UNIVERSAL DEFINITION MI HISTORIQUE

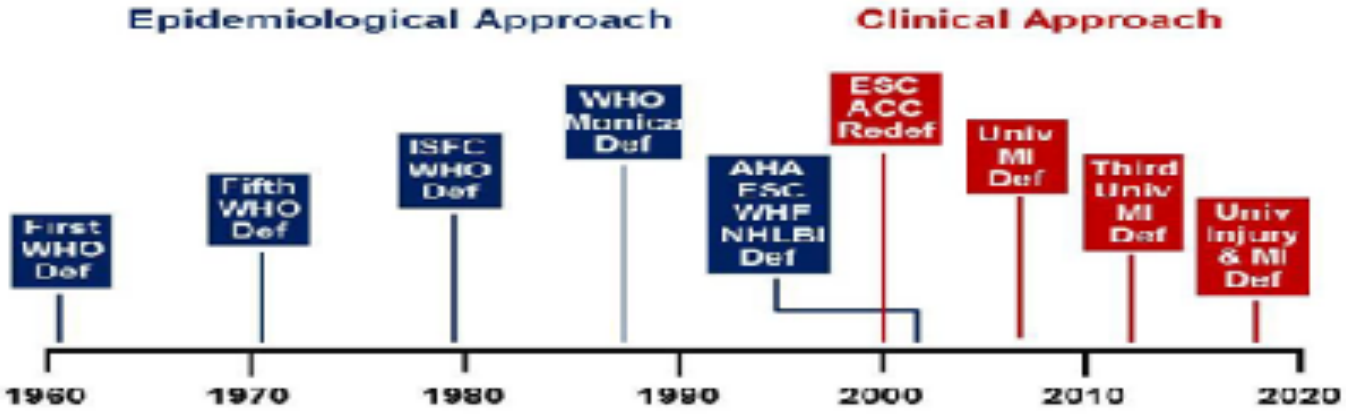


Figure1. Changing criteria for the definition of myocardial infarction (MI). ACC = American College of Cardiology; AHA = American Heart Association; ESC = European Society of Cardiology; ISFC = International Society and Federation of Cardiology; NHLBI = National Heart, Lung, and Blood Institute; WHF = World Heart Foundation; WHO = World Health Organization.

ACUTE MYOCARDIAL INFARCTION(TYPE1MI)

Acute myocardial infarction (type1MI)
Acute Myocardial injury
Acute myocardial ischaemia.

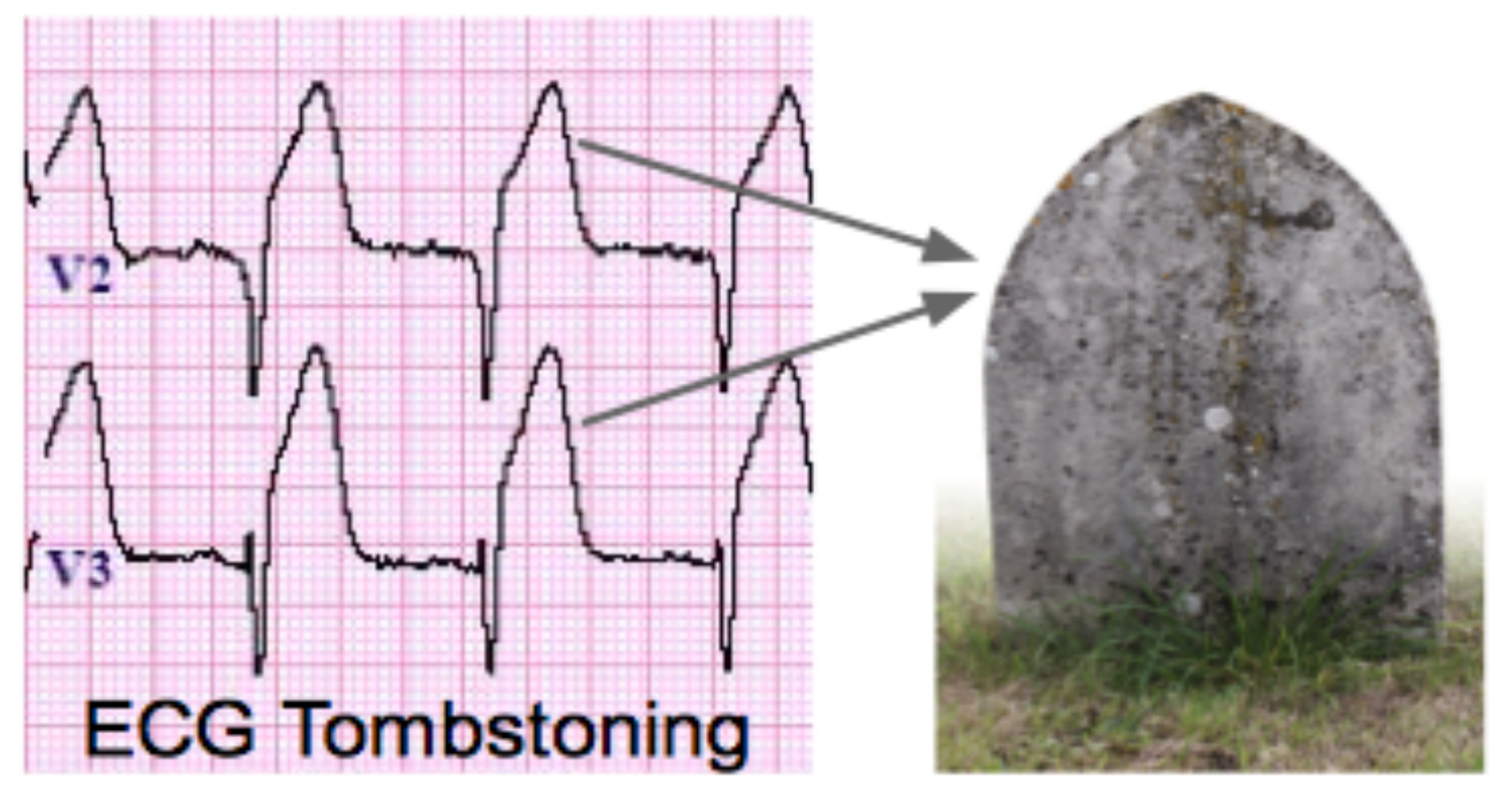
-Injury: Detection of a **rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)]** with at least **one value above the 99$_{th}$ percentile upper reference limit (URL)** and with at least **one of the following:**

-Ischemia.
-Clinic
-ECG Changes ST-T LBBB Q-waves
-Imaging evidence
-**Identification of an intracoronary thrombus by angiography or autopsy.**

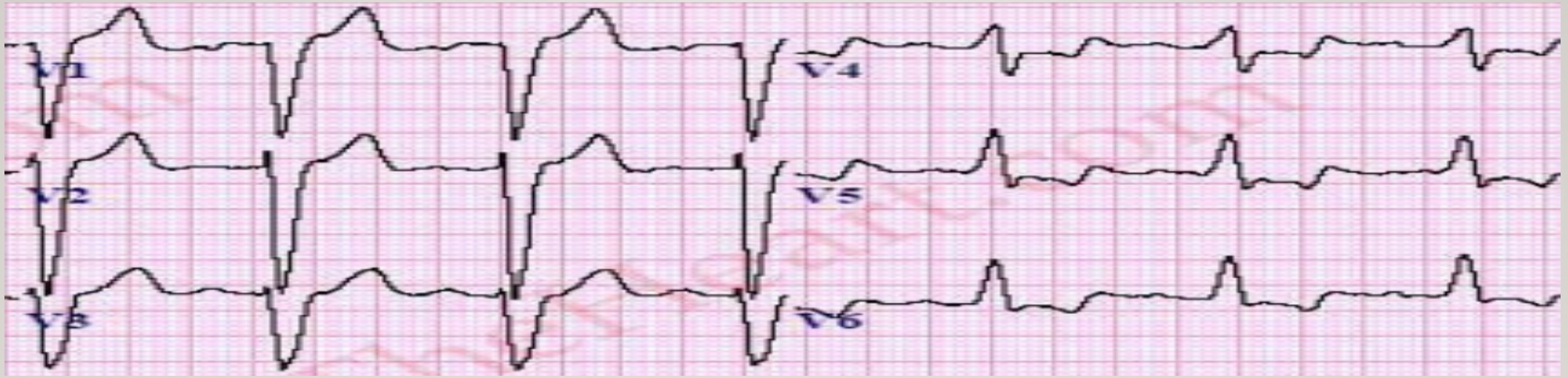
TYPE 1 MI

- **STEMI**
- ST-segment elevation Myocardial Infarction
- **N-STEMI**
- non ST-segment elevation Myocardial Infarction
- The case of **Unstable Angina**

