

Troponin

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LIFE BEFORE TROPONIN

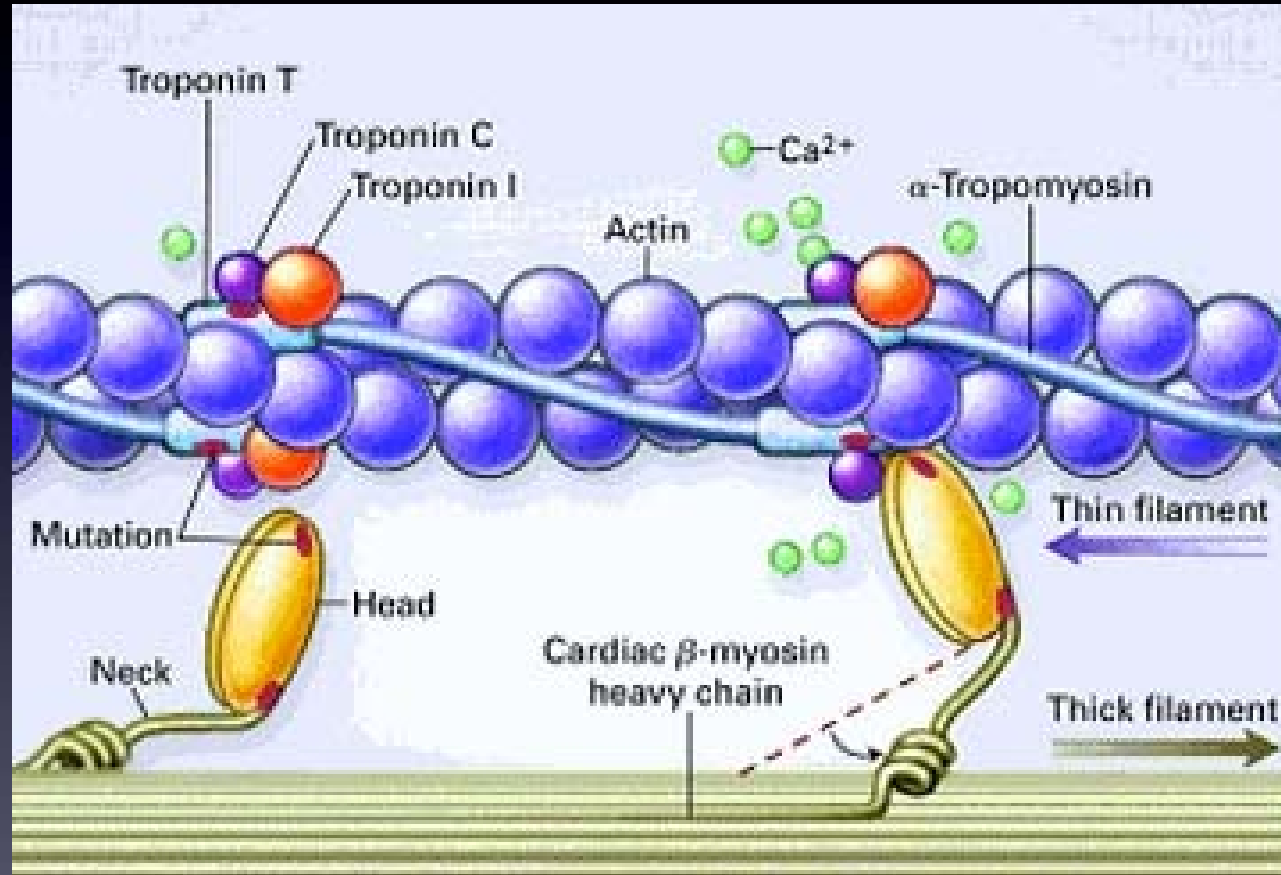
HOW *DID* WE SURVIVE?

