

**Table 1. Common cause of chest pain<sup>a</sup>**

<b>Cardiac</b>	<b>Pulmonary</b>	<b>Others</b>
<i>Ischemic syndromes</i>	Bronchitis	<i>Vascular</i>
Stable angina	Bronchospasm	Aortic dissection
Unstable angina	Empyema	Pulmonary embolism
Variant angina	Pleural effusion	Pulmonary hypertension
AMI	Pleuritis	<i>Gastrointestinal</i>
<i>Valvular disease</i>	Pneumonia	Esophageal spasm
Mitral valve prolapse	Pneumothorax	Gastroesophageal reflux
Aortic stenosis	Pulmonary edema	Mallory-Weiss tear
Subaortic stenosis	Aortic dissection	Esophagitis/gastritis
<i>Cardiomyopathy</i>	Pulmonary embolism	Gastric/duodenal ulcer
<i>Pericarditis</i>	Pulmonary hypertension	Biliary colic
		<i>Musculoskeletal</i>
		Costochondritis
		Muscle strain/spasm
		Cervical radiculopathy
		<i>Neurologic: Herpes Zoster</i>

<sup>a</sup>Taken from Green GB, Green SF. Markers of myocardial injury in the evaluation of the emergency department patient with chest pain. In: Wu et al. ed., Cardiac Markers, Totowa NJ: Humana Press, 1998, p. 77.

**Table 2. Rate of inappropriate discharge from the ED for patients with AMI**

<b>Study</b>	<b>Year</b>	<b>Percentage</b>
Pozen et al. (5)	1984	7%
Tierney et al. (6)	1986	13%
Lee et al. (7)	1987	4%
Rouan et al. (8)	1987	10%
McCarthy et al. (9)	1993	2%
Puleo et al. (10)	1994	5%
Graff et al. (11)	1997	4.5%

**Table 3. Summary of studies on biochemical markers for determination of reperfusion success following intravenous thrombolytic therapy**

No. Patients <sup>a</sup>	Marker	Sen/Spec	Aniography Time interval <sup>b</sup>	Reference
7/35	myoglobin	85/100	2 h	78
17/46	CK-MB	85/71	1 1/2 h	79
	myoglobin	94/88		
	cTnT	80/65		
8/17	CK-MB	65/100	1 1/2 h	80
	myoglobin	76/100		
	cTnI	82/100		
12/12	CK-MB	83/100	1 h	81
	cTnT	83/100		
17/32	CK-MB	100/100	1 h	82
	myoglobin	100/100		
52/45	CK-MB	57/54	1 1/2 h	83
	myoglobin	84/40		
	MM isoforms	53/65		
	cTnT	64/54		
8/19	CK-MB	100/61	1 h	84
	myoglobin	100/94		
	cTnI	100/67		
61/146	CK-MB+myo	83/78	1 1/2 h	85

<sup>a</sup>non-reperused group/reperused group. <sup>b</sup>Time interval between initiation of therapy and collection of blood.

**Table 4. Summary of early biochemical markers for acute coronary syndromes<sup>a</sup>**

<b>Marker</b>	<b>Biochemical function</b>	<b>size, kDa</b>	<b>Clinical utility</b>
<u>markers of inflammation</u>			
C-reactive protein	acute phase reactant	~120	non-specific
amyloid protein A	acute phase reactant	12.5 (monos) 220-235 (polys)	markers of inflammation
<u>coagulation factors and proteins</u>			
soluble fibrin monomers	soluble protein precursor	??	early detection of
thrombus precursor protein	insoluble fibrin	??	thrombus form.
<u>platelet function</u>			
soluble P-selectin	platelet activation	140	platelet aggregation
<u>ischemic marker</u>			
glycogen phosphorylase BB	enzyme of glycogenolysis	~200	reversible injury
<u>biochemical markers</u>			
carbonic anhydrase III	converts $\text{HCO}_3^-$ to $\text{H}_2\text{CO}_3$	28	skeletal muscle protein (used w/ myo)
fatty acid binding protein	cytosolic fatty acid carrier	15	non-specific early AMI marker

<sup>a</sup>Modified from Clin Lab News 1996;22:wall poster. Used with permission from the American Association for Clinical Chemistry.

**Table 5. Continuing analytical issues for implementation of cardiac troponin as an accepted standard for myocardial injury.**

lack of assay standardization for cTnI

lack of standardization between laboratory-based and point-of-care testing platforms

lack of good analytical correlation (e.g.,  $r > 0.950$ ) among commercial cTnI assays for clinical specimens

variability in imprecision for all cardiac troponin assays

variability in acceptable blood collection tubes

appropriate cutoff concentrations not documented

potential for false positive results due to presence of fibrin and human anti-mouse antibodies.