Emergency Department ACS HEART Pathway 2021 - BUMC Supplement

Supplement (Version 7.21)

The BSW emergency department (ED) comprehensive acute coronary syndrome (ACS) HEART pathway is an evidence-based decision support system that uses validated ED risk stratification methods and shared decision making to improve care. The HEART pathway was initially rolled out throughout the BSW healthcare system in June of 2016 and has successfully helped our EDs safely risk stratify chest pain patients with excellent outcomes. The pathway also has proved to decrease ED length of stay, hospitalization rates, and avoid unnecessary stress testing and imaging that ultimately has saved our healthcare system millions of dollars in associated costs. At BUMC specifically, the pathway avoided over 10,700 admissions and reduced healthcare cost by $37 million dollars over a 3-year period, while also decreasing 30- and 90-day mortality rates.

This educational supplement outlines important updates to the pathway pending transition to our new Roche high sensitivity troponin T assay (projected rollout July 27, 2021). **All ED patients with chest pain or symptoms suspicious for ACS will be evaluated using the 2021 BSW ED comprehensive HEART pathway.** The goal is to rapidly identify patients with acute coronary occlusion MI that will benefit from immediate reperfusion and distinguish them from other patients with ACS who need hospitalization for medical treatment, observation, and less urgent cardiac testing. The pathway also supports rapid identification and safe discharge of patients with low risk of ACS who will not benefit from further testing and treatment in the acute setting. Patients with symptoms not suspicious for ACS are excluded from the HEART pathway, and do not require troponin testing specifically for the evaluation of ACS (used for risk stratification in other etiologies, PE, HF, etc.).

ED evaluation starts with a thorough history, exam, and careful interpretation of ECGs. ED risk stratification is communicated through EPIC documentation of the calculated HEART score and disposition guided using the BSW ED chest pain ADP (see separate pdf). This educational supplement to the pathway provides detailed guidance on the ED evaluation of ACS and highlights the importance of ED risk stratification in clinical decision making. It contains everything you need to know and provides useful clinical pearls to help guide disposition. Links to recommended reading and full references in support of this evidence-based pathway are attached.

Disclaimer: Ultimately, a thorough history, physical, and expertise in ECG interpretation will guide management of ACS. Clinical decision rules and pathways should not be used in isolation and clinical judgment may be used to override them at the discretion of the provider.

TABLE OF CONTENTS

Emergency Department ACS HEART Pathway 2021 - BUMC Supplement

BACKROUND & DEFINITIONS OF ACS

- There are roughly 10 million ED chest pain related visits per year in the US alone[1]
- < 20% of these patients will diagnosed with acute myocardial infarction (MI) or unstable angina pectoris (UA)[21]
- MI is the acute manifestation of CAD and ischemic heart disease, which is the leading cause of death in the US and many parts of the developed world, accounting for ~1/3 of all deaths in people > 35 years of age.
- The symptoms of acute coronary syndromes (ACS) range from asymptomatic to sudden death[2]
- ACS refers to a spectrum of coronary disease involving atherosclerotic plaque rupture and platelet-rich thrombus formation, causing stenosis or occlusion and diminished blood flow that results in hypoperfusion and ischemia, with or without infarction of the myocardium
- **The diagnosis of ACS should be considered in any patient with evidence or symptoms of acute myocardial ischemia (based on HPI & ECG)**
- **The spectrum of ACS presentations includes:**
    - **Acute myocardial infarction (AMI)**
        - **Type I MI** = caused by atherothrombotic CAD and usually precipitated by atherosclerotic plaque rupture or erosion. Requires clinical evidence of acute myocardial ischemia with detection of a rise and/or fall of troponin (at least 1 troponin value greater than the 99th percentile URL).[3]
            - **Need at least one of the following to diagnose Type I AMI;** symptoms of acute myocardial ischemia, new ischemic ECG changes, development of pathologic Q waves, imaging evidence of new myocardial viability loss or regional wall motion abnormality, or identification of coronary thrombus by angiography
        - **Type 2 MI** = related to ischemia due to increased oxygen demand or decreased supply. Requires detection of a rise and/or fall of troponin (at least 1 troponin value greater than the 99th percentile URL) and evidence of oxygen supply/demand imbalance, unrelated to coronary atherothrombosis.[3]
            - **Need at least one of the following to diagnose Type II AMI;** symptoms of acute myocardial ischemia, new ischemic ECG changes, development of pathologic Q waves, imaging evidence of new myocardial viability loss or regional wall motion abnormality
        - Type 3 MI = Patients who suffer cardiac death, with symptoms suggestive of myocardial ischemia accompanied by presumed new ischemic ECG changes or ventricular fibrillation, but die before

[1] Implementation of an Emergency Department High-Sensitivity Troponin Chest Pain Pathway in the United States. PMID: 30747757
[2] Meyers HP, Smith SW. Acute Coronary Syndromes. Corependium EM Textbook 2021
[3] Fourth Universal Definition of Myocardial Infarction. PMID: 30153967
[4] 2013 AHA STEMI Guideline PMID: 23247304, 2017 ACEP STEMI Clinical Policy PMID: 29056206, 2014 AHA NSTEMI Guideline PMID: 25260718, 2018 ACEP NSTEMI Clinical Policy PMID: 30342745
[4] 2013 AHA STEMI Guideline PMID: 23247304, 2017 ACEP STEMI Clinical Policy PMID: 29056206, 2014 AHA NSTEMI Guideline PMID: 25260718, 2018 ACEP NSTEMI Clinical Policy PMID: 30342745

BACKROUND & DEFINITIONS OF ACS (continued)

blood samples for biomarkers can be obtained, or before increases in cardiac biomarkers can be identified, or MI is detected by autopsy examination
        - Type 4 MI = associated with PCI (4a) or stent thrombosis (4b)
        - Type 5 MI = associated with cardiac surgery
    - Current US guidelines categorize acute MI into NSTEMI/STEMI which have considerable overlap: [4]
        - **NSTEMI** (elevated troponin without ECG STEMI criteria)
        - **STEMI** (occlusion MI that specifically meets STEMI ECG criteria)
    - **Occlusion MI (OMI)**
        - Type 1 MI resulting in total occlusion or near-occlusion of the infarct related epicardial vessel, with insufficient collateral circulation, such that full-thickness infarction will occur unless flow is restored immediately
    - **Non-occlusion MI (NOMI)**
        - Any MI (Type 1-5) that does not satisfy the description of occlusion MI above
    - **Unstable angina (UA)**
        - New or worsening symptoms of ischemia (or changing pattern of symptoms), experienced at rest or with minimal exertion, but with normal troponin (below the 99% URL)
        - UA is also referred to as "crescendo angina or pre-infarction angina", differentiating its acuity from stable angina and highlighting its short-term risk of acute MI in classically described cases
        - To date, changes in high sensitivity troponin below the 99% reference value have not been established as a factor for ruling in or out unstable angina[1]

Key Clinical Pearl: Just like acute MI, UA does not necessarily manifest with any ECG abnormalities, beware of this in patients with concerning symptoms with a normal or nonspecific initial ECG

- The pathophysiology of ACS is extremely dynamic and complete occlusion or spontaneous reperfusion can happen at any time. Hence, the ECG may show dynamic ischemic changes that must be looked for with serial ECGs when the history is suspicious
- **The goal in the ED is to quickly identify patients with acute coronary occlusion who may benefit from immediate reperfusion therapy.** However, in ACS without acute occlusion, reperfusion therapy has not proven beneficial and can in-fact be harmful, making early distinction critical

[1] Meyers HP, Smith SW. Acute Coronary Syndromes. Corependium EM Textbook 2021

Emergency Department ACS HEART Pathway 2021 - BUMC Supplement

BACKROUND & DEFINITIONS OF ACS (continued)

Over time, STEMI has become synonymous with acute occlusion MI, but it is not!

