Introduction: Hypoglycemia is associated with increased cardiovascular mortality in patients with and without established coronary artery disease. We have previously demonstrated that insulin induced hypoglycemia is associated with a significant reduction in myocardial blood flow reserve (MBFR). Using an ultrasensitive cardiac troponin I (us-cTnI) assay that has a limit of detection at least one order magnitude below that of current commercial assays, we investigated whether myocardial injury occurred as determined by a change in us-cTnI following a period of symptomatic insulin induced hypoglycemia and the associated reduction in MBFR.

Methods: Twenty-three subjects without obstructive coronary artery disease underwent hyperinsulinemic clamps beginning with hyperinsulinemic euglycemia (HE) followed by hyperinsulinemic hypoglycemia (HH), each for 60 minutes. Serum samples for us-cTnI, (limit of detection: 0.2 pg/ml), were taken at baseline, after 60 minutes of HE and then 60 minutes of HH with concurrent measurements of MBFR. MBFR was measured using low-power, real-time myocardial contrast echocardiography and low dose dipyridamole.

Results: Plasma glucose was 4.9±0.4 mmol/L at baseline, 4.9±0.2 mmol/L during HE and 2.8±0.1 mmol/L during HH. Mean MBFR was 2.48±0.33 at baseline, increasing during HE to 2.92±0.59 (p=0.003) and decreasing to 2.20±0.26 during HH - a fall of 11.3% and 24.7% with respect to baseline (p=0.003) and HE (p<0.001), respectively. Us-cTnI levels at baseline, HE and HH were 1.82±1.9 pg/ml, 1.71±1.7 pg/ml and 1.83±1.8 pg/ml, respectively. No significant differences in us-cTnI between different stages were detected (p=0.28).
Conclusion: In subjects without obstructive CAD, a 60 minute period of insulin induced hypoglycemia and reduction in MBFR was not associated with an increase in us-cTnI levels.