

Best Original Resuscitation Science, Moderated Poster Session

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Abstract P126: Apoptosis Is Not Involved in the Mechanisms of Postresuscitation Myocardial Dysfunction in a Rat Model of Cardiac Arrest and CPR

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Background: Postresuscitation myocardial dysfunction (PRMD) has been recognized as the main cause of early death after successful resuscitation from cardiac arrest. However, the mechanisms of PRMD remain controversial. Since myocyte apoptosis plays an important role in myocardial dysfunction following ischemia/reperfusion, it has also been proposed as a potential mechanism of PRMD. We therefore investigated the presence of apoptosis during either cardiac arrest/cardiopulmonary resuscitation (CPR) or myocardial ischemia/reperfusion and related it to the severity of postresuscitation myocardial dysfunction.

Methods: fifteen male Sprague-Dawley rats weighing 450 –550 g were randomized to:

1. 8 min of untreated cardiac arrest followed by 6 min of CPR;
2. Left anterior descending coronary artery (LAD) occlusion for 45 min followed by 4 h of reperfusion; and
3. LAD sham occlusion group.

Postresuscitation and postreperfusion cardiac function measured by dp/dt_{40} were continuously monitored for 4 hours. Animals were then euthanized and the hearts were harvested for TUNEL analysis.

Results: Myocardial function was significantly impaired after both resuscitation from cardiac arrest and reperfusion from LAD occlusion ($P<0.01$). There was no difference in percentage of apoptotic cells between CPR animals and sham operated animals. However, greater apoptosis ($P<0.05$) was observed in animals subjected to LAD occlusion.

Conclusion: Myocardial function was significantly impaired after both cardiac arrest/CPR and ischemia/reperfusion. However, apoptosis was not involved in the mechanism of postresuscitation myocardial dysfunction.