Effects of pretreatment hypothermia during resuscitated porcine hemorrhagic shock

Titre	Effects of pretreatment hypothermia during resuscitated porcine hemorrhagic shock
Type de publication	Article de revue
Auteur	Groger, M. [1], Scheuerle, A. [2], Wagner, F. [3], Simon, F. [4], Matallo, J. [5], McCook, O. [6], Seifritz, A. [7], Stahl, B. [8], Wachter, U. [9], Vogt, J. A [10], Asfar, Pierre [11], Matejovic, M. [12], Moller, P. [13], Lampl, L. [14], Bracht, Hendrik [15], Calzia, Enrico [16], Georgieff, M. [17], Radermacher, P. [18], Stahl, W. [19]
Editeur	Lippincott, Williams & Wilkins
Type	Article scientifique dans une revue à comité de lecture
Année	2013
Langue	Anglais
Date	2013
Numéro	7
Pagination	e105 - 17
Volume	41
Titre de la revue	Critical care medicine
ISSN	1530-0293
Mots-clés	Animals [20], Blood Chemical Analysis [21], Female [22], Glucose/metabolism [23], Hemodynamics [24], Hypothermia, Induced/methods [25], Male [26], Random Allocation [27], Resuscitation/methods [28], Shock, Hemorrhagic/blood/therapy [29], Swine [30], Time Factors [31]

Résumé en anglais	OBJECTIVES: Accidental hypothermia increases mortality and morbidity after hemorrhage, but controversial data are available on the effects of therapeutic hypothermia. Therefore, we tested the hypothesis whether moderate pretreatment hypothermia would beneficially influence organ dysfunction during long-term, porcine hemorrhage and resuscitation. DESIGN: Prospective, controlled, randomized study. SETTING: University animal research laboratory. SUBJECTS: Twenty domestic pigs of either gender. INTERVENTIONS: Using an extracorporeal heat exchanger, anesthetized and instrumented animals were maintained at 38 degrees C, 35 degrees C, or 32 degrees C core temperature and underwent 4 hours of hemorrhage (removal of 40% of the blood volume and subsequent blood removal/retransfusion to maintain mean arterial pressure at 30 mm Hg). Resuscitation comprised of hydroxyethyl starch and norepinephrine infusion titrated to maintain mean arterial pressure at preshock values. MEASUREMENTS AND MAIN RESULTS: Before, immediately at the end of, and 12 and 22 hours after hemorrhage, we measured systemic and regional hemodynamics (portal vein, hepatic and right kidney artery ultrasound flow probes) and oxygen transport, and nitric oxide and cytokine production. Hemostasis was assessed by rotation thromboelastometry. Postmortem biopsies were analyzed for histomorphology (hematoxylin and eosin staining) and markers of apoptosis (kidney Bcl-xL and caspase-3 expression). Hypothermia at 32 degrees C attenuated the shock-related lactic acidosis but caused metabolic acidosis, most likely resulting from reduced carbohydrate oxidation. Although hypothermia did not further aggravate shock-related coagulopathy, it caused a transitory attenuation of kidney and liver dysfunction, which was ultimately associated with reduced histological damage and more pronounced apoptosis. CONCLUSIONS: During long-term porcine hemorrhage and resuscitation, moderate pretreatment hypothermia was associated with a transitory attenuation of organ dysfunction and less severe histological tissue damage despite more pronounced metabolic acidosis. This effect is possibly due to a switch from necrotic to apoptotic cell death, ultimately resulting from reduced tissue energy deprivation during the shock phase.
URL de la notice	http://okina.univ-angers.fr/publications/ua8231 [32]
DOI	10.1097/CCM.0b013e31827c0b1f [33]
Lien vers le document	http://dx.doi.org/10.1097/CCM.0b013e31827c0b1f [33]
Titre abrégé	Crit Care Med

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