Réadaptation Cardiaque et MINOCA: quel niveau de preuves?

Marie Christine ILIOU

Paris

Conflits d’intérêts

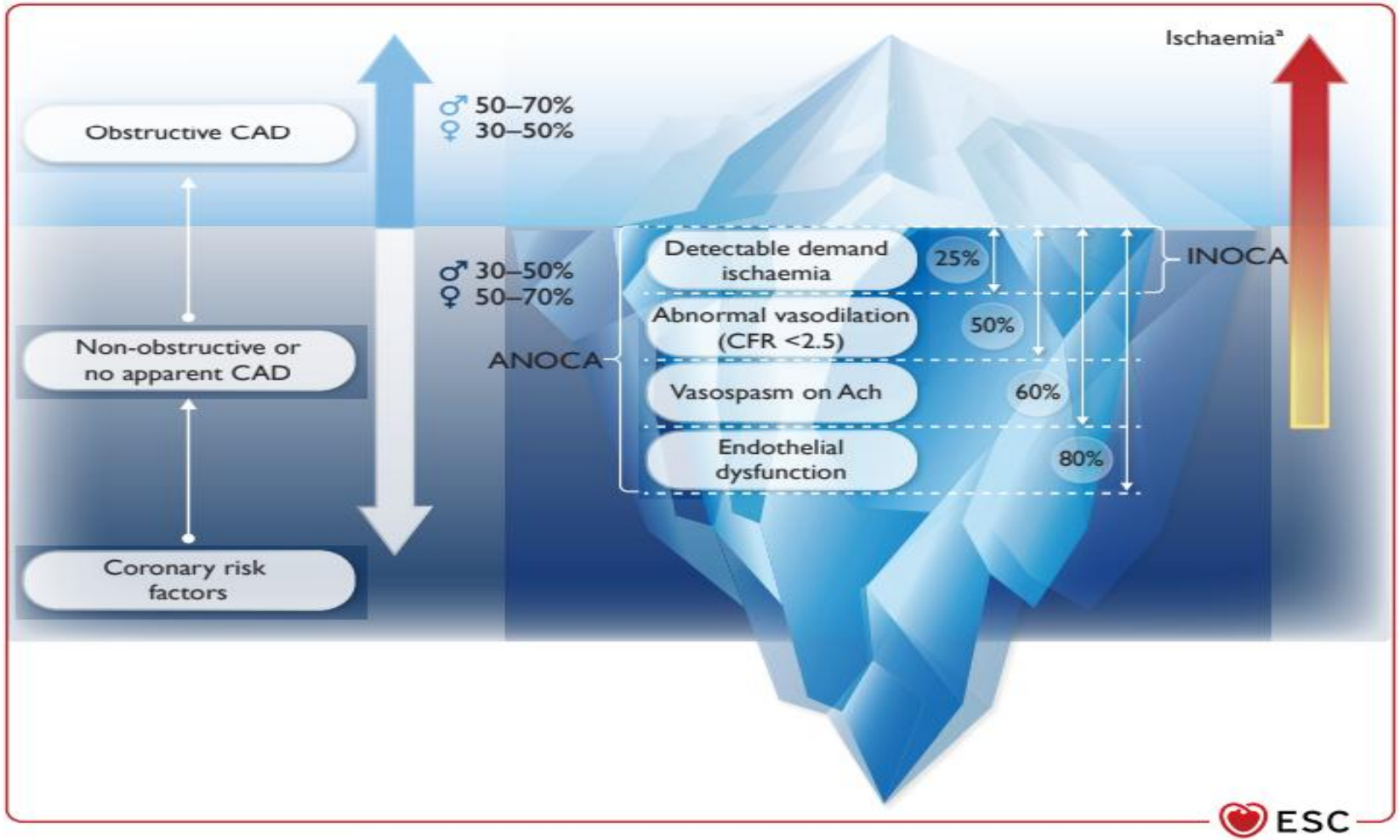
Aucun

NonObstructive Coronary Artery disease

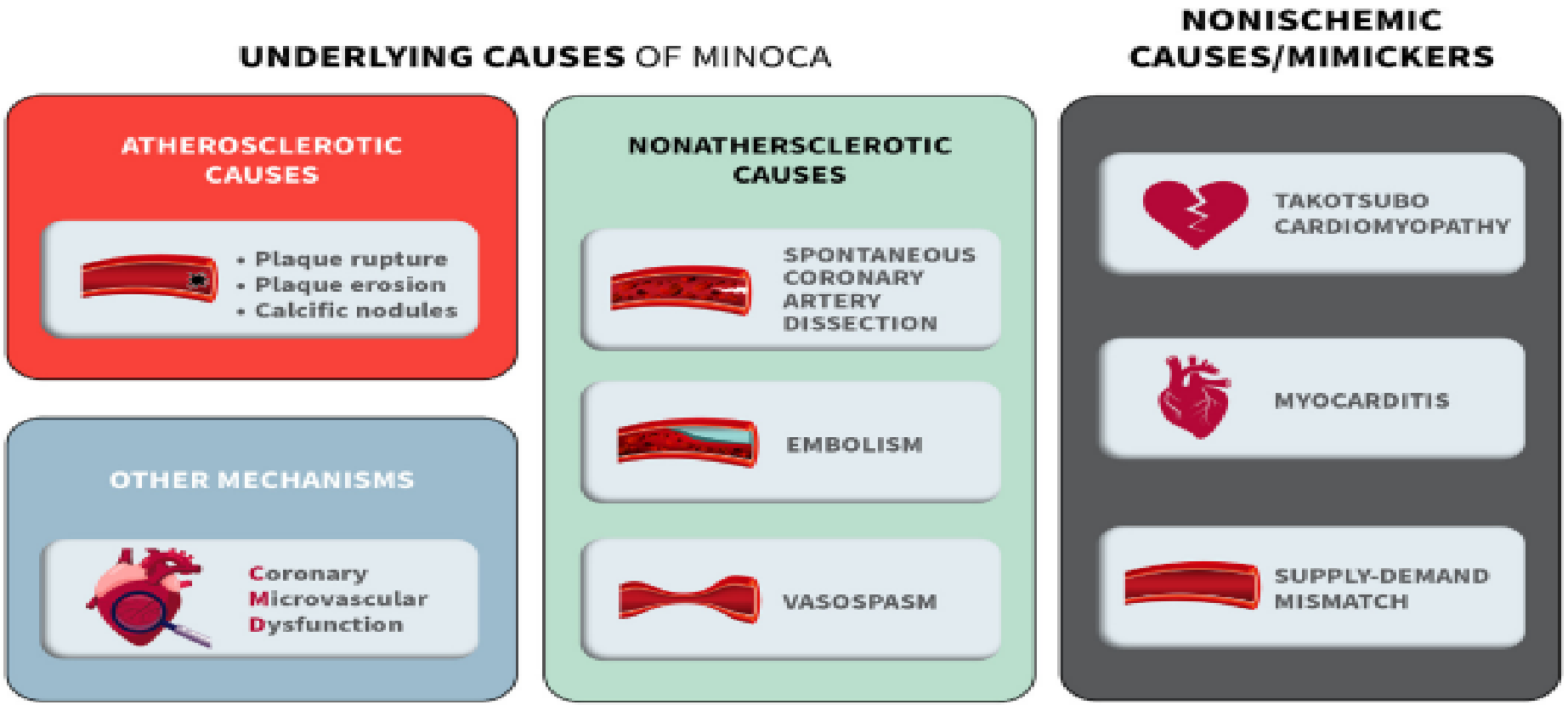
A ngina = **ANOCA**

I schemic= **INOCA**

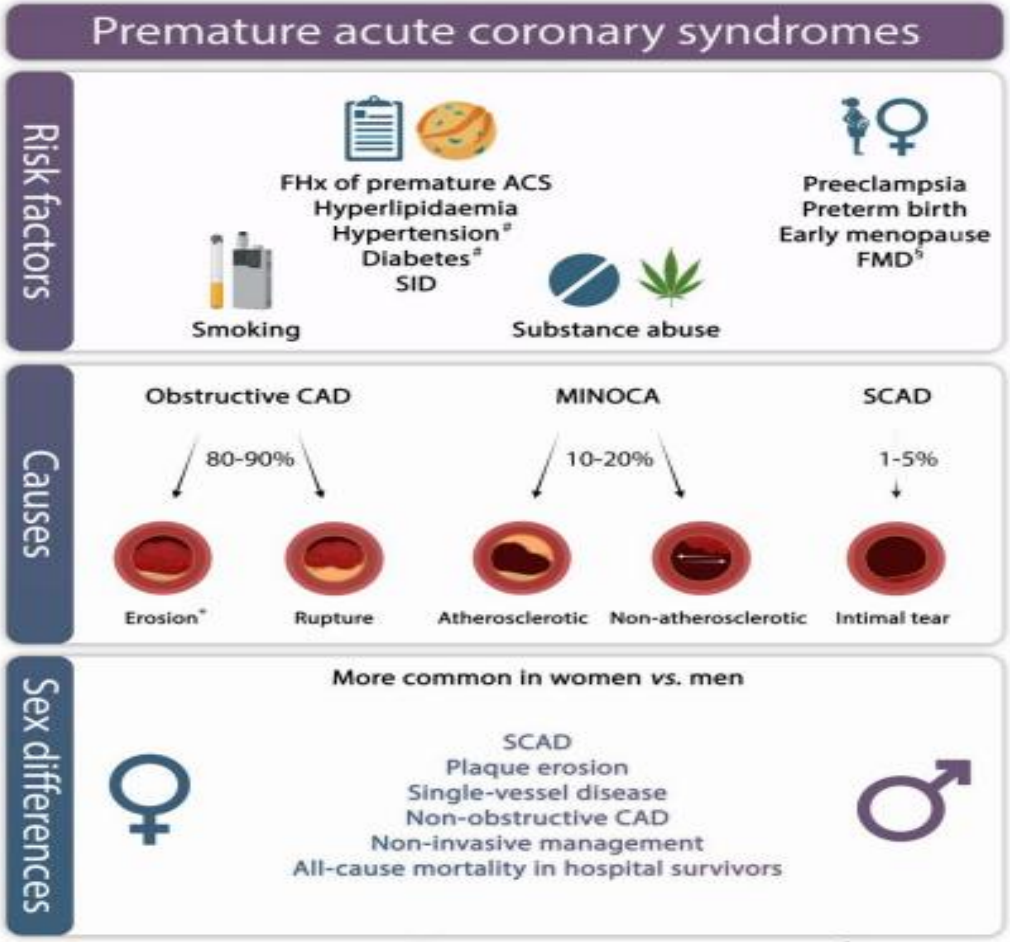
