Immediate inhalation of hydrogen (H₂) has been demonstrated to improve post-resuscitation (PR) myocardial dysfunction. However, applying inhaled H₂ early is not practical especially in out of hospital cardiac arrest due to safety issues. In the present study, we investigated the effects of delayed treatments with H₂ on post resuscitation myocardial function in a porcine model of prolonged cardiac arrest (CA). We hypothesized delayed inhalation of H₂ reduces the severity of post resuscitation myocardial dysfunction in a CA porcine model.

**Methods**

**Animal Model**

Twenty male domestic pigs weighing 39±2 kg were utilized. Ventricular fibrillation was induced electrically and CPR was initiated after 10 minutes of untreated VF. All the animals were resuscitated successfully and randomized into two groups: delayed inhalation of H₂ (DH group) or continuous inhalation of room air (C group). Animals in DH group were ventilated with 2% H₂/21% oxygen from PR 2hours until PR 4hours. Left ventricle pressure (LVP) was recorded continuously and cardiac output (CO) was measured by thermo-dilution at baseline (BL) and then hourly after resuscitation for 6 hours. Serum levels of troponin T (TnT) and N-terminal probrain natriuretic peptide (NTpro-BNP) were measured by ELISA at BL, PR 180 min and PR 360 min.

**Results**

Deterioration in the maximal rate of LVP increased (dp/dtmax), maximal rate of LVP declined (−dp/dtmax) and CO were observed in all animals after PR 4 hours (Figure 1). However, better dp/dtmax, −dp/dtmax and CO were achieved in the DH group after PR 5 hours when compared with the C group. The levels of both TnT and NTpro-BNP in serum were lower in animals treated with DH compared with those of C group (Figure 2).

**Conclusions**

Delayed treatment of H₂ attenuates myocardial injury and reduces post resuscitation myocardial dysfunction after resuscitation.

**References**


**Disclosure**

None