Title: EVALUATION OF LEFT VENTRICULAR VOLUMES AND CONTRACTILITY DURING INCREMENTAL PEEP USING TWO-DIMENSIONAL TRANSESOPHAGEAL ECHOCARDIOGRAPHY

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Introduction: With addition of positive end-expiratory pressure (PEEP), stroke volume (SV) is known to decrease in both experimental animals and humans. The mechanism by which SV is decreased remains controversial due to the limitations of the methods available to date. Previous PEEP studies using M-mode echocardiography have produced contrary findings concerning left ventricular (LV) preload and contractility impairments as possible causes of the decrease in SV.[1,2,3] These controversies might be the result of limitations to the use of M-mode echocardiography to measure LV dimensions. The cross section of the LV imaged by M-mode echocardiography is assumed to be representative of the LV as a whole, and the presence of septal wall motion abnormalities as well as changes of cardiac geometry cannot taken into account.[4] We therefore studied the effects of incremental PEEP on LV volumes and contractility by means of two-dimensional transesophageal echocardiography (2D-TEE), which may circumvent the shortcomings of M-mode echocardiographic techniques.

Methods: 12 ASA class I patients (mean age 33±13, range15-48 years) undergoing a variety of surgical procedures were studied. All patients gave written informed consent. Anesthetic management was similar in all patients, consisting of induction with intravenous etomidate (0,2 mg/kg), fentanyl (3 µg/kg) and endotracheal intubation after paralysis with vecuronium (0.1 mg/kg). Anesthesia was maintained with 60% N_2O in O_2 and fentanyl (3-7 µg/kg). The investigation was performed in the pre-operative phase after induction of anesthesia. All patients were at a stable hemodynamic state, exhibiting <10% variation in blood pressure and heart rate. The study was performed with a TEE probe (Hewlett Packard) connected to a Hewlett Packard echo-Doppler unit (model 77020).The probe was gently introduced into the patients' esophagus and positioned to afford the most spherical short axis view of the LV at the level of the papillary muscles. PEEP was incrementically raised from 0 to 16 cm H_2O in 4-cmH_2O steps each for 5 min. When PEEP altered the position of the heart, the position of the transducer was slightly adjusted to maintain the maximal spherical midpapillary muscle view. Heart rate (HR) and systolic blood pressure (SBP) of A. radialis were continuously monitored and the 2D-echocardiograms were recorded on videotape for later analysis. Left ventricular end-diastolic area (LVE DA) and end-systolic area (LVESA) from 10 consecutive cardiac cycles were traced by a computer-assisted outlining by the endocardial image with exclusion of the papillary muscles. Ejection fraction area (EFA) was calculated as ((LVEDA-LVESA)/LVEDA) 100 . Volume calculations in the 2D examination were based on a modified elipsoid model for single-plane data using the following formula: $V = SA^{3/2}$ (SA+36/ SA+12), where V stands for volume and SA for short-axis area.[5] The stroke volume index (SVI) was derived by the difference between LVEDVI and LVES VI. Cardiac index (CI) was calculated as the product of SVI and heart rate, while ejection fraction (EF) was obtained by the formula ((LVEDVI-LVESVI)/LVED VI) 100 .The endsystolic pressure-volume relationship[6] of the LV was estimated by the peak systolic arterial pressure (as an approximate equivalent of LVES pressure) and the LV end-systolic area (ESP/ LVESA) and volume (ESP/ LVESV), respectively.[7] Data were expressed as mean±SEM. Statistical significance was estimated using paired t-test.

Results: The results are displayed in Table 1. Figure 1 and 2 show the relationship between the LV volume indices and SVI (mean values of all patients) during incremental PEEP.

Table 1

PEEP (cm H20)	0	4	8	12	16
SBP (mmHg)	121±5	117±4,5	121±5	122±8	122±6
HR (beats/min)	61±4	60±4	63±5	63±4	66±5*
LVEDA (cm^2)	15.9±0.8	15.0±0.8*	14.7±1.0*	13.6±0.9*	12.5±0.8*
LVESA (cm^2)	7.3±0.6	6.8±0.6*	6.6±0.5*	6.1±0.5*	5.9±0.4*
LVEDVI (ml/m^2)	62.7±3.5	58.4±3.4*	56.8±4.0*	51.6±3.9*	46.6±2.9*
LVESVI (ml/m^2)	23.7±2.2	21.6±2.1*	20.7±1.8*	18.9±1.7*	17.7±1.5*
SVI ($ml/beat/m^2$)	39.0±2.6	36.8±2.2	36.0±3.2	32.7±3.0*	28.9±2.5*
CI (l/m^2)	2.4±0.3	2.2±0.2	2.2±0.2	2.0±0.2	1.8±0.1*
EF (%)	62.3±2.7	63.3±2.5	62.9±2.9	62.7±3.1	61.4±2.8
EFA (%)	53.9±2.6	54.6±2.4	54.2±2.8	53.5±3.1	51.8±2.8
ESP/LVESA ($mmHg/cm^2$)	17.9±2.0	18.7±2.1	19.7±2.0*	21.2±2.0*	21.9±1.7*
ESP/LVESV (mmHg/ml)	3.1±0.4	3.5±0.5*	3.5±0.5*	3.8±0.5*	4.0±0.4*

mean ± SEM,* $p < 0.05$ vs. PEEP 0

Figure 1

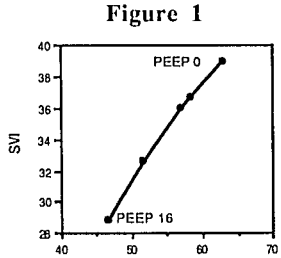
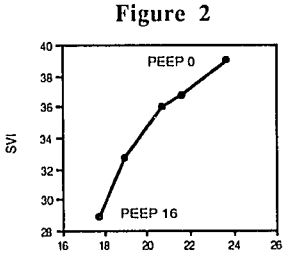


Figure 2



Discussion: The present 2D-TEE study clearly demonstrates that incremental PEEP significantly decreases LV preload in patients without cardiac disease or respiratory failure. The proportional relationship between LVEDVI and SVI indicates that the decrease in SVI is closely associated with a decrease in LVEDVI. PEEP did not decrease EF and EFA. This might suggest that LV systolic function is not altered by PEEP. However, these inotropic parameters may have been affected by the changes of preload.Using the load-independent end-systolic pressure/volume ratio for the assessment of LV contractility, we found a significant positive inotropic effect of PEEP. This unexpected finding should act as an incentive for further studies because at this time one may only speculate as to the causes of this effect.We conclude, that the PEEP-induced decrease of SV is caused mainly by a decrease in LV preload and obviously not by a depressed LV systolic function.

References:

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