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Pranidipine, a 1,4-Dihydropyridine Calcium Channel Blocker that Enhances Nitric Oxide-Induced Vascular Relaxation

Toyoki Mori*, Hiromichi Takase*, Kiyotaka Toide*, Takahiro Hirano*, Toshimi Kambe*, Natsuki Nakayama**, and Arnold Schwartz***

**First Institute of New Drug Research, and*
***Division of Clinical Research & Development,*
Otsuka Pharmaceutical Co., Ltd., Tokushima, Japan;
****Institute of Molecular Pharmacology and Biophysics,*
University of Cincinnati College of Medicine, Cincinnati, Ohio, USA.

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ABSTRACT

Pranidipine, a long acting 1,4-dihydropyridine calcium channel blocker, prolongs nitric oxide (NO)-mediated relaxation of rat aorta; it prolongs acetylcholine-induced relaxation in presence of endothelium as well as nitroglycerin-induced relaxation in absence of endothelium. In rat aorta the effect of pranidipine on NO-mediated relaxation is cyclic guanosine monophosphate (cGMP)-independent, but in guinea pig carotid artery the same effect of pranidipine is cGMP-dependent. It has been reported that in co-cultured human endothelial and smooth muscle cells pranidipine, at a higher concentration (10^{-6} M), enhances vasorelaxant effect of NO by blocking NO decomposition. The enhancement of NO action by pranidipine differs from the direct NO-releasing action of other 1,4-dihydropyridines. It is expected that enhancement of NO-induced vasodilatation will lead to a venodilator action *in vivo* and less peripheral edema. The target organ protective effects of pranidipine are also reviewed in this article.