CAT devant des troponines élevées en Réanimation

Leila Abid
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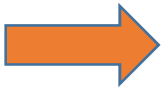
FMS

A Challenge……

- L’interprétation d’une élévation de la troponine sans une clinique d’infarctus du myocarde représente un défi au quotidien dans les services de soins aigus



la lésion myocardique sans ischémie, aiguë ou chronique, est devenue une entité de plus en plus reconnue

Situations d’élévation de la troponine sans ischémie myocardique!!!

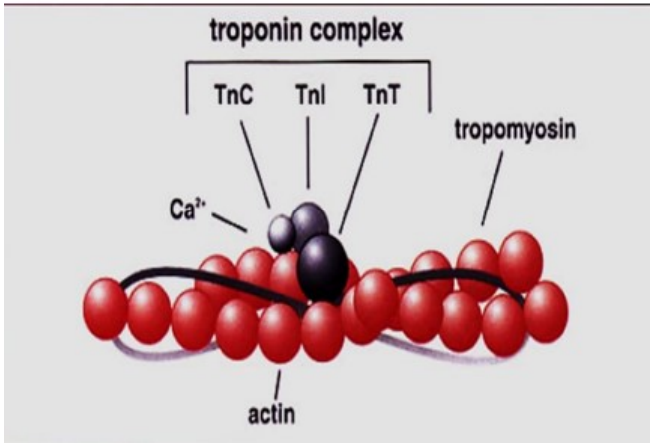
A challenge…

Le clinicien se retrouve confronté à un nombre croissant de cas d'élévation isolée du taux de hs-cTn, sans critères clairs d'IM

Un problème diagnostique et thérapeutique!!

Les troponines

- Protéines de structure du muscle cardiaque présentes dans le muscle cardiaque et les muscles squelettiques: Marqueurs ayant une spécificité myocardique élevée



- Isoformes cardiaque et musculaire de la TnI et de la TnT sont différentes : dosage spécifique de l'isoforme cardiaque (anticorps spécifique)

- Tn C: pas d'intérêt en cardiologie

Les troponines sont-elles spécifiques du myocardes ?

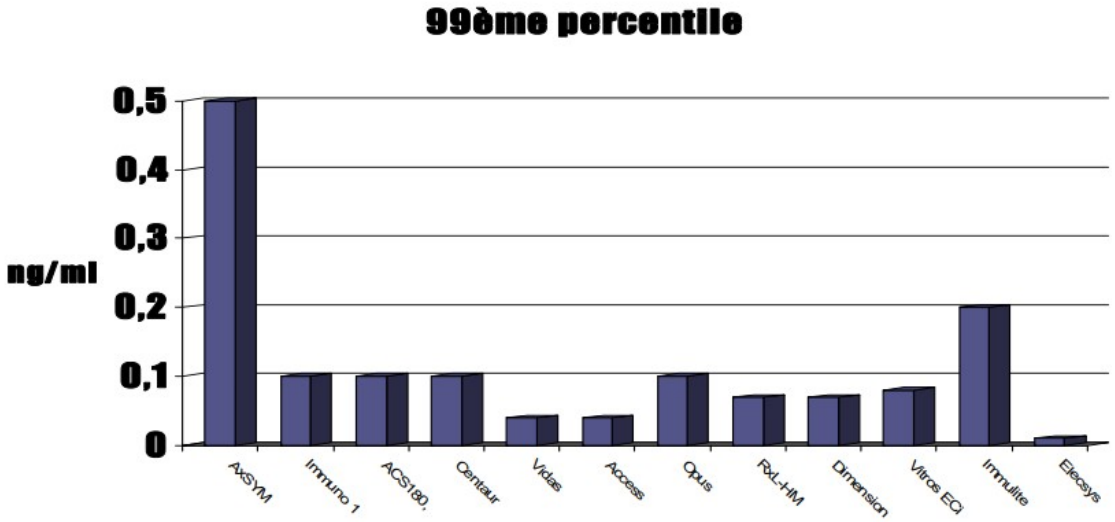
- Pour la troponine I : OUI

- Pour la troponine T : ça se discute

- Les plus sensibles et les plus spécifiques des dommages myocardiques: **Gold standard du diagnostic et pronostic des SCA**
- Libération si nécrose ou précocement si ischémie: Tn ultrasensible!
- Leur augmentation ne pose pas le diagnostic d'IM!!!!

Fiabilité des tests et valeur seuil

- Immunodosages très sélectifs: les anticorps utilisés dans ces dosages sont dirigés contre les formes cardiaques de ces protéines, et ainsi, une élévation des troponines signe habituellement exclusivement une **atteinte myocardique**



Mécanismes?

Stress mécanique de paroi en réponse à une surcharge en pression ou en volume (IC, EP…)

Athéro-thrombose coronarienne

Tachycardie

Elévation de troponine

Myocardite, ou atteinte inflammatoire des cardiomyocytes

Libération massive de catécholamines (AVC ischémique ou Hémorragique….)

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OPEN

CARDIAC TROPONIN RELEASE IS ASSOCIATED WITH BIOMARKERS OF INFLAMMATION AND VENTRICULAR DILATATION DURING CRITICAL ILLNESS

Marlies Ostermann,[*] Salma Ayis,[†] Emma Tuddenham,[‡] Jessica Lo,[§] Katie Lei,[*] John Smith,[*] Barnaby Sanderson,[*] Carl Moran,[||] Paul Collinson,[¶] Janet Peacock,[†] Andrew Rhodes,[¶] and David Treacher[*]

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Prevalence of troponin rise

One hundred forty-five patients (84%) had at least one cTnT value ≥15 ng/L during their stay in ICU. Among them,

Table 1. **Baseline characteristics of patients in the four groups**

aracteristics	All groups	Definite MI	Possible MI	Raised cTnT only	No cTn rise	*P* value*
up size (n)	172	21	51	73	27	
al number of cTnT results	997	138	318	415	126	
dian number of cTnT results per patient (min–max)	6 (1–11)	7 (1–11)	6 (1–11)	6 (1–11)	3 (1–11)	
e, mean (SD)	63.0 (16.6)	61.4 (14.4)	68.6 (14.4)	64.9 (15.2)	48.4 (17.7)	<0.01
nale sex, n (%)	72 (42%)	8 (38%)	22 (43%)	34 (47%)	8 (30%)	0.48
ite ethnicity, n (%)	142 (83%)	18 (86%)	40 (80%)	63 (90%)	21 (84%)	0.33
ACHE II score on admission to ICU, mean (SD)	18.2 (6.6)	18.0 (6.22)	18.9 (6.2)	20.0 (6.5)	12.5 (4.8)	<0.01
rum creatinine ≥140 μmol/L at any time during study period, n (%)	76 (44.2%)	10 (47.6%)	26 (51%)	39 (53.4%)	1 (3.7%)	<0.001
atment with RRT, n (%)	54 (31.4%)	7 (33.3%)	16 (31.4%)	30 (41.1%)	1 (3.7%)	0.005
T or serum creatinine >140 μmol/L, n (%)	87 (50.6%)	10 (47.6%)	31 (60.8%)	45 (61.6%)	1 (3.7%)	<0.001
morbidities						
hemic heart disease, n (%)	33 (20%)	6 (29%)	12 (24%)	13 (18%)	2 (7%)	0.23
pertension, n (%)	67 (39%)	8 (38%)	30 (59%)	24 (33%)	5 (19%)	<0.01
betes, n (%)	51 (30%)	8 (38%)	20 (39%)	20 (27%)	3 (11%)	0.06
form of vascular disease, n (%)	34 (20%)	4 (19%)	15 (29%)	14 (19%)	1 (3.7%)	0.06
rtality						
hospital, n (%)	42 (24%)	6 (29%)	16 (31%)	19 (26%)	1 (3.7%)	0.03
CU, n (%)	32 (19%)	5 (24%)	11 (22%)	15 (21%)	1 (3.7%)	0.44

values are for the test of heterogeneity among the four proportions or means as appropriate.
ACHE indicates acute physiology and chronic health; cTnT, cardiac troponin T; ICU, intensive care unit; RRT, renal replacement therapy; SD, standard deviation.

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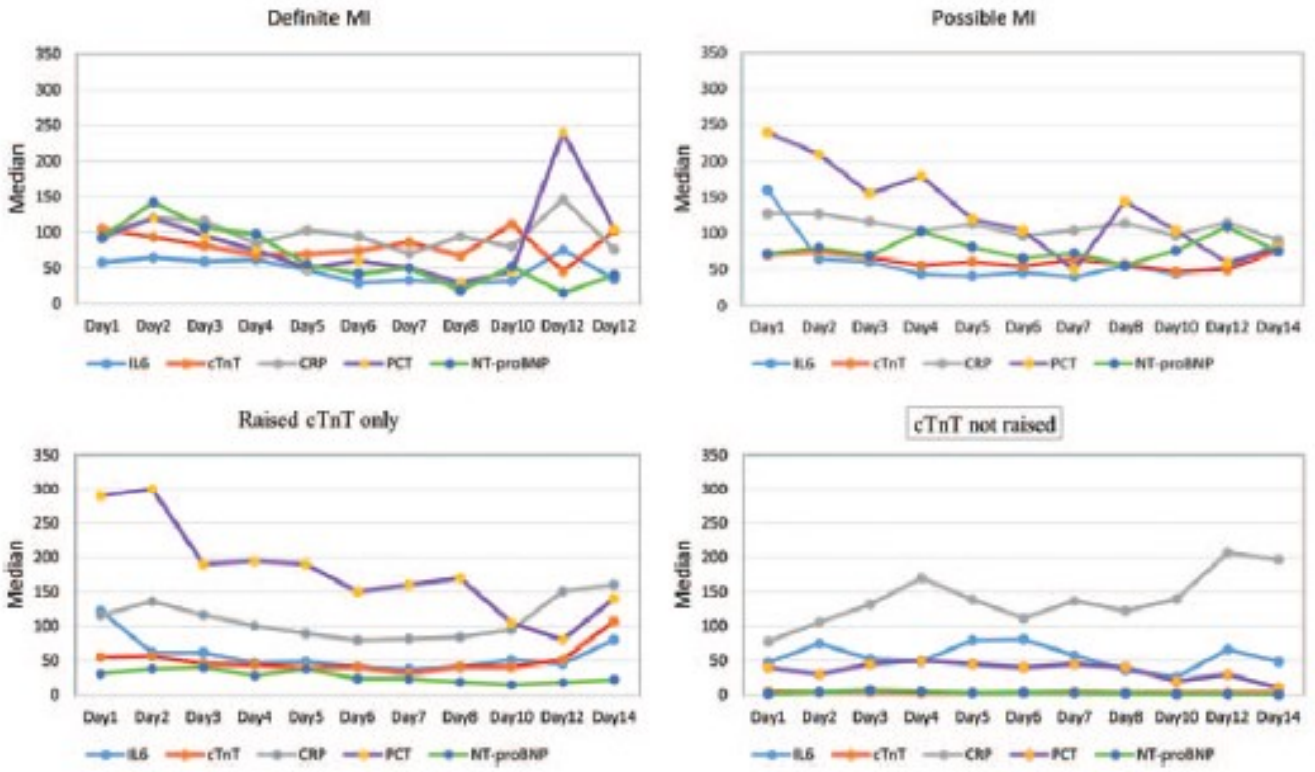

FIG. 1. **Patterns of variations described by medians over time from Day 1 to Day 14 for the outcome cardiac troponin T and biomarkers of inflammation and ventricular strain in four groups.** The daily median values of CRP, IL-6, PCT, NT-proBNP, and cTnT in their original units were graphically displayed to illustrate the variations of each biomarker over time in each of the four groups. NT-proBNP was scaled down for the graph by dividing it by 100 to allow the presentation on the same scale as other parameters. CRP indicates C-reactive protein; cTnT, cardiac troponin T; IL-6, interleukin-6; MI, myocardial infarction; NT-proBNP, N-terminal pro brain natriuretic peptide; PCT, procalcitonin.