Myocardial **I**nfaction= **MINOCA**



Vrints C. European Heart Journal (2024) 45, 3415–3537



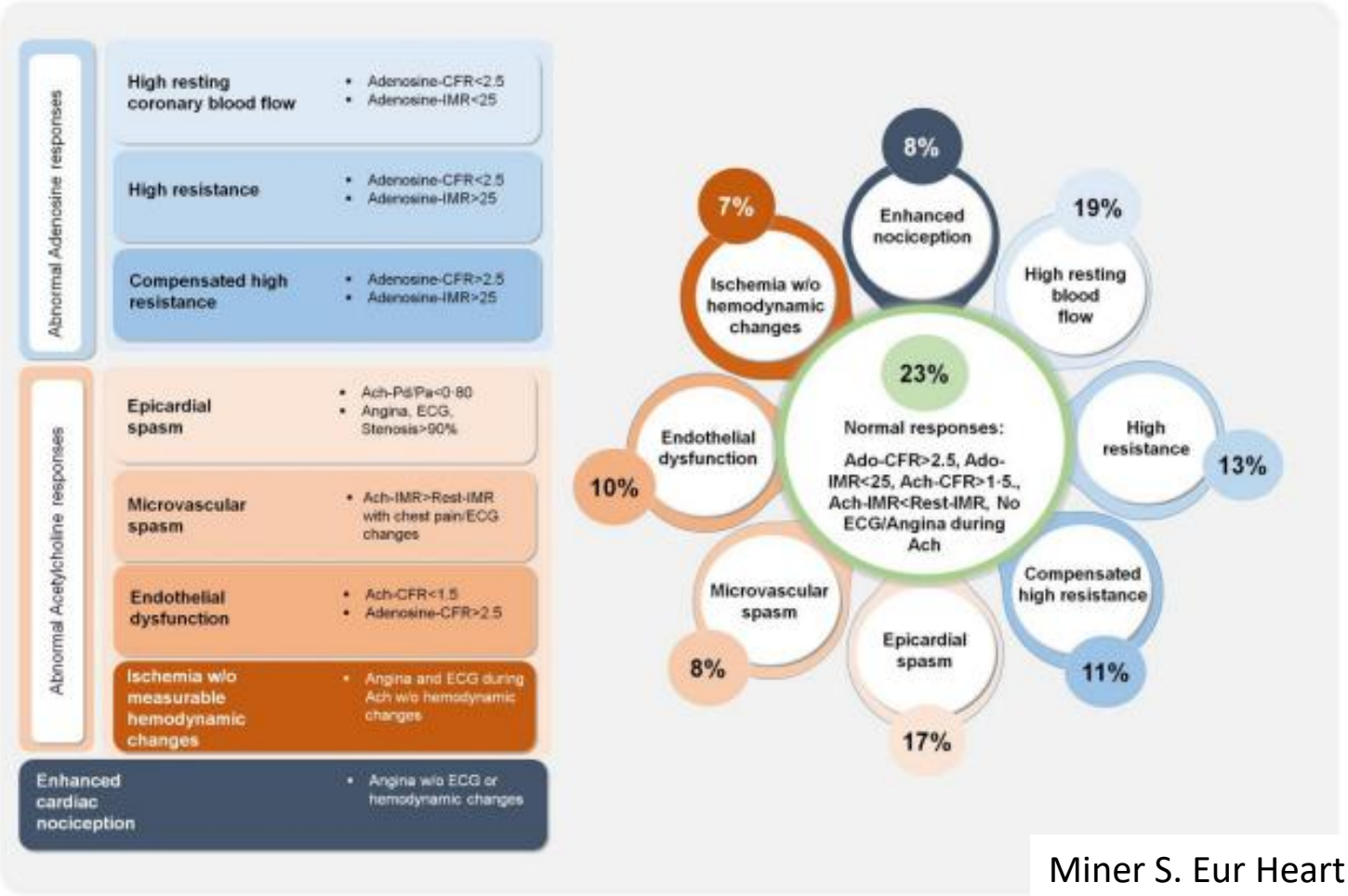
Pacheco C. Can J Cardiol 2024;40:953-68



10 -20 % des SCA

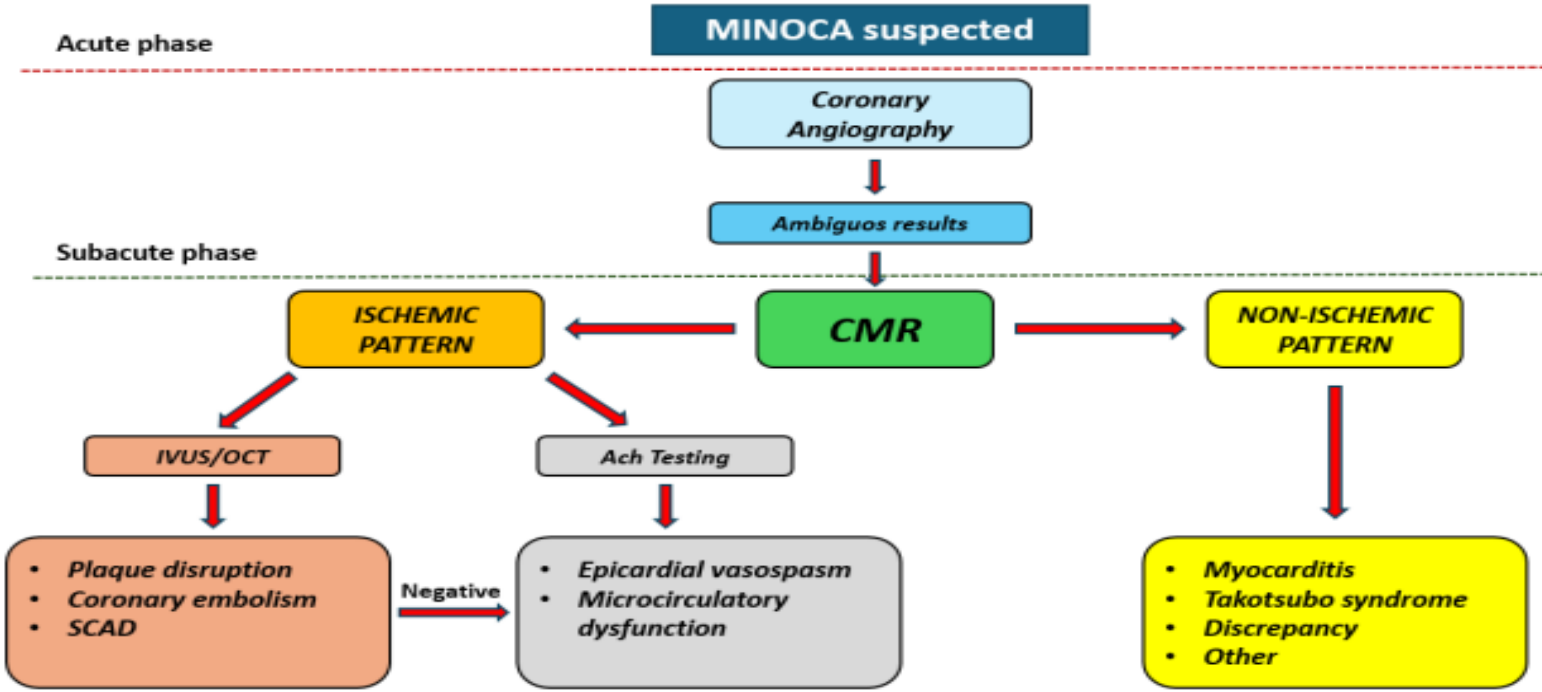
Kraler S. European Heart Journal (2025) 46, 2866–2889