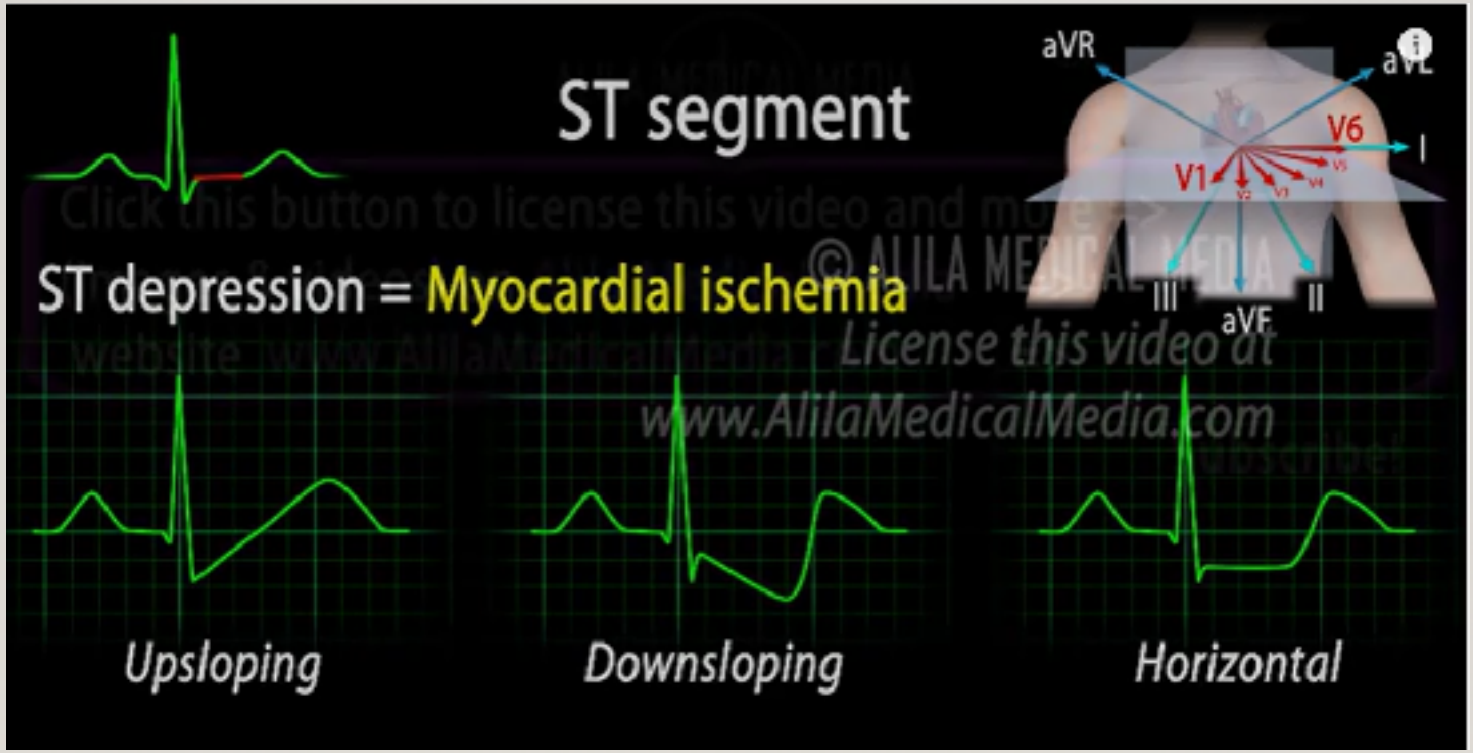
STEMI



STEMI-LBBB



N-STEMI ECG

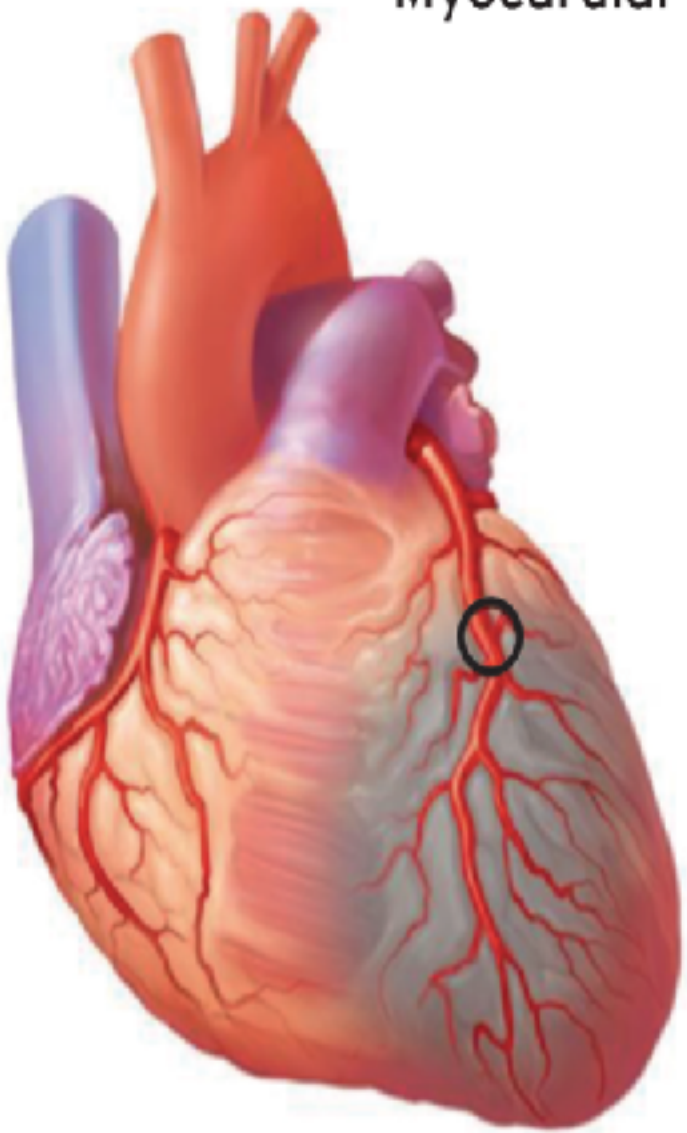

ST segment
ST depression = Myocardial ischemia
aVR
aVL
V6
I
V1
III
aVF
II
Upsloping
Downsloping
Horizontal

THE 99TH PERCENTILE UPPER REFERENCE LIMIT

The 99th percentile URL is designated as the decision level for the presence of myocardial injury and **must be determined for each specific assay** with quality control materials.

Clinicians should be aware that for all cTn assays, including hs-cTn assays, there is still **no expert opinion or consensus about specific criteria for how the 99th percentile URL should be defined…**.