- STEMI criteria were not designed to diagnose acute coronary occlusion, and they are unfortunately far too insensitive and inaccurate especially in early ischemia [2]
- STEMI criteria misses more than ¼ of patients with acute occlusions that are categorized as NSTEMI and also results in a substantial burden of unnecessary false positive cath lab activations[3]
- NSTEMI with occlusion (STEMI (-) OMI) have roughly double the short- and long-term mortality of NSTEMIs without occlusion (STEMI (-) NOMI) [3]

Key Clinical Pearl: It is important to understand that you can use the ECG to detect patterns suggestive of occlusion MI that do not fulfill the classic STEMI criteria

- The earlier in the evolution of occlusion MI that the diagnosis is made, the sooner reperfusion can be achieved. Time is myocardium!
- ED providers who take care of ACS patients must be experts in ECG interpretation and identification of acute coronary occlusion to successfully advocate for patients who may not meet strict STEMI criteria but have evidence of OMI (See ECG section)
- **Despite the challenges, it is possible to safely diagnose and treat ACS in the ED while also avoiding unwarranted evaluation and unnecessary testing that may cause harm in patients who are not having an acute coronary event[4]**

HISTORY OF PRESENTING ILLNESS IN ACS

- **Textbook HPI:** Aged patient with acute onset chest discomfort radiating the left arm, shoulder, or jaw, with associated diaphoresis, nausea/vomiting, shortness of breath, often in context of exertion or stress
  - In reality, ACS is notorious for atypical presentations, especially among women, diabetics, the elderly, and non-white populations
  - 33% of both STEMI & NSTEMI, as many as 75% of patients > 75 years old, and 40% of women with AMI present without any chest pain whatsoever[1]
  - **All of the following patient descriptions of chest discomfort are associated with the same incidence of AMI:** Burning, pressure, squeezing, indigestion, crushing, tightness, numbness, and nondescript chest discomfort[2]
  - Only "stabbing" chest discomfort is associated with a lower probability of ACS
  - Clinical severity of symptoms ranges from silent MI to vague complaints in well appearing patients, to electrical/hemodynamic instability or sudden cardiac arrest

[2] PMID: 29020244, PMID: 25458652, PMID: 20920642, PMID: 20723851, PMID: 19332201, PMID: 15464674, PMID: 12231080, PMID: 11713132
[3] STEMI: A transitional fossil in MI classification? 2021PMID: link
[4] A Risk Assessment Score and Initial High-sensitivity Troponin Combine to Identify Low Risk of Acute Myocardial Infarction in the Emergency Department. PMID: 29131477

[1] Association of age and sex with myocardial infarction symptom presentation and in-hospital mortality PMID: 22357832, Prevalence, clinical characteristics, and mortality among patients with myocardial infarction presenting without chest pain PMID: 10866870
[2] Acute Chest Pain in the Emergency Room PMID: 3970650

HISTORY OF PRESENTING ILLNESS IN ACS (continued)

- **Physician Gestalt** (blinded to everything except initial history and physical) showed the following categories and likelihood ratios for ACS:[3]
  - "Definite" (4.0)
  - "Probable" (1.8)
  - "Could be" (0.66)
  - "Probably not" (0.20)
  - "Definitely not" (0.36)
  - Compare this with structured ED risk assessment with the HEART score, which was very useful for the diagnosis of ACS (high risk = 13.0), and identifying patients at low risk of ACS (0.20)
- **Summary of the most useful features & findings that increase the probability for ACS in the acute setting (+LR):**[4]
  - High risk HEART score > 6 (13.0)
  - High risk overall gestalt (4.0)
  - Abnormal previous stress test (3.1)
  - Hx of peripheral arterial disease (2.7)
  - Pain radiating to right or bilateral arms (2.6), also consider radiation to shoulder and jaws
  - Pain similar to prior ischemia (2.2)
  - Change in pattern of symptoms over previous 24h (2.0)
  - Prior known CAD (2.0)
  - Exertional chest pain
  - Diaphoresis, vomiting, older age
- **Summary of the most useful features & findings that decrease the probability of ACS in the acute setting (-LR):**[1]
  - Low risk HEART score ≤ 3 (0.20)
  - Pain fully reproducible by palpation (0.28)
  - Stabbing pain
  - Pain localized to a fingertip area
  - Pain that is fully pleuritic or fully positional
  - Symptoms that last only for a second
  - Improvement with exertion
  - Constant pain lasting many hours without ECG or troponin changes
  - Younger age
- **Risk Factors (RFs) in ACS:**
  - RFs for CAD are commonly elicited during evaluation for ACS; prior CAD, hypertension, diabetes, tobacco use, obesity, hyperlipidemia, cerebrovascular disease, peripheral vascular disease, a family history of CAD before 55 years of age, HIV/AIDS, and autoimmune disorders
  - A history of atherosclerotic disease (MI, CVA/TIA, PVD, etc.) is automatically assigned 2 points
  - Although important to ask about and look for, these are risk factors for CAD and may not be good surrogates for risk of ACS in acute settings. Multiple systemic reviews have confirmed the poor accuracy of any single risk factor, symptoms, or sign for the diagnosis of ACS

[3] Does This Patient With Chest Pain Have Acute Coronary Syndrome? PMID: 26547467
[4] Meyers HP, Smith SW. Acute Coronary Syndromes. Corependium EM Textbook 2021
[1] Meyers HP, Smith SW. Acute Coronary Syndromes. Corependium EM Textbook 2021

Emergency Department ACS HEART Pathway 2021 - BUMC Supplement

HISTORY OF PRESENTING ILLNESS IN ACS: CLINICAL PEARLS

Key Clinical Pearls:

- Ask the questions that matter and will change the probability of ACS
- Figure out how suspicious the history is for ACS. Document presence or absence of signs and symptoms described above and translate to a score of slightly suspicious (0), moderately (1), or highly (2) suspicious.
- If the history is not at least slightly suspicious for ACS, do not order unwarranted tests, and find an alternative cause of symptoms.
- **Be cautious of classifying any patient scored a 2 for history (highly suspicious) as low risk even if the HEART score total is ≤3.**

Key Clinical Pitfalls:

