Chapter 76 – Acute Coronary Syndromes Part B

Episode Overview:

1. List Sgarbossa criteria for AMI in pre-existing LBBB
2. What is Takotsubo cardiomyopathy and how does it present?
3. Describe the kinetics of cardiac biomarkers (Troponins and CK)
4. List DDx for ↑ Troponin
5. What is the utility of CTA in the diagnosis of MI?
6. What is the role of ED-based chest pain centers?
7. List 3 phases of delay in the management of AMI; and describe the time-points in ED management of AMI.
8. What are door-to-needle and door-to-balloon timelines by AHA recommendations?
9. Describe the mechanism of action and indications/contraindications for
   - Nitroglycerin
   - Morphine
   - B-Blockers
   - ACE-I
   - Antiplatelet Therapies
   - Anti-thrombins
10. Describe eligibility criteria for Fibrinolytics
    a. When should prehospital thrombolytics be used?
11. List contraindications to Fibrinolytic therapy in MI
12. What is the utility of Rescue PCI and Facilitated PCI?
13. List 5 indications for Rescue PCI
14. Describe factors assisting with decision to utilize PCI or thrombolytics
15. In NSTEMI, who will benefit from an early invasive strategy of management?

Wise Cracks:

1. What is the management of ACS in the setting of recent cocaine use?
2. How is STEMI diagnosed in the setting of LBBB?
3. How is STEMI diagnosed in the setting of a ventricular pacemaker?
4. When should you be getting a 15 lead ECG?

Rosen's in Perspective

For the RIP in this chapter let's recap two things from last episode: - the patho of ACS and why serial ECGs are key.

- "The pathophysiology of an acute coronary event includes (1) endothelial damage through plaque disruption, irregular luminal lesions, and shear injury; (2) platelet aggregation; (3) thrombus formation causing partial or total lumen occlusion; (4) coronary artery vasospasm; and (5) reperfusion injury caused by oxygen free radicals, calcium, and neutrophils. In patients with non infarction ACS, spontaneous fibrinolysis of the thrombus occurs rapidly, minimizing ischemic insult; persistence of the occlusive thrombus, however, results in MI."
- Let's review the importance of serial ECGs before we dive into today's content.
    - As many as 20% of people diagnosed with an AMI had a nonspecific or normal ECG at the time of their presentation.

- A single ECG does not rule out ACS - a single ECG is only 60% sensitive and 90% specific in making the diagnosis of AMI
- Serial (q20 min ECG's) are extremely helpful in the patient who may have ACS
  - Remember someone can have a perfectly normal workup - and still be in the class of Unstable Angina - (i.e. No myocardial necrosis has occurred)

Now let's get back to our questions!

1) List Sgarbossa criteria for AMI in pre-existing LBBB

(1) ST segment elevation of at least 1 mm that is concordant with the QRS complex;
(2) ST segment depression of at least 1 mm in lead V1, V2, or V3; and
(3) ST segment elevation of at least 5 mm that is discordant with the QRS complex.

"These findings were assigned weighted scores of 5, 3, and 2, respectively. For accuracy in diagnosis, a specificity of 90% requires a score of at least 3. Thus if an ECG features only discordant ST segment elevation of 5 mm or more but neither of the other two criteria, further testing is recommended before one can conclude that the ECG is indicative of AMI."

"Ultimately the approach to the patient with LBBB and possible MI remains complicated; diagnostic adjuncts to the history and physical examination (e.g., serial ECGs, comparison with prior ECGs, echocardiography, serum cardiac marker measurement) should be liberally used when the ECG does not show obvious evidence of AMI as noted by the Sgarbossa criteria."

2) What is Takotsubo cardiomyopathy and how does it present?

"Takotsubo cardiomyopathy is referred to as left apical ballooning or "broken heart" syndrome. Takotsubo cardiomyopathy features ST segment elevation (or deep T wave inversions) without evidence of obstructive CAD.

Positive serum markers for cardiac ischemia may be present, as well as hemodynamic compromise.

It occurs principally in postmenopausal women and characteristically is triggered by intense emotional stress. Ballooning of the left ventricular apex is seen on ventriculography or echocardiography. Prognosis is excellent, typically with recovery of normal wall motion within a month or less."

3) Describe the kinetics of cardiac biomarkers (Troponins and CK)

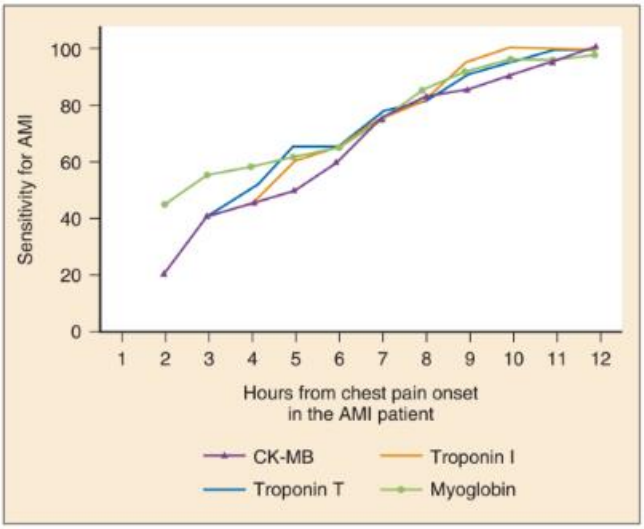


Figure 78-22. Serum marker sensitivity relative to the time of onset of chest pain in the patient with acute myocardial infarction. Data obtained from the medical literature. *AMI,* acute myocardial infarction; *CK-MB,* creatine phosphokinase MB fraction.