Myocardial Infarction Type 1



Plaque rupture/erosion with occlusive thrombus

Plaque rupture/erosion with non-occlusive thrombus

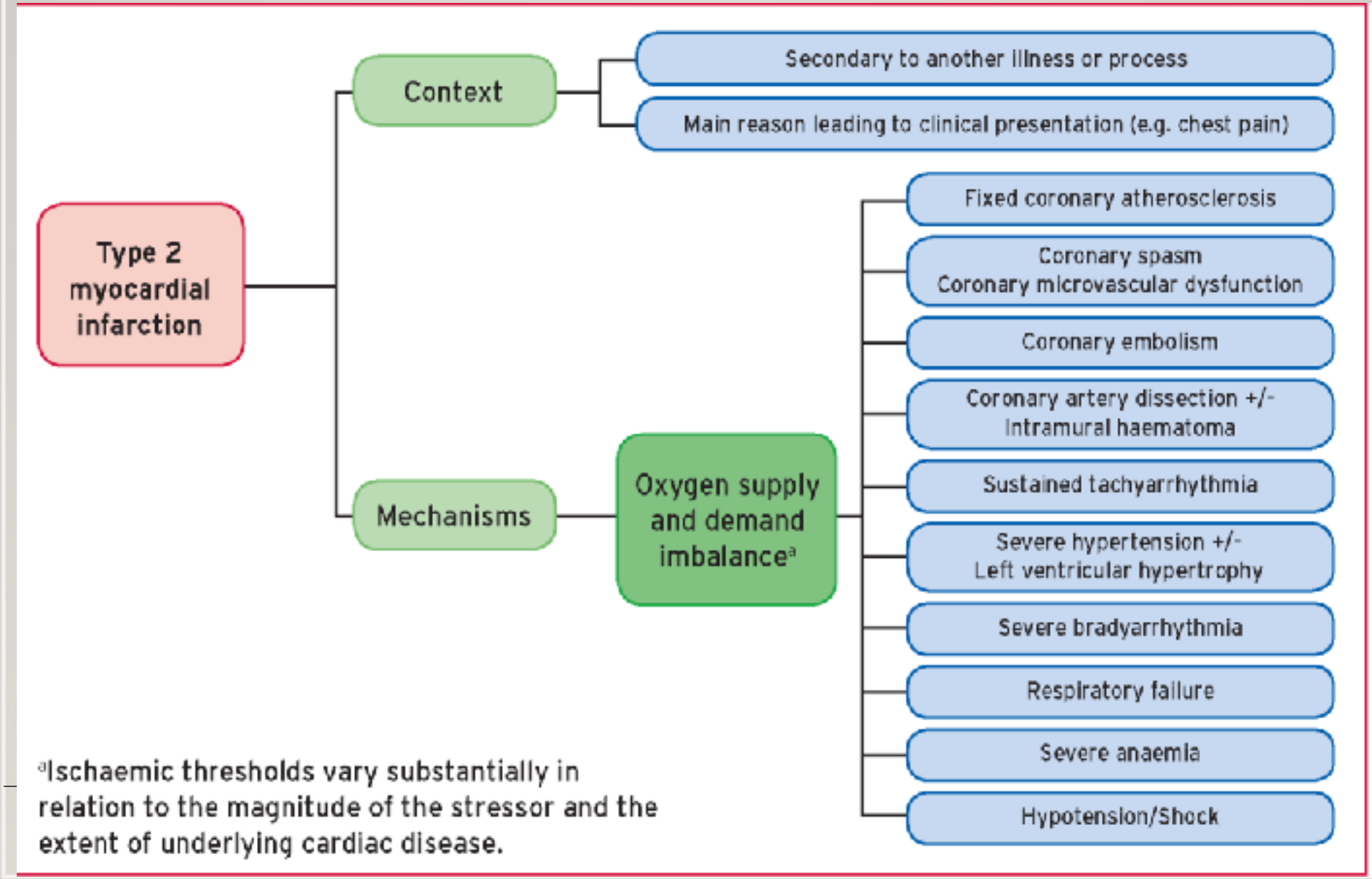
N-STEMI/UA INVASIVE STRATEGY

- Increased cardiac biomarkers (troponin, CK-MB)
- New ST segment depression
- Signs or symptoms of congestive heart failure (rales on examination, hypoxia with pulmonary edema on chest X-ray)
- Hemodynamic instability
- Sustained ventricular tachycardia or ventricular fibrillation
- Recent coronary intervention within 6 months
- Prior coronary artery bypass grafting
- High TIMI risk score
- Reduced left ventricular systolic function (EF < 40%)
- Recurrent angina at rest or with low level activity
- High-risk findings from non-invasive testing

MYOCARDIAL INJURY RELATED TO ACUTE MYOCARDIAL ISCHAEMIA BECAUSE OF OXYGEN SUPPLY/DEMAND IM BALANCE TYPE 2 MI

Reduced myocardial perfusion,

Increased myocardial oxygen demand,


Type 2 myocardial infarction
Context
Secondary to another illness or process
Main reason leading to clinical presentation (e.g. chest pain)
Mechanisms
Oxygen supply and demand imbalance[a]
Fixed coronary atherosclerosis
Coronary spasm
Coronary microvascular dysfunction
Coronary embolism
Coronary artery dissection +/-
Intramural haematoma
Sustained tachyarrhythmia
Severe hypertension +/-
Left ventricular hypertrophy
Severe bradyarrhythmia
Respiratory failure
Severe anaemia
Hypotension/Shock
[a]Ischaemic thresholds vary substantially in relation to the magnitude of the stressor and the extent of underlying cardiac disease.
©ESC/ACC/AHA/WHF 2018

