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ABSTRACT & CONFERENCE BOOK

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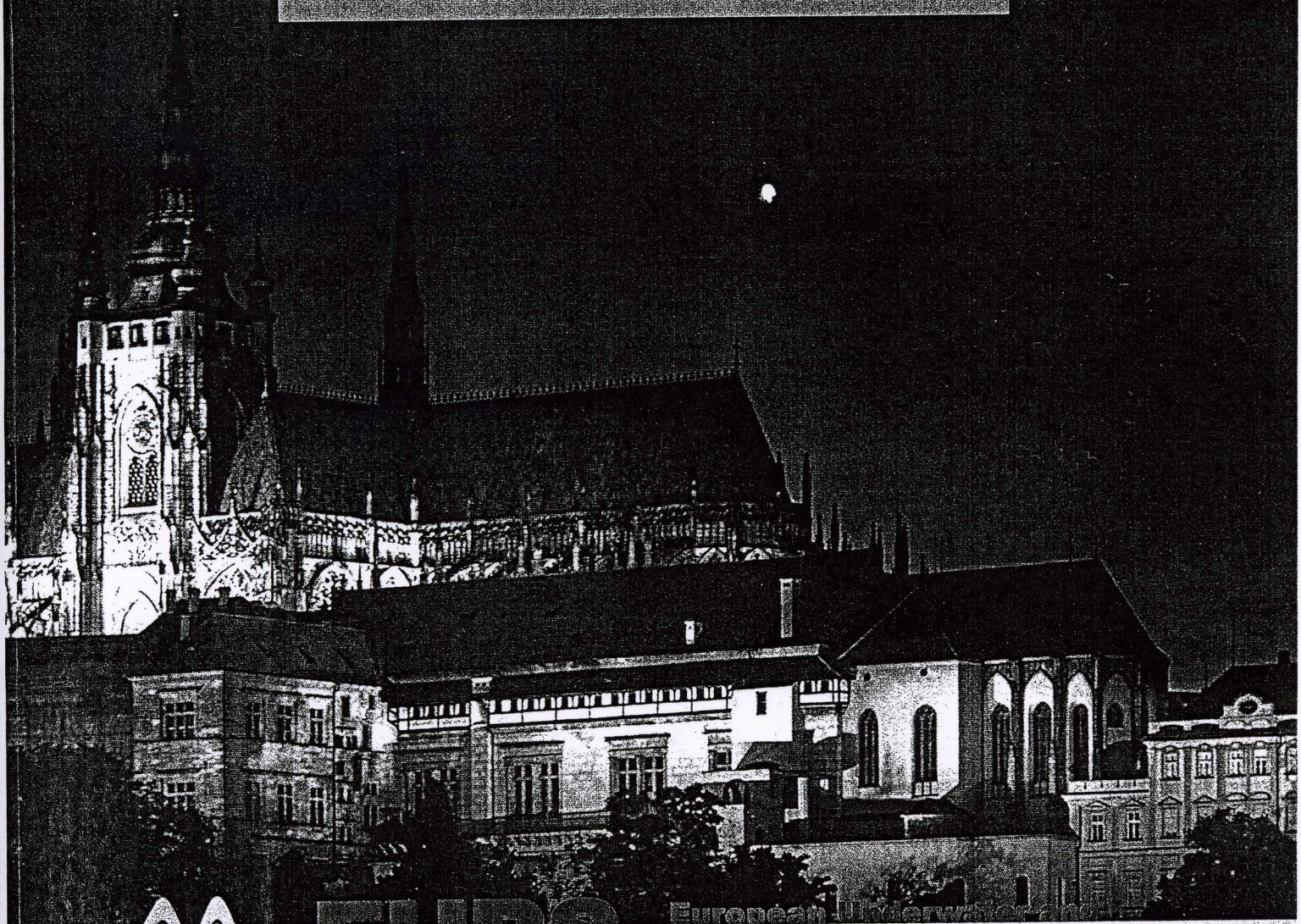
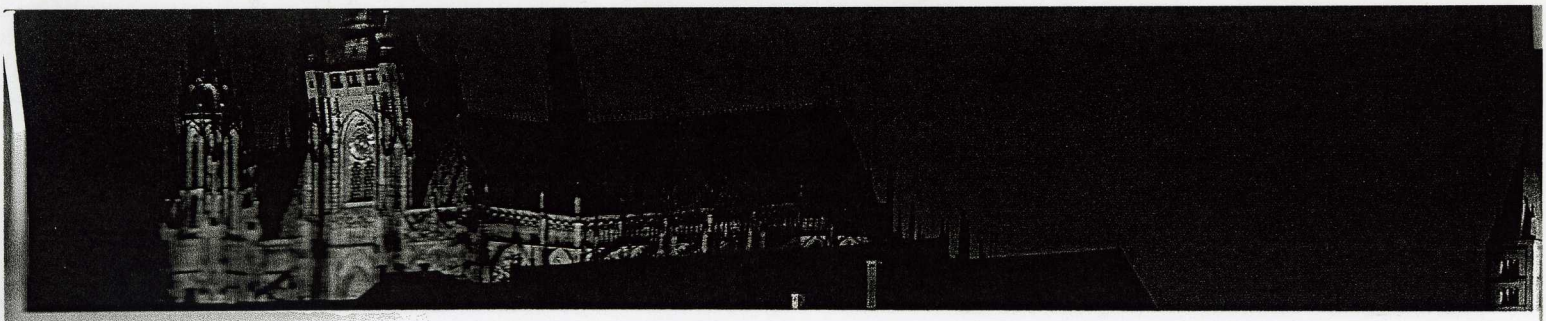


