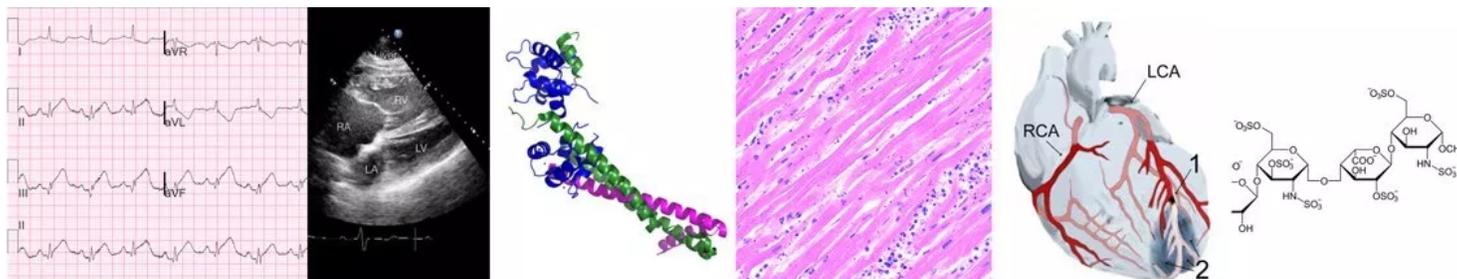


The Internet Book of Critical Care

Troponin elevation in non-cardiac critical illness

November 2, 2016 by [Josh Farkas](#)



(<https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/mieval2.jpg>)

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introduction

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This chapter is about how to approach ischemia evaluation for the “non-cardiac” critically ill patient (someone admitted for a problem such as sepsis, stroke, or DKA). This is tough. We want to be thorough enough not to miss significant infarctions. However, excessive testing risks over-diagnosis of ischemia, leading to a host of iatrogenic harms.

baseline cardiac investigation in the non-cardiac patient

- Investigating a patient's cardiac function can be useful for many reasons:

- This will occasionally show an unexpected *acute* disease processes (e.g. tamponade, massive pulmonary embolism).
- More often this will reveal a *chronic* disease that may affect how the patient responds to acute illness (e.g. severe aortic stenosis, chronic pulmonary hypertension, cardiomyopathy).
- Baseline EKG is advisable in almost all critically ill patients.
 - This will help interpret any changes in rhythm or morphology which may occur during the patient's ICU course.
- Baseline point-of-care echocardiography may be helpful as well.
 - If significant abnormalities are found, a formal echocardiogram may be ordered to confirm, quantify, and document abnormalities.

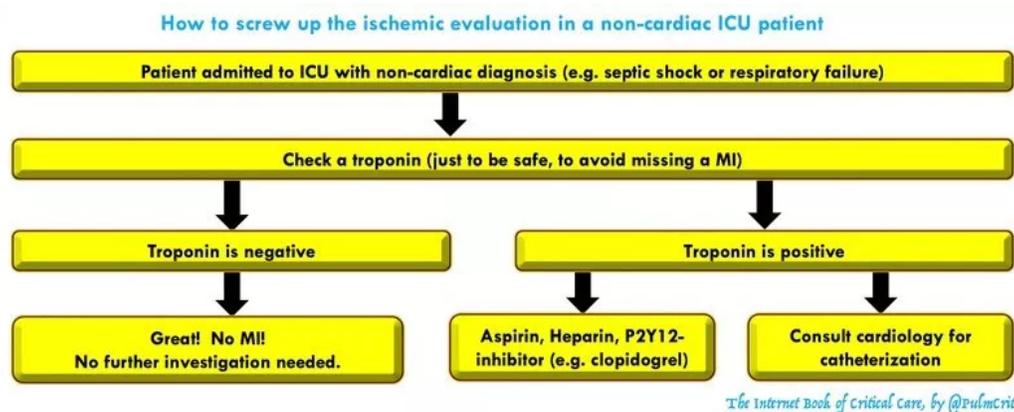
risks of over-diagnosing myocardial ischemia