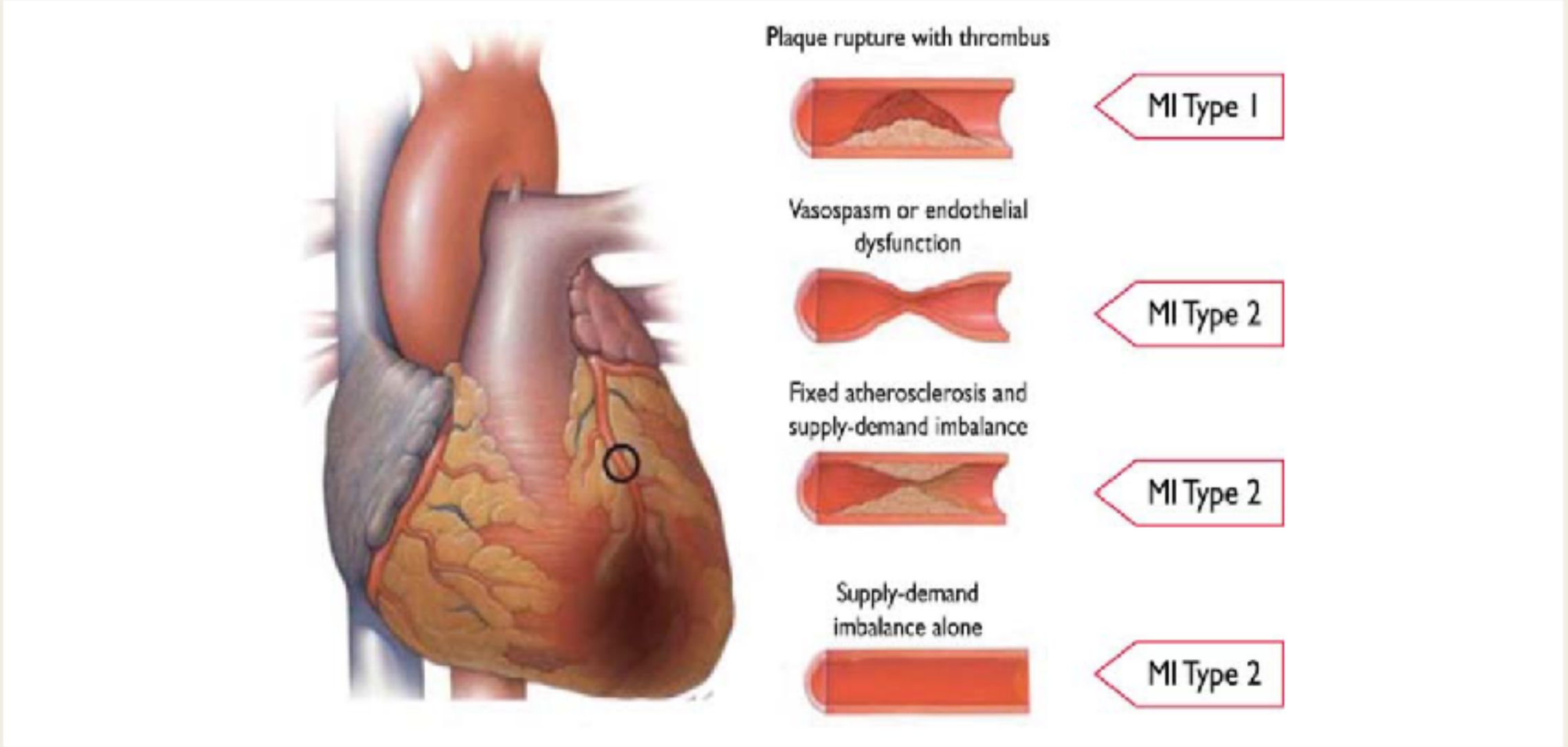


Figure 2 Differentiation between myocardial infarction (MI) types 1 and 2 according to the condition of the coronary arteries.

Type 1: Spontaneous myocardial infarction
Spontaneous myocardial infarction related to atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection with resulting intraluminal thrombus in one or more of the coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis. The patient may have underlying severe CAD but on occasion non-obstructive or no CAD.
Type 2: Myocardial infarction secondary to an ischaemic imbalance
In instances of myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or demand, e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/brady-arrhythmias, anaemia, respiratory failure, hypotension, and hypertension with or without LVH.
Type 3: Myocardial infarction resulting in death when biomarker values are unavailable
Cardiac death with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or new LBBB, but death occurring before blood samples could be obtained, before cardiac biomarker could rise, or in rare cases cardiac biomarkers were not collected.
Type 4a: Myocardial infarction related to percutaneous coronary intervention (PCI)
Myocardial infarction associated with PCI is arbitrarily defined by elevation of cTn values >5 x 99th percentile URL in patients with normal baseline values (≤99th percentile URL) or a rise of cTn values >20% if the baseline values are elevated and are stable or falling. In addition, either (i) symptoms suggestive of myocardial ischaemia, or (ii) new ischaemic ECG changes or new LBBB, or (iii) angiographic loss of patency of a major coronary artery or a side branch or persistent slow- or no-flow or embolization, or (iv) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required.
Type 4b: Myocardial infarction related to stent thrombosis
Myocardial infarction associated with stent thrombosis is detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac biomarkers values with at least one value above the 99th percentile URL.
Type 5: Myocardial infarction related to coronary artery bypass grafting (CABG)
Myocardial infarction associated with CABG is arbitrarily defined by elevation of cardiac biomarker values >10 x 99th percentile URL in patients with normal baseline cTn values (≤99th percentile URL). In addition, either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (III) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

MYOCARDIAL INFARCTION WITH NON-OBSTRUCTIVE CORONARY ARTERIES (MINOCA)

It is increasingly recognized that a group of MI patients has no angiographic obstructive (≥ 50% diameter stenosis) CAD and the term **Myocardial infarction with non-obstructive coronary arteries (MINOCA) has been coined for this entity**.

The prevalence of MINOCA is estimated to be 6−8% among patients diagnosed with MI.

Atherosclerotic plaque disruption and coronary thrombosis may be a cause of MINOCA, i.e. type 1 MI. However, **coronary spasm and spontaneous coronary dissection** may be involved as well, i.e. type 2 MI.

MYOCARDIAL INJURY

Small amounts of myocardial injury with necrosis may be detected without Ischemia

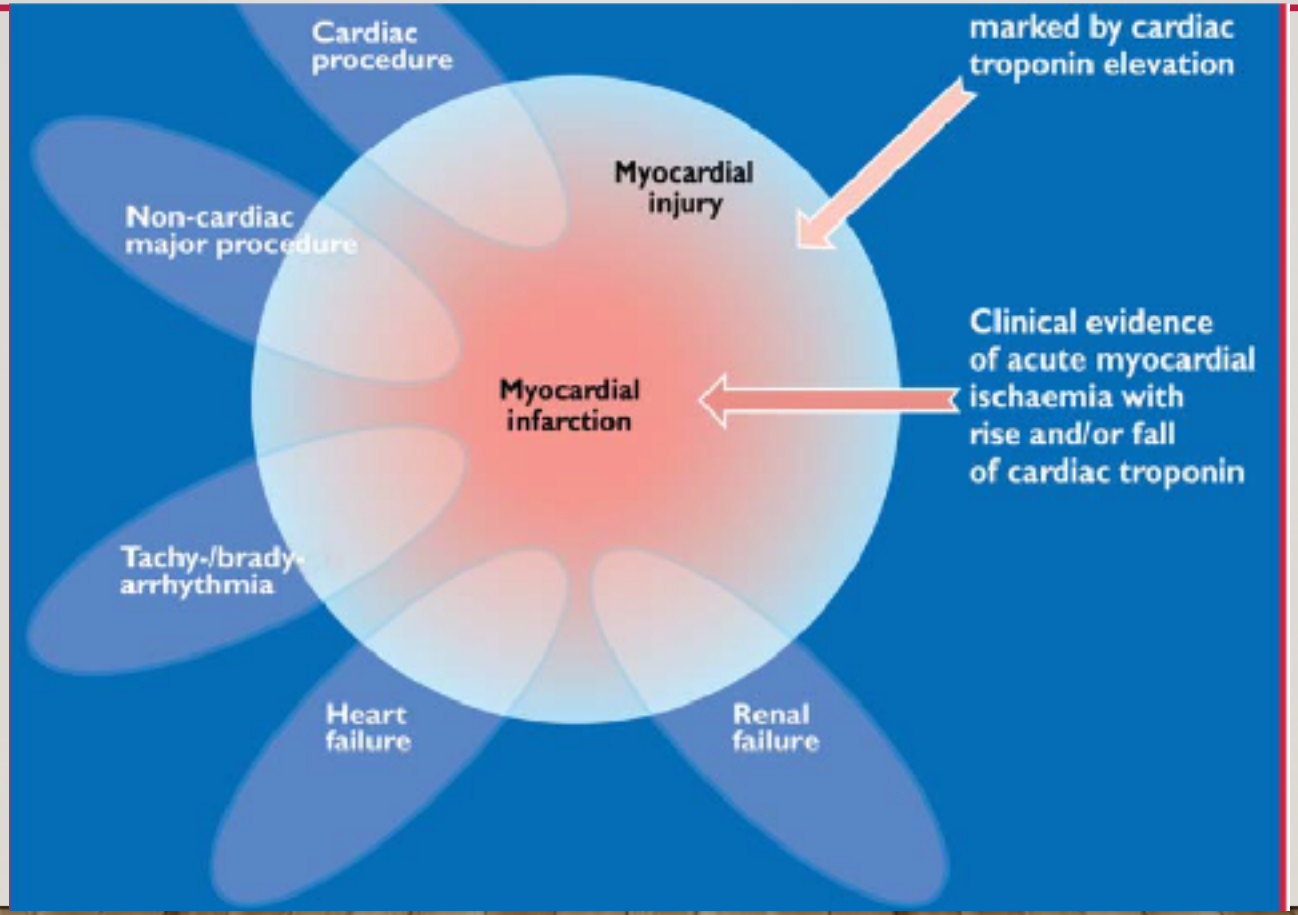
Ischémie — Wikipédia

Une **ischémie** (prononcer /is.ke.mi/ ; du grec ancien ἴσχω, ískhô (« tenir ») et αἷμα, haîma (« sang ») est la diminution de l'apport sanguin artériel à un organe