- Antacids should not be used as a diagnostic test for ruling in or out ACS. In fact, antacids may be associated with pain relief in as many as 25% of acute MI cases.
- Nitroglycerin should also not be used as a diagnostic test for ruling in or out ACS, as response to nitro has been found to be unhelpful (LR ~1.0)
- Be cautious when diagnosing chest pain as a GI etiology, cardiac ischemia can produce symptoms that mimic GI pathology and vice versa.
- **No single risk factor, symptom, sign, or physical exam feature in isolation has a high enough sensitivity or specificity for the diagnosis of ACS**

HISTORY OF PRESENTING ILLNESS IN ACS: Deep Dive

Summary of the mechanisms of ACS and their implications for therapy (PMID: 23697515 & 30605419)

- It has been traditionally thought that stenosis of atherosclerotic coronary arteries would progressively narrow the lumen of vessels, until a small thrombus of platelets would cause complete occlusion
- Stress testing and perfusion scanning have been used evaluate the ischemia that results from established or fixed stenosis, and coronary angiography used to visualize the degree of intraluminal stenosis
- Newer investigations into the pathogenesis of ACS show that the characteristic plaques actually expand outward into the arterial wall first, minimizing intraluminal encroachment during growth
- Luminal stenosis occurs late in the process of atherogenesis, when plaque growth outpaces the ability of the arterial wall to further expand outward
- Sizable plaques will live and grow in the coronary arterial walls and may evade detection on arteriograms
- Hence, the degree of intraluminal stenosis seen on traditional arteriograms is not indicative of the actual size or composition of the underlying plaques
- Intravascular ultrasound (IVUS) studies in have shown that culprit lesions often lie proximal to the sites of maximal stenosis (traditional targets of revascularization)
- More recent investigations also point to superficial erosion of plaques as another cause of ACS distinct from plaque rupture
- Nevertheless, there is a dissociation between the degree of luminal stenosis and

Emergency Department ACS HEART Pathway 2021 - BUMC Supplement

HISTORY OF PRESENTING ILLNESS IN ACS: Deep Dive (continued)

likelihood of ACS, which may explain why MI can occur without the classic demand-induced symptoms of angina that result from high-grade stenosis

Plaque vulnerability and likelihood of rupture is based on 3 major factors:

1. Size and integrity of the fibrous cap
2. Size of the underlying lipid core
3. Inflammatory cell composition in the lipid core

KEY POINT: Plaque vulnerability is more important than lumen stenosis or the size of the plaque

Recent stress testing and angiography cannot predict new plaque rupture!

- Problems with stress testing
  - Smaller plaques with thin-capped fibroatheromas may be more unstable and prone to rupture than thicker capped plaques. Both can evolve over time, and any observations of plaque stability represent a snapshot in time of a moving target
  - These smaller plaques can still rupture and cause complete occlusion and acute MI
    - Studies of infarct related arteries have shown that they often have non-obstructing plaques before rupture and MI
  - Smaller plaques are often associated with negative stress tests
    - A small study looking at the frequency of significant CAD in ED patients with chest pain and a recent negative or inconclusive stress test, showed that more than 20% of these patients were still found to have significant CAD (PMID: 21079714)
- Problems with coronary angiography
  - Cannot distinguish "stable" vs. "unstable" plaque composition
    - No information about fibrous cap
    - No information about lipid core
  - Cannot identify "coronary artery remodeling", a newer concept in atherogenesis
    - Can be identified on IVUS and autopsy studies
  - Cannot reliably identify recent plaque rupture
    - For example, classic unstable angina with plaque rupture but without complete occlusion

Key Clinical Pearls:

- **You can't always rely on a negative stress test or even "unremarkable" recent coronary angiogram/cath**
  - **Plaque vulnerability and stability is much more important than plaque size/stenosis**
  - **Although a completely normal angiogram with "clean coronary arteries" is reassuring, be careful with "unremarkable or non-significant" results that may not have captured information about plaque vulnerability and stability**
  - **Nothing risk stratifies to zero!**
- **The history of presenting illness and clinical suspicion for ACS should guide management**

Emergency Department ACS HEART Pathway 2021 - BUMC Supplement

THE ELECTROCARDIOGRAM (ECG/EKG) IN ACS

- Despite its limited sensitivity and specificity, the ECG remains the most important initial test in the workup of ACS
- **Obtain a STAT ECG in the first 10 minutes of arrival to help identify patients with acute coronary occlusion MI (OMI) that may benefit from emergent reperfusion, as this is a time sensitive decision that carries the greatest mortality benefit of all interventions in ACS (aside from aspirin)**
- **Always compare the initial ECG (which may be initially normal in ACS) to previous/EMS ECGs and document findings. In patients with ongoing symptoms that are highly suspicious of ACS but have a non-diagnostic initial ECG, repeat ECGs every 15-30 mins for the first hour or get continuous 12-lead ST segment monitoring[1] When in doubt, one ECG begets another!**
- The ECG undergoes a predictable pattern of changes during occlusion and reperfusion, and is a better identifier of successful reperfusion and a better predictor of viable myocardium than time since onset of symptoms

Epidemiology of ECGs in ACS

- The "classic STEMI criteria" traditionally used to identify OMI are neither sensitive nor specific for acute coronary occlusion
  - On initial ECG, STEMI criteria are 6.4% sensitive for any AMI, 35% sensitive for retrospectively adjudicated STEMI, and 21% sensitive for occlusion MI[2]
  - On serial ECGs, STEMI criteria are 9.4%, 51%, and 30% sensitive, respectively[2]
- True positive STEMI cases have near or total occlusion with insufficient collateral circulation (occlusion MI) vs. at least 25%-30% of NSTEMI cases who are also found later to have occlusion[3]
- NSTEMI with acute coronary occlusion (STEMI (-) OMI) have roughly double the short- and long-term mortality of NSTEMIs without coronary occlusion (STEMI (-) NOMI)
- Both Type I & II MI's may present with or without STEMI criteria depending on the degree and extent of ischemia, although fewer than 5% of type 2 MI cases have ischemic ST elevation[4]

Key Clinical Pearls:

- **Many patients with ACS will present with initial ECGs that show no evidence of acute ischemia, much less STEMI. Non-specific ECG abnormalities that are non-diagnostic in patients with concerning history or symptoms must be repeated**
- **Always compare repeat ECGs with the initial & prior/EMS ECGs**
- **When in doubt, always perform serial ECGs looking for dynamic changes!**
- **Practice recognizing ECG patterns suggestive of OMI to more accurately identify patients that need emergent reperfusion**

[1] 2013 AHA STEMI Guideline PMID: 23247304, 2017 ACEP STEMI Clinical Policy PMID: 29056206,
[2] *Heart Disease and Stroke Statistics—2014 Update:* Circulation, Vol 129.; 2014. PMID: 24352519
[3] PMID: 29020244, PMID: 25458652, PMID: 20920642, PMID: 20723851, PMID: 19332201, PMID: 15464674, PMID: 12231080, PMID: 11713132
[4] Diagnosis of Type I versus Type II Myocardial Infarction in Emergency Department Patients with Ischemic Symptoms. PMID: Link

