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Abstracttitel: THE EFFECT OF PROTAMINE ON PULMONARY ARTERY PRESSURE DURING
NEUTRALIZATION OF HEPARIN

Background: Heparin-protamine(H-P) complexes produce both prostacyclin(PGI₂) which dilates vascular smooth muscle and thromboxane A₂(TxA₂) which constricts vascular smooth muscle during neutralizing heparin on cardiothoracic anaesthesia. The imbalance between these products causes untreatable hypotension due to catastrophic pulmonary vasoconstriction(PV). We experienced this fatal PV before. However, this untreatable hypotension occurs very rare. We hypothesized that the protamine injection intravenously to neutralize heparin after discharging cardiopulmonary bypass(CPB) induces temporary PV without severe hemodynamic changes.

Methods: Retrospectively, we investigated 21 adult patients without pulmonary hypertension, each of ASA physical status III, for elective cardiovascular surgery at the Saitama Cardiovascular and Respiratory Center, Saitama, Japan. All patients were induced with midazolam 0.05mg/kg, vecuronium bromide 0.1mg/kg, fentanyl 0.05mg/kg intravenously, and maintained with sevoflurane/fentanyl/propofol. We administered heparin at 300U/kg for anticoagulation before CPB established. After CPB, protamine was injected at 3.9mg/kg intravenously for the neutralization. An activated clotting time was ensured more than 400 seconds after heparin administration, less than 150 seconds after protamine injection. (The case which needs second injection of heparin or protamine was excluded.) Dopamine, dobutamine, milrinone, and nitroglycerin were used for hemodynamic stability. We analyzed the means of heart rate, radial artery pressure, pulmonary artery pressure(PAP) 10 minutes before and 20 minutes after protamine administration by paired-t test. $P < 0.05$ was considered statistically significant.

Results: Only the changes of PAP(systolic, mean, diastolic). revealed a statistically significant. ($P=0.0001, 0.0029, 0.0202$)

Conclusion: Protamine administration to neutralize heparin after CPB induces temporary PV without severe hemodynamic changes during cardiothoracic anaesthesia.