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11AP2-7

Effects of therapeutic hypothermia during resuscitation from porcine hemorrhagic shock

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Background: The "lethal triad" consisting of accidental hypothermia, acidosis, and coagulopathy is associated with increased mortality after trauma [1]. In contrast, therapeutic hypothermia may decrease mortality possibly due to hypometabolism and a reduced inflammatory response [2]. Furthermore, pretreatment hypothermia attenuated lactic acidosis and organ damage in a porcine model of hemorrhagic shock (HS) [3]. Therefore, we tested the hypothesis whether posttreatment hypothermia would also improve organ dysfunction and metabolic parameters in a long-term model of resuscitated porcine HS, reflecting the clinical situation of trauma patients.
Methods and Measurements: Up to now, 13 anaesthetized, mechanically ventilated, and instrumented pigs underwent a volume and pressure controlled HS of 3 hrs (withdrawal of 30% of the calculated blood volume and titration to mean arterial pressure (MAP) of 40 mmHg). Directly after HS resuscitation was started and pigs were randomized either to normothermia (38°C; n=7) or to hypothermia (34°C; n=6). Hypothermia was induced for 12 hrs with an extracorporeal heat exchanger, afterwards animals were rewarmed to 38°C. The resuscitation period of 23 hrs comprised retransfusion of shed blood, fluids, and noradrenaline to maintain MAP at pre-shock levels. Systemic hemodynamics and inflammation, coagulation, metabolic parameters, and parameters of visceral organ function were measured before and after shock, at 12 and 23 hrs of resuscitation. Markers of barrier dysfunction and oxidative/nitrosative stress were analyzed in postmortem kidney biopsies with immunohistochemistry.
Results and Discussion: Whereas there were no significant intergroup differences concerning hemodynamics, mean noradrenalin dose, and organ function (creatinine and creatinine clearance), hypothermic animals showed a significantly higher hemoglobin concentration and suffered from significantly aggravated acidosis (lower pH and base excess, higher lactate) at 12 hrs of resuscitation. One factor contributing to the negative effects of hypothermia might be increased barrier dysfunction.
Conclusion: Induced hypothermia after HS showed no benefit in this porcine model, however, it even worsened shock- and resuscitation-related acidosis.
References:
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11AP2-8

Effects of ventilation with 100 % O_2 during resuscitation after porcine haemorrhagic shock

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Background and Goal of Study: Haemorrhagic shock accounts for 30-40% of trauma mortality [1], due to secondary multiple organ failure, which is triggered by hypoxaemia and tissue ischaemia [2]. Maintaining sufficient oxygen tension therefore seems vital, but stands in contrast to the toxic effects of long-term use of inspiratory pure oxygen, especially in the lung [3]. Therefore, we tested the hypothesis whether pure O_2 ventilation would attenuate systemic inflammation and thereby organ dysfunction after long-term porcine haemorrhage and resuscitation.
Materials and Methods: Anaesthetized and instrumented animals underwent 4 hrs of haemorrhage (removal of 30% of the blood volume and subsequent titration of mean arterial pressure (MAP) at 35mmHg). Thereafter, swine were randomly assigned to either standard resuscitation ("control" group, comprising of re-transfuion of shed blood, fluid resuscitation, and continuous i.v. noradrenaline titrated to maintain MAP at pre-shock values; n=9) or the hy-

systemic and regional haemodynamics, visceral organ function (creatinine clearance, neutrophil gelatinase-associated lipocalin (NGAL), troponin), and cytokine production. Immunohistochemistry allowed the analysis of kidney nitrotyrosine formation.
Results and Discussion: Animals in the hyperoxia group presented with significantly lower troponin levels after 12 and 22 hrs of resuscitation and showed a trend towards attenuated lactic acidosis and lower noradrenaline infusion rates needed to achieve haemodynamic targets. Hyperoxia was associated with significantly higher PaO_2/FiO_2 ratios and a trend towards higher kidney blood flow, higher diuresis, and higher creatinine clearance, as well as a significantly lower NGAL formation. This coincided with significantly lower pro-inflammatory blood cytokine levels and kidney tissue nitrotyrosine formation in the hyperoxia group.
Conclusion: During early resuscitation from severe hemorrhagic shock, pure O_2 ventilation attenuated systemic inflammation as well as oxidative and nitrosative stress, which may ultimately result in improved organ function.
References:
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11AP2-9

Hemorrhagic shock, importance of fibrinogen

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Background: It is of crucial importance to correct hemostatic parameters and replace the lost blood with blood and plasma products besides surgical repair when facing hemorrhagic shock situation. Human Recombinant Fibrinogen product (Haemocomplettan P/Riastap, CSL Behring, Marburg, Germany) have been used for this purpose recently. We aimed to present our surgical and medical approach to hemorrhagic shock condition in a young adult and emphasize the significance of fibrinogen product.
Case report: A 30 year-old male (BMI: 21.7 kg m^{-2}) presented with hemorrhagic shock after abdominal gunshot injury. He was scheduled for emergency operation with ASA-VE physical status. Blood analysis revealed: pH: 6.9, HCO_3: 10 mEq/L, Hb: 3 g/dL, Hct: 10%. With the collaborative work of General Surgery, Cardiovascular Surgery and Neurosurgery departments, at the end of 2.5 hours, the patients active bleeding was stopped. The patient experienced deep hypotension and intravenous dopamine
(30 $\mu g\ kg^{-1}\ min^{-1}$) and noradrenaline 20 ($\mu g\ min^{-1}$) was administered throughout the operation; with adrenaline (20 $\mu g\ min^{-1}$) during the last 1.5 hours. Bicarbonate treatment was made for metabolic acidosis. The patient received 4 units of erythrocyte suspension, 4 units of fresh frozen plasma, 1 g fibrinogen; and these supports continued throughout the Intensive Care Unit follow-up. Supplemental 3 g of fibrinogen was given in the early postoperative period until the fibrinogen values raised to desired levels. The patient was extubated on the 2nd, and discharged on the 6th postoperative days.
Discussion: We again emphasize the importance of fibrinogen product during acute hemorrhagic shock. Fibrinogen concentrate, applied 1 g intra- and 3 g postoperatively, provided hemostasis together with surgical repair. Fibrinogen was raised from 50 to 282 mg/dL after the treatment
(normal range is 200-400 mg/dL). Three inotropic agents and bicarbonate treatment was applied to the patient throughout the operation to treat deep hypotension and acidosis. For all that, the patient's mean arterial pressure was below 40 mmHg for 1.5 hours and brain edema was developed. Still the patient was successfully recovered and extubated on the 2nd postoperative day.
Learning points: Fibrinogen product is of quite importance for hemostasis management. Patients with long-term hypotension incompatible with life can sometimes recover completely.