- Troponins
  - TnT, and TnI are the most sensitive and specific serum proteins for myocardial injury
  - They start rising within 2 hrs of symptom onset and stay elevated longer than CK-MB
    - They are nearly 100% sensitive for AMI at 12 hrs from symptom onset
  - High-sensitivity troponins are increasingly becoming used - and become elevated within a few hours from symptom onset
- CK-MB
  - Is a myocardial CK protein previously used to diagnose AMI
  - "In the setting of AMI, CK-MB is released and is detectable in the serum as early as 3 hours after onset of the necrosis. CK-MB characteristically peaks at 20 to 24 hours and becomes normal within 2 to 3 days after injury."
  - "Elevated CK-MB values identify a patient at considerable risk for a poor outcome but do not correlate well with infarct size. Unfortunately, skeletal muscle does contain small amounts of CK-MB, particularly the pelvic musculature. Abnormal CK-MB elevations may be seen in patients with trauma, muscular dystrophies, myositis, and rhabdomyolysis and after extremely vigorous exercise."
  - It isn't used anymore because of time constraints and lower diagnostic sensitivity
  - May have a role - in places without troponin testing

At this time there is no strong recommendation for multi-marker testing in the ED due to their poor specificity

4) List DDx for ↑ Troponin

Causes of Elevated Troponin – From UptoDate

Myocardial Ischemia

ACS
- STEMI
- NSTEMI

Other coronary ischemia
- Arrhythmia: Tachy- or brady-
- Cocaine/methamphetamine use
- Coronary intervention (PCI or cardiothoracic surgery)
- Coronary artery spasm (variant angina)
- Stable coronary ahterosclerotic disease in setting of increased O2 demand (eg. tachycardia)
- Severe hypertension
- Coronary embolus
- Aortic dissection
- Coronary artery vasculitis (SLE, Kawasaki's)

Non-coronary ischemia
- Shock (hypotension)
- Hypoxia
- Hypoperfusion
- PE
- Global ischemia
- Cardiothoracic surgery

Myocardial Injury with No Ischemia

Comorbidities
- Renal failure
- Sepsis
- Infiltrative disease
- Acute respiratory failure
- Stroke
- SAH

Specific identifiable precipitants
- Extreme exertion
- Cardiac contusion
- Burns >30% BSA
- Cardiotoxic meds: anthracyclines, herceptin
- Electrical shock
- CO exposure

Other

- Apical ballooning (Takotsubo)
- Myocarditis
- Myopericarditis
- Rhabdomyolysis involving cardiac muscle
- Hypertrophic cardiomyopathy
- Peripartum cardiomyopathy
- Heart failure, malignancy, stress cardiomyopathy

Broad categories: Lippism: "ACSss"

- Acute coronary ischemia
    - ACS
    - Cocaine
    - Variant angina
    - Coronary embolism/vasculitis
- Comorbidities causing myocardial injury
    - Renal failure, sepsis, ARDS, stroke, SAH
- Systemic shock states
    - Distributive shock states (sepsis, CO poisoning, burns)
    - Cardiogenic shock states (myocarditis / myocardial contusion / cardiomyopathy)

"The patient's history remains the most vital portion of the diagnostic evaluation of potential ACS. Serial testing substantially improves the sensitivity of these tests"

5) What is the utility of CTA in the diagnosis of MI?

Don't have a clearly beneficial role in the ED workup of chest-pain patients at this time. More research and implementation studies are needed before they become part of mainstream use. They (as well as the triple-rule out multi-detector CTA) are quite sensitive, but also can be non-specifically positive (as in the case of multiple calcifications / perfusion defects in someone known to have symptomatic CAD).

Again, we need to take a good history, do a comprehensive physical, serial ECGs, serial serum troponins, and arrange appropriate follow up!

6) What is the role of ED-based chest pain centers?

Don't have a role in our centre currently, but many American ED's (30% according to Rosen's) use them.

To quote the textbook:

"Approximately 80% of patients with chest pain can be safely evaluated in the ED with ultimate discharge to home. The resources required for a successful CPC [chest pain centres]-based operation in which patients undergo rapid exclusion of ACS through serial testing, continuous monitoring, and immediate provocative stress testing are consid- erable.

Although studies suggest that CPCs decrease the number of admissions, they may increase the number of patients seen in the ED for chest pain, and physicians may overuse the CPC accelerated diagnostic protocol approach in patients whom they would otherwise have discharged."