Endotypes of ANOCA



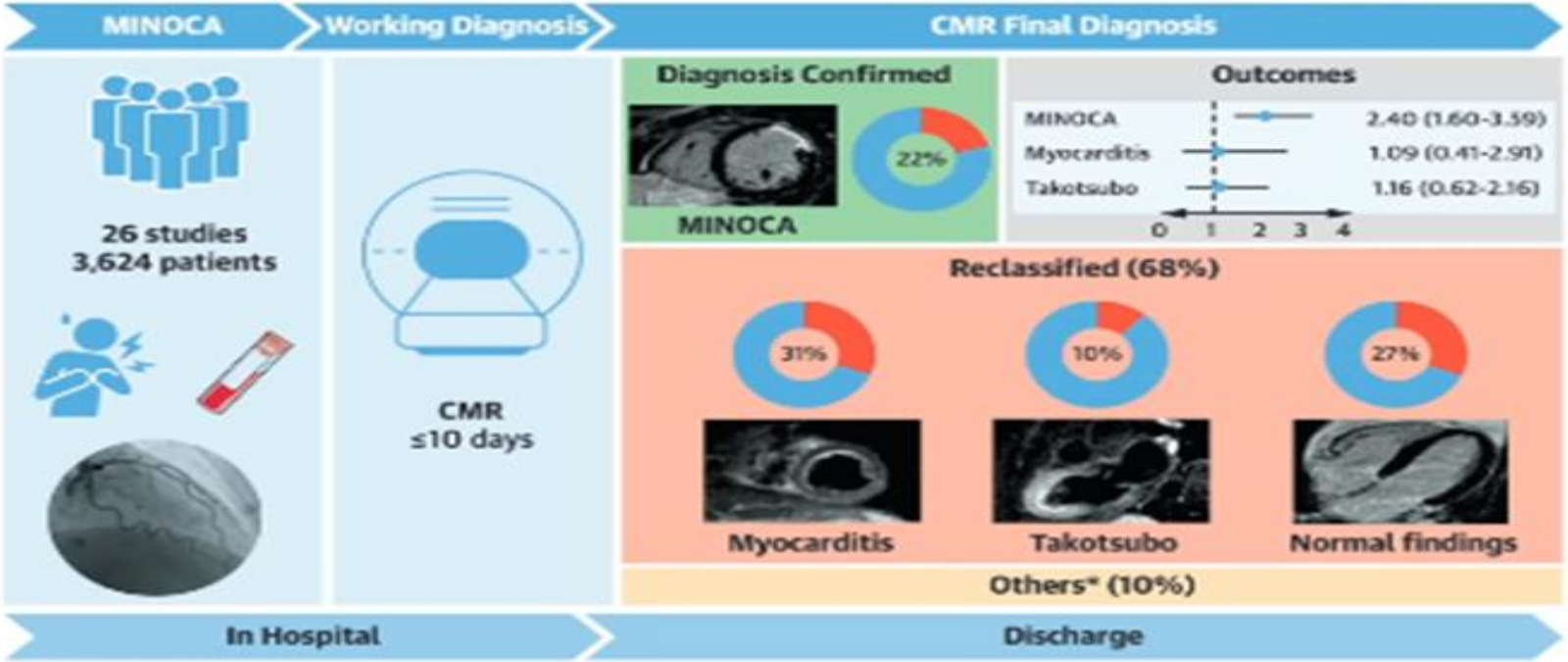
Miner S. Eur Heart J (2026) 47, 594–604

Suspicion MINOCA



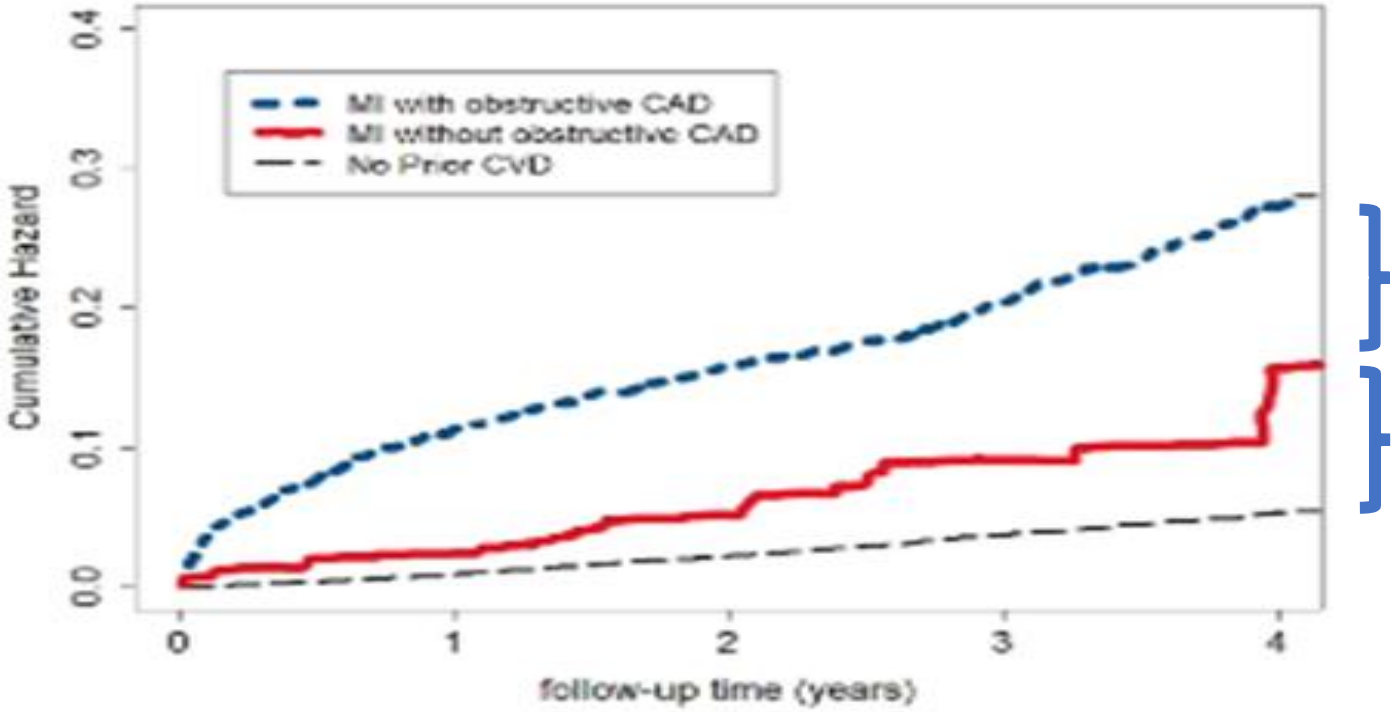
Parlati A. J. Cardiovasc. Dev. Dis. 2025, 12, 64

Pas de Dg = aggravation du pronostic



Mileva N, et al. J Am Coll Cardiol Img. 2023;16(3):376–389.

Mortalité et MACE

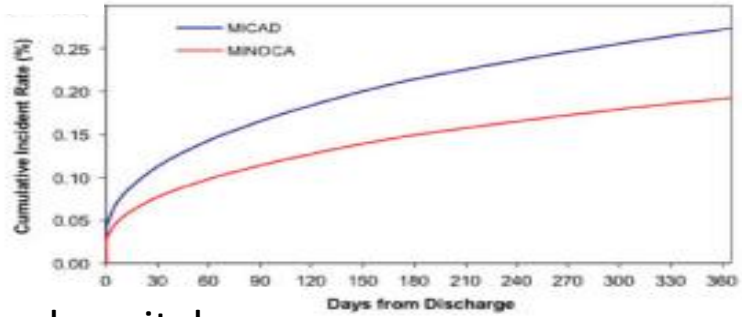


Barr PR; Heart Lung Circ. 2018;27(2):165-174.

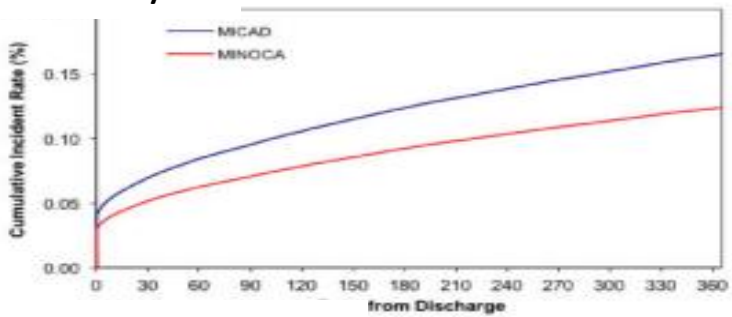
Pronostic USA

276 522 patients SCA,
16 849 (5.9%) had MINOCA.

