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Relationship between autonomic activity, beta receptors and catecholamines after sympathectomy

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Background: Different approaches of autonomic modulation have been proposed in the treatment of HF. However, the knowledge of the physiological effects of the drugs used upon the heart (no diseased model) is limited.

Purpose: The aim of this study was to compare the effects of sympathectomy by chemical ablation of the stellate ganglion regarding mainly to heart physiology.

Material and methods: 75 Wistar rats were divided into 5 groups: control (CT), left unilateral sympathectomy (UNI), bilateral sympathectomy (BIL), left unilateral sympatholysis + atenolol (UNI A) and atenolol without sympathectomy (CT A). Thoracic sympathectomy was performed by percutaneous chemical ablation of the stellate ganglion under isoflurane anesthesia. All procedures were performed in accordance with guidelines established and approved by the Ethical Animal Use Committee of the University of São Paulo according the rules of Brazilian College of Animal Studies and Universities Federation for Animal Welfare. Clinical confirmation was assessed by observing ipsilateral non-reversible ptosis.

After six weeks we evaluated autonomic nervous system, myocardial catecholamines, beta receptors and maximal exercise test parameters. It was achieved using treadmill test analyzing rest and peak maximal heart rate, mean arterial blood pressure (carotid artery cannulated), heart rate variability using heart rate (HR) and systolic blood pressure (SPB) measured in the time and frequency domain. The β1 and β2 receptor densities, and the myocardial β-adrenoreceptors, receptors, peroxidase and myocardial catecholamines were obtained by RT-PCR, ELISA and High Performance/Liquide Chromatography, respectively.

Results: The BIL group presents basal tachycardia immediately after the exercise test and still more tachycardia at peak exercise. (CT: 387±7 x BIL: 46±22 bpm, p=0.0234). The same pattern regarding blood pressure was observed (CT: 104±13 x BIL: 121±8 mmHg, p=0.0365). The variables related to autonomic modulation have no statistical significance except the high frequency (HF) variable which had significant differences with all groups except UNI. We observed that sympatholysis had no statistical significance except the high frequency (HF) variable which had significant differences with all groups except UNI. We observed that sympatholysis + atenolol (UNI A) and atenolol without sympathectomy (CT A).

Conclusions: These findings suggest an extra cardiac compensatory pathway raising the sympathetic tone and keeping higher HR, higher levels of peripheral norepinephrine and downregulation of beta receptors in the procedure group. The increase in HF activity can be interpreted as an attempt to increase the parasympathetic tone and to balance the greater sympathetic activity raising the sympathetic tonus and keeping higher HR, higher levels of peripheral norepinephrine in BIL group (CT: 1.08±1.22 ng/ml × BIL: 3.94±1.73 ng/ml, p=0.0001).

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Array comparative genomic hybridization as the first-line investigation for neonates with congenital heart disease

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Background: The purpose of this study was to analyze the results of array comparative genomic hybridization (aCGH) performed in neonates with congenital heart disease (CHD) in a tertiary hospital and investigate the advantages and disadvantages of verifying genetic abnormality using aCGH immediately after diagnosis of CHD.

Methods: Among neonates under the age of 28 days who underwent echocardiography from January 1, 2014 to April 30, 2016, neonates whose chromosomal and genomic abnormalities were tested using aCGH in cases of an abnormal finding on echocardiography were enrolled. Their medical records were reviewed.

Results: Two hundred and forty-seven neonates underwent echocardiography during the research period. Of the patients diagnosed with CHD, 81 underwent a-CHD and 11 patients (11/81, 13.5%) had abnormal findings on aCGH. 22q11.2 deletion syndrome was the most common test result with each being found in four patients. On the first a-CHD, four patients were negative (4/81, 5%). Three of them were finally diagnosed with Williams syndrome using fluorescent in-situ hybridization (FISH), one patient was diagnosed with Noonan syndrome through genetic sequencing of them exhibited diffuse pulmonary artery branch hypoplasia, as well as increased velocity of blood flow, on repeated echocardiography. Five patients started rehabilitation therapy at mean 6 months old in outpatient clinics and seizures were suspected in two patients due to their abnormal movements or seizing. Two patients (22q11.2 deletion syndrome and Patau syndrome) parents refused treatment due to the anticipated prognosis.

Conclusions: Screening tests for genetic abnormalities using aCGH in neonates with CHD has the advantage of early diagnosis of genetic abnormality during the neonatal period in which there is no obvious symptom of genetic abnormality. Furthermore, early rehabilitation therapy was feasible as the neurologist prognosis could be predicted. However, there are disadvantages that some genetic abnormalities cannot be identified on aCGH.