

BIOMARKER TIME PROFILES FOR ACUTE MYOCARDIAL INFARCTION

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Biomarker time profiles? Why? What does this have to do with respiratory therapy? If you have ever worked in a hospital you know all about codes, both on the floors and in the emergency department. These are often acute myocardial infarction (AMI) patients. These patients generally are intubated and placed on a ventilator, and, therefore, become our patients. A patient's metabolic processes differs depending on the amount of initial damage to the cardiac muscle as well as how fast and how well the muscle heals after an AMI. The internal and external respiration as well as cellular metabolism are all affected, depending on the amount of damage and/or stage of healing the patient finds themselves in. You will give better care by having a good handle on the patients' current condition. A good review of the chemistry results in the laboratory section of the chart can be very helpful in this regard.

A precursor state of AMI is myocardial ischemia, in which obstruction of a coronary artery leads to severe oxygen deprivation of the myocardium before necrosis. The major causes of AMI are atherosclerotic plaque rupture and thrombus formation. Ischemia and subsequent infarction usually begin in the endocardium and spread toward the epicardium. Necrosis to the full thickness of the myocardium is termed transmural necrosis. Irreversible cardiac injury occurs if occlusion is complete for at least 15 to 20 minutes. Irreversible injury occurs maximally when occlusion is sustained for 4 to 6 hours.

Historically, cardiac enzymes have been the hallmark tests, along with the ECG, for identifying active AMI, as well as follow-

ing the healing process. The three major enzymes that are used for cardiac evaluation are; creatine kinase (CK) or creatine phosphokinase (CPK), aspartate aminotransferase (AST, old term SGOT), and Lactic dehydrogenase (LD) or (LDH). The bad news is that each of these enzymes can be found in other organ systems. The good news is that two of the enzymes, CK and LD, have isoenzyme forms (isoforms) which are more specific for myocardium. In addition, these three enzymes tend to occur in differing amounts during the acute and healing stages of AMI. Taken as a whole, these enzymes make a nice diagnostic and prognostic tool. The normal values (generalized for both sexes) for these enzymes are as follows:

CK	15-160 IU/L
CK-MB	0%-6% of total CK
AST	5-30 IU/L
LD	8-200 IU/L
LD	114-26% of total LD
LD	229%-39% of total LD

These values and units will vary depending on the method your hospital laboratory uses for analysis.

The CK-MB is a particular isoenzyme form (isoform) of CK that is specific for myocardium. CK-MM and CK-BB also exist, but we are specifically interested in the CK-MB for AMI. You will notice there are also two isoenzymes (isoforms) listed for LD. There are actually five LD isoenzymes (isoforms). LD-1 comes from heart tissue, LD-2 from renal cortex, LD-3 from lung, lymphocytes, spleen and pancreas, LD-4 and LD-5 from Liver and skeletal muscle. LD-2 actually makes up the largest fraction of LD (see chart above); however, during AMI there is a LD-1:LD-2 "flip" in which LD-1 percent is greatly increased over LD-2, another useful diagnostic tool.

Now to the time profiles. These enzymes and isoenzyme forms (isoforms) have a predictable pattern during AMI and each rises and decreases at a different rate in the AMI patient. The following table shows these predictable patterns. The patterns are useful not only in identifying that a patient has had an AMI but also helps identify the healing process.

Myoglobin is not an enzyme but rather an oxygen-binding protein of cardiac and skeletal muscle. The protein's low molecular weight and cytoplasmic location probably account for its early appearance in the circulation after muscle injury. Increases in serum myoglobin occur after trauma to either skeletal or cardiac muscle, as in crush injuries or AMI. Serum myoglobin methods are unable to distinguish the tissue of origin. Even minor injury to skeletal muscle may result in an elevated concentration of serum myoglobin, which, by itself, may lead to the misdiagnosis of AMI. When evaluated in conjunction with cardiac enzymes; however, the myoglobin value may help to "complete the picture."

The contractile proteins of all myofibrils include the regulatory protein troponin. Troponin is a complex of three protein subunits-troponin C (the calcium-binding component), troponin I (the inhibitory component), and troponin T (the tropomyosin-binding

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Are the Current Efforts Enough?