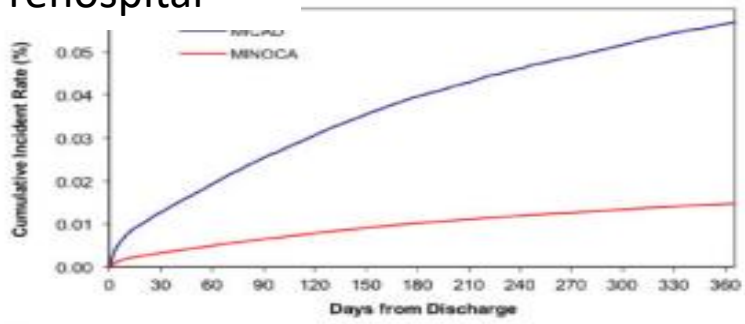
MACE



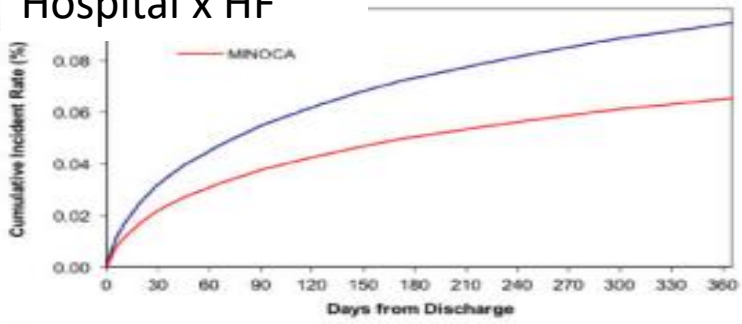
Mortality



MI rehospital

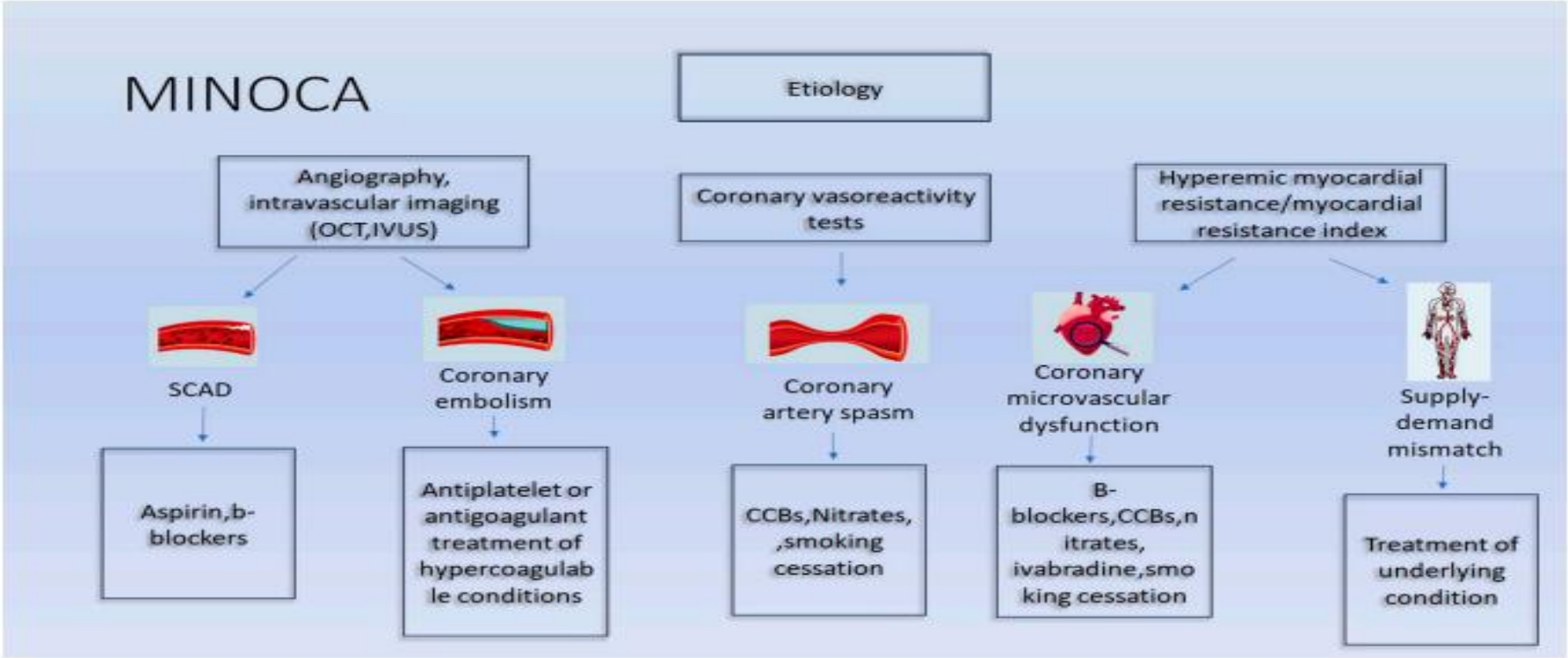


Hospital x HF

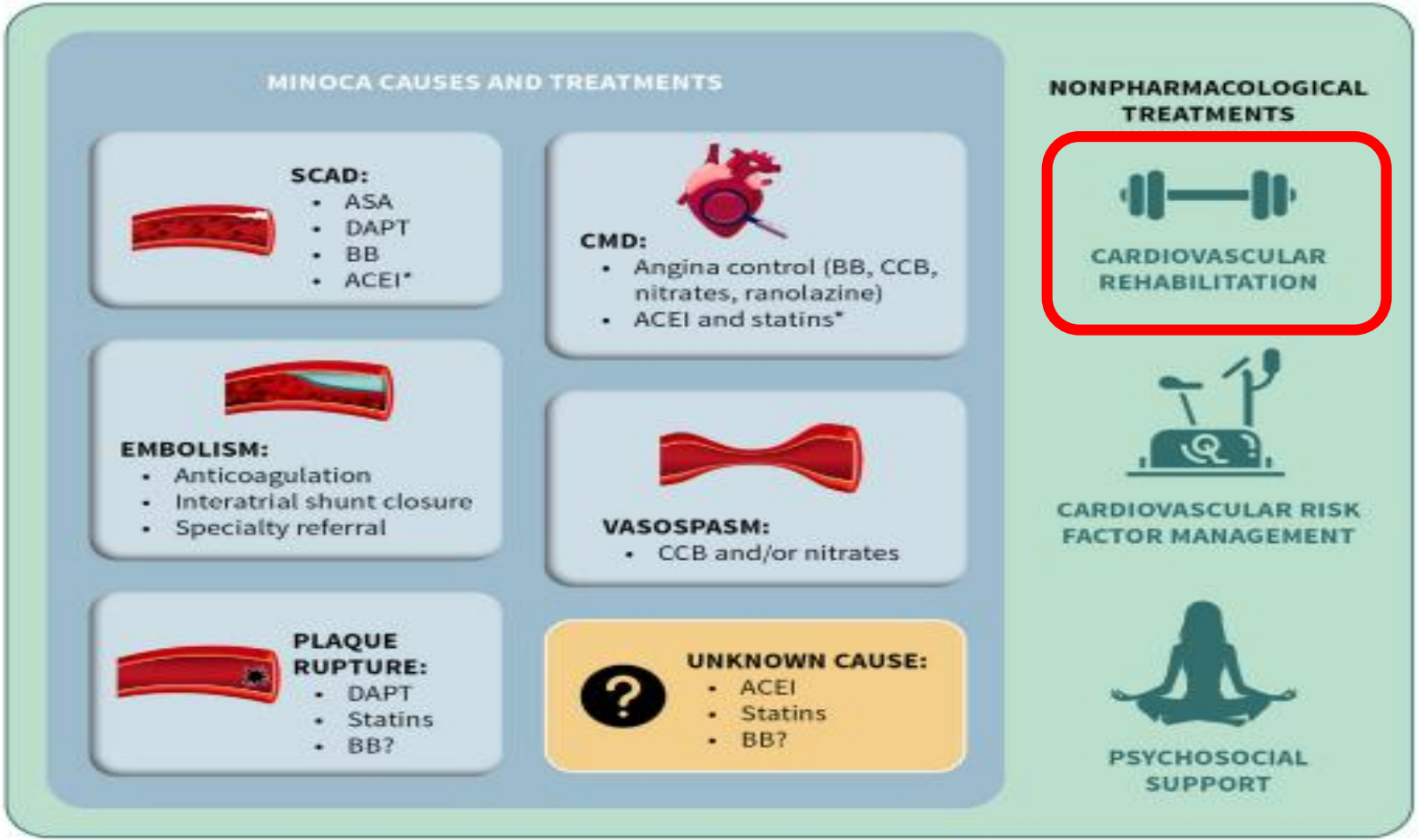


Dreyer R. European Heart Journal (2020) 41, 870–878

ttts MINOCA

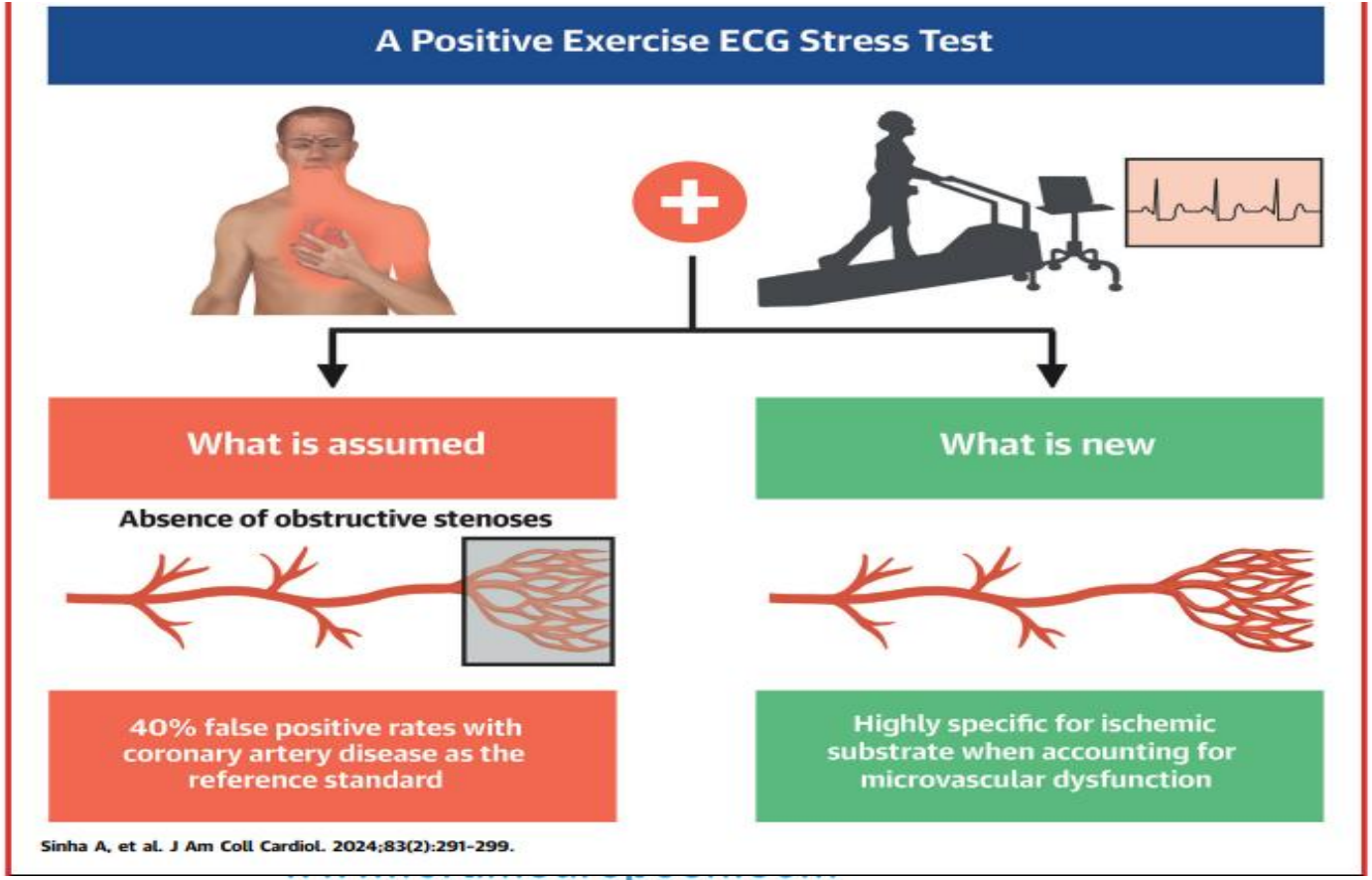


Khattab E. Biomedicines 2024, 12, 2457



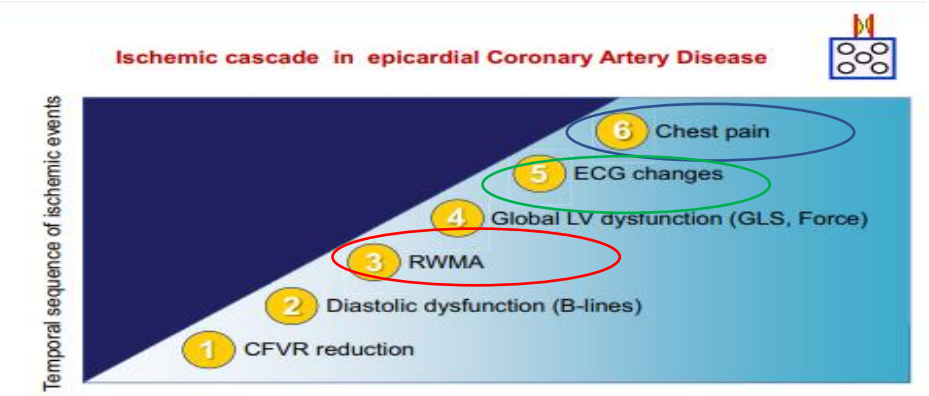
Pacheco C. Can J Cardiol 2024;40:953-68

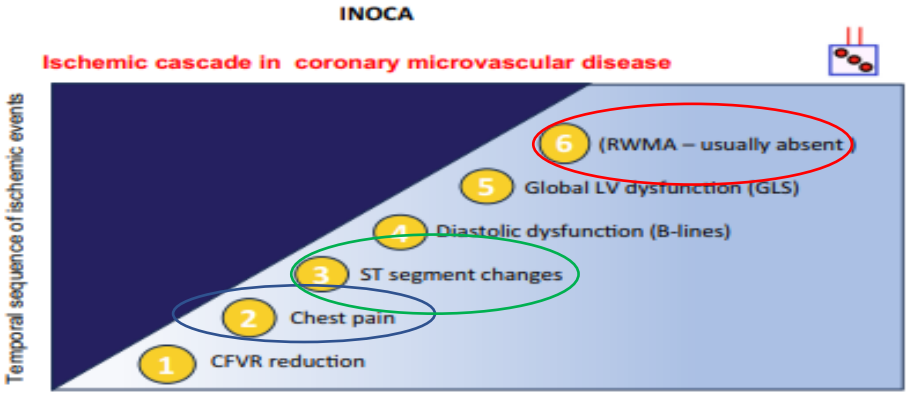
Rethinking False Positive Exercise Electrocardiographic Stress Tests by Assessing Coronary Microvascular Function