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TABLE 3. **Unadjusted and adjusted estimates of associations of cTnT with sepsis and CRP, IL-I, PCT, and NT-proBNP in different troponin groups**

	Unadjusted estimates: effect of factor on cTnT as a percentage per unit cTnT, except sepsis* (95% CI)				Adjusted estimates: effect of factor on cTnT as a percentage per unit cTnT, except sepsis* (95% CI)				Standardized effect sizes for adjusted analysis equivalent to a SD change in the relevant marker		
Parameter	Effect size (%)	LCI	UCI	*P* value	Effect size (%)	LCI	UCI	*P* value	Effect size (%)	LCI	UCI
Patients with definite MI (n = 21)											
Sepsis	33	6.0	68	0.01	28.85	1.99	62.79	**0.03**			
CRP	0.13	−0.07	0.33	0.20	0.12	−0.08	0.32	0.25	9.70	−6.16	28.23
IL-6 (log)†	N/A			0.006	N/A			**0.02**	**23.37**	3.68	46.80
PCT	1.62	−0.06	3.33	0.06	1.30	−0.42	3.04	0.14	14.20	−4.21	36.15
NT-proBNP	0.00	−0.0003	0.0012	0.23	0.0004	−0.0004	0.0012	0.31	0.01	−0.01	0.02
Patients with a possible MI (n = 51)											
Sepsis	10.01	−5.48	28.03	0.22	5.98	−8.81	23.18	0.45			
CRP	0.13	0.03	0.23	0.01	0.11	0.01	0.21	**0.03**	**10.08**	0.85	20.16
IL-6 (log)†	N/A			<0.001	N/A			**<0.001**	**17.45**	8.14	27.56
PCT	1.32	0.36	2.29	0.007	1.20	0.25	2.15	**0.01**	**16.86**	3.31	32.17
NT-proBNP	0.00	0.0006	0.0016	<0.001	0.0010	0.0005	0.0015	**<0.001**	**0.02**	0.01	0.03
Patients with raised cTnT only (n = 73)											
Sepsis	22.12	6.56	39.95	0.004	23.17	4.99	37.80	**0.01**			
CRP	0.09	0.00	0.17	0.045	0.08	0.00	0.16	0.07	7.87	−0.47	16.92
IL-6 (log)†	N/A			0.001	N/A			**<0.001**	**15.68**	6.58	25.56
PCT	0.68	0.30	1.07	<0.001	0.70	0.28	1.03	**0.001**	**12.58**	5.23	20.45
NT-proBNP	0.00	0.0010	0.0028	<0.001	0.0018	0.0006	0.0024	**0.001**	**0.02**	0.01	0.03
Patients without raised cTnT (n = 27)											
Sepsis	−1.58	−15.80	15.03	0.84	−8.72	−21.03	5.51	0.22			
CRP	0.06	−0.03	0.15	0.16	0.00	−0.08	0.09	0.92	0.36	−6.35	7.55
IL-6 (log)†	N/A			0.31	N/A			0.31	−3.32	−9.45	3.24
PCT	2.86	1.35	4.38	<0.001	2.21	0.89	3.54	**0.001**	**13.74**	5.35	22.79
NT-proBNP	0.01	0.0036	0.0089	<0.001	0.0049	0.0025	0.0073	**<0.001**	**0.02**	0.01	0.03

Adjusted mixed model gives average cTnT (outcome) after allowing for gender, age, ischemic heart disease, hypertension, RRT use, or creatinine >140 μmol/L, diabetes, and any form of vascular disease.

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EXPERT CONSENSUS DOCUME

Fourth Universal Definition of Myocardial Infarction (2018)

Joint ESC/ACC/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction

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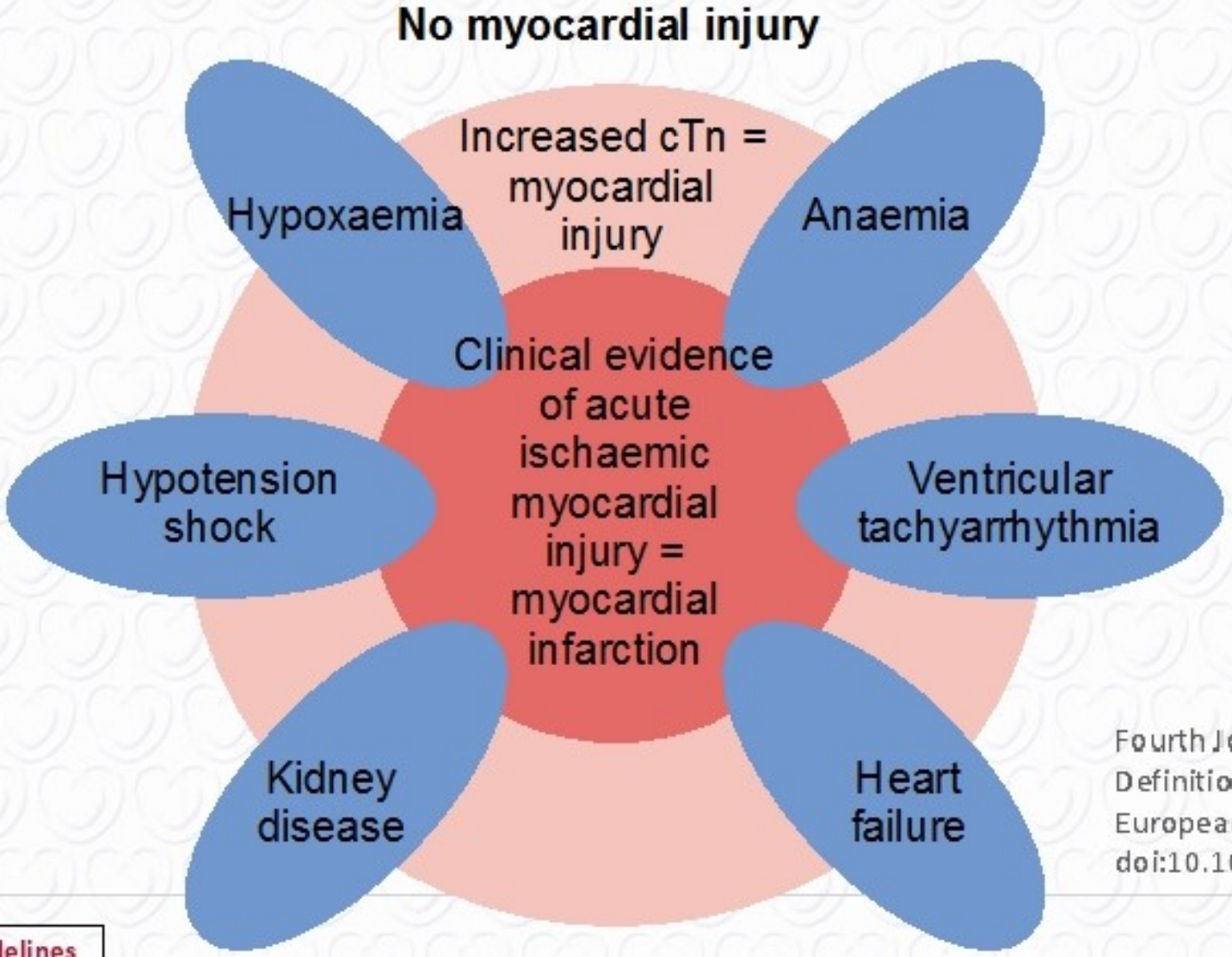
Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Spectrum of Myocardial Injury, ranging from no Injury to Myocardial Infarction



Fourth Joint ESC/ACC/AHA/WHF Unive
Definition of Myocardial Infarction
European Heart Journal 2019; 40: 237-
doi:10.1093/eurheartj/ehy462

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Reasons for Elevation of Cardiac Troponin Values because of Myocardial Injury (1)

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Myocardial injury related to acute myocardial ischaemia
Atherosclerotic plaque disruption with thrombosis.
Myocardial injury related to acute myocardial ischaemia because of oxygen supply/demand imbalance
Reduced myocardial perfusion, e.g. • Coronary artery spasm, microvascular dysfunction • Coronary embolism • Coronary artery dissection • Sustained bradyarrhythmia • Hypotension or shock • Respiratory failure • Severe anaemia
Increased myocardial oxygen demand, e.g. • Sustained tachyarrhythmia • Severe hypertension with or without left ventricular hypertrophy

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Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Reasons for Elevation of Cardiac Troponin Values because of Myocardial Injury (2)

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Other causes of myocardial injury
Cardiac conditions, e.g. • Heart failure • Myocarditis • Cardiomyopathy (any Type) • Takotsubo syndrome • Coronary revascularization procedure • Cardiac procedure other than revascularization • Catheter ablation • Defibrillator shocks • Cardiac contusion

Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Reasons for Elevation of Cardiac Troponin Values because of Myocardial Injury (3)

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Other causes of myocardial injury
Systemic conditions, e.g. • Sepsis, infectious disease • Chronic kidney disease • Stroke, subarachnoid haemorrhage • Pulmonary embolism, pulmonary hypertension • Infiltrative diseases, e.g. amyloidosis, sarcoidosis • Chemotherapeutic agents • Critical ill patients • Strenuous exercise

Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Etiology of troponin elevation in critically ill patients

Wendy Lim MD, MSc[a,b,*], Richard Whitlock MD, PhD[c,d], Vikas Khera MD[a,b], Philip J. Devereaux MD, PhD[a,d,e], Andrea Tkaczyk MN[b], Diane Heels-Ansdell MSc[d,e], Michael Jacka MD, MSc[f], Deborah Cook MD, MSc[a,d,e]

Etiology of troponin elevation in critically ill patients

Table 1 Frequency of each etiology adjudicated as being the most likely cause for troponin elevation in critically ill patients

Etiology for elevated troponin	Adjudicator 1 (n = 49)	Adjudicator 2 (n = 49)	Consensus (n = 49)
MI	21 (42.9)	24 (49.0)	26 (53.1)
Sepsis	7 (14.3)	9 (18.4)	9 (18.4)
Renal failure	8 (16.3)	7 (14.3)	6 (12.2)
Cardiac contusion/ cardiopulmonary resuscitation	2 (4.1)	2 (4.1)	3 (6.1)
Chronic obstructive pulmonary disease	2 (4.1)	2 (4.1)	3 (6.1)
Congestive heart failure	3 (6.1)	1 (2.0)	2 (4.1)
Pulmonary hypertension	1 (2.0)	1 (2.0)	0 (0)
Hypotension	1 (2.0)	1 (2.0)	0 (0)
Left ventricular hypertrophy/strain	0 (0)	1 (2.0)	0 (0)
Other[a]	4 (8.2)	1 (2.0)	0 (0)

Values are presented as no. (%).

[a] Eclamptic seizure, malignancy, loss of airway, cocaine-induced vasospasm.

Journal of Critical Care (2010) 25, 322–328

Table 2 Baseline patient characteristics according to etiology

	MI (n = 26)	Other etiology (n = 23)	No elevated troponin (n = 51)	P[a]		
				MI vs other etiology	MI vs no elevated troponin	Other etiology vs no elevated troponin
Age, mean (SD)	69.8 (15.2)	66.7 (19.2)	59.3 (16.9)	.53	.01	.10
Female sex, no. (%)	11 (42.3)	8 (34.8)	24 (47.1)	.77	.81	.45
APACHE II score, mean (SD)	26.4 (9.1)	26.5 (10.5)	16.2 (8.3)	.97	<.001	<.001
Medical, no. (%)	21 (80.8)	19 (82.6)	23 (45.1)	1.0	.003	.003
Prior coronary artery disease,[b] no. (%)	10 (38.5)	5 (21.7)	9 (17.6)	.23	.06	.75
Baseline life support interventions,[c] no. (%)						
Invasive MV	21 (80.8)	10 (43.5)	25 (49.0)	.009	.008	.80
Noninvasive MV	0 (0)	2 (8.7)	2 (3.9)	.22	.55	.58
Baseline inotropes and vasopressors,[c] no. (%)						
Epinephrine	0 (0)	3 (13.0)	0 (0)	.10	–	.03
Dopamine[d]	2 (7.7)	3 (13.0)	1 (2.0)	.66	.26	.09
Norepinephrine	3 (11.5)	8 (34.8)	3 (5.9)	.09	.40	.003
Dobutamine	0 (0)	2 (8.7)	0 (0)	.22	–	.09
Phenylephrine	2 (7.7)	1 (4.3)	0 (0)	1.0	.11	.31
Vasopressin	0 (0)	1 (4.3)	0 (0)	.47	–	.31
Any of the above	5 (19.2)	10 (43.5)	4 (7.8)	.12	.16	<.001
Hemodialysis,[c] no. (%)						
Intermittent	3 (11.5)	4 (17.4)	2 (3.9)	.69	.33	.07
CRRT	0 (0)	1 (4.3)	0 (0)	.47	–	.31

CRRT indicates continuous renal replacement therapy; MV, mechanical ventilation.

