

Ask the Clinical Instructor

A Q&A column for those new to the cath lab

Questions are answered by:
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“We had a patient that came to the cath lab with a non-ST-elevation MI. The patient had been in the ICU overnight before coming to the cath lab. The physician stated that the troponin levels were rising. I’m not really sure what that meant.”

— Anonymous email

The physician was talking about the cardiac biomarkers that indicate the likelihood that the patient has experienced a myocardial infarction of some degree. It can make sense when you understand where these come from.

During a myocardial infarction, cardiac cells die due to lack of blood flow caused by the obstruction. The number of cells that die is directly related to the size of the vessel that is occluded. The vessels that can be occluded can be small branches, or can be the large main arteries. The size of the “territory” covered will determine the number of cells that are cut off from blood flow.

As cells die, they release chemicals as their cellular structure falls apart. That is how these chemicals are picked up in the bloodstream and identified during laboratory tests. It should be noted that these biomarkers are released upon the cell death. Therefore, biomarkers will not be released in cases of myocardial ischemia.

Creatinine Kinase (CK) / CK-MB

When heart muscle dies, a chemical from within the cell is released into the circulation. The releases have a specific time of occurrence and concentration levels in the blood that are fairly reliable (Chart 1). The different types of CK are BB (brain- and kidney-specific), MM (skeletal muscle-specific, even though skeletal muscle does contain some MB) and MB (which can also be present in the diaphragm, small intestine, uterus and prostate).

CK/CK-MB can also pick up skele-

tal muscle damage from exertion or trauma. If the CK-MB level is above the normal limits for cardiac assessment, skeletal muscle damage would be assumed. A couple of examples would be a patient who was previously defibrillated or received recent intramuscular injections. Defibrillation and injections creates muscle damage. Another example could be someone who is in severe respiratory distress. One of the things that happens in severe respiratory distress is the use of chest muscles to help breathe. This use of muscle that results in overexertion can create CK-MB levels in the bloodstream, as those muscles are fatigued from use.

The measurement of CK/CK-MB should not be used as a one-time interpretation, but comparative over a period of time. CK/CK-MB is always present in the body, so it should be monitored for increases and an eventual plateau.

Myoglobin

Before troponins were available, myoglobin was a standard laboratory test for identification of suspected myocardial infarction. It can be obtained from urinalysis or blood levels. Unfortunately, myoglobin is release into the system following damage to any muscle cell. Because of its lack of cardiac specificity, it is not used as a stand-alone test, but can be used with other tests, such as troponins.