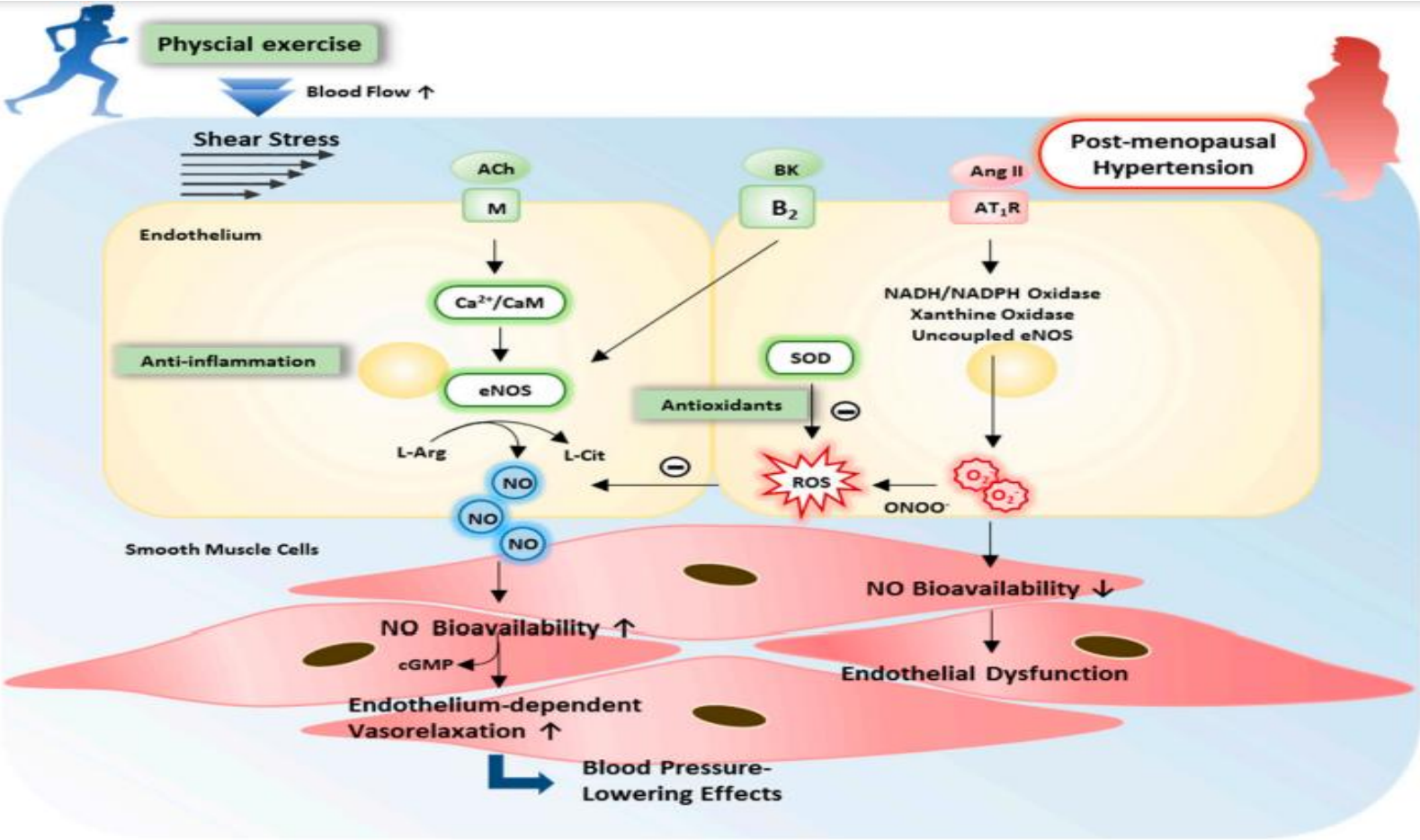
coronary flow velocity reserve

regional wall motion abnormality

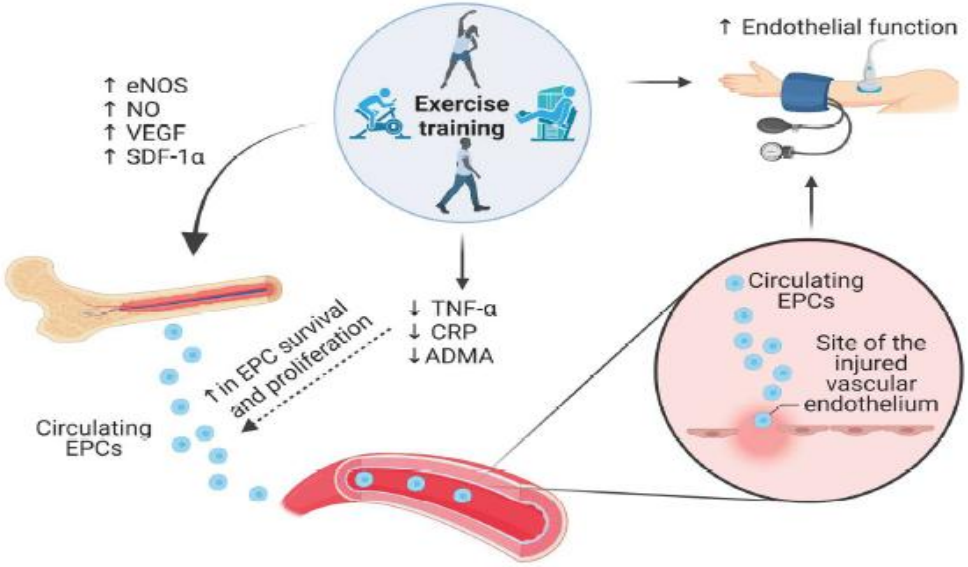




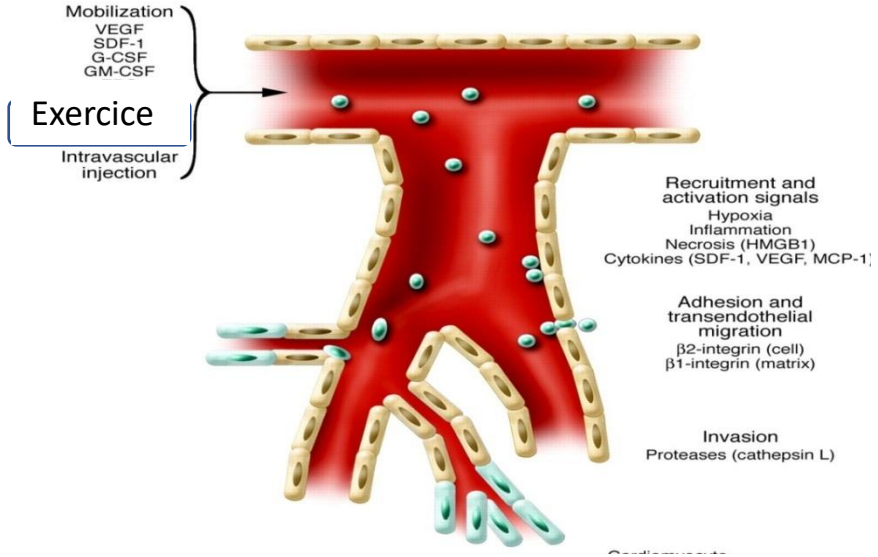
Picano E. Eur Heart J - Cardiovascular Imaging (2024) 25, e65–e90



Int. J. Mol. Sci. 2018, 19, 2523



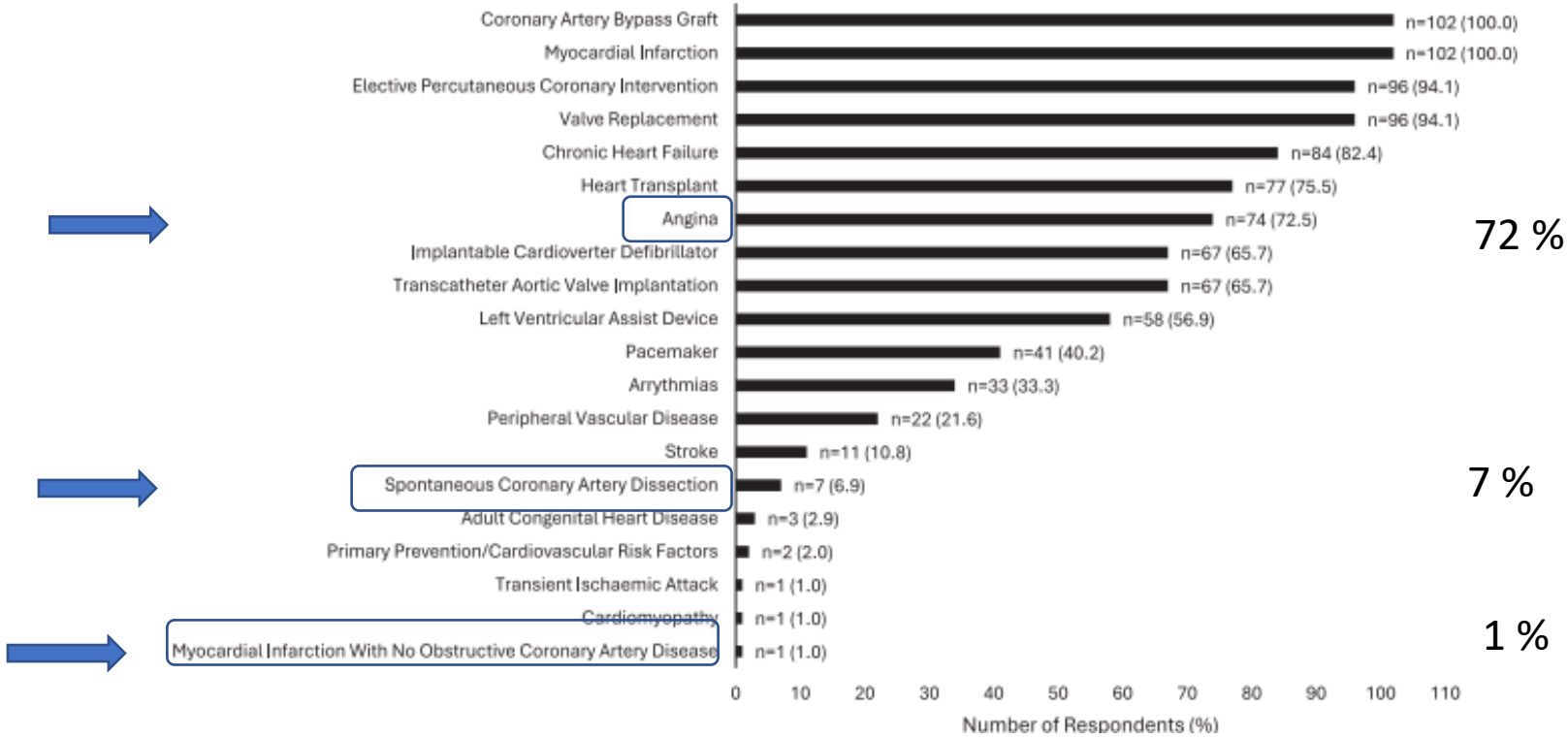
Ferrentinos P. Eur J App Physiol 2022;122:815



Dimmeler S, J Clin Invest 2005;115:572-583

CR survey UK

114 Reponses / 931 professionels de santé en Read Card



72 %

7 %

1 %

Nichols S. Journal of Advanced Nursing, 2025; 0:1–10

Physical Training in Syndrome X

Physical Training Counteracts Deconditioning and Pain in Syndrome X

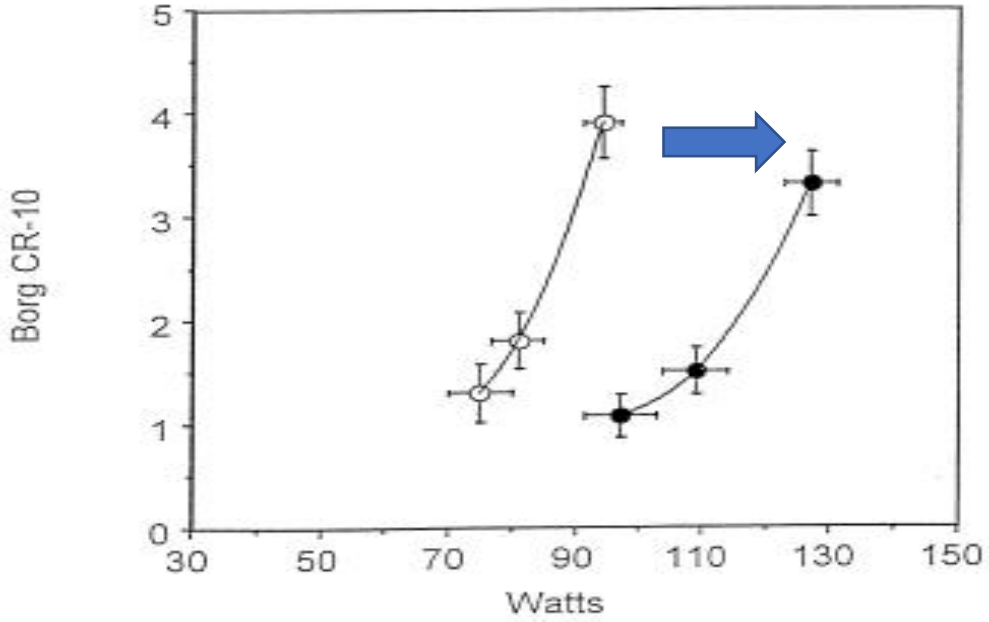


Figure 2. Pain response to increased workload in the training groups (A + B) before **(open dots)** and after training **(solid dots)**.

Erikson B. JACC 2000;36 (5)

Entrainement Physique

	n	programme	Results
Erikson (2000) **R**	26 femmes	8 semaines Relaxation + 8 ExT vs ExT vs control	↑ Delai de angor Améliore fon endothel ↑ Pic VO2 30%
Asbury (2008) **R**	64 femmes	8 semaines Ex vs relaxation	↑ QOL (HAD) ↑ Shuttle walk test + 29 %
