La troponina nel paziente anziano con patologia acuta: significato e ruolo prognostico

Intissar Sleiman
✅ Causes of troponin elevation
✅ Prognostic roles
✅ Significance
Causes of troponin elevation
Causes of troponin elevation

- Myocardial infarction
- In general population without any acute process:
  - Heart failure
  - Left ventricular hypertrophy
  - Chronic kidney disease
  - Diabetes
- A variety of clinical scenarios other than acute coronary syndromes
Elevations of troponin in the absence of overt ischemic heart disease

- Cardiac contusion, or other trauma including surgery, ablation, pacing, etc.
- Congestive heart failure—acute and chronic
- Aortic dissection
- Aortic valve disease
- Hypertrophic cardiomyopathy
- Tachy- or bradyarrhythmias, or heart block
- Apical ballooning syndrome
- Rhabdomyolysis with cardiac injury
- Pulmonary embolism, severe pulmonary hypertension
- Renal failure
- Acute neurological disease, including stroke or subarachnoid haemorrhage
- Infiltrative diseases, e.g. amyloidosis, haemochromatosis, sarcoidosis, and scleroderma
- Inflammatory diseases, e.g. myocarditis or myocardial extension of endo-/pericarditis
- Drug toxicity or toxins
- Critically ill patients, especially with respiratory failure or sepsis
- Burns, especially if affecting >30% of body surface area
- Extreme exertion
✓ Prognostic roles
Troponin as a Risk Factor for Mortality in Critically Ill Patients Without Acute Coronary Syndromes

Peter Ammann, MD,* Marco Maggiorini, MD,† Osmund Bertel, MD,* Edgar Haenseler, MD,§ Helen I. Joller-Jemelka, MD,‖ Erwin Oechslin, MD,¶ Elisabeth I. Minder, MD,† Hans Rickli, MD,¶ Thomas Fehr, MD‡

Zurich, Switzerland

A

B

Serum Cardiac Troponin T as a Prognostic Marker in Early Sepsis*

Claudia Spies, MD; Volker Haude, MD; Rudolf Fitzner, MD; Klaus Schröder, MD; Maria Overbeck, MD; Norbert Runkel, MD; and Walter Schaffartzik, MD
Table 1—Basic Patient Characteristics*

<table>
<thead>
<tr>
<th></th>
<th>High S-TnT (n=18)</th>
<th>Low S-TnT (n=8)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>59 (21-89)</td>
<td>61 (41-69)</td>
<td>0.636</td>
</tr>
<tr>
<td>Body surface area, m²</td>
<td>1.8 (1.3-2.4)</td>
<td>1.7 (1.6-2.0)</td>
<td>0.470</td>
</tr>
<tr>
<td>Male/female</td>
<td>11/7</td>
<td>4/4</td>
<td>0.603</td>
</tr>
<tr>
<td>APACHE III</td>
<td>48 (24-67)</td>
<td>47 (26-68)</td>
<td>0.861</td>
</tr>
<tr>
<td>CHF</td>
<td>2/18</td>
<td>1/8</td>
<td>0.920</td>
</tr>
</tbody>
</table>

*Median (range); frequency; APACHE=acute physiology and chronic health evaluation score; CHF=chronic heart failure; p=difference between groups.
Table 2. Screening of Troponin-Positive Patients for Coronary Artery Disease*

<table>
<thead>
<tr>
<th></th>
<th>Survivors (n = 19)</th>
<th>Deaths (n = 13)</th>
<th>Total (n = 32)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tests performed</td>
<td>17 (89%)</td>
<td>8 (62%)</td>
<td>25 (78%)</td>
</tr>
<tr>
<td>Test results</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>15</td>
<td>6</td>
<td>21</td>
</tr>
<tr>
<td>Pathologic</td>
<td>2†</td>
<td>2‡</td>
<td>4</td>
</tr>
<tr>
<td>Relevant coronary artery disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative</td>
<td>17 (89%)</td>
<td>6 (46%)</td>
<td>23 (72%)</td>
</tr>
<tr>
<td>Positive</td>
<td>0</td>
<td>2 (15%)</td>
<td>2 (6%)</td>
</tr>
<tr>
<td>Patient not tested§</td>
<td>2 (11%)</td>
<td>5 (38%)</td>
<td>7 (22%)</td>
</tr>
</tbody>
</table>