THE ELECTROCARDIOGRAM (ECG/EKG) IN ACS (continued)

ECG evidence of occlusion MI

- **Diagnostic ECGs**
  - **Current STEMI criteria[1]:** New or presumed new, ST-segment elevation (STE) ≥ 1.0 mm (measured at the J-point in 2 contiguous leads) is required in all leads (except V2, V3, V3R, V4R, V7-V9)
    - **Leads V2 & V3 are sex and age specific:**
      - Women: ≥ 1.5 mm
      - Men ≥ 40 years old: ≥ 2.0 mm
      - Men < 40 years old: ≥ 2.5 mm
    - **Right sided leads V3R & V4R (RV STEMI)**
      - Women: ≥ 0.5 mm
      - Men ≥ 30 years old: ≥ 0.5 mm
      - Men < 30 years old: ≥ 1.0 mm
    - **Posterior leads V7-V9 (Posterior STEMI)**
      - Women: ≥ 0.5 mm
      - Men ≥ 40 years old: ≥ 0.5 mm
      - Men < 40 years old: ≥ 1.0 mm
  - **Sgarbossa Criteria for LBBB & Paced Rhythms[2]**
    - Presence of LBBB or RV paced rhythms (LBBB pattern) does not obviate the ability to diagnose acute occlusion and can be detected with good specificity
    - AHA & ESC guidelines recommend that LBBB (regardless of chronicity) with positive original Sgarbossa criteria should be considered a "STEMI equivalent" requiring emergency reperfusion
  - **Current national guidelines also recommend emergency reperfusion therapy for STEMI, as well as NSTEMI with the following situations:**
    - electrical or hemodynamic instability
    - acutely worsening heart failure
    - ongoing ischemia despite maximal medical management (aspirin, antiplatelet, anticoagulant, nitroglycerin)

KEY ECG INTERPRETATION PEARL:

- **Measure your ST segments accurately -** The criteria state that ST elevation is measured at the J-Point, relative to the QRS onset (PQ junction). However, in patients with a stable baseline, the TP segment is a more accurate method to evaluate the magnitude of ST-segment deviation. Tachycardia and baseline shift can make this determination difficult

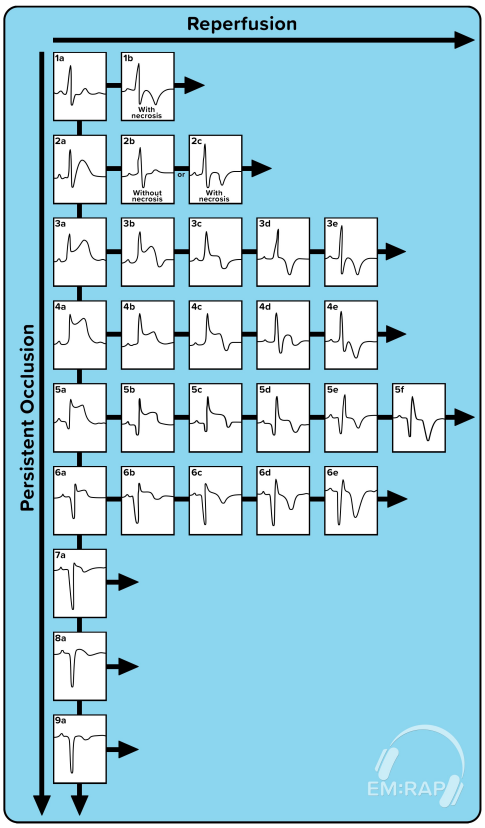
[1] PMID: 30153967- Fourth Universal Definition of Myocardial Infarction
[2] PMIDs: 28886621, 8880802, 8602576, 21079708, 22939607, 24016487, 26678648, 32627643.
[2] PMID: 29020244, PMID: 25458652, PMID: 20920642, PMID: 20723851, PMID: 19332201, PMID: 15464674, PMID: 12231080, PMID: 11713132

THE ELECTROCARDIOGRAM (ECG/EKG) IN ACS (continued)

Earliest ECG evidence of occlusion MI

- Expert ECG skills in identification of OMI is complex and out of the scope for this supplement, feel free to reach out to me for references and resources as needed. The following highlights some important evidence of OMI to consider:
- The earliest ECG sign of ischemia is QT lengthening, followed by an increased area under the T wave (**hyperacute T wave**) which signals a viable but ischemic myocardium
- As ischemia progresses, the ST segments begin to elevate, T wave inversion and Q wave formation occurs depending on timing and completeness of reperfusion
- Irreversible infarction is signaled by Q wave formation, but Q waves may also form in salvageable myocardium (especially in anterior MI)[2]. Hence, Q waves should not be used as a reason to withhold immediate reperfusion when otherwise indicated
- Truly ischemic ECGs should evolve over time, but the occluded artery may undergo reperfusion and reocclusion at any time, see patterns associated with this process below:

- With reperfusion, resolution of hyperacute T waves and ST segment is expected, before progression of terminal, then full T wave inversion over the course of hours to days
- Current guidelines suggest using improvement in chest pain, and > 70% resolution of STE or reperfusion arrhythmias (AIVR) as indicators of reperfusion, but they remain imprecise
- Evidence suggests that even asymptomatic patients with persistent ECG evidence of full thickness infarction should have immediate angiogram & PCI considered[1]
- **ECG experts can use the ECG to infer when the infarct related artery is reperfusing or re-occluding in real time, with greater accuracy than observing symptoms alone[2]**



[2] Appearance of abnormal Q waves early in the course of acute myocardial infarction: implications for efficacy of thrombolytic therapy PMID: 7897120
[1] Mechanical reperfusion in patients with acute myocardial infarction presenting more than 12 hours from symptom onset: a randomized controlled trial. JAMA. 2005;293(23):2865-2872. PMID: 15956631
[2] Early continuous ST segment monitoring in unstable angina: prognostic value additional to the clinical characteristics and the admission electrocardiogram. PMID: 8800982
[3] New Insights Into the Use of the 12-Lead Electrocardiogram for Diagnosing Acute Myocardial Infarction in the Emergency Department PMID: 29407007

THE ELECTROCARDIOGRAM (ECG/EKG) IN ACS (continued)

STEMI (-) ECG evidence of acute coronary occlusion MI[3]

