Effects of propofol on cyclic strain-induced endothelin-1 expression in human umbilical vein endothelial cells

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Abstract

***Background*:** Since the introduction of propofol into clinical practice, it has become the intravenous induction agent of choice for anesthesia providers. Experimental results revealed that propofol exerted hypotensive and antioxidative effects. However, the intracellular mechanism of propofol remains to be delineated. The aims of this study were to test the hypothesis that propofol may alter strain-induced endothelin-1 (ET-1) secretion and nitric oxide production, and to identify the putative underlying signaling pathways in human umbilical vein endothelial cells.

***Methods*:** Cultured human umbilical vein endothelial cells were exposed to cyclic strain in the presence of propofol, ET-1 expression was examined by Northern blotting and enzyme-linked immunosorbent assay kit. Activation of extracellular signal-regulated protein kinase, endothelial nitric oxide synthase, and protein kinase B were assessed by Western blot analysis.

***Results*:** We show that propofol inhibits strain-induced ET-1 expression. Propofol also inhibits strain-increased reactive oxygen species formation and extracellular signal-regulated protein kinase phosphorylation. On the contrary, nitric oxide production, endothelial nitric oxide synthase activity, and protein kinase B phosphorylation were enhanced by propofol treatment in human umbilical vein endothelial cells. Furthermore, in the presence of PTIO, a nitric oxide scavenger, and KT5823, a specific inhibitor of cyclic guanosine monophosphate-dependent protein kinase, the inhibitory effect of propofol on strain-induced extracellular signal-regulated protein kinase phosphorylation and ET-1 release was reversed.

***Conclusions*:** In summary, we demonstrate for the first time that propofol inhibits strain-induced ET-1 secretion and enhances strain-increased nitric oxide production in human umbilical vein endothelial cells. Thus, this study delivers important new insight in the molecular pathways thatmay contribute to the proposed hypotensive effects of propofol in the cardiovascular system.