*Troponin-positive surviving patients were tested by dobutamine stress echocardiography within three months after hospital discharge (n = 16) or by coronary angiography during hospitalization (n = 1). Troponin-positive patients who died were autopsied. †One patient had an inferior myocardial scar on the electrocardiogram and a corresponding occlusion of the right coronary artery on the coronary angiogram; one patient showed a myocardial scar on stress echocardiography. Because these two patients had no signs of active myocardial ischemia, they were classified as negative for relevant ischemic heart disease in the final analysis. ‡One patient had high-grade stenosis of the left circumflex artery and subacute myocardial infarction; one patient showed stenosing coronary artery disease and individual cardiomyocyte necrosis. §Stress echocardiography could not be performed in two patients because of their personal refusal. Autopsy could not be performed in five patients because of refusal by their relatives. Data represented as the number (%) of patients.
DEMAND ISCHEMIA

A MISMATCH BETWEEN MYOCARDIAL OXYGEN DEMAND AND SUPPLY

Increased myocardial oxygen demand:
- Tachycardia
- Changes in cardiac loading conditions
- Increases in cardiac output
- Myocardial depression

Reduced myocardial oxygen delivery:
- Reduced coronary perfusion
- Decreased oxygen delivery to the heart
<table>
<thead>
<tr>
<th>Variable</th>
<th>Patients With Septic Shock</th>
<th>Patients With SIRS Sepsis Without Shock</th>
<th>Total Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Troponin Positive (n = 18)</td>
<td>Troponin Negative (n = 6)</td>
<td>Troponin Positive (n = 32)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cytokine</td>
<td>14.8 (11.5–53.4)</td>
<td>14.9 (10.4–32.6)</td>
<td>10.6 (5.8–15.7)</td>
</tr>
<tr>
<td>Tumor necrosis factor-alpha (pg/ml)</td>
<td>0.71 (0.53–1.80)</td>
<td>0.40 (0.27–0.47)</td>
<td>0.71 (0.53–1.80)</td>
</tr>
<tr>
<td>Soluble tumor necrosis factor-alpha receptor (ng/ml)</td>
<td>1.40 (0.86–3.15)</td>
<td>1.44 (0.68–2.07)</td>
<td>0.78</td>
</tr>
<tr>
<td>Interleukin-1-beta (pg/ml)</td>
<td>0.35 (0–7.78)</td>
<td>1.03 (0.13–3.91)</td>
<td>0.70 (0.10–5.25)</td>
</tr>
<tr>
<td>Interleukin-6 (pg/ml)</td>
<td>830 (99–6,786)</td>
<td>5311 (250–17,564)</td>
<td>72 (57–364)</td>
</tr>
<tr>
<td>Interleukin-8 (pg/ml)</td>
<td>141 (61–694)</td>
<td>1242 (63–8,020)</td>
<td>54 (38–202)</td>
</tr>
<tr>
<td>Soluble intercellular adhesion molecule-1 (ng/ml)</td>
<td>2018 (948–2,307)</td>
<td>1124 (1,030–1,239)</td>
<td>852 (447–1,056)</td>
</tr>
<tr>
<td>C-reactive protein (mg/l)</td>
<td>279 (189–317)</td>
<td>218 (184–259)</td>
<td>221 (178–259)</td>
</tr>
</tbody>
</table>

*Median values over time were calculated for each patient, and the median value (interquartile range) per group is indicated. The upper normal values are indicated as follows: tumor necrosis factor-alpha < 6.3 pg/ml, soluble tumor necrosis factor-alpha receptor < 0.49 ng/ml, interleukin-1-beta < 0.1 pg/ml, interleukin-6 < 3.1 pg/ml, interleukin-8 < 0.1 pg/ml, soluble intercellular adhesion molecule-1 < 300 ng/ml, C-reactive protein < 5 mg/l.

