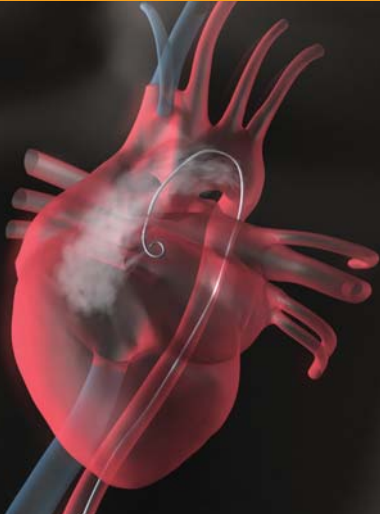


Risk Matters



Oceania

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When Is a Heart Attack a Heart Attack?

Most existing heart attack definitions in critical illness (CI) insurance policies are likely to be out of step with clinical heart attack definitions that cardiologists use with their patients. Contributing to this disconnect are current heart attack definitions (issued in 2007) and the clinical application of highly sensitive Troponin, which will increase frequency of heart attack diagnosis.

Constants within heart attack definitions used in CI policies are “death of a portion of heart muscle” due to inadequate blood supply resulting in typical symptoms (chest pain) and new ECG changes. A third measure is Troponin; this article considers the evolution of this biomarker and the implications in advances in its sensitivity.

Troponin

The enzymes Aspartate Aminotransferase (AST) and Lactate Dehydrogenase (LDH), which used to confirm cardiac muscle injury, were superseded by Creatinine Phosphokinase (CPK) almost 50 years ago. Troponin, a more specific marker, was discovered in the late 1980s and incorporated in the 2000 redefinition of myocardial infarction (MI).¹ Biomarkers had by then become acknowledged as the cornerstone of MI diagnosis.

Troponin is a protein found in the actin myosin complex of muscle cells and is essential for muscle contraction. There are three forms found in the heart – Troponin I, T and C. As Troponin C is also found in “slow twitch” skeletal muscle, it is not used in MI assessment. Troponin I is only found in heart muscle, and whilst Troponin T can be expressed in diseased skeletal muscle, modern assays are specific for heart muscle Troponin T only.² When cardiac muscle damage occurs, Troponin is released into the blood. These proteins are typically not present or found at very low concentrations in the blood of healthy individuals.

Several Troponin I assays are available and absolute cutoff values (higher than the 99th percentile) vary from one manufacturer to another and cannot be compared. The 99th percentile is that level of Troponin higher than 99% of the healthy population. Early generations of the assays were unable to measure low concentrations with a sufficient degree of precision. It is also important to note

that biochemical testing has become so refined that Troponins can reflect even microscopic areas of necrosis³ and not just larger areas of irreversible muscle damage that were traditionally considered typical and diagnostic of MI. Following the revision of the MI definition in 2000, one study indicated a 50% increase in diagnosis when using the more sensitive Troponin assay compared to CPK alone.⁴

Interestingly, the motive to adopt the 99th percentile of Troponin in a normal population for the evaluation of patients with suspected acute coronary syndrome (ACS) was primarily aimed at improving risk stratification⁵ rather than as a refinement of the MI definition. ACS incorporates MIs and unstable angina (the latter having no associated cardiac biomarker rise). Those with an elevated Troponin, regardless of cause, have a worse cardiac prognosis. Figure 1 summarises the differences between unstable angina and myocardial infarction.

Figure 1 – Differences between unstable angina and myocardial infarction

Unstable Angina versus Myocardial Infarction		
Condition	Unstable Angina	Myocardial Infarction – NSTEMI/STEMI
Severity	Less severe	More severe
Cause	Partial occlusion with microemboli	Near total or total occlusion +/- microemboli
BioMarker	Tn & CPK undetectable	Tn & CPK detectable
EKG	ST segment decreased, transient increase or normal	ST segment decreased increase or normal: may evolve Q waves
LV function	No LV dysfunction	Varies from no LV dysfunction to severe systolic dysfunction/LV dilatation/heart failure
Myocyte necrosis amount	< 1 gm	> 1.0 gm to > 25 gm
12 month mortality rate	Approx. 7%	Approx. 10%-15%

CI insurers in Oceania generally cover the more severe heart attacks by stipulating diagnostic thresholds for Troponin in the policy wording.