- **Hyperacute T waves**
  - STE may be preceded by large hyperacute T waves which might be a subtle finding of early OMI
  - The term has never been formally defined, but has been shown in the past to be an independent marker of benefit from thrombolytics
  - QRS voltage should be proportional to the size of the T wave, beware of large broad based T waves, especially when larger than the associated QRS complex
- **De Winter's T waves[4]**
  - Abnormal T waves that are high risk for acute anterior MI and suggestive of acute proximal LAD occlusion
  - 1-3mm ST depression at the J-point in mid precordial leads, leading to tall symmetric T waves
- **Modified Sgarbossa Criteria for LBBB & Barcelona Criteria[5]**
  - AHA guidelines recommend that LBBB (regardless of chronicity) with positive original Sgarbossa criteria should be considered a "STEMI equivalent" requiring emergency reperfusion
  - Modified criteria have been validated to perform better
    - Modified Sgarbossa for LBBB, any 1 of 3 criteria, 80% sensitive, 99% specific for OMI
    - Modified Sgarbossa for Paced rhythms, any 1 of 3 criteria, 67% sensitive, 99% specific for OMI
- **Modified Sgarbossa Criteria for Paced rhythms**
  - Included in ESC 2017 Guidelines, uses relative rather than absolute values of discordant STE
- **ST elevation in aVR associated with widespread ST depression**
  - Included in ESC 2017 Guidelines, many non-cardiac conditions can also present with this pattern, but highly concerning for proximal or multivessel disease in patients with symptoms concerning for ACS
- **Subtle/Minor inferior STE with reciprocal STD in aVL**
  - With any amount of STD in aVL, any STE in II, III, & aVF is MI until proven otherwise, as this can be an indicator of early reciprocal changes of occult MI (LCx lesions classically)
  - 99% sensitive, 100% specific
- **Subtle anterior STE, terminal QRS distortion**
  - Differentiating normal STE from ischemic STE from LAD occlusion
  - Absence of S wave and J wave in either V2 or V3, 20% sensitive, 100% specific (terminal QRS distortion)
- **Non-contiguous STE**
  - **South African flag pattern**
    - STE in aVL and V2 with concurrent inferior ST depression
    - Suggestive of occlusion of first diagonal branch of LAD

[4] PMIDs:18987380, 19620137, 21146650, 20591442, 23863685, 24176590
[5] PMIDs: 28886621, 8880802, 8602576, 21079708, 22939607, 24016487, 26678648, 32627643

STEMI (-) ECG evidence of acute coronary occlusion MI[3] (continued)

- **Aslanger's pattern**
  - Inferior STE isolated to lead III, with concomitant STD in any of V4-V6, with positive/terminally positive T wave, ST segment in V1>V2
  - Suggestive of occlusion MI in patients with concomitant multivessel disease, does not display contiguous ST elevation or fulfil STEMI criteria
- **STEMI Mimics –** Several conditions may cause ST elevation, not just MI, resulting in false positive cath lab activation that may result in harm. Consider the following:
  - LVH
  - LV aneurysm
  - Acute pericarditis
  - Early repolarization
  - Peri/myocarditis
  - Takotsubo cardiomyopathy
  - LBBB & paced rhythms, etc.

KEY PEARLS:

- **Take caution in scoring patients with highly suspicious history (2-point Hx) or acute ECG changes suggestive of ischemia (2-point ECG - ST depression, ischemic T wave inversions) as low risk regard less of total score.**
- **Focusing only on ST elevation that meets "STEMI criteria" will simply result in missed OMIs - STEMI criteria will miss more than 1/4 of patients with angiogram proven OMI (false negative per STEMI criteria, classified as NSTEMI) and also result in a significant amount of unnecessary cath lab activations (false positives)[2]**

[4] PMIDs:18987380, 19620137, 21146650, 20591442, 23863685, 24176590

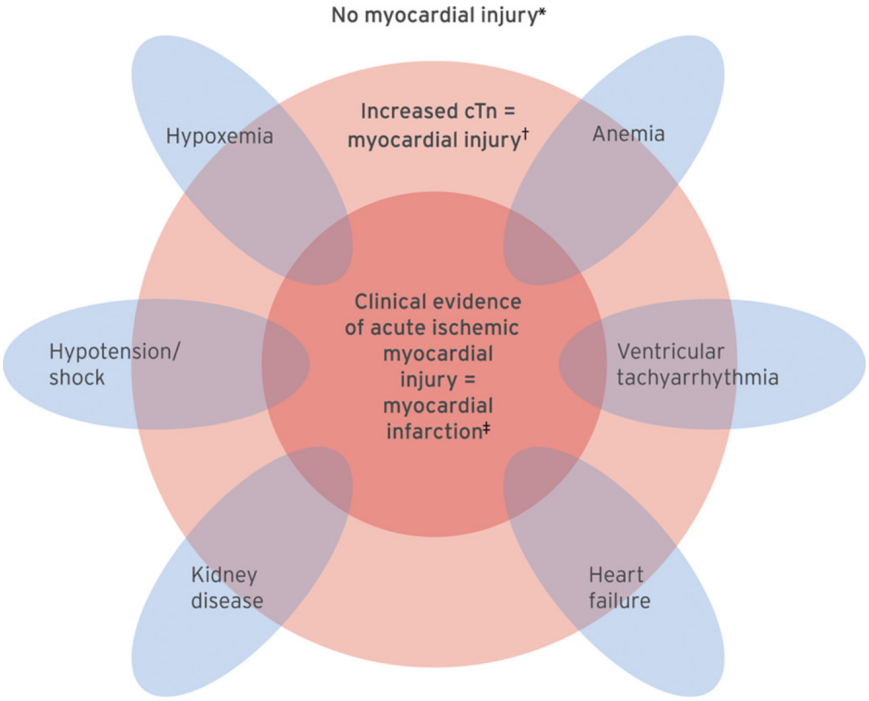
Emergency Department ACS HEART Pathway 2021 - BUMC Supplement

UTILIZATION OF TROPONIN (hs-TnT) IN ACS

- Troponin is an important part of the work-up in patients with ACS. However, the decision to perform immediate reperfusion should only involve troponin when the ECG is not diagnostic, and the symptoms persist
- **Troponin elevation must be interpreted in the context of clinical presentation**
    - Troponin may be elevated in many disease states in addition to MI
    - Among ED patients with at least one elevated troponin level, up to 85% are found in conditions other than type 1 MI (i.e., type 2 MI, non-MI acute myocardial injury, and chronic myocardial injury)
    - Elevated troponin of any etiology, regardless of presence of ACS, is associated with higher mortality than in its absence.
        - Any troponin (myocardial injury or infarction) is worse than no troponin
        - Higher troponin is worse than lower troponin
- **High-sensitivity troponins (hs-TnT)** are defined by the fact that > 50% of a normal population have a measurable troponin level with a good coefficient of variation (the population that is less than the 99th percentile reference range). The following terminology is helpful in clarifying how a hsT assays performs:
    - 99th percentile clinical decision values: 99th percentile of normal healthy individuals has been selected as a consensus decision point. Lower thresholds result in excessive false positive results.
        - These levels are sex specific: < 14 ng/L (female) and < 22 ng/L (male) are below the 99th percentile upper reference level (URL)
    - Coefficient of variation: measure of assay imprecision at any given concentration. Should be 10% or less at the 99th percentile URL for hsT assays. Good precision allows for confident identification of small changes in biomarker concentration
    - Limit of blank (LoB): background noise present in measurement system when no troponin is present
    - Limit of detection (LoD): lowest concentration of detectable troponin in 95% of measurements. Imprecision at LoD is often high, making measurements inaccurate
    - Limit of quantitation (LoQ): lowest troponin concentration that can be reported as a number with specified certainty
- **hs-T were introduced in an effort to improve detection of MI, and are able to detect much lower concentrations of the troponin protein, hence shortening the time interval required to identify myocardial injury**

