Title : ANTIDIURESIS DURING PEEP IS NOT MEDIATED BY AN INHIBITION OF ATRIAL NATRIURETIC FACTOR RELEASE

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INTRODUCTION : The observed antidiuresis and antinatriuresis during positive end expiratory pressure (PEEP) has been related to changes in cardiovascular hemodynamics and consequent impairment of renal function[1]. However, the mechanisms involved remain debated. The physiological control and the renal effects of the recently discovered atrial natriuretic factor (ANF) lead to the idea that the inhibition of its secretion during PEEP could account for antidiuresis and antinatriuresis[2]. This study was designed to test this hypothesis in patients demonstrating a normal physiological ANF response to right atrial distension.

MATERIAL AND METHODS : 8 patients (44 ± 15 SD yrs) were studied 6 hours after cardiac surgery for valvular replacement. Criteria for inclusion were an angiographically pre-operative left ventricular ejection fraction over 45% and a post-operative hemodynamic stability with a diastolic arterial pulmonary pressure of less than 20 mmHg, without any cardiovascular agent. Anesthesia (high dose fentanyl, 75 mcg/kg), cardiopulmonary bypass management (partial hemodilution, moderate hypothermia, cardioplegic arrest) were similar for all patients.

Parameters : pleural pressure variations were measured using the oesophageal balloon technique, the position of the balloon being controlled by a chest X Ray and the validity of the pressure tracing during mechanical ventilation ; oesophageal pressure (P oeso) was measured by an accurate differential transducer (Validyne) and amplified for paper recording. Right atrial pressure (RAP) was measured via a 15 cm long transatrial fluid-filled catheter. Zero pressure was cautiously taken at the mid-axillary level in strict supine position. Transmural RAP (RAP TM) was calculated as RAP minus P oeso and averaged during a full respiratory cycle. All the tracings were recorded at 50 mm/sec.

Renal function : urine was collected through an intra-vesical catheter and urinary output (Vu) was expressed in ml/min.

Plasma ANF concentrations (pANF) were measured in right atrial blood samples by mean of radio-immunoassay (RIA), according to the Gutkowska technique, normal value 64 ± 14 pg/ml[3]. Because of a possible interaction between fentanyl and ANF release, residual fentanyl plasma concentrations were measured : 7.4 ± 5.6 mcg/ml (RIA).

Protocol : The following sets of measurements were performed after a 20 min steady state period : before PEEP (T1), PEEP 15 cm H_2O (T2), relapse PEEP (T3), combined PEEP 15 + anti-G suit (T4).

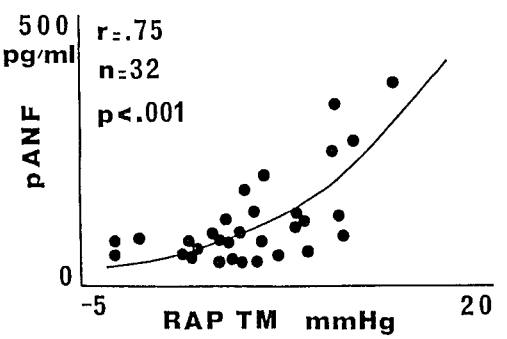
Statistical analysis was performed using the non-parametric Wilcoxon test.

RESULTS : data are summarized on the table (means ± SD)

	T1	T2	T3	T4
pANF pg/ml	112 ± 104	142 ± 111	72 ± 54	97* ±67
RAP mmHg	8.8 ± 2.8	12.9# ± 3.2	9.4 ± 2.4	15.6# ± 2.6
RAPTM mmHg	3.4 ± 4.2	6.5* ± 4.4	4.0 ± 3.7	7.7# ± 5.5
Vu ml/mn	1.24 ± .36	.58# ± 0.3	1.1 ± .2	0.4# ± .14

* $p < .01$; # $p < .001$ vs control (T2 vs T1 and T4 vs T3.

Furthermore, pANF was exponentially correlated to RAP TM (fig).



DISCUSSION : This study clearly demonstrates that PEEP does not inhibit ANF release, accordingly to an absence of decrease in RAP TM. Therefore, this mechanism does not account for the antidiuresis observed under PEEP. Morover, all the patients demonstrated an increased ANF release under combined PEEP and anti-G suit, simultaneously with an increase in RAP TM, indicating a normal response of ANF secretion to right atrial distension[4]. However antidiuresis persisted. Moreover this study confirms that RAP TM is a major determinant of ANF release.

REFERENCES :

1- Berry et al : Anesthesiology, 55 : 655, 1981
2- Urzua : Anesthesiology, 67, 608, 1987.
3- Gutkowska et al : Biochem Biophys Res Comm 122 : 593, 1984
4- Edwards et al : Circ Res, 62 : 191, 1988.