Feizi (202) **R**	40 femmes	12 semaines Exercice T vs relaxation	↑ QOL
Carvalho (2015)	**12 pts**	12 semaines Ex T	↑Pic VO2 14% ↑ Qol
Szot (2015)	55 femmes	12 semaines Ex T	↑ 24 % METs ↑ QOL
Tyni-Lenne (2020) **R**	24 femmes	8 semaines Ex ± relaxation	↑ Pic VO2 + 15 % ↑ QOL (2 gpes)
Rahmani (2020) **R**	**30 pts**	4 semaines Ex	↑ VO2 et pouls 02 Améliore FC récup
Bove (2020) **R**	56 femmes BMI > 25	24 semaines	↓ angor, poids et Lipides = réserve coronaire (echo)

Modalities and safety of cardiac rehabilitation in a population managed for spontaneous haematoma or coronary disruption: insights from the READAPT-DISCO study

Clément Nguyen Ngoc[1], Nicolas Combaret [2], Hervé Douard[3], Marc Bonnet[4], Yann Valy[5], Nicolas Méneveau[6], Louis le Bivic[7], Gilles Rioufol [8,9], Grégoire Rangé[10], Philippe Brunel[11], Brahim Harbaoui [12,13], Benoît Lattuca[14], Johanne Silvain[15], Matthieu Godin[16], Thibault Lhermusier [17], Arnaud Fluttaz[18], Christophe Saint-Etienne[19], Grégory Ducrocq[20,21], Hakim Benamer[22,23,24], Vincent Roule[25], Didier Bresson[26], Norbert Mayaud[27], Geraud Souteyrand[2], Julien Nardoux[3], Pascal Motreff [2], and Edouard Gerbaud [1,28]*