7) List 3 phases of delay in the management of AMI; and describe the time-points in ED management of AMI.

...time is muscle…and life…

"The opening of the occluded artery causes less adverse ventricular modeling, reduces occurrence of ventricular aneurysm, increases blood flow to myocardium, and improves electrophysiologic stability……each additional 30 minutes of delay to PCI compounding relative mortality risk by 7.5% at 1 year even when adjusted for baseline characteristics."

- Patient-bystander
    - Factors that prevent the patient to make initial contact with medical personnel (EMS)
- Preadmission
    - Time from decision to seek medical care to first contact at the ED
- Hospital
    - Delay to PCI / lysis

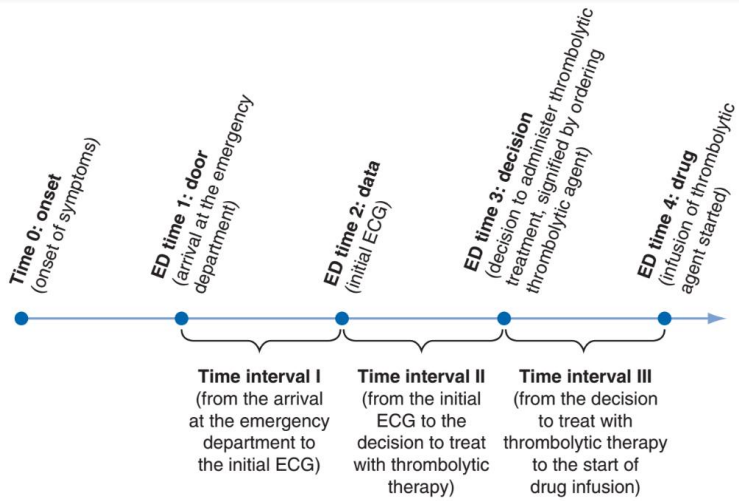


Figure 78-24. The four Ds of emergency department (ED)–based diagnosis and management of the patient with acute myocardial infarction (AMI). Shown are the process time points and intervals through which the patient with AMI passes until treatment in the emergency department. *ECG,* electrocardiogram. (From U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Heart, Lung, and Blood Institute [NIH Publication No. 93-3278], September 1993, p 10.)

Put another way, there are also four Ds that need to be addressed when brainstorming ways to achieve AHA recommendations….and save that myocardium! Door (recognition and contact with medical providers), data (get that ECG ASAP! < 10 mins!), decision (is this a STEMI? Lysis or PCI?), drug (Get the patient to what they need!).

8) What are door-to-needle and door-to-balloon timelines by AHA recommendations?

"The AHA recommends that all patients with STEMI (patients with typical and uncomplicated presentations of AMI with ST segment elevation) receive fibrinolytic therapy within 30 minutes of arrival or undergo primary PCI (i.e., device across culprit artery) no later than 90 minutes after arrival."

- Door to needle/drug: 30 mins
- Door to balloon: 90 mins

9) Describe the mechanism of action and indications/contraindications for

Nitroglycerin

- End goal: reduce pain - titrate therapy to prn
- No current studies in the post-fibrinolysis era to support the use of NTG
- Long story:
    - "Nitrates decrease myocardial preload and, to a lesser extent, afterload. Nitrates increase venous capacitance and induce venous pooling, which decreases preload and myocardial oxygen demand. Direct vasodilation of coronary arteries may increase collateral blood flow to ischemic myocardium. Most studies of intravenous NTG in the setting of AMI are from the prefibrinolytic era. Although a meta-analysis of multiple small trials noted a 35% mortality reduction with intravenous NTG, no contemporary evidence supports the routine use of any form of nitrate therapy in patients with AMI.
    - Patients with possible ACS and a systolic blood pressure greater than 90 mm Hg should receive a sublingual NTG tablet (0.4 mg or 400 µg) on presentation. If symptoms and pain are not fully relieved with three sublingual tablets, intravenous NTG should be considered.
- Contraindications:
    - bradycardia, hypotension, inferior wall AMI, and right ventricular infarction, a sudden decrease in preload associated with NTG can result in profound hypotension.

Morphine

- End goal: pain reduction
- Bottom line: can be a reasonable option in the right patient with severe pain resistant to NTG
- Long story:
    - "Morphine is a potent opioid analgesic with weak sympathetic blockade, systemic histamine release, and anxiolysis. If a patient with possible ACS is unresponsive to NTG or has recurrent symptoms despite maximal anti-ischemic therapy, administration of morphine sulfate is reasonable."
- Contraindications:
    - Hypotension, allergy

B-Blockers

- End goal: slow HR, prevent tachydysrhythmias, decrease myocardial demand
- Bottom line: no clear role for ED use of beta-adrenergic blockers
- Long story:
    - "The early intravenous use of beta-adrenergic blocking agents, when coupled with contemporary therapy, does not appear to offer significant benefit and is associated with an increased rate of adverse events. **Oral administration to patients without contra- indication during the first day of management is an appropriate approach to the ACS patient.** Empirical therapy in the ED, however, should be reconsidered and reserved for only those patients who have adverse effects from significantly elevated blood pressure despite application of NTG, or significant tachydysrhythmia.
- Contraindications: hypotension, bradycardia, cardiogenic shock, allergy, CHF

Side notes: "Unless speci cally used for rate control of supraventricular dysrhythmia in a patient who cannot tolerate beta-blockade, calcium channel blocker agents are not recommended for ACS."