[a] P values for continuous data based on unpaired t test; and for binary data, on Fisher exact test.

[b] Coronary artery disease = angina, MI.

Prognostic Value of Initial Elevation in Cardiac Troponin I Level in Critically Ill Patients Without Acute Coronary Syndrome

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Table 3 Primary and secondary outcomes differences between cardiac troponin I (CTnI)-positive and CTnI-negative groups[a]

Variable	CTnI-positive (n = 40)	CTnI-negative (n = 50)	Relative risk	95% CI	P[b]
Hospital mortality	14 (35)	6 (12)	1.35	1.05-1.73	.01
Length of stay, median (interquartile range)					
Intensive care units	4 (2-6.5)	3 (2-6)	—	—	.52
Hospital	9 (4-11.5)	8 (6.1-16)	—	—	.44
Admitting diagnosis					
Sepsis	15 (38)	11 (22)	1.23	0.94-1.65	.16
Shock	16 (40)	10 (20)	1.33	0.99-1.78	.06
Acute renal failure	4 (10)	3 (6)	1.04	0.92-1.18	.48
COPD/asthma exacerbation	9 (22)	6 (12)	1.87	0.72-4.82	.19
Use of intravenous vasopressor(s)	15 (38)	9 (18)	1.31	0.99-1.72	.06
Intubated any time during intensive/cardiac care unit admission[c]	15 (41)	8 (17)	1.34	1.02-1.76	.02
COPD patients who required intubation[c]	7 (47)	2 (25)	1.24	0.44-3.54	.68
Successful extubation among intubated patients[c]	5 (33)	5 (62)	1.03	0.89-1.19	.18
Confirm pulmonary embolism[d]	1 (14)	0 (0)	5.25	0.24-114	.29
Disposition at discharge[e]					
Home	10 (25)	23 (46)	0.72	0.52-0.98	.04
Nursing home	8 (20)	10 (20)	1.00	0.81-1.23	.92
Rehabilitation	6 (15)	7 (14)	1.01	0.85-1.20	.90
Inpatient psychiatry unit	2 (5)	1 (2)	1.03	0.95-1.10	.41
Expired during hospitalization	14 (35)	6 (12)	1.35	1.05-1.73	.01
Transfer to other facility	0 (0)	1 (2)	0.98	0.92-1.04	.41

Abbreviation: COPD, chronic obstructive pulmonary disease.

[a] Data are presented as absolute numbers (percentage) unless otherwise indicated.

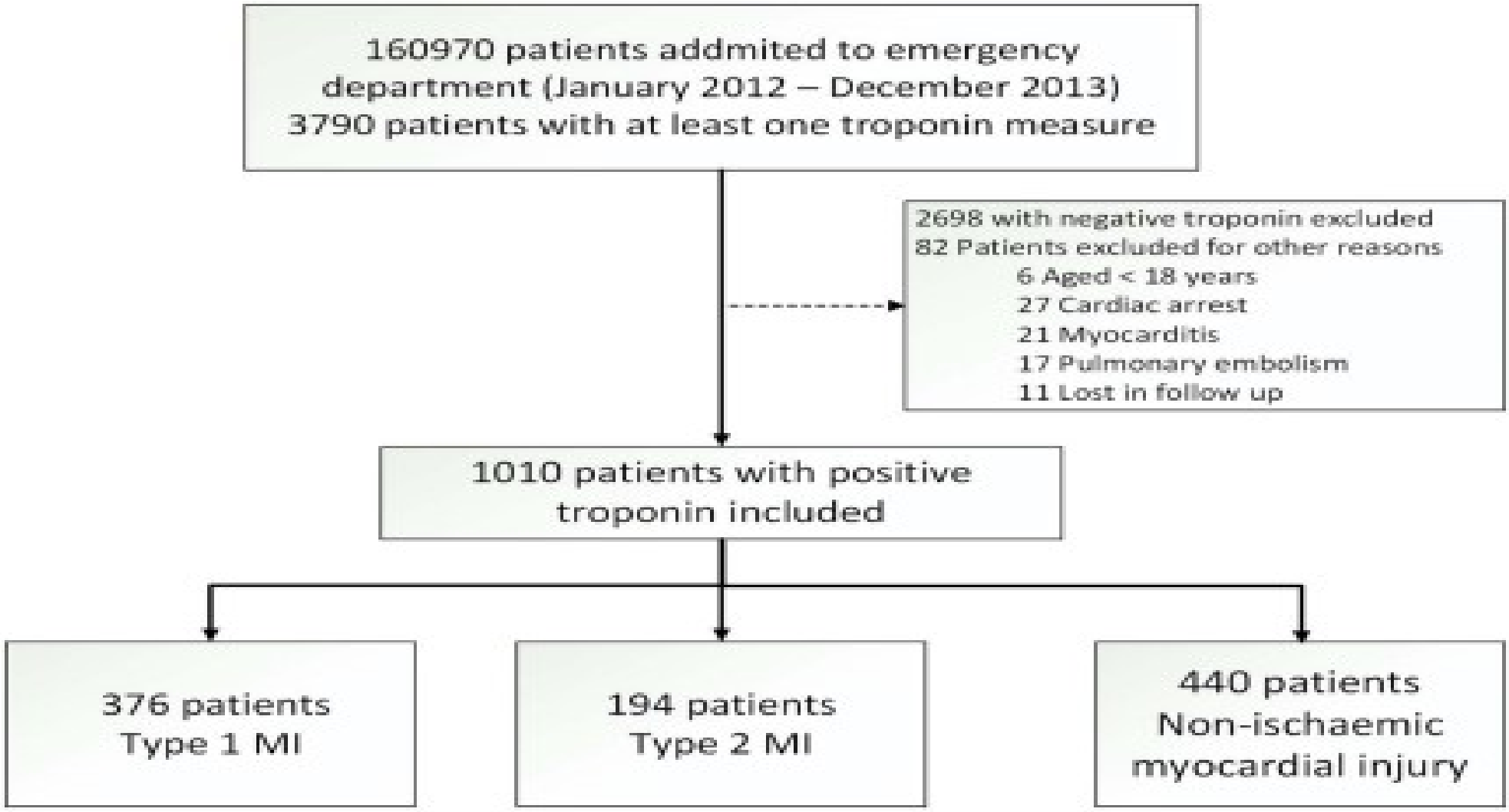
[b] For comparison between CTnI-positive and CTnI-negative groups, the Fisher exact test was used for categorical variables and the Mann-Whitney test was used for categorical independent variables and continuous dependent variables. The Kruskal-Wallis test was used for disposition of patients after discharge from the hospital. *P* values less than .05 were considered statistically significant.

[c] Excludes patients who were dependent on invasive mechanical ventilation before the current admission (3 patients in the CTnI-positive group and 4 patients in the CTnI-negative group) and patients who did not require intubation during the current admission (22 patients in the CTnI-positive group and 38 patients in the CTnI-negative group). Exclusion of those patients leaves 15 patients in the CTnI-positive group and 8 patients in the CTnI-negative group who required intubation at any time during the intensive care/cardiac care unit admission.

[d] Was performed on 7 of the CTnI-positive group and 13 of the CTnI-negative group.

[e] For the CTnI-negative group, 2 patients (4%) had not been discharged by the end of the study period.

(Critical Care Nurse. 2015;35[2]: e1-e10



Cediel G, *et al. Heart* 2016;**0**:1–7. doi:10.1136/heartjnl-2016-310243

Cediel G, Gonzalez-del-Hoyo M, Carrasquer A, et al. Outcomes type 2 myocardial infarction compared with non-ischemic myocardial injury. Heart 2017;103:61

Table 2 Main diagnoses of patients with type 2 myocardial infarction (MI) and with non-ischaemic myocardial injury (NIMI) at hospital discharge

	Type 2 MI (n=194)	NIMI (n=440)	p Value
Heart failure	55 (28.35)	83 (18.86)	0.008
Tachyarrhythmia	36 (18.56)	36 (8.18)	<0.001
Respiratory infection or COPD	36 (18.56)	70 (15.91)	0.410
Bradycardia	21 (10.82)	0	<0.001
Anaemia	10 (5.15)	0	<0.001
Hypertensive crisis	1 (0.52)	5 (1.14)	0.457
Renal failure	9 (4.64)	9 (2.05)	0.070
Syncope	1 (0.52)	25 (5.68)	0.003
Gastrointestinal bleeding	5 (2.58)	5 (1.14)	0.180
Other gastrointestinal pathology	1 (0.52)	31 (7.05)	0.001
Other infections	1 (0.52)	18 (4.09)	0.015
Other diagnoses	9 (4.64)	76 (17.27)	<0.001
Sepsis	5 (2.58)	10 (2.27)	0.816
Neurological disease	1 (0.52)	25 (5.68)	0.003
Neoplasia	2 (1.03)	7 (1.59)	0.583
Chest pain	0	34 (7.73)	<0.001

Data are presented as no. (%).
COPD, chronic obstructive pulmonary disease.

Cediel G, Gonzalez-del-Hoyo M, Carrasquer A, et al. Outco
type 2 myocardial infarction compared with non-ischemic myocardial injury. Heart 2017;10

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Distinct etiologies of high-sensitivity troponin T elevation predict different mortality risks for patients hospitalized with COVID-19

Pegah Khaloo [1], Ayman Shaqdan [1,1], Pablo A. Ledesma [1], Uwajachukwumma A. Uzomah [1], Jennifer Galvin [1], Leon M. Ptaszek [1,2], Jeremy N. Ruskin [*,1,2]

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Table 3
Predictors of in-hospital mortality in patients admitted with COVID-19.

Predictors of Mortality: Logistic Regression Based on Medical Comorbidities	OR	95% CI	P value
Age (per 1 year)	1.06	1.05-1.07	<0.001
Pre-existing AV block	2.5	1.4-5.3	0.003
Previous cerebrovascular accidents	2.3	1.4-3.8	0.001
Incident stroke	3.6	1.0-12.5	0.043
Incident shock	3.2	2.3-4.5	<0.001
Incident acute kidney injury	1.9	1.4-2.6	<0.001
Elevated troponin due to primary cardiac cause	4.6	2.7-7.6	<0.001
Elevated troponin due to primary non-cardiac cause	2.7	1.6-4.5	<0.001

Quatrième définition universelle de l'infarctus du myocarde

Cinétique de la troponine dans les différentes entités de l'ischémie cardiaque

- Lésion du myocarde (myocardial injury) = cTn* > 99e percentile URL+
- Lésion aiguë du myocarde = cTn avec une cinétique d'augmentation et/ou baisse
- Lésion chronique du myocarde = cTn avec cinétique stable (≤ 20% de variation entre dosages)
- Infarctus du myocarde = clinique typique d'ischémie cardiaque associée à une lésion aiguë du myocarde

Classification des cinq types d'Infarctus du myocarde**

Type I :	Rupture de plaque coronarienne avec athérothrombose
Type II :	Déséquilibre entre le débit sanguin coronarien (DO_2) et la demande en O_2 (VO_2)
Type III :	Mort subite d'origine cardiaque
Type IVa :	Associé à une intervention coronarienne percutanée
Type IVb :	Thrombose de stent
Type IVc :	Resténose instent ou resténose après angioplastie au ballon seul
Type V :	Associé à bypass aorto-coronarien

(D'après réf.[4]).

