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"Vasodilator effects of β-agonist Isoprenaline in Doxorubicin-induced model of heart failure "

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Abstract:

We investigated the vasodilatory effect of isoprenaline at large vessels (aorta, renal ans saphenous arteries, vena cava, renal and saphenous veins) in doxorbicin-induced model of heart failure. Thirty saline-treated (normal group) and thirty doxorubicin treated rabbits (1 mg/kg administered intravenously twice weekly for 8 weeks) were studied after 16 weeks of treatment. Chronic heart failure was confirmed by histopathology. Arteries and veins were cut as rings and so bathed in Krebs maintained at 37°C and gassed with 95% O2 and 5% CO2. Then all tissues were placed under different resting tensions and allowed to equilibrate for 1 hour. Then all the tissues were contracted with U-46619 (0.1 μM) nearly ten minutes before initial applications of isoprenaline. When the U-46619 (0.1 μM). induced contraction reached a plateau, concentration-response curves to isoprenaline were obtained. Isoprenaline was chosen as vasodilator resulting from stimulating beta-receptors in blood vessels. Maximum effect (Ema) and median effective concentration (EC50) were determined from each concentration-response curve and pD2 was calculatd as-log (EC50). Isoprenaline induced relaxations in all vessels. Aorta and renal artery were the most sensitive ones and had the maximum relaxations (15-20%). In relaxation due to β-adrencoeptor agonist isoproterenaol, the aorta and renal artery were the most sensitive vessels. Compared with control, in doxorubicin treated rabbits, Emax of isoprenalin was not modified in all the studied vessles. Relaxation responses were negligible and maximum responses in vena cava, and renal vein were only-5-10 percent. Of all vessels there was no significant difference between control and doxorubicin induced of heart failure in response to isoprenaline.

Keywords:

Doxorubicin ، Heart failure ، Isoprenaline ، Vasodilators

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