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# **IX CONGRESSO NAZIONALE ECOCARDIOCHIRURGIA 2017**

28 - 29 MARZO 2017 MILANO, 27 - 28 - 29 MARZO 2017 MILANO, 27 - 28 - 29 MARZO 2017 MILANO  
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17 MILANO, 27 - 28 - 29 MARZO 2017 **MILANO, 27 - 28 - 29 MARZO 2017**

## **MINI CORSO MEDICINA D'URGENZA**

### **Il trattamento delle urgenze cardiorespiratorie time-dipendenti**

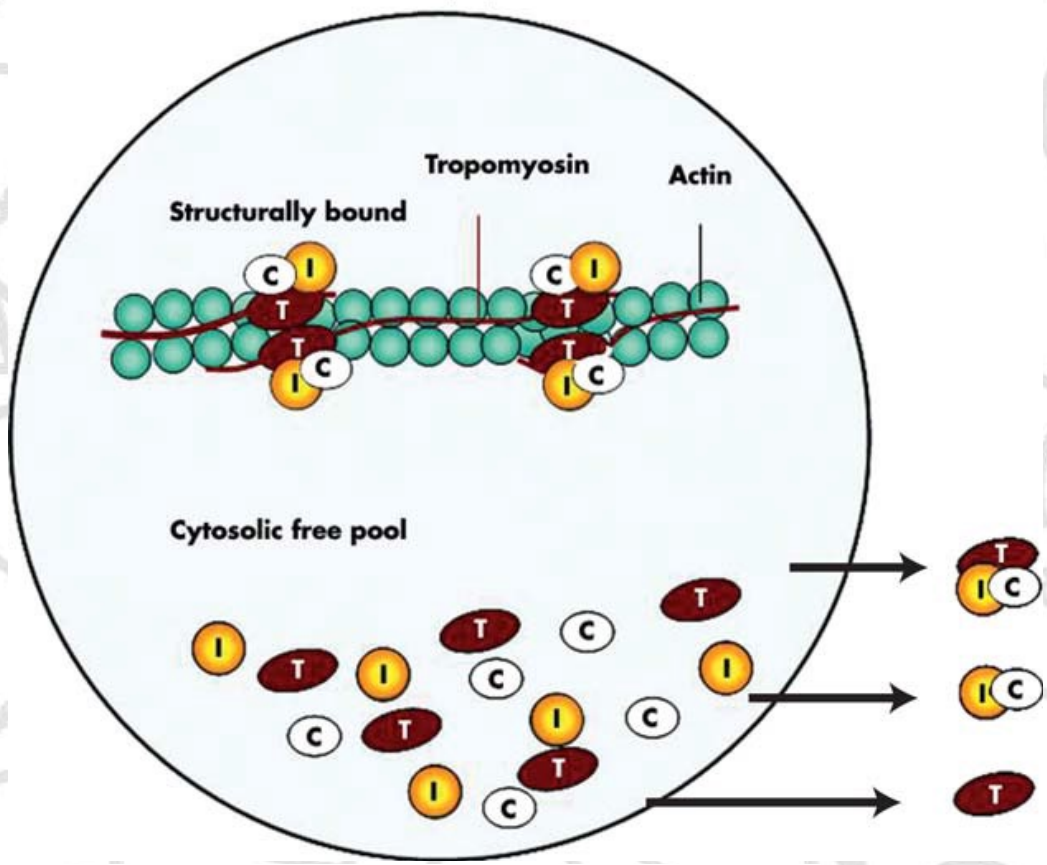
**Troponina ultrasensibile in PS: cosa cambia nella processazione del paziente  
con dolore toracico in PS**

**29/03/2017**

Andrea Barbieri  
U.O. Cardiologia  
Policlinico di Modena

**SERVIZIO SANITARIO REGIONALE  
EMILIA-ROMAGNA**  
Azienda Ospedaliera Policlinico di  
Modena





**Table 1**

**Pathobiological Classification of Types of Potential Mechanisms Causing Troponin Elevations**

Type 1	Myocyte necrosis
Type 2	Apoptosis
Type 3	Normal myocyte turnover
Type 4	Cellular release of proteolytic troponin degradation products
Type 5	Increased cellular wall permeability
Type 6	Formation and release of membranous blebs

J Am Coll Cardiol 2011;57::2406–8

Eur Heart J 2011;32:404–411

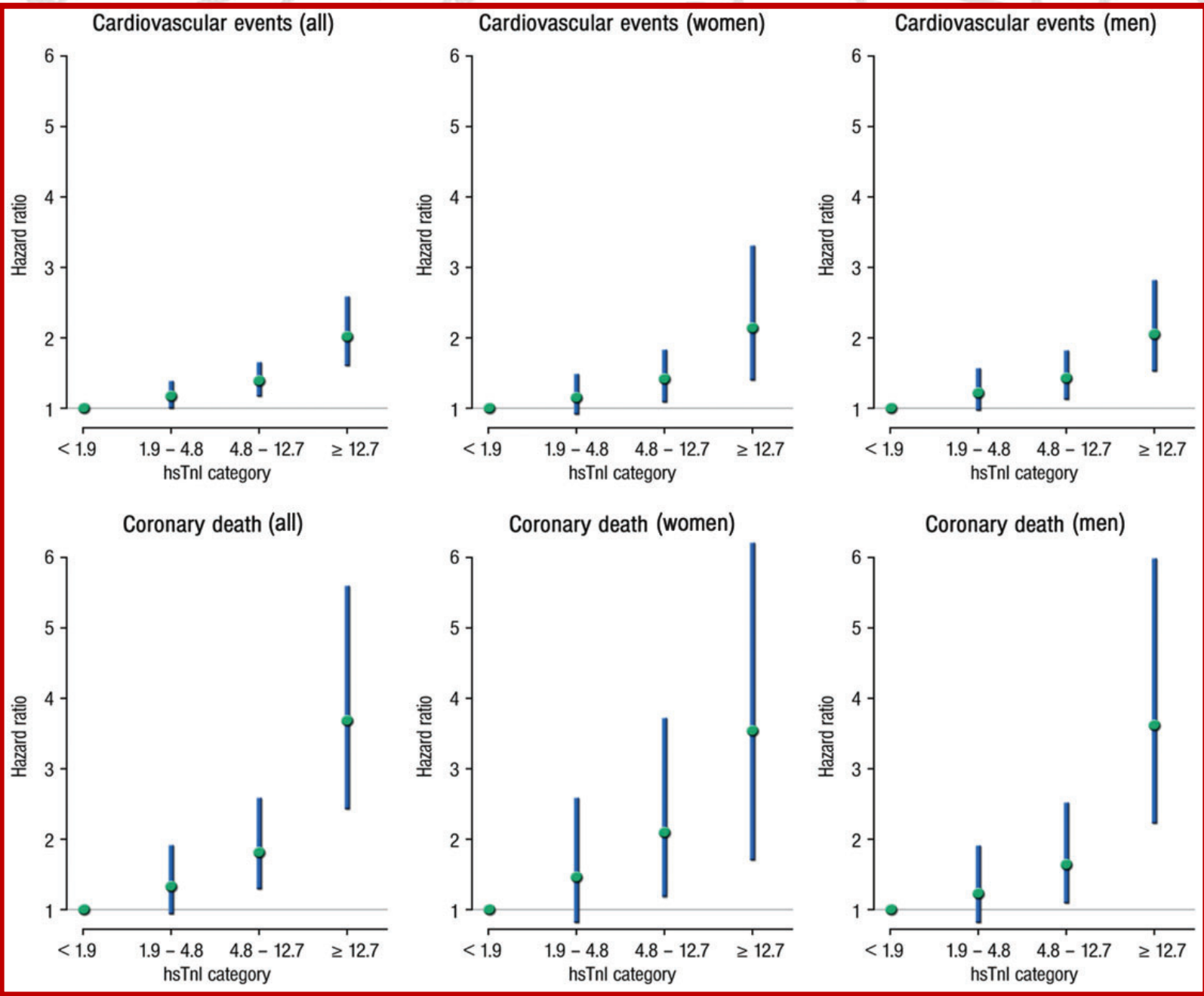


European Heart Journal (2014) **35**, 271–281  
doi:10.1093/eurheartj/eh406

**CLINICAL RESEARCH**  
*Prevention and epidemiology*

# High population prevalence of cardiac troponin I measured by a high-sensitivity assay and cardiovascular risk estimation: the **MORGAM Biomarker Project Scottish Cohort**

**Tanja Zeller<sup>1</sup>, Hugh Tunstall-Pedoe<sup>2\*</sup>, Olli Saarela<sup>3,4</sup>, Francisco Ojeda<sup>1</sup>, Renate B. Schnabel<sup>1</sup>, Tarja Tuovinen<sup>4</sup>, Mark Woodward<sup>2,5,6</sup>, Allan Struthers<sup>7</sup>, Maria Hughes<sup>8</sup>, Frank Kee<sup>8</sup>, Veikko Salomaa<sup>4</sup>, Kari Kuulasmaa<sup>4</sup>, and Stefan Blankenberg<sup>1\*</sup>, for the MORGAM Investigators**



# Improving our understanding of hsTn

1. These strategies do ***not apply to everyone*** (varies widely from  $\approx 9.8\%$  to  $77\%$ )
2. Understand the ***analytical performance*** of the assay (embedded in the local standard ED operating procedures)
3. Understand the ***performance metrics***
4. It is not a definitive diagnostic strategies such as pregnancy-test but a ***risk stratification strategies***

# Improving our understanding of hsTn

1. These strategies do ***not apply to everyone*** (varies widely from  $\approx 9.8\%$  to 77%)
2. Understand the *analytical performance* of the assay (embedded in the local standard ED operating procedures)
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4. It is not a definitive diagnostic strategies such-as pregnancy-test but a *risk stratification strategies*

Low Likelihood

High Likelihood

1. Presentation



2. ECG



3. Troponin

-

+

++

4. Diagnosis

Noncardiac

UA

Other  
Cardiac

NSTEMI

STEMI

# Caveats: high-risk features

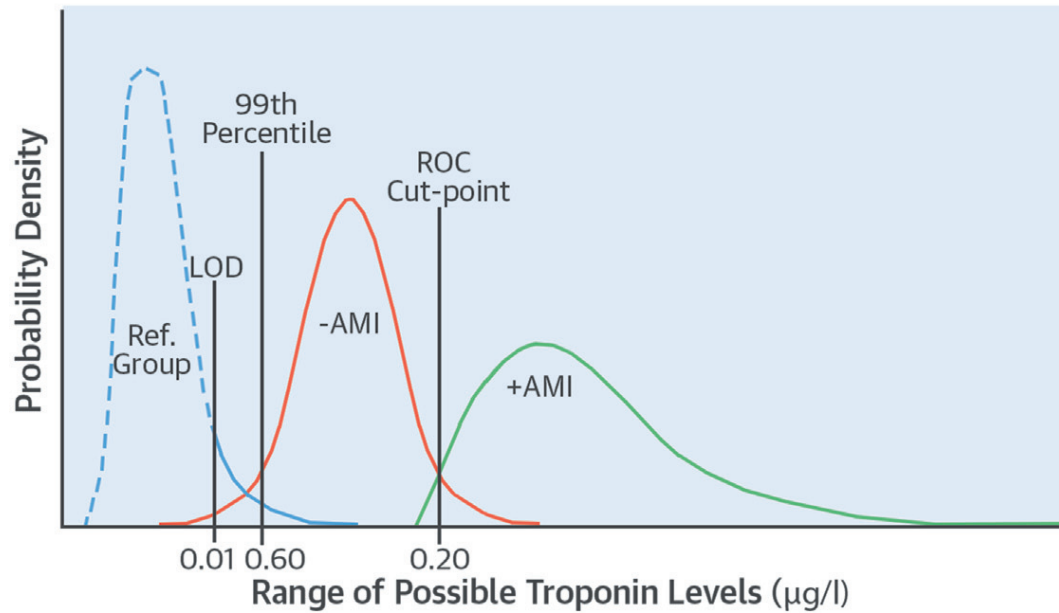
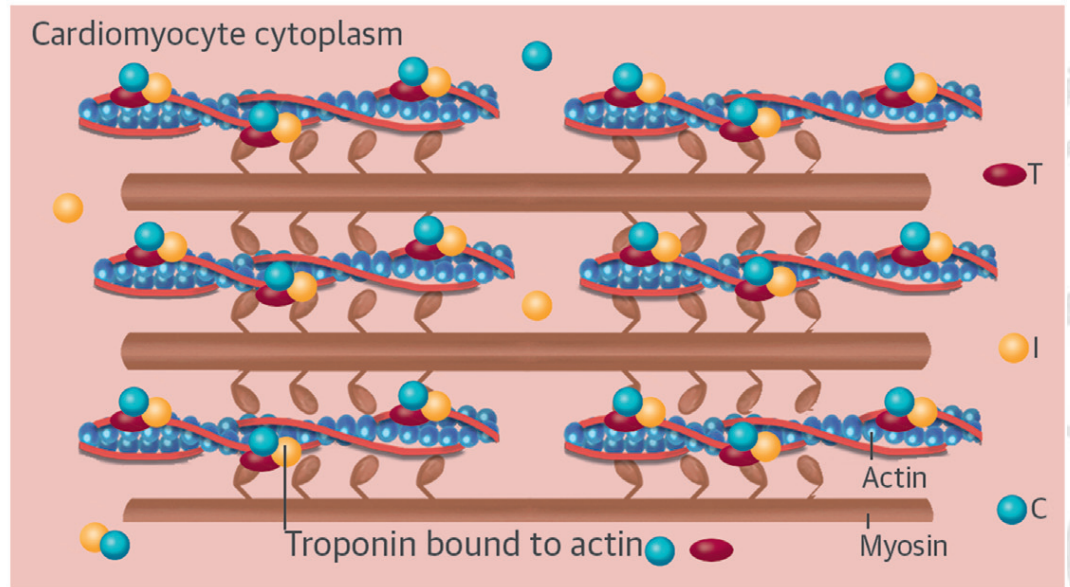
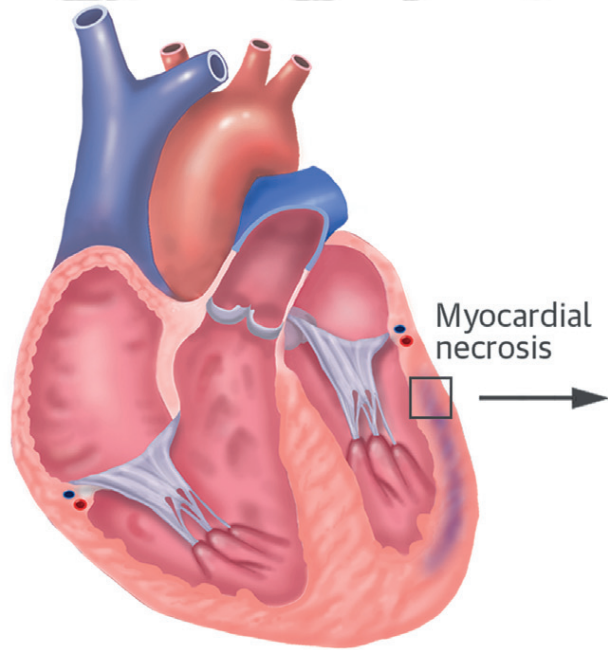
- *Prior history of CAD (PCI)* or positive cardiac exercise test result
- ECG with ST-segment depression 0.05-0.10 mV and/or flat or inverted T waves <0.20 mV deep
- Typical angina symptoms, acceleration of previously stable angina
- LBBB, pacing rhythm
- Cardiovascular risk factors (diabetes, CKD)
- Signs of atherosclerosis on physical examination
- .....