CRITERIA FOR MYOCARDIAL INJURY

Detection of an elevated cTn value above the 99th percentile URL is defined as myocardial injury.The injury is called **acute if there is a rise and/or fall of cTn values**

MYOCARDIAL INJURY



Myocardial injury that may occur unrelated to acute myocardial ischaemia

Cardiac conditions

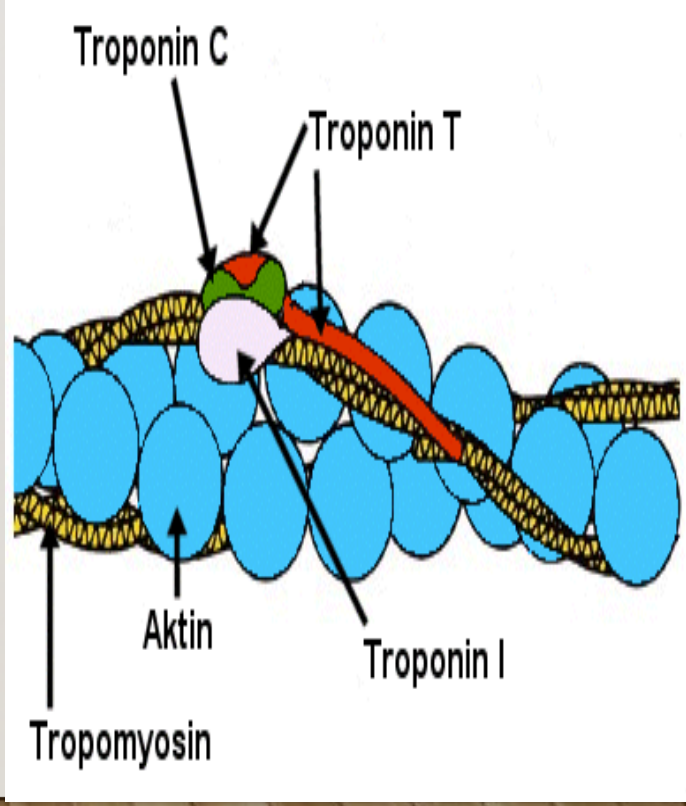
Heart failure
Myocarditis
Cardiomyopathy
Takotsubo syndrome
Coronary revascularization procedure
Non-coronary cardiac surgery
Transcatheter valve replacement
Ablation, defibrillator shocks
Cardiac contusion

Systemic conditions

Sepsis, infectious disease
Chronic kidney disease
Stroke, subarachnoid haemorrhage
Pulmonary embolism
Chemotherapeutic agents
Strenuous exercise

TROPONINE

- Troponin is a myofibrillar regulatory protein complex of exclusively muscular origin
- TnT fixes troponin-complex to tropomyosin
- TnI has Ca-binding site
- Cardiac specific isoforms of TnT and TnI



BIOMARKER DETECTION OF MYOCARDIAL INJURY

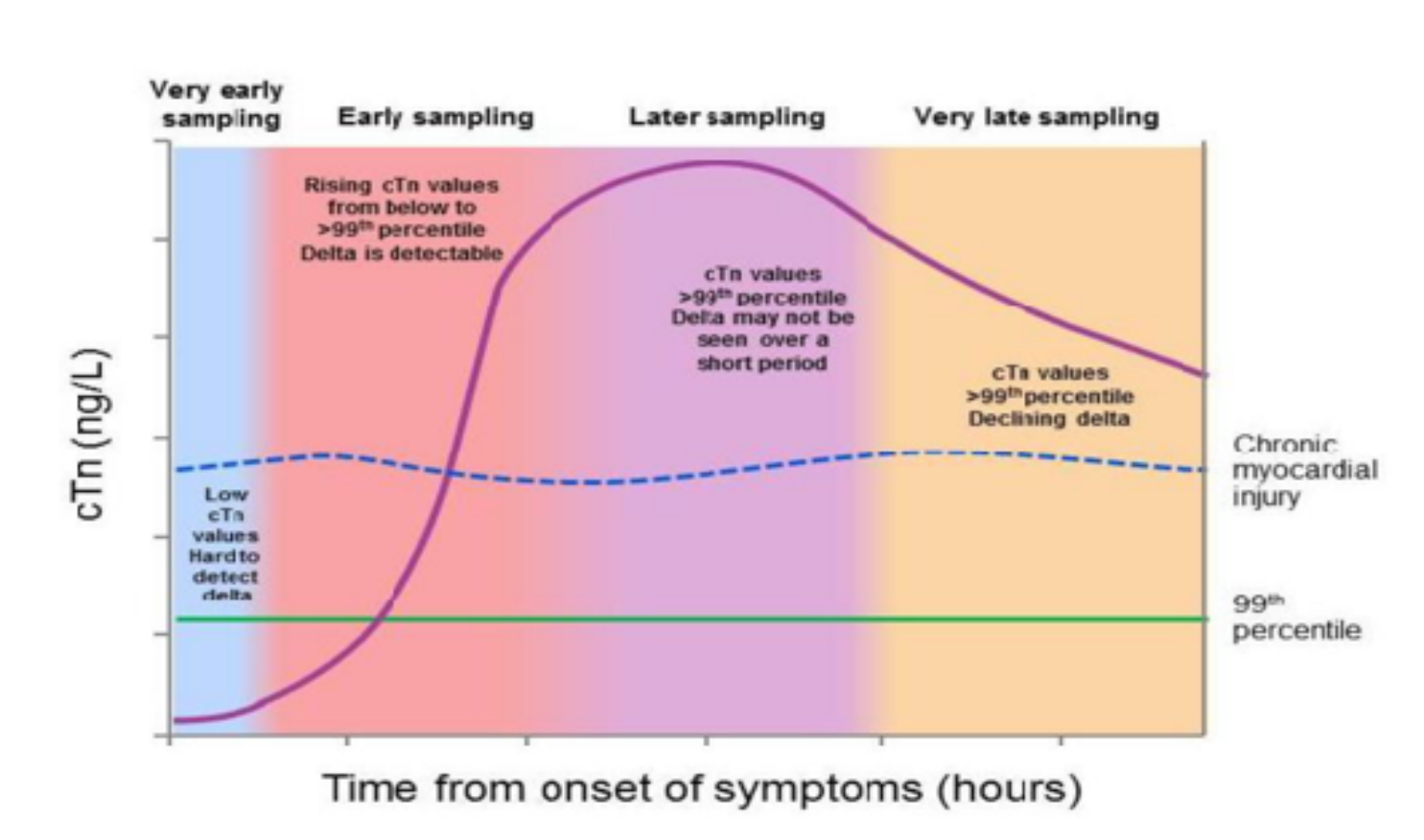
Cardiac troponin I (cTnI) and T (cTnT) are components of the contractile apparatus of myocardial cells and are expressed almost exclusively in the heart.

Increases in cTnI values have not been reported to occur following injury to non-cardiac tissues.

