

Sphincter of Oddi motility is stimulated by acute intravenous ethanol

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The relationship between ethanol abuse and acute and chronic pancreatitis is well established. However, the role of sphincter of Oddi (SO) motility in the induction of alcoholic acute pancreatitis is still unclear. Previous studies examining the effect of acute ethanol administration on SO motility have produced conflicting results. AIM: To assess the effect of intravenous ethanol on common bile duct (CBD) pressure and SO motility in anaesthetised Australian possums. Methods: Possums were anaesthetised for the duration of experiments (anaesthesia induction intramuscular xylazine (10 mg/kg) and ketamine (20 mg/kg) and maintained a constant intravenous infusion of sodium thiopentone (5-10 mg/kg/h)). In separate animals, ethanol (1g/kg) or saline was infused over a 1h period via the left femoral vein. In separate groups of animals, CBD pressure was measured as an indication of SO spasm (ethanol n=5; saline n=2), blood and bile ethanol concentrations were determined at 30min intervals (n=2) and SO motility was measured by manometry (n=5). Blood pressure was measured continuously in all animals. Results: CBD pressure, SO basal pressure and contractile activity increased progressively during the ethanol infusion with maximal changes of about 2mmHg, 1.5mmHg, two fold respectively at 1h and then declined. Blood ethanol levels peaked at 313 ± 26 mg/100ml by 1h and then declined. Bile ethanol concentrations displayed a very similar profile to blood. Blood pressure was not significantly changed. Conclusion: Ethanol (and/or metabolites) stimulates SO activity to elevate CBD and probably pancreatic duct pressure which could contribute to the onset of acute pancreatitis. The active agents could act via the blood and/or bile. Ethanol in bile could also have down stream effects which contribute to the overall effects of ethanol on biliary and pancreatic physiology.

Supported in part by the NH&MRC of Australia (grant # 229901).