
Déviation axiale gauche	De -30° à -90°
Elargissement atrial gauche	Onde P>120 ms en I ou II avec portion négative de l'onde P ≥1mm de profondeur et ≥40 ms de durée en V1
Aspect d'hypertrophie ventriculaire droite	R en V1 + S en V5> 10,5 mm ET déviation axiale droite> 120°
Extrasystole ventriculaire **Arythmie ventriculaire**	≥2 ESV / 10 s de tracé ECG Doublets, triplets et tachycardie ventriculaire non soutenue



***Major Criterion**

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

— Ratio of RV end-diastolic volume to BSA ≥110 mL/m2 (male) or ≥100 mL/m2 (fem

— *or* RV ejection fraction ≤40%

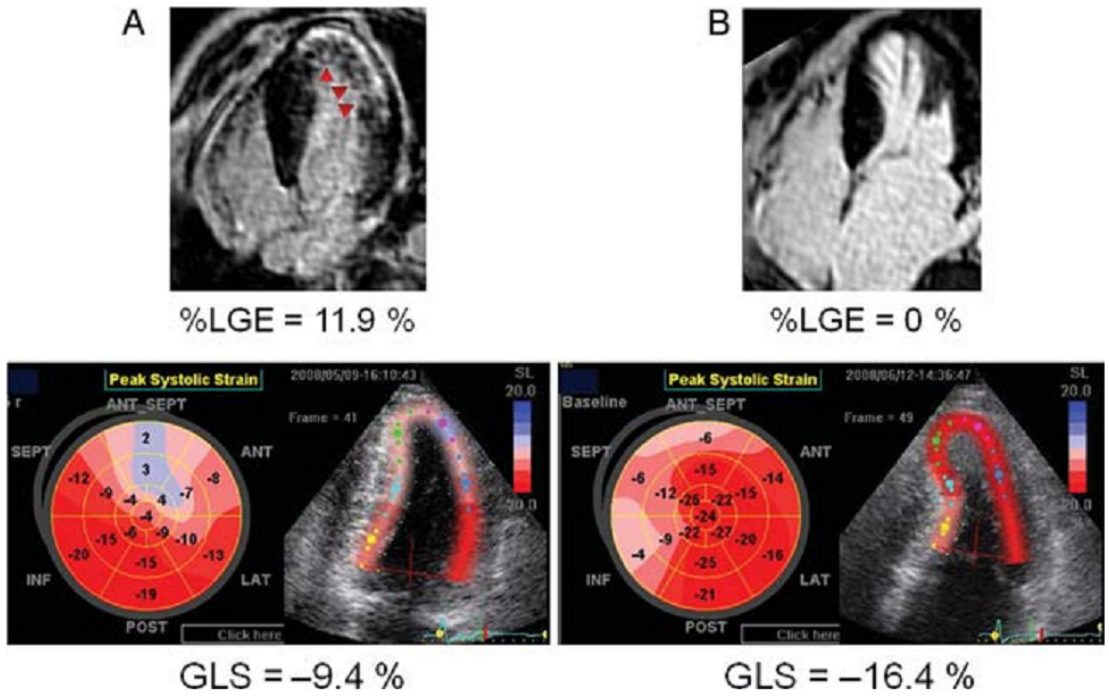
Minor criterion:

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

— Ratio of RV end-diastolic volume to BSA ≥100 to <110 mL/m2 (male) or ≥90 to <100 mL/m

— *or* RV ejection fraction >40% to ≤45%

European Heart Journal – Cardiovascular Imaging (2015) **16**, 353



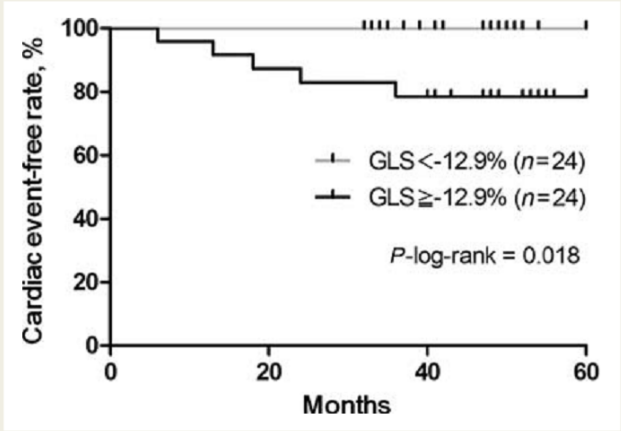
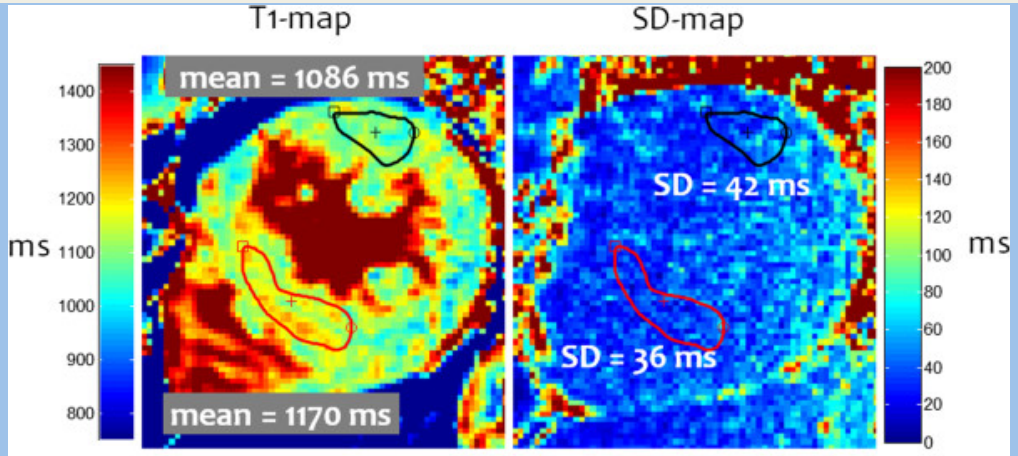