SIRS = systemic inflammatory response syndrome.
Cardiac Troponin Elevation, Cardiovascular Morbidity, and Outcome After Subarachnoid Hemorrhage

Andrew M. Naidech, MD, MSPH; Kurt T. Kreiter, PhD; Nazli Janjua, MD; Noeleen D. Ostapkovitch, MS; Augusto Parra, MD, MPH; Christopher Commichau, MD; Brian-Fred M. Fitzsimmons, MD; E. Sander Connolly, MD; Stephan A. Mayer, MD

Background—Cardiac troponin I (cTI) release occurs frequently after subarachnoid hemorrhage (SAH) and has been associated with a neurogenic form of myocardial injury. The prognostic significance and clinical impact of these elevations remain poorly defined.

Methods and Results—We studied 253 SAH patients who underwent serial cTI measurements for clinical or ECG signs of potential cardiac injury. These patients were drawn from an inception cohort of 441 subjects enrolled in the Columbia University SAH Outcomes Project between November 1998 and August 2002. Peak cTI levels were divided into quartiles or classified as undetectable. Adverse in-hospital events were prospectively recorded, and outcome at 3 months was assessed with the modified Rankin Scale. Admission predictors of cTI elevation included poor clinical grade, intraventricular hemorrhage, loss of consciousness at ictus, global cerebral edema, and a composite score of physiological derangement (all \( P \leq 0.01 \)). Peak cTI level was associated with an increased risk of echocardiographic left ventricular dysfunction (odds ratio [OR], 1.3 per quintile; 95\% CI, 1.0 to 1.7; \( P = 0.03 \)), pulmonary edema (OR, 2.1 per quintile; 95\% CI, 1.6 to 2.7; \( P < 0.001 \)), hypotension requiring pressors (OR, 1.9 per quintile; 95\% CI, 1.5 to 2.3; \( P < 0.001 \)), and delayed cerebral ischemia from vasospasm (OR, 1.3 per quintile; 95\% CI, 1.07 to 1.7; \( P = 0.01 \)). Peak cTI levels were predictive of death or severe disability at discharge after controlling for age, clinical grade, and aneurysm size (adjusted OR, 1.4 per quintile; 95\% CI, 1.1 to 1.9; \( P = 0.02 \)), but this association was no longer significant at 3 months.

cTI elevation after SAH is associated with an increased risk of cardiopulmonary complications, delayed cerebral ischemia, and death or poor functional outcome at discharge

Circulation, 2005
90-day outcome
- Independent (mRS 0-3)
- Dependent (mRS 4-5)
- Dead (mRS 6)

Percent of Patients

Peak cTI (µg/L)
Syndrome of neurogenic stunned myocardium

- Reversible left ventricular systolic dysfunction
- Cardiogenic shock
- Pulmonary edema

Excessive release of norepinephrine from the cardiac sympathetic nerves

Circulation, 2005
Cardiac Troponin I Is Associated With Impaired Hemodynamics, Progressive Left Ventricular Dysfunction, and Increased Mortality Rates in Advanced Heart Failure

Tamara B. Horwich, MD; Jignesh Patel, MD; W. Robb MacLellan, MD; Gregg C. Fonarow, MD

Background—Cardiac troponin I (cTnI), a sensitive and specific marker of myocardial cell injury, is useful in diagnosing and assessing prognosis in acute coronary syndromes. Small studies report that cTnI is elevated in severe heart failure (HF) and may predict adverse outcomes.