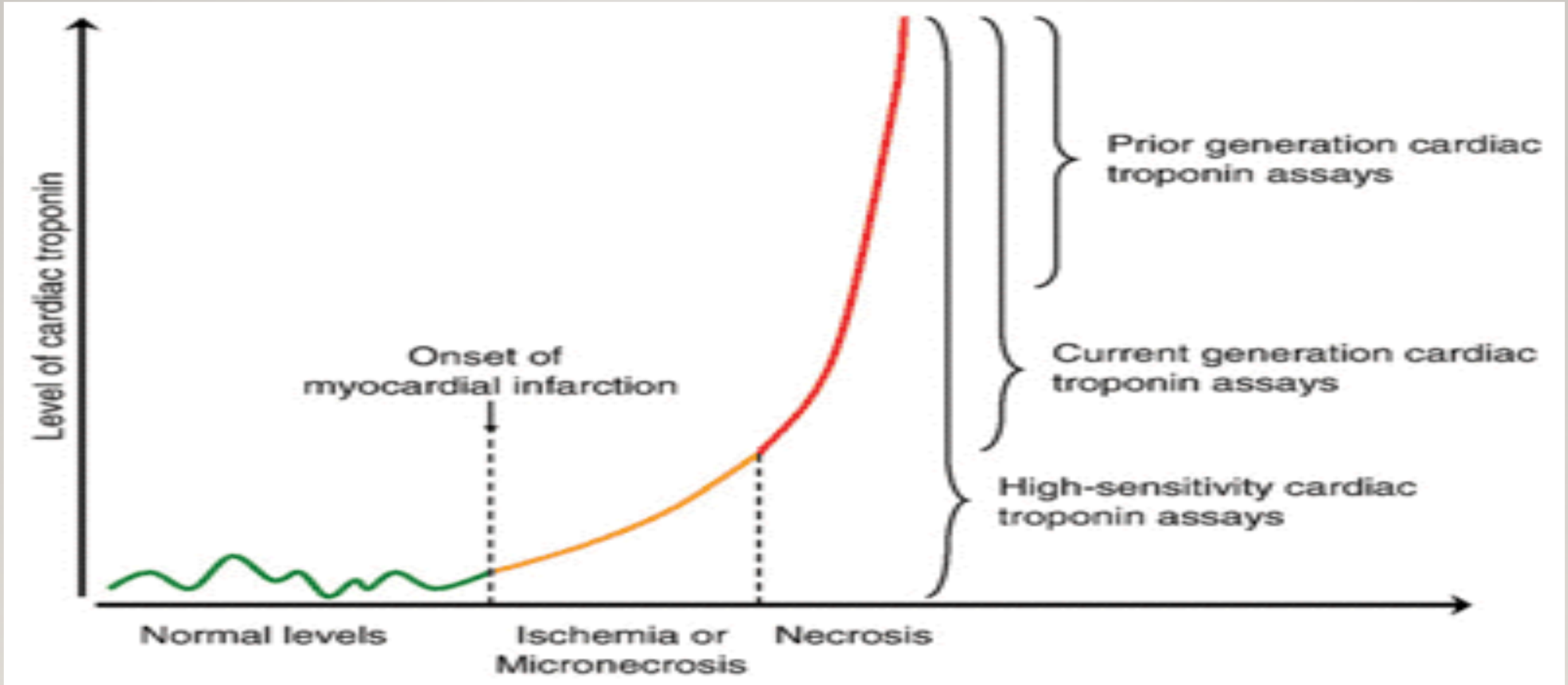
The situation is **more complex for cTnT**. Biochemical data indicate that injured skeletalmuscle can manifest the ability to express proteins that are detected by the cTnT assay

cTnI and cTnT are the preferred biomarkers for the evaluation of myocardial injury,and when available, **hs-cTn assays are recommended for routine clinical use.**