Myocardial Infarction Type 1

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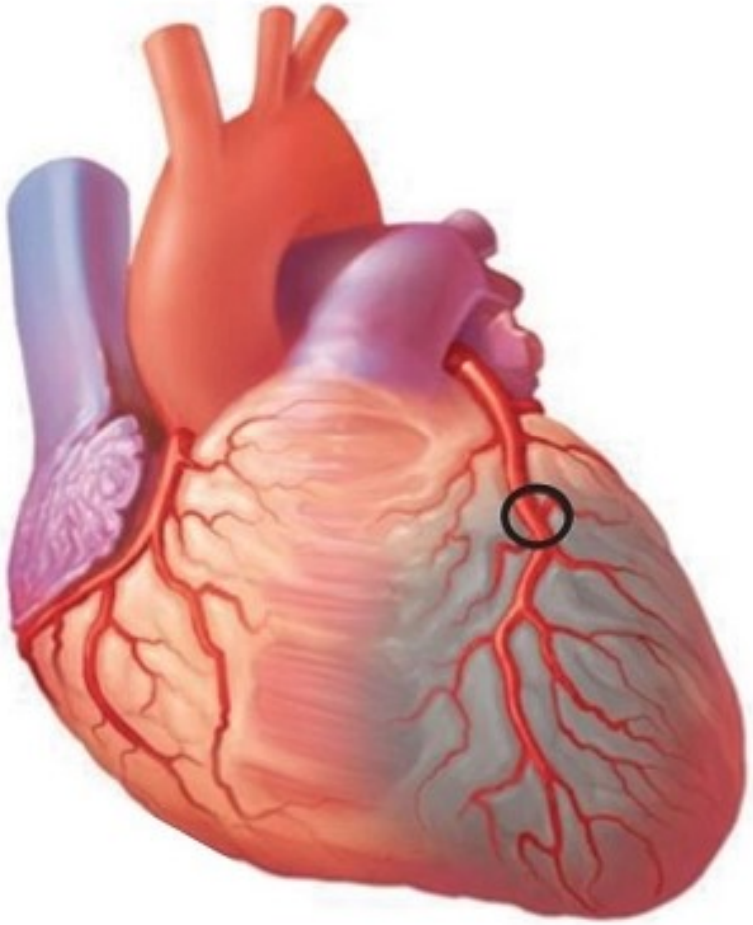

Plaque rupture/erosion with occlusive thrombus

Plaque rupture/erosion with non-occlusive thrombus

SCAST
STEM

SCAST
NSTEM

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European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Criteria for Type 1 Myocardial Infarction

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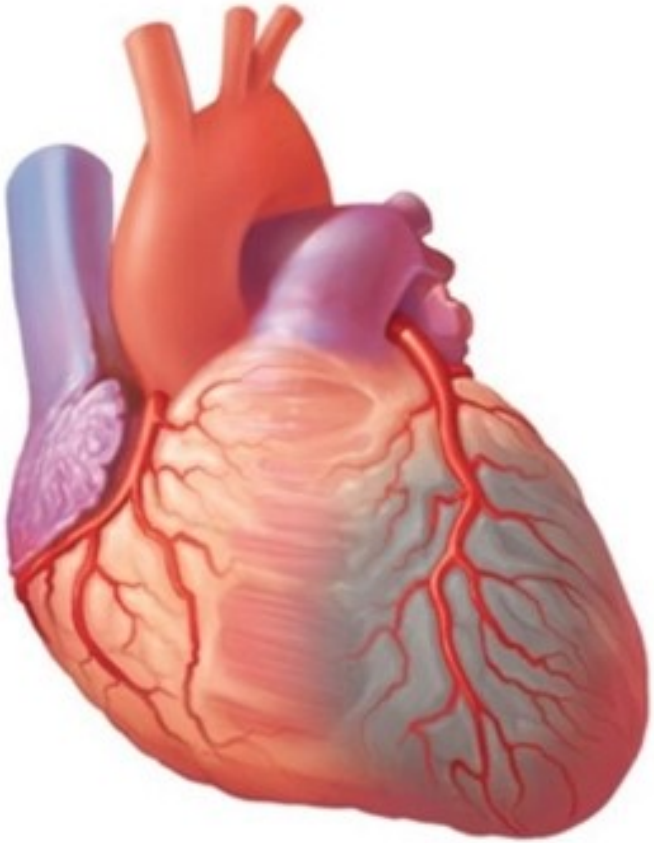
Detection of a rise and/or fall of cTn with at least one value above the 99th percentile URL and with at least one of the following:

- **Symptoms of acute myocardial ischaemia;**
- **New ischaemic ECG changes;**
- **Development of pathological Q waves;**
- **Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology;**
- **Identification of a coronary thrombus by angiography including intracoronary imaging or by autopsy.**

Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Myocardial Infarction Type 2



Atherosclerosis and oxygen supply/demand imbalance

Vasospasm or coronary microvascular dysfunction

Non-atherosclerotic coronary dissection

Oxygen supply/demand imbalance alone

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European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Criteria for Type 2 Myocardial Infarction

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Detection of a rise and/or fall of cTn with at least one value above the 99th percentile URL and evidence of an imbalance between myocardial oxygen supply and demand unrelated to coronary athero-thrombosis, requiring at least one of the following:

- **Symptoms of acute myocardial ischaemia;**
- **New ischaemic ECG changes;**
- **Development of pathological Q waves;**
- **Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology.**

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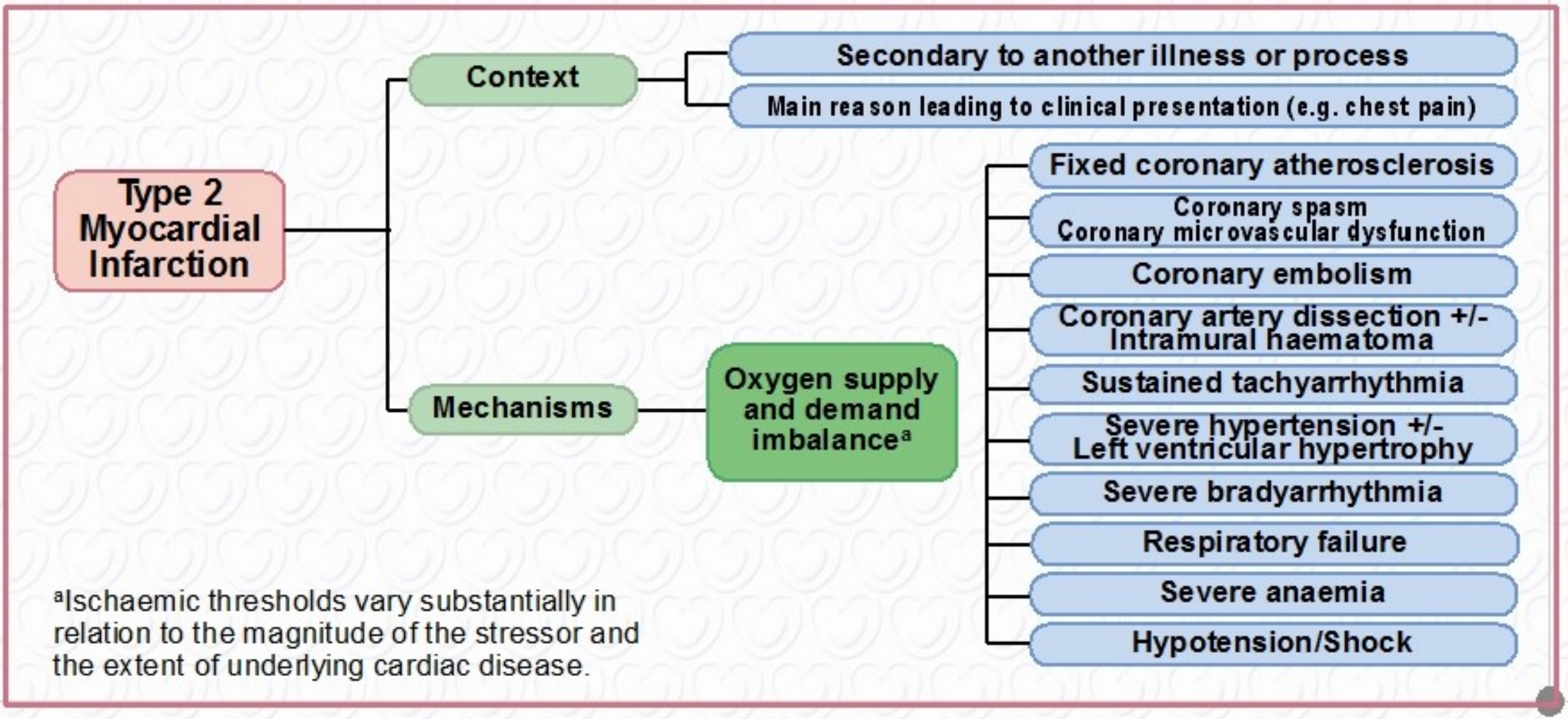
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Framework for Type 2 MI considering Context and Mechanisms attributable to Acute Myocardial Ischaemia



[a]Ischaemic thresholds vary substantially in relation to the magnitude of the stressor and the extent of underlying cardiac disease.

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European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Criteria for Type 3 Myocardial Infarction

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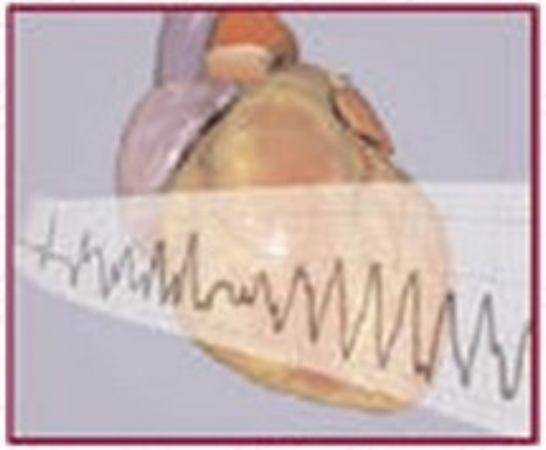
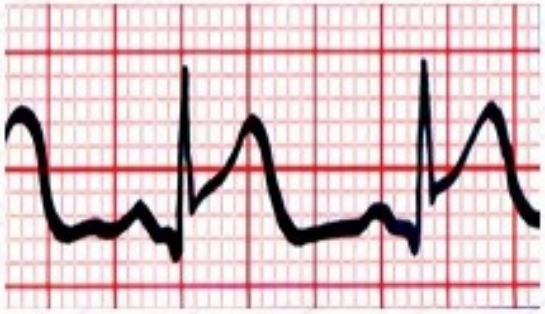
Patients who suffer cardiac death, with symptoms suggestive of myocardial ischaemia accompanied by presumed new ischaemic ECG changes or ventricular fibrillation, but die before blood samples for biomarkers can be obtained, or before increases in cardiac biomarkers can be identified or myocardial infarction detected by autopsy examination.





Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Myocardial Infarction Type 4a

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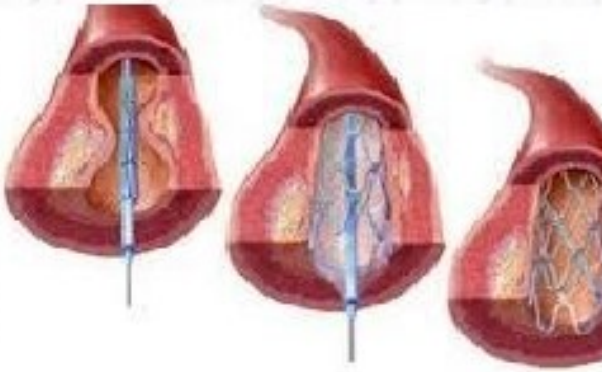
PCI-related MI ≤48 h after the index procedure is defined by elevation of cardiac troponin values >5 times 99th percentile URL. In addition, either

- New ischaemic ECG changes or
- Imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality consistent with an ischaemic aetiology
- Angiographic findings consistent with a procedural flow-limiting complication such as coronary dissection, occlusion of a major epicardial artery or a side-branch occlusion/ thrombus, disruption of collateral flow or distal embolization



Isolated development of ne
Q waves meets the criteria
cTn values are elevated an
rising but less than the pre
specified thresholds for PC

If cTn values are not >5 X 99th percentile URL, then the term myocardial injury should be used

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European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Myocardial Infarction Type 4b

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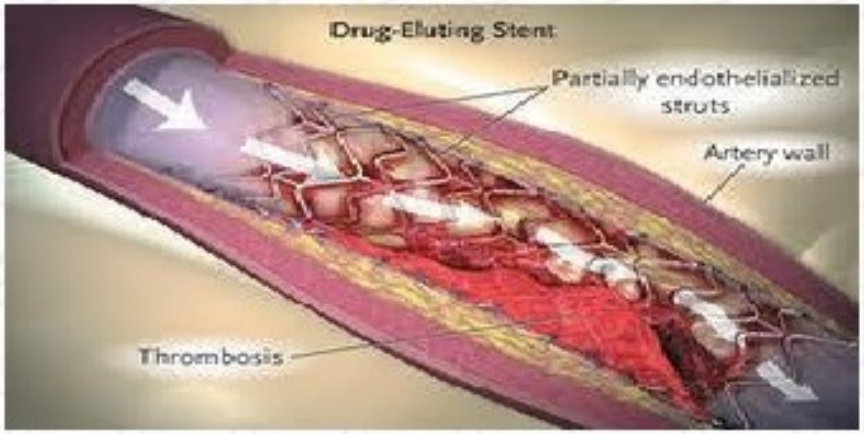
Myocardial infarction related to stent-thrombosis is detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac troponin values with at least one value >99th percentile URL.

