**Background** Oxidative stress contributes to tissue damage after perinatal asphyxia. The thiol-containing free radical scavenger N-acetylcysteine amide (NACA) is a promising new antioxidant with good penetration into the mitochondria. The objective was to investigate the protective effect of NACA in a piglet model of birth asphyxia.

**Methods** Anesthetized newborn piglets (n=51) were subjected to global hypoxemia and block-randomized to either
1. intravenous administration of NACA 300 mg/kg and resuscitated with 21% oxygen for 30 min,
2. saline and 21% oxygen,
3. NACA and 100% oxygen or
4. saline and 100% oxygen.

After resuscitation, the piglets were followed for 9 hours and samples for several markers of injury and oxidative stress were collected. Reported here are clinical parameters and measurements of reduced to oxidized glutathione (GSH/GSSG).

**Results** Thirty minutes after end-resuscitation metabolic acidosis was less pronounced in the 100%-NACA group compared to 100%-oxygen-alone (lactate 8.1±2.6 vs 10.9±3.4, p<0.05). This difference was not shown for the 21%-oxygen groups. Mean arterial blood pressure and hemoglobin levels remained similar between the groups. The GSSG values were generally low. At end-resuscitation GSH was lower in 100%-NACA compared to 100%-oxygen-alone group (164±111 vs 255±113 µmol, p<0.05) and delta-GSH during resuscitation greater (143±49 vs 32±66 µmol p<0.001).

**Conclusions** The data indicate that NACA may enhance immediate recovery, improve mitochondrial glutathione metabolism and restore the cell to a normal metabolism following asphyxia and resuscitation. Upcoming analyses of histopathology and injury markers will further elucidate neuroprotective effect of NACA treatment following birth asphyxia.

**Abstracts**

**306 A NOVEL ANTI-OXIDANT TO COUNTERACT OXIDATIVE STRESS DURING RESUSCITATION AFTER BIRTH ASPHYXIA**

![Image](https://example.com/306.png)

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**307 EFFECTS OF THE ISCHEMIC POSTCONDITIONING IN A NEONATAL STROKE MODEL**

![Image](https://example.com/307.png)

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The lack of efficient neuroprotective strategies for neonatal stroke could be ascribed to pathogenic ischemic processes differentiating adults and neonates. In the present study, we tested the hypothesis that ischemic postconditioning applied immediately after ischemia provides neuroprotection in a neonatal stroke model. Neonatal ischemia was generated by permanent occlusion of the left distal middle cerebral artery combined with 50 min of occlusion of both common carotid arteries (CCA) in P7 rats. Postconditioning was performed by repetitive brief release and occlusion (30 s, 1 or 5 min) of CCA after 50 min of CCA occlusion. Alternative reperfusion was generated by controlled release of the bilateral CCA occlusion. Blood-flow velocities in the left internal carotid artery were measured using ultrasound imaging with sequential Doppler recordings. Cortical perfusion was measured using laser Doppler. Local cerebral blood-flow was measured by iodine-125 antipyrine autoradiography. None of the procedures of serial mechanical interruptions of blood flow applied at reperfusion induced a reduction of infarct volume after 72 hours. In contrast to adult ischemia, a gradual perfusion was found during early re-flow both in the left internal carotid artery and in the cortical penumbra. No hyperemia was detected on autoradiograms. In addition, vasodilation to hypercapnia remained unchanged. Absence of acute hyperemia during early CCA re-flows and absence of increased cerebrovascular reactivity could at least in part explain the inefficiency of ischemic postconditioning in the developing brain.