Sodium Nitroprusside Increases Renal Synthesis of cGMP and Reduces Free Water Clearance in Human

C.B.	Claus Brøckner	Nielsen
------	----------------	---------

Department of Medicine and Nephrology C, Skejby University Hospital, Aarhus, Denmark

Claus Brøckner Nielsen, MD, Department of Medicine and Nephrology C, Skejby University Hospital, DK-8200 Aarhus N (Denmark)

Dear Sir,

I read with interest the review article entitled 'Nitric oxide: An inflammatory mediator of glomerular mesangial cells' in volume 64 of Nephron [1]. Recently, we completed a study entitled 'Enhanced renal production of cyclic GMP and reduced free water clearance during sodium nitroprusside infusion in healthy man' [2]. In this study we investigated the effects of sodium nitroprusside (SNP), which is believed to act via a nitric oxide (NO) release [3], on renal hemodynamics, tubular function and the plasma levels of angiotensin II (Ang II), aldosterone (Aldo) and atrial na-triuretic peptide (ANP) and the tubular transport of cGMP. Our results confirm some of the animal studies and extend the understanding of the NO actions in the kidney reviewed in the above-mentioned article [1]. We believe that this is the first human study to show that SNP and thus NO can stimulate renal production of cGMP, and that this effect is associated with intrarenal actions. During a 90-min infusion period of SNP (0.7 µg/kg/min, mean value) or placebo to healthy control subjects, we observed a significant decrease in mean arterial blood pressure (from 89.5 to 81.5 mm Hg) urinary output (from 7.7 to 4.5 ml/min) and free water clearance (from 4.0 to 1.3 ml/min) and plasma level of ANP (from 3.3 to 2.5 pmol/min) together with an increase in the tubular transport of cGMP (from 28.8 to 214.4 pmol/min) indicating an increased production in the SNP infusion group. Heart rate, Ang II and Aldo were also increased, whereas renal haemodynamics, urinary sodium excretion and lithium clearance, a marker for proximal tubular reabsorption of sodium and water, were unchanged.

We concluded that the results could indicate that SNP and thus NO have a direct effect on the distal tubular handling of water and that this effect might be mediated via cGMP release.

References

Pfeilschifter J, Kunz D, Mühl H: Nitric Oxide: An inflammatory mediator of glomerular mesangial cells Nephron 1993;64:518-525.

Nielsen CB, Eiskjær H, Pedersen EB: Enhanced renal production of cyclic GMP and reduced free water clearance during sodium nitroprusside infusion in healthy man. Eur J Clin Invest 1993;23:375-381.

Smith RP, Kruszyna H: Nitroprusside produces cyanide poisoning via a reaction with haemoglobin. J Pharmacol Exp Ther 1974; 191: 557-563.

0028-2766/94/
0682-0273\$8.00/0