Importance of ischemia

The current 2007 definition⁶ (see Figure 2) defines acute MI as a clinical event consequent to the death of cardiac myocytes (myocardial necrosis) that is caused by prolonged ischemia – as distinct from other etiologies, such as myocarditis or trauma. The notation of ischaemia as a causative event is key in both definitions for clinical and CI settings because Troponin, whilst relatively specific for heart muscle, will rise and fall with any cardiac cellular damage regardless of the underlying cause. For example, a Troponin rise and fall can occur whilst running a marathon, during right heart strain against a large pulmonary embolus (originating from the venous system), and during cardiac arrhythmias – without any coronary artery disease. Also noteworthy is the diagnosis under the 2007 definition of post angioplasty procedural MI based on raised Troponin alone – without a requirement for accompanying ECG changes or typical symptoms.

Figure 2 – Definition of myocardial infarction

Criteria for acute myocardial infarction

The term myocardial infarction should be used when there is evidence of myocardial necrosis in a clinical setting consistent with myocardial ischaemia. Under these conditions, any one of the following criteria meets the diagnosis for myocardial infarction:

- Detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit (URL) together with evidence of myocardial ischaemia with at least one of the following:
 - Symptoms of ischaemia
 - ECG changes indicative of new ischaemia [new ST-T changes or new left bundle branch block (LBBB)]
 - Development of pathological Q waves in the ECG
 - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
- Sudden, unexpected cardiac death, involving cardiac arrest, often with symptoms suggestive of myocardial ischaemia, and accompanied by presumably new ST elevation, or new LBBB, and/or evidence of fresh thrombus by coronary 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.
- For percutaneous coronary interventions (PCI) in patients with normal baseline troponin values, elevations of cardiac biomarkers above the 99th percentile URL are indicative of peri-procedural myocardial necrosis. By convention, increases of biomarkers greater than 3 x 99th percentile URL have been designated as defining PCI-related myocardial infarction. A subtype related to a documented stent thrombosis is recognized.
- For coronary artery bypass grafting (CABG) in patients with normal baseline troponin values, elevations of cardiac biomarkers above 99th percentile URL are indicative of peri-procedural myocardial necrosis. By convention, increases of biomarkers greater than 5 x 99th percentile URL plus either new pathological Q waves or new LBBB, or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium have been designated as defining CABG-related myocardial infarction.

Pathological findings of an acute myocardial infarction.

Criteria for prior myocardial infarction

Any one of the following criteria meets the diagnosis for prior myocardial infarction:

- Development of new pathological Q waves with or without symptoms.
- Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of non-ischaemic cause.
- Pathological findings of a healed or healing myocardial infarction.

Source: Kristian Thygesen, Joseph S. Alpert and Harvey D. White on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction.

Highly sensitive Troponin – a new biomarker

The new highly sensitive Troponin assay (hs-cTnT) greatly improves diagnostic precision, and with time will most likely replace standard Troponin I and T tests.⁷ The highly sensitive Troponins are capable of measuring levels in the healthy population and are more reliable at the 99th percentile cutoff stipulated in the 2007 MI definition.

Key points to note regarding hs Troponin are:

- The higher sensitivity of hs Troponin in identifying minor degrees of cardiac injury, even in the absence of acute coronary syndrome,⁸ all the more necessitates presentation of other clinical features consistent with a clinical “ischaemic” heart attack rather than just an observed rise and fall in Troponin levels.
- The lack of direct correlation between hs Troponin T, results and current cutoff values for traditional Troponin I and T, creates uncertainty in assessing CI claims against an outdated definition.
- Using hs Troponin reclassifies more people from suffering unstable angina without evidence of myonecrosis to a clinical heart attack. One study diagnosed 20% more patients with non-STEMI heart attacks.⁹

Conclusion

The advent of hs Troponin assays has revolutionised the ability of clinicians to detect and diagnose MI, ACS and other events with minimal cardiac damage, “non ACS” raised Troponins. Latest generation highly sensitive Troponin assays overcome the limitations of low sensitivity and precision at lower concentrations above the 99th percentile, thereby satisfying the current consensus definition of MI.