DISCO : 373 pts -> 101 pts en RC (50 ans, 91 % femmes, 27 % PCI)

Délai :42 js Durée : 4-8 semaines

Entraînement : MCT 71 % , Interval training 50 % , Renf muscul 19%
Intervention psychologique : 44 %

	Avant RC	Fin RC
Charge max (watts)	105 ± 30	128 ± 35
FC max (b/min)	120 ± 23	134 ± 20
VO2 pic (ml/kg/min)	22.3 ± 7	24.4 ± 6
FC SV1 (b/min)	94 ± 20	107 ± 22
SV1 (ml/kg/min)	16.7 ± 6	21.4 ± 17

Eur J Prev Cardiol 2025;32:708-11

Effects of cardiac rehabilitation on the severity of angina, health-related quality of life, and exercise capacity among adults living with microvascular angina: a systematic review and meta-analysis

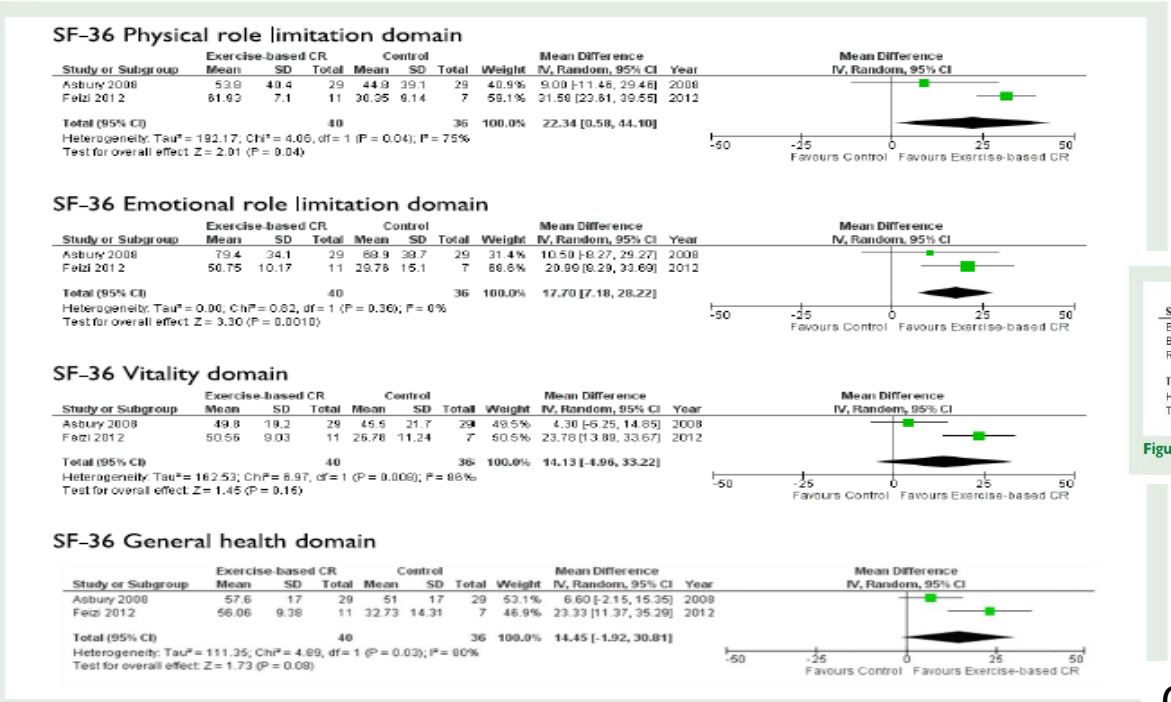


Figure 3 Comparison 1 exercise-based cardiac rehabilitation vs. control for microvascular angina, primary outcome 2 health-related quality of life

5 studies
Mean ages 51 -64 years
97.3% were women.

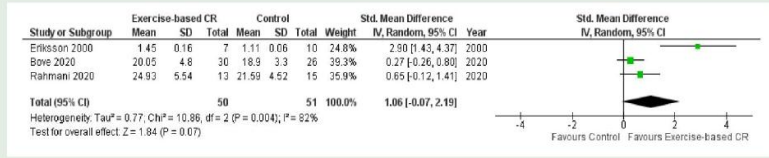


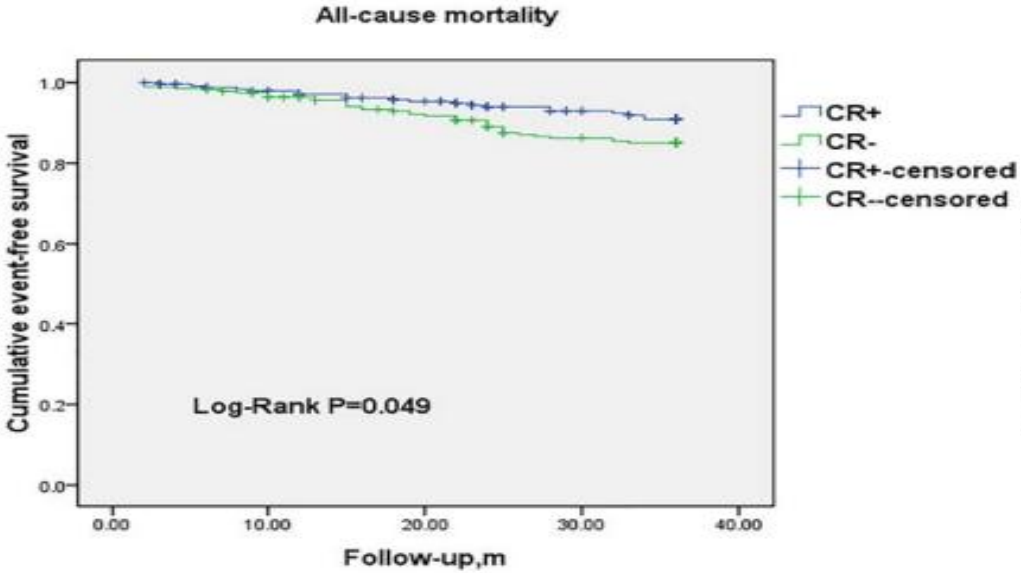
Figure 4 Comparison 1 exercise-based cardiac rehabilitation vs. control for microvascular angina, primary outcome 3 exercise capacity.

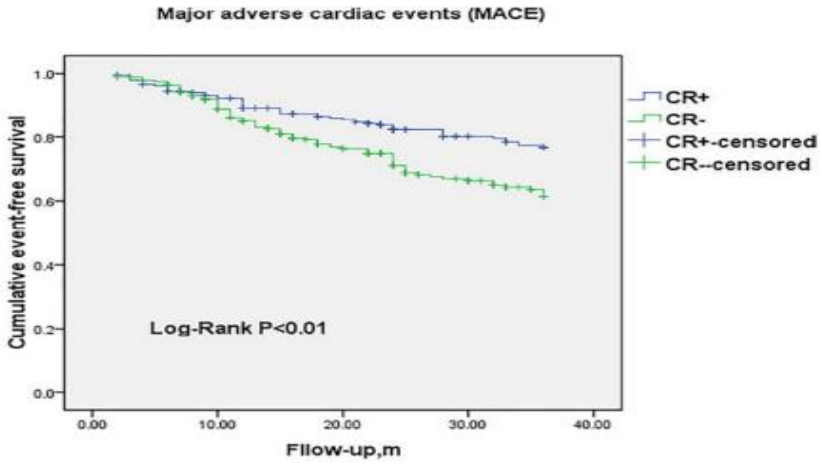
Oliveira J. Eur J Prev Cardiol 2025

Read card longue durée à domicile

524 patients MINOCA , randomisés (CR+) vs (CR−)

CR+ = Entrainement (MCT à 65%–75% de FC au max de l'EE) 3/sem



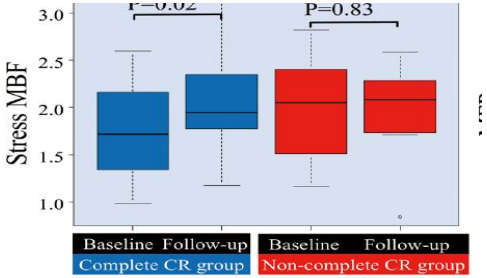


He C. Int J Cardio l 2020;315:9-14

Clinical Effects of Supervised Cardiac Rehabilitation in Patients With Angina and Non-Obstructive Coronary Artery Disease and Impaired Myocardial Flow Reserve Assessed Using ^{13}N-Ammonia Positron Emission Tomography

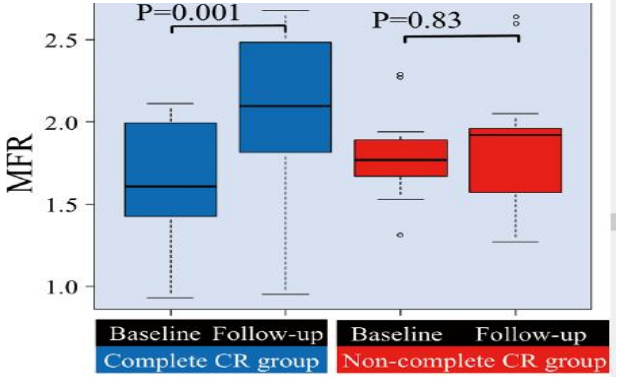
29 pts ANOCA, programme 16 RC (5 mois) complet, 13 incomplet

