Context: Biomarkers are indiscriminately ordered and repeated both in the ER and floors; 79% of elevated troponin not from ACS[1]

Current: Gap between actual and optimal care, using high-value care: significant increase in cost savings and test-free patient days, no worsening in patient safety endpoints[2]

Cutting Edge: Implementing a computerized clinical decision support tool or algorithm helps reduce unnecessary testing.

Biomarker	Indications	Level of evidence (AHA/ACC)
BNP or NT-proBNP	Diagnosis of HF	Class I, Level A
	Prognosis in acute decompensated HF	Class I, Level A
	Chronic HF risk stratification	Class IIb, Level B
Troponin	Suspected myocardial injury/ACS, prognosis in acute decompensated HF	Class I, Level A
	MINS* (high risk pts)	AHA: "Recommended" in 2021 Scientific Statement CCS: "Moderate-quality evidence" ESA: Class 1c (strong rec, low quality evidence)

Table 1. Diagnostic Criteria for MINS

Elevated postoperative cTn with ≥1 cTn measurement above the 99th percentile of the URL for the cTn assay, with a rise/fall pattern indicative of acute myocardial injury*†
Occurs in the first 30 d (and typically within 72 h) after surgery
Myocardial injury is attributable to a presumed ischemic mechanism (ie, supply-demand mismatch or atherothrombosis) in the absence of an overt precipitating nonischemic cause (eg, pulmonary embolism)
Clinical symptoms may be masked by sedation or analgesia in the perioperative setting, so an ischemic feature (eg, ischemic symptoms, electrocardiographic changes) is not required

MINS: Myocardial Injury after Non-Cardiac Surgery

Context: Due to cardiac and intraoperative risk factors, the incidence of MINS can be as high as 20%, most happened < 48h postop, 65% asymptomatic, increased 30d mortality and 1-year mortality

Current: Variability between what guidelines recommend: serial troponin for "high risk" patients (age, male, elevated RCRI≥ 1, cardiac hx, elevated BNP).[2]

Cutting Edge: Guidance is still evolving if the diagnosis of MINS considers testing based on the mechanism of injury.

Plaque rupture: serial troponin, echo/stress testing, invasive testing/cardiology.

Supply-demand mismatch: low quality evidence but consider pt goals. Still unclear what to do, consider statin, ASA, or noninvasive cardiac testing. More studies are needed, and there is no guidance on follow-up/timing and no evidence that interventions improve outcomes.

What is ACS? A spectrum of disease

Context: The primary difference between NSTEMI is partial occlusion; STEMI causes total occlusion.
NSTEMI vs UA: same process, same EKG changes, but NSTEMI is biomarker positive and more common than STEMI. Clinical history, EKG, with biomarkers are critical.

Type 1, Type 2 MI, & Non-Ischemic Myocardial Injury

Type 1 MI
- **Due to ACS (plaque rupture or erosion)**
- Distinction from T2MI generally a clinical dx
- hsTn elevations >5x URL → >90% PPV for T1MI
- Coronary thrombus on angiography confirms dx

Type 2 MI
- **NOT due to ACS**
- ↓ myocardial perfusion
  - Coronary artery spasm, embolism, dissection
  - HoTN, sustained bradycardia, anemia
- ↑ myocardial demand
  - Sustained tachycardia, hypertension

Acute myocardial injury
- **↑ Tn w/o s/s ischemia**
- HF, myocarditis, TTC
- Ablation, defib, contusion
- PE, PH
- Sepsis, CKD
- Stroke, SAH
- Strenuous exercise

Current: High sensitivity troponin has greater precision at lower levels, more false negative diagnoses in younger women and false positive dx in older men.[4]

Cutting Edge: For anti-ischemic therapy, use oral beta blockers to avoid acute HF/shock as the long-term benefit may be just in those with HFrEF; nitrates have no mortality benefit, CCB is for ischemia, and O2 is only for hypoxemia. 1) Troponin elevation ≠myocardial infarction, 2) Recognize the pattern of biomarker elevation, 3) Clinical history and ECG are critical to MI dx

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