The following temporal categories are suggested:

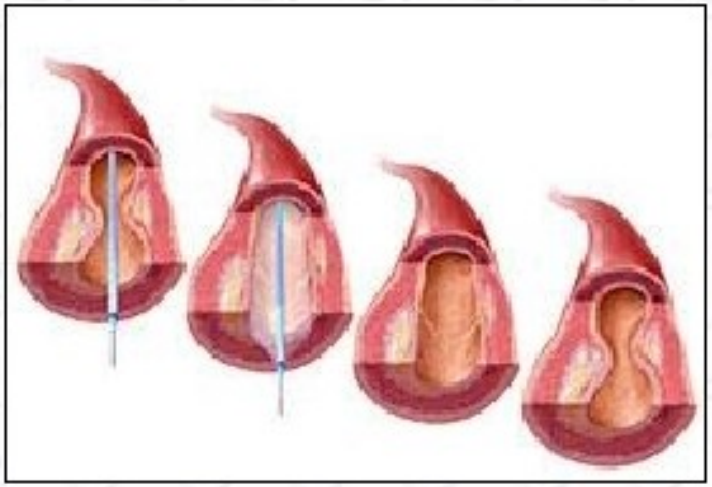
- **Acute, 0–24 h**
- **Subacute, > 24 h to 30 days**
- **Late, > 30 days to 1 year**
- **Very late > 1 year**



Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Myocardial Infarction Type 4c

Myocardial infarction related to in-stent restenosis, or restenosis following balloon angioplasty in the infarct territory is detected by coronary angiography in the setting of myocardial ischaemia and with a rise and/or fall of cardiac troponin values with at least one value >99th percentile URL



Fourth Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 226 - https://doi.org/10.1093/eurheartj/ehy856

Myocardial Infarction Type 5

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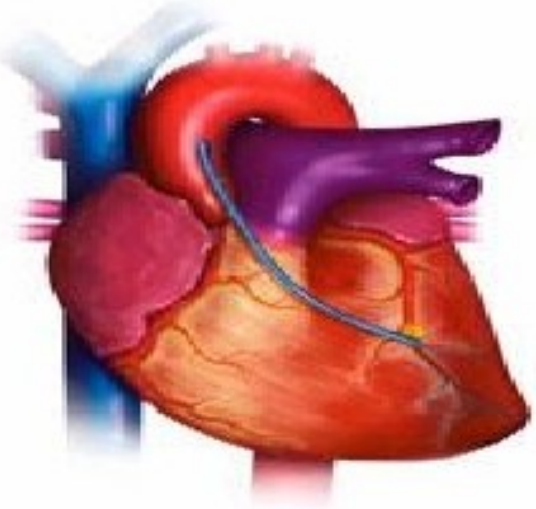
CABG-related MI ≤48 h after the index procedure is defined by elevation of cardiac troponin values >10 times 99th percentile URL. In addition, either

- **new pathological Q waves or**
- **angiographic documented new graft or new native coronary artery occlusion, or**
- **imaging evidence of new loss of viable myocardium or new regional wall motion abnormality and in a pattern consistent with an ischaemic aetiology.**



Isolated development of new Q waves meets the criteria if cTn values are elevated and rising but less than the pre-specified thresholds for CABG

If cTn values are not >10 X 99th percentile URL, then the term myocardial injury should be used

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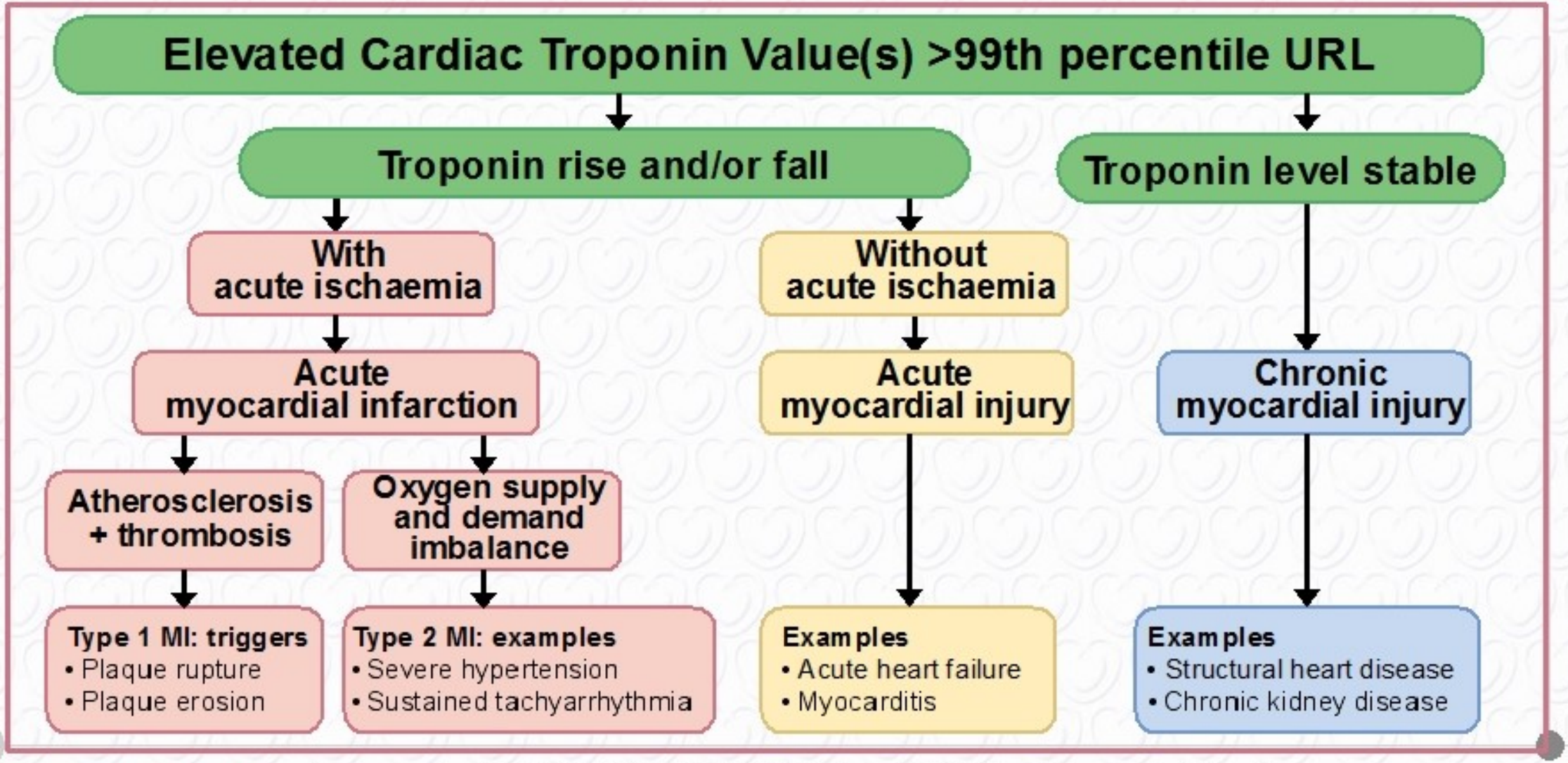
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Model for interpreting Myocardial Injury and Myocardial Infarction



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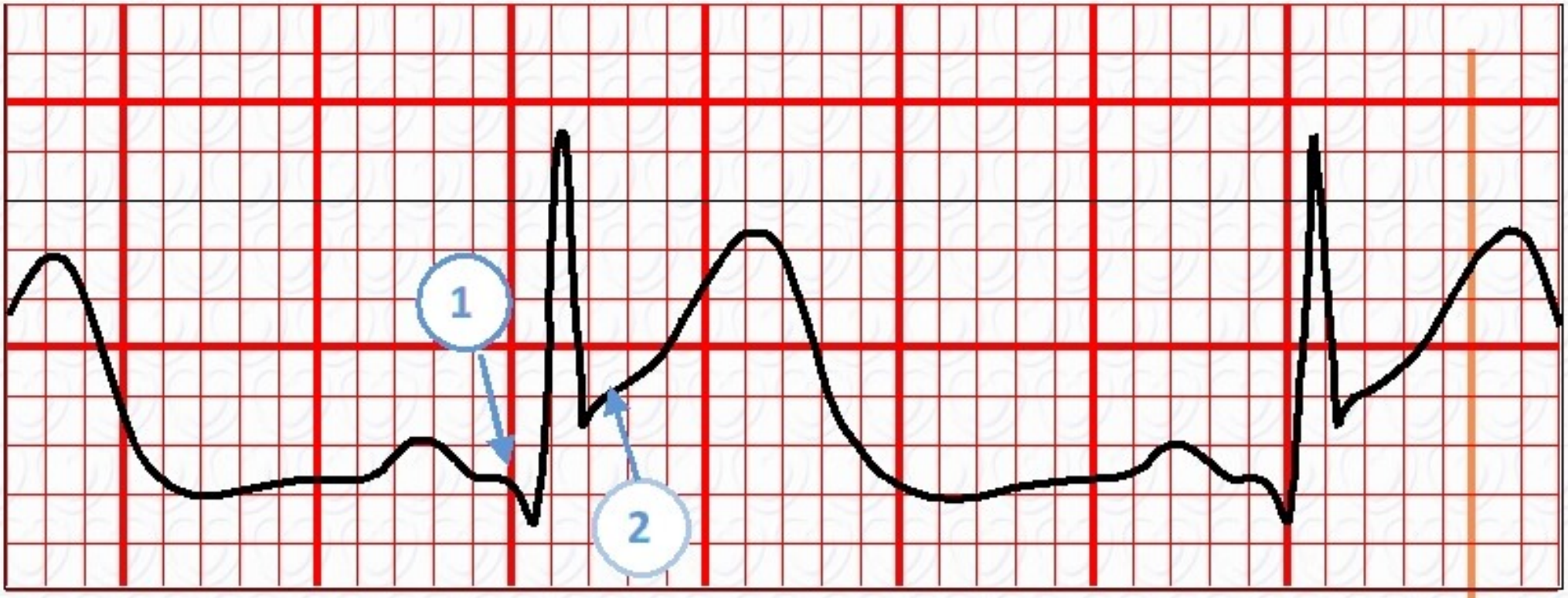
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

How to assess ST-segment elevation



Arrow 1 indicates the onset of the Q wave. Arrow 2 Indicates the onset of the ST-segment or J-point. The difference between points 1 and 2 denotes the magnitude of the ST-segment elevation

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European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Electrocardiographic Changes* suggestive of Acute Myocardial Ischaemia

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ST-elevation
New ST-elevation at the J-point in two contiguous leads with the cut points: ≥1 mm in all leads other than leads V_2–V_3 where the following cut points apply: ≥2mm in men ≥40 years; ≥2.5 mm in men <40 years, or ≥1.5 mm in women regardless of age.
ST-depression and T wave changes
New horizontal or down-sloping ST-depression ≥0.5 mm in two contiguous leads and/or T inversion >1 mm in two contiguous leads with prominent R wave or R/S ratio >1.