Methods and Results—The present study evaluated 238 patients with advanced HF referred for cardiac transplantation evaluation who had cTnI assay drawn at the time of initial presentation. Patients with acute myocardial infarction or myocarditis were excluded from analysis. cTnI was detectable (cTnI ≥0.04 ng/mL) in serum of 117 patients (49.1%). Patients with detectable cTnI levels had significantly higher B-type natriuretic peptide (BNP) levels (P<0.001) and more impaired hemodynamic profiles, including higher pulmonary wedge pressures (P=0.002) and lower cardiac indexes (P<0.0001). A significant correlation was found between detectable cTnI and progressive decline in ejection fraction over time. Furthermore, detectable cTnI was associated with increased mortality risk (RR, 2.05; 95% CI, 1.22 to 3.43). After adjustment for other factors associated with adverse prognosis including age, sex, ejection fraction, and coronary artery disease, cTnI remained a significant predictor of death. cTnI used in conjunction with BNP further improved prognostic value.

Conclusions—cTnI is associated with impaired hemodynamics, elevated BNP levels, and progressive left ventricular dysfunction in patients with HF. cTnI may be a novel useful tool in identifying patients with HF who are at increased risk for progressive ventricular dysfunction and death. (Circulation. 2003;108:833–838.)
Kaplan-Meier cumulative survival curves for 56 patients with severe pulmonary hypertension. In the cTnT() group (solid line), the overall survival rates at 6, 12, and 24 months were 98%, 93%, and 81%, respectively, compared with 59% (P0.0003), 59% (P0.005), and 29% (P0.01) in group cTnT() (dashed line). P estimated by log-rank test.
Elevated cTnI is a strong and independent predictor of in-hospital death in patients admitted for acutely exacerbated COPD (OR: 6.52; CI: 1.23-34.47)
Volume and pressure overload of both the right and left ventricle can produce excessive wall tension.
Elevated Cardiac Troponin Measurements in Critically Ill Patients

Wendy Lim, MD, MSc(Epid); Ismael Qushmaq, MD; P. J. Devereaux, MD, PhD(Epid); Diane Heels-Ansdell, MSc; François Lauzier, MD; Afisi S. Ismaila, MSc; Mark A. Crowther, MD, MSc(Epid); Deborah J. Cook, MD, MSc(Epid)

Background: The clinical significance of elevated cardiac troponin (cTn) level in patients in the intensive care unit (ICU) is uncertain. We reviewed the frequency of cTn elevation and its association with mortality and length of ICU stay in these patients.

Methods: Studies were identified using MEDLINE, EMBASE, and reference list review. We included observational studies of critically ill patients that measured cTn at least once and reported the frequency of elevated cTn or outcome (mortality and length of ICU or hospital stay). We pooled the odds ratios (ORs) using the inverse variance method in studies that conducted multivariable analysis to examine the relationship between elevated cTn and mortality (adjusted analysis). We calculated the weighted mean difference in length of stay between patients with and without elevated cTn and pooled the results using the inverse variance method (unadjusted analysis).

Results: A total of 23 studies involving 4492 critically ill patients were included. In 20 studies, elevated cTn was found in a median of 43% (interquartile range, 21% to 59%) of 3278 patients. In adjusted analysis (6 studies comprising 1706 patients), elevated cTn was associated with an increased risk of death (OR, 2.5; 95% confidence interval [CI], 1.9 to 3.4; P < .001). In the unadjusted analysis (8 studies comprising 1019 patients), elevated cTn was associated with an increased length of ICU stay of 3.0 days (95% CI, 1.0 to 5.1 days; P = .004) and an increased length of hospital stay of 2.2 days (95% CI, −0.6 to 4.9; P = .12).

Conclusions: Elevated cTn measurements among critically ill patients are associated with increased mortality and ICU length of stay. Research is needed to clarify the underlying causes of elevated cTn in this population and to examine their clinical significance.

