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Troponin

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Also known as: Tnl; TnT; cTnl; cTnT

Formal name: Cardiac-specific Troponin I and Troponin T

Related tests: [CK](#); [CK-MB](#); [Myoglobin](#); [Cardiac Biomarkers](#)



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At a Glance

Why Get Tested?

To determine if you have had a [heart attack](#) or injury to heart muscle; to determine if your [angina](#) (chest pain related to heart trouble) is worsening

When to Get Tested?

Immediately, then followed by a series of troponin tests over several hours when you are having signs and symptoms that may be due to a heart attack, such as pain in your chest, shoulders, neck, jaw and/or shortness of breath; when your angina worsens, especially if it does not resolve with rest

Sample Required?

A blood sample drawn from a vein in your arm

Test Preparation Needed?

None

The Test Sample

What is being tested?

Troponins are a family of [proteins](#) found in skeletal and heart (cardiac) muscle fibers that produce muscular contraction. Troponin tests measure the level of cardiac-specific troponin in the blood to help detect heart injury.

There are three types of troponin proteins: troponin C, troponin T, and troponin I. Troponin C initiates contraction by binding calcium and moves troponin I so that the two proteins that pull the muscle fiber shorter can interact. Troponin T anchors the troponin complex to the muscle fiber structure. There is little or no difference in troponin C between skeletal and cardiac muscle, but the forms of troponin I and troponin T are different. Measuring the amount of cardiac-specific troponin T or troponin I in the blood can help identify individuals who have experienced damage to their heart.

Normally, troponin is present in very small to undetectable quantities in the blood. When there is damage to heart muscle cells, troponin is released into the blood. The more damage there is, the greater the concentration in the blood. Primarily, troponin tests are used to help determine if an individual has suffered a [heart attack](#). They may also be helpful in evaluating someone for other forms of heart injury.

When a person has a heart attack, levels of cardiac-specific troponins I and T can become elevated in the blood within 3 or 4 hours after injury and may remain elevated for 10 to 14 days.

How is the sample collected for testing?

A blood sample is taken by needle from a vein in the arm.

Is any test preparation needed to ensure the quality of the sample?

No test preparation is needed.

The Test

Because troponin tests are measuring cardiac muscle-specific troponin, the test is not affected by damage to skeletal muscles, so injections, accidents, and drugs that can damage muscle do not affect cardiac troponin levels. Troponin may rise following strenuous exercise, although in the absence of signs and symptoms of heart disease, it is usually of no medical significance.

Is there anything else I should know?

Increased troponin levels should not be used by themselves to diagnose or rule out a [heart attack](#). A physical exam, clinical history, and [ECG](#) are also important. Your healthcare provider may also need to see whether the troponin levels from a series of tests are stably elevated or show a rise over several hours.

Very rarely, people who have a heart attack will have normal troponin concentrations, and some people with increased troponin concentrations have no apparent heart injury.

Common Questions

1. What does heart attack mean?

[Heart attack](#) means that some of the muscle tissue in your heart is severely injured or has died. The medical term for the death of cardiac muscle is myocardial infarction. Because it is not always possible to determine whether there is only serious injury or there is infarction, the general term for the condition is acute coronary syndrome (ACS).

Most commonly, a heart attack starts with a sensation of heavy pressure or pain in the chest, often extending into the neck or left arm. You may have trouble catching your breath or you may feel weak and break into a cold sweat. Read about [what a heart attack feels like](#) on the National Heart, Lung, and Blood Institute web site.

A heart attack usually occurs because one of the blood vessels (called coronary arteries) that bring blood to your heart muscle is blocked. This usually happens when a blood clot forms in a blood vessel that is already partially closed. The partial closure is usually due to [atherosclerosis](#) (often called hardening of the arteries). Obstruction occurs gradually over many years as lipid [plaques](#) are deposited along the walls of the blood vessels. These plaques narrow and stiffen the arteries and can rupture unexpectedly, totally blocking off the affected artery.

2. If I have chest pain, does that mean I am having a heart attack?

Many other problems can cause chest pain, and it is not possible to tell from the type of chest pain whether or not you are having a [heart attack](#). Many people have chest pain from straining the muscles in their chest, from heartburn or other problems involving the stomach and esophagus, from emotional stress, and with some lung problems. Chest pain that occurs during exercise, hard work, or at times of stress, lasts for a few minutes, and goes away with rest is often caused by [angina](#). A relatively rare form of chest pain may be due to temporary heart spasms called variant angina. These spasms usually occur at night when a person is resting and can cause severe but temporary pain.

