

# Effects of propofol on mitochondrial function and intracellular calcium shift in bovine aortic endothelial model

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摘要

Abstract

**BACKGROUND:** Hypotension was commonly encountered in clinical practice during induction of anesthesia with 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 and morphology 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. The membrane potential of mitochondria was measured by DiOC6 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 (e.g. mitochondria) after the addition of ionomycin was visualized to decrease dramatically within the endothelial cells after preincubation with propofol. The membrane potential of mitochondria was significantly inhibited by pretreatment of propofol at 0.01 mM, 37 degrees C for 30 min. Morphologically, the integrity of mitochondria was distorted and fragmented in the presence of propofol as compared with that of control. **CONCLUSIONS:** Our data showed that propofol in clinical concentration, 0.01 mM, could inhibit intracellular calcium shift from the intracellular stores and decrease the membrane potential and distort the morphology of mitochondria 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.