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Effects of vardenafil, a selective phosphodiesterase-5-inhibitor, on cardiovascular function in a rat model

Sivakkanan Loganathan, Tamás Radovits, Iwan R Wirotanseng, Gábor Veres, Siegfried Hagl and Gábor Szabó*

Address: Department of Cardiac Surgery, University of Heidelberg, Germany

Email: Gábor Szabó* - Gabor.Szabo@urz.uni-heidelberg.de

* Corresponding author

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Background

Phosphodiesterase-5-inhibitors are vasoactive drugs used in the treatment of erectile dysfunction and pulmonary hypertension. Their beneficial effects in the pulmonary vasculature are widely described, but it remained unclear, how they influence cardiac performance. In the present study we investigated the effects of the phosphodiesterase-5-inhibitor vardenafil on myocardial and vasomotor function in a rat model.

Methods

We performed left ventricular pressure-volume analysis in young adult rats by using a Millar microtip conductance catheter. Pressure-volume loops were recorded before and after a single iv. injection of vardenafil (3, 10, 30, 100, 300 μg/kg, n = 6/group) and myocardial contractility parameters (E_{max} (slope of ESPVR), PRSW, +dP/dt/EDV) were calculated. Furthermore, in vitro organ bath experiments with isolated aortic rings of the treated rats were performed to investigate endothelium-dependent (using acetylcholine) and -independent (using sodium nitroprusside) vasorelaxation.

Results

Treatment with vardenafil resulted in a significant dose-dependent increase in the load independent cardiac contractility parameters reaching its maximum at the dose of 100 μg/kg (ESPVR: 2.15 ± 0.15 vs. 3.29 ± 0.26 mmHg/μl; PRSW: 93.28 ± 4.04 vs. 134.90 ± 6.27 mmHg; +dP/dt/EDV: 38.73 ± 7.97 vs. 53.02 ± 3.74 mmHg/s/μl, $p < 0.05$). Results of in vitro organ-bath experiments showed a dose-dependent improvement in the vasorelaxation of aortic rings after vardenafil-treatment.

Conclusion

Our current results show that the phosphodiesterase-5-inhibitor vardenafil improves myocardial contractility and vasodilatory functions. Therefore it may represent a novel therapeutic approach in the treatment of acute heart failure.