ACE-I

- End goal: reduction in cardiovascular mortality, reduced AMI recurrence, reduced CHF
- Bottom line: "Therapy should be initiated within the first 24 hours, although ED administration is usually not indicated."
- Long story: "These bene ts increase when ACE inhibitors are used in conjunction with other agents, such as aspirin and brinolytics. The mechanism of action regarding a reduction in recurrent AMI is unknown but may involve a reduction in plaque rupture related to decreased intra- coronary shear force or neurohumoral infuences."
    - Patients should continue on ACE/ARB for at least 4 months post-event
- Contraindications: allergy, hypotension, severe kidney disease, hypovolemia

HMG-CoA reductase inhibitors (Statins);

End goal: stabilize and prevent fibro-fatty plaque growth/dislodgement
Bottom line: "Administration of statin therapy before elective or urgent PCI for ACS is reasonable to decrease the incidence of periprocedure AMI; however, there are no specific risk or safety data regarding its use in this setting."
Long story: "A number of investigations have demonstrated a reduction in inflammation and reinfarction, angina, and lethal arrhythmia with the administration of statin drugs in the first few days after an ACS event. Initiation of this therapy should occur within the first 24 hours or should continue if patients are already undergoing statin therapy, as discontinuation during hospitalization is associated with an increase in near-term mortality and adverse events."
Contraindications: Allergy

Antiplatelet Therapies

- ASA 325 mg chewed
    - Bottom line: Best, most cost-effective strategy for anyone with suspected ACS (UA or AMI)
    - "It irreversibly acetylates platelet cyclooxygenase, thereby removing all activity for the life span of the platelet (8-10 days). Thus aspirin stops the production of proaggregatory thromboxane A2 and is an indirect antithrombotic agent. Aspirin also has important nonplatelet effects because it inactivates cyclooxygenase in the vascular endothelium, thereby diminishing formation of antiaggregatory prostacyclin.
    - Contraindications:
        - Significant allergy; concern for possible aortic dissection or other major hemorrhage
- Glycoprotein IIb/IIIa receptor inhibitors
    - Not to be given in patients receiving fibrinolytics
    - Usually not given in people receiving PCI because a P2Y12 inhibitor is preferred
    - If given, it is administered after angiography
- PSY12/P2Y12 receptor inhibitor agents:
    - Bottom line: Usually Clopidogrel 300 or 600 mg PO is given
    - Long story: "The thienopyridines—ticlopidine, clopidogrel, and prasugrel—are more potent platelet inhibitors than aspirin. They inhibit the transformation of the PSY12 receptor into its high-affinity ligand-binding state, irreversibly inhibiting platelet aggregation for the duration of the life of the platelet."
    - ***some cardiologists prefer ticagrelor 180 mg PO over clopidogrel for those patients receiving primary PCI***
    - Contraindications: allergy, CABG candidate (impossible for us to know in the ER)

Antiplatelet bottom line: "In accordance with the 2013 AHA Guidelines for STEMI management, patients should receive a loading dose of clopidogrel, prasugrel, or ticagrelor in addition to standard ACS care (ASA, anticoagulants, and reperfusion therapy) assuming there are no contraindications to its use, prior to PCI. For patients with moderate-to high-risk NSTEMI, the administration of a PSY12 receptor inhibitor should be deferred "downstream" to the attending cardiologist as the best revascularization strategy is determined."

Antithrombins

"Antithrombotic therapy is indicated in ACS patients with recurrent anginal pain, AMI (NSTEMI and STEMI), a positive serum marker, and a dynamic 12-lead ECG."

- Heparin
    - "At standard doses, UFH binds to antithrombin III, forming a complex that is able to inactivate factor II (thrombin) and activated factor X. This prevents the conversion of fibrinogen to fibrin, thus preventing clot formation. Heparin by itself has no anticoagulant property."