The home care advocacy groups have undertaken a noble effort to change the framework of the current laws and despite the success in influencing members of Congress the industry must embark on lasting solutions that withstand the test of time and most importantly the changes that occur in Congress. Industry leaders and those who work in home care/HME must continue to educate policy makers but also take a parallel track to work with the National Institutes of Health (NIH) to develop tools to collect data that are consistent throughout the industry and define ways to quantify both economic and healthcare outcomes that demonstrate the value of the home care/HME industry. The money, resources and time invested in lobbying must not be the "one-stop-shop" because we have seen for years that the efforts are only slightly effective – at best - when the appropriate members are in Congress; and regardless of party affiliation of members or the presidential administration, the challenges have continued for years and the only way to deal with the issues once and for all is to take a different approach. Albert Einstein once said "The definition of insanity is doing the same thing over and over again and expecting different results". The original quote came from one of the fathers of our nation Benjamin Franklin. We would do well as an industry to listen to both of these great thinkers or be destined for repeated failures in our efforts. There have been fragmented efforts to quantify data yet these efforts are in my estimation, "one-hit-wonders" that do not "wow" anyone. The efforts are short-sighted yet the right idea; however must be expanded to create long-term results that are collaborative with the NIH to establish validity and reliability. After all, we are not the most trusted of industries in healthcare and anything we sponsor or commission will immediately fall under scrutiny by policy makers regarding motive, methodology and more importantly credibility. We must begin the effort to work with the NIH now! We've had many discussions for years yet have not taken hold and progressed in a manner that yields hope for positive changes in the perceptions not only with policy makers but with others in the healthcare continuum including physicians, nurses and other allied health professionals. In fact I recall presenting an abstract of a study conducted during my tenure with my former employer at the American College of Chest Physicians (ACCP) annual conference in Montreal Quebec Canada; in which a very prominent physician stopped by, reviewed the study, chuckled and stated that I was trying to improve the credibility of the industry - a noble cause he said since everyone is not focused on the same effort across the board. The study was published as an abstract but is certainly not enough to reverse the tide that seems to be gaining momentum against the industry. Benjamin Franklin made another important quote: "He that speaks much is much mistaken". We've spoken to CMS and policy makers tirelessly about our essential need in the continuum and either they don't care, aren't listening or misunderstand us due to the missteps taken by some in the industry regarding fraud and abuse issues.

So, now is the time to create lasting solutions that help the industry or simply continue wasting our time, efforts and money and prepare to react to the additional actions that will be undertaken by CMS.

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component). Remember this stuff from Anatomy and Physiology class? Troponin is localized primarily in the myofibrils (94% to 97%), with a smaller cytoplasmic fraction (3% to 6%).

The troponin subunits exist in a number of isoforms. The distribution of these isoforms varies between cardiac muscle and slow- and fast-twitch skeletal muscle. Cardiac-specific troponin T (cTnT) and troponin I (cTnI) isoforms have been identified. A number of studies have shown that troponin T has a clinical sensitivity similar to that of CK-MB (CK-2) during the first 48 hours after the onset of chest pain. The same is true of troponin I.

Marker	Time (h) until Marker Increases above Upper Reference Limit	Time (h) until Peak Concentration	Time (Days) until Return to within Reference Interval
CK	3 to 8	10 to 24	3 to 4
CK-MB	3 to 8	10 to 24	2 to 3
LD, LD-1	8 to 12	72 to 144	8 to 14
Myoglobin	1 to 3	6 to 9	1
Troponins I and T	3 to 8	24 to 48 (I st peak) 72 to 100 (T st peak)	3 to 5 (I) 5 to 10 (T)

A number of new studies are looking at C-reactive protein (CRP) as a potential marker for cardiovascular disease and risk assessment. The CRP rises significantly in response to injury, infection, or other inflammatory conditions and is not present in appreciable amounts in healthy individuals. At this time, the CRP is more useful as a predictor of future cardiovascular events and inflammatory processes that might be going on in the body in general rather than specifically for an AMI. There is a new reliable, automated high sensitivity assay for CRP known as the hs-CRP that does allow detection of small increases of CRP often seen in cardiac disease in general. I hope this article has given you additional tools for your diagnostic arsenal of respiratory skills. Our patients deserve the best we can provide.

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