TITLE: CARDIAC OUTPUT FAILS TO INCREASE WITH PACING IN CARDIAC SURGICAL PATIENTS

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INTRODUCTION: The ability of heart rate to increase cardiac output (CO) is limited by venous return and ventricular filling. In euvolemic animals, CO remains constant when heart rate increases, since stroke volume progressively decreases (1). In contrast, increasing heart rate in hypervolemic animals causes CO to increase since stroke volume remains constant. Whether increasing heart rate by pacing improves CO in patients after cardiac surgery is not known.

METHODS: Following IRB approval and written informed consent, 9 patients undergoing elective coronary artery bypass surgery were atrially paced at 80, 95, and 110 beats/minute before and after cardiopulmonary bypass. The sequence of pacing frequency was randomized at each interval. Right ventricular ejection fraction (RVEF), end-diastolic volume (RVEDV), end-systolic volume (RVESV), and stroke volume were determined by thermodilution using a volumetric catheter (Baxter Edwards, Santa Ana, CA). Triplicate values of CO were obtained and averaged. Data were analyzed by analysis of variance for repeated measures.

RESULTS: Preliminary results are reported in Table 1. Before bypass, increasing heart rate caused significant decreases in RVEDV and stroke volume, although RVESV remained unchanged. RVEF serially decreased since the decrement in stroke volume was greater than the decrease in RVEDV. Consequently, CO remained unchanged with pacing. After bypass, RVEDV, stroke volume, and CO were significantly greater. However, pacing produced similar results by decreasing RVEDV and stroke volume so that RVEF declined significantly. Again, pacing at higher heart rates failed to increase CO.

DISCUSSION: Increasing heart rate caused RVEDV and stroke volume to decrease so that CO remained unchanged. In addition, right ventricular function curves were similar in these patients before and after bypass. Although cardiac pacing may be used to suppress ventricular dysrhythmias, its ability to increase CO is limited by ventricular filling.

REFERENCES:

1. Guyton AC. Circulation 64:1076, 1981.

TABLE 1: HEMODYNAMIC PARAMETERS BEFORE AND AFTER BYPASS

	BEFORE BYPASS Heart Rate			AFTER BYPASS Heart Rate		
Parameters	80 bpm	95 bpm	110 bpm	80 bpm	95 bpm	110 bpm
CO L/min	5.0 ± .4	4.8 ± .4	4.7 ± .4	6.1 ± .4	6.3 ± .5	6.0 ± .5
Stroke Volume ml	63 ± 7	52 ± 4*	42 ± 4*	74 ± 6	66 ± 5*	54 ± 4*
RVEDV ml	151 ± 13	134 ± 10*	128 ± 9*	170 ± 13	160 ± 11*	148 ± 9*
RVESV ml	88 ± 9	82 ± 7	86 ± 6	96 ± 8	94 ± 7	94 ± 6
RVEF%	.42 ± .02	.38 ± .01*	.33 ± .02*	.44 ± .01	.41 ± .02*	.36 ± .01*

* $p < 0.05$ compared to previous value

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TITLE : EPHEDRINE vs PHENYLEPHRINE FOR HYPOTENSION DUE TO THORACIC EPIDURAL ANESTHESIA ASSOCIATED WITH GENERAL ANESTHESIA: EFFECTS ON LEFT VENTRICULAR FUNCTION

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Thoracic epidural anesthesia associated with light general anesthesia (TEA + GA) is increasingly proposed to high risk patients. However, despite prophylactic intravenous hydratation, TEA + GA often causes systemic hypotension requiring vasopressor therapy. Ephedrine (E) and phenylephrine (P), which are widely used to counteract this hypotension may differently affect left ventricular (LV) function in high risk patients. Accordingly, we determine the effects of E vs P on LV function assessed by transoesophageal echocardiography (TEE), when E or P was used to treat TEA + GA induced hypotension.

Twenty patients, with preoperative radionuclide ejection fraction > 40 %, scheduled for abdominal aortic surgery were randomly assigned to receive either E (n=10) or P (n=10) to restore blood pressure under TEA + GA. All gave informed consent. In all patients radial artery, pulmonary artery and thoracic epidural (T8) catheters were established. While patients were receiving 1,5 l of lactated ringer solution, TEA was induced at T3 level by injecting 7-9 ml of mixture of plain bupivacaine 0.5 % and lidocaine 2 %. GA was induced by midazolam and fentanyl (6 mcg/kg) and the ventilation was controlled. E (5 mg increments) or P (20-60 mcg/min continuous infusion) was given to achieve a systolic blood pressure 15 % lower than the preoperative value.

Three sets of hemodynamic and TEE measurements were performed 1) awake before TEA (Control), 2) TEA + GA under controlled ventilation, 3) after E or P infusion (Vasopressor). Ejection fraction area (EFa) was determined using TEE short axis view of the LV.

TABLE : mean ± SD, 2 ways ANOVA. ▲ $p<0.05$ E vs P; * $p<0.05$ vs the preceeding value.

		CONTROL	TEA + GA	VASOPRESSOR
MAP	E	98 ± 9	68 ± 6 *	89 ± 10 *
mmHg	P	90 ± 10	66 ± 10 *	86 ± 8 *
HR	E	78 ± 16	67 ± 14 *	65 ± 16 ▲
b/min	P	73 ± 10	58 ± 7 *	50 ± 8 *
CI	E	3.7 ± 0.6	2.8 ± 0.5*	3.5 ± 0.7*▲
$L/min/m^2$	P	3.5 ± 1.0	2.6 ± 0.6*	2.1 ± 0.6*
EFa	E	55 ± 14	59 ± 14	60 ± 13 ▲
%	P	60 ± 10	59 ± 12	51 ± 14 *

Our study demonstrates that the differences between the effects of E vs P on myocardial function are readily apparent when these two agents are given to treat TEA + GA induced hypotension. When P was given, both CI and EFa were significantly compromized. By constrast, E possessed the advantage of maintaining LV function without increasing heart rate.