    - "UFH has a profound synergistic effect with aspirin in preventing death, AMI, and refractory angina in ACS patients, particularly those with AMI and, to a lesser extent, high-risk UA. UFH should be administered early in patients with the following ACS features: recurrent or persistent chest pain, AMI, positive serum marker, and dynamic ECG."
- LMWH (enoxaparin)
    - More potent antithrombotic effects, better bioavailability, more consistent therapeutic response than UFH
    - In summary, the LMWH enoxaparin demonstrates some degree of benefit compared with UFH in patients at higher risk for non–ST-segment elevation-ACS who are treated conservatively without immediate PCI (i.e., beyond 24 hours).150 For STEMI patients managed aggressively with rapid PCI, UHF is the preferred over enoxaparin. Enoxaparin is administered in a twice-daily regimen subcutaneously at a dose of 1 mg/kg for all ACS patients. If patients have renal dysfunction with an estimated glomerular filtration rate of less than 30 mL/min, the dose should be reduced to 1 mg/kg in a single daily administration. Few safety data are available for enoxaparin in ACS patients with renal insufficiency, and UFH may be preferable.
- Contraindications to heparins:
    - Allergy, ongoing hemorrhage, high predisposition to hemorrhage

- Direct thrombin inhibitors (fondaparinux, bivalirudin)
    - Both have their role, which is determined by local guidelines.
    - "In the large Optimal Antiplatelet Strategy for Interventions (OASIS) trial, fondaparinux was found to be similar to enoxaparin in the short-term reduction of ischemic events, yet substantially reduced major bleeding and improved long-term outcome."

Antithrombin bottom line:

- Primary PCI (or likely to have PCI) = UFH
    - Max bolus 5000 Units, then 12 U/kg/hr
- Fibrinolytics =
    - Low bleeding risk: enoxaparin
        - IV bolus followed by SC dose q12 hrs
        - Adjust dose based on CrCl and age > 75 yrs
    - High bleeding risk: fondaparinux
        - 2.5 mg IV, then 2.5 mg SC daily (not approved by the FDA)
        - NOT given in ppl with a CrCl < 30 ml/min
- No reperfusion = fondaparinux is preferred to enoxaparin or UFH

10) Describe eligibility criteria for Fibrinolytics

This is the third of the three pillars of AMI management. Antiplatelets, antithrombins, revascularization therapy. This is the only one that actually "breaks up the clot" and it has a class I recommendation!

"The 2013 American College of Cardiology Foundation/American Heart Association guideline for the management of ***STEMI recommends the use of fibrinolytic therapy in patients with symptom onset within 12 hours who cannot receive primary percutaneous coronary intervention within 120 minutes of first medical contact****. Fibrinolytic therapy has generally not improved outcomes in patients presenting at 12 hours or later and is therefore not indicated in those who are stable and asymptomatic. However, fibrinolysis can be considered up to 24 hours after symptom onset if the patient has ongoing or stuttering chest pain and PCI is not available. " - Uptodate 2017.*

- No mortality benefit in people treated 12-24 hrs from symptom onset
- "Treatment beyond that time [12 hrs from onset] is not supported by the literature. The single exception may be a patient with a "stuttering" nature of chest pain 12 to 24 hours after symptom onset, which emphasizes the importance of an adequate history."

"At the present time it appears that TNK is marginally more effective, minimally safer, and easier to administer than t-PA and thus is recommended; furthermore, cost differences are minimal and likely will not affect medical decision-making in the ED."

"Combined With The Patient's history and physical examination, the 12-lead ECG is the key determinant of eligibility for fibrinolysis.

The electrocardiographic findings include two basic issues:

(1) ST segment elevation of 1 mm or more in two or more anatomically contiguous standard limb leads or elevation of 2 mm or more in two or more contiguous precordial leads, and
(2) New or presumed new LBBB.
(3) Acute posterior MI (may appear as NSTEMI anteriorly!) aka. Inferiobasal AMI

No evidence of benefit from fibrinolytic therapy is found in patients with ischemic chest pain who lack either appropriate ST segment elevation or the development of a new LBBB.

Although fibrinolysis has widespread availability and a proven ability to improve coronary flow, limit infarct size, and improve survival in AMI patients, many individuals with acute infarction are not suitable candidates.

Patients with absolute contraindications to fibrinolytic therapy, certain relative contraindications, cardiogenic shock, and UA may not be eligible. The temporal constraints and other limitations of fibrinolytic therapy suggest that rapidly performed PCI is often the treatment of choice in the STEMI patient. To provide the most significant benefit, PCI must be performed as soon as possible after the initial presentation. In other settings and situations, PCI that is delayed is inferior to rapidly administered fibrinolysis."

11) List contraindication to Fibrinolytic therapy in ACS.

Box 78-1 Fibrinolysis in Acute Myocardial Infarction: Absolute and Relative Contraindications

- Recent (within 10 days) major surgery (e.g., coronary artery bypass graft, obstetric delivery, organ biopsy, previous puncture of noncompressible vessels)
- Cerebrovascular disease
- Recent gastrointestinal or genitourinary bleeding (within 10 days)
- Recent trauma (within 10 days)
- Hypertension: systolic BP 180 mm Hg or diastolic BP 110 mm Hg
- High likelihood of left heart thrombus (e.g., mitral stenosis with atrial fibrillation)
- Acute pericarditis
- Subacute bacterial endocarditis
- Hemostatic defects, including those secondary to severe hepatic or renal disease
- Significant liver dysfunction
- Diabetic hemorrhagic retinopathy or other hemorrhagic ophthalmic condition
- Septic thrombophlebitis or occluded AV cannula at seriously infected site
- Advanced age (older than 75 years)
- Patients currently receiving oral anticoagulants (e.g., warfarin sodium)
- Any other condition in which bleeding constitutes a significant hazard or would be particularly difficult to manage because of its location

"Trauma within 10 days of your 75th birthday means that you can't get Fibrinolytics because your INR and BP are high."