KEY CLINICAL PEARL: Up to 50% of patients without ACS will have a detectable (but not abnormal) hsT, so it is critical to learn how to interpret these values

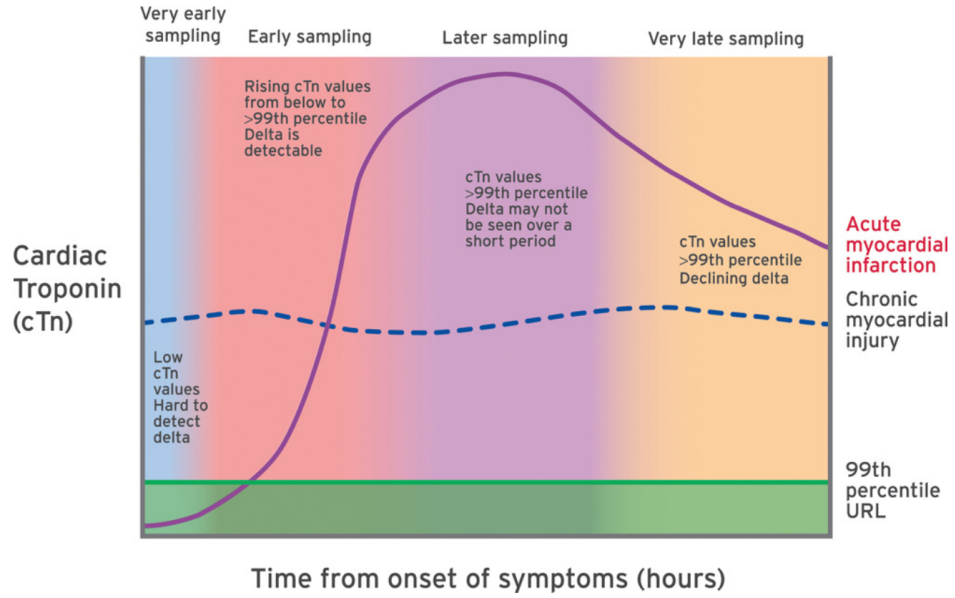
UTILIZATION OF TROPONIN (hs-TnT) IN ACS (continued)



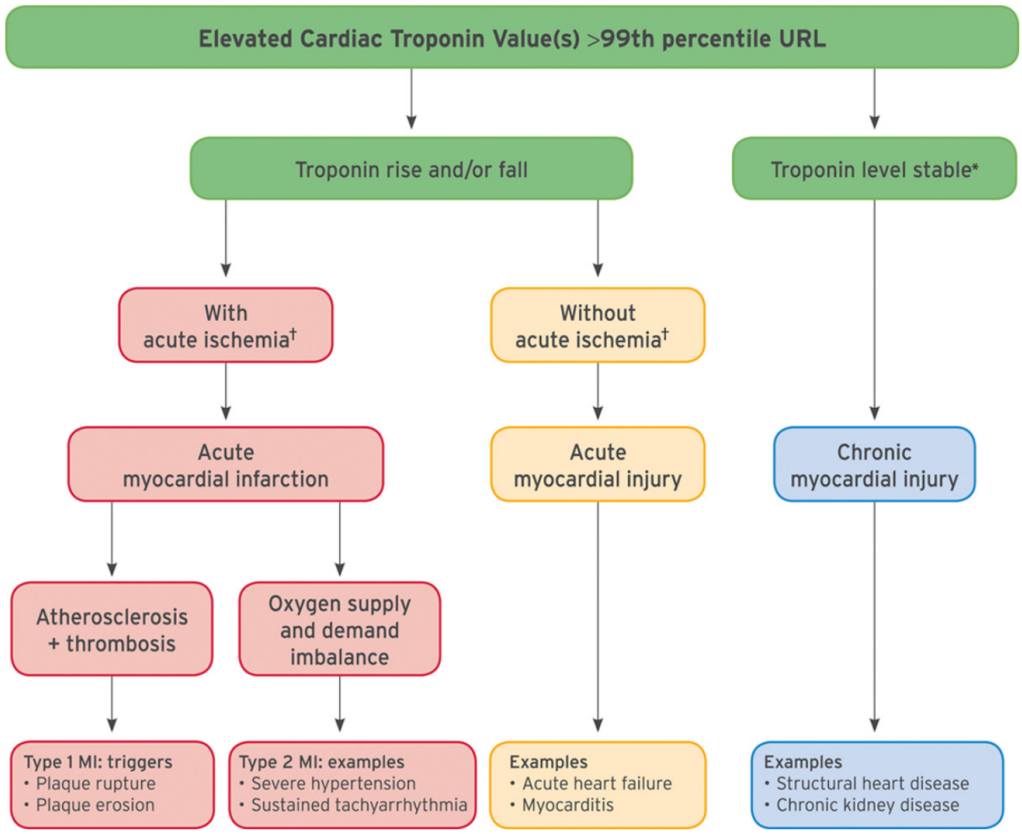
- **Criterial for Myocardial Injury:**
  - Detection of any elevated troponin level above the 99th percentile URL
  - Myocardial infarction (MI) is only one cause of myocardial injury[1]
  - Various clinical entities may cause myocardial injury:
    - Cardiac conditions
      - Heart failure, myocarditis, ventricular tachyarrhythmias, cardiomyopathy (any type), takotsubo syndrome, coronary revascularization & other cardiac procedures, catheter ablation, defibrillator shocks, cardiac contusion
    - Systemic conditions
      - Sepsis/infectious disease, chronic kidney disease/ESRD, stroke, subarachnoid hemorrhage, pulmonary hypertension, pulmonary embolism, infiltrative diseases (amyloidosis, sarcoidosis, etc.), chemotherapeutic agents, critical illness, strenuous exercise
  - Injury is considered acute of there is a rise and or fall of Troponin values
  - Troponin does not easily differentiate acute from chronic injury
    - Acute injury seen in ACS should have a rise in troponin over time, a Δ > 5 ng/L is typically consistent with acute injury
    - Chronic conditions that produce troponin elevation rarely show increase over time intervals of 2 - 6 hours
    - Timing matters and serial sampling is important

[1] Fourth Universal Definition of Myocardial Infarction PMID: 30153967

UTILIZATION OF TROPONIN (hs-TnT) IN ACS (continued)



KEY CLINICAL PEARL: A decrease in troponin over time can actually indicate an acute injury that occurred days ago but is less specific for ACS and more often associated with non-ACS conditions. When troponin is declining during serial testing, you need to have clinical criteria (see AMI definition) to make the diagnosis of ACS/MI



UTILIZATION OF TROPONIN (hs-TnT) IN ACS (continued)

