

**Bassuk JA, Wu D, Lozano H, Aria J, Kurlansky P, Lama GA, Adams JA.  
Non-selective cyclooxygenase inhibition before periodic acceleration (pGz) cardiopulmonary resuscitation (CPR) in a porcine model of ventricular fibrillation. Resuscitation. 2008, January 30 (Epub ahead of print)**

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Whole body periodic acceleration (pGz) along the spinal axis is a novel method of cardiopulmonary resuscitation (CPR). Oscillatory motion of the supine body in a horizontal fashion provides ventilation and blood flow to vital organs during cardiac arrest and pulsatile shear stress to the vascular endothelium. We previously showed in pigs that pGz-CPR affords better overall survival, post resuscitation myocardial function, and neurological outcomes compared to conventional chest compression CPR. pGz through pulsatile shear stress on the vascular endothelium elicits acute production of prostaglandins and endothelial-derived nitric oxide (eNO) in whole animal models and in vitro preparations. The salutary effects associated with pGz-CPR compared to chest compression CPR are in part related to endothelial-derived nitric oxide. Both eNO and prostaglandins are cardioprotective in ischemia reperfusion models. To differentiate between the roles of these mediators, indomethacin a non-selective cyclooxygenase inhibitor (COX) was used as a tool to investigate prostaglandin effects during pGz-CPR by acute outcomes of survival, cardioprotection and regional blood flows (RBF). Two groups of anesthetized, intubated pigs weighing 25-36kg were studied. Prior to electrical induction of ventricular fibrillation (VF) animals received equal volumes of either saline placebo Control (CONT) (n=9) or indomethacin (INDO), (n=8), (2mg/kg). After 3min of unsupported VF, both groups received 15min of pGz-CPR followed by pharmacologic and electrical attempts for resuscitation. Return of circulation (ROSC) to 3h occurred in (78%) in CONT and (63%) in INDO pretreated animals. There was no statistically significant difference in hemodynamics between groups at baseline or during the protocol. At baseline, INDO caused a decrease in brain RBF. Two hours after ROSC, INDO blunted the hyperemia response to brain and heart. Echocardiographic evidence of myocardial dysfunction was most notable for the INDO group in the wall motion score index (WMSI). After 3h of ROSC there was a 4-fold difference in both creatine phosphokinase (CPK) and Troponin I concentration between INDO and CONT. Therefore, non-specific acute inhibition of COX in part blunts the salutary effects of pGz-CPR. These data suggest that prostaglandins in part are involved in the cardio protection induced by pGz during CPR.