If chest pain lasts longer than just a few minutes, especially if it occurs when you are resting, seek immediate medical attention.

3. What if I am not sure if I am having a heart attack?

Many people are not sure if they are experiencing a [heart attack](#). If you are unsure, don't wait, but get help. If you have ongoing pain in the upper body such as the chest, shoulders, arms, neck or jaw or pain in the upper part of your stomach, or if you have been previously diagnosed with [angina](#) and the drugs you were prescribed do not ease the pain, seek immediate medical attention. Shortness of breath, nausea, sweating and dizziness are also signs and symptoms. Getting help right away can help you survive and treatments given soon after a heart attack can help limit the damage to your heart.

4. What are high-sensitivity troponin (hs-troponin) tests?

High-sensitivity troponin tests measure the same protein in the blood as the standard test but at much lower levels. Because these new generation of tests are more sensitive than standard ones, they become positive sooner and may help detect [acute coronary syndrome](#) earlier. The hs-troponin test may also be positive in people with stable [angina](#) and even in people with no symptoms. In these cases, a positive test indicates an increased risk of future heart events such as heart attacks. Hs-troponin is not approved in the U.S. at this time, but research is ongoing and it may become available soon. It is already routinely used in Europe, Canada, and other countries as well.

Related Pages

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American Academy of Family Physicians: [Heart Attack](#)

National Heart, Lung, and Blood Institute: [What is a Heart Attack?](#)

How is it used?

Troponin tests are primarily ordered to help diagnose a **heart attack** and rule out other conditions with similar signs and symptoms. Either a troponin I or troponin T test can be performed; usually a laboratory will offer one test or the other. The concentrations are different, but they basically provide the same information.

Troponin I and troponin T are **proteins** found in heart muscle and are released into the blood when there is damage to the heart.

Troponin tests are also sometimes used to evaluate people for heart injury due to causes other than a heart attack or to distinguish signs and symptoms such as chest pain that may be due to other causes. Testing may also be done to evaluate people with **angina** if their signs and symptoms worsen.

Troponin tests are sometimes ordered along with other **cardiac biomarkers**, such as **CK-MB** or **myoglobin**. However, troponin is the preferred test for a suspected heart attack because it is more specific for heart injury than other tests (which may be elevated in the blood with skeletal muscle injury) and remain elevated for a longer period of time.

A test called high-sensitivity troponin detects the same protein that the standard test does, just at much lower levels. Because this version of the test is more sensitive, it becomes positive sooner and may help detect heart injury and acute coronary syndrome earlier than the standard test. The hs-troponin test may also be positive in people with stable angina and even in people with no symptoms. When it is elevated in these individuals, it indicates an increased risk of future heart events such as heart attacks. Currently, this test is not approved in the U.S., but research is ongoing and it may become available in the near future. It is already routinely used as a cardiac biomarker in clinical practice in Europe, Canada, and other countries as well.

When is it ordered?

A troponin test will usually be ordered when a person with a suspected **heart attack** first comes into the emergency room, followed by a series of troponin tests performed over several hours.

A heart attack may be suspected and testing done when a person has **signs** and **symptoms** such as those listed below. Note that not everyone will experience chest pain, and women are more likely than men to have sign and symptoms that are not typical.

- Chest pain, discomfort and/or pressure (most common)
- Rapid heart rate, skipping a beat
- Shortness of breath and/or difficulty breathing
- Fatigue
- Nausea, vomiting
- Cold sweat
- Lightheaded
- Undue fatigue
- Pain in other places: back, arm, jaw, neck, or stomach

In people with stable **angina**, a troponin test may be ordered when:

- Symptoms worsen
- Symptoms occur when a person is at rest
- Symptoms are no longer eased with treatment

These are all signs that the angina is becoming unstable, which increases the risk of a heart attack or other serious heart problem in the near future.

What does the test result mean?

A high troponin and even slight elevations may indicate some degree of damage to the heart. When a person has significantly elevated troponin levels and, in particular, a rise in the results from a series of tests done over several hours, then it is likely that the person has had a **heart attack** or some other form of damage to the heart. Levels of troponin can become elevated in the blood within 3 or 4 hours after heart injury and may remain elevated for 10 to 14 days.

In people with **angina**, an elevated troponin may indicate that their condition is worsening and they are at increased risk of a heart attack.

Troponin levels may also be elevated with other heart conditions such as myocarditis (heart inflammation), weakening of the heart (cardiomyopathy), or **congestive heart failure**, and with conditions unrelated to the heart, such as severe infections and **kidney disease**.

