Title : **EFFECTS OF PROLONGED ALMITRINE INTRAVENOUS INFUSION ON GAS EXCHANGE AND HEMODYNAMICS IN ARDS PATIENTS.**

Authors: **P Plaisance MD, JF Loriferne MD, C Brun-Buisson MD, F Lemaire MD**

Affiliation : ICU H. Mondor Hospital. Créteil. and Anest. Dept Univers. Lariboisière Paris.

**Introduction** : Short time (30 min) IV infusion of Almitrine(A) induces a rapid increase of pulmonary arterial pressure (PAP) and an improvment of gas exchange in ARDS patients. Its was speculated that PAP increase recruits pulmonary vessels, allowing to reduce $\dot{V}A/\dot{Q}$ mismatching. This study was designed 1) to know if A has a sustained effect during long term infusion ; 2) if it is the case, is there a relationship between increases in PAP and $PaO_2$ ?

**Materials and methods** : 10 ARDS patients, mechanically ventilated ($FiO_2$ .4 to .85) were studied. After 2 hrs of hemodynamic and ventilatory stability, A was infused (8-16 mcg/kg/min for 60 min followed by 2-4 mcg/kg/min) during 16.4 ± 10.5 hrs. Eventual adjuvent vasoactive drugs (dopamine) and ventilatory patterns were maintained constant. Just before (baseline) and 60 min, 120 min, 22 hrs (n=6) after the beginning of the infusion, the following variables were measured or calculated: 1) arterial and venous blood gases (arterial and Swan-Ganz catheters), venous admixture ($\dot{Q}VA/\dot{Q}T$); 2) pulmonary and systemic hemodynamic parameters; 3) right ventricular ejection fraction (RVEF) in 3 patients (RVEF Swan-Ganz catheter). Statistics were performed by analysis of variance.

**Results** : There was an immediate and significant increase in $PaO_2$ during A infusion. $\dot{Q}VA/\dot{Q}T$ decreased unsignificantly. These effects persisted in the 6 patients who were still under perfusion at the 22nd hour. Cardiac index (CI) and systemic hemodynamic parameters (including RVEF) remained unchanged. Mean PAP transiently increased during the 1st hr. and then returned to its baseline value.

| Mean ± SD | N | Baseline | T 60' | T 120' | T 22 h |
|---|---|---|---|---|---|
| $PaO_2$ | 10 | 85.9±23.6 | 105.7*±28.7 | 103.7*±27.5 | |
| (mmHg) | 6 | 85.1±19.1 | | | 104.3*±25 |
| $P\bar{v}O_2$ | 10 | 38.9±8.9 | 43.3±9.9 | 39.7± 9.7 | |
| (mmHg) | 6 | 37.8±5.9 | | | 38.6 ±8.2 |
| $\dot{Q}VA/\dot{Q}T$ | 10 | 38.2±16.8 | 30.7±9.9 | 31.6±10. | |
| (%) | 6 | 34.3±14.1 | | | 27.1±10 |
| $\overline{PAP}$ | 10 | 23.4±6.5 | 26.1±7 | 24.4±4.7 | |
| (mmHg) | 6 | 24.4±6 | | | 23.5±3.9 |
| CI | 10 | 4.7±2.2 | 5±2.4 | 4.9±2.6 | |
| (l/min/$m_2$) | 6 | 4.3±1.5 | | | 3.7±1.1 |

*:p<.001 vs control.

**Discussion** : Whatever CI and $P\bar{v}O_2$ variations during the study period and although PAP returned to control, $\dot{Q}VA/\dot{Q}T$ did not change whereas $PaO_2$ increase lasted during 22 hrs. The mechanism by which continuous A infusion improves $PaO_2$ remains to be elucidated.



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Title: Acetyl-salicylic acid reverses the nitroglycerin-induced fall in $PaO_2$ in anethetized obese patients

Authors: H.In-nami,M.D.,M.Takasaki,M.D., T.Kawaguchi,M.D.,K.Okada,M.D.

Affiliation: Anes. Dept., Teikyo University School of Medicine,Tokyo 173

Introduction:Nitroglycerin(NTG) is known to cause a fall in $PaO_2$. Release of hypoxic pulmonary vasoconstriction(HPV) has been suggested as the underlying mechanism(1).Although acetyl-salicylic acid(ASA) has been reported to potentiate HPV(2), it is not clear whether ASA reverses the NTG-induced fall in $PaO_2$ in anesthetized patients. The present study was undertaken to investigate whether ASA reverses the NTG-induced impairment in gas exchange in obese patients.

Methods: Eighteen concenting obese patients undergoing tympanoplasty were studied. Anesthesia was maintained with $N_2O$-$O_2$-enflurane and ventilation was controlled(IPPV). $FiO_2$( 0.4), Vt(10 ml/kg) and f(12/min) were kept constant throughout the study. After a stabilization period, blood gas was measured( control stage) and a NTG infusion(1.0 $\mu$g/kg/min) was started. At 40 min after the start of NTG infusion, blood gas measurement was made(stage A). Then, ASA dissolved in saline solution was administered(10 mg/kg) to nine patients(group I), while to the other nine patients(group II) the same volume of saline solution without ASA was administered. At 40 min after the i.v. injection of ASA or plain saline solution, blood gas was determined( stage B). The Student's t test was used to analyze the results.

Results: The NTG infusion produced significant decreases in mean arterial pressure(MAP ) and in $PaO_2$ both in the group I and II. However, $PaO_2$ increased significantly from 91 ± 15 to 106 ± 15 mm Hg after the administration of ASA, while no significant change in $PaO_2$ was observed after the injection of plain saline solution. $PaCO_2$ and pH did not change with the NTG or ASA administration.

Conclusion: The present study demonstrated that ASA reversed partially the NTG-induced decrease in $PaO_2$ in anesthetized obese patients. Since ASA is known to potentiate HPV, the reversal of the NTG-induced fall in $PaO_2$ is likely due to a potentiation of HPV.

Table

| | | Control | NTG | |
|---|---|---|---|---|
| | | Stage | Stage A | Stage B |
| MAP | G. I | 87 ± 6 | 64 ± 6 * | 65 ± 8 * |
| (mm Hg) | G.II | 85 ± 7 | 68 ± 6 * | 67 ± 7 * |
| $PaO_2$ | G. I | 134 ± 21 | 91 ± 15* | 106 ± 15 * @ |
| (mm Hg) | G.II | 131 ± 22 | 89 ± 15* | 94 ± 19 * |

*:p<0.05(vs Control),@:p<0.05(vs Stage A)

References: 1. Am J Med 65: 911, 1978.
2. J Appl Physiol 45: 33, 1978.