- Incorrectly diagnosing a patient with MI can cause a variety of problems.
- (1) Bleeding risk from anticoagulation
 - Heparin increases the risk of hemorrhage. Most of these are non-lethal GI bleeds, but occasionally heparin will cause intracranial hemorrhage. The risk for any individual patient is low, but over time indiscriminate use of heparin will eventually cause severe harm.
- (2) Cardiac catheterization & stent risk
 - Risks of catheterization include hemorrhage and kidney injury. Renal toxicity is particularly problematic, as critically ill patients frequently have some degree of kidney injury already.¹
 - Stent placement obligates the patient to remain on dual anti-platelet therapy. This may be problematic if the patient subsequently requires surgery or develops bleeding (frequent occurrences in a complex ICU patient).
- (3) Interference with other essential procedures
 - Once the patient is labeled as having a myocardial infarction, this will often scare off anyone from doing procedures on the patient (especially anesthesiologists and surgeons).
 - This can derail the patient's entire plan of care. Rather than fixing the patient's actual problem, the focus of care may shift towards treatment of a "myocardial infarction" that doesn't really exist.

how to screw this up

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I never received formal instruction on this topic, so I screwed it up for years. Seriously, *mea culpa*. It's easy for smart and well-intentioned folks to do this wrong and cause harm.



<https://i0.wp.com/emcrit.org/wp-content/uploads/2016/11/screwy.jpg>

Superficially this strategy might seem OK, but there are a *ton* of problems here. Many of these problems will be explored further below, but one is worth mentioning here.

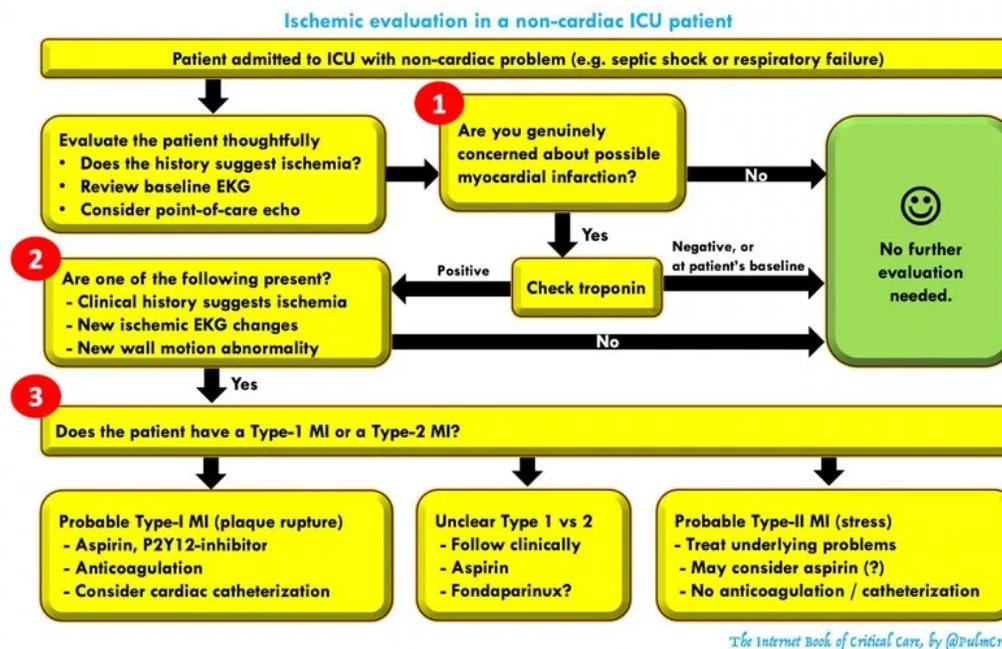
checking troponin without checking an EKG

- EKG is the first-line test for myocardial infarction. An EKG tells you in *real time* how much tissue is ischemic. For example, a patient with acute myocardial occlusion may have dramatic EKG changes with a negative troponin (troponin takes a couple hours to rise).

- Cycling troponin without checking EKGs is just plain wrong. If the patient does have a massive MI, this strategy won't diagnose it until substantial myocardial damage has already been done.

approach to ischemia evaluation

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<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/MIalgo.jpg>. The approach to ischemia boils down to three decision points. Let's walk through them.

step 1- should we check troponin?