Normal troponin values in a series of measurements over several hours means that it is unlikely that a person's heart has been injured. Signs and symptoms may be due to a cause unrelated to the heart.

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Table 1 Causes of elevated troponins, prevalence, and underlying mechanism

Troponin elevation (disease)	Prevalence of troponins (cut-offs)	Key references	Mechanism of troponin release
ACS related			
AMI	100% per definition	Alpert JS. <i>J Am Coll Cardiol</i> 2000; 36 :959–69.	Thrombotic occlusion of coronary artery (STEMI), microembolisation (NSTEMI)
Post-PCI	31% (cTnI) – 40% (cTnI); 24% (cTnT)	Nageh T. <i>Heart</i> 2005; 91 :1181–5. Okmen E. <i>J Invasive Cardiol</i> 2005; 17 :63–7.	Side branch occlusion, coronary dissection, bulky devices causing transient ischaemia and microembolisms
Open heart surgery	100% (cTnT)	Lehrke S. <i>Clin Chem</i> 2004; 50 :1560–7.	Myocardial infarction, incomplete cardioprotection, reperfusion injury, direct surgical trauma
Non-ACS related			
Acute pulmonary embolism	Variable, depending on cut-off; 32% at cTnT >0.1 ng/ml – 50% at cTnT >0.01 ng/ml	Giannitsis E. <i>Circulation</i> 2000; 102 :211–7. Pruszczyk P. <i>Chest</i> 2003; 123 :1947–52.	Right ventricular strain
Asymptomatic patients with ESRD	Variable, depending on cut-off; 99 th centile/10% CV/ROC: 82%/53%/20% for cTnT and 6%/1%/0.4% for cTnI	Apple FS. <i>Circulation</i> 2002; 106 :2941–5.	Several possible reasons including coronary and non-coronary cardiac origin; prolonged renal elimination; non-dialysable, intact cTnT; differences to cTnI may be related to higher affinity to dialysis membrane, unstable molecule (fragments), smaller protein
Pericarditis/myocarditis	32–49% (cTnI)	Smith SC. <i>Circulation</i> 1997; 95 :163–8. Imazio M. <i>J Am Coll Cardiol</i> 2003; 42 :2144–8. Bonnefoy E. <i>Eur Heart J</i> 2000; 21 :832–6.	Direct damage of myocytes
Aortic dissection Stanford A	24% (cTnI >1.5 ng/ml)	Bonnefoy E. <i>Acta Cardiol</i> 2005; 60 :165–	Dissection of coronary artery

Chronic HF	15% (cTnT >0.1 ng/ml) –23% (stable and unstable) (cTnI >0.3 ng/ml)	Missov E. <i>Am Heart J</i> 1999; 138 :95–9. La Vecchia L. <i>Am J Cardiol</i> 1997; 80 :88–90.	Global wall stretch, degradation of contractile protein and cellular injury due to oxidative stress and neurohumoral factors
Acute HF	52% (cTnT ≥0.02 ng/ml) –55% (cTnT ≥0.1 ng/ml)	Setsuda K. <i>Am J Cardiol</i> 1999; 84 : 608–11. Perna ER. <i>Am Heart J</i> 2002; 143 :814–20.	Global wall stretch, hypoxaemia, systemic hypoperfusion, coronary malperfusion
Strenuous exercise/ultra-endurance athletes	26% (cTnT), 9% (cTnI); 23% (cTnT), 32% (cTnI)	Rifai N. <i>Am J Cardiol</i> 1999; 83 :1085–9. Urhausen A. <i>Am J Cardiol</i> 2004; 94 :696–8.	Ventricular stretch, release of soluble troponin, underlying cardiac disease
Cardiotoxic chemotherapy	Unknown	Yeh ET. <i>Circulation</i> 2004; 109 :3122–31.	Direct toxic effect on myocytes
High frequency ablation/ current cardioversion-defibrillator shocks	90% (cTnI)	Madrid AH. <i>Am Heart J</i> 1998; 136 :948–55.	Direct myocardial damage
Cardiac infiltrative disorders (amyloidosis)	Unknown	Dispenzieri A. <i>Lancet</i> 2003; 361 :1787–9.	Myocyte compression
Heart transplant	100% (up to 3 months) variable after 3 months	Zimmermann R. <i>Br Heart J</i> 1993; 69 :395–8. Labarrere CA. <i>JAMA</i> 2000; 284 :457–64.	Inflammatory/immune mediated
Cardiac contusion after blunt chest wall trauma	12% (cTnI) – 15% (cTnT)	Edouard AR. <i>Anesthesiology</i> 2004; 101 :1262–8. Collins JN. <i>Am J Surg</i> 2001; 67 :821–5.	Direct myocardial damage
Sepsis/critical ill patients	36% (cTnT ≥0.1 ng/ml) – 85% (cTnT >0.1 ng/ml)	ver Elst KM. <i>Clin Chem</i> 2000; 46 :650–7. Ammann P. <i>Intensive Care Med</i> 2001; 27 :965–9	Oxygen supply/demand mismatch, cytokine/endotoxin mediated toxicity, heterophilic antibodies (false positive)
Rhabdomyolysis	Unknown	Lavoigne A. <i>Clinical Chemistry</i> 1998; 44 :667–8.	Cross-reactivity between skeletal and cardiac muscle isoforms of troponins in first and second generation troponin assays