History of chest symptoms

ECG

Biomarkers--AST, LDH ratio, CPK MB

Cardiac Biomarkers

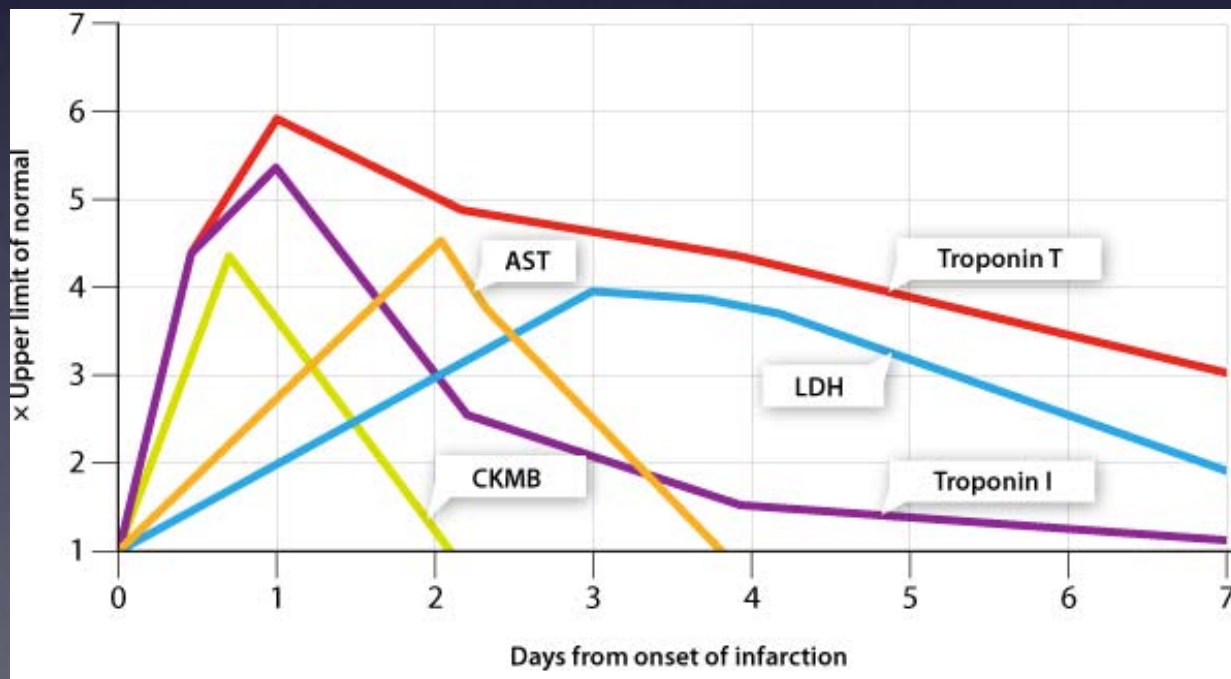


Troponin

Cardiac Tn--Earlier marker compared to CPK

Tn positive: 50% at 4 hrs, 75% at 5 hrs, 95% at 7 hrs

CPK-MB: 50% at 5 hrs, 75% at 6 hrs, 95% at 12 hrs



Troponin

NOT ALL T_n ASSAYS CREATED EQUALLY

TEST HAS GOTTEN BETTER WITH IMPROVED TECHNOLOGY

CUT OFFS FOR “POSITIVITY” HAVE GOTTEN MORE SPECIFIC

BUT THE TEST IS NOT PERFECT

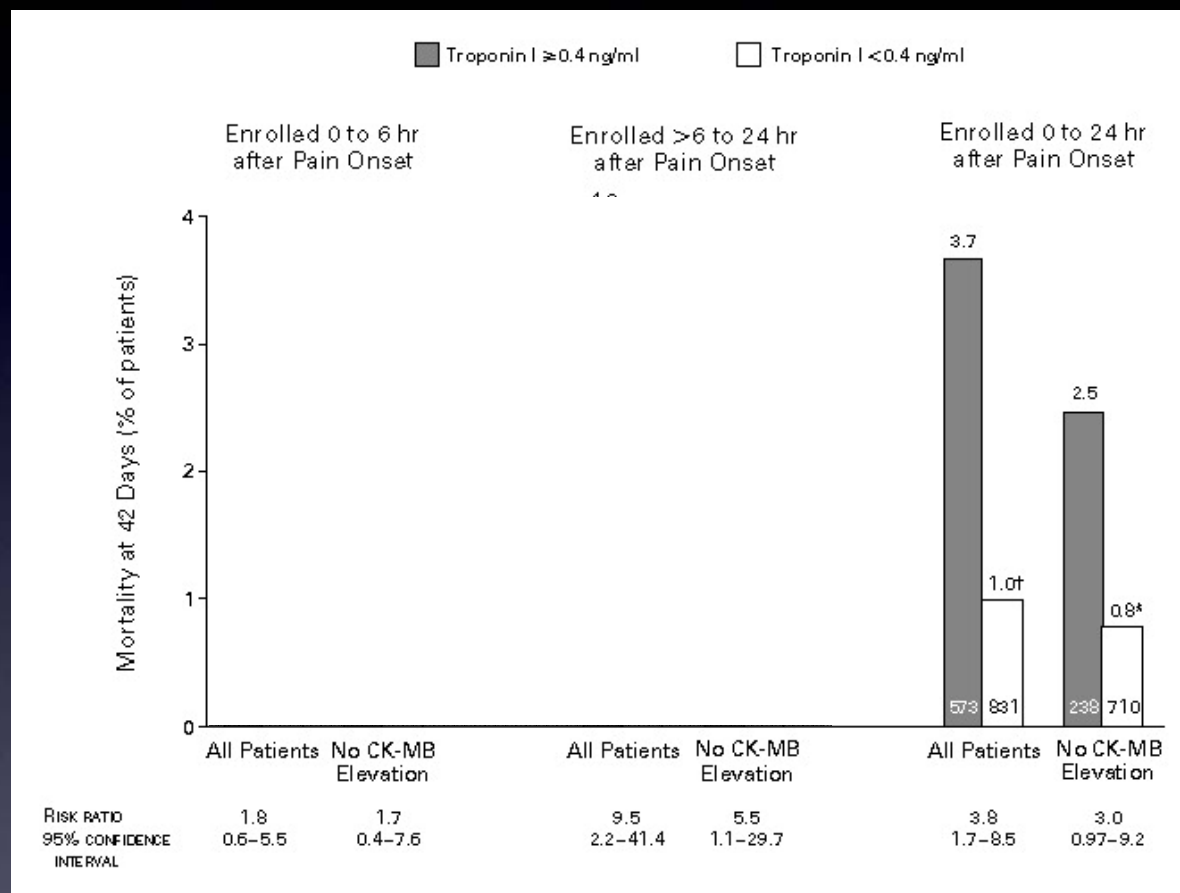
TN IS OBTAINED FREQUENTLY AND THERE IS INCREASED RISK FOR:

INCORRECT DIAGNOSIS MI

UNNECESSARY TESTS AND RISK TO PATIENT

PROBLEMS WITH PATIENTS BEING LABELED AS HAVING MI HISTORY (IF INCORRECT)

TROPONIN ASSOCIATED WITH INCREASED MORTALITY



Antman EM et al. N Engl J Med
1996;335:1342-1349.



The NEW ENGLAND
JOURNAL of MEDICINE

EMERGENCY ROOM TRIAGE OF PATIENTS WITH ACUTE CHEST PAIN BY MEANS OF RAPID TESTING FOR CARDIAC TROPONIN T OR TROPONIN I

CHRISTIAN W. HARRIS, M.D., BRITA U. GOLDMANN, M.D., CHRISTOPHER H. HERSHORN, M.D., GREGG KIRBYMANN, M.D.,
JEREMY BRIDGES, Ph.D., AND THOMAS M. PERL, M.D.

