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

Methods

Animal Model

Eighteen male domestic pigs weighing 39 ± 2 kg were utilized. Ventricular fibrillation (VF) was induced electrically and CPR was initiated after 10 minutes of untreated VF. All the animals were resuscitated successfully and randomized into two groups immediately following resuscitation: delayed inhalation of H₂ (DH group, N=9) or inhalation of room air (C group, N=9). Animals in the DH group were ventilated with 2% H₂/21% oxygen from 2h post resuscitation (PR) to PR 4h. Serum levels of S100B and neuron-specific enolase (NSE) were measured by ELISA at baseline and PR 360 minutes. The neurological deficit score (NDS) and survival were evaluated daily for a total of 72 hours.

Results

Table 1. Survival Outcomes

<table>
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<tr>
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<th>NDS</th>
<th>Survival duration, hours</th>
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<tbody>
<tr>
<td></td>
<td>At 24h</td>
<td>At 48h</td>
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<tr>
<td>C group</td>
<td>380 ± 40</td>
<td>387 ± 38</td>
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<tr>
<td>DH group</td>
<td>270 ± 122*</td>
<td>224 ± 168*</td>
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</tbody>
</table>

Values are presented as mean ± SD. NDS, neurological deficit score; C group, control group; DH group, two hours delayed inhalation of H2 group. * p < .05 vs. the C group.

Figure 1. Changes in serum concentration of S100B and NSE.

Conclusions

Delayed treatment of H₂ mitigates neurological injury and reduces the severity of neurological dysfunction after resuscitation.

References


Disclosure

None