*in absence of left ventricular hypertrophy and bundle branch block

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European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Electrocardiographic Changes* associated with Prior Myocardial Infarction

Any Q wave in leads V_2–V_3 >0.02 s or QS complex in leads V_2–V_3
Q-wave ≥0.03 s and ≥1 mm deep or QS complex in leads I, II, aVL, aVF or V_4–V_6 in any two leads of a contiguous lead grouping (I, aVL; V_1–V_6; II, III, aVF)
R wave >0.04 s in V_1–V_2 and R/S >1 with a concordant positive T wave in absence of conduction defect

*in absence of left ventricular hypertrophy and bundle branch block

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European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Prior or Silent/Unrecognized Myocardial Infarction

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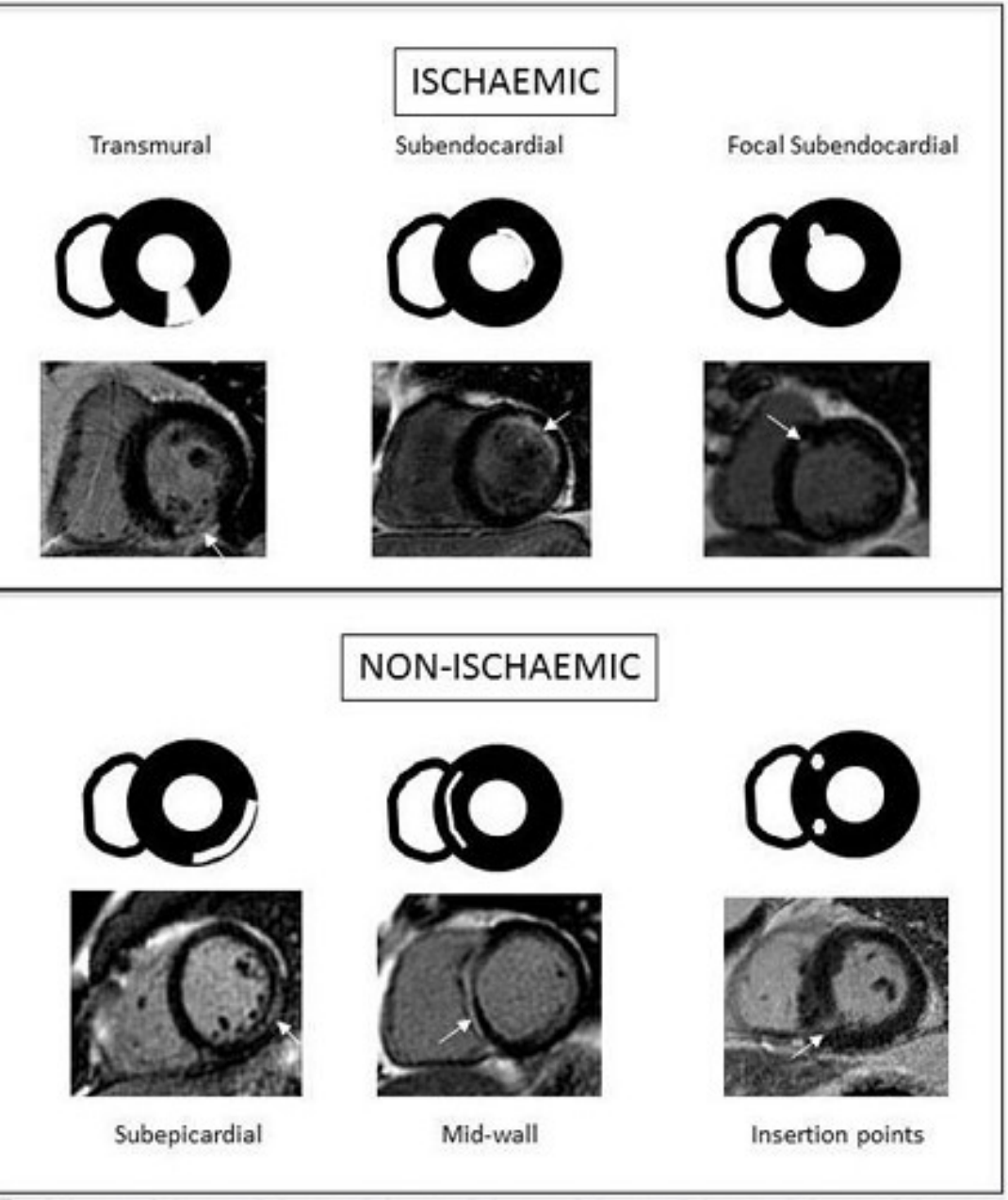
Criteria for Prior or Silent/Unrecognized Myocardial Infarction
Any one of the following criteria meets the diagnosis for prior or silent/unrecognized MI: • Abnormal Q waves with or without symptoms in the absence of non-ischaemic causes. • Imaging evidence of loss of viable myocardium in a pattern consistent with ischaemic aetiology. • Patho-anatomical findings of a prior MI



Cardiac Magnetic Resonance Images

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Gadolinium-based contrasts wash out from myocardium with increased extracellular space such as fibrosis, thus enhancing areas of scar (white arrows).

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Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 237-269 - doi:10.1093/eurheartj/ehy462

Ten commandments for the Fourth Universal Definition of Myocardial Infarction (1)

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Myocardial Injury and Myocardial Infarction

1) Myocardial injury is defined by the presence of cardiac troponin values (cTn) above the 99th percentile of the upper reference limit (URL).

2) Myocardial injury may be acute (rise and/or fall of cTn values) as in acute heart failure or chronic (≤20% variation of cTn values) as in chronic kidney disease.

3) Myocardial injury may occur in a variety of situations including after coronary procedural intervention and/or with cardiovascular and non-cardiovascular illnesses.

4) Occurrence of acute myocardial injury in the setting of acute myocardial ischaemia defines acute myocardial infarction.

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Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 226 - https://doi.org/10.1093/eurheartj/ehy856

Ten commandments for the Fourth Universal Definition of Myocardial Infarction (2)

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Myocardial Infarction – Spontaneous Types

5) Myocardial infarction type 1 is acute myocardial injury related to acute athero-thrombotic coronary artery disease. It is usually precipitated by atherosclerotic plaque disruption that reduces blood supply to the myocardium.

6) Myocardial infarction type 2 is acute myocardial injury related to an imbalance between myocardial oxygen supply and demand secondary to stressors unrelated to acute coronary athero-thrombosis.

7) Myocardial infarction type 3 is related to patients who suffer cardiac death, with symptoms suggestive of acute myocardial ischaemia accompanied by new ischaemic ECG changes and die before biomarker values could be obtained.

Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 226 - https://doi.org/10.1093/eurheartj/ehy856

Ten commandments for the Fourth Universal Definition of Myocardial Infarction (3)

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Myocardial Infarction – Procedural Types

8) Myocardial infarction type 4a denotes PCI-related increases of cTn values >5 times the 99th percentile URL from a normal or if elevated, stable pre-procedural baseline. New myocardial ischaemia evidenced by ECG or imaging, or complications leading to reduced coronary blood flow are required.

9) Myocardial infarction type 4b is acute myocardial ischaemic injury related to stent thrombosis, and myocardial infarction type 4c is acute myocardial ischaemic injury associated with restenosis.

10) Myocardial infarction type 5 is CABG-related increases of cTn values >10 times 99th percentile URL from a normal or if elevated, stable pre-procedural baseline. New myocardial ischaemia or new loss of myocardial viability is required.

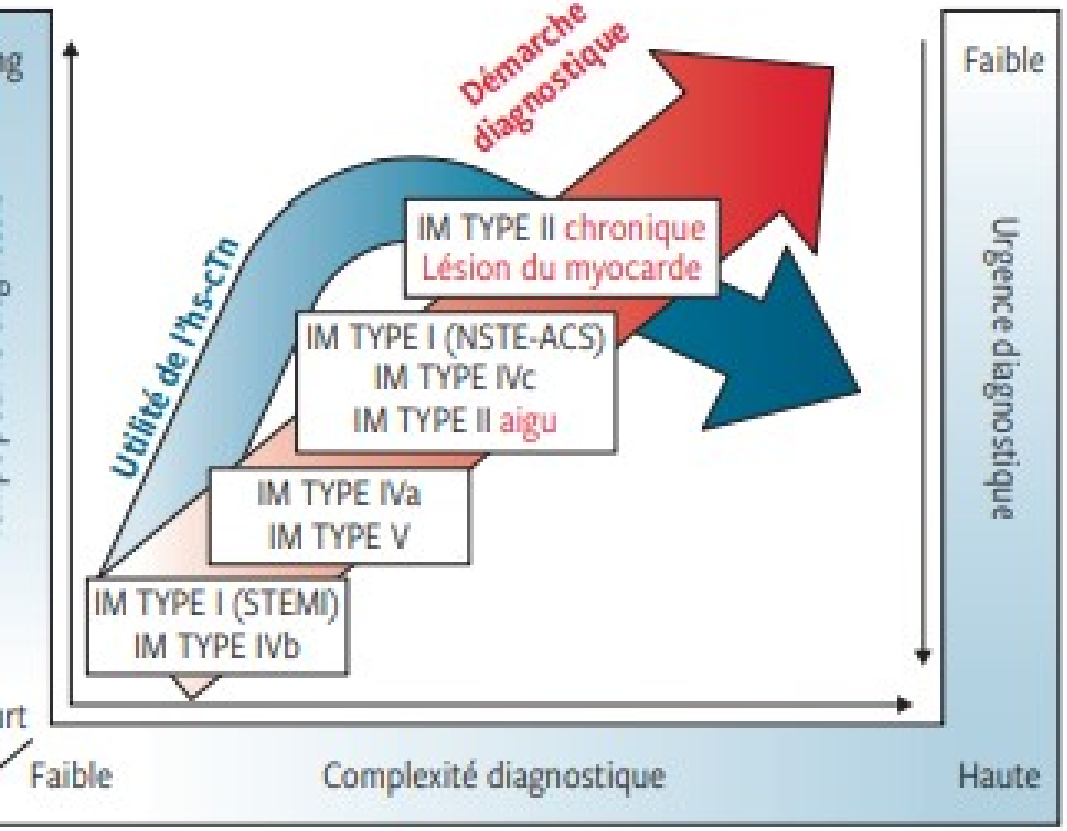
Fourth Joint ESC/ACC/AHA/WHF Universal Definition of Myocardial Infarction
European Heart Journal 2019; 40: 226 - https://doi.org/10.1093/eurheartj/ehy856

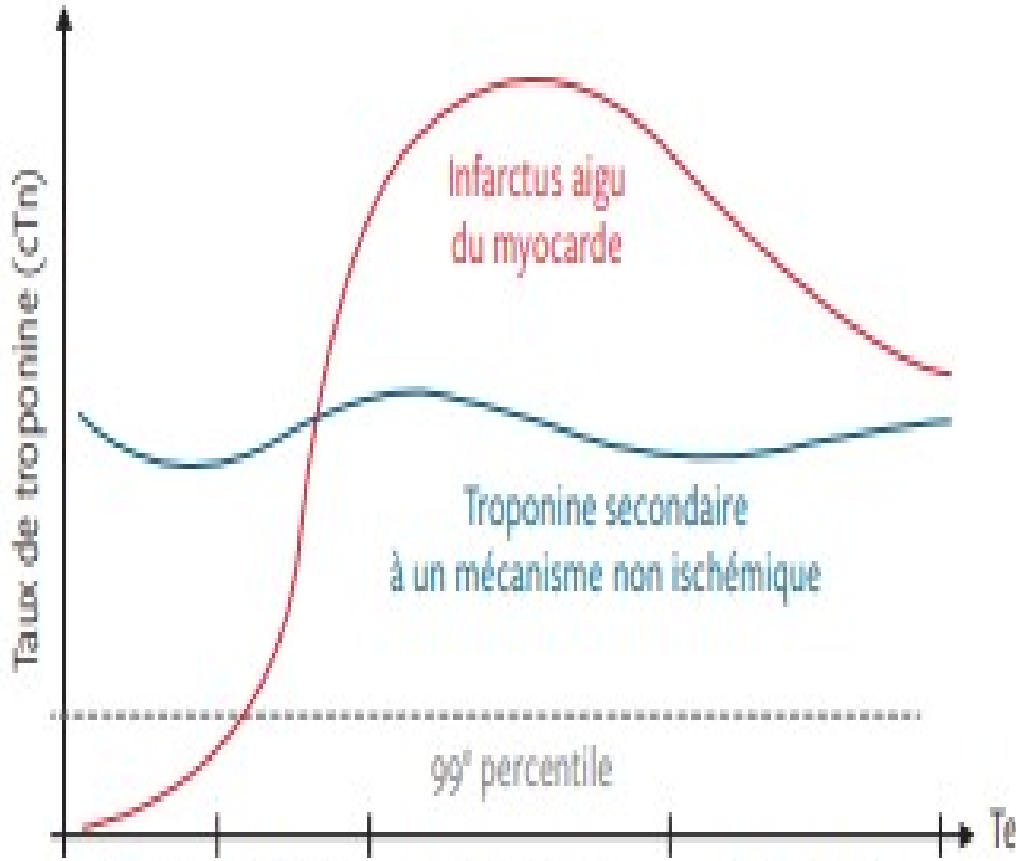
Démarche diagnostique

L'atteinte **ischémique aiguë du myocarde**, en particulier secondaire à un déséquilibre entre apport et besoin en oxygène, doit être différenciée de **l'atteinte sans ischémie, aiguë ou chronique**, ce que met en évidence la Quatrième définition universelle de l'IM