**Clinical judgment by an experienced ED physician remains crucial**



# Improving our understanding of hsTn

1. These strategies do *not apply to everyone* (varies widely from  $\approx 9.8\%$  to  $77\%$ )
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## 1. Analytical sensitivity (LOD)

Concentrations below the 99th percentile detectable above the assay's LOD (% of healthy individuals in the population of interest)

“contemporary”: <50% (“sensitive”: ≈20% to 50%)

“high sensitive”: >50%

1st hsTn generation: 50-75%

2nd hsTn generation: 75-95%

3d hsTn generation: >95%

## 2. Precision (CV%) 99° percentile

“guideline acceptable”  $\leq 10\%$

“clinically usable” 10-20%

“not acceptable” >20%

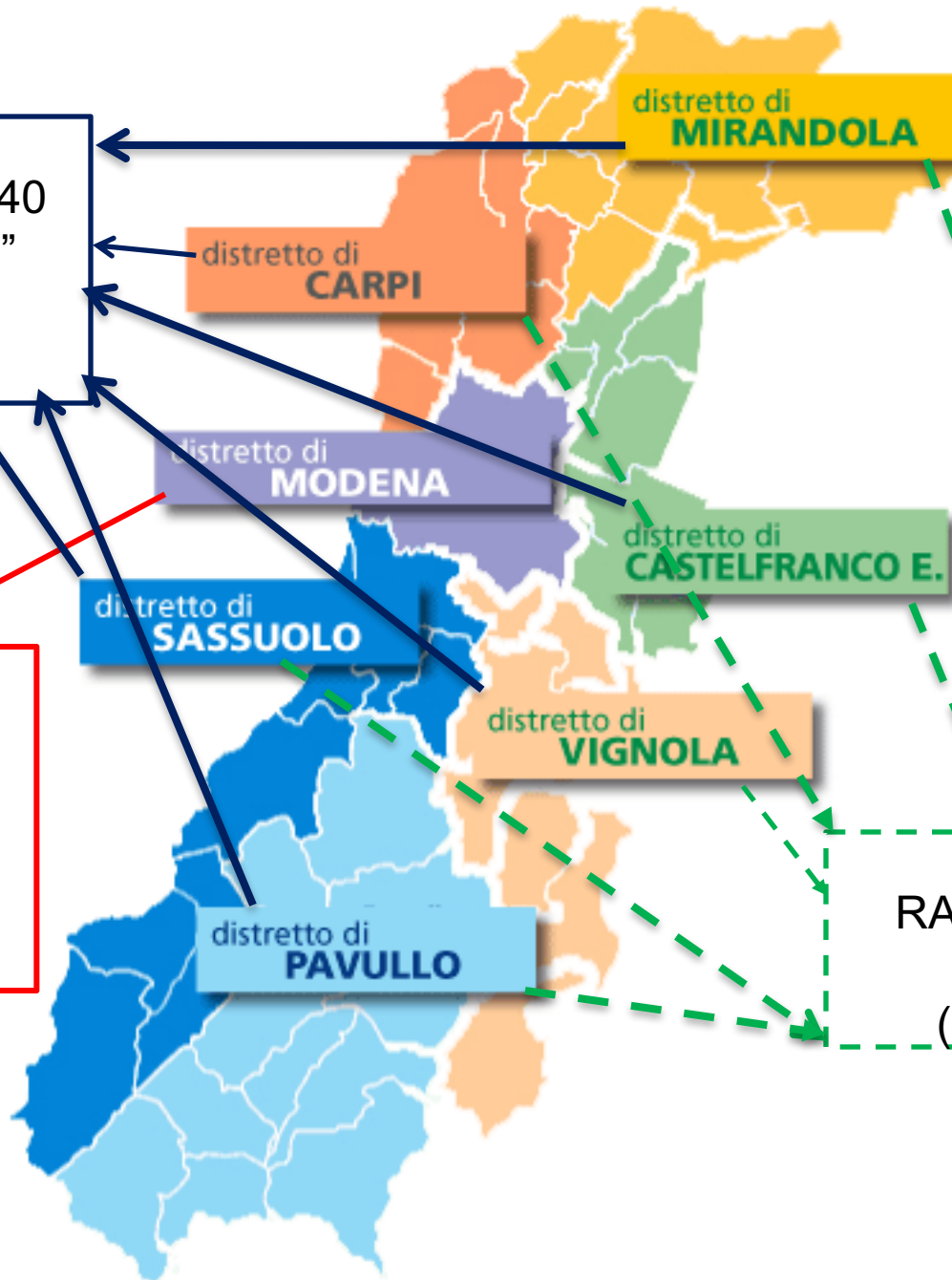
## 3. Analytical variation (delta)

Pre-analytic biological variability

	Limit of Detection (ng/L)	99% (CV) (ng/L)	10% CV (ng/L)
<b>Hs-cTn-T</b>			
Roche Elecsys	5.0	14 (13%)	13
<b>Hs-cTn-I</b>			
Abbot ARCHITECT	1.2	16 (5.6%)	3.0
Beckman ACCESS	2 to 3	8.6 (10%)	8.6
Mitsubishi Pathfast	8.0	29 (5%)	14
Nanosphere	0.2	2.8 (9.5%)	0.5
Radiometer AQT90	9.5	23 (17.7%)	39
Singulex Erenna	0.09	10.1 (9.0%)	0.88
Siemens Vista	0.5	9 (5.0%)	3
Siemens Centaur	6.0	40 (10%)	30

**NOCSAE:** Beckman  
Coulter AccuTnl+3 (< 40  
ng/L) “clinically usable”  
10-20% “hs-first  
generation”.

**Policlinico:** Ortho-  
Clinical Diagnostics  
Vitros ES  
(<34 ng/L) “guideline  
acceptable” ≤10%  
“contemporary”



POCT  
RADIOMETER  
AQT 90  
(< 20 ng/L)

# ED/Laboratory collaboration is essential

Issues to be shared with the laboratory include:

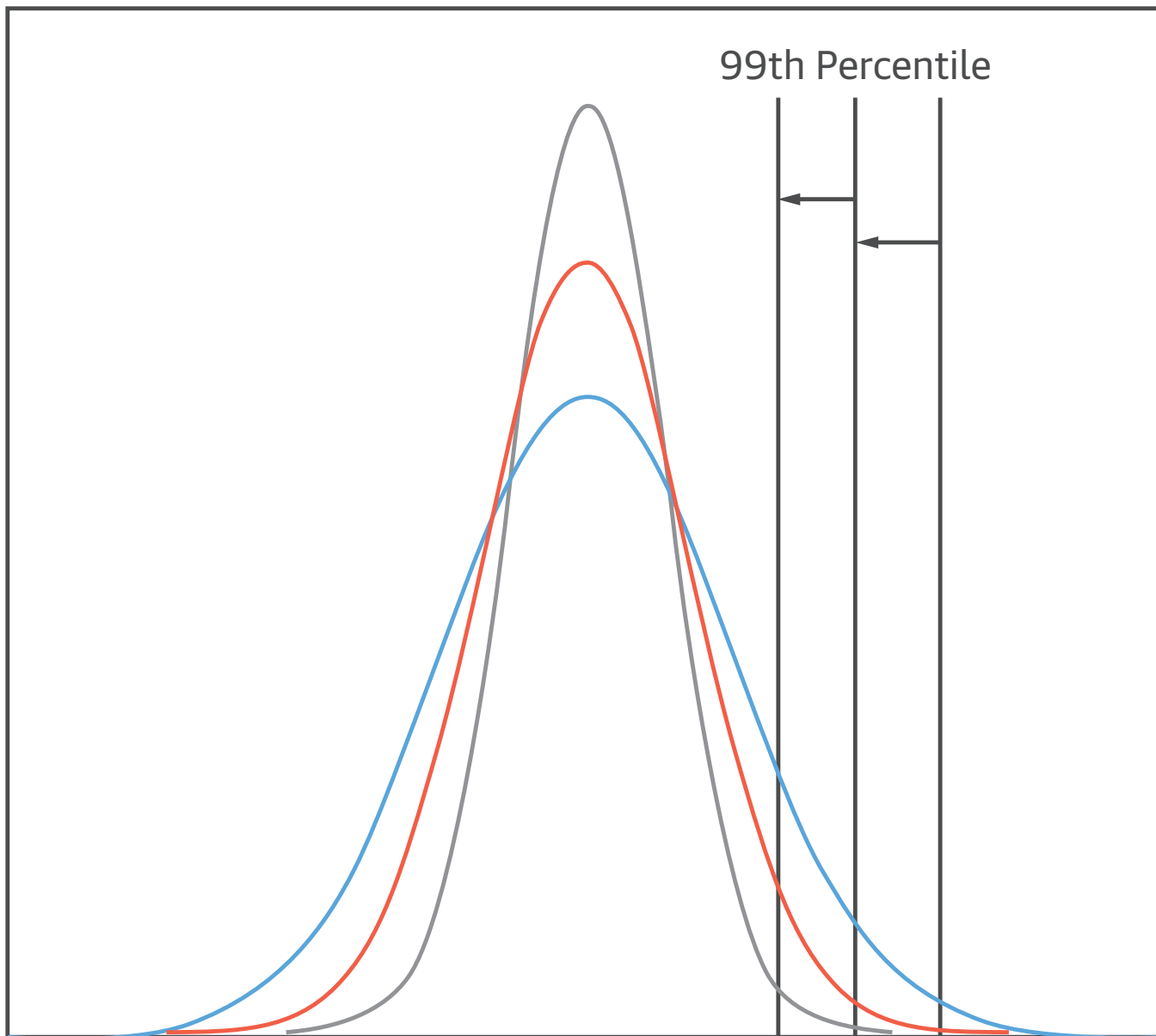
- when and how to evaluate potential FP and FN values
- what cut-off values to use
- how to decide when a changing pattern is present

Many without a dynamic pattern can be evaluated as outpatients, but only if there is agreement concerning a facile pathway for that activity