FIBRIN

Points for discussion/further reading in Rosen's:

- AGE: "age alone shouldn't be a contraindication to fibrinolytic therapy" although it comes with a higher risk of bleeding.
- HIGH BP: "a persistently elevated blood pressure higher than 200/120 mm Hg is generally considered to be an absolute contraindication to fibrinolytic therapy."
    - Try to lower it to a safe range before pushing lytics!
- HYPOTENSION: people in cardiogenic shock have a very high risk of death; ideally they go for a primary PCI if it is available on site, but cardiogenic shock and CHF are not absolute contraindications for fibrinolytics.
- RETINOPATHY IN DIABETICS: "Active diabetic hemorrhagic retinopathy is a strong relative contraindication to fibrinolytic therapy because of the potential for permanent blindness caused by intraocular bleeding. There is no reason, however, to withhold the use of a fibrinolytic agent in a diabetic patient with evidence of simple background retinopathy. Patients with diabetes mellitus who sustain an AMI have an almost doubled incidence of mortality."

- ONGOING CPR: “CPR is not a contraindication to fibrinolytic therapy unless CPR is prolonged—more than 10 minutes—or extensive chest trauma from manual compression is evident.”
- HX OF STROKE: a hemorrhagic stroke hx is an ABSOLUTE contraindication to fibrinolytics, ischemic strokes are a relative CI.
- PREVIOUS CABG:
    - PCI is preferred
- “RECENT” SURGERY
    - Recent has different definitions (10 days to 6 weeks).
    - Recent surgery in the last 10 days is a relative-absolute CI to lysis...

12) What is the utility of Rescue PCI and Facilitated PCI?

- R-PCI:
    - For patients who despite fibrinolytics fail to revascularize (either ongoing chest pain OR persistent ST elevation) they should undergo PCI.
    - No mortality benefit shown, but there is a lower incidence of CHF and recurrent infarction.
    - NO role for repeat fibrinolysis
- F-PCI
    - “Facilitated percutaneous coronary intervention refers to combination therapy involving fibrinolysis coupled with emergent PCI. This concept originally was developed to maximize therapy in STEMI patients who would be transferred urgently for PCI; the patient would receive the additive benefit of medical therapy (a fibrinolytic agent) before transfer, optimizing the culprit artery for the benefit of mechanical therapy before arrival at the PCI-capable institution.”
    - Less beneficial than fibrinolysis or PCI alone….ongoing scientific investigation is needed!

13) List 5 indications for Rescue PCI

1. Persistent chest pain
2. Persistent ST elevation
3. Cardiogenic shock
4. Post-reperfusion ischemia
5. Ventricular dysrhythmias

14) Describe factors assisting with decision to utilize PCI or thrombolytics

It all comes down to time and resources….

How long should the ERP wait for PCI in a patient who is fibrinolytic eligible?

- Considerations:
    - Time to therapy question
    - Time from onset of symptoms

        - Infarct duration
    - Patient age
    - Infarct location
        - E.g. cardiogenic shock

"Thus, emergency revascularization with PCI or CABG is preferred in patients with STEMI complicated by cardiogenic shock irrespective of the delay to treatment (i.e., more than 120 minutes first medical contact to PCI time usually measured for transferring these patients)."

"In fact, the ACC/AHA guidelines note that in hospitals without PCI capability, immediate transfer for primary PCI is a treatment option when it can be accomplished within 120 minutes of first medical contact.140 If delays in PCI performance are anticipated and the patient is an acceptable candidate for fibrinolysis, the fibrinolytic should be started before or during transport to the receiving hospital."

Patient presentations with the "maximal allowable" time to catheter placement across the lesion are as follows:

- Within 2 hours of symptom onset—94 min
- Beyond 2 hours of symptom onset—190 min
- Younger than 65 years—71 min
- Older than 65 years—155 min
- Anterior STEMI—115 min
- Nonanterior STEMI—112 min

Further analysis combined commonly encountered clinical variables in typical STEMI presentations:

- Patient presentation within 2 hours of symptom onset and:
    - Anterior STEMI with age younger than 65 years—40 min
    - Anterior STEMI with age older than 65 years—107 min
    - Nonanterior STEMI with age younger than 65 years—58 min
    - Nonanterior STEMI with age older than 65 years—168 min
- Patient presentation beyond 2 hours of symptom onset and:
    - Anterior STEMI with age younger than 65 years—43 min
    - Anterior STEMI with age older than 65 years—148 min
    - Nonanterior STEMI with age younger than 65 years—103 min
    - Nonanterior STEMI with age older than 65 years—179 min

"PCI initiated within 3 hours of initial hospital arrival is also superior to fibrinolysis."
….. **just call your local interventional cardiologist…..**

15) In NSTEMI, who will benefit from an early invasive strategy of management?