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troponin is often positive among critically ill patients

- Using standard troponin assays, ~40% of patients in an ICU will have an elevated troponin.² Most of these troponin elevations don't represent MI – this simply reflects global stress on the heart due to critical illness. Numerous disorders are capable of causing troponin elevation in critical illness (e.g. sepsis, chronic kidney disease, stroke, PE, heart failure, defibrillation, takotsubo cardiomyopathy).³
- About half of healthy people who run a marathon will also develop an elevated troponin.⁴ Neither marathon runners, nor most ICU patients with elevated troponin, require specific treatment for this lab abnormality.
- Elevated troponin *does* correlate with increased mortality. Thus, for most critically ill patients troponin functions as a *death marker* (not an *MI marker*).

broad application of troponin destroys its use as a clinical test

- Among sick, non-cardiac ICU patients, troponin has the following performance:
 - Sensitivity for MI is extremely high (nearly 100%)
 - Specificity might be ~60% for MI.²
- This creates the following positive and negative likelihood ratios:
 - Positive likelihood ratio = 2.5
 - Negative likelihood ratio = 0
- Therefore, if we apply this test to a patient with low pre-test probability for MI (let's say, 5% pre-test probability), then a positive test only increases the post-test probability marginally (from 5% to 12%). Even with a *positive* troponin, the patient is *unlikely* to have a myocardial infarction.
 - Broad application of troponin testing to a low-risk population will yield almost entirely *false-positive results*.

- This isn't the test's fault, it's our fault. Inappropriate application of *any* clinical test to patients with low pre-test probability will yield more false-positive than true-positive results.

every ICU patient needs an EKG – not a troponin

- There is myth that every ICU patient needs a troponin level “just to be safe.” This is wrong. Troponin should only be obtained if there is genuine concern for myocardial ischemia based on history and EKG.
- EKG is a good initial test for ischemia because minor, nonspecific abnormalities won't trigger a huge ischemia evaluation.
 - *Physician judgement is built into the EKG test itself.* So you can order tons of EKGs without causing harm, whereas if you start shotgun ordering troponin this leads to iatrogenic chaos.
- Point-of-care ultrasonography is an underutilized tool, which can help guide thoughtful evaluation for myocardial ischemia.
 - If echo shows a hyperkinetic, underfilled ventricle then the heart is working fine. Focus on treating the underlying problem (e.g. sepsis, hemorrhage).
 - If echo shows hypokinesis or wall-motion abnormalities, that's more concerning for a primary cardiac problem. Clinical judgement and additional tests may be needed to sort out whether these abnormalities are chronic or acute.

step 2- does this trop-positive patient have a MI?

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diagnosis of an MI requires the following:

1. A dynamic rise and fall in troponin (>20% variation), *plus*
2. At least one of the following:⁵
 1. Clinical history suggestive of MI
 2. New ischemic EKG changes
 3. New wall motion abnormality on echocardiography

positive troponin usually doesn't indicate an MI (myocardial infarction)

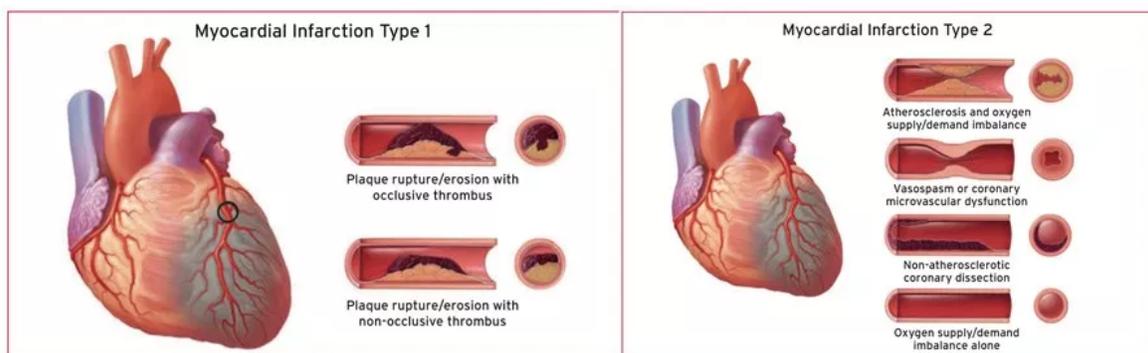
- Most troponin elevations in the ICU *don't* actually represent an MI.
- Beware of patients with renal failure and chronically elevated troponin – if the troponin isn't rising/falling then it's not an MI. Review the patient's prior troponin values, if possible.
- Patients with elevated troponin who don't meet the definition of a MI (myocardial ischemia) are described as having *myocardial injury*.³

don't treat non-MI troponin elevations (myocardial injury)