- **Troponin and Acute Myocardial Infarction**
  - Diagnosis is made based on a rise or fall of troponin, with at least 1 measurement exceeding the 99th percentile URL (myocardial injury), in the context of reasonable suspicion for coronary ischemia (symptoms, ECG changes, evidence for loss of myocardial function, or demonstration of obstructive coronary disease)
  - Although changes below the 99th percentile may reveal acute coronary events, the use of lower concentrations is not yet endorsed by the 4th universal definition of MI[1]
  - AMI is classified into one of 5 different types, but type 1 and type 2 MI are most commonly encountered. However, an elevated troponin does not indicate the underlying cause of injury, and the abnormal result may be chronic (i.e., CKD/ESRD, etc.) or due to one of several other possible conditions

KEY CLINICAL PEARL: MI is a clinical diagnosis that is not defined by troponin alone, evidence of myocardial ischemia is required. Do not diagnose myocardial infarction based on troponin alone, consider diagnosis of myocardial injury instead

  - When determining whether there has been a rise or fall on serial samples, absolute change has greater diagnostic accuracy for AMI than relative change. The rise can be faster than the fall in values
    - Generally, a change threshold of 50-80% of the baseline concentration is significant (ex. Baseline of 14 ng/L, with change of 7ng/L or greater)
  - Serial testing becomes even more important with chronic comorbid conditions (elderly, CKD/ESRD, HF, etc.). Chronic myocardial injury in these settings is valid and should not be considered a false positive. Even if not suggestive of Type 1 MI, troponin elevation is still suggestive of poor cardiovascular prognosis

KEY CLINICAL PEARL: In general, hsT measurements with lower change criteria have higher sensitivity and lower specificity. No hsT change criteria have perfect sensitivity and specificity for acute MI, thus clinical judgment remains essential to confirm or refute the diagnosis

  - The differential for abnormal hsT is very broad at low concentrations, and the differential narrows with higher values. The absolute baseline concentration, as well as the delta will often determine whether acute MI has occurred
  - When interpreting an abnormal troponin, it is important to consider the other possible causes, including but not limited to PE, heart failure, myocarditis, and sepsis
  - In Type 1 MI, it is common to see rapid and substantial increases in hsT over a few hours
    - Of note, myocarditis can also cause large rises that overlaps expected changes in Type 1 MI. Stress cardiomyopathy, PE, and critical illness are other considerations

[1] Recommendations for Institutions Transitioning to High-Sensitivity Troponin Testing: JACC Scientific Expert Panel. PMID: 30798981, Fourth Universal Definition of Myocardial Infarction. PMID: 30153967

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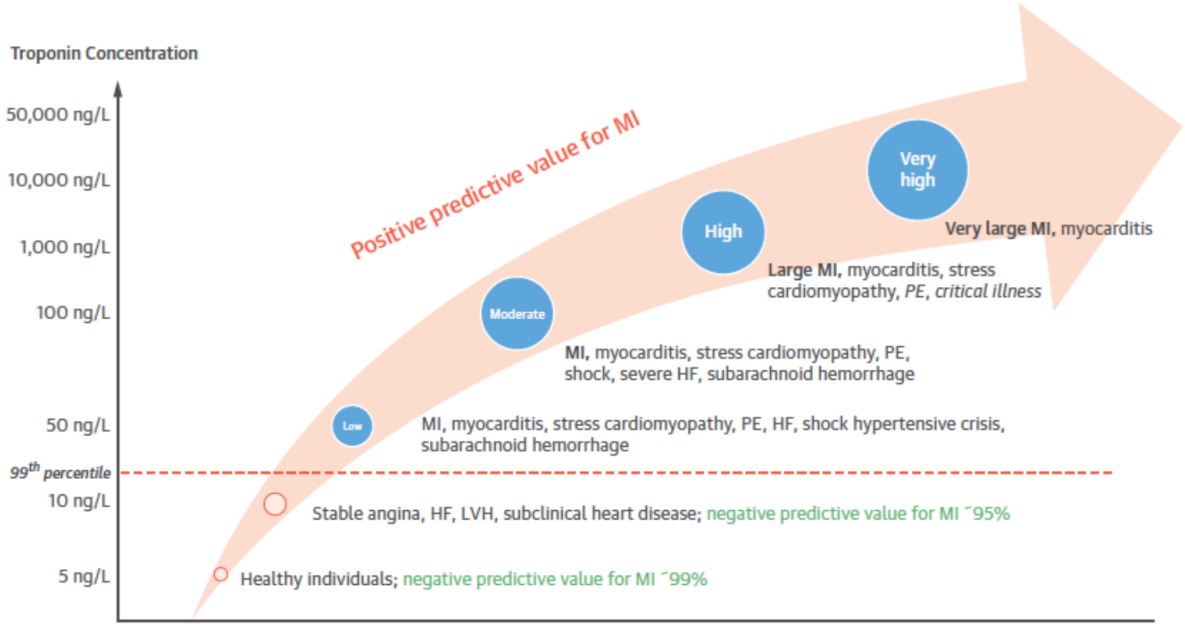
UTILIZATION OF TROPONIN (hs-TnT) IN ACS (continued)



- BUMC will be implementing new Roche chemistry system with Gen 5 (higher sensitivity) Troponin T (July 27, 2021 expected rollout)
- Any troponin ≥ 52 ng/L or Δ > 5 ng/L @ 1 hour, or Δ > 7 ng/L @ 3 hours is abnormal
- Troponin lower than age specific cutoffs below the 99th percentile URL (< 14 (female) or < 22 (male)) with Δ ≤ 3 ng/L @ 1 hour, or Δ ≤ 5 ng/L @ 3 hours are ruled out for MI
- When calculating HEART score utilizing hsT, the "T" is scored:
  - 0 = < 14 ng/L (Female), < 22 ng/L (Male)
  - 1 = 14-41 ng/L (Female), 22-51 ng/L (Male)
  - 2 = ≥ 42 ng/L (Female), ≥ 52 ng/L (Male)

KEY CLINICAL PEARL: When suspicion for ACS is below the testing threshold for troponin, do not order troponin for ACS. When ordered, consider routinely ordering a 1-hour delta unless patient is very low risk for ACS and the initial troponin is undetectable (<6ng/L) and onset of symptoms is > 3h from initial result.

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BUMC ED: DISPOSITION OF ACS

Admission (Cardiology/HMD)

- Patients judged to potentially benefit from immediate reperfusion should have prompt consultation from interventional cardiology, and be admitted
  - **STEMI per code STEMI institutional guideline**
    - STEMI diagnosis to balloon time should be as short as possible, with benefit accruing even at times < 60-90 minutes
  - **ECG findings that do not meet STEMI guidelines but are suggestive of acute occlusion MI (STEMI (-) OMI (+)) should have prompt consultation from interventional cardiology and consideration of immediate reperfusion**
  - **Other indications for urgent or emergent cath s/p consultation with interventional cardiology**
    - electrical or hemodynamic instability
    - acutely worsening heart failure
    - ongoing ischemia despite maximal medical management (aspirin, antiplatelet, anticoagulant, nitroglycerin)
- **Patients with high-risk HEART scores (≥ 7) and all other patients with the diagnosis of ACS or myocardial infarction/unstable angina, without indication for emergent cath should ideally have consultation with the on-call cardiologist** (consult service, not interventional on call) for consideration of admission to BHVH/cardiology, with timing and benefit of angiography vs. non-invasive therapy to be determined by Cardiology team
- MD/DO discretion may be used for consultation to admit patients with concern for ACS directly to cardiology for reasons not covered above