Immediate angiography and revascularization — Patients who have a non-ST elevation ACS and one or more of the following characteristics are at extremely high risk of an adverse cardiovascular event in the short term:

- *Hemodynamic instability or cardiogenic shock*
- *Severe left ventricular dysfunction or heart failure*
- *Recurrent or persistent rest angina despite intensive medical therapy*
- *New or worsening mitral regurgitation or new ventricular septal defect*
- *Sustained ventricular arrhythmias*

We recommend that patients with any of these five characteristics be referred for immediate coronary arteriography and revascularization.

-- Uptodate 2017

Early means within 24 hrs.

Wisecracks

1) Describe the HEART score

We're discussing this because the previous capstone score (the TIMI score) has limited utility in helping us determine which of our low risk patients do/don't need further workups.

The HEART score has been validated in the Netherlands and the USA.

From the original authors (big shout out to the Netherlands!)

The HEART Score

By assigning zero, one, or two points — towards an atypical patient **history**, **ECG anomalies**, the patient's **age**, any **risk factors** present, and elevated **Troponin** — patients score on a scale of 0–10.

Discriminative Power

This graph shows that the HEART score is a perfect predictor of MACE within six weeks after presentation at the emergency room. The X axis represents the increasing HEART score of a patient, while the Y axis indicates the increasing risk of a cardiac event. The S shaped curve represents a discrimination between low risk and high risk patients. Low HEART scores accompany a low likelihood of MACE; high HEART scores predict high incidence of MACE.

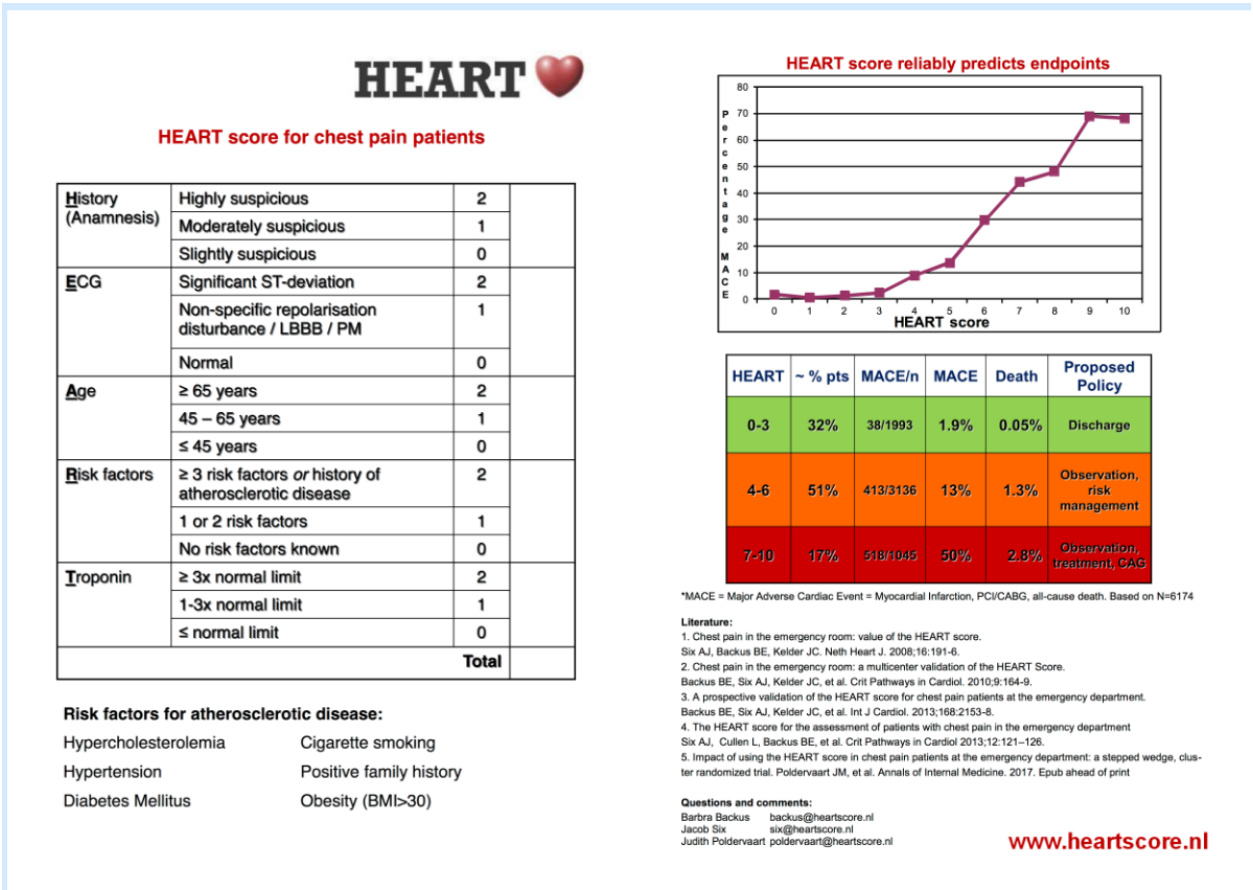
Proposed Policy

Patients can be divided into three distinct groups:

A score of 0-3 indicates a risk of 1.6% for reaching a MACE, and therefore supports a policy of early discharge.