- Isolated troponin elevation shouldn't be treated as an MI (e.g. with anticoagulation).
- Treat the underlying disease (e.g. sepsis, hypoxemia, anemia).

step 3- type-1 versus type-2 MI?

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The key difference between Type I vs. Type II MI is the presence/absence of plaque rupture (From 4th Universal Definition of MI, 2018, PMID 30165617)

<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/type1type2.jpg> what is a type-1 or type-2 MI?

- **Type-I MI:** *Primary plaque rupture* causing a myocardial infarction (for example, a patient who presents to the ED with chest pain and acute occlusion of a coronary artery).
 - As discussed below, these patients may benefit from traditional MI therapy.
- **Type-II MI (demand ischemia):** Myocardial infarction *not involving* unstable coronary plaque. This is usually due to *stable coronary stenoses* in the context of physiologic stress (e.g. anemia, hypoxemia, inotropes, tachycardia). However, Type-II MI can also occur in the setting of normal coronaries due to severe stress (e.g. sustained tachyarrhythmia).
 - Type-II MI can generally be conceptualized as a patient with a *positive stress test*. The patient usually *does* have coronary disease, and myocardial ischemia *did* occur. However, the patient *doesn't* have unstable coronary plaque. Once the acute stressor is removed, the patient isn't in immediate danger from coronary ischemia.
 - These patients *don't* require traditional MI therapy (e.g. catheterization, anticoagulation).

differentiating type-I vs. type-II MI

- Sorting this out is difficult, often relying heavily on clinical judgement.
- A key consideration is comparing the severity of physiologic stress vs. the severity of myocardial ischemia. For example, if you give someone 500 mcg of subcutaneous epinephrine and they develop transient chest pain and a troponin of 0.7 ng/ml, that's probably a type-2 MI. If the same symptoms developed in the absence of any stressor (chest pain and troponin elevation out of the blue), then that's probably a type-1 MI.
- The table below includes some factors which can help guide this differentiation. Unfortunately, none of these factors in isolation are 100% reliable.
- In unclear situations, serial EKG can be helpful. For example, a patient might present with chest pain, EKG changes, and a GI hemorrhage. If treating the GI bleed causes the EKG changes and chest pain to disappear, that suggests a type-2 MI. Alternatively, if you fix the GI bleed and the patient continues having chest pain and ischemic EKG changes, then that's more worrisome for type-1 MI.