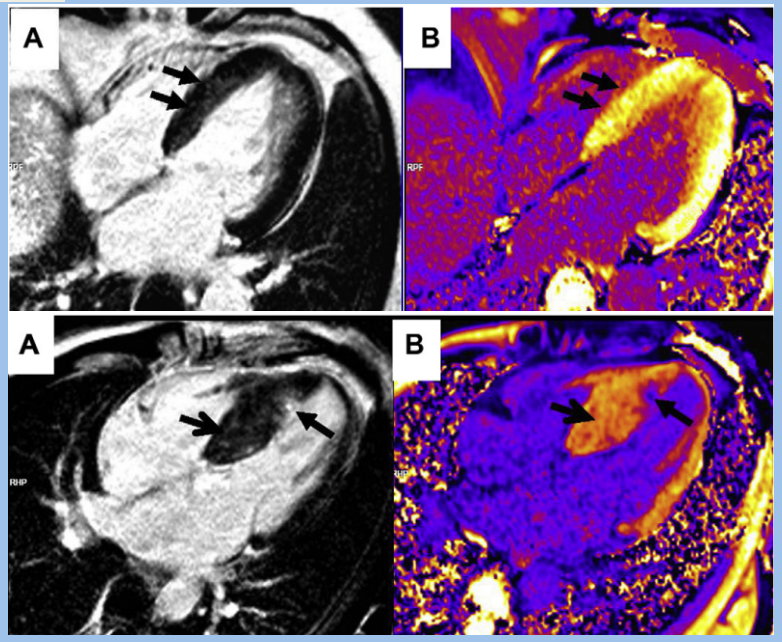
Figure 2 Kaplan–Meier event-free curves of patients with HCM stratified in two groups according to the median level of GLS. HCM, hypertrophic cardiomyopathy; GLS, global longitudinal strain.

European Heart Journal – Cardiovascular Imaging (2012) **13**, 617–623



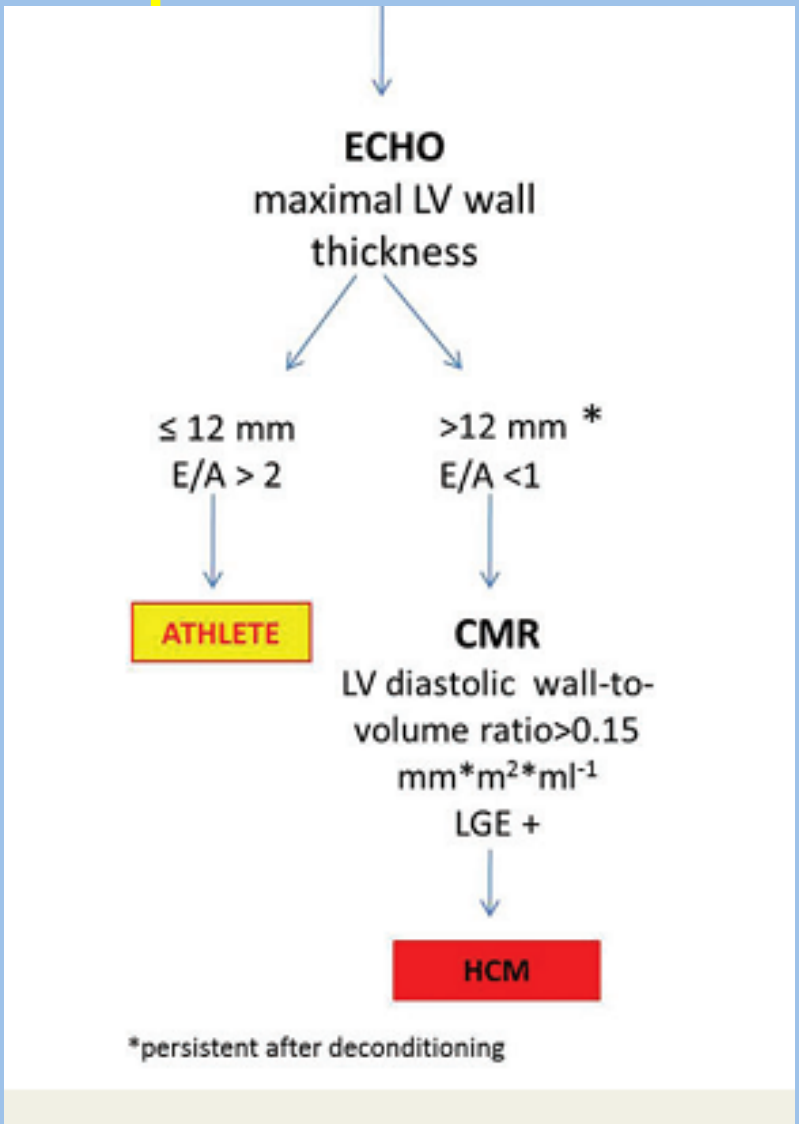
T1 in Septal Region is elevated 84 ms (2.1 SD) using MOLLI 5(3s)3 protocol

