

## **THE EFFECT OF NITRIC OXIDE ON HUMAN INTRAUTERINE ARTERY RESPONSES TO VASOPRESSIN**

ANNA KOSTRZEWSKA

Department of Biophysics, Medical Academy of Białystok, 15-230 Białystok, Poland

Nitric oxide (NO) is an endogenous vasodilator synthesised by the vascular endothelium and smooth muscles. It has been suggested that NO plays a role in the modulation of the vascular action of arginin vasopressin (AVP).

The aim of the present study was to investigate the involvement of endogenous NO in the regulation of human intrauterine artery responses to AVP and the effect of an NO-donor (sodium nitroprusside) on AVP-evoked contraction of these arteries.

Uterine arteries (diameter 0.6 – 1.2 mm) were obtained from non-pregnant women, aged 41– 51 years, undergoing hysterectomy for benign gynaecological disorders. The local ethical committee approved the study.

Experiments were performed on artery rings without endothelium. The endothelium was removed mechanically by gently rubbing the intimal surface with a stainless-steel wire.

Artery responses to AVP were recorded under isometric conditions.

The incubation (15 min) of artery rings with an NO synthase blocker (L-NNA) resulted in an increase in the basal tone of the muscles. In the presence of L-NNA, the responses of the human intrauterine arteries to vasopressin were also significantly enhanced. Sodium nitroprusside (SNP) caused a slight yet statistically significant decrease in the muscle basal tone. Pre-treatment with SNP did not influence the artery responses to AVP. However, administered on pre-contracted tissues, SNP caused a concentration – dependent relaxation of the artery rings. Apamin, a blocker of small conductance  $Ca^{2+}$  dependent potassium channels significantly attenuated the relaxant effect of SNP.

In conclusion, endogenous NO is involved in the modulation of responses of human intrauterine arteries to AVP. Pre-treatment with SNP does not influence the sensitivity of human intrauterine arteries to AVP. Apamin – sensitive potassium channels are involved in the SNP-induced relaxation of human intrauterine arteries pre-contracted with AVP.