

## 허혈성 급성 신부전 흰쥐모델에서 산화질소계의 역할

전남대학교 내과학교실<sup>1</sup>, 전남대학교 생리학교실<sup>2</sup>

배은희<sup>1</sup> · 이종은<sup>2</sup> · 마성권<sup>1</sup> · 김인진<sup>2</sup> · 나진희<sup>2</sup> · 김남호<sup>1</sup> · 최기철<sup>1</sup> · 김수원<sup>1</sup>

### Alteration of Nitric Oxide Synthase and Guanylyl Cyclase Activity in Rats Following An Ischemic Insult

Eun Hui Bae<sup>1</sup>, JongUn Lee<sup>2</sup>, Seong Kwon Ma<sup>1</sup>, In Jin Kim<sup>2</sup>  
Jin Hee Na<sup>2</sup>, Nam Ho Kim<sup>1</sup>, Ki Chul Choi<sup>1</sup>, Soo Wan Kim<sup>1</sup>

Department of Internal Medicine<sup>1</sup> and Physiology<sup>2</sup>, Chonnam National University Medical School, Gwangju, Korea

**Purpose** : Nitric oxide (NO) not only regulates blood flow and Na balance, but also has cytotoxic effects. Among the mechanisms underlying the ischemia/perfusion (I/R) injury in the kidney, a role of NO has been suggested. We investigated the expression of different isoforms of NO synthases (NOS) and determined guanylyl cyclase (GC) activity following an ischemic insult in the kidney.

**Methods** : I/R injury was experimentally induced by clamping the both renal pedicle for 40 min in Sprague-Dawley male rats. The renal expression of NOS isoforms was determined by western blot analysis, and the activity of guanylyl cyclase was determined by the amount of guanosine 3',5'-cyclic monophosphate (cGMP) formed in response to sodium nitroprusside (SNP), NO donor.

**Results** : I/R injury resulted in renal failure associated with decreased urine osmolality. The expression of inducible NOS was increased, while that of endothelial NOS and neuronal NOS decreased. The urinary excretion of NO metabolites was decreased. The cGMP production provoked by SNP was decreased in the papilla, but not in glomerulus.

**Conclusion** : These results indicate an altered regulation of NOS expression and guanylyl cyclase activity in I/R-induced nephropathy.