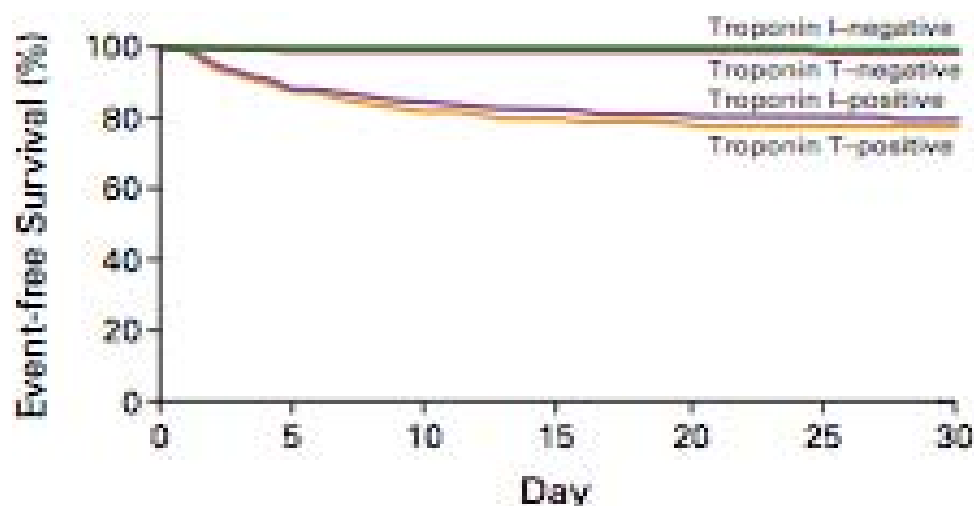


Figure 1. Survival without Cardiac Events (Death or Nonfatal Acute Myocardial Infarction) during 30 Days of Follow-up According to Troponin T and Troponin I Status.
Events that occurred during the initial 24 hours are excluded.

Positive Troponin

Ischemic

Acute Coronary Syndrome/MI

Non Acute Coronary Syndrome (Still ischemic)

Demand MI

HTN/small vessel disease

Spasm

Embolism

Procedure related (PCI/CABG)

Positive Troponin

Myocardial Damage (*non* ischemic)

Myocarditis/Pericarditis

Surgery

Cardioversion

Takasubo's/stress myocarditis/stroke

CHF

Pulmonary embolism

Renal failure ?

Sepsis

- Tn and Renal Disease

Tn is a large molecule degraded into smaller molecules detected by assay and renal clearance

Elevated Tn not solely due to renal dysfunction

Elevated Tn (above baseline) associated with poor prognosis and increased mortality

False Positive Troponin

TEST PERFORMANCE PROBLEMS

NOT ALL T_n ASSAYS CREATED EQUALLY

Poor performance

Calibration errors

Interfering substances

Interfering antibodies

Clinical Context

A “Positive” Troponin MUST be interpreted differently depending on the *clinical context*

Clinicians need to differentiate between

True positive

ACS

Other injury pattern

False positive

MI DEFINED

1980's

2/3:

Chest pain

Biomarker (CPK)

ECG

2007

Clinical setting consistent with ischemia *and*

1. A rise and fall in biomarkers (preferably Tn)

Serial Tn suggested (aside from ST elev)

BAYE'S THEOREM

The predictive value of ANY given test relates to:

1. The Prevalence of disease in the population tested

Estimated by the pretest probability of CAD for individual patients

2. Test Sensitivity and Specificity

PRETEST PROBABILITY OF ACS

RISK FACTORS FOR CAD

CHEST PAIN:

