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Introduction:
The aim of the study was to investigate into effectiveness of lithium chloride (LiCl) as agent that prevents damage to the monolayer of endothelial cells under the action of serum from multiple trauma patients with septic shock.

Methods:
Serum from 5 pts with septic shock (Sepsis-3) and 5 healthy donors was withdrawn. Monolayer of Ea.hy926 endothelial cells were incubated for 3 hrs at 37 ° C with healthy person’s serum and with septic patient’s serum without LiCl and with it at concentrations of 0.01 mmol, 0.1 mmol, 1 mmol, 10 mmol. LiCl was added 1 hour before the change of serum. After incubation cells were washed and fixed with 2% paraform solution and permeabilized with 1% Triton X-100 solution. Fixed cells were stained with primary antibodies to VE-cadherin and then incubated with secondary antibodies conjugated with Oregon Green 488 fluorescent dye as well as with phalloid red and Hoechst dye 33342. Images were processed by fluorescence microscope and ImageJ 1.44p and MetaVue 4.6 programs. Western blotting was used to detect antibodies to VE-cadherin, claudin and GSK-3beta. Statistics included Mann-Whitney test and chi-square test.

Results:
Incubation of a monolayer of endothelial cells with 5% serum of septic shock patients led to loss of VE-cadherin contacts and decrease of claudine. Preincubation with LiCl 0.01 mmol did not prevent dismantling of claudine, actin, VE-cadherins; 0.1 mmol LiCl prevented it (p>0.05), but at higher concentrations (1 mmol, 10 mmol) almost completely protected endothelial monolayer from destruction of intercellular contacts (p<0.05). Serum had almost no effect on the phospho-GSK-3β level after 5 min, 15 min, 30 min and 1 hr, but caused a significant (60%) decrease in its level after 2 and 4 hrs. LiCl (1 mmol) caused a significant increase in phospho-GSK-3β already 15 mins and up to 4 hrs after exposure.

Conclusion:
LiCl prevents septic damage to the monolayer of endothelial cells in vitro in a GSK-3beta mediated way.