ACS, acute coronary syndrome; AMI; acute myocardial infarction, ESRD; end stage renal disease; HF; heart failure; NSTEMI, non-ST elevation myocardial infarction; PCI; percutaneous coronary intervention; STEMI, ST elevation myocardial infarction.

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Table 2 Analytical causes of false-positive troponins

- Rhabdomyolysis in first and second generation troponin assays
- Heterophilic antibodies
- Rheumatoid factor
- Fibrin clots
- Microparticles
- Analyser or analyte malfunction

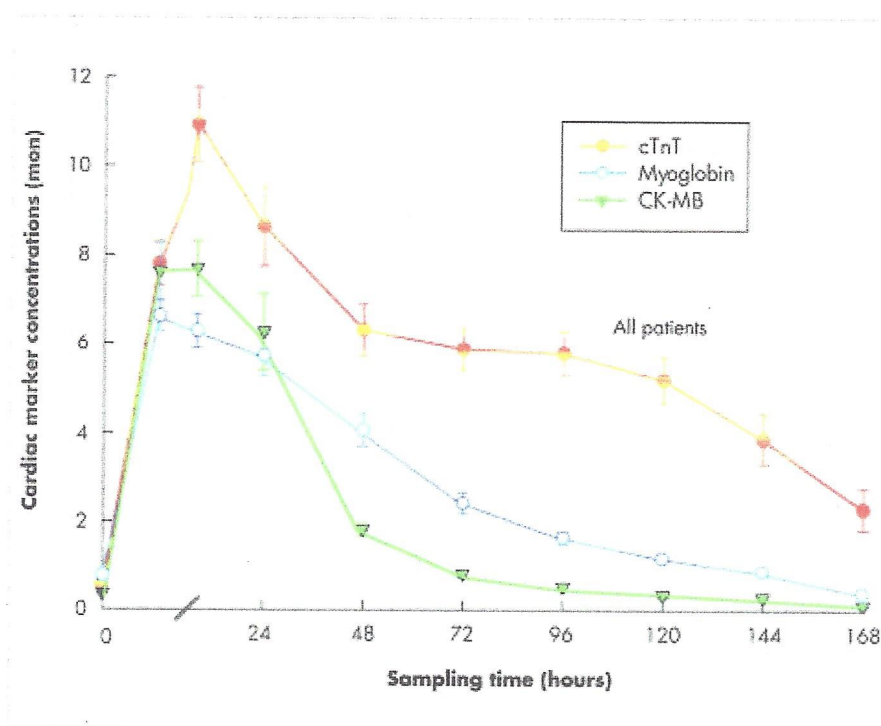
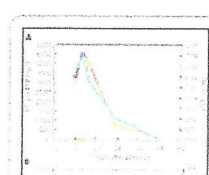
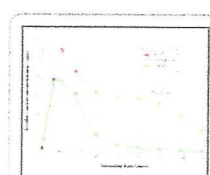
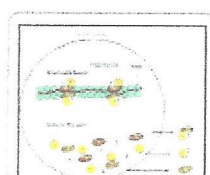


Figure 2 Release curves of cardiac markers cTnT, myoglobin and CK-MB in 204 patients following open heart surgery. Concentrations are displayed as multiples of normal (mon). Cardiac markers are elevated up to seven days after open heart surgery.

Images in this article



Do false-positive troponin results exist?

By definition, 1% of healthy volunteers with minor elevation of cTnT must be labelled false positive because only 99% of tested healthy volunteers had cTnT values below the lower limit of detection (99th centile). In clinical practice, an elevation of cTn is more often misinterpreted as false-positive when significant coronary artery disease has been ruled out by coronary angiography in a symptomatic or asymptomatic patient. This labelling is inappropriate and frequently leads to cessation of further diagnostic work-up or adequate treatment. Usually, cTn confirms and supports a clinical suspicion or diagnosis. The usefulness of troponin largely depends on the quality of the clinical judgement and the level of clinical probability.

