

# i-STAT Fact Sheet

## PoC Testing for Cardiac Troponin I

Point of Care Testing (PoCT) for cardiac Troponin-I (cTnI) using the Abbott i-STAT device is available in your local facility. The local laboratory will continue to provide cTroponin testing. While there are similarities between the two forms of testing, there are some important differences to be aware of and that may impact the interpretation of test results:

- a) The laboratory based cardiac Troponin I assay is a highly sensitive (hs-cTnI) method.
- b) The PoCT cTnI assay is less analytical sensitivity than the laboratory-based hs-cTnI assay.
- c) Currently, two different units are used. PoCT cTnI results are expressed in  $\mu\text{g/L}$  and the laboratory hs-cTnI results in  $\text{ng/L}$ . This, in effect gives a 3 orders of magnitude difference, i.e. three decimal places or one thousand times; e.g.  $0.10 \mu\text{g/L}$  from a PoCT cTnI is equivalent to  $100 \text{ng/L}$  in the laboratory unit. See points f) & g) following.
- d) The laboratory hs-cTnI and PoCT cTnI assays have different clinical decision points.
- e) The clinical decision point for i-STAT PoCT cTnI assays is  **$0.04 \mu\text{g/L}$** . Refer to your local laboratory for the laboratory decision point(s).
- f) **In many cases the PoCT cTnI and laboratory based hs-cTnI tests are not directly comparable and therefore results measured on the PoCT device should not be converted to  $\text{ng/L}$  and interpreted against the laboratory based hs-cTnI assay.**
- g) It is also important that when the baseline PoCT cTnI test is followed up with the second cTnI test to meet the Universal Definition of Myocardial Infarction (see below) ***the retesting is performed using the same assay*** and the interval is in accordance with the NSW Health “Acute Coronary Syndrome Assessment” guidelines.
- h) When a patient may to be transferred in the follow-up period to a hospital where a different or laboratory cTnI or Troponin T assay is available, the PoCT cTnI data will not be applicable assessing the magnitude of change of Troponin. In such cases it is suggested to take a baseline venous sample for a laboratory Troponin at the same time the PoCT cTnI test is collected and send this sample accompanying the patient.

### Interpretation of cardiac Troponin results:

- Elevation of cTroponin I is a highly specific marker of myocardial damage.
- According to an Expert Consensus, the Universal Definition of Acute Myocardial Infarction is: “In a clinical setting consistent with acute myocardial ischaemia.....detection of a rise and/or fall of cardiac Troponin.....with at least one value above the 99<sup>th</sup> percentile of a reference population” (*Thygesen et al. 2012;JACC 60(16):1581–98*).
- Also consider, an increased c-TnI above the 99<sup>th</sup> percentile (clinical decision point) may be caused by non-ischaemic or non-acute events and must be interpreted in the clinical context of the patient (for more information see Table 1).
- Clinicians must be aware that “false positive” and “false negative” results, although rare, may occur in all immunoassays, including troponin assays. This may be due to but not limited

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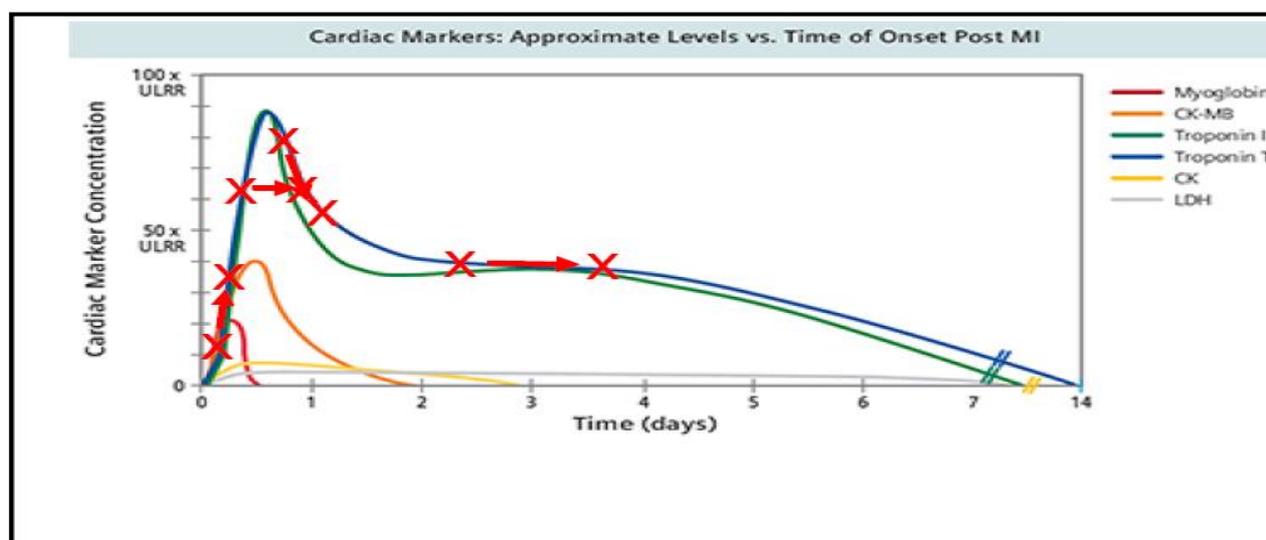
to interference from heterophilic antibodies and/or human auto-antibodies. For PoCT Troponin testing, micro clots pre-analytical are a significant risk. Good phlebotomy and an appropriately preserved well mixed sample is essential. Lithium Heparin anticoagulant only.

- In many cases the PoCT cTnI assay will have a lower diagnostic sensitivity than the laboratory-based hs-cTnI assay. The PoCT Troponin is useful to rule in the diagnosis but may give less assurance when used to rule out AMI.
- To ensure clinical diagnosis of Acute Myocardial Infarction, serial testing is essential and helps also in distinguishing an acute from a chronic or “false positive” elevation of cTnI.

**Table 1: Causes of increased Cardiac Troponin I**

<ul style="list-style-type: none"> <li>• Acute Myocardial Infarction</li> <li>• Tachy or bradyarrhythmias</li> <li>• Aortic dissection or severe aortic valve disease</li> <li>• Severe hypo or hypertension, e.g. haemorrhagic shock, hypertensive emergency</li> <li>• Acute or chronic heart failure</li> <li>• Hypertrophic cardiomyopathy</li> <li>• Coronary vasculitis, e.g. SLE, Kawasaki synd.</li> <li>• Coronary artery spasm, e.g. cocaine</li> <li>• Severe pulmonary embolism or pulmonary hypertension</li> <li>• Dialysis dependent renal failure</li> </ul>	<ul style="list-style-type: none"> <li>• Cardiac contusion or surgery. Including but not limited to CABGS &amp; stenting.</li> <li>• Rhabdomyolysis with cardiac involvement</li> <li>• Myocarditis, severe sepsis</li> <li>• Cardiotoxic agents, e.g. anthracyclines, CO poisoning</li> <li>• Severe burns affecting &gt; 30% body surface</li> <li>• Severe acute neurological conditions, e.g. stroke, trauma</li> <li>• Infiltrative diseases, e.g. amyloidosis, sarcoidosis</li> <li>• Extreme exertion, e.g. marathon running</li> <li>• Frequent defibrillator shocks</li> </ul>
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Table from: Jones GDR, Finckh A, Wilson S. *Introducing Highly Sensitive Troponin into Routine Use - A Worked Example. The Clinical Biochemist Reviews Troponin Monograph 2012; pp97-102*



**For interpretation and timing of serial troponin testing please refer to the current NSW Health “Pathway for Acute Coronary Syndrome Assessment”.**