# Improving our understanding of hsTn

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2. Understand the *analytical performance* of the assay (embedded in the local standard ED operating procedures)
3. Understand the ***performance metrics***
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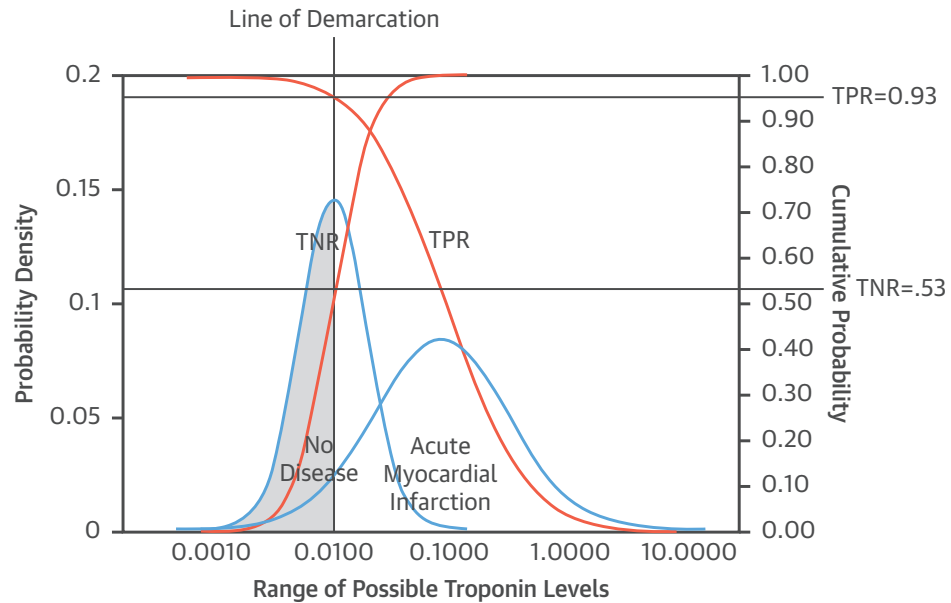
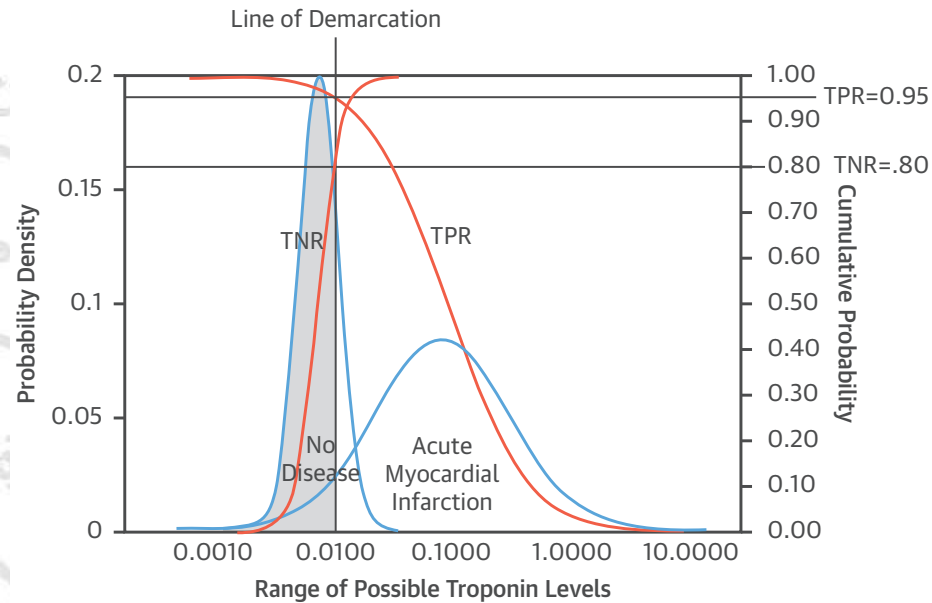
Probability Density



Range of Possible Troponin Levels



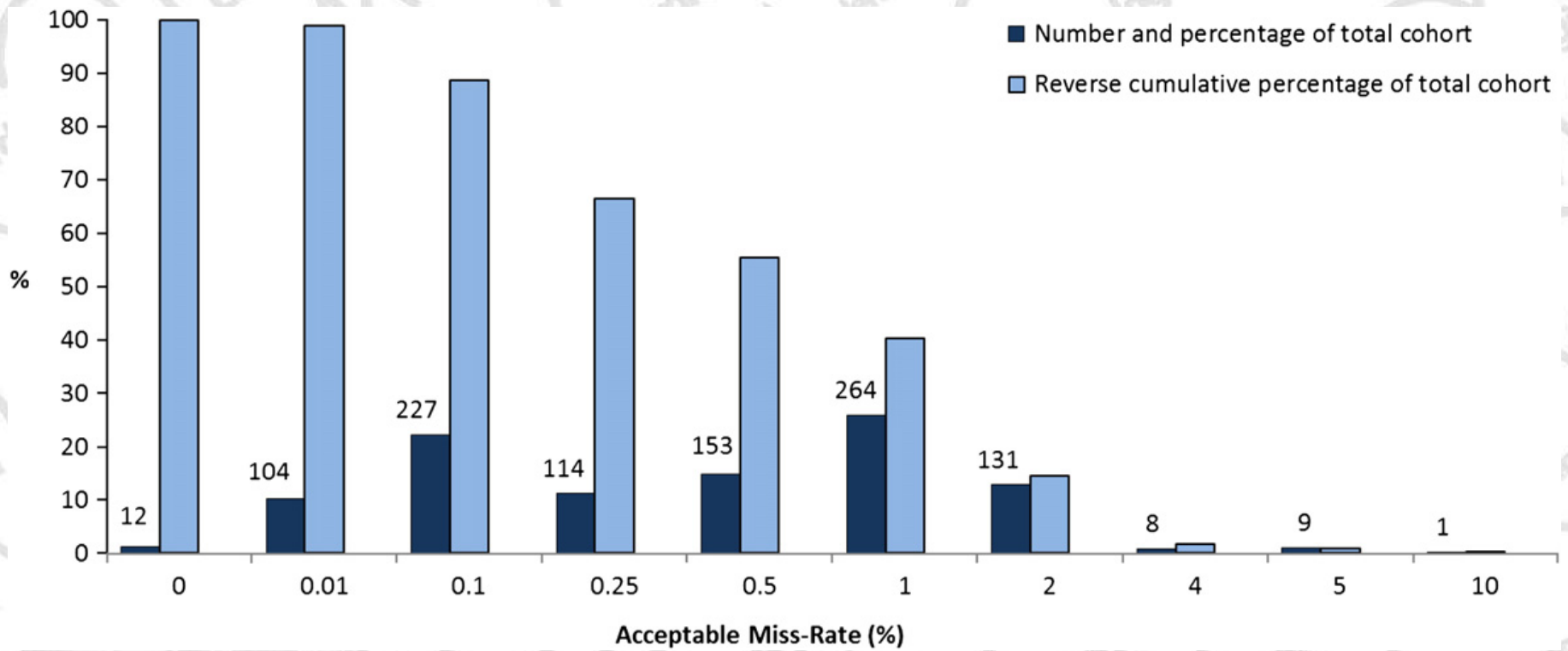
# Spectrum bias



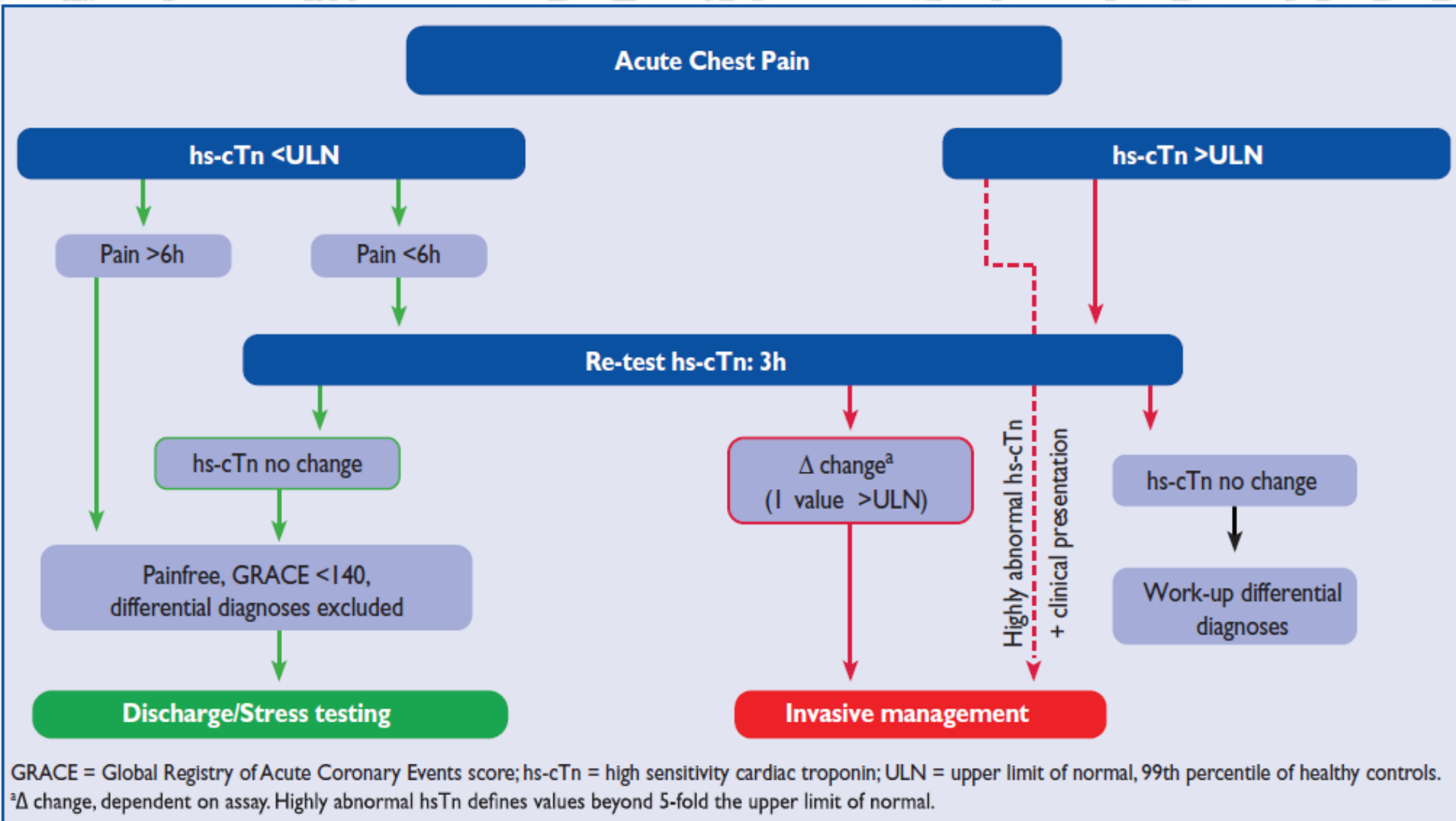
**Table 2. Summary of Biomarker Strategies for Rapid Assessment of Patients With Potential ACS in the ED**

	Very Low cTn	cTn and Copeptin	0- and 1-h Algorithm	0- and 2-h Algorithm	2-h ADP	0- and 3-h ESC
Clinical scoring system	None	None	None	None	TIMI score $\leq 1$ ECG normal at 0 and 2 h	GRACE $<140$ and pain free
Blood draws, n	1	1	2*	2*	2*	2*
Indication	Rule out	Rule out	Rule out and rule in	Rule out and rule in	Rule out	Rule out and rule in
NPV for AMI, %	98–100	92.4–99 96–99 with hs-cTn	99.1–100	99.5–99.9	99.1–100†	99.6–100
Eligible population size	+(+)	++	+++	+++	++	++(+)
Biomarker rule-out criteria‡						
Using hs-cTnT	hs-cTnT $<5$ ng/L	hs-cTnT $<14$ ng/L AND copeptin $<10$ pmol/L	hs-cTnT $<12$ ng/L AND 1-h $\Delta <3$	hs-cTnT $<14$ ng/L at 0 and 2 h AND 2-h $\Delta <4$	hs-cTnT $<14$ ng/L at 0 and 2 h	hs-cTnT $<14$ ng/L at 0 and 3 h
Using hs-cTnI	hs-cTnI $<2$ – $5$ ng/L	hs-cTnI $<26$ ng/L AND copeptin $<10$ pmol/L	hs-cTnI $<5$ ng/L AND 1-h $\Delta <2$	hs-cTnI $<6$ ng/L at 0 and 2 h AND 2-h $\Delta <2$	hs-cTnI $<26$ ng/L at 0 and 2 h	hs-cTnI $<26$ ng/L at 0 and 3h
Biomarker rule-in criteria						
Using hs-cTnT			hs-cTnT $\geq 52$ ng/L OR 1-h $\Delta \geq 5$	hs-cTnT $\geq 53$ ng/L OR 2-h $\Delta \geq 10$		
Using hs-cTnI			hs-cTnI $\geq 52$ ng/L OR 1-h $\Delta \geq 5$	hs-cTnI $\geq 64$ ng/L OR 2-h $\Delta \geq 15$		
Feasibility	High	Low; Requires 2 biomarkers requiring different analyzers	High	High	Medium; Requires use of TIMI score	Medium; Requires GRACE score