In patients with a high probability of ACS, absence of a significant lesion does not contradict the presence of an unstable plaque. In patients with low or intermediate probability of ACS other differential diagnoses should be considered. Thus, frequently cTn is the only indicator of cardiac pathology in numerous cardiac diseases or systemic diseases with cardiac involvement. Elevated troponins could therefore open an avenue for more precise or specific diagnostic procedures.

The term false positive troponin should be restricted to analytical issues. A summary of potential reasons is given in table 2. The continuous improvement of assay performance has reduced rates of analyte-related false positive results over the years by eliminating heparin interference, or cross-reactivity with skeletal muscle. For example, first and second generation troponin assays often yielded positive results in patients with severe skeletal muscle injury because of an unspecific binding of skeletal muscle troponin T to the test tube.²⁰ Using a third generation cTnT assay, in a recent study on 10 ultra-marathon athletes, we found no cross-reactivity between cTnT and CK, neither in exercise-induced skeletal muscle trauma nor after rhabdomyolysis (unpublished data).

False Positive Cardiac Troponin Results in Patients Without Acute Myocardial Infarction

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Lab Med. 2006;37(9):546-550.



Abstract and Introduction

Abstract

Cardiac troponin I (cTnI) and T (cTnT) are highly sensitive and specific biochemical markers for myocardial necrosis and are generally not elevated in cases other than acute myocardial infarction (AMI) and acute coronary syndrome (ACS). However, if the patient's clinical picture for AMI or ACS do not match an elevated troponin result, the laboratory should suspect a false positive troponin value caused by analytical interferences with this assay. These analytic interferences include fibrin clots, microparticles in sample, heterophile and human anti-animal antibodies, rheumatoid factor, interference by bilirubin, hemolysis, lipemia, elevated alkaline phosphatase activity, macro immunocomplex formation, and analyzer malfunction. In general, analytical interferences resulting in false positive troponin results are associated with a specific manufacturer's troponin assay and are not encountered in all troponin assays. This review discusses steps which the laboratory should consider taking in the investigation of suspected analytical interference with the troponin assay. Awareness of the possibility of a false positive troponin result may assist the physician in the management of the patient without AMI or ACS and may spare the patient additional diagnostic procedures especially if the troponin result is not consistent with these diagnoses. A false positive troponin result is a reminder that although troponin plays an important role in the diagnosis of AMI and ACS, it should not be the only criterion for establishing these diagnoses.

Introduction

Cardiac troponin I (cTnI) and T (cTnT) are the most sensitive and specific biochemical markers of myocardial cell damage and have replaced creatine kinase MB as the preferred marker for diagnosis of myocardial injury.^[1,2] A consensus document issued by the European and American College of Cardiology Committee for the redefinition of myocardial infarction promoted cardiac troponin I and T as the preferred markers for myocardial damage because of their nearly absolute myocardial tissue specificity, high sensitivity, and the ability of these marker to reflect microscopic zones of myocardial necrosis.^[3] Cardiac troponins are recommended as the preferred marker in the evaluation of all patients who present with chest discomfort consistent with acute coronary syndromes (ACS), including ST-segment elevation myocardial infarction, non-ST elevation myocardial infarction, and unstable angina, which are characterized by a common pathophysiology of cardiac ischemia.^[4]

A variety of clinical conditions other than myocardial infarction may be associated with elevated cardiac troponin levels; these conditions include acute pulmonary embolism, acute or severe heart failure, myocarditis, sepsis/critically ill patients, acute pericarditis, renal failure, hypovolemia, cerebrovascular accidents, tachyarrhythmias, myocardial contusion,^[5-7] and takotsubo cardiomyopathy,^[8] reversible left ventricular dysfunction associated with emotional stress with evidence of exaggerated sympathetic activation.^[8] In addition, false elevations in patients without myocardial infarction may also occur because of analytical interferences with the troponin immunoassay. In this regard, in August, 2005, the Office of In Vitro Diagnostic Device Evaluation and Safety (OVID) of the United States Food and Drug Administration (FDA), using input provided by Advanced Medical Technology Association (AdvaMed) issued an advisory regarding false elevations in cardiac troponin results in response to many manufacturers' and users' reports of false positive troponin levels and to published articles in the medical literature.^[9] The purpose of the advisory was to inform laboratories about the possibility of false positive troponin values when this marker is used in the diagnosis of acute myocardial infarction and to recommend steps to be followed by the laboratory to identify and to confirm cases of false positive troponin results.^[9]