TITLE: RIGHT VENTRICULAR FUNCTION DURING ORTHOTOPIC LIVER TRANSPLANTATION

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Patients undergoing orthotopic liver transplantation (OLT) may develop significant hemodynamic instability, especially on reperfusion of the grafted liver.[1,2] A study using transesophageal echocardiography (TEE) has suggested that isolated right ventricular (RV) failure may contribute to this instability.[3] The aim of this study was to determine RV function during OLT using the RV ejection fraction (RVEF) catheter.

With institutional approval and informed consent, RV function was studied in 10 patients undergoing OLT. Anesthetics consisted of fentanyl, isoflurane, pancuronium, and oxygen-air. Venovenous bypass was used during the anhepatic stage. RV function was determined using a RVEF catheter (REF-ejection fraction/cardiac output computer, Baxter, Irvine, CA), allowing calculation of RVEF, RV end-diastolic volume index (RVEDVI) and RV end-systolic volume index (RVESVI) in addition to cardiac index (CI) and stroke volume index (SVI). Standard hemodynamic variables, including mean arterial pressure (MAP), heart rate (HR), mean pulmonary artery pressure (MPAP), central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), and right ventricular stroke work index (RVSWI), were measured. Hemodynamic profiles were determined 60 min after surgical incision (I+60), 60 min after onset of the anhepatic stage (II+60), 5 min before graft reperfusion (III-5), and 5, 30, and 120 min after graft reperfusion (III+5, III+30, and III+120). Statistical analysis was performed using analysis of variance for repeated measures. Simple regression analysis was used to correlate CVP and PCWP, CVP and RVEDVI, and RVEDVI and SVI.

Hemodynamic variables are presented in table 1. CVP and PCWP remained fairly constant throughout the procedure and were correlated (r=0.681). However, RVEDVI decreased during the anhepatic stage. No correlation was found between CVP and RVEDVI (r=0.348). RVEDVI and SVI were highly correlated (r=0.908). RVEF (SVI/RVEDVI) remained fairly constant over a wide range of RVEDVI, and it was always greater than normal values (0.40-0.45). A small decrease in RVEF was seen during the anhepatic stage; this was not associated with pulmonary hypertension or increased PVRI. Hemodynamic variables returned toward I+60 values after graft reperfusion.

The lack of a correlation between CVP and RVEDVI indicates that CVP is an unreliable indicator of RV preload. This may be due to changes in RV compliance. RV function was well preserved during the entire procedure, as indicated by a relatively constant and supranormal RVEF, even during the anhepatic stage. The cause of the small decrease in RVEF during the anhepatic stage is unknown, and its clinical significance is unclear.

Although unstable blood temperature precluded the determination of RVEF during the first 5 min after reperfusion, significant right ventricular dysfunction does not appear to occur on reperfusion.

References
1. Aggarwal S, et al. Transplant Proc 19 Suppl 3: 54-55, 1987
2. Estrin J, et al. Transplant Proc 21: 3500-3505, 1989
3. Ellis J, et al. Anesth Analg 68: 777-782, 1989

Table 1. Hemodynamic variables during OLT.

	I+60	II+60	III-5	III+5	III+30	III+120
MAP	79 ± 14	80 ± 8	76 ± 8	74 ± 15	70 ± 6	77 ± 11
HR	92 ± 11	94 ± 13	93 ± 13	90 ± 13	101 ± 13 *	96 ± 16 *
MPAP	21 ± 7	17 ± 3	15 ± 2 *	20 ± 5 #	22 ± 8 #	19 ± 4
CVP	11 ± 3	11 ± 4	9 ± 3	11 ± 3	12 ± 7 #	11 ± 4
PCWP	12 ± 3	9 ± 3	9 ± 2 *	12 ± 3 #	14 ± 6 #	13 ± 5 #
CI	5.6 ± 2.0	4.0 ± 1.1 *	3.1 ± 0.8 *	5.7 ± 2.0 #	5.8 ± 1.6 #	5.0 ± 1.5 #
SVI	61 ± 17	44 ± 14 *	34 ± 10 *	63 ± 20 #	59 ± 17 #	54 ± 18 #
SVRI	1110 ± 504	1440 ± 433 *	1822 ± 471 *	981 ± 381 #	878 ± 372 #	1134 ± 409 #
PVRI	126 ± 103	162 ± 80	155 ± 61	107 ± 56	119 ± 56	100 ± 57
RVEF	0.54 ± 0.09	0.48 ± 0.08*	0.48 ± 0.07*	0.57 ± 0.07#	0.54 ± 0.07#	0.54 ± 0.07#
RVEDVI	112 ± 28	92 ± 24 *	70 ± 14 *	111 ± 33 #	111 ± 36 #	100 ± 31 #
RVESVI	52 ± 16	48 ± 13	36 ± 7 *	48 ± 16 #	51 ± 22 #	46 ± 15 #
RVSWI	8.4 ± 6.4	3.8 ± 3.1 *	2.7 ± 1.6 *	7.9 ± 5.6 #	7.9 ± 4.8 #	6.7 ± 3.9 #