A likely impact is an increase in numbers of both admitted and declined CI insurance claims. Estimates are difficult to make with any confidence at the present time due to lack of published data and the relative novelty of hs Troponin as a cardiac biomarker assay.

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Endnotes

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Financially Verifying Occupational Duties

Having an accurate understanding of a claimant’s specific work duties and occupation is vital for the assessment of claims. Medical practitioners, on whom insurers rely to provide objective opinions regarding a claimant’s work capacity, need to know not only the claimant’s job title, but also details of his or her specific work duties.

Our article “Working It Out: The Occupational Side of Assessing Return to Work” (*Risk Matters Oceania*, April and May 2011) offered suggestions on how to better establish occupational duties in risk protection insurance claims. Some tools proposed are Job Analysis or Functional Job Description, Workplace Assessment and other qualitative information.

While many occupations share common titles and functions, they are also quite individual in terms of what specific duties a person is responsible for, which can be attributable to the management structure of the business and the services it aims to provide.

Getting to know the claimant’s occupation

Financial records are one of the most definitive signatures of a business, and these documents can help enhance knowledge of a claimant’s occupation.

While an internal job description provides a point of reference regarding a claimant’s work duties, such records may not exist for some self-employed people, such as sole trader plumbers. Taxation returns may not be accurate for insurance purposes because the individual chooses a work title from a list of occupations provided by the government bureau, based on the closest match to the person’s line of work.

Source documents of a business ultimately assist most in developing an understanding of duties and occupations. These records are generally those contained in Profit and Loss Statements, which summarise business activities. Income and expense items may establish an individual’s work duties.

Case study

A sole trader plumber states in his initial claim form for Income Protection benefits that his job is 90% hands-on plumbing and 10% administration. Aspects of a self-employed tradesperson’s occupation that can sometimes be overlooked are the time he or she spends traveling to and from job sites, producing quotes and purchasing materials.

Financial records are useful for verifying a plumber’s activities. Many sole traders use credit cards for business purchases, and these transactions record the locations where debits are incurred. The attainment of credit card statements can confirm business activity stated in the Profit and Loss Statement, but also provide insight into the regularity of purchasing activity.

A Profit and Loss Statement should include expense items such as petrol and tolls. Receipts for vehicle tolls document the dates and times tolls are paid, and petrol is often charged to his/her clients in kilometres travelled to the worksite. An invoice to a plumber’s client should include a description of services rendered, the duration of the service including travel time, and expenses incurred in carrying out the work. For privacy reasons client details may be hidden. Tolls, petrol expenses, invoices to clients and business credit card statements reviewed in combination capture how time is used and how expense is occurred.

Analysis of the above records revealed the time this particular plumber had spent over a whole year, as follows:

- 50% hands-on plumbing
- 20% travel time
- 10% quotation for services
- 15% administration including bookkeeping
- 5% purchasing materials for jobs

This time allocation is very different from the 90% hands-on plumbing and 10% administration disclosed in the insurer's initial claim form.

It is recommended to consider at least one year's evidence to allow for work seasonality and often a person's work duties in the 12 months prior to claim can be considered his/her usual occupation. Own occupation policies offer some flexibility with respect to a claimant changing occupations after policy inception.

Conclusion

Using a claimant's own business financial records to verify his/her occupation is a technique to establish a claimant's work duties. A more expensive and time consuming method, this approach is truly effective because the evidence is specific to the individual claimant and strengthened by links between the claimant's business source documents and work duties. Insurers should consider the approach on a case-by-case basis. For sole traders or claimants who have complicated self-employed business structures, such as Trust Accounts, this technique works particularly well. A definitive understanding of a claimant's work duties and occupation informs medical providers including treating doctors, independent doctors and rehabilitation consultants with an evidence-based description of a claimant's work duties on which to provide opinion.

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The Eurozone Crisis

The declining ability of governments to attenuate economic stress will affect the capital markets for years to come, writes chief investment officer John Gilbert of General Re New England Asset Management.

Click here <<http://www.genre.com/sharedfile/pdf/Reflections201111-en.pdf>> to read the November issue of *Reflections*.



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