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Title : BLOCK OF CHRONOTROPIC RESPONSE TO CONTROLLED HYPOTENSION

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Introduction. The use of sodium nitroprusside (SNP) is often associated with an increase in heart rate (1). The objective of the present study was to investigate the relative importance of heart rate and left ventricular stroke volume for the regulation of cardiac output during controlled hypotension with SNP. Accordingly, we studied the cardiovascular effects of a derivative of clonidine, a new substance with selective negative chronotropic action (2), on the hemodynamic responses during SNP-infusion.

Methods. 22 patients, 29 to 65 years of age, were studied when they underwent plastic surgery. Each patient was informed about the nature and the risk of the proposed investigation and informed consent as well as institutional approval were obtained. All patients had normal cardiopulmonary findings on clinical examination. Arterial and pulmonary artery pressures were measured by means of catheters in the radial and pulmonary artery and monitored and registered synchronously with the ECG and the heart rate. Cardiac output was determined using the thermodilution method. Cardiac rate was decreased by a bolus injection of 2-{N-allyl-N-(2,6-dichlorophenyl)-amino}-2-imidazoline, St 567, 0.5 mg/kg i.v. Measurements were made before and after induction of neuroleptanesthesia, during SNP-infusion (3.0±1.8 µg/kg i.v.) and at 10 min intervals after administration of St 567 (n = 7). In 15 patients, St 567 was given before infusion of SNP. Statistical analysis was performed by means of the analysis of variance.

Results. SNP decreased mean arterial pressure by 30% from 100 ± 10 mmHg, mean pulmonary artery pressure by 22% from 15 ± 2 mmHg, cardiac output by 10% from 5.9 ± 0.6 l/min and calculated total peripheral vascular resistance by 21% from 1296 ± 207. Heart rate rose to 20% above control (97 ± 20 beats/min). All these changes were significant ($p<0.05$).

St 567 during infusion of SNP decreased heart rate to the pre-infusion level. No changes in cardiac output, blood pressure and pulmonary artery pressure were encountered. However, left ventricular stroke volume was increased by 43% from 48 ± 10 ml ($p<0.05$).

In those patients who received St 567 before SNP, controlled hypotension failed to elicit changes in heart rate.

A significant correlation ($p<0.01$) was found between the initial cardiac rate and the maximum decrease after St 567.

Discussion. While the use of SNP is generally held to be a safe method for inducing controlled hypotension, the secondary increase in heart rate may necessitate the use of larger doses of SNP. Moreover, tachycardia during controlled hypotension has been found to be indicative of the onset of tachyphylaxis (1). The principal finding of the present investigation is that tachycardia does not play an important role for the maintenance of an adequate cardiac output during infusion of SNP. Second, a derivative of clonidine can be used to avoid or to abolish the secondary increase in heart rate as it occurs with controlled hypotension. Third, the negative chronotropic effects of St 567 are a linear function of the initial cardiac rate. Thus, St 567 may exert beneficial effects during controlled hypotension with SNP.

References.

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