FIG 1 **Démarche diagnostique lors d'une élévation de la hs-cTn**

e diagnostique en présence ou en absence d'une clinique d'ischémie
enne.
roponine cardiaque ultrasensible; NSTE-ACS: IM de type I sans élévation
ent ST (non ST-segment elevation acute coronary syndrome); STEMI:
avec élévation du segment ST (ST-segment elevation myocardial
n).





gesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction. J Am Coll Cardiol 2018;72:223

Démarche diagnostique

Elévation de troponine en milieu de réanimation

atteinte aiguë du myocarde **ischémique**

Atteinte **sans ischémie**, aiguë ou chronique

\+

Anamnèse, clinique, ECG

\-

e en urgence un SCA ST+: La clinique +++ et l'ECG+++ (I, IVb)!!!

ion ≥ 5 fois de la hs-cTn de base ou > 20 % en cas de
de base pathologique, dans un contexte de cathétérisme
chirurgie cardiaques, permet d'identifier les IM de type
V

étique dynamique de type rise and fall: Il faut exclure d'un
type I sans élévation du segment ST (SCAST-) et d'un IM
pe II, secondaire à une diminution de la perfusion
ardique ou à une augmentation de la demande en
ne: anamnèse, clinique, ECG,

Elévation persistante avec une cinétique fruste (≤ 2
variation de la hs-cTn: atteinte non ischémique liée
mécanisme aigu ou chronique

Origine Cardiaque:

syndrome de Takotsubo, décompensation cardiaqu

Origine systémique:

état septique, AVC, IRC,
post-chirurgie non cardiaque

Implication pratique!!

- Une élévation de la hs-cTn ne correspond pas forcément à un infarctus du myocarde
- Un infarctus myocardique de type I doit être exclu en premier lieu
- La démarche diagnostique se fait par exclusion

Table 1 Clinical baseline characteristics of patients

Baseline characteristic*	Type 1 MI (n=376)	Type 2 MI (n=194)	NIMI (n=440)	p Value	p Value (type 2 MI vs NIMI)
Male sex	272 (72.34)	112 (57.73)	242 (55.00)	<0.001	0.523
Age, median (IQR), years	66 (55–78)	79 (69–84)	78 (67–85)	<0.001	0.620
Clinical history					
Prior MI	120 (31.91)	41 (21.13)	118 (26.82)	0.021	0.128
Congestive heart failure	15 (3.99)	31 (15.98)	87 (19.77)	<0.001	0.258
Peripheral arterial disease	52 (13.83)	21 (10.82)	60 (13.64)	0.560	0.328
Stroke or TIA	38 (10.11)	29 (14.95)	65 (14.77)	0.099	0.954
COPD	52 (13.83)	53 (27.32)	126 (28.64)	<0.001	0.734
Diabetes	132 (35.11)	73 (37.63)	155 (35.23)	0.813	0.561
Arterial hypertension	270 (71.81)	153 (78.87)	330 (75.00)	0.179	0.292
Renal disease	42 (11.17)	51 (26.29)	96 (21.82)	<0.001	0.219
Current or previous smoker	218 (57.98)	62 (31.96)	128 (29.09)	<0.001	0.468
Charlson Index, median (IQR)	1 (0–3)	2 (1–4)	2 (1–4)	<0.001	0.358
Clinical symptoms					
Chest pain	337 (89.63)	42 (21.65)	108 (24.55)	<0.001	0.429
Dyspnoea	37 (9.84)	90 (46.39)	146 (33.18)	<0.001	0.002
Syncope	5 (1.33)	15 (7.73)	38 (8.64)	<0.001	0.705
Other	21 (5.59)	62 (31.96)	194 (44.09)	<0.001	0.004
ECG†					
Sinus rhythm	334 (89.30)	98 (55.37)	252 (61.92)	<0.001	0.138
Atrial fibrillation	37 (9.92)	67 (37.85)	135 (33.25)	<0.001	0.283
RBBB or LBBB	72 (19.25)	40 (22.60)	97 (23.83)	0.290	0.746
ST segment elevation	92 (24.60)	5 (2.82)	7 (1.72)	<0.001	0.387
ST segment depression	97 (25.94)	13 (7.34)	25 (6.14)	<0.001	0.588
Negative T wave	94 (25.13)	16 (9.04)	45 (11.08)	<0.001	0.458
Vital signs on admission					
Heart rate, median (IQR), bpm	78 (67–90)	93 (70–123)	87 (70–110)	<0.001	0.022
SBP, median (IQR), mm Hg	140 (123–160)	140 (115–163)	132 (119–152)	<0.001	0.078
SaO_2, median (IQR),	98 (96–99)	94 (88–98)	97 (94–99)	<0.001	<0.001
Laboratory tests					
Glucose, median (IQR), mmol/L	7.38 (5.66–10.27)	7.60 (6.1–10.43)	7.16 (5.77–10.27)	0.446	0.194
GFR, median (IQR), mL/min/1.73 m^2	82.3 (55.6–99.6)	51.3 (31.5–64.8)	57.5 (40.2–79.9)	<0.001	<0.001
Haemoglobin, median (IQR), g/L	138 (123–150)	116 (92–138)	127 (115–141)	<0.001	<0.001
Maximum level of TnI, median (IQR), µg/mL	6.05 (0.61–31.84)	0.15 (0.08–0.56)	0.10 (0.06–0.27)	<0.001	<0.001
Clinical management					
Echocardiogram	359 (95.48)	79 (40.72)	137 (31.14)	<0.001	0.019
Exercise stress test	21 (5.59)	2 (1.03)	5 (1.14)	<0.001	0.820
Coronary angiography	278 (73.94)	11 (5.67)	23 (5.23)	<0.001	0.907
Assessment by cardiologist	376 (100)	107 (65.64)	234 (62.73)	<0.001	0.519
Hospital admission	368 (97.87)	126 (64.95)	215 (48.86)	<0.001	<0.001

*Data are presented as no. (%) unless otherwise indicated.
†Data on ECG were available for 374 patients with type 1 MI, 177 with type 2 MI and 407 with NIMI.
COPD, chronic obstructive pulmonary disease; GFR, glomerular filtration rate; LBBB, left bundle branch block; MI, myocardial infarction; NIMI, non-ischaemic myocardial injury; RBBB,

Cediel G, Gonzalez-del-Hoyo M, Carrasquer A, et al. Outco
type 2 myocardial infarction compared with non-ischemic myocardial injury. Heart 2017;10

30 Januzzi JL, Filippatos G, Nieminen M, et al. Troponin elevation in patients with heart failure: on behalf of the third Universal definition of myocardial infarction global task force: heart failure section. Eur Heart J 2012;33:2265-71.

28 Putot A, Derrida SB, Zeller M, et al. Short-term prognosis of myocardial injury, type 1, and type 2 myocardial infarction in the emergency unit. Am J Med 2018;131:1209-19

31 Jacobs LH, van de Kerkhof J, Mingels AM, et al. Haemodialysis patients longitudinally assessed by highly sensitive cardiac troponin T and commercial cardiac troponin T and cardiac troponin I assays. Ann Clin Biochem 2009;46: 283-90. 32 Unger ED, Dubin RF, Deo R, et al. Association of chronic kidney disease with abnormal cardiac mechanics and adverse outcomes in patients with heart failure and preserved ejection fraction. Eur J Heart Fail 2016;18:103- 12

Troponine négative faussement rassurante!

Algorithme rapide de troponine de l'ESC:
Hs-cTn: Troponine c de haute sensibilité

0 h
hs-cTn
1 h

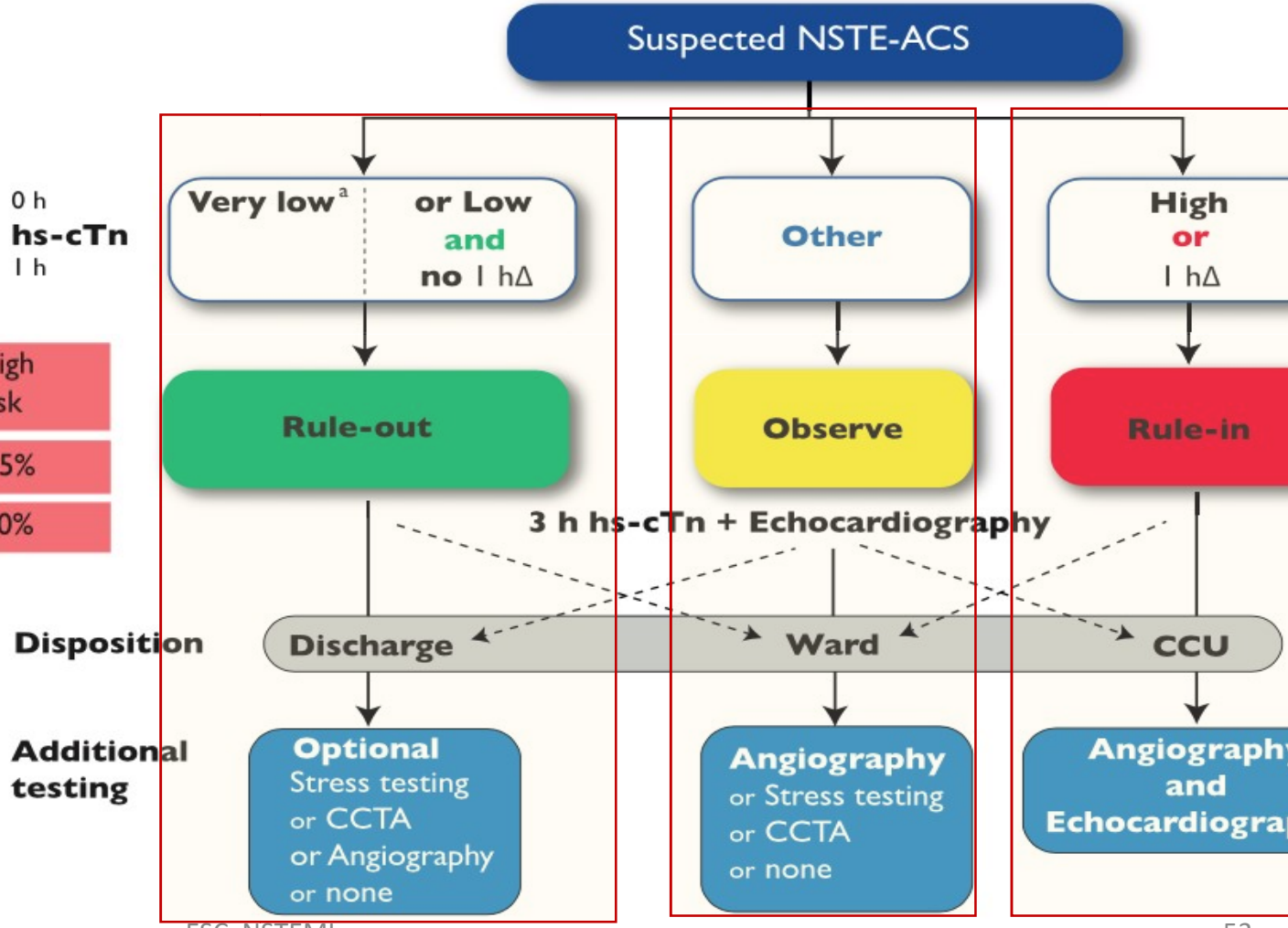
0 h/2 h
Hs-cTn
algorithm

C 0 h/1 h algorithm with blood sampling at 0 h and 1 h is recommended if an hs-cTn test with a validated 0 h/1 h hm is available.[30,33,35,36,39,68,69,75,76]	I

pared with standard cardiac troponin assays, hs-cTn assays:
Have higher NPV for AMI.
Reduce the 'troponin-blind' interval leading to earlier detection of AMI.
Result in ~4% absolute and ~20% relative increases in the detection of type 1 MI and a corresponding decrease in the diagnosis of unstable angi
Are associated with a 2-fold increase in the detection of type 2 MI.