# Acceptable miss-rate of MACEs



# 0 h/3 h rule-out algorithm of NSTEMI-ACS using hs-cTn



# Guideline endorsement of advanced imaging when ACS is suspected but ECG and biomarker are inconclusive

Modality	Guidelines	Endorsement
2D-TTE	<ul style="list-style-type: none"> <li>• ESC guidelines for NSTEMI-ACS (2011)<sup>12</sup></li> <li>• ACCF/AHA/AHA Appropriate Use Criteria for Echocardiography (2011)<sup>14</sup></li> </ul>	<ul style="list-style-type: none"> <li>• Primary bedside modality</li> <li>• To assess resting RWMA</li> </ul>
Stress Echo	<ul style="list-style-type: none"> <li>• ESC guidelines for NSTEMI-ACS (2011)<sup>12</sup></li> <li>• ACCF/AHA/AHA Appropriate Use Criteria for Echocardiography (2011)<sup>14</sup></li> </ul>	<ul style="list-style-type: none"> <li>• In all suspected ACS to assess RWMA</li> </ul>
CTCA	<ul style="list-style-type: none"> <li>• ESC guidelines for NSTEMI-ACS (2011)<sup>12</sup></li> <li>• ACCF/AHA/AHA Appropriate Use Criteria for Cardiac Computed Tomography (2010)<sup>44</sup></li> </ul>	<ul style="list-style-type: none"> <li>• In low/intermediate likelihood of CAD to assess coronary anatomy</li> <li>• In patients with suspected coronary anomalies</li> </ul>
Rest MPS	<ul style="list-style-type: none"> <li>• ESC guidelines for NSTEMI-ACS (2011)<sup>12</sup></li> <li>• ACCF/AHA/AHA Appropriate Use Criteria for Cardiac Radionuclide Imaging (2009)<sup>68</sup></li> </ul>	<ul style="list-style-type: none"> <li>• In all suspected ACS to assess myocardial scar</li> </ul>
CMR	<ul style="list-style-type: none"> <li>• ESC guidelines for NSTEMI-ACS (2011)<sup>12</sup></li> <li>• ACCF/AHA/AHA Appropriateness Criteria for Cardiac Computed Tomography and Cardiac Magnetic Resonance Imaging (2006)<sup>103</sup></li> </ul>	<ul style="list-style-type: none"> <li>• In intermediate likelihood of CAD to assess myocardial scar or RWMA</li> <li>• In patients with suspected coronary anomalies, using MR coronary angiography</li> </ul>

ACCF, American College of Cardiology Foundation; ACS, acute coronary syndrome; ASE, American Society of Echocardiography; CAD, coronary artery disease; CMR, cardiovascular magnetic resonance; CTCA, computed tomography coronary angiography; ECG, electrocardiogram; Echo, echocardiography; MPS, myocardial perfusion scintigraphy; MR, magnetic resonance; NSTEMI, non-ST-segment elevation; RWMA, regional wall-motion abnormalities; TTE, transthoracic echocardiography.

# The “test-threshold”

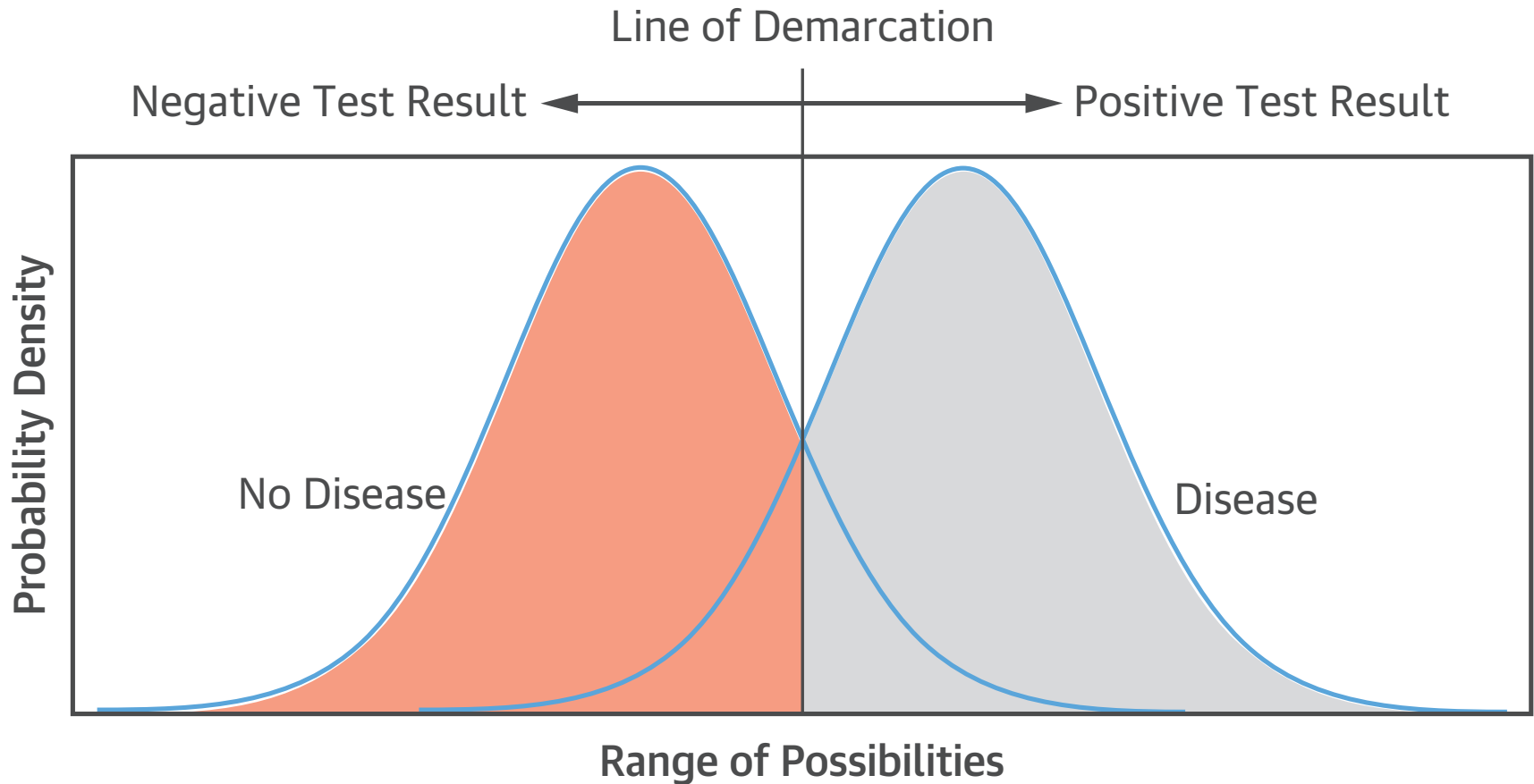
- Represents the point of probability at which the risks from FP testing are balanced with the risk of harm from untreated disease
- Mathematically, patients with a disease probability below the test-threshold will not benefit (and will be harmed) from further testing
- This point is  $\approx 2\%$  for Pts with suspected cardiac chest pain



# **Attempts to improve the clinical sensitivity of hs-cTn assays**

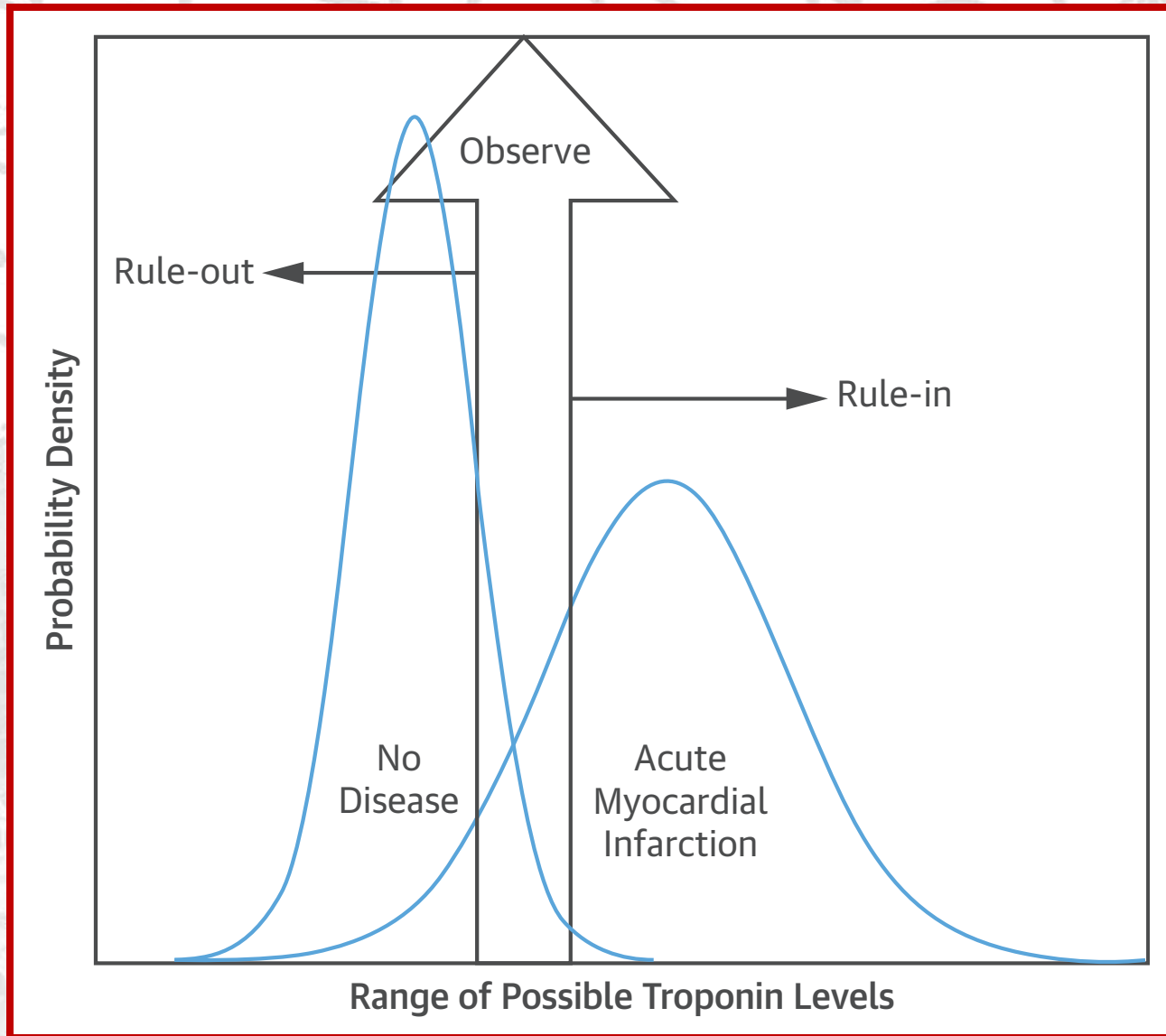


# Single cut-point strategy

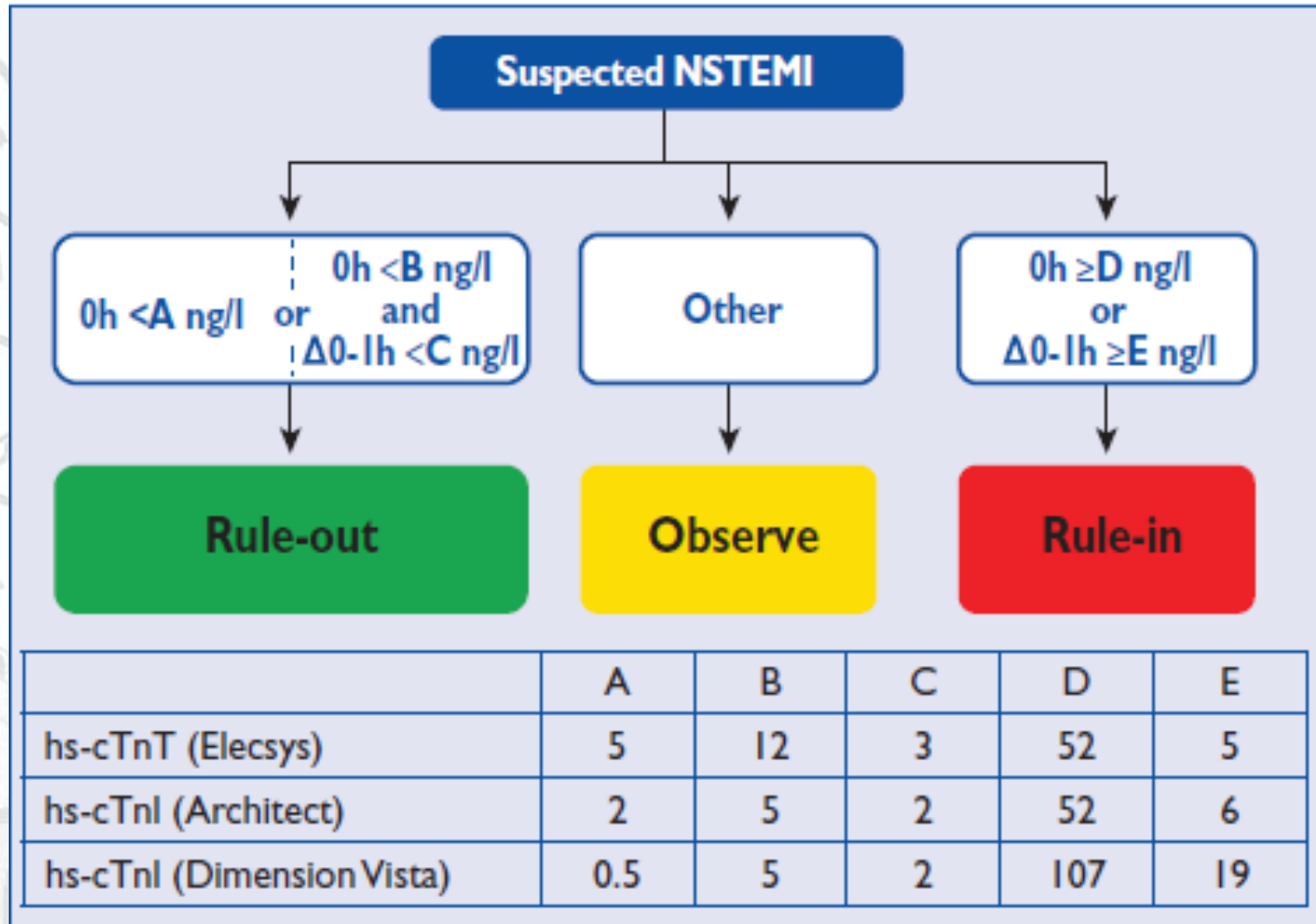




# Double cut-point strategy



# 0 h/1 h rule-out and rule-in algorithms of NSTEMI-ACS using hs-cTn



# High-sensitivity cardiac troponin I at presentation in patients with suspected acute coronary syndrome: a cohort study

Anoop SV Shah\*, Atul Anand\*, Yader Sandoval, Kuan Ken Lee, Stephen W Smith, Philip D Adamson, Andrew R Chapman, Timothy Langdon, Dennis Sandeman, Amar Vaswani, Fiona E Strachan, Amy Ferry, Alexandra G Stirzaker, Alan Reid, Alasdair J Gray, Paul O Collinson, David A McAllister, Fred S Apple, David E Newby, Nicholas L Mills; on behalf of the High-STEACS investigators†

