Abstract – Part I (clinical study)

Background: Patients with injuries to multiple organs or organ systems are in a serious risk of shock, multiorgan failure and death. Although there are scoring systems available to assess the extent of polytrauma and guide prognosis, their usefulness is limited by their considerably subjective nature. As the production of nitric oxide (NO) by many cell types is elevated by tissue injury, we hypothesized that serum concentration of NO (and its oxidation products, NOx) represents a suitable marker of polytrauma correlating with prognosis.

We wanted to prove that nitric oxide can serve as an indicator for severity of injury in polytrauma.

Methods: We measured serum NOx and standard biochemical parameters in 93 patients with various degrees of polytrauma, 15 patients with minor injuries and 20 healthy volunteers.

Results: On admission, serum NOx was higher in patients with moderate polytrauma than in both controls and patients with minor injury, and it was even higher in patients with severe polytrauma. Surprisingly, NOx on admission was normal in the group of patients that required cardiopulmonary resuscitation or died within 48 hours after admission. In groups where it was elevated on admission, serum NOx dropped to normal values within 12 hours. Blood lactate levels on admission were elevated in proportion to the severity of subsequent clinical course.

Abstract – Part II (experimental study)

Background: The finding of the practically normal NOx concentrations in fatal polytrauma was very surprising. We tried this fact to explain in experiment. The experimental study was undertaken to test the hypothesis that serum NOx is reduced in severe polytrauma by concomitant overproduction of reactive oxygen species (ROS).

Methods: Polytrauma was induced in rats under anesthesia by bilateral fracture of femurs and tibiae plus incision of the right liver lobe through laparotomy. Serum NOx was measured by chemiluminescence after hot acidic reduction. The role of ROS was assessed by treatment with an antioxidant, N-acetyl-L-cysteine (NAC).

Results: Experimental polytrauma elevated NOx from 11.0 ± 0.7 to 23.8 ± 4.5 ppb. This was completely prevented by NAC treatment (9.1 ± 2.2 ppb).

Abstract – Conclusion:

Elevated serum NOx and blood lactate in patients with polytrauma are markers of serious clinical course, while normal NOx combined with very high lactate may signal fatal prognosis. Serum NOx is elevated in severe polytrauma, and this is not reduced by ROS. On the contrary, ROS are necessary for the NOx elevation. In conclusion, the experimental polytrauma causes elevation in serum NOx concentration that is prevented by antioxidant treatment with N- acetylcysteine. This implies that radical production is necessary for the NOx to rise after polytrauma.