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Double-edged effect of early reperfusion on arrhythmias and death after transient coronary artery occlusion in the rat

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Abstract:

Reperfusion of blood supply following a sustained period of ischemia can be injurious to the heart. Here, we monitored cardiac reperfusion in rats subjected to 10 min of wide-area coronary artery occlusion (CAO) by injecting them with the Evans blue dye, a visible inert tracer. The rats were then divided into three groups depending on their rate of reperfusion. Group 1 consisted of rats that showed no signs of early reperfusion within the 10 min period of ischemia, and these rats demonstrated a moderate rate of sudden cardiac death within the ischemic period. Group 2 included rats that showed early signs of reperfusion, and these rats demonstrated a higher rate of cardiac death than those in Group 1. Rats in Group 3 had poor ligation such that they sustained partial reperfusion almost immediately post-CAO, yet surprisingly, this group demonstrated highest rate of cardiac death during the ischemic period. In contrast to mortality during the ischemic period, rats that survived the first 10 min of ischemia then received ligation-removal and thus full-blown reperfusion. Interestingly, surviving rats from Group 3 now has best chance of continued survival post-reperfusion, whereas rats from Group 2 had poor survival rate and no rats from Group 1 survived reperfusion. To better understand the pre-conditioning effect of early reperfusion on full-reperfusion-mediated sudden cardiac death, we challenged rats to 5 min of small-area CAO. This model produced 100% survival rate during the ischemic period, and therefore all rats survived to receive full-reperfusion. In this model, rats from Group 1 demonstrated highest rate of sudden cardiac death after full-reperfusion, whereas rats from Group 2 demonstrated higher rate of survival and all rats from Group 3 survived reperfusion. Taken together, early reperfusion of blood supply may induce sudden death of the ischemic heart, but if the heart survived this earlier attack, it becomes more resistant to full-reperfusion-mediated sudden cardiac death.

Author Disclosure Information: K.H. Liao: None. D.K. Hong: None. K. Chen: None. E.Y. Kuo: None. T.W. Lai: None.

Topic Category (Complete): 3000-ASPET Cardiac Pharmacology – Injury, remodeling, protection

Sponsor (Complete):

: Pharmacology - American Society for Pharmacology and Experimental Therapeutics (ASPET) - Sponsoring Society

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Additional Information (Complete):

Grant Funding Source: : Supported by the National Science Council of Taiwan

Presentation Preference (Complete): Oral

Payment (Complete): Your credit card order has been processed on Friday 8 November 2013 at 11:05 AM.

Status: Complete

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