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Title : ECHOCARDIOGRAPHY AND LV FUNCTION DURING ANESTHESIA

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INTRODUCTION: Echocardiography is a sensitive noninvasive technique which provides direct visual information concerning LV function including wall motion abnormalities, chamber size, fiber shortening and mechanical/valvular dysfunction indicative of ischemia. Echo changes may occur in the absence of other indication of ischemia. Patients with coronary artery disease (CAD) have been studied with echo during induction of anesthesia to evaluate the occurrence of ischemia and its relationship to hemodynamic changes and to such obligatory maneuvers as laryngoscopy and intubation.

METHODS AND MATERIALS: Echocardiography was performed on 24 adults with CAD undergoing anesthesia for cardiac surgery. The 17 male and 7 female patients, age 53.8 ± 23 had persistent symptoms of CAD despite medical management; 4 had unstable angina, 8 had compensated CHF. 21 patients were taking Inderal, average dose 187 mg daily. Patients were sedated with diazepam, morphine and scopalamine. Standard 6 limb leads and V_5 ECG, intraarterial, CVP and Swan Ganz catheters were established under local anesthesia. Baseline cardiac output and derived hemodynamic variables were determined. Anesthesia was induced with diazepam or fentanyl or both. Continuous infusion and/or bolus sodium nitroprusside or IV nitroglycerine were used for control of SAP when indicated throughout the induction period. 6 patients received dopamine infusion 3-10 mcg/kg/min for low CI. M-mode echocardiograms were taken continuously from the time of baseline CO until intubation was complete. Echoes were analyzed for septum and wall motion abnormalities, change in end-systolic and enddiastolic LV dimension and percent shortening, and the data correlated and compared statistically with concurrent direct and derived hemodynamic data.

RESULTS: 10/24 patients demonstrated echocardiographic changes indicative of ischemia with an increase in end-systolic (4 ± 3.3mm) and end-diastolic (3 ± 1.6mm) LV dimensions, a decrease in % fiber shortening (7 ± 6.7) and septal or posterior wall (PW) motion abnormalities. Despite active preventive measures, all but two developed an increase in SAP (24.8 ± 24), HR (8 ± 6.7) and rate pressure product RPP (2513 ± 1950) all $p<.01$; $\overline{PAo}$, CI, SV, SVR, and LVSWI changes were NS for the group, although in 2 patients $\overline{PAo}$ increased by 21 and 26 Torr. Only one patient developed ischemic ST-T changes. 6 patients displayed pronounced impairment of wall thickening affecting the septum in 2 and the PW in 4. 14/24 patients demonstrated either no changes or an increase in LV contractility with a decrease in transverse cavity dimensions at end-systole and diastole. The 24 patients had an increase in SAP, HR and RPP significant at $p<.01$ with NS changes in $\overline{PAo}$, SV, LVSWI and SVR. A total of 3/24 had ST-T changes: in one there were echo changes of ischemia; the other two had ST-T changes in V_5 only and no echo changes of ischemia.

DISCUSSION: Echocardiography is a sensitive method for determination of myocardial ischemia during induction of anesthesia and intubation: 10/24 patients had significant changes in LV dimension or wall motion, only 3 of whom could have been diagnosed by other means (2 by increase $\overline{PAo}$, 1 by ECG change). 8/10 had significant increase in SAP, HR and RPP. Fisher's exact test was performed comparing absence of SAP increase ($\leq$ 15mm Torr) with the absence of decrease % fiber shortening and was highly significant, $p<.003$. SAP increase need not be large as shown in Fig. 1.

CONCLUSION: M-mode echocardiography is a sensitive and appropriate means for determining LV dysfunction and ischemia during induction of anesthesia and intubation. The primary determinants of such dysfunction and ischemia are elevation in SAP, HR and RPP, which may be of surprisingly low magnitude in relation to the LV dysfunction produced.

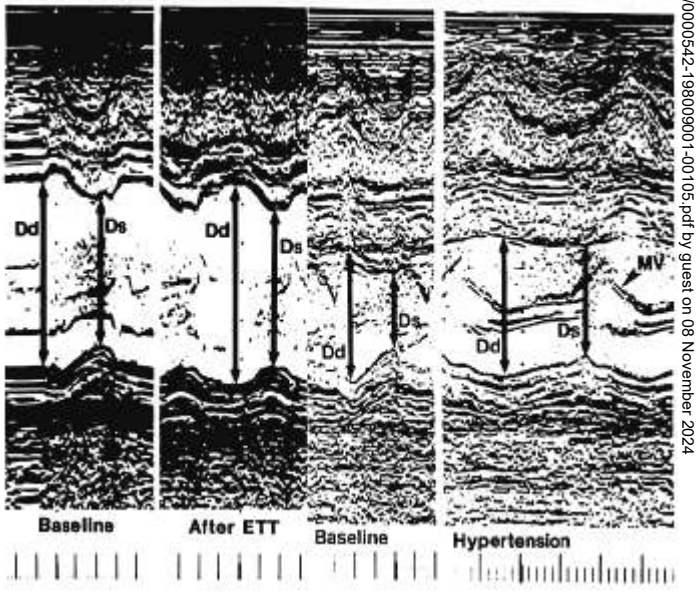

Figure 1 Figure 2

FIGURE 1: Systolic SAP increased 31 to 148 but PAo decreased; the increase was sudden but to a level no greater than preop SAP.

FIGURE 2: Hypertension following laryngoscopy. LV thickening and excursion severly deranged and mitral valve (MV) open during systole, indicating ischemic papillary muscle dysfunction.