Prise en charge Initiale selon la Probabilité de NSTEMI

k of	Low risk	Intermediate risk	High risk
t index visit	<0.3%	≈10%	>65%
ay MACE	<0.5%	15–20%	>70%



Stratification du risque ischémique: Pronostic

ommendations on biomarker measurements for prognostic stratification

ommendations	Class[a]	L
nd its diagnostic role, it is recommended to measure hs-cTn serially for the estimation of prognosis.[12,13,119,120]	I	
suring BNP or NT-proBNP plasma concentrations should be considered to gain prognostic information.[121,125,126]	IIa	
measurement of additional biomarkers, such as mid-regional pro-A-type natriuretic peptide, high-sensitivity active protein, mid-regional pro-adrenomedullin, GDF-15, copeptin, and h-FABP is not recommended for ne risk or prognosis assessment.[50,127,129]	III	
re to risk stratify in NSTE-ACS		
CE risk score models should be considered for estimating prognosis.[137–139]	IIa	
use of risk scores designed to evaluate the benefits and risks of different DAPT durations may be considered.[153,154]	IIb	
stimate bleeding risk, the use of scores may be considered in patients undergoing coronary angiography.[155,156]	IIb	

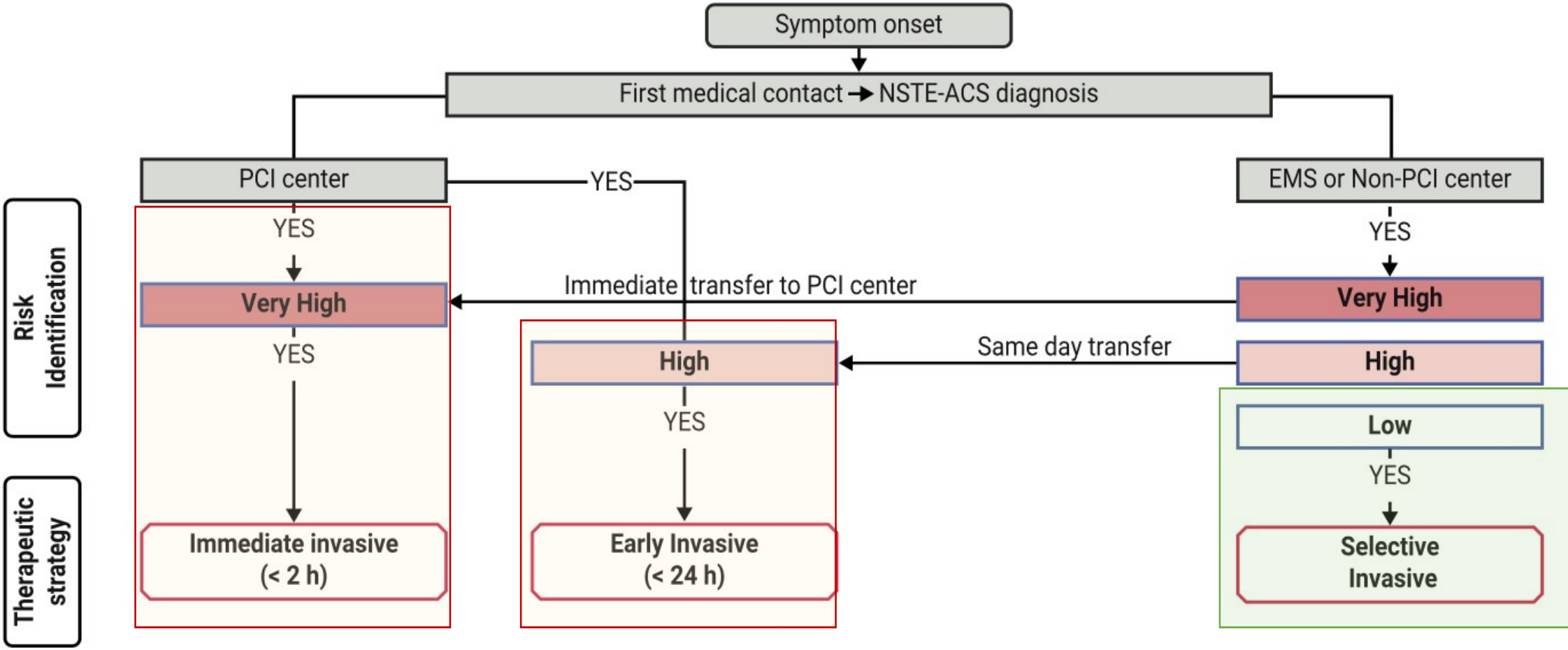
Stratification du risque ischémique: Pronostic
3 catégories de risque de NSTE-ACS

Very high risk
- Haemodynamic instability
- Cardiogenic shock
- Recurrent/refractory chest pain despite medical treatment
- Life-threatening arrhythmias
- Mechanical complications of MI
- Acute heart failure clearly related to NSTE-ACS
- ST-segment depression >1 mm/6 leads plus ST-segment elevation aVr and/or V1

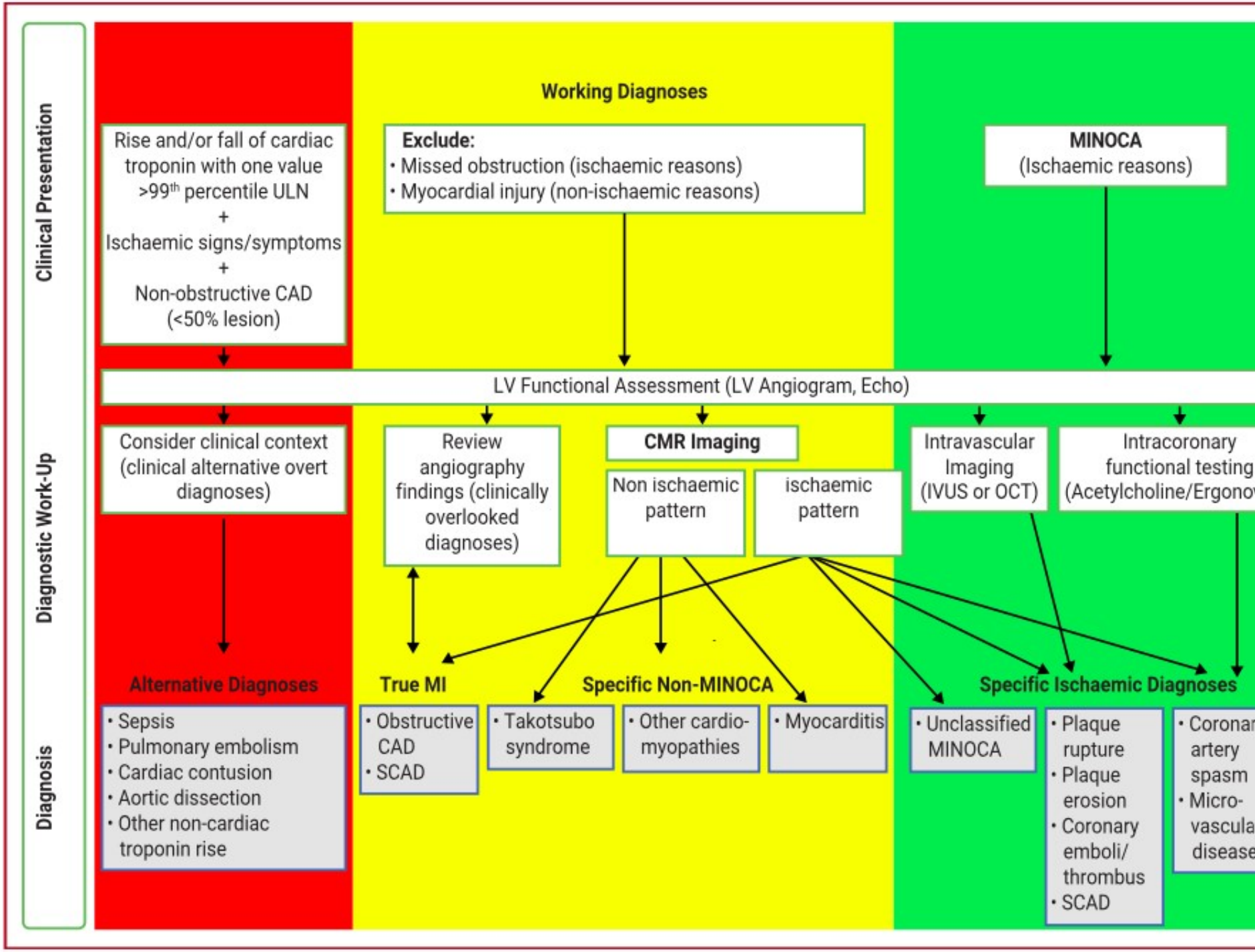
High risk
- Established NSTEMI diagnosis
- Dynamic new or presumably new contiguous ST/T-segment changes (symptomatic or silent)
- Resuscitated cardiac arrest without ST-segment elevation or cardiogenic shock
- GRACE risk score >140

Low risk
Lack of any of the very high or high risk characteristics

Timing de la Coronarographie: selon la catégorie de risque initiale



gorithme
agnostic



Conclusions

- Marqueur très sensible et spécifique d'atteinte myocardique ne préjugeant pas du mécanisme lésionnel
- Utilisation très large dans le syndrome coronaire
- Une élévation de la hs-cTn ne correspond pas forcément à un infarctus du myocarde
- Un infarctus myocardique de type I doit être exclu en premier lieu
- Ne pas oublier son utilisation dans les situations suivantes : Embolie pulmonaire - Myopéricardite - Sepsis - Contusion myocardique……..;

Causes d'élévation des troponines

Myocardite et péricardite	50 – 70 %
Dissection aiguë de l'aorte	25 %
« Hyper-exercices »	8 – 75 %
Insuffisance cardiaque	29 – 89 %
Greffé cardiaque	50 %
Insuffisance rénale	4 – 32 %
Accidents vasculaires cérébraux	17 – 65 %
Sepsis graves	25 – 85 %
Chirurgie cardiaque	100 %
Embolie pulmonaire	32 – 47 %
Contusions myocardiques	17 – 42 %
Ablations par radiofréquence	50 – 100 %
Chimiothérapies anticancéreuses	8 – 32 %
Cathétérismes interventionnels	25 – 37 %
Angioplastie coronaire	13 – 31 %
Chirurgie non cardiaque	5 – 70 %
Nouveau-né de mère hypertendue	5 – 78 %

Merci

MINOCA: Définition

The diagnosis of MINOCA is made in patients with AMI fulfilling the following criteria:

1. AMI (modified from the 'Fourth Universal Definition of Myocardial Infarction' criteria):

- Detection of a rise or fall in cardiac troponin with at least one value above the 99th percentile upper reference limit and
- Corroborative clinical evidence of infarction as shown by at least one of the following:
  a. Symptoms of myocardial ischaemia
  b. New ischaemic electrocardiographic changes
  c. Development of pathological Q waves
  d. Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic cause
  e. Identification of a coronary thrombus by angiography or autopsy

2. Non-obstructive coronary arteries on angiography:

- Defined as the absence of obstructive disease on angiography (i.e. no coronary artery stenosis ≥50%) in any major epicardial vessel[a]

This includes patients with:

- Normal coronary arteries (no angiographic stenosis)
- Mild luminal irregularities (angiographic stenosis <30% stenoses)
- Moderate coronary atherosclerotic lesions (stenoses >30% but <50%)

3. No specific alternate diagnosis for the clinical presentation:

- Alternate diagnoses include, but are not limited to, non-ischaemic causes such as sepsis, pulmonary embolism, and myocarditis