sorting out type-1 versus type-2 MI

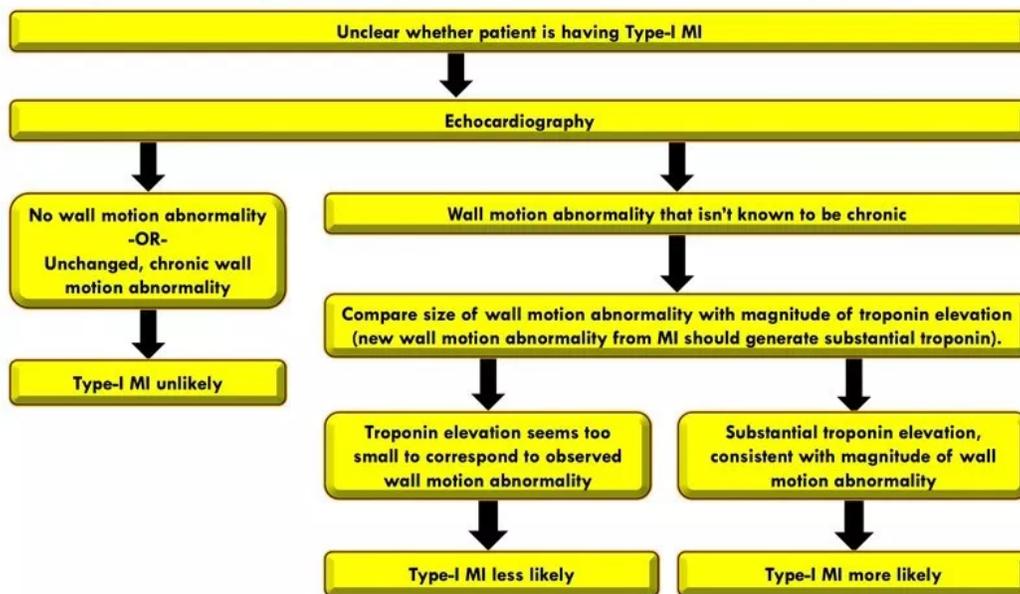
	Type-1 MI (Acute plaque rupture)	Type-2 MI (demand ischemia)
Clinical context	- Initial presentation suggestive of myocardial ischemia	- Patient initially presents with non-cardiac problem (e.g. sepsis, hemorrhage, trauma)
Amount of clinical stress	- Low	- High (e.g. high-dose vasopressors, anemia, hypoxemia, hypotension, hypoperfusion)
Echo findings	- New wall-motion abnormality or reduced ejection fraction	- Echo shows hyperkinetic, under-filled heart
EKG findings	- Strongly diagnostic of ischemia (e.g. occlusive MI)	- Nonspecific or minimal EKG changes - EKG changes resolve with resolution of stress
Extent of troponin elevation	- Larger troponin elevations are more worrisome for Type-1 MI (e.g. Troponin I > 10 ng/ml)	- Troponin elevation usually moderate (however, a severe fixed lesion such as left-main stenosis can produce marked troponin elevation due to stress)

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(<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/sortmi.jpg>) **echocardiographic strategy to sorting type-I vs. type-II MI**

- Every single patient with a troponin elevation doesn't require an echocardiogram. However, echocardiography can be helpful if there is difficulty differentiating between type-1 and type-2 MI.
- Available evidence suggests that in ICU patients without echocardiographic features of ischemia, catheterization is unlikely to reveal a treatable coronary lesion (Type-I MI)².
- Echocardiography may be particularly useful in situations of global myocardial ischemia (e.g. due to anemia, hypoxemia, and inotropes). Diffuse ischemia may cause a very scary EKG pattern (e.g. diffuse STD with STE in aVR) and substantial elevation in troponin. However, echocardiography will reveal a hyperkinetic ventricle without wall motion abnormalities, arguing against Type-I MI.
- The main limitation of echocardiography is that in the context of an aging population, lots of patients have *chronic* wall motion abnormalities. In the absence of a prior echocardiogram, such abnormalities could masquerade as an acute MI. One clue that the wall motion abnormality may be chronic is mismatch between a *significant* wall motion abnormality versus a *minor* troponin elevation.
 - The key is always integration of multiple features (e.g., echocardiography, EKG, history, and troponin).
- The following is an example of how echocardiography could be used:

Echocardiography strategy for determining if patient might be having a Type-1 MI



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<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/echostrat.jpg>

treatment of type-1 MI

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This involves the traditional management of MI that we all learned on the cardiology service (e.g. aspirin, P2Y12-inhibitor, anticoagulation, beta-blockade, potentially cardiac catheterization). There will eventually be an entire chapter on this eventually ([here](#)).

treatment of type-2 MI

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this is an evidence-free zone

- To my knowledge no RCTs exist on the treatment of ICU patients with demand ischemia.
- If you know of such a study please let me know and I'll add it.

reasonable management strategy?

- The most important treatment is management of the underlying process.
- Aspirin is reasonable in the absence of contraindications. This is supported by one observational study.⁶
- Transfusion to a slightly higher hemoglobin target than usual (>8 mg/dL).
- Hemodynamic optimization?
 - If the patient is on vasopressors, consider trying to reduce the amount of beta-agonist stimulation.
 - If the patient is hypertensive and otherwise hemodynamically stable, consider treating hypertension with a beta-blocker.

treatment when type-1 vs. type-2 is unclear

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As discussed above, it's not always possible to immediately classify a patient into type-1 vs. type-2 MI. In these situations, the following management strategy may be reasonable (noting that this is another evidence-free zone). These patients will be followed carefully over time (e.g. with serial troponin and EKGs) which may help ultimately clarify whether they have type-1 versus type-2 MI.