Observation (HMD hospitalization/CDU)

- **Patients with moderate risk HEART scores (4-6) requiring further testing or observation that cannot be reasonably obtained from the ED in 3 hours should be placed in observation units when available or hospitalized as needed**
- ED MD/DO discretion may also be used as criteria for placement in observation, when it is truly thought that patients may benefit from further observation and testing
  - For example, otherwise low or moderate risk HEART score with continued clinical concern for ACS or undifferentiated active symptoms requiring further testing or non-emergent cardiology consultation. When diagnosing ACS or UA, consider consultation with cardiology to discuss individualized plan of care prior to placement in observation
- Stress testing and coronary CTAs are recommended by guidelines to exclude myocardial ischemia or obstructive CAD among patients with acute chest pain. However, this paradigm is historically associated with over testing, a low yield of true positive findings, ED/observation unit overcrowding, radiation exposure and high cost
  - **When to consider cardiology consultation:**
    - Prior to stress testing in patients with down trending troponin (with at least one value above 99th percentile URL)

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Observation (HMD hospitalization/CDU) continued

- **When to consider cardiology consultation: (continued)**
  - Uncertainty with what type of stress test is most appropriate
  - Active chest pain with unclear cardiovascular stability
  - Any concern that patient may have an indication for urgent or emergent cath
- **It may be safe to perform a stress test in the following situations:**
  - Active chest pain, but ruled out by troponin & clinically stable
  - No active chest pain or resolved pain as clinically indicated (indeterminant troponins or ruled out by troponin)

KEY CLINICAL PITFALL: Do not perform a stress test on a patient with active chest pain AND a rising troponin

- **Stress Testing/CCTA considerations:**
  - **No testing**: low risk HEART scores ruled out by serial troponins/ECGs have very low rate of major adverse cardiac events, testing this population may cause harm
  - **Consider treadmill stress/echo**; when patient can exercise/walk on treadmill, BMI < 40, no significant ECG abnormalities (i.e., conduction abnormalities, paced rhythms, LVH with strain, etc.)
  - **Consider pharmacological stress echo**; when patient cannot exercise/walk, has lung disease, no significant ECG abnormalities (i.e., conduction abnormalities, paced rhythms, LVH with strain, etc.)
  - **Consider nuclear medicine perfusion**; if patient unable to exercise or has features that make obtaining sufficient echo images technically difficult (prior MI/baseline wall motion abnormalities, dilated cardiomyopathy, morbid obesity, COPD/emphysema, etc.), history of atrial fibrillation or arrhythmia
  - **Consider coronary CTA;** GFR > 30, normal sinus rhythm, no known CAD, no significant IV contrast allergy, able to get HR < 65 (with or without beta blocker)

KEY CLINICAL PEARL: Routine hospitalization or cardiology consultation without clinical criteria for MI is not indicated

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Discharge from the ED

- Our HEART pathway combines risk stratification with the HEART score and serial hs-TnT measurements to improve the sensitivity and NPV of just using the HEART score or troponin in isolation
- A prospective study using a HEART pathway identified 30.7% of patients as low risk with a NPV of 99.6% for 30-day death or MI[1]. Pathway implementation is associated with decreased hospitalization, increased identification of index visits MIs, and a very low death and MI rate among low-risk patients

KEY CLINICAL PEARL: For a patient to be considered low risk (HEART score 0-3) and eligible for early discharge, our pathway requires; a detailed history not highly suspicious for ACS, expert ECG interpretation that is not suggestive of acute ischemia, and troponin results that meets rule out criteria

- Very low risk patients with symptoms that reliably began ≥ 3h may be discharged with a single undetectable hsT (<6 ng/L) after shared decision making when clinicians think this is appropriate, consider serial troponin testing when history is unreliable or with any clinical concern
- When clinical suspicion is low and other potential emergent causes of symptoms have been evaluated, all other low & moderate risk HEART score (<7) patients that rule out for MI by ECG & troponin criteria may be discharged after shared decision making with strict return precautions and PCP follow up advised, no cardiology follow up required
- Low risk patients with minimal changes in repeat troponins that do not rule in (Δ < 5 ng/L @ 1 hours, or Δ <7 ng/L @ 3 hours) not thought to be clinically relevant; may be discharged with close outpatient follow up after shared decision making conversation with strict return precautions advised, consider scheduled follow up with cardiology as needed
- Moderate risk patients with minimal changes in repeat troponins (Δ < 5 ng/L @ any time) not thought to be clinically relevant; may be discharged with close outpatient follow up after shared decision making conversation with strict return precautions advised, consider scheduled outpatient cardiology follow up within 72hrs (Amb referral to BHVH at dispo)

KEY CLINICAL PITFALL: Be cautious of classifying any patient with history scored a 2 (highly suspicious) as low risk even if the HEART score total is ≤3. This pathway supports sound clinical judgement and is not designed to promote early discharge in patients where clinical concerns persist. Discharging any patients with high clinical suspicion for ACS/UA even when initial troponins and ECGs are normal is strongly discouraged.

[1] Safely Identifying Emergency Department Patients with Acute Chest Pain for Early Discharge: HEART Pathway Accelerated Diagnostic Protocol. PMID: 30571347

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RECOMMENDED READING - TOP 10 - (full references below):

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6. Allen BR, Christenson RH, Cohen SA, et al. Diagnostic Performance of High-Sensitivity Cardiac Troponin T Strategies and Clinical Variables in a Multisite US Cohort. *Circulation*. Published online 2021:1659-1672. PMID: 33474976
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10. Sandoval Y, Jaffe AS. Using High-Sensitivity Cardiac Troponin T for Acute Cardiac Care. *Am J Med*. 2017;130(12):1358-1365.e1. PMID: 28843652

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2. Gara PTO, Kushner FG, Ascheim DD, et al. ACCF / AHA Guideline 2013 ACCF / AHA Guideline for the Management of ST-Elevation Myocardial Infarction A Report of the American College of Cardiology Foundation / American Heart Association Task Force on Practice Guidelines. Published online 2013:362-425. PMID: 23247304
3. Torbati SS. Clinical Policy: Emergency Department Management of Patients Needing Reperfusion Therapy for Acute ST-Segment Elevation Myocardial Infarction. *Ann Emerg Med*. 2017;70(5):724-739. PMID: 29056206
4. Amsterdam EA, Wenger NK, Brindis RG, et al. AHA / ACC Guideline 2014 AHA / ACC Guideline for the Management of Patients with Non - ST-Elevation Acute Coronary Syndromes A Report of the American College of Cardiology / American Heart Association Task Force on Practice Guidelines.; 2014. PMID: 25260718
5. Subcommittee P, et al. Clinical Policy: Critical Issues in the Evaluation and Management of Emergency Department Patients With Suspected Non - ST-Elevation Acute Coronary Syndromes. *Ann Emerg Med*. 2018;72(5):e65-e106. PMID: 30342745

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