Centrally applied vasopressin prevents posthemorrhagic hypotension in WKY rats

Marcin Ufnal and Tymoteusz Żera
Department of Clinical and Applied Physiology, Medical University of Warsaw, 26/28 Krakowskie Przedmieście St., 00-927 Warsaw, Poland, E-mail: tyzer@amwaw.edu.pl

INTRODUCTION AND METHODS. Increasing evidence indicates that the intrabrain vasopressinergic system plays an essential role in regulation of the cardiovascular system. Previous findings have provided evidence that centrally applied vasopressin (AVP) may produce both pressor-tachycardiac and hypotensive-bradycardiac responses, acting through V1 receptors located in circumventricular organs (1, 3). It has been shown previously that hemorrhage increases the intrabrain release of vasopressin. In spontaneously hypertensive rats (SHR) the intracerebral level of AVP is decreased compared to their maternal strain Wistar-Kyoto rats (WKY) (2). The present study reports that in WKY rats intraventricular administration of AVP during hemorrhage elicits tachycardia and reduces hypotension. Male, 12-14 week-old WKY rats, were used for the experiment. The rats were subjected to the two surgical procedures performed under chloride hydrate anesthesia (360 mg/kg i.p.). During the first surgery a cannula was implanted into the left cerebral ventricle (LCV) (1). After the recovery period of 6 days, the abdominal aorta was catheterized for blood pressure measurements and blood withdrawal. The experiments were performed on freely moving rats one day after the catheterization (1). Animals were divided into two groups; control group (n = 6) subjected to LCV infusion of artificial cerebrospinal fluid (5 μl/h, aCSF), and experimental group (n = 6) subjected to LCV infusion of AVP (100 ng/h). After 40 min of LCV infusion the arterial hemorrhage was performed (1.3% of body weight, 2.5 ml/min) (1). Mean arterial blood pressures was recorded during initial 40 and 30 min after the hemorrhage. Heart rate (HR) was calculated from the heart rate period (HRp).

RESULTS AND DISCUSSION. Under control conditions the WKY rats subjected to LCV infusion of aCSF responded with nonsignificant changes of HR (Fig. 1 A) which were associated with a significant decrease of blood pressure (Fig.1 B). In contrast, the animals receiving LCV infusion of AVP manifested a significant increase of HR (Fig. 1 A) and only slight change of MAP (Fig. 2 B). However, the difference of MAP was insignificant between groups. Lack of difference in changes of MAP between control and AVP groups in presence of significant increase of HR in the latter group indicates that the intrabrain AVP may potently modulate hemodynamic adjustment to hemorrhage. It appears that excessive fall in blood pressure in AVP experiments is to great extent prevented by improvement of cardiac output while in control experiments it is achieved mainly by an increase of the peripheral resistance. Therefore it is likely that the subpressor dose of AVP may promote enhanced activation of the cardiac component of the sympathetic system during hemorrhage.

Fig. 1. Effect of LCV infusion of AVP on heart rate (A) and blood pressure (B). H, hemorrhage; HR, heart rate; MAP, mean arterial pressure. Difference between groups; *, P<0.05; **, P<0.01. Difference from prehemorrhagic value; +, P<0.05; ++, P<0.01; ++++, P<0.001 by ANOVA.


Accepted 14 January 2002