

MEMO

SUBJECT:	Changes to the Cardiac Analyzer
FROM:	William F. Fitter, M.D., Chief of Chemistry and Mary Pille, M.T. (ASCP), Manager, Clinical Chemistry
то:	All SJMC Medical Staff and Nursing Units

SUMMARY

Effective April 1st, 2013, substantial changes are being made to the cardiac analyzer to incorporate the most recently revised expert consensus guidelines for evaluating patients for the detection of myocardial infarction, as outlined in the "Third Universal Definition of Myocardial Infarction", jointly proposed by the European Society of Cardiology, American College of Cardiology Foundation, American Heart Association and World Heart Federation.

The cardiac analyzer ordering name, CPEU AN will not change. A single order for a cardiac analyzer will generate two blood draws, initially at 0 hours and subsequently at 6 hours. Troponin I will be run on each specimen and reported against a reference range where the upper limit of normal will be at the 99th percentile of the normal population. Measurement of Creatine Kinase (CK) and the heart associated Creatine Kinase isoenzyme (CK-MB) will no longer routinely be performed. A written pathologist interpretation will no longer be provided.

Any elevation of troponin above the limit of the reference range, greater than 0.12 ng/ml, is indicative of myocardial injury from any cause. Significant interval decrease or increase between elevated troponin values at 0 and 6 hours (> 20%) would, in the context of related clinical and EKG findings favor ischemic myocardial injury.

The abbreviated sampling time advocated by the revised guidelines is anticipated to significantly hasten clinical decision making and patient triage times. Where clinical concern for myocardial injury persists, despite a negative study at 6 hours, it is suggested that a second profile be initiated. As in the past, the new guidelines strongly caution against the exclusion of ischemic myocardial injury based upon a single negative troponin test.

BACKGROUND

More recent evaluation of the role of troponin in evaluating patients for myocardial ischemia indicates that elevation of troponin alone cannot distinguish between non-ischemic myocardial injury such as seen with viral myocarditis, traumatic injury, hypoxemia from any cause, operative injury, infective endocarditis, amyloidosis or involvement by neoplastic disorders and myocardial ischemic injury, where injury occurs as a result of restricted blood supply to the myocardium, as in coronary atherosclerosis. The revised guidelines advocate distinguishing between ischemic and non-ischemic injury based upon EKG changes, clinical signs and symptoms and serial troponin evaluations at 0 and 6 hours where an increase or decrease in serial troponin measurements of 20% would favor ischemic myocardial injury. Sustained, relatively unchanging troponin values, absence of EKG changes of ischemia, and absence of characteristic signs and symptoms would favor non-ischemic injury.

REFERENCES

Third Universal Definition of Myocardial Infarction, Thygesen, K et al, Journal of the American College of Cardiology, Vol. 60, No. 16, 2012 (http://www.sciencedirect.com/science/article/pii/S0735109712028963)

ACCF 2012 Expert Consensus Document on Practical Clinical Considerations in the Interpretation of Troponin Elevations, Journal of the American College of Cardiology, Vol. 60. No. 23. 2012 (http://www.sciencedirect.com/science/article/pii/S0735109712042398)

QUESTIONS

If you have questions or concerns, please feel free to contact either Dr William Fitter at 918-744-2555, ext 15525, wfitter@sjmc.org or Mary Pille, Manager, Chemistry at 918-744-2555 ext 15788 Mary.Pille@sjmc.org

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