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## MYOCARDIAL ISCHEMIA AND INFARCTION

### NON-INVASIVE IMAGING OF THE LOCALIZATION AND TIME COURSE OF CELL DEATH IN A CANINE MODEL OF ACUTE MYOCARDIAL INFARCTION AND REPERFUSION: DEMONSTRATION OF EARLY AND LATE PHASES OF REPERFUSION CELL DEATH

ACC Poster Contributions

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Controversy remains regarding the time of myocardial cell death in ischemic/reperfused myocardium. Using real-time MRI and cardiac enzyme release, this critical time course is elucidated in a canine model.

**Methods:** The left anterior descending artery (LAD) was occluded using a 3 mm PTCA balloon catheter (n = 12). A constant gadolinium (Gd) infusion was administered during MRI to assess MI. Regional myocardial signal enhancement and function were recorded every 10 min during 90 min of ischemia followed by 120 min of R, and again at 24 and 48 hr post R. A catheter was inserted into the coronary sinus to measure creatine kinase (CK) leak across the coronary circulation.

With occlusion of the LAD, the risk region became hypokinetic/akinetic. There was no difference in myocardial enhancement between the ischemic and non-ischemic regions during the ischemic period and no change in CK release. CK did not rise significantly until 10 min of R and reached a peak and then began to fall after 30 min of R when infarct/LV% was 66% of maximum. No myocardial cell death was detected during ischemia. Within the first 30 min of R, 66% of the observed infarction occurred which was followed by a second phase with further progressive increase in cell death over the next 48 hrs (Fig).

These results show that R injury consists of an early phase of myocyte death followed by a gradual progressive phase of injury and indicate that while treatment to prevent R injury should start at the onset of R, treatment out to 24 hr may still result in myocardial salvage.