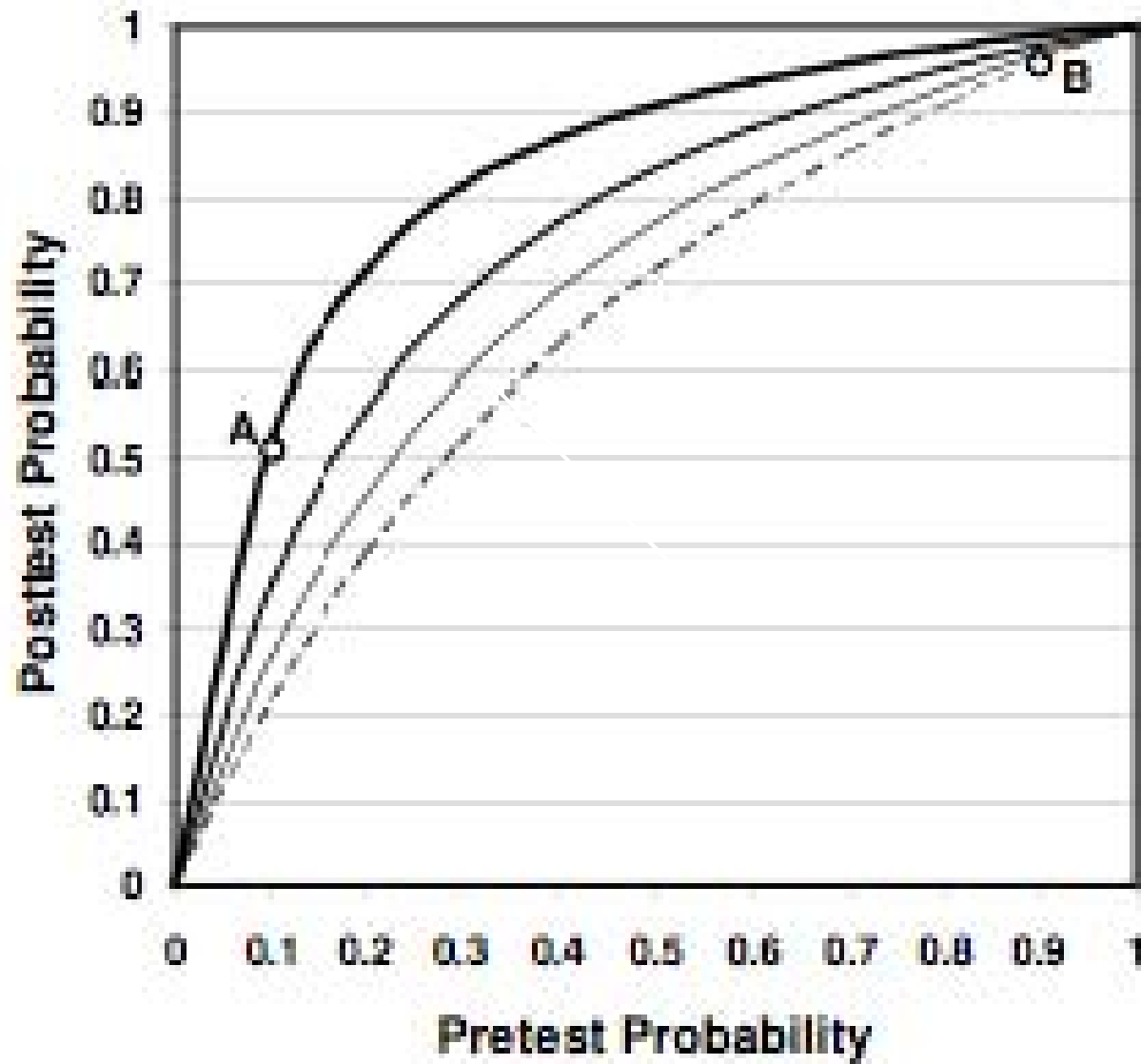
TYPICAL SYMPTOMS (REST/CRESCENDO ANGINA)

ISCHEMIC ECG CHANGES OR WALL MOTION

ABNORMALITY ON ECHO

KNOWN CAD

BAYE'S THEOREM



BAYE'S THEOREM

It follows the laws of probability

A test obtained in a low pretest probability population is more likely to be a *false positive* OR related to *ischemia/injury that is not ACS*

