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Theme: The changing face of cardiothoracic and vascular anesthesiology

BOOK OF ABSTRACTS

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II – 3 (059)

PRE- AND POST-CONDITIONING EFFECTS OF MILRINONE AGAINST MYOCARDIAL STUNNING IN THE SWINE

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Myocardial stunning is defined as the prolonged contractile dysfunction following an ischemic episode that does not result in necrosis. The present study was carried out to clarify the cardioprotective effect against myocardial stunning of milrinone administered before ischemia or just after reperfusion in anesthetized open-chest swine. Twenty-eight swine were subjected to 12-min ischemia followed by reperfusion to make myocardial stunning. Myocardial contractility was assessed by percentage segment shortening (%SS). Group A (n = 9) received intravenous milrinone at a rate of 5 μg/kg/min for 10 min followed by 0.5 μg/kg/min for 10 min until 30 min before coronary occlusion. Group B (n = 7) received the same dose of milrinone starting at 1 min after reperfusion. Group C (n = 12) received saline in place of milrinone. Five swine in group C and 2 swine in group A had ventricular fibrillation or tachycardia after reperfusion, and thus they were excluded from further analysis. There were no significant differences in systemic hemodynamics, or coronary blood flow among the groups throughout the time course. The percentage changes of %SS from baseline at 90 min after reperfusion in groups A and B were 78 ± 9% and 79 ± 7%, respectively, which were significantly higher than that in group C (43 ± 13%). We conclude that milrinone administered before ischemia or just after reperfusion provides cardioprotection against myocardial stunning.

II – 4 (073)

VASCULAR STIFFNESS, VISCOSITY, AND INERTIA IN THE PATIENTS WITH CARDIOVASCULAR DISEASE

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Introduction: In the patient with cardiovascular disease, vascular structure and function are both damaged to render blood pressure unstable during anaesthesia. We have previously proposed a vascular impedance measurement, which showed drastic change along sympathetic stimulus. In this study, we investigated these vascular impedance parameters during anaesthesia in the patients with or without cardiovascular disease.
Methods: Thirty patients scheduled for neurosurgery were enrolled and divided into 2 groups with (group D, n=15) or without (group N, n=15) cardiovascular disease. Electrocardiogram, radial arterial pressure, and plethysmograph were computed to analyze vascular impedance: stiffness (K), viscosity (B) and inertia (M). These parameters were measured before and after anaesthesia induction on-line with a personal computer.
Results: Prior to anaesthesia, mean arterial pressure (MAP) was higher in group D, and anaesthesia reduced MAP in both groups to the comparative level. K, B and M before anaesthesia were almost equal in both groups, and were reduced after anaesthesia. However, K (10.7+/-4.85 vs 6.59+/-3.34%mmHg-1), B (0.97+/-0.62 vs 0.54+/-0.25%s-1mmHg-1), and M (0.06+/-0.04 vs 0.03+/-0.03%s-2mmHg-1) after anaesthesia were higher in group D. These changes in K, B and M were greater in group N, while MAP changes were greater in group D.
Conclusion: Reduced range and elevated value in vascular impedance during anaesthesia induction may represent the impairment in regulation in vascular constriction/dilatation in cardiovascular disease. Vascular dysfunction could be anticipated using vascular impedance measurements during anaesthesia.
Reference:
1. A Sakane, et al: Discrimination of vascular conditions Using a probablistic Neural Network, J Robotics and Mechatronics, 16(2), 138-45, 2004.