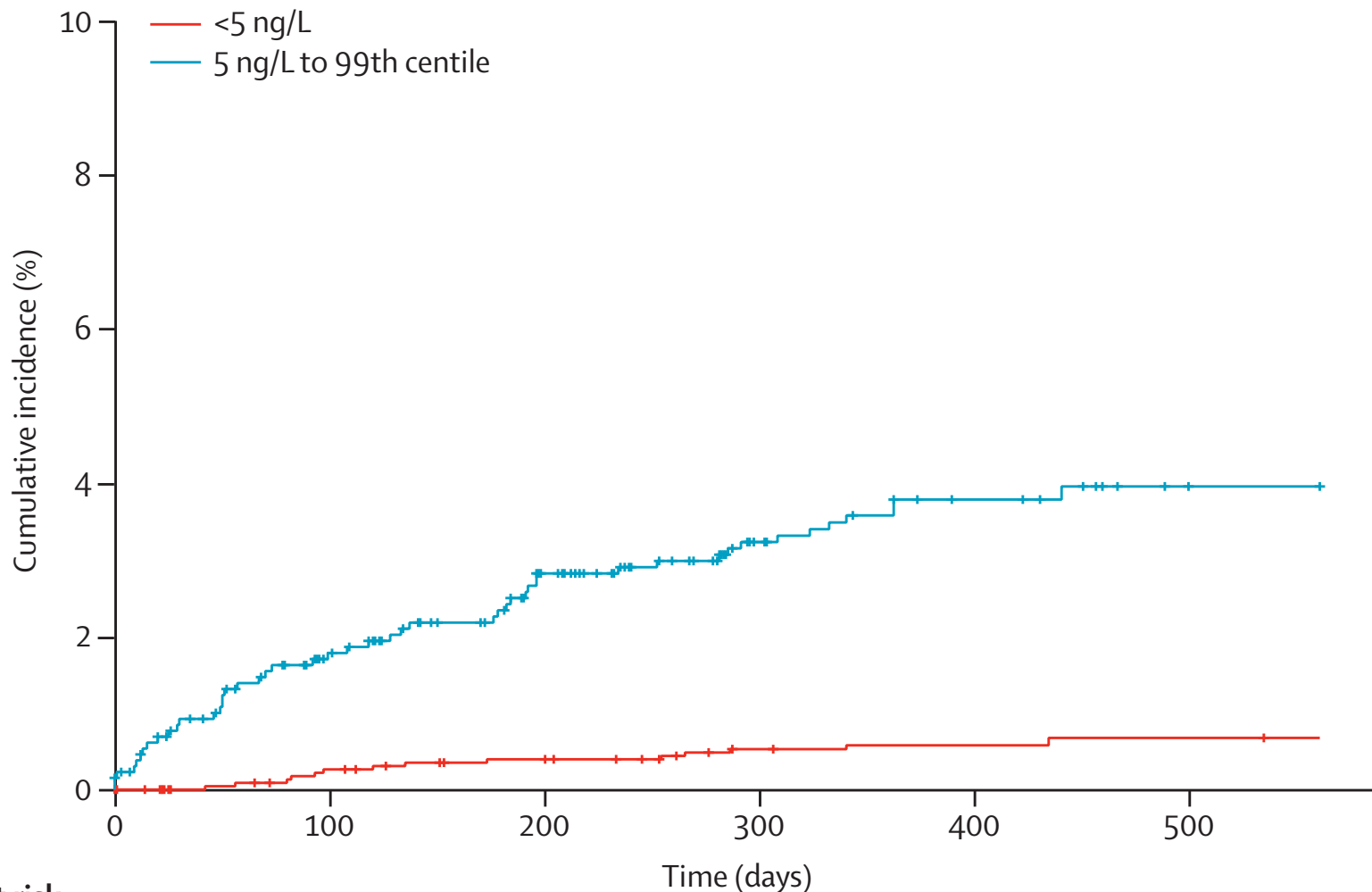
## Summary

**Background** Suspected acute coronary syndrome is the commonest reason for emergency admission to hospital and is a large burden on health-care resources. Strategies to identify low-risk patients suitable for immediate discharge would have major benefits.

**Methods** We did a prospective cohort study of 6304 consecutively enrolled patients with suspected acute coronary syndrome presenting to four secondary and tertiary care hospitals in Scotland. We measured plasma troponin concentrations at presentation using a high-sensitivity cardiac troponin I assay. In derivation and validation cohorts, we evaluated the negative predictive value of a range of troponin concentrations for the primary outcome of index myocardial infarction, or subsequent myocardial infarction or cardiac death at 30 days. This trial is registered with ClinicalTrials.gov (number NCT01852123).

**Findings** 782 (16%) of 4870 patients in the derivation cohort had index myocardial infarction, with a further 32 (1%) re-presenting with myocardial infarction and 75 (2%) cardiac deaths at 30 days. In patients without myocardial infarction at presentation, troponin concentrations were less than 5 ng/L in 2311 (61%) of 3799 patients, with a negative predictive value of 99·6% (95% CI 99·3–99·8) for the primary outcome. The negative predictive value was consistent across groups stratified by age, sex, risk factors, and previous cardiovascular disease. In two independent validation cohorts, troponin concentrations were less than 5 ng/L in 594 (56%) of 1061 patients, with an overall negative predictive value of 99·4% (98·8–99·9). At 1 year, these patients had a lower risk of myocardial infarction and cardiac death than did those with a troponin concentration of 5 ng/L or more (0·6% vs 3·3%; adjusted hazard ratio 0·41, 95% CI 0·21–0·80;  $p < 0·0001$ ).

**Interpretation** Low plasma troponin concentrations identify two-thirds of patients at very low risk of cardiac events who could be discharged from hospital. Implementation of this approach could substantially reduce hospital admissions and have major benefits for both patients and health-care providers.



**Number at risk**

	0	100	200	300	400	500
<5 ng/L	2160	2144	2136	2126	1303	374
5 ng/L to 99th centile	1453	1385	1346	1309	830	297

**Circulation**



**Direct Comparison of Four Very Early Rule-Out Strategies for Acute Myocardial Infarction Using High-Sensitivity Cardiac Troponin I**

Jasper Boeddinghaus, Thomas Nestelberger, Raphael Twerenbold, Karin Wildi, Patrick Badertscher, Janosch Cupa, Tobias Bürge, Patrick Mächler, Sydney Corbière, Karin Grimm, Maria Rubini Giménez, Christian Puelacher, Samyut Shrestha, Dayana Flores Widmer, Jakob Fuhrmann, Petra Hillinger, Zaid Sabti, Ursina Honegger, Nicolas Schaerli, Nikola Kozhuharov, Katharina Rentsch, Òscar Miró, Beatriz López Barbeito, F. Javier Martin-Sanchez, Esther Rodriguez-Adrada, Beata Morawiec, Damian Kawecki, Eva Ganovská, Jiri Parenica, Jens Lohrmann, Wanda Kloos, Andreas Buser, Nicolas Geigy, Dagmar I. Keller, Stefan Osswald, Tobias Reichlin and Christian Müller

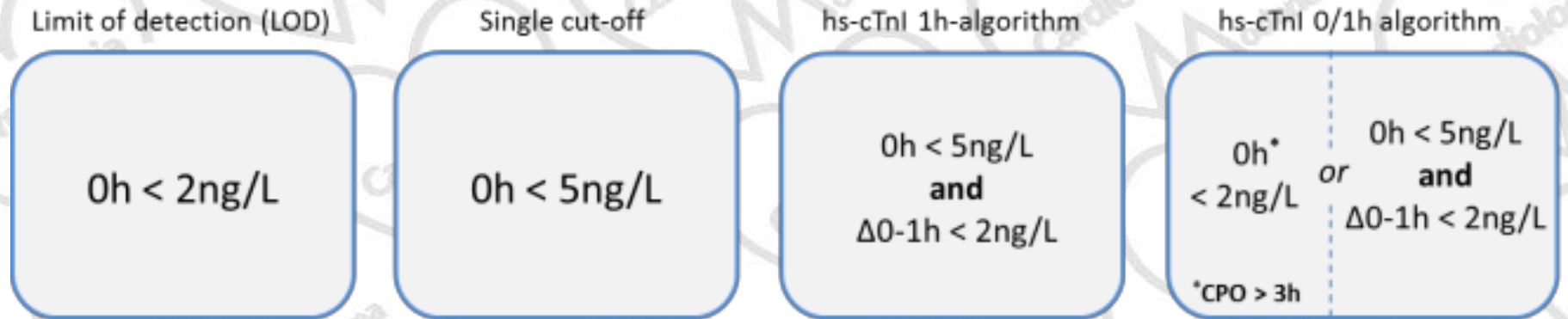
*Circulation*. published online March 10, 2017;

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# Very early rule-out strategies for AMI



1. Very low concentrations ( $<LOD$  for the specific assay)
2. Single cut-off approach
3. 1h-algorithm
4. Combined very-low concentrations and 1h-algorithm

# Suspected NSTEMI

n = 2828

Limit of detection (LOD)

0h < 2ng/L

		NSTEMI	
		-	+
-	1924	451	
+	453	0	

453/2828 (16%)  
**NPV: 100% (99.2-100)**  
**Sens.: 100% (99.2-100)**  
 Prevalence of AMI: 0%

Single cut-off

0h < 5ng/L

		NSTEMI	
		-	+
-	874	438	
+	1503	13	

1516/2828 (54%)  
**NPV: 99.1% (98.5-99.5)**  
**Sens.: 97.1% (95.1-98.3)**  
 Prevalence of AMI: 0.9%