Troponins

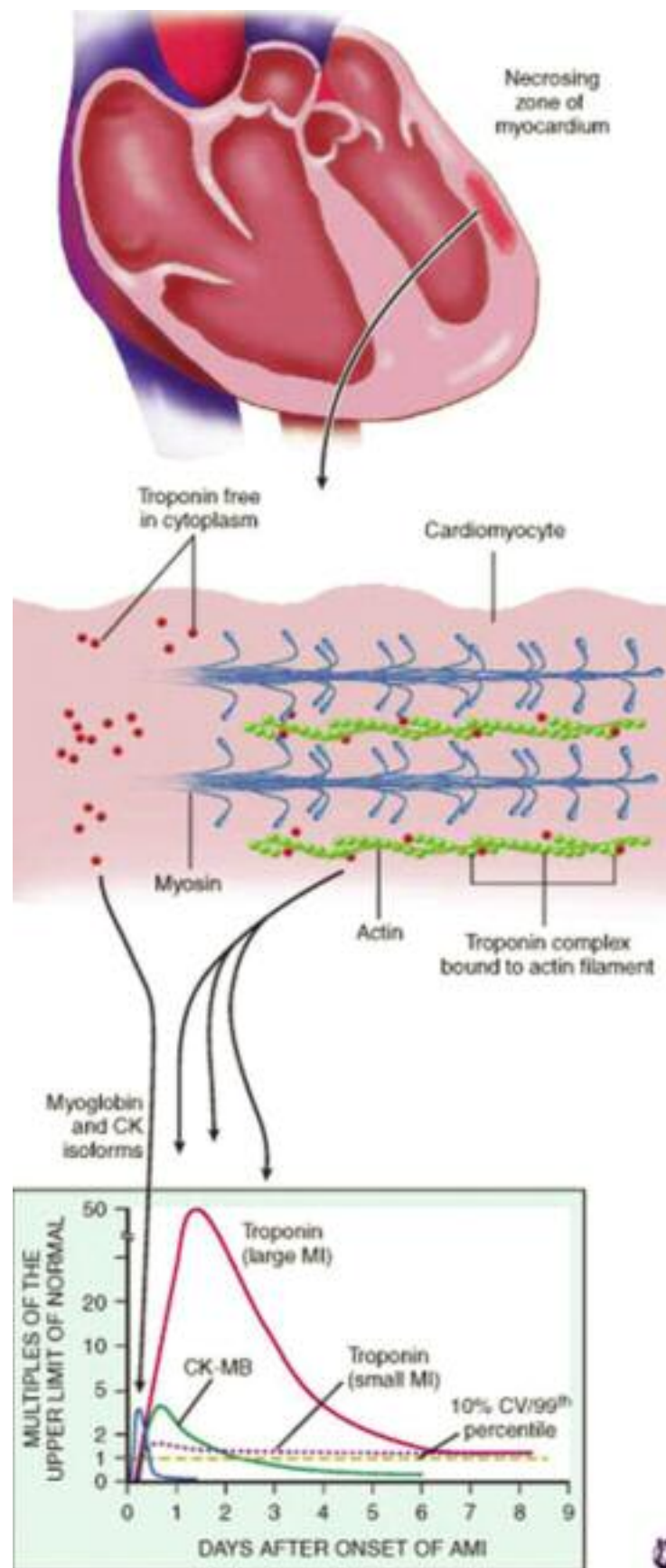
Troponins are actually present in cardiac muscle as well as skeletal muscle. However, the cardiac muscle

Chart 1. Interpretation of upper reference values must take into account the sensitivity and specificity of the type of testing. No standardized “normals” are available.³

Troponin Marker	Reference Values
Troponin I (TnI)	<0.6 ng/ml > 1.5 ng/ml consistent with myocardial infarction (MI)
Troponin T (TnT)	> 0.1 – 0.2 ng/ml consistent with MI

Figure 1. This figure helps to demonstrate where troponin levels in the blood come from. When an area of myocardium is without oxygen and dies, cell structures start to break down. Troponins are present in the cardiac cell and are attached to actin and myosin. Those chemicals are then released into the circulatory system as the cardiac cells die. The graph at the bottom of Figure 1 also depicts the changes in those levels based upon the amount of damage to the myocardium.

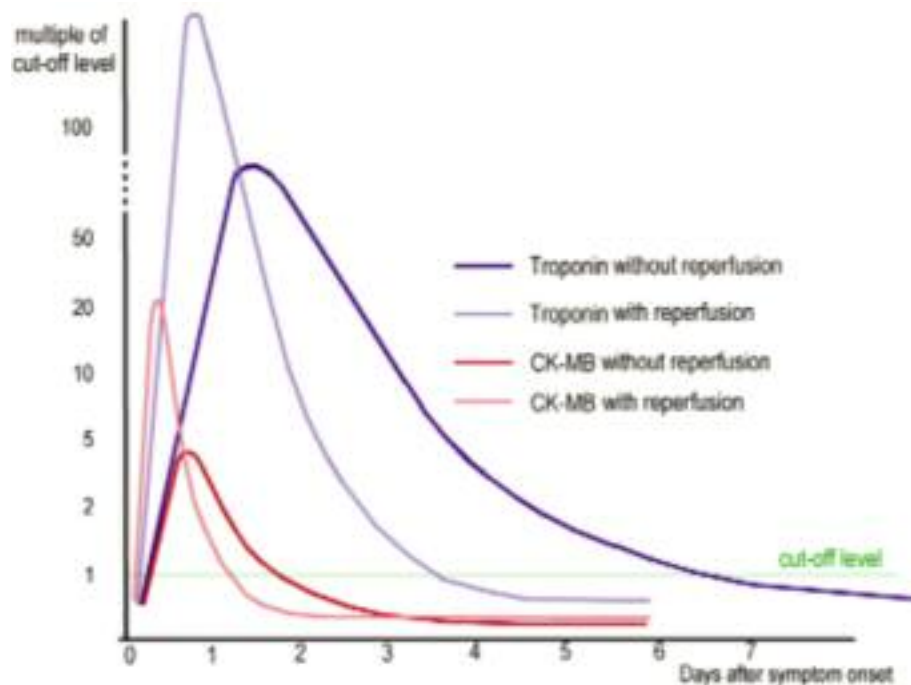
(This figure is reprinted with permission from Chapter 50, page 1225 of Libby P, Bonow RO, Mann DL, Zipes DP. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 8th Edition. Philadelphia: Elsevier Science; 2007.)