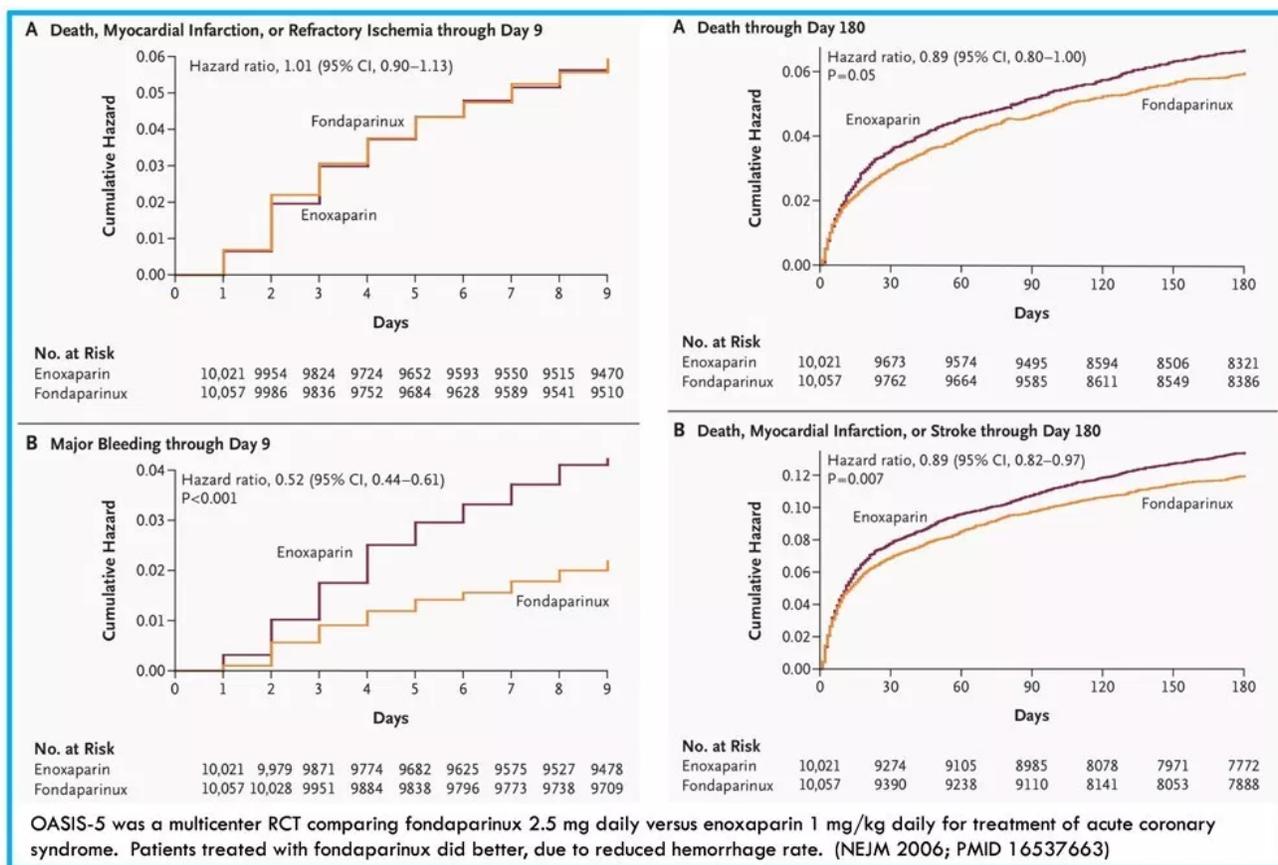
basic initial management

- Aspirin if not contraindicated

- Treatment of anemia, with a target hemoglobin >8 mg/dL.
- Reduce stress on the heart, as able:
 - If on pressors, try to reduce beta-agonist stimulation.
 - If hypertensive, consider addition of a beta-blocker.