hs-cTnl 1h-algorithm

0h < 5ng/L  
**and**  
 $\Delta 0-1h < 2ng/L$

		NSTEMI	
		-	+
-	925	444	
+	1452	7	

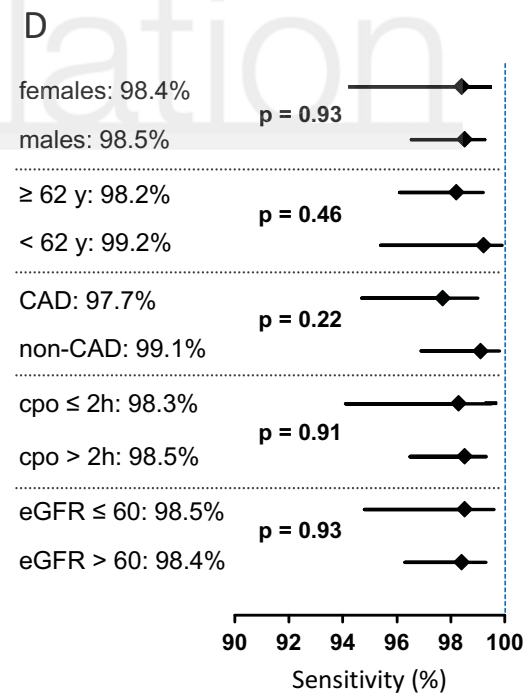
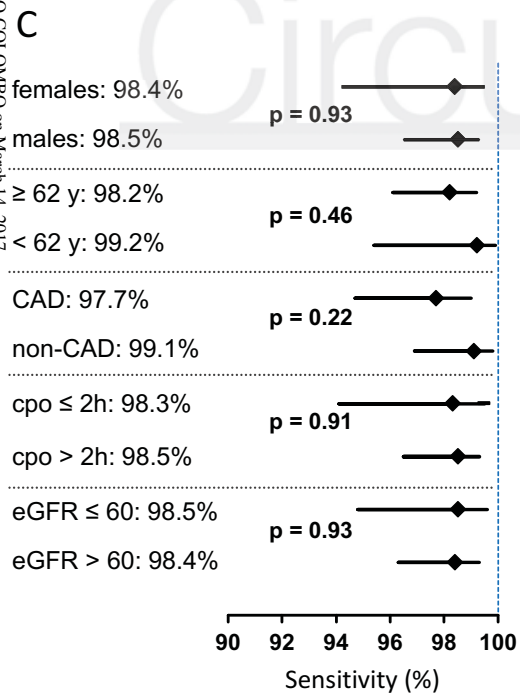
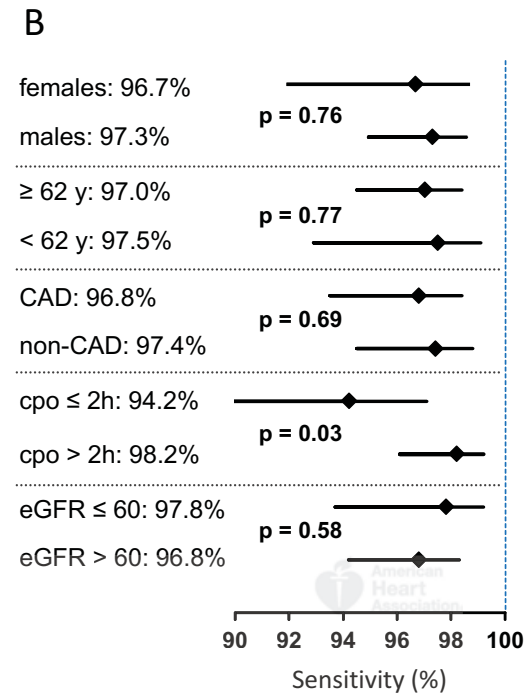
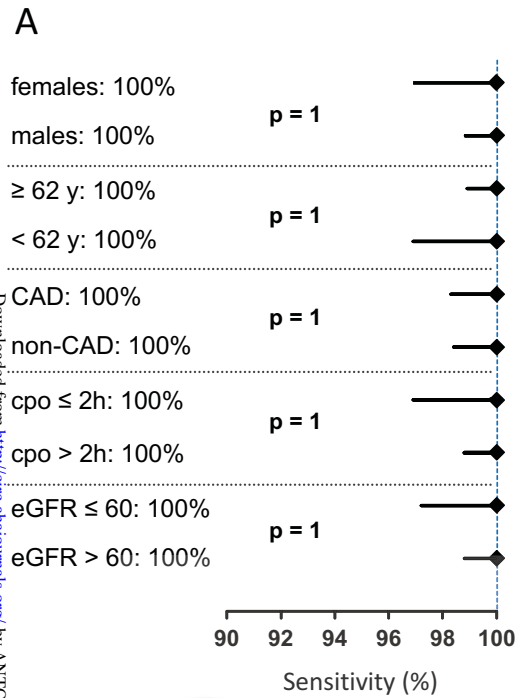
1459/2828 (52%)  
**NPV: 99.5% (99.0-99.8)**  
**Sens.: 98.4% (96.8-99.2)**  
 Prevalence of AMI: 0.5%

hs-cTnl 0/1h algorithm

0h\* < 2ng/L **or** 0h < 5ng/L  
**and**  
 $\Delta 0-1h < 2ng/L$

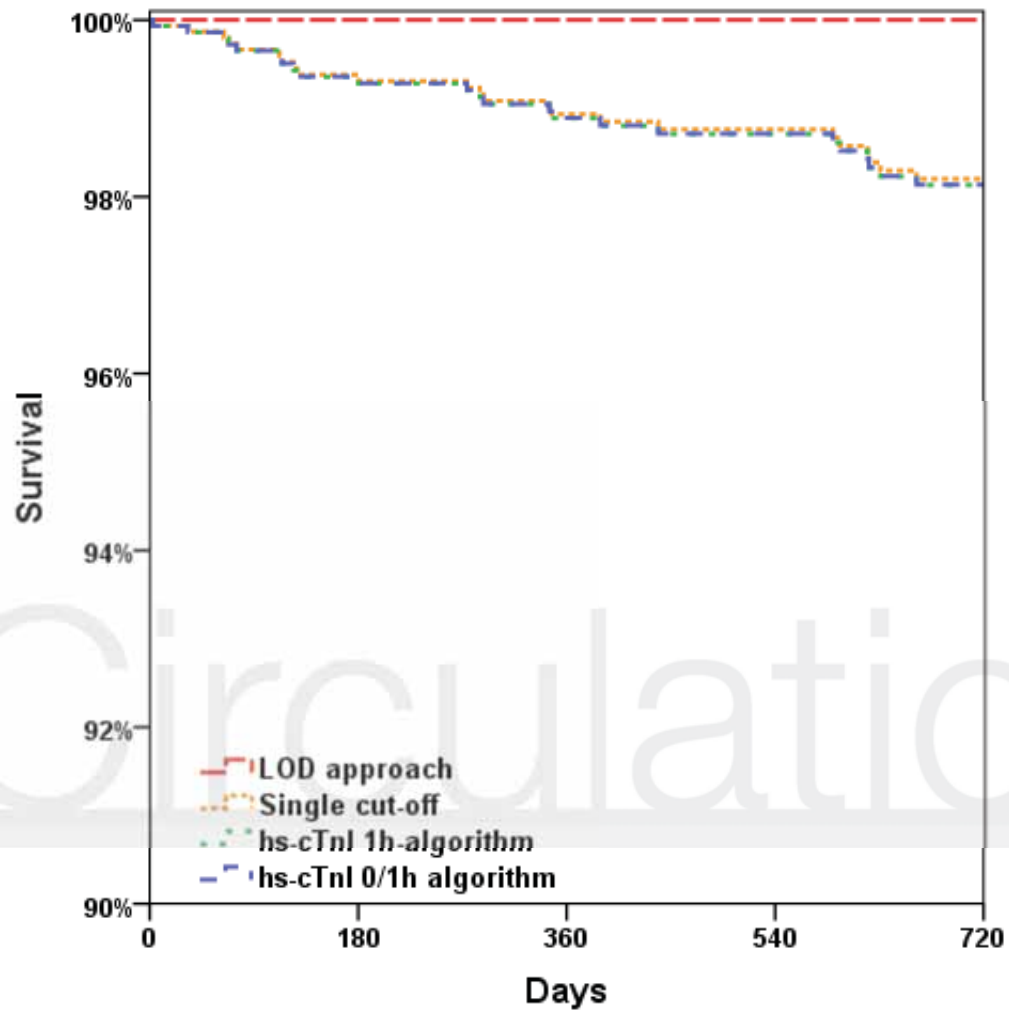
		NSTEMI	
		-	+
-	921	444	
+	1456	7	

1463/2828 (52%)  
**NPV: 99.5% (99.0-99.8)**  
**Sens.: 98.4% (96.8-99.2)**  
 Prevalence of AMI: 0.5%



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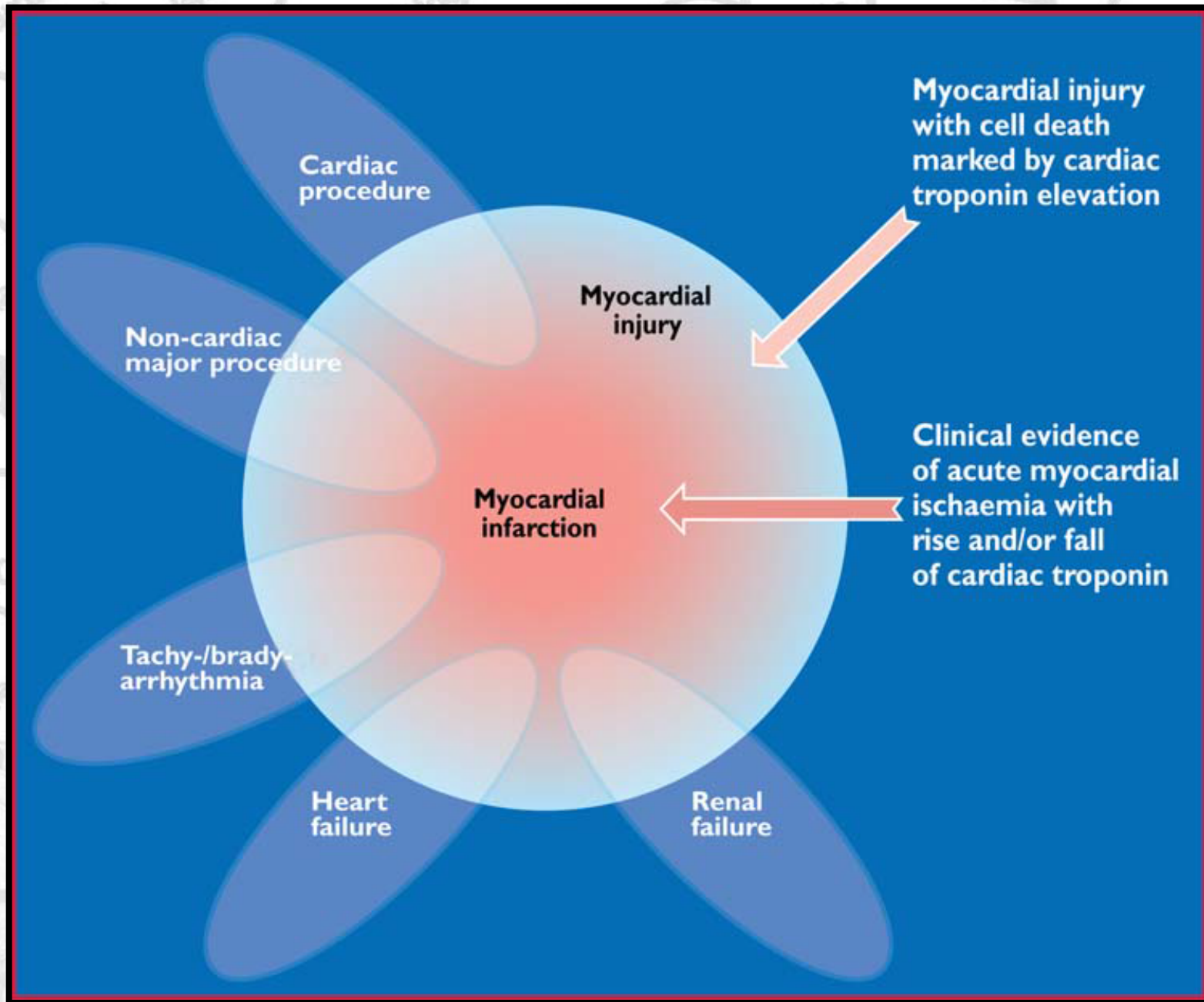