A tool is only as good as its operator

A test is only as good as its interpretation
Order in proper context and interpret

Approach to Chest Pain and Troponin

ACC Guidelines

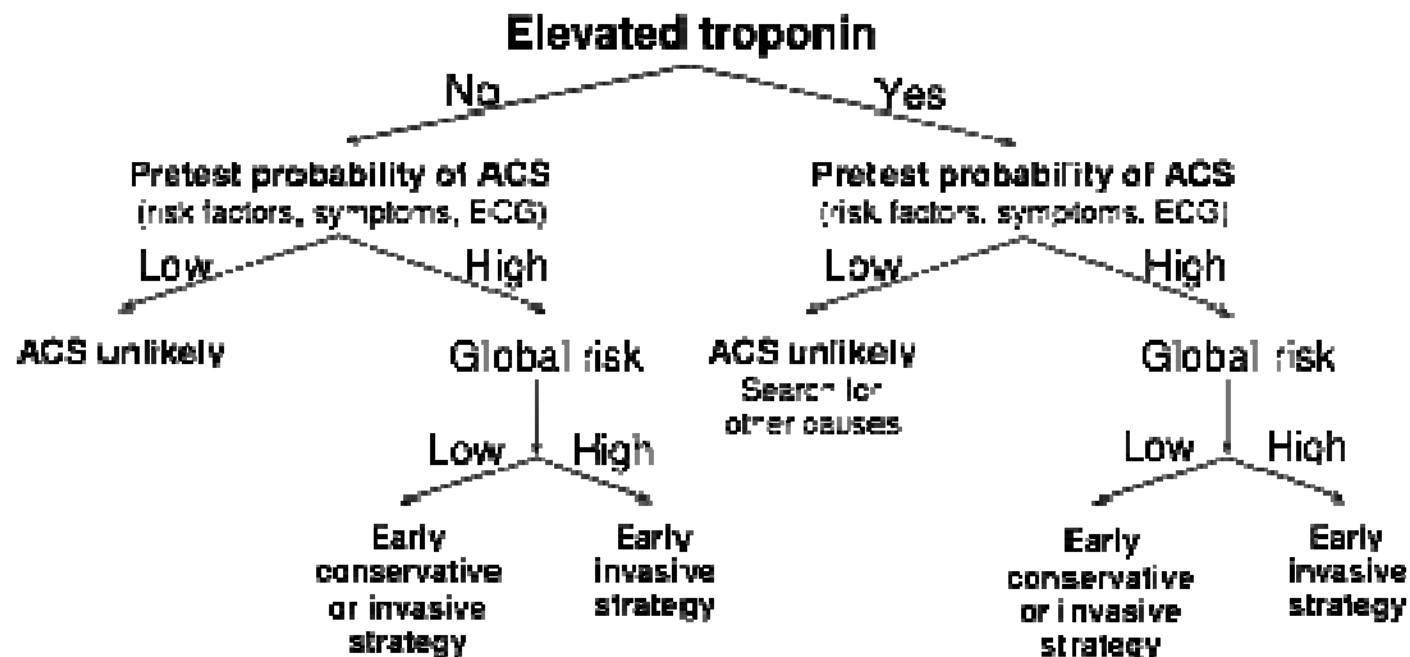
Avoid evaluation of chest pain over the phone

All need an evaluation: Hx, PE and ECG

Patients with suspected ACS should be sent to a
EW/CPU and get serial Tn

Probably no role for Tn as an outpatient/office test at
this time

Risk Stratify



Tn Summary

Troponin is a sensitive and specific for myocardial injury

Elevation does clarify the cause of injury

Myocardial necrosis is a lab diagnosis

Myocardial Infarction is a *clinical* diagnosis

Elevated Troponin in a setting that is not suggestive of MI is probably *not* an MI