consider fondaparinux 2.5 mg daily

- Administration of 2.5 mg daily [fondaparinux](https://reference.medscape.com/drug/arixtra-fondaparinux-342172) (https://reference.medscape.com/drug/arixtra-fondaparinux-342172) is a reasonable anticoagulation strategy here. This is a completely legitimate treatment for Type-1 MI, in fact it is suggested as front-line therapy in the European Society of Cardiology guidelines.^{7 8}
 - Fondaparinux has been shown to improve mortality compared to low molecular-weight heparin (OASIS 5 trial,⁹ see below). This finding was replicated in the OASIS-6 trial¹⁰ and also a real-world registry.¹¹
 - Fondaparinux should probably be used more for the treatment of MI among patients who aren't going for immediate catheterization.¹² Unfortunately, heparin infusions remain over-utilized in the United States due to habit and convenience.
- 2.5 mg fondaparinux is the dose studied for use in myocardial infarction. It is also the dose used for DVT *prophylaxis* (compared to 7.5 mg daily, which is the full therapeutic dose for DVT/PE).
 - Thus, 2.5 mg fondaparinux can be used to serve a *dual purpose* of anticoagulation for MI as well as DVT prophylaxis.
 - The fact that 2.5 mg fondaparinux is the *DVT prophylactic* dose should indirectly show that it has a low bleeding risk (considerably lower than a heparin infusion).
- Fondaparinux is contraindicated in patients with renal dysfunction (e.g. GFR <30 ml/min).



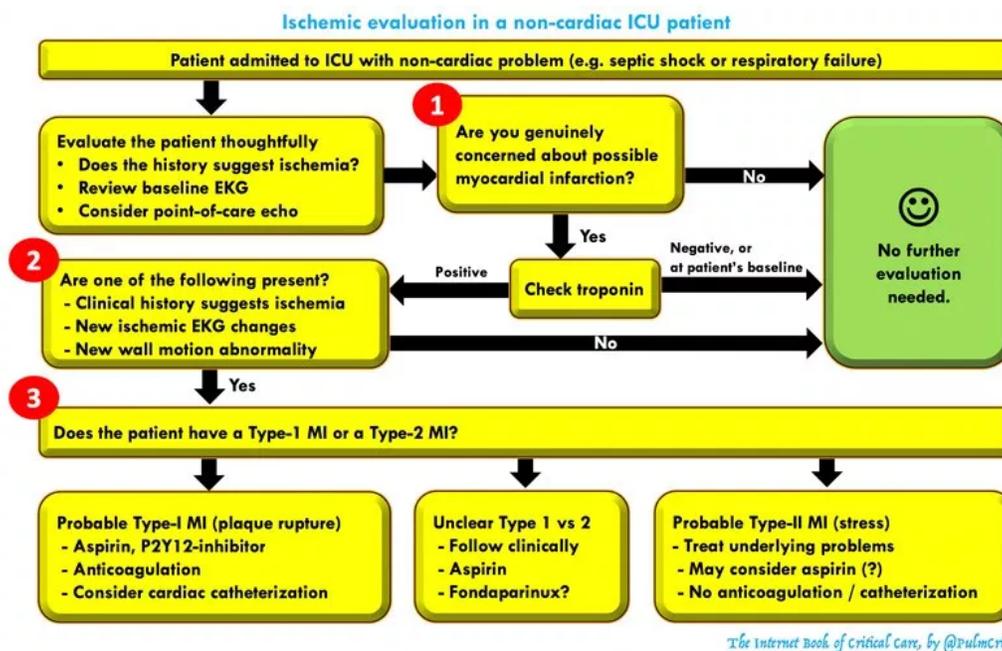
(<https://io.wp.com/emcrit.org/wp-content/uploads/2016/11/oasis5.jpg>) **avoid heparin infusions**

- First, let's consider some basic [principles of heparin use in MI](https://emcrit.org/pulmcrit/mythbusting-heparin-isnt-beneficial-for-noninvasive-management-of-nstemi/) (https://emcrit.org/pulmcrit/mythbusting-heparin-isnt-beneficial-for-noninvasive-management-of-nstemi/):
 - Heparin causes a *transient* reduction in the rate of ischemic events while the patient is anticoagulated. Unfortunately, after heparin is stopped, there is a *rebound* in ischemic events. Ultimately, the risk of an ischemic event is the same, with or without heparin therapy.
 - There is no *sustained net benefit* from providing a patient with 48 hours of heparin as “medical management” for MI.¹³ This exposes patients to the risks of anticoagulation, without causing any sustained reduction in the rate of re-infarction or death.