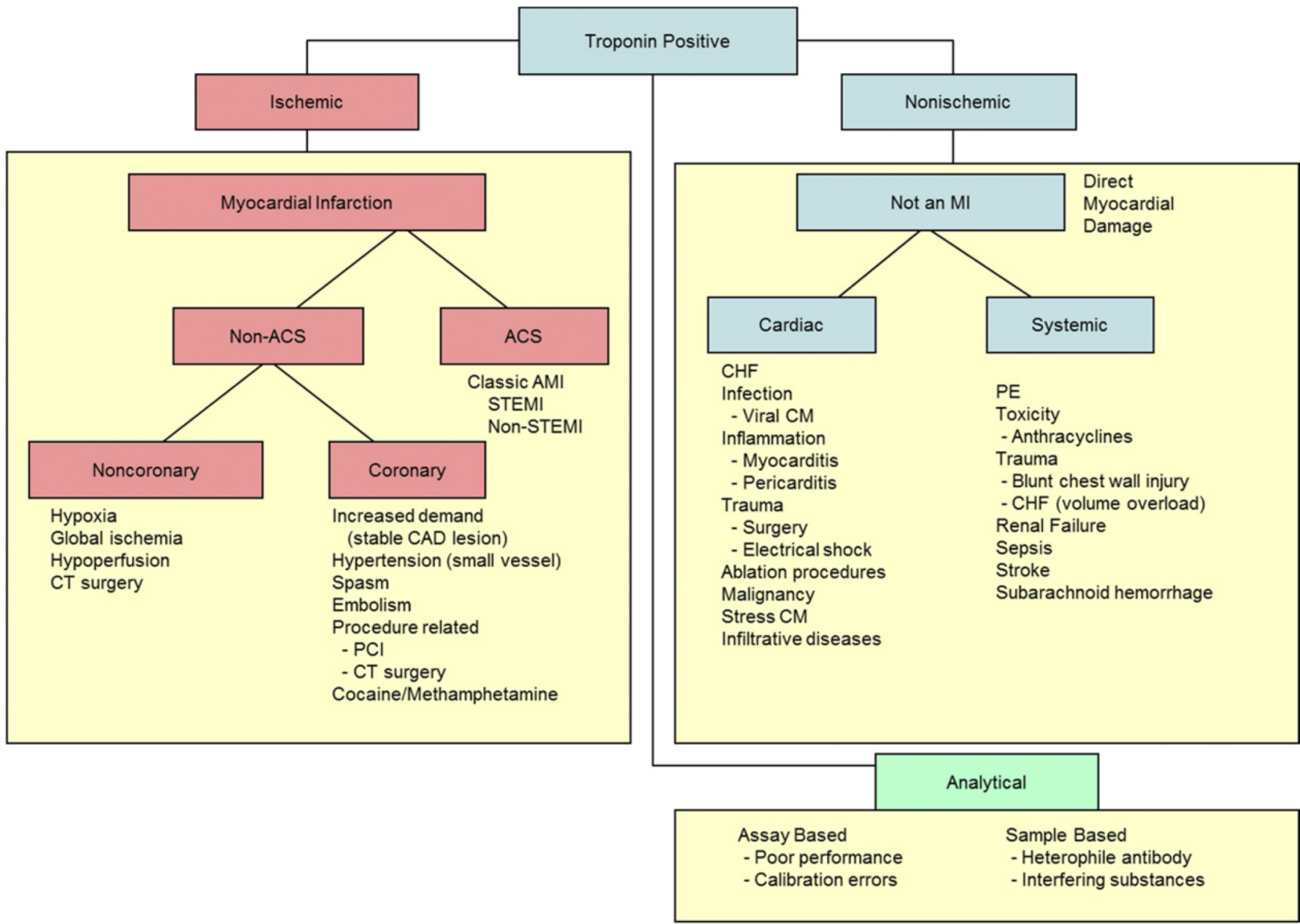
**No. at risk**

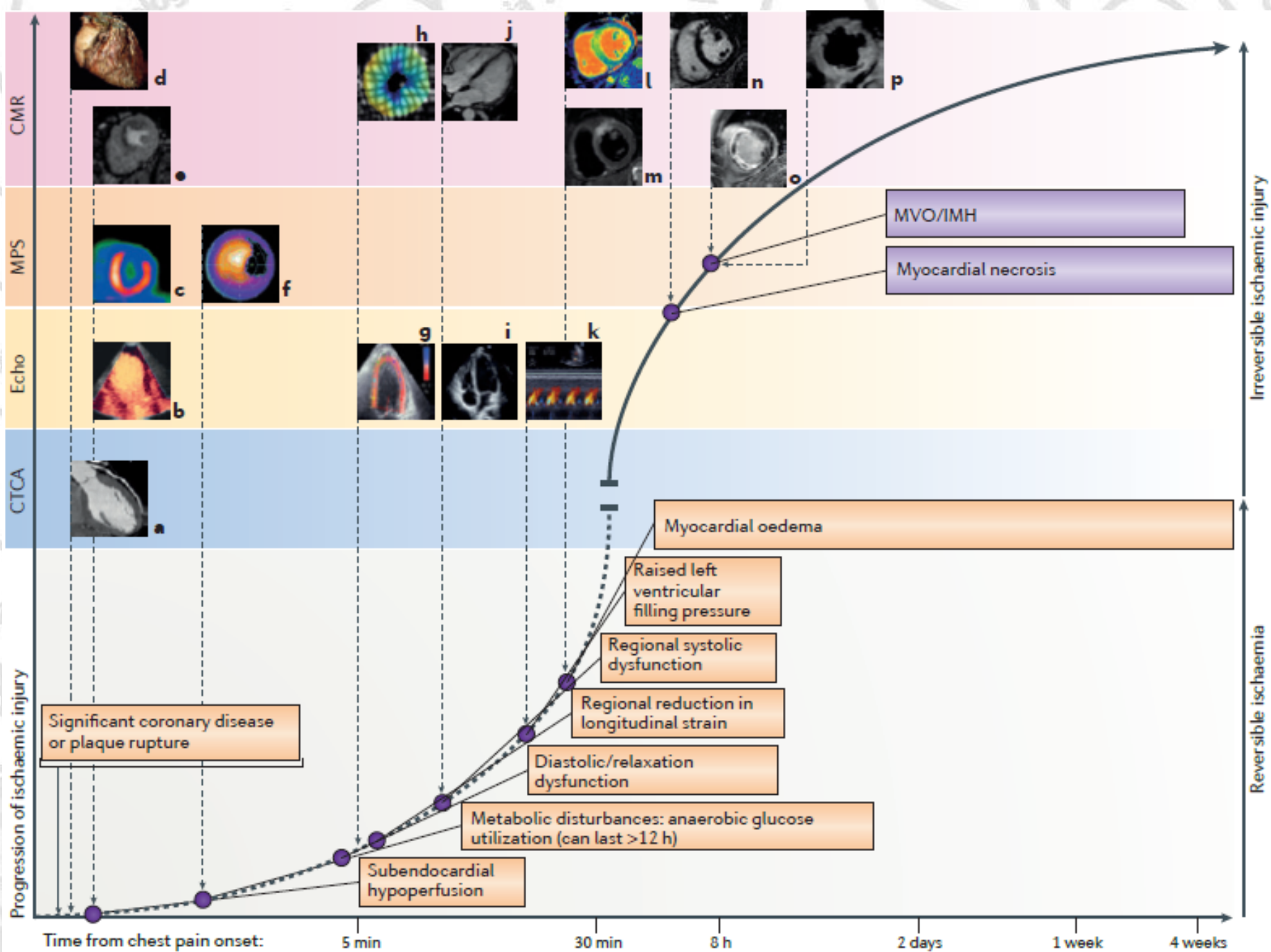
	0	180	360	540	720
LOD approach	453	405	388	314	305
Single cut-off	1516	1356	1297	1074	1042
hs-cTnl 1h-algorithm	1459	1303	1246	1035	1004
hs-cTnl 0/1h algorithm	1463	1305	1248	1036	1004



# Attempts to improve the specificity of hs-cTn assays



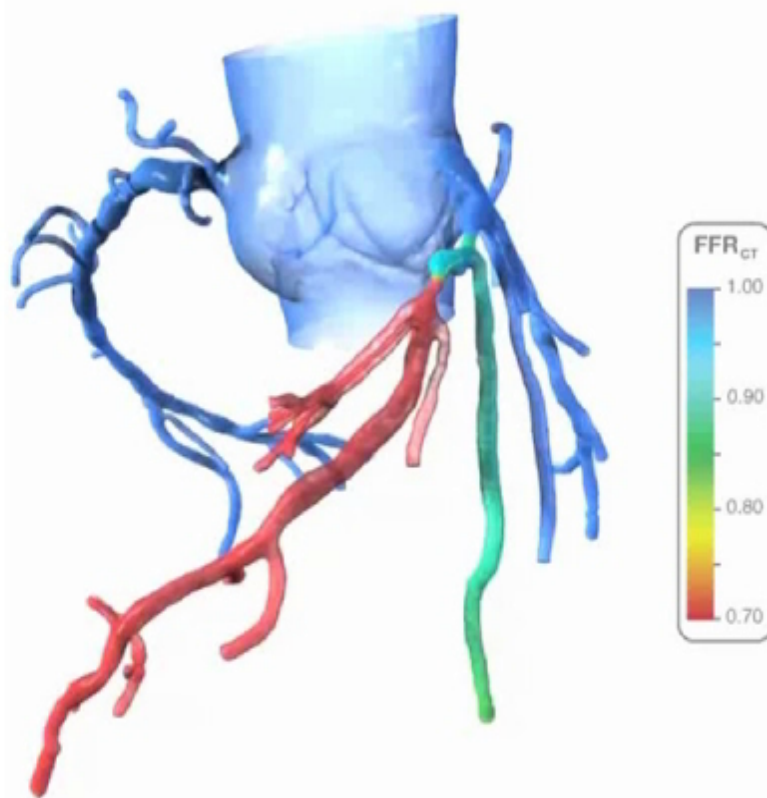




# Everything will change in the near future?

## The PLATFORM Study: Prospective Longitudinal Trial of FFR<sub>CT</sub>: Outcome and Resource Impacts)

The objective of the PLATFORM Study is to compare clinical outcomes, resource utilization, and quality of life (QOL) of FFRCT-guided evaluation versus standard practice evaluation in patients with suspected CAD in order to further inform patients, health care providers, and other stakeholders about which technologies are most effective and efficient in the diagnosis of CAD



Simulation of Heartflow's FFR<sub>CT</sub> Technology

*Simulation of coronary pressure and flow*

# The role of cardiovascular magnetic resonance imaging and computed tomography angiography in suspected non–ST-elevation myocardial infarction patients: Design and rationale of the CARdiovascular Magnetic rEsoNance imaging and computed Tomography Angiography (CARMENTA) trial

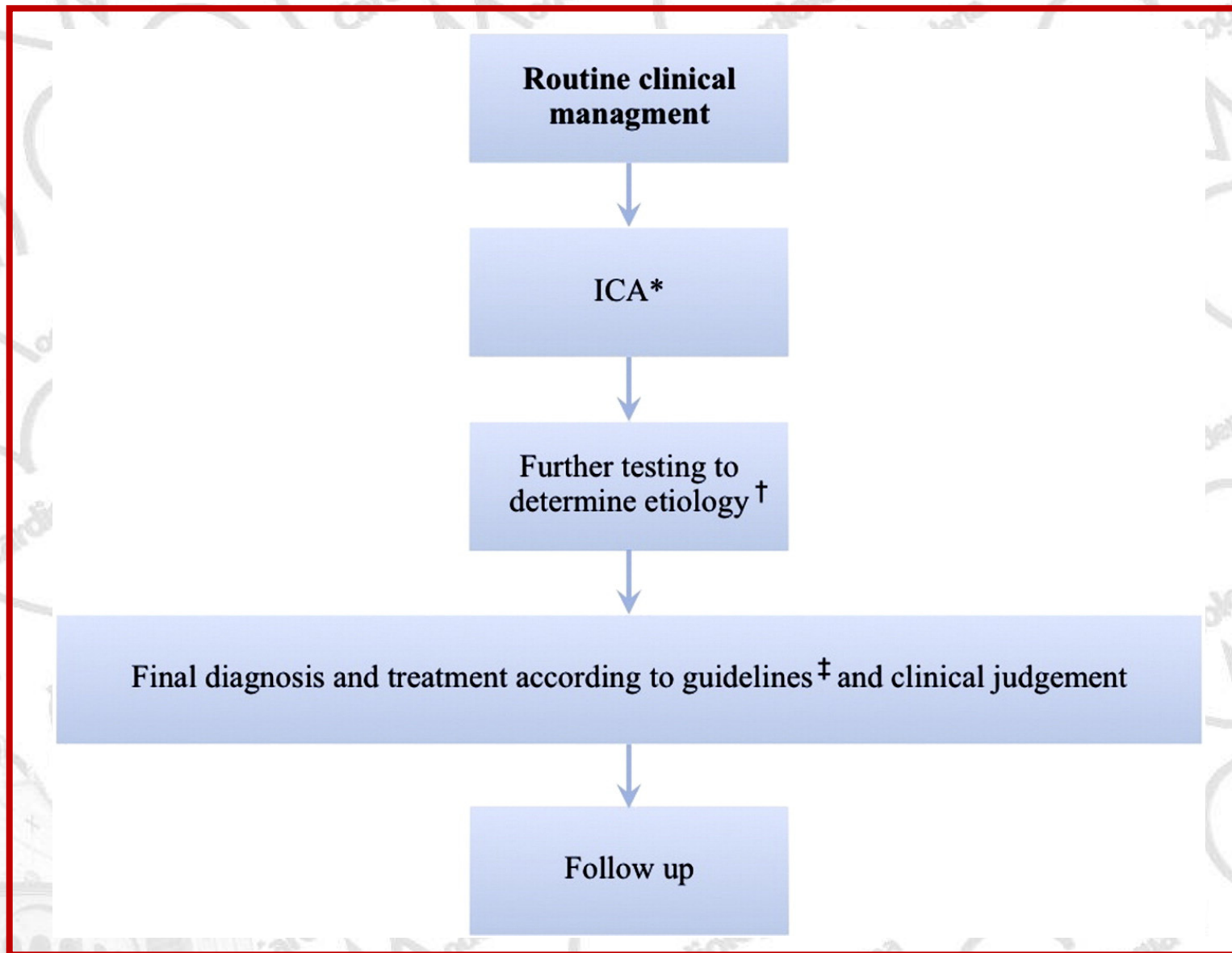
Martijn W. Smulders, MD,<sup>a,b,j</sup> Bastiaan L. J. H. Kietselaer, MD, PhD,<sup>a,b,c,j</sup> Marco Das, MD, PhD,<sup>b,c</sup> Joachim E. Wildberger, MD, PhD,<sup>b,c</sup> Harry J. G. M. Crijns, MD, PhD,<sup>a,b</sup> Leo F. Veenstra, MD,<sup>a</sup> Hans-Peter Brunner-La Rocca, MD, PhD,<sup>a,b</sup> Marja P. van Dieijen-Visser, PhD,<sup>d</sup> Alma M. A. Mingels, PhD,<sup>d</sup> Pieter C. Dagnelie, PhD,<sup>e,f</sup> Mark J. Post, MD, PhD,<sup>b,g</sup> Anton P. M. Gorgels, MD, PhD,<sup>a,b</sup> Antoinette D. I. van Asselt, PhD,<sup>h</sup> Gaston Vogel,<sup>h</sup> Simon Schalla, MD,<sup>a,b</sup> Raymond J. Kim, MD,<sup>i</sup> and Sebastiaan C. A. M. Bekkers, MD, PhD<sup>a,b</sup>  
*Maastricht, The Netherlands; and Durham, NC*