Mean ± SD * $p < 0.05$ compared to I+60 # $p < 0.05$ compared to III-5

Units for MAP : mmHg; HR : bpm; MPAP, CVP and PCWP : mmHg; CI : l·min^{-1}·m^{-2} ; SVI : ml·m^{-2} ; SVRI and PVRI : dynes·sec^{-1}·m^{-2} ; RVEDVI and RVESVI : ml·m^{-2}; RVSWI : g·mm^{-2}.

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Title: The effects of oral clonidine as the sole premedication on the hemodynamic and catecholamine responses to both brief and prolonged laryngoscopy.

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INTRODUCTION: Laryngoscopy and endotracheal intubation are potent stimuli that may dangerously increase HR and BP especially if laryngoscopy is prolonged to 45 seconds. Clonidine has been shown to decrease the dose of fentanyl needed to prevent the hyperdynamic response to laryngoscopy and intubation. For these reasons, we sought to determine if oral clonidine alone would mute the response to brief and/or prolonged laryngoscopy as measured by changes in the HR, BP and venous norepinephrine (NE) levels.
METHODS: The study was approved by our IRB. Forty ASA I-II patients 20-60 years old gave informed consent and were randomized to receive, in a double-blinded manner, either placebo or clonidine 5 mcg/kg to a maximum dose of 300 mcg. Patients with hypertension, diabetes, heart disease or with an abnormal EKG were excluded. BP and HR were measured prior to and 90 minutes after ingestion of the medication. After the second evaluation, a venous blood sample was obtained for NE levels and the patient was taken to the O.R. where noninvasive monitors were placed and vital signs recorded each minute for 12 minutes. Induction was standardized such that after mask 0_2, the patient received thiopental 5 mg/kg and succinycholine 1.5 mg/kg. Direct laryngoscopy was initiated and maintained for either 15 or 45 seconds at which time an endotracheal tube was placed and mechanical ventilation begun. Four minutes post intubation, a second venous blood sample was obtained.
RESULTS: All four groups were similar in respect to distribution of age, weight, ASA classification, and baseline vital signs. In each group, BP and HR rose significantly after the stress of laryngoscopy. Among the patients undergoing 15 second laryngoscopy, maximum systolic and diastolic blood pressures attained were significantly lower in the group who received clonidine than in the comparable placebo group ($p<0.05$ Mann-Whitney U test). As measured by maximal systolic and diastolic BP attained, clonidine provided no protection from the stress of a 45 second laryngoscopy. For both the 15 and 45 second laryngoscopies, the maximal heart rates attained were significantly lower in the clonidine than in the corresponding placebo groups. NE data showed large variation with no relationship to drug used or to the duration of stress. There were no complications or untoward effects recognized in any of our patients.
DISCUSSION: Oral clonidine at 5 mcg/kg as the sole preoperative medication served to blunt the maximum systolic and diastolic blood pressures attained during 15 second laryngoscopy. This dose also resulted in lowered maximal heart rates attained in both brief and prolonged laryngoscopy groups. However, the agent was unable to blunt the hypertensive response to a more prolonged stress. Oral clonidine will provide hemodynamic protection from the stress of 15 second intubation.