- Heparin should be used only as a *temporary bridge* to stabilize a patient while they are awaiting cardiac catheterization.
- There may be a temptation to start a heparin infusion here. However, heparin is generally the wrong move for the following reasons:
 1. If the patient has adequate renal function, fondaparinux 2.5 mg daily is safer (see above).
 2. Heparin is only useful as a temporary bridge to cardiac catheterization. The vast majority of patients in this group won't go for urgent catheterization, so they won't benefit from this therapy.

algorithm

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<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/M1algo.jpg>

podcast

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questions & discussion

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To keep this page small and fast, questions & discussion about this post can be found on another page [here](https://emcrit.org/pulmcrit/troponin/) (<https://emcrit.org/pulmcrit/troponin/>).



<https://i.wp.com/emcrit.org/wp-content/uploads/2016/11/pitfalls2.gif>

- Don't order a troponin level unless you truly suspect that the patient is having a myocardial infarction.
- Don't order a troponin level without getting an EKG as well.
- Consider *echocardiography* as a diagnostic tool to evaluate for plaque-rupture MI (type-I MI).
- Avoid heparin infusions, with consideration of 2.5 mg fondaparinux instead.
- Don't be surprised when your critically ill patient is found to have an elevated troponin level – this is extremely common among ICU patients.

Going further

- There is precious little published about MI in the non-cardiac ICU patient. The best evidence review seems to be Carroll 2016.²
- Blogs
 - [Heparin is non beneficial for noninvasive treatment of MI](https://emcrit.org/pulmcrit/mythbusting-heparin-isnt-beneficial-for-noninvasive-management-of-nstemi/) (<https://emcrit.org/pulmcrit/mythbusting-heparin-isnt-beneficial-for-noninvasive-management-of-nstemi/>) (PulmCrit)
 - [Non-MI troponin elevation](http://www.emdocs.net/the-elevated-troponin-what-else-besides-acs-could-cause-troponin-elevation/) (<http://www.emdocs.net/the-elevated-troponin-what-else-besides-acs-could-cause-troponin-elevation/>) (EMDocs by Christina Smith et al.)

Image credits: [troponin](https://en.wikipedia.org/wiki/Troponin) (<https://en.wikipedia.org/wiki/Troponin>)

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5. Technically a fourth option here is identification of a coronary thrombus on cardiac catheterization. However in clinical practice catheterization is rarely used as a primary tool to diagnose myocardial infarction.
6. Kortbaoui R, Allyn J, Guglielminotti J, et al. Pattern and consequences of cardiologic medications management for patients with elevated troponin I upon admission into an intensive care unit not linked to type 1 acute myocardial infarction: A prospective observational cohort study. *Ann Pharm Fr.* 2017;75(4):285-293. [[PubMed](https://www.ncbi.nlm.nih.gov/pubmed/28454759) (<https://www.ncbi.nlm.nih.gov/pubmed/28454759>)]
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10. Yusuf S, Mehta S, Chrolavicius S, et al. Effects of fondaparinux on mortality and reinfarction in patients with acute ST-segment elevation myocardial infarction: the OASIS-6 randomized trial. *JAMA.* 2006;295(13):1519-1530. [[PubMed](https://www.ncbi.nlm.nih.gov/pubmed/16537725) (<https://www.ncbi.nlm.nih.gov/pubmed/16537725>)]
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12. Fondaparinux is associated with an increase in catheter thrombosis at time of cardiac catheterization. This can be managed by bolusing patients with heparin immediately prior to cardiac catheterization. Fondaparinux isn't the ideal drug for patients going straight to the cath lab, but that's not the patient population we're dealing with here.
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