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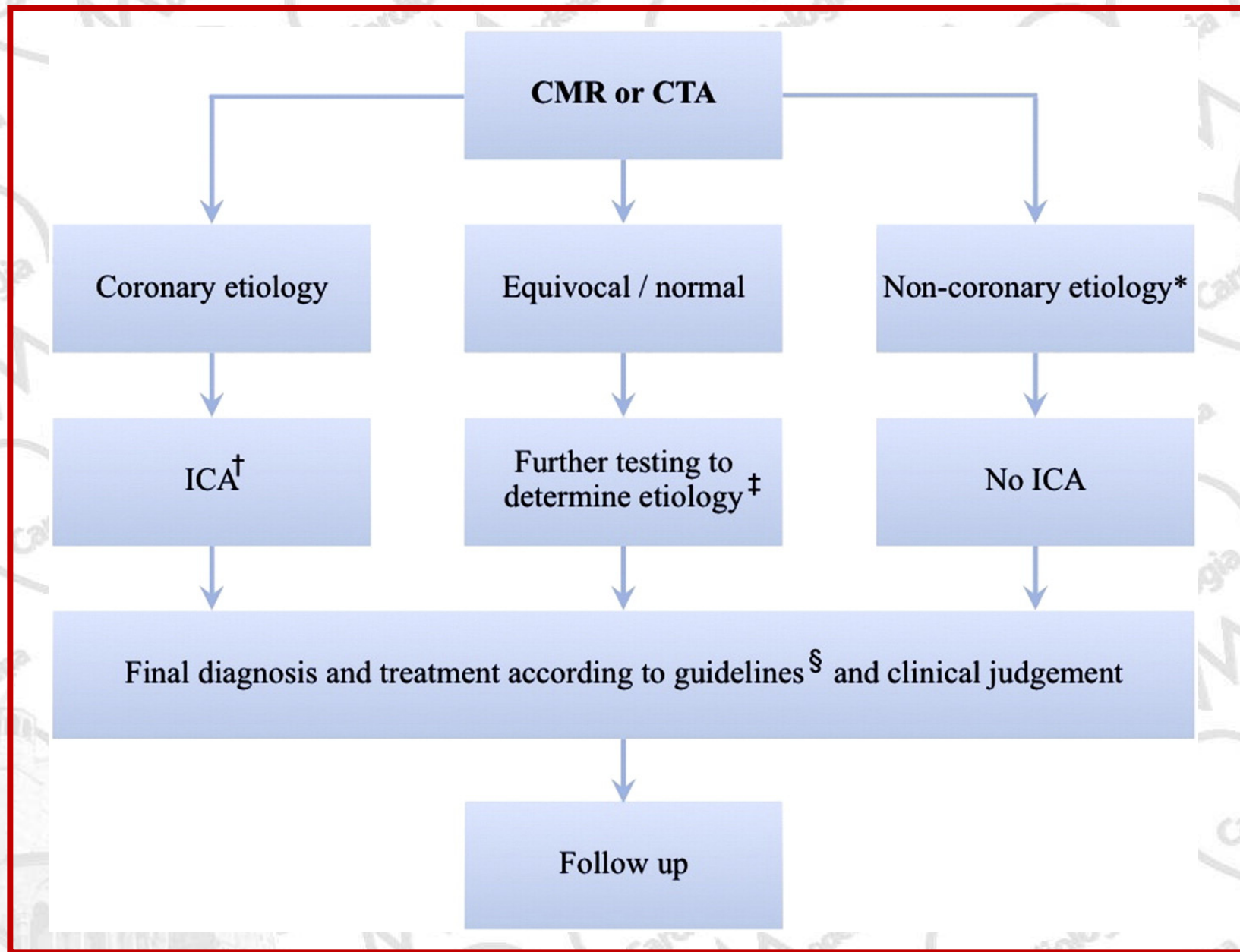
**Background** Although high-sensitivity cardiac troponin (hs-cTn) substantially improves the early detection of myocardial injury, it lacks specificity for acute myocardial infarction (MI). In suspected non–ST-elevation MI, invasive coronary angiography (ICA) remains necessary to distinguish between acute MI and noncoronary myocardial disease (eg, myocarditis), unnecessarily subjecting the latter to ICA and associated complications. This trial investigates whether implementing cardiovascular magnetic resonance (CMR) or computed tomography angiography (CTA) early in the diagnostic process may help to differentiate between coronary and noncoronary myocardial disease, thereby preventing unnecessary ICA.

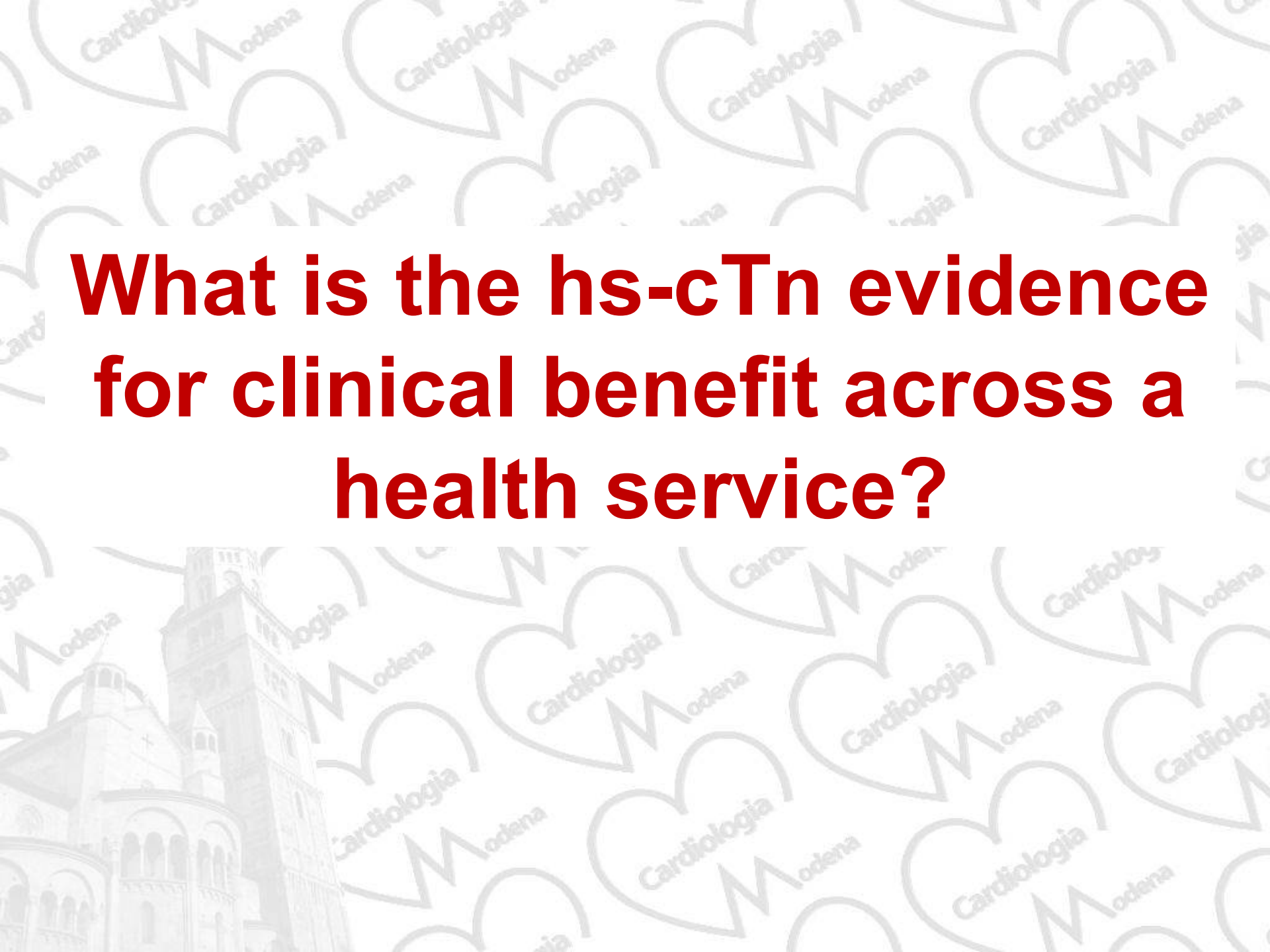
**Study Design** In this prospective, single-center, randomized controlled clinical trial, 321 consecutive patients with acute chest pain, elevated hs-cTnT, and nondiagnostic electrocardiogram are randomized to 1 of 3 strategies: (1) CMR, or (2) CTA early in the diagnostic process, or (3) routine clinical management. In the 2 investigational arms of the study, results of CMR or CTA will guide further clinical management. It is expected that noncoronary myocardial disease is detected more frequently after early noninvasive imaging as compared with routine clinical management, and unnecessary ICA will be prevented. The primary end point is the total number of patients undergoing ICA during initial admission. Secondary end points are 30-day and 1-year clinical outcome (major adverse cardiac events and major procedure-related complications), time to final diagnosis, quality of life, and cost-effectiveness.

**Conclusion** The CARMENTA trial investigates whether implementing CTA or CMR early in the diagnostic process in suspected non–ST-elevation MI based on elevated hs-cTnT can prevent unnecessary ICA as compared with routine clinical management, with no detrimental effect on clinical outcome. (Am Heart J 2013;166:968-75.)









**What is the hs-cTn evidence  
for clinical benefit across a  
health service?**



## ORIGINAL ARTICLE

## High-sensitivity versus conventional troponin for management and prognosis assessment of patients with acute chest pain

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## ABSTRACT

**Objectives** High-sensitivity troponin (hs-cTn) is substituting conventional cTn for evaluation of chest pain. Our aim was to assess the impact on patient management and outcome.

**Methods** A total of 1372 consecutive patients presenting at the emergency department with non-ST-elevation acute chest pain were divided into two periods according to the cTn assay used, conventional (n=699, March 2008 to July 2010) or hs-cTn (n=673, November 2010 to March 2013). Management policies were similar and according to guidelines. The primary endpoint was major adverse cardiac events (MACE) at 6 months (death, myocardial infarction, readmission by unstable angina or postdischarge revascularisation).

**Results** There were minor differences in baseline characteristics. In the hs-cTn period, more patients elevated cTn (73% vs 37%, p=0.0001) leading to more coronary angiograms (77% vs 55%, p=0.0001) and revascularisations (45% vs 31%, p=0.0001); conversely, fewer patients were initially assigned to exercise testing (14% vs 36%, p=0.0001) and, therefore, discharged early after a negative result (7% vs 22%, p=0.0001). At 6 months, 135 patients suffered MACE, including 54 deaths. After adjusting for a Propensity Score, hs-cTn use was not significantly associated with MACE (HR=0.99; 95% CI 0.70 to 1.41; p=0.98) or mortality (HR=1.02; 95% CI 0.59 to 1.77; p=0.95), though the risk of longer hospitalisation stay increased at the index episode (OR=1.35, 95% CI 1.07 to 1.71, p=0.02).

**Conclusions** hs-cTn simplified chest pain triage on avoiding a more complex evaluation with non-invasive tests in the chest pain unit, but prompted longer hospitalisations and more invasive procedures without impacting on the 6-month outcomes.

within the vast population of acute chest pain who would otherwise go undetected using conventional cTn.<sup>5-7</sup> In addition, cTn release can be detected as early as 2 h from AMI onset.<sup>8-10</sup> However, ruling in AMI when hs-cTn is mildly elevated or ruling out unstable angina when hs-cTn is normal remains matters of debate.

Currently, hs-cTn is substituting conventional cTn in many hospitals, but there is scarce information regarding how this change is modifying patient management and outcome. Some data suggest that hs-cTn use might improve patient prognosis, but more information is needed to confirm these findings.<sup>11</sup> In this study, two consecutive series of patients with acute chest pain managed with conventional or hs-cTn assay were compared. The main purpose was to investigate the impact of hs-cTn on postdischarge outcome as well as on the in-hospital diagnostic work-up and management.

## METHODS

## Study design

This prospective cohort study included 1372 patients who presented at the emergency department with acute chest pain. The study group comprised two cohorts corresponding to different periods according to the cTn assay used: (1) the conventional cTn period (from 1 March 2008 to 1 July 2010, n=699) and (2) the hs-cTn period (from 1 November 2010 to 1 March 2013, n=673). In the time interval between the two periods, a different provisional cTn assay was used until the new high-sensitivity assay was implemented, and these interim patients were not considered. The study was reviewed and approved by the ethics

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# THM-1

- Current hs-cTn assays now *rule out AMI with high confidence* and challenge the need for additional testing (this does not apply for clinical high-risk features)

# THM-2

- The reduced specificity of the hs-cTn assays for AMI confronts ED physicians with a new problem and *warrant a more sophisticated approach*

# THM-3

- However, studies that optimally define “markedly elevated and/or substantially rising hs-cTn levels” and the most appropriate imaging modality strategy are needed