Arch Intern Med. 2006;166:2446-2454
Mortality associated with an elevated cardiac troponin (cTn) level (adjusted analysis). CI indicates confidence interval; OR, odds ratio.
Incidence of post-operative troponin I rises and 1-year mortality after emergency orthopaedic surgery in older patients

Carol P. Chong, Que T. Lam, Julie E. Ryan, Rabindra N. Sinnappu, Wen Kwang Lim

Age & Ageing; 2009
Table 2. Univariate and multivariate analysis of mortality at 1 year

<table>
<thead>
<tr>
<th></th>
<th>Unadjusted OR (95% CI)</th>
<th>Unadjusted (P)-value</th>
<th>Adjusted OR (95% CI)(^a)</th>
<th>Adjusted (P)-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.1 (1.1–1.2)</td>
<td>0.004</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Troponin rise</td>
<td>27.6 (3.6–216.1)</td>
<td>0.002</td>
<td>12.0 (1.4–104.8)</td>
<td>0.025</td>
</tr>
<tr>
<td>Number of comorbidities</td>
<td>1.8 (1.3–2.5)</td>
<td>&lt; 0.001</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Premorbid comorbidities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>3.4 (1.2–9.1)</td>
<td>0.017</td>
<td>1.3 (0.3–5.3)</td>
<td>0.689</td>
</tr>
<tr>
<td>Congestive cardiac failure</td>
<td>9.4 (2.8–31.1)</td>
<td>&lt; 0.001</td>
<td>2.3 (0.4–12.3)</td>
<td>0.327</td>
</tr>
<tr>
<td>Dementia</td>
<td>5.3 (1.9–14.8)</td>
<td>0.002</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Renal failure</td>
<td>11.5 (1.9–14.8)</td>
<td>&lt; 0.001</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Aspirin use</td>
<td>3.1 (1.1–8.3)</td>
<td>0.027</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Nitrate use</td>
<td>7.7 (1.9–20.6)</td>
<td>0.004</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Post-operative complications</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood transfusion</td>
<td>4.5 (1.6–12.3)</td>
<td>0.004</td>
<td>2.7 (0.7–10.1)</td>
<td>0.140</td>
</tr>
<tr>
<td>Any cardiac event</td>
<td>9.7 (3.2–30.0)</td>
<td>&lt; 0.001</td>
<td>6.6 (1.7–25.6)</td>
<td>0.006</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>3.9 (1.1–14.4)</td>
<td>0.041</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Congestive cardiac failure</td>
<td>21.2 (5.6–79.3)</td>
<td>0.001</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>7.7 (1.9–30.6)</td>
<td>0.004</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>6.8 (2.2–21.2)</td>
<td>&lt; 0.001</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Renal failure</td>
<td>7.3 (2.4–21.8)</td>
<td>&lt; 0.001</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Delirium</td>
<td>10.2 (3.4–30.3)</td>
<td>&lt; 0.001</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

\(^a\)Adjusted OR presented only for variables included in the final multiple logistic regression model.
Elevated Troponin I Levels in Acute Liver Failure: Is Myocardial Injury an Integral Part of Acute Liver Failure?


Fig. 1. Apparent dose effect of troponin levels in relation to coma grade, presence of arrhythmias, and outcomes. Higher troponin levels were associated in a general way with greater degrees of coma, greater likelihood of arrhythmias and death. Lower likelihood of transplantation was observed with higher troponin levels.
✓ Significance
Troponin I = Myocyte death
Myocyte death ≠ Acute Myocardial Infarction
The term **myocardial infarction** should be used when there is evidence of myocardial necrosis in a clinical setting consistent with myocardial ischemia.

Under these conditions any one of the following criteria meets the diagnosis for myocardial infarction:

1. Detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least value above the 99th percentile of the upper reference limit (URL) together with evidence of myocardial ischemia with at least one of the following

   ✓ Symptoms of ischemia
   ✓ ECG changes indicative of new ischemia (new ST-T changes or new LBBB)
   ✓ Development of pathological Q waves in the ECG
   ✓ Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

Circulation, 2007
Interpreting troponin elevations: do we need multiple diagnoses?