(Modified from Antman EM: Decision making with cardiac troponin tests. *N Engl J Med* 348:2078, 2002 and Jaffe AS, Babian L, Apple FS: Biomarkers in acute cardiac disease: The present and the future. *J Am Coll Cardiol* 48:1, 2006.)

Chart 2. Time relationships with the different biomarkers

Biomarker	Time to Initial Elevation	Time to Peak Elevation	Time to Return to Normal
CK-MB	3-6 hours	12-24 hours	72-96 hours
Myoglobin	1-3 hours	8-10 hours	24 hours
cTnI	4-6 hours	12 hours	3-10 days
cTnT	4-6 hours	12-48 hours	7-10 days



troponins have specific microbiological differences that allow the cardiac-specific form to be picked up on lab tests.

Troponins are attached to specific sites within the cardiac cell. When the cells die during a myocardial infarction, these chemicals are released into the bloodstream and can be measured by laboratory tests. The specific troponins used for cardiac analysis are troponin I (cTnI) and troponin T (cTnT). Both of these components are attached to specific structures within the cardiac cell (sarcomere). Troponin I binds to actin and the troponin T binds to a part of the myosin. Actin-myosin are the key components to the contraction mechanism of the sarcomere. It now should make sense that when a cardiac cell dies, the structure of the cell is somewhat decompiled, resulting in a chemical release of these items into the circulation. These provide specific measurements related to cardiac cell death.

Measurements

If your facility has a chest pain center, you would likely see orders for biomarker testings upon the arrival of the patient, and at least 6 hours later. The physician will evaluate these samples to look for increases in the biomarkers over time. Increases can be interpreted to mean that some heart muscle is dying as a result of no blood flow to a part of the myocardium. If there are no changes in the biomark-

ers, the physician may rule out myocardial infarction, OR order one more test 6 hours later and then make the determination.

Troponin levels are now considered the criterion standard in defining and diagnosing myocardial infarction, according to the American College of Cardiology (ACC)/American Heart Association (AHA) consensus statement on myocardial infarction. Cardiac troponin levels (troponin T and troponin I) have a greater sensitivity and specificity than CK-MB levels in detecting myocardial infarction. They have important diagnostic and prognostic roles. Positive troponin levels are considered virtually diagnostic of myocardial infarction in the most recent ACC/AHA revisions, as they are without equal in combined specificity and sensitivity in this diagnosis.^{1,2}

Troponins are not intended as an interpretive diagnosis in the presence of ECG changes as seen in a ST-elevation myocardial infarction (STEMI). Labs can be drawn initially to compare against later results.^{1,2}

Values

Again, these biomarkers are usually run based over specific periods of time (serial) and comparison of the results over time are the standard of measurement.

Chart 2 demonstrates the time relationships with the different biomarkers. These times are approximations

based on several sources, as differences in testings and professional interpretation of research findings are not standardized.³ n

References

1. Braunwald E, Antman EM, Beasley JW, et al. ACC/AHA guidelines for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Unstable Angina). *J Am Coll Cardiol* Sep 2000;36(3):970-1062.
2. Braunwald E, Antman EM, Beasley JW, Califf RM, Chaitlin MD, Hochman JS, et al. ACC/AHA guidelines for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction: executive summary and recommendations. A report of the American College of Cardiology/American Heart Association task force on practice guidelines (committee on the management of patients with unstable angina). *Circulation* Sep 5 2000;102(10):1193-209.
3. Corbett, JV. *Laboratory Tests and Diagnostic Procedures with Nursing Diagnoses*. 6th ed. Upper Saddle River, NJ: Pearson/Prentice Hall; 2003.

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