EFFECTS OF PROPOFOL ON LIPID PEROXIDATION AND CELLULAR DAMAGE AGAINST WARM HEPATIC ISCHEMIA/REPERFUSION INJURY IN RATS

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INTRODUCTION: Propofol exerts protective effects against lipid peroxidation and hypoxia/reoxygenation injury of hepatocytes *in vitro*. However, as a complex process in which various mediators and cells interplay causes liver cell injury during ischemia and reperfusion, the results of the *in vitro* studies may not be applicable to an *in vivo* model of hepatic ischemia/reperfusion injury (IRI).

METHODS: With Research Ethics Committee approval, to evaluate whether continuous infusion of propofol (Diprivan[®], AstraZeneca, UK) could protect the liver from warm IRI *in vivo*, livers of male Sprague-Dawley rats were subjected to 60 minutes of partial normothermic ischemia allowing perfusion to right and caudate lobes and subsequent 45 minutes of reperfusion using a microsurgical clip after 30 minutes of stabilization. Either propofol (Propofol group, n = 11, 10 mg/kg/h) or saline (Control group, n = 11, same volume) was continuously administered during the stabilization period and throughout the experiment via the tail vein. At the end of reperfusion blood and liver tissue samples for SGOT and SGPT, lipid peroxidation assay, and histological examination for a morphometric tissue injury scoring system¹ were taken. Additional eight rats were sacrificed for obtaining normal values of SGOT and SGPT. Data were analyzed using Mann-Whitney-U test and ANOVA with *post hoc* Scheffe test, P < 0.05 being significant.

RESULTS: The liver malondialdehyde concentration ($\mu\text{mol/g}$ tissue, mean $\pm$ SD) was decreased in Propofol group (1.39 ± 0.21 (perfused lobes) and 1.85 ± 0.27 (ischemic reperfusion lobes)) compared with Control group (1.97 ± 0.20 (perfused lobes) and 2.39 ± 0.28 (ischemic reperfusion lobes)) (P < 0.01) (Figure 1). Hepatic injury scores, mild in both groups, were decreased in Propofol group compared with Control group (P < 0.01), with no difference of SGOT and SGPT between the two groups.

DISCUSSION: Continuous infusion of propofol before ischemia significantly attenuates lipid peroxidation in the livers subjected to IRI. However, propofol provides only minimal protective effects against cellular damage during the early phase of warm hepatic IRI in rats. These results suggest that propofol is effective mainly in attenuation of oxidant injury during hepatic IRI *in vivo*.

REFERENCE: 1. Hepatology 26: 1513-1520.

Figure 1. Changes of malondialdehyde concentrations in Control and Propofol groups after 45 min of reperfusion. Data were expressed as mean $\pm$ SE. * P < 0.05 vs. perfused lobes; [†]P < 0.01 vs. control perfused lobes; NS, not significant; MDA, malondialdehyde.