Gordon L. Pierpont\textsuperscript{1,2,*} and Edward O. McFalls\textsuperscript{1,2}

\textsuperscript{1}Cardiology Division, Minneapolis Veterans Administration Medical Center, 1 Veterans Drive, Minneapolis, MN 55417, USA; and \textsuperscript{2}Cardiology Division, Department of Medicine, University of Minnesota, Minneapolis, MN, USA

Received 31 July 2008; accepted 23 October 2008; online publish-ahead-of-print 29 November 2008
B.G. Donna, 71 anni

**Motivo del ricovero:**
- Sepsi da polmonite a focolai multipli
- Insufficienza respiratoria globale acuta con acidosi respiratoria

**Storia clinica:** Ipertensione arteriosa, diabete mellito tipo 2

**ESAMI EMATOCHIMICI:**
- EAB: pH: 6.9  pO2: 45  pCO2: 60
- G.B: 26X10$^3$, Creatinina: 0.5 mg/dl, Troponina: 6.5 ng/ml,

**ECG:** tachicardia sinusale, FC 120 B/min. BBDx. EAS.

**E. OBIETTIVO:** soporosa ma risvegliabile. Toni ritmici tachifrequenti. Al torace m.v. aspro. Rantoli grossolani su tutto l’ambito.
Orientamento diagnostico

- Sepsi da polmonite a focolai multipli
- Insufficienza respiratoria globale con acidosi respiratoria
- NSTEMI (?)
ECOCARDIOGRAMMA:

Ipocinesia della parete anteriore, FE conservata. Ventricolo sx non dilatato. Ventricolo dx ipercinetico, non dilatato. IM lieve. Sclerosi aortica. IT di grado moderato, PAPs 50 mmHg.

CORONAROGRAFIA:

Coronarie prive di lesioni significative
Clinical classification of different types of myocardial infarction

Type 1
Spontaneous myocardial infarction related to ischaemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection

Type 2
Myocardial infarction secondary to ischaemia due to either increased oxygen demand or decreased supply, e.g. coronary artery spasm, coronary embolism, anaemia, arrhythmias, hypertension, or hypotension

Type 3
Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischaemia, accompanied by presumably new ST elevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood

Type 4a
Myocardial infarction associated with PCI

Type 4b
Myocardial infarction associated with stent thrombosis as documented by angiography or at autopsy

Type 5
Myocardial infarction associated with CABG
Diagnosi di dimissione

- Sepsi da polmonite a focolai multipli
- Insufficienza respiratoria globale con acidosi respiratoria
- Infarto acuto del miocardio non ST sopraelevato (tipo II)
ECOCARDIOGRAMMA:

Normale cinetica con FE conservata. Ventricolo sx non dilatato. Ventricolo dx ipercinetico, non dilatato. IM lieve. Sclerosi aortica. IT di grado moderato, PAPs 50 mmHg.

CORONAROGRAFIA:

Coronarie prive di lesioni significative
Diagnosi di dimissione

- Sepsi da polmonite a focolai multipli con danno miocardico secondario
- Insufficienza respiratoria globale con acidosi respiratoria
Troponin is a highly sensitive biomarker that aids in the detection of myocardial cell damage, which is often, but not always, due to thrombotic obstruction of a coronary artery.

Determining whether a troponin elevation is due to an ACS can be difficult.

Troponin may be useful to "rule out" a non-ST-segment elevation MI (NSTEMI), it is less useful to "rule in" this event because it is not specific for an acute coronary syndrome (ACS).

Factors that suggest CHD and an ACS include ischemic ECG changes, chest pain, wall-motion abnormalities on echocardiography, and the presence of atherosclerotic risk factors.

As a result, if troponin testing is applied indiscriminately in broad populations with a low pretest probability of thrombotic disease, the positive predictive value for NSTEMI is greatly diminished.
Troponin elevation in the absence of an ACS still retains significant prognostic value, and screening may be justified on this basis.

Troponin elevations in a variety of settings predict worse short- and long-term survival.

Regardless of the reason for poorer prognosis, patients with troponin elevation require appropriate diagnostic evaluation and therapy aimed at the underlying disorder.

Currently no data from randomized, controlled trials evaluating the efficacy of therapies aimed at reducing risk in patients with troponin elevations in the absence of an ACS.