Effects of propofol on mitochondrial function and intracellular cacium shift in bovine aortic endothelial model.

陳瑞明
Chang HC;Tsai SY;Wu GJ;Lin YH;Chen RM and Chen TLP

Abstract

propofol. The purpose of this study is to investigate the effect of propofol on mitochondrial membrane potential and morphology so as to infer its relation with intracellular calcium mobilization in bovine aortic endothelium. Methods : In this study, we used the cultured bovine aortic endothelial cells (Gm 7372a) to elucidate the impact of propofol upon the membrane potential andmorphology of mitochondria in correlation with its effect on intracellular calcium shift. The intracellular calcium mobilization within the cells preincubated with or without propofol was evaluated using a fluorescent spectrophotometer (confocal microscope) after being treated with Fluo-3. The mobilization of intracellular calcium was demonstrated by the appearance of "hot spots" released from intracellular stores after the addition of an ionophore, ionomycin, to the incubation system. Themembrane potential of mitochondriawasmeasured byDiOC6 and the morphology of the mitochondria was evaluated by the treatment of TM Ros and compared with that by the treatment of the uncoupler, FCCP, as control. Results : The release of calcium "hot spots" from the intracellular stores (eg. mitochondria) after the addition of ionomycin was visualized to decrease dramatically within the endothelial cells after preincubation with propofol. The membrane potential of mitochondriawas significantly inhibited by pretreatment of propofol at 0.01 mM, 37°C for 30min. Morphologically, the integrity ofmitochondriawas distortedand fragmented in thepresence of propofol as compared with that of control. Conclusions : Our data showed that propofol in clinical concentration, 0.01mM, could inhibit intracellular calcium shift from the intracellular stores and decrease the membrane potential and distort the morphology of itochondria in bovine aortic endothelial cells. These inhibitions of the function and disfiguration of the morphology of mitochondria signify that the clinical hypotension induced by propofol might be of a potential mechanism.