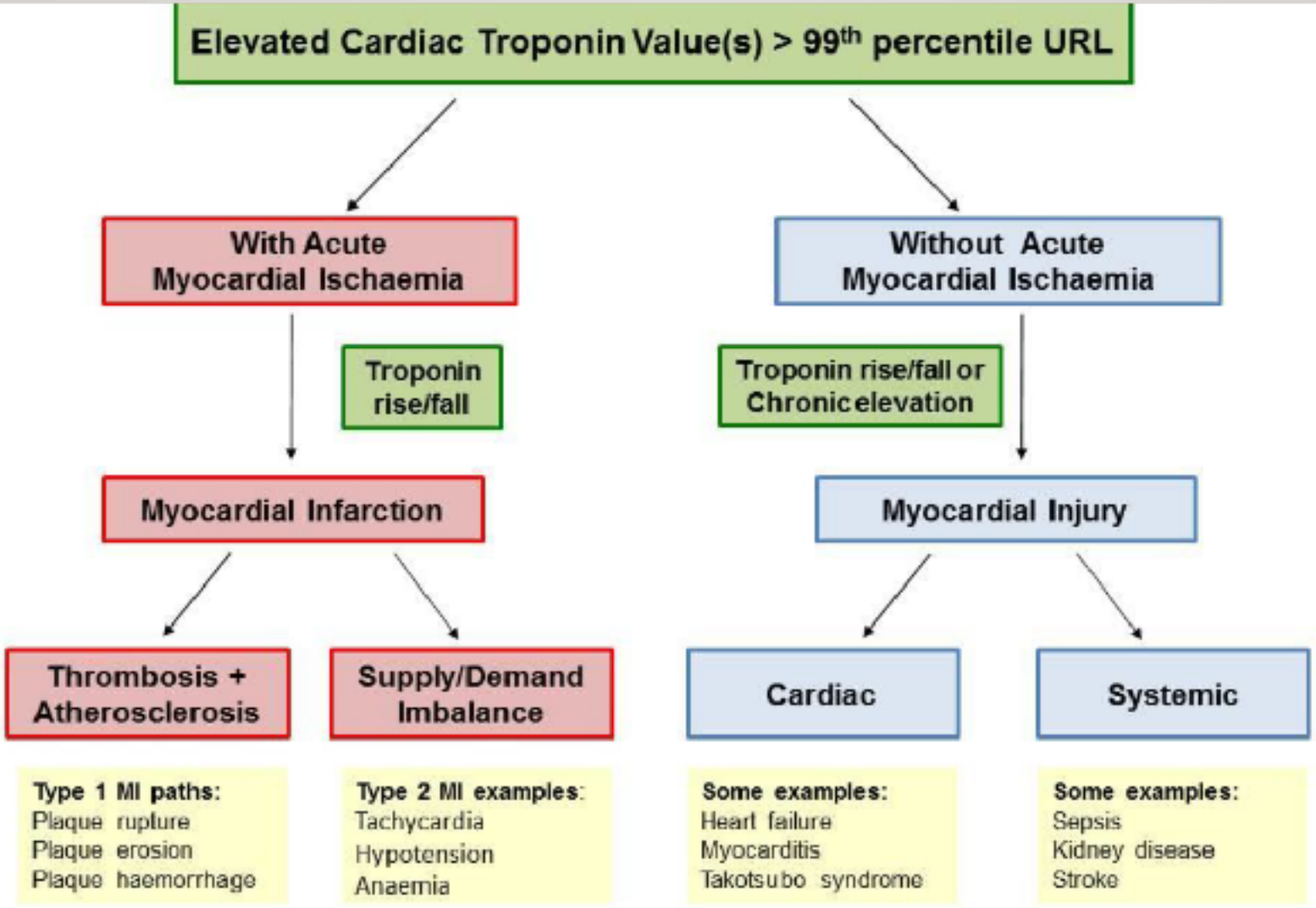
TROPONIN: INFARCTION AND CHRONIC INJURY



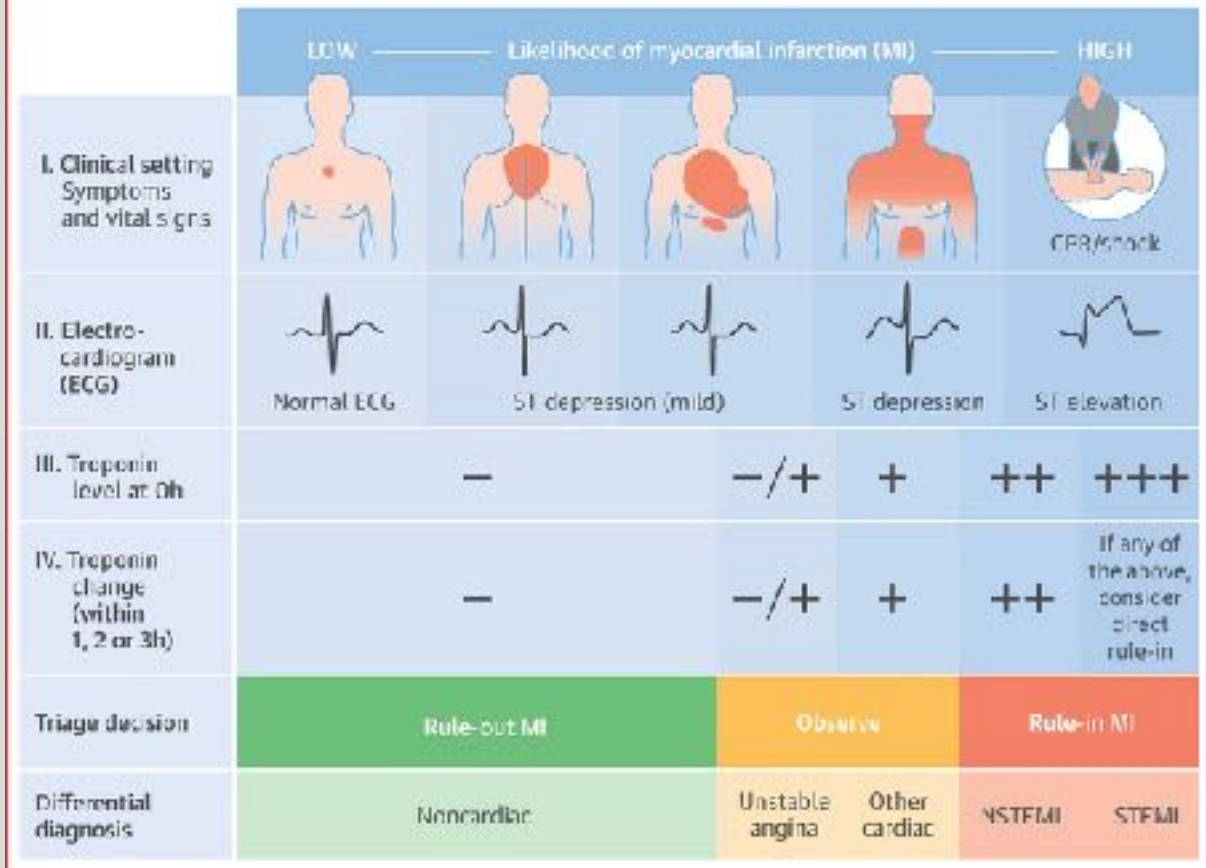
HS-TROPONIN


Level of cardiac troponin
Onset of myocardial infarction
Prior generation cardiac troponin assays
Current generation cardiac troponin assays
High-sensitivity cardiac troponin assays
Normal levels
Ischemia or Micronecrosis
Necrosis

TROPONINE EN CLINIQUE

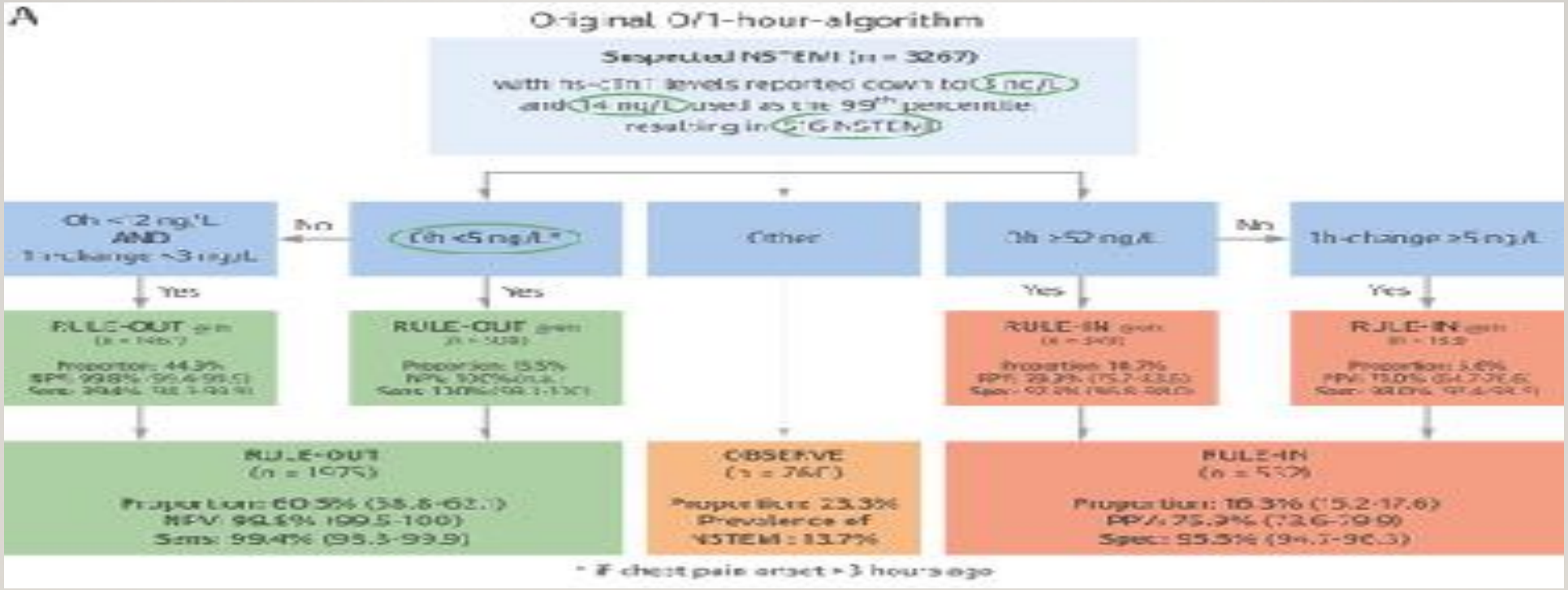


CENTRAL ILLUSTRATION: Patient Assessment With Suspected ACS



Twerenbold, R. et al. J Am Coll Cardiol. 2017;70(8):996-1012.

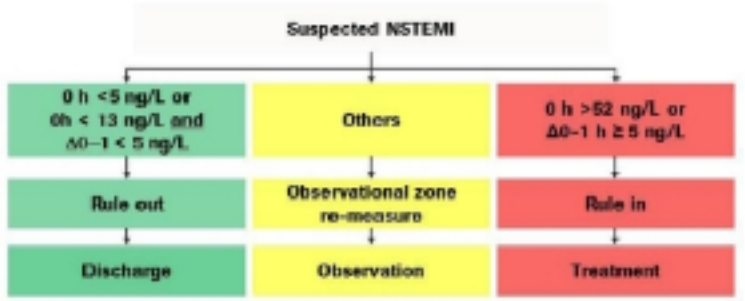
RULE-IN/RULE-OUT



RULE-IN RULE OUT À UNE HEURE ROCHE ELECSYS

Algorithme à 1h[4]

Conformément aux directives européennes, seuls les tests de troponine cardiaque ultra-sensibles (voir définition ci-dessus) conviennent à l'application de l'algorithme à 1h. Ce protocole permet une exclusion sûre ainsi qu'une confirmation après seulement 1 heure. Ce nouvel algorithme garantit sécurité et gain de temps pour tous. L'algorithme se caractérise par une excellente validation et peut être appliqué en toute sécurité.[4-11]



Vue détaillée

Les valeurs sont spécifiques à l'essai et données ici pour hs-TropT de Roche.
L'algorithme 0/1h s'applique pour la douleur mammaire > 3h.

