A983

DOES LOW DOSE APROTININ REDUCE BLOOD LOSS FOLLOWING CARDIOPULMONARY BYPASS?

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Introduction: The blood saving effects of aprotonin (Apt) in cardiac surgery have been previously reported (1). Apt improves the platelet's hemostatic activity by preserving the adhesive platelet receptor GPIIb. However, to date the optimal dose of Apt has not yet been established. The aim of this study was to evaluate the effect of two therapeutic regimens: Low dose Apt(LDA) vs High dose Apt(HDA). This latter corresponds to the classical dose suggested in the literature.

Patients and Methods: After ethical approval and informed consent, 15 patients(pts) undergoing cardiac operation (7 CABG, 8 valve replacements) were included in a randomized double blind study. Three groups were individually assigned:

Group I (n=5) HDA: Pts received a bolus of 2.10^6 KIU Apt at induction of anesthesia followed by 1ml/kg of saline before cannulation. Continuous infusion of 0.510^5 KIU/hour of Apt were administered during the whole operative period. In addition 2.10^6 KIU Apt were added in the pump prime solution.

Group II (n=5) LDA: Pts received saline at induction, 25. 10^3 KIU Apt/kg at cannulation and saline at all other corresponding times. Group III (n=5) Placebo: No Apt was administered and equivalent volumes of saline were injected at all corresponding times. Several parameters were analyzed:

1) Blood loss was measured intraoperatively from deccanulation till skin closure and postoperatively until drains were withdrawn.
2) Platelet count(PC) was obtained at different times: To (after induction) T1 (10 minutes after starting cardiopulmonary bypass (CPB) T2 (during deccanulation) T3 (90 minutes after skin closure) T4 (24 hours postoperatively) and T5 (4 days postoperatively).
3) Bleeding time was measured at T0 and T3. Statistical analysis was performed using Kruskall-Wallis, Mann-Whitney, Chi-square and Anova. Values are means ± S.E.

Results: Patients' data of the three groups were comparable. Total postoperative blood loss was significantly lower in groups I and II compared to group III (Table). However, no statistical difference could be noted between pts in group I and group II. Although the decrease of PC was identical in the 3 groups, BT at T3 was significantly shorter in groups I & II compared to group II: 53±27, 47±3 and 84±22 seconds respectively.

In Conclusion, this double blind randomized study confirms the blood saving effects of Apt and establishes that a lower dose than that classically recommended is effective in decreasing blood loss following cardiac surgery.

BLOOD LOSS Inteperative Postoperative Total

<table>
<thead>
<tr>
<th>Group</th>
<th>Value (ml)</th>
<th>Value (ml)</th>
<th>Total (ml)</th>
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<tbody>
<tr>
<td>I</td>
<td>203 ± 9*</td>
<td>336 ± 43</td>
<td>539 ± 103*</td>
</tr>
<tr>
<td>II</td>
<td>322.5 ± 76</td>
<td>397.5 ± 33*</td>
<td>720 ± 65*</td>
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<tr>
<td>III</td>
<td>512 ± 109</td>
<td>675 ± 74</td>
<td>1 187 ± 77</td>
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* p < 0.05 compared to placebo (Group III) values.

References:

A984

TITLE: INHALED NITRIC OXIDE (NO) IN PULMONARY HYPERTENSION FOLLOWING MITRAL VALVE REPLACEMENT

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The purpose of this study was to determine the hemodynamic effects of NO, believed to be Endothelin Derived Relaxing Factor (1). We thus investigated the vasodilating effect of inhaled NO in patients with pulmonary artery hypertension.

Six patients underwent mitral valve replacement for mitral stenosis and presented with postoperative mean pulmonary arterial pressure over 25 mmHg. They had given informed consent and the protocol was approved by the Ethical Committee, Claude Bernard Univ., Lyon and Univ. Hosp., Lyon. Monitored parameters included heart rate (HR), mean systemic arterial pressure (MAP), systolic (SPAP), diastolic (DPAP) and mean (MPAP) pulmonary arterial pressures, pulmonary capillary wedge pressure (PCWP) and central venous pressure (CVP). Thermodynamics cardiac index (CI), systemic (SVR) and pulmonary (PVR) vascular resistances, arterio-venous oxygen content difference (AVDO2), oxygen delivery (O2 DEL) consumption (O2 CONS) and extraction (O2 EXT) were calculated. Under mechanical ventilation (FiO2 = 0.5), NO inhalation (40 ppm) was maintained during 10 minutes. Data were recorded before inhalation (T1), after 10 min of NO inhalation (T2), and 30 min after the end of inhalation (T3). In all patients, the hemodynamic response to inhaled NO consisted of transient decrease in SPAP, DPAP, MPAP and PVR with an increase in mixed venous O2 saturation (SVO2) (table). In the same time, we noted an increase in O2 DEL and a decrease in AVDO2 and O2 EXT. Methemoglobin levels were studied in all patients and were found to be below 1%.

Inhalation of a 40 ppm concentration of NO causes pulmonary artery vasodilation and hemodynamic improvement in patients with pulmonary artery hypertension following mitral valve replacement. The absence of effect on systemic arterial pressure preserves right coronary perfusion pressure.


<table>
<thead>
<tr>
<th>TABLE: HEMODYNAMIC EFFECTS OF NO (40 ppm)</th>
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<tbody>
<tr>
<td>T1</td>
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<tr>
<td>-----</td>
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<tr>
<td>HR (bpm)</td>
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<tr>
<td>MAP (mmHg)</td>
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<td>SPAP (mmHg)</td>
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<td>DPAP (mmHg)</td>
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<td>MPAP (mmHg)</td>
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<tr>
<td>CI (l/min/m²)</td>
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<tr>
<td>02 (bVol%)</td>
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<tr>
<td>02 (bVol%)</td>
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<tr>
<td>02 CONS (m/min/m²)</td>
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<tr>
<td>02 EXT (%)</td>
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</tbody>
</table>

Data are expressed as mean ± SEM and analyzed using Wilcoxon test; p < 0.05 was considered significant (* versus T1; ** versus T2).