JACC: CARDIOVASCULAR IMAGING, VOL. 6, NO. 4, 2013

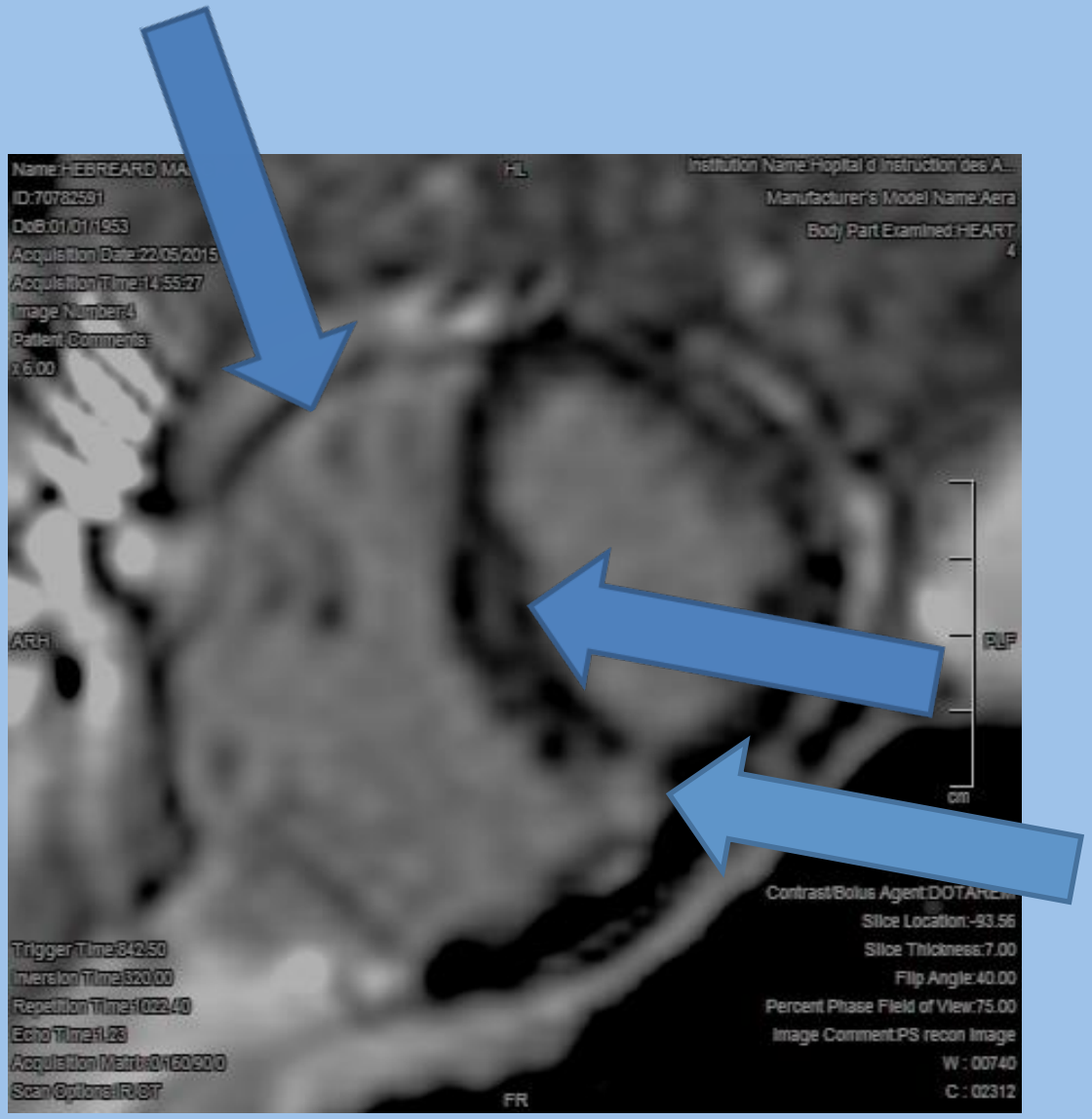


European Journal of Radiology 82 (2013) e225–e231

Suspicion de CMH



European Heart Journal – Cardiovascular Imaging (2015) **16**, 353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