TITLE: DETERMINANTS OF VENTRICULAR PERFORMANCE DURING INDUCTION OF ANESTHESIA OF HEART TRANSPLANT RECIPIENTS

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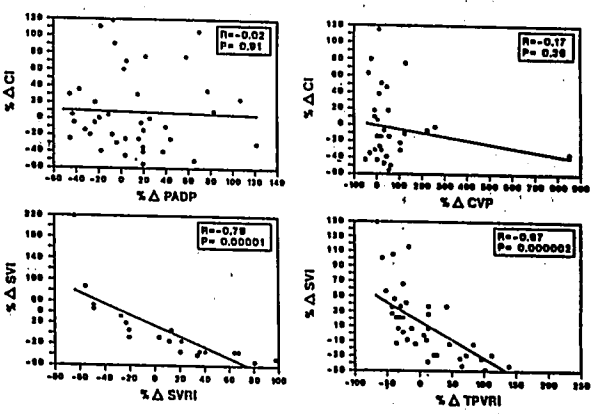
To determine the cause of ventricular dysfunction during induction of anesthesia (I) of heart recipients we analyzed pre and post I hemodynamic data (HR, MAP, CVP, MPAP, PADP, PAWP, CI, SVI, SVRI, TPVRI, PVRI and SVI/PADP and SVI/CVP ratios) of 104 recipients (NYHA IV, male/female ratio 3, 46 ± 1 year [mean ± SEM], 71 ± 2 kg, 1.82 ± .03 m^2 BSA, ischemic cardiomyopathy 44% primary cardiomyopathy 51%, others 5%, EF 19 ± 1%, PVRI 346 ± 2 dynes cm^{-5} sec/m^2) who were anesthetized as previously reported[2]. Least square linear regression and two-tailed Student t tests were used to determine correlation and significance, respectively.

Baseline data and percent changes from pre I were: HR 97 ± 2, -1 ± 2%; MAP 86 ± 2, -3 ± 3%; CVP 13 ± 1, 53 ± 17% ($p < .00017$); MPAP 35 ± 1, 6 ± 8%; PADP 26 ± 1, 11 ± 8%; CI 2.1 ± .07, 13 ± 7%; SVI 23 ± 1, 18 ± 9%; SVRI 2738 ± 152, 6 ± 10%; TPVRI 1448 ± 57, 14 ± 10%; PVRI 346 ± 22, 12 ± 14%; SVI/PADP 1.01 ± .07, 20 ± 11%; SVI/CVP 2.95 ± .38, 6 ± 15%. Percent changes of indices of ventricular performance (CI and SVI) were predicted well by changes in SVRI (Fig, bottom left, R = -.79, p = .00001) and TPVRI (Fig, bottom right, R = .67, p = .000002) but not by changes in PADP (Fig, top left), CVP (Fig, top right) or HR (R = .10, p = .58). In turn, changes in CVP and TPVRI were predicted [CVP (R = .48, p = .0012) and TPVRI (R = .46, p = .002)] well by changes in PADP, an index of left ventricular performance. When ventricular dysfunction occurs during I of heart recipients, it is predominantly due to an acute rise in vascular impedances, the left ventricular dysfunction being the main determinant of that of the right heart.

References

1. Anesth and Analg 70(S2):S136. 1090.
2. Anesth and Analg 67(S5):S50, February 19, 1988.



TITLE: COMPARISON OF THE ABILITY OF ESMOLOL AND ALFENTANIL TO ATTENUATE TACHYCARDIA AND HYPERTENSION DURING EMERGENCE FROM INHALATIONAL ANESTHESIA.

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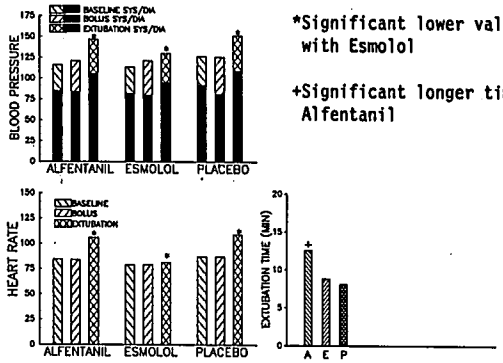
Introduction: Emergence and extubation can be as stimulating as intubation, resulting in tachycardia and hypertension. Esmolol (E), an ultra-short acting beta blocker, and Alfentanil (A), short-half-life narcotic, have both been shown to be effective in reducing the responses to the transient stimuli of induction and intubation.

This study was designed to evaluate the effectiveness of these agents in controlling the heart rate (HR) and blood pressure (BP) responses to emergence and extubation.

Methods: This protocol was approved by the university Human Research Board and all participants gave informed consent. Forty-two ASA I or II patients were included in this randomized double blind study. No premedication was given. Sodium thiopental, ≤6 mg/kg, was given for induction; intubation was facilitated by succinylcholine after 3 mg DTC. Anesthesia was maintained with $N_2O:O_2$, 70:30% and isoflurane (F) as clinically indicated. No narcotics were given. Patients were excluded if their systolic BP was < 100 or their HR < 60 at the end of surgery. The F was turned off as surgery finished. When the end tidal F concentration was less than 0.25% the patients were given either: (1) a bolus of normal saline (NS) followed by an infusion of the same, or (2) a bolus of 5 ug/kg of A followed by an infusion of NS, or (3) a bolus of 500 ug/kg of E followed by an infusion of 300 ug/kg/min of E. After three minutes of the infusion the N_2O was stopped and the patient extubated when awake. The infusion was stopped as the patient was extubated. Statistical analysis was by ANOVA, $p < 0.05$ considered significant.

Results:



*Significant lower values with Esmolol

+Significant longer time with Alfentanil

Conclusions: Emergence and extubation after inhalational general anesthesia result in significant increases in HR and BP. E, given as above, significantly attenuated the responses to emergence and extubation: A minimized the responses to emergence however, it significantly increased time to extubation. By extubation the protective effect of the A was no longer evident. There were no differences between the groups in either the quality of extubation or in the immediate postoperative course.