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TITLE: POTENTIAL INTRAOPERATIVE UNDERESTIMATION OF THE SEVERITY OF MITRAL REGURGITATION BY TEE

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Transesophageal Echocardiography (TEE) is frequently used intraoperatively to estimate the severity of mitral regurgitation (MR). This estimation is performed after the induction of anesthesia when the systemic blood pressure is generally below the patient's usual value. The echocardiographic assessment of MR may be significantly effected by the lower blood pressure. Previous investigations have generally ignored this factor. This study examined the effects of restoring the patient's blood pressure to preinduction values on the estimation of severity of MR by TEE.

The protocol was approved by the IRB and written informed consent was obtained from 17 patients with clinical or angiographic MR undergoing cardiac surgery. Each patient had a radial and pulmonary arterial catheter placed prior to the induction of anesthesia, and recordings of the hemodynamics were obtained. Anesthesia was induced with a high-dose opioid/relaxant technique, and a 5MHz TEE probe was then inserted. In the absence of surgical stimulation, and during brief periods of apnea the mitral valve was evaluated. Color flow mapping (CFM) of the entire left atrium was performed, and pulmonary vein flow was then measured by pulsed-wave Doppler (PWD) (A). A bolus of phenylephrine (PE) (40-80 mcgs) followed by an infusion (20-80 mcgs/min) was administered to restore the patient's preinduction blood pressure, and the assessment was repeated (B). Simultaneous hemodynamic recordings were obtained in both periods. MR by CFM was graded by two observers as I-IV using standard echocardiographic criteria, and the PWD recordings were analyzed to obtain the peak systolic and diastolic velocities (VEL), and the systolic and diastolic velocity time integrals (VTI). The ratios of the systolic to diastolic (S/D) values were also calculated. Comparison of the echo findings during A (BP sys = 102±17, PCWP=17±7) to those during B (BP sys = 136±12, PCWP=22±9) was performed using a paired Student's t-test.

In 10 patients after PE the MR by CFM increased by one grade, and in 4 patients by two grades. 2 patients with grade IV MR, and 1 patient with grade II during A did not change following PE. As shown in the table, after PE there were significant reductions in S/D pulmonary vein flow parameters. This is consistent with increased MR.

TABLE: PWD PULMONARY VEIN FLOW PARAMETERS

	A	B	DIFF
S/D VEL	1.02±0.59	.62±.76	.40±.61*
S/D VTI	1.44±1.14	.89±.98	.55±.89*

* = P<0.05

These data show that TEE estimations of MR are very sensitive to changes in systemic blood pressure and that every attempt to recreate the patient's usual hemodynamic conditions should be made prior to clinical decision making. Additionally, the use of pharmacologic stress during echocardiographic monitoring can be used to predict which patients will most benefit by afterload reduction.

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TITLE: HORMONAL RESPONSES DURING HYPOTENSIVE EPIDURAL ANESTHESIA

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Introduction: The hormonal responses to hypotension, which include release of renin and catecholamines, may be altered by epidural anesthesia(EA).[1,2] This study was designed to define the hormonal responses to deliberate hypotension (DH) under high EA.

Methods: After institutional approval, we measured plasma renin activity (PRA), epinephrine(Epi) and norepinephrine (NE) before and during DH in 28 healthy patients undergoing total hip replacement. Patients were randomly assigned to receive infusions of either low dose Epi (<0.1 ug/kg/min), phenylephrine(Phe) (<0.5ug/ kg/min) or fluid to maintain mean arterial pressure (MAP) at 50-60 mmHg. Baseline blood samples were drawn 30 minutes after placement of arterial and CVP catheters under intravenous sedation. EA was induced with bupivacaine 0.75% or lidocaine 2%, 15-25 ml to produce a sensory level of T2-T4. Blood samples during DH were drawn after MAP had been stable for a minimum of 45 minutes. Data were analyzed using ANOVA or Wilcoxon signed-rank (Epi levels) and alpha was set at 0.05.

Results: There were no differences between groups in age (42-79, mean= 62±7.3) or baseline values for PRA, Epi and NE (table). During DH, plasma NE decreased in all patients. Epi levels fell during DH in patients receiving fluid and tended to fall in the Phe treated patients. PRA was elevated only in the Epi treated group.

Discussion: In contrast to other studies,[1,2] renin did not rise in response to hypotension except in patients treated with Epi, in whom renin secretion was probably due to direct beta-adrenergic stimulation. This reflects the importance of the sympathetic nervous system in modulating renin secretion and agrees with prior studies showing that acute renal denervation markedly blunts renin secretion in response to hypotension.[3,4] In summary, DH under high EA did not cause activation of the renin-angiotensin system and was associated with low or normal catecholamine levels.

References:

1 Anesth 62:294-297, 1985
2 Br J Anesth 52:305-310, 1980
3 Clin Sci and Molec Med 51:453-461, 1976
4 Circ Res 44: 645-652, 1979

	Before Epidural			During Epidural Hypotension		
	Epi (n = 10)	Phe (n = 11)	Fluid (n = 7)	Epi (n = 10)	Phe (n = 11)	Fluid (n = 7)
MAP (mmHg)	97 ± 10	95 ± 13	102 ± 14	54 ± 3 *	54 ± 4 *	54 ± 3 *
HR (beats/min)	76 ± 14	65 ± 12	68 ± 7	74 ± 15 §	51 ± 9 *	55 ± 7 *
CVP (mmHg)	6 ± 3	5 ± 1	4 ± 2	5 ± 2	3 ± 2 *	5 ± 2
PRA (ng/mL/hr)	0.8 ± 0.5	1.0 ± 0.7	1.0 ± 0.8	2.8 ± 2.2 *§	0.9 ± 1.1	0.6 ± 0.4
Plasma Epi (pg/mL)	43 ± 30	39 ± 34	49 ± 16	1552 ± 431 *§	28 ± 8	23 ± 5 *
Plasma NE (pg/mL)	204 ± 68	208 ± 85	244 ± 86	85 ± 55 *	62 ± 45 *	50 ± 17 *

All values expressed as mean ± standard deviation
* p < 0.05 vs Before Epidural
§ p < 0.05 vs Phenylephrine and Fluid