Test hs TropT pour la stratification du risque[4]

Outre le diagnostic d'infarctus aigu du myocarde, la hs TropT convient aussi à titre de biomarqueur pour l'évaluation du risque face à des événements cardiaques à court et long terme. La pertinence d'un pronostic est encore meilleure si l'on associe les paramètres hs TropT et NT proBNP.

IT IS IMPORTANT TO HIGHLIGHT 5 ASPECTS WHEN APPLYING TROPONIN-BASED STRATEGIES IN CLINICAL PRACTICE

First, they *should be used only in conjunction with* ***full clinical assessment***, including a pre-test probability assessment

Second**,** these strategies should be considered **triage strategies rather than definite diagnostic strategies**

Third**, the percentage of patients eligible for rule-out or rule-in varies widely from ≈9.8% to 77%** depending on the underlying algorithm, the cTn assay used, and the clinical setting, including the prevalence of MI .

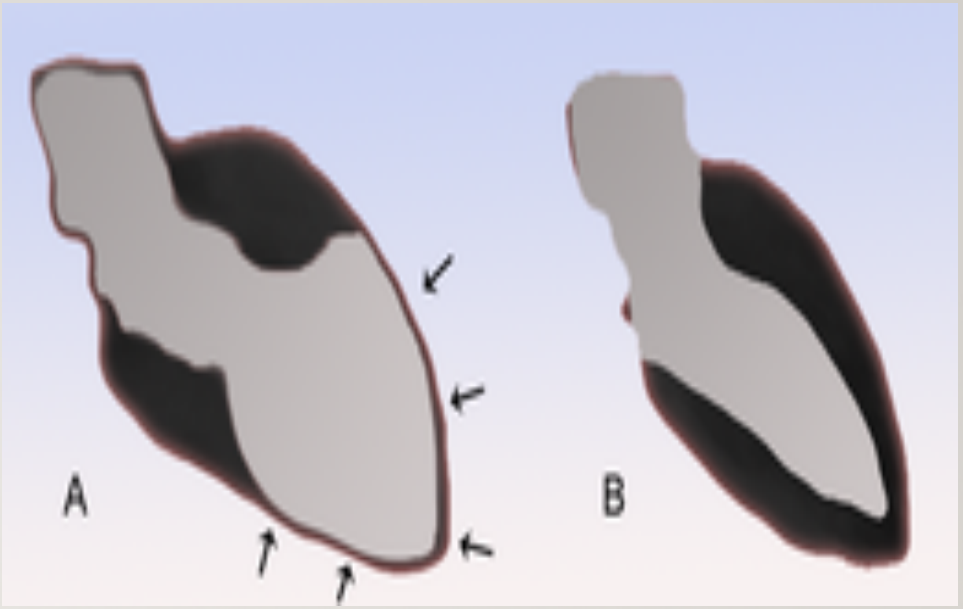
Fourth, **these strategies should only be applied after the initial ECG has excluded ST-segment elevation myocardial infarction (STEMI)**

Fifth, all triage strategies should be embedded in the local standard operating procedures of the ED

Takotsubo and Stroke Heart:

Two examples of Myocardial Injury

Le **syndrome de *tako-tsubo*** (蛸壺), littéralement « piège à poulpe »), appelé également **syndrome des cœurs brisés** ou **ballonnisation apicale**



Japanese octopus pot

STRESS CARDIOMYOPATHY/TAKOTSUBO SYNDROME

An increasingly recognised acute **HF syndrome with myocardial injury** which mimics MI is known as stress cardiomyopathy (SCM) or Takotsubo syndrome (TTS)

SCM/TTS occurs in approximately **1–2% of patients presenting with suspected STEMI**, and when presentation is delayed it can also mimic non-STEMI.

The onset of SCM/TTS is frequently, but not always, triggered by **intense emotional or physical stresses such as bereavement**

Over 90% of patients reported in the largest cohorts are postmenopausal women, although younger women and men may also develop SCM/TTS.

Acute cardiovascular complications occur in approximately 50% of patients presenting with SCM/TTS, and the inpatient mortality is similar to STEMI (4–5%) due to cardiogenic shock, ventricular rupture or malignant arrhythmias.[160]

SUSPICION OF TAKOTSUBO

The diagnosis of SCM/TTS should be suspected when the clinical manifestations and electrocardiographic abnormalities are out of proportion to the degree of elevation of cTn values and **when the distribution of the LV wall motion abnormalities do not correlate with a single coronary artery distribution.**

STROKE HEART

Acute cardiac troponin increases (with rising and/or falling patterns) in stroke patients might either be caused by a concomitant primary coronary event resulting in acute myocardial ischemia (type 1 MI) or **acute myocardial injury secondary to the stroke.**

Acute alterations in the autonomic control of the heart with exaggerated catecholamine release have been suggested as a possible noncoronary mechanism of acute cardiac injury in ischemic stroke

The INSULA is important for the central autonomic regulation and is frequently affected in anterior circulation stroke, and an association between Right insula cortex stroke and increased cardiac troponin has been shown .

CKD (CHRONIC KIDNEY DISEASE)

Thus, baseline elevation of cTn values is common, and because they reflect myocardial injury, such elevation is highly prognostic over time.

PROMISE
TROPONIN IN STABLE OUTPATIENTS

Background **Evaluation of stable symptomatic outpatients** with suspected coronary artery disease (CAD)
Objectives This study sought to evaluate the prognostic meaning of single-molecule counting high-sensitivity troponin I (hsTnI) (normal range <6 ng/l) among outpatients with stable chest symptoms and suspected CAD.

Results The study sample consisted of 4,021 participants; 98.6% had measurable hsTnI concentrations. The median hsTnI value was 1.6 ng/l. In upper hsTnI quartiles, patients had higher-risk clinical profiles. Higher hsTnI concentrations were associated with greater event probabilities for death, acute MI, or hospitalization for unstable angina.

Conclusions **In symptomatic outpatients with suspected CAD, higher concentrations of hsTnI within the normal range were associated with heightened near-term risk for death, acute MI, or hospitalization.**

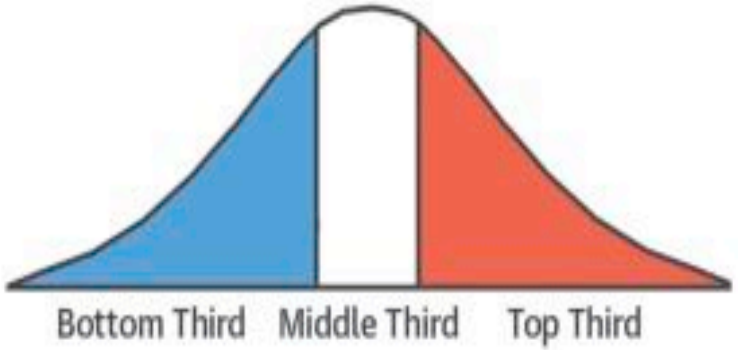
CENTRAL ILLUSTRATION: Troponin and Risk of CVD Outcomes

Study Data

154,052 participants without CVD
recruited by 28 prospective studies

Cardiac Troponin Concentration

Detectable in 80% with high-sensitivity assays



Risk of a First-ever CVD Event

Relative risk comparing top vs. bottom third

CVD	+43%
Fatal CVD	+67%
CHD	+59%
Stroke	+35%

Willeit, P. et al. J Am Coll Cardiol. 2017;70(5):558-68.

When Troponin was a lousy essay, it was a great test. Now that it's a great assay it's a lousy test

Dr Bob Jesse +2017