A HEART score of 4-6 points, with a risk of MACE of 13%, immediate discharge is not an option. These patients should be admitted for clinical observation and subjected to non-invasive investigations such as repeated troponin or advanced ischemia detection.

A HEART score ≥ 7 points, with a risk of 50% for a MACE, calls for early aggressive treatments possibly including invasive strategies without preceding non-invasive testing.



HEART

HEART score for chest pain patients

History (Anamnesis)	Highly suspicious	2	
	Moderately suspicious	1	
	Slightly suspicious	0	
ECG	Significant ST-deviation	2	
	Non-specific repolarisation disturbance / LBBB / PM	1	
	Normal	0	
Age	≥ 65 years	2	
	45 – 65 years	1	
	≤ 45 years	0	
Risk factors	≥ 3 risk factors *or* history of atherosclerotic disease	2	
	1 or 2 risk factors	1	
	No risk factors known	0	
Troponin	≥ 3x normal limit	2	
	1-3x normal limit	1	
	≤ normal limit	0	
		Total	

Risk factors for atherosclerotic disease:

Hypercholesterolemia	Cigarette smoking
Hypertension	Positive family history
Diabetes Mellitus	Obesity (BMI>30)

HEART score reliably predicts endpoints

HEART	~ % pts	MACE/n	MACE	Death	Proposed Policy
0-3	32%	38/1993	1.9%	0.05%	Discharge
4-6	51%	413/3136	13%	1.3%	Observation, risk management
7-10	17%	518/1045	50%	2.8%	Observation, treatment, CAG

*MACE = Major Adverse Cardiac Event = Myocardial Infarction, PCI/CABG, all-cause death. Based on N=6174

Literature:

1. Chest pain in the emergency room: value of the HEART score.
Six AJ, Backus BE, Kelder JC. Neth Heart J. 2008;16:191-6.
2. Chest pain in the emergency room: a multicenter validation of the HEART Score.
Backus BE, Six AJ, Kelder JC, et al. Crit Pathways in Cardiol. 2010;9:164-9.
3. A prospective validation of the HEART score for chest pain patients at the emergency department.
Backus BE, Six AJ, Kelder JC, et al. Int J Cardiol. 2013;168:2153-8.
4. The HEART score for the assessment of patients with chest pain in the emergency department
Six AJ, Cullen L, Backus BE, et al. Crit Pathways in Cardiol 2013;12:121–126.
5. Impact of using the HEART score in chest pain patients at the emergency department: a stepped wedge, cluster randomized trial. Poldervaart JM, et al. Annals of Internal Medicine. 2017. Epub ahead of print

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http://rebelem.com/heart-score-new-ed-chest-pain-risk-stratification-score/

2) What is the management of ACS in the setting of recent cocaine use?

According to UptoDate:

- Anyone with cocaine-related ACS is managed in a similar way
    - Except that beta-blockers are NOT given in the early phases of treatment (due to theoretical concerns of unopposed alpha-adrenergic stimulation)
- These are the drugs we should be using:
    - ASA 325 mg
    - NTG and/or CCB (for chest pain symptoms)
    - Benzodiazepines
        - For sedation, HR and BP control
    - Early reperfusion, preferably with PCI rather than fibrinolysis (when PCI is available)

3) How is STEMI diagnosed in the setting of LBBB?

See our discussion above in Question 1. We can use Sgarbossa's criteria.

4) How is STEMI diagnosed in the setting of a ventricular pacemaker?

A modified version of Sgarbossa's criteria can be used.

"Limited data exist to guide the clinician in interpretation of the 12-lead ECG in this setting. As with the LBBB scenario, the VPR pattern represents a significant confounding variable in the evaluation of the patient with chest pain suspected of having ACS. **Sgarbossa and associates advanced criteria for detection of AMI in the presence of VPR that are similar to those for LBBB.** These, too, are derived from the GUSTO-I database, but from a smaller group of patients.

The criteria are essentially the same as the LBBB criteria: (1) ST segment elevation of at least 5 mm that is discordant with the QRS complex; (2) ST segment elevation of at least 1 mm that is concordant with the QRS complex; and
(3) ST segment depression of at least 1 mm in lead V1, V2, or V3. (Copied from Rosen's 7th Ed.)"

5) When to get a 15 lead ECG?

- When should be we get a 15 lead ecg?
    - (1) ST segment changes (depression or elevation) in leads V1 to V3, either in an isolated lead or in more than one;
    - (2) Equivocal ST segment elevation in the inferior (II, III, aVF) or lateral (I, aVL) limb leads or both;
    - (3) All inferior STEMI; and
    - (4) Hypotension in the setting of ACS