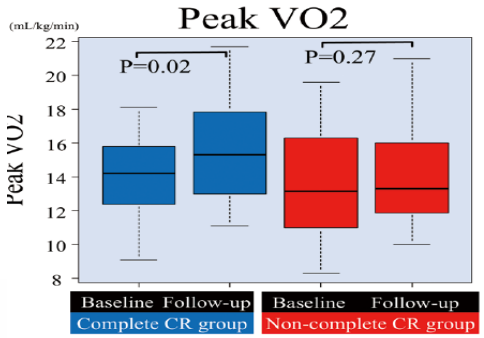
Stress Myocardial Blood Flow

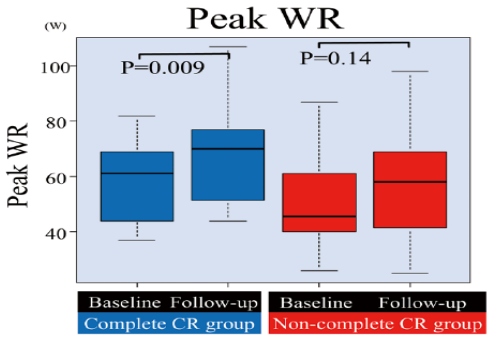


Test hyperhemie Petscan

Myocardial Flow Reserve

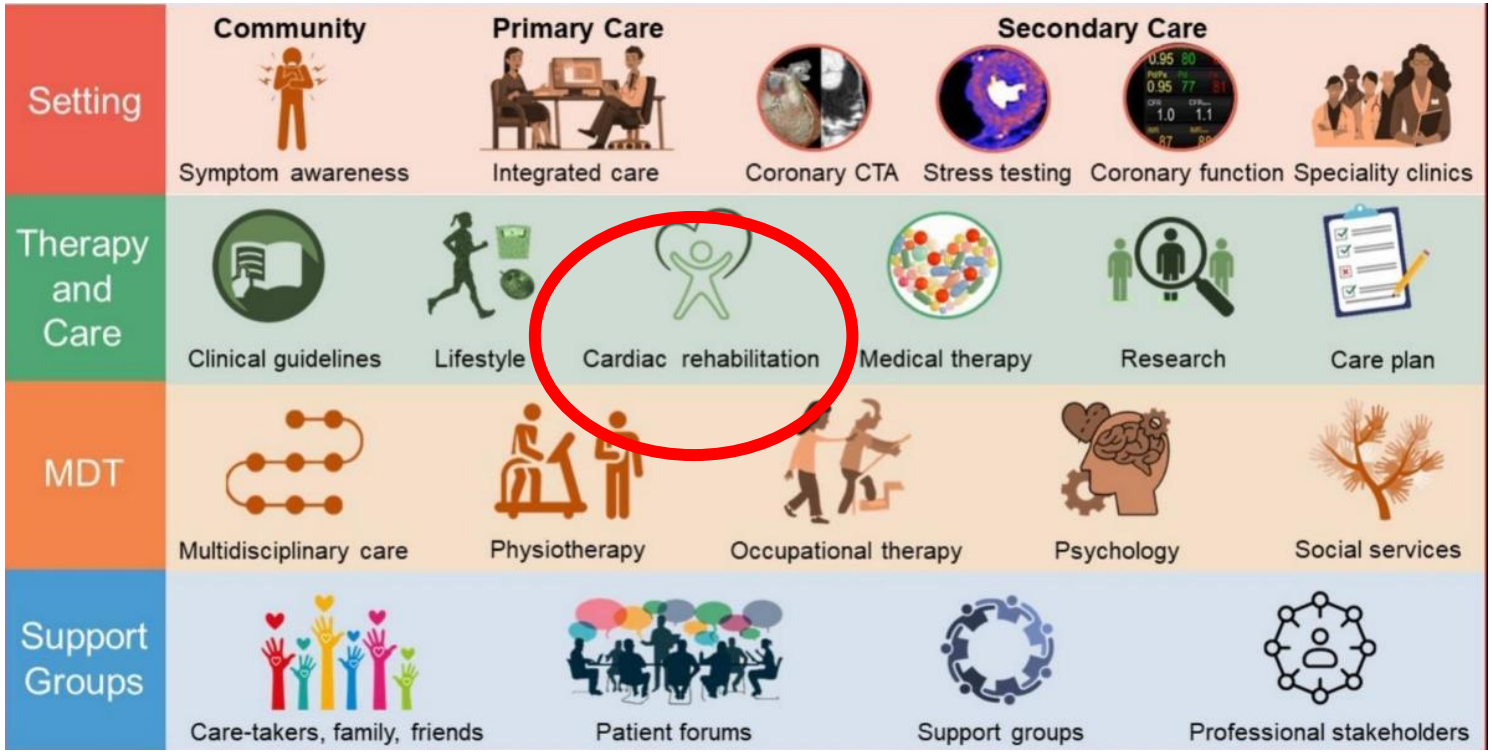






Miura S. Circ J 2025;89:1162-71

Clinical standards in angina and non-obstructive coronary arteries: A clinician and patient consensus



Int J Cardiol . 2025 ;5:429:133-162

Conclusions

La réadaptation cardiaque a toute sa place dans la prise en charge des coronaropathies non-épicardiques.

- Prise en charge globale : contrôle des FDR, psychologique, exercice
- L’ exercice physique est un traitement de la dysfonction endothéliale

Toutefois, le niveau de preuves est très faible et nécessite des essais cliniques complémentaires

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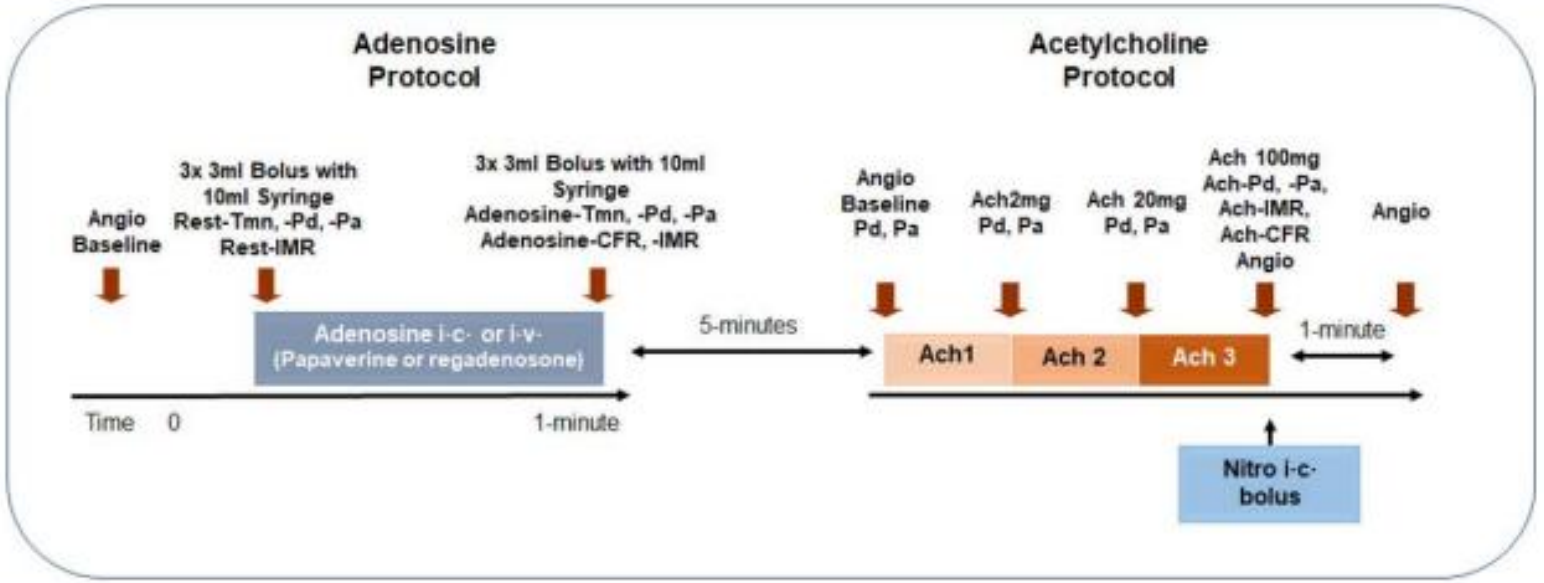

Figure 1 Protocol for the haemodynamic assessment. The protocol duration was approximately 9 min

Miner S. Eur Heart J (2026) 47, 594–604

Abnormal adenosine responses

(1) Adenosine-CFR < 2.5 with adenosine-IMR < 25 (high resting coronary blood flow)
(2) Adenosine-CFR < 2.5 with adenosine-IMR > 25 (high resistance)
(3) Adenosine-CFR > 2.5 with adenosine-IMR > 25 in which compensation mechanisms are recruited to maintain CFR (compensated high resistance)

Abnormal acetylcholine responses

(1) Acetylcholine-Pd/Pa < 0.80 and/or >90% coronary diameter reduction with ECG changes and angina during acetylcholine (epicardial spasm)
(2) Acetylcholine-IMR > rest-IMR (i.e. measurable evidence of increased microvascular resistance during acetylcholine) with ECG changes and angina during acetylcholine (microvascular spasm)
(3) Acetylcholine-CFR < 1.5 with preserved Adenosine-CFR and no microvascular spasm impaired endotheliumdependent vasodilation)
(4) Angina AND ischaemic ECG changes during acetylcholine without measurable haemodynamic abnormalities

Miner S. Eur Heart J (2026) 47, 594–604