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PIYARAT SREESAWANG : THE LACK OF NITRIC OXIDE ACTIVITY
MODULATES RENAL HEMODYNAMICS AND RENAL HANDLING OF WATER
AND ELECTROLYTES FOLLOWING ISCHEMIA/REPERFUSION IN RAT. THESIS
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The present study was designed to evaluate the correlation between NO activity and kidney function after ischemia/reperfusion (I/R) and to determine the agent(s) that could enhance the recovery of kidney function under this circumstance. The renal hemodynamics as well as renal handling of water and electrolytes were determined using clearance techniques before and after ischemia induced by 20 minutes of renal artery occlusion. The change in urinary NO_x ($\text{NO}_2 + \text{NO}_3$) excretion under various circumstances were measured as the indicators of NO activity. It was found that renal vascular resistance (RVR) was increased by about 6 fold in the first reflow period, whereas renal blood flow (RBF) and glomerular filtration rate (GFR) were decreased by about 77% and 80%, respectively. A reduction in urine output (~25%) and increase in sodium and potassium excretion by 25% also occurred. Without any treatment, all parameters of kidney function mentioned above could revert to normal within 100 minutes. Infusion of either L-arginine, NO donor, or L^G-nitro L-arginine -methyl ester (L-NAME), NO inhibitor, during the reflow period had no effect on the increased RVR, decreased RBF, GFR or the increased sodium and potassium excretion after ischemia. In contrast, infusion of sodium nitroprusside (SNP), an endothelium-independent vasodilator, prevent the changes in kidney function that occurred after I/R. There is a positive correlation between an increase in urinary NO_x excretion and the tubular handling of water and electrolytes but no correlation with renal hemodynamics after I/R. It is concluded that the changes in renal hemodynamics after I/R are only temporary and partly due to the lack of NO activity which can amplify the action of any vasoconstrictor systems that are active under these conditions. This study provides further evidence concerning the effect of SNP on renal handling of water and electrolytes after I/R.