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Focus
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Copeptin

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Copeptin (CP) is a by-product polypeptide produced from the degradation of pro-vasopressin. Pro-vasopressin is degraded into:

- **Vasopressin (VP)** : 9 aa
- **Copeptin (CP)** : 42 aa (C terminal section)
- **Neurophysin II (NPII)** : 82 aa

Copeptin was discovered some time ago (Nature 1989), but it is only recently that the Brahms Laboratory showed that there is a strict stoichiometric relationship between CP and VP.
Pathologists have had great difficulty in quantifiably measuring VP as it requires the use of a laborious radio immunology method, platelet binding and *in vitro* rapid degradation etc.
It therefore seemed obvious that quantification of copeptin could replace quantification of VP. Copeptin quantification can be performed using two antibodies which is a method that is quick and easy and one that avoids the problems encountered with VP quantification.

As a result it has highlighted the significance of VP in syndromes such as Schwartz-Barter Syndrome and other disorders that are linked with sodium. This was indeed the case, but an advance in the significance of measuring CP arose following the publication in 2009 in the *American College of Cardiology* of: an article describing a link between CP and troponin as a preliminary exclusion marker for myocardial infarction with a negative predictive value (NPV) of 99.7%.

Chest pain represents a very large proportion of emergency consultations and so needs to be investigated further. The aim is to exclude the hypothesis that a myocardial infarction is, at present, time consuming and costly. It has been shown that 60 – 80% of patients with chest pain and a normal ECG do not have a myocardial infarction.

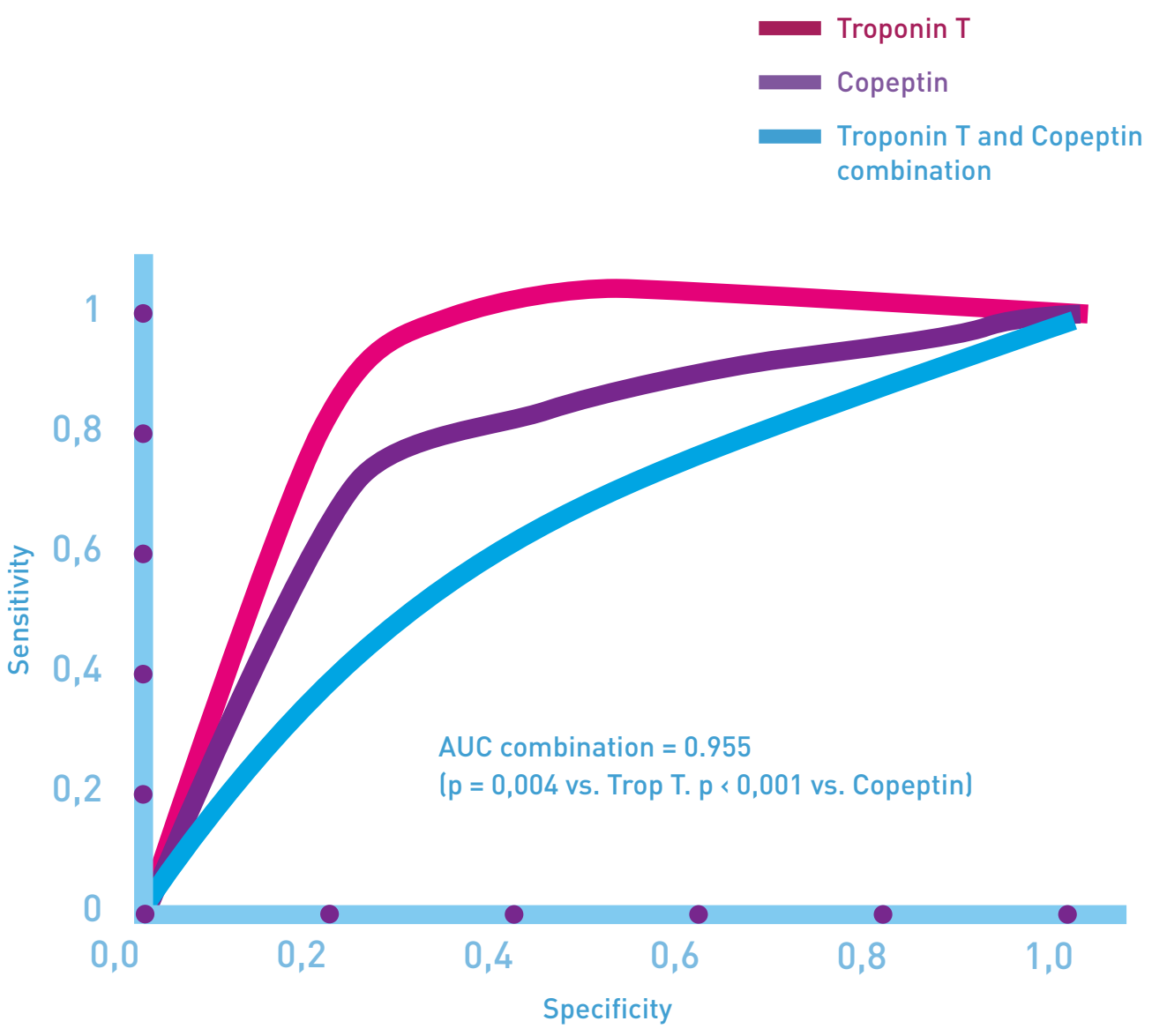
- The levels of troponin US, (the gold standard for MI), rise only during the later stages and do not allow us to exclude the possibility of MI in less than 6 hours.
  In the first 6 hours of chest pain, patients with suspected Acute Coronary Syndrome, with a normal ECG, a negative CP result and a negative tropin US result, allow the emergency services worker to exclude MI with a sensitivity level of 98.8% and a NPV of 99.7%.

  However, CP is not specific to MI as it is often found to be modestly raised in a number of conditions including severe sepsis.
  Recently, a publication in Kidney Int (2010; 77:21-36) showed a strong association between albuminuria and copeptin (CP) levels. This reinforced the hypothesis that vasopressin is implicated in the urinary excretion mechanisms of albumin. This means that making the older generation drink more could improve their renal function!

- Another observation that promotes the quantification of CP was described in regards to the future of cardiac failure patients (*European J.Clin. Investigation*). VP antagonists could become the therapeutic drug of choice in these patients.

Diagnostic performance of the Copeptin/Troponin T combination (cTnT)

Copeptin and Troponin T for the diagnosis of myocardial infarction



Reichlin et al. J Am Coll Cardiol - June 2009

Text from Dr Claude Bohuon