EUBS European Underwater Baromedical Society



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IMMUNOHISTOCHEMICAL EXPRESSION OF HEME OXYGENASE-1, 4-HYDROXYNONENAL AND HYPOXIA INDUCIBLE FACTORS (1α, 1β, 2α) AFTER HBO PRECONDITIONING IN POSTISCHEMIC ACUTE KIDNEY INJURY INDUCED IN SPONTANEOUSLY HYPERTENSIVE

Sanjin Kovacevic[1], Milan Ivanov[2], Maja Zivotic[3], Zoran Miloradovic[2], Predrag Brkic[4], Rada Jeremic[4], Danijela Karanovic[2], Una Jovana Vujic[2], Nevena Mihailovic Stanojevic[2], Djurdjica Jovovic[2], Jelena Nesovic Ostojic[1]

[1] *Department of Pathophysiology, Medical Faculty, University of Belgrade, Belgrade, Serbia*
[2] *Department of Cardiovascular Physiology, Institute for Medical Research, University of Belgrade, Belgrade, Serbia*
[3] *Department of Pathology, Medical Faculty, University of Belgrade, Belgrade, Serbia*
[4] *Department of Medical Physiology, Medical Faculty, University of Belgrade, Belgrade, Serbia*

Introduction
Renal ischemia/reperfusion injury (IRI) is common cause of acute kidney injury (AKI). Recent studies suggest beneficial effects of hyperbaric oxygen (HBO) preconditioning in IRI, so the aim of this study was to examine those effects in renal IRI performed hypertensive conditions.

Methods
Animals were randomly selected in 3 experimental groups: sham-operated group (SHAM, n=7), AKI control group (AKI, n=9) and AKI group with HBO preconditioning (AKI+HBO, n=12). HBO preconditioning was performed by exposing to pure oxygen (2.026 bar) twice a day for two consecutive days for 60 minutes and 24 hours before AKI induction. AKI was induced by removal of the right kidney and atraumatic clamp occlusion of the left renal artery for 45 minutes.

Results
AKI induction significantly decreased creatinine, urea and phosphate clearances. Significant improvement was observed in group with HBO preconditioning. Also, significantly increased KIM-1 plasma levels in AKI group were decreased in AKI + HBO group. HO-1 activity in kidney tissue, in AKI+HBO group was significantly increased compared to AKI group, without difference between SHAM and AKI group. Considering the immunohistochemical expression, HO-1 expression in SHAM group was diffuse with weak intensity on the apical surface of the proximal tubular cells. In AKI group, the expression was moderate and diffuse in the cytoplasm of the proximal tubular epithelial cells, with strong expression in some tubules. In AKI + HBO group, the intensity of HO-1 expression was diffuse, but with weak intensity in the cytoplasm and on the apical surface of the proximal epithelial tubular cells, as previously noticed in SHAM group. Sham-operated rats did not express 4-HNE in any parenchymal structure. AKI induced abundant and strong glomerular expression of 4-HNE along with expression in interstitial compartment. HBO preconditioning significantly decreased 4-HNE expression both in glomeruli and interstitium. Expression of hypoxia inducible factors: HIF 1α, HIF 1α and HIF 2α, in SHAM group were minimal, with expression in glomeruli and interstitium. AKI caused a pronounced and abundant expression in the glomeruli and interstitium. HBO preconditioning significantly reduced the expression of these parameters, restoring to the expression pattern noticed in SHAM group.

Conclusions
Considering our results, even in hypertensive conditions, we can expect protective effects of HBO preconditioning in experimental model of AKI.