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TITLE : TRANSESOPHAGEAL ECHOCARDIOGRAPHY (T.E.E.) IN HEART SURGERY

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Introduction. During cardiac surgery, the ventricular function is influenced by the composite of intrinsic and extrinsic factors. A more complete understanding of the relative contribution of each of these factors to cardiac performance would have clinical importance. It has been our consistent observation that the cardiac output in the early post bypass period is approximately equal to that of pre-bypass or in the case of patients with valvular disease somewhat higher, yet by the end of surgery in both coronary and valvular cases the cardiac output has generally fallen. Currently employed monitoring techniques such as pressure and cardiac output measurements provide an incomplete picture of ventricular function in this situation. In an attempt to elucidate this phenomenon we evaluated the dimensional changes, contractility, and diastolic indices of the left ventricle using our TEE method.

Methods. Twenty-four consecutive patients, 16 male and eight female, who underwent open heart surgery were studied. Their mean age was 58, ranging from 25 to 79. Twelve patients underwent saphenous vein bypass grafting for coronary artery disease (CAD), eleven patients underwent valvular surgery and one patient had both coronary and valvular surgery. Premedication consisted of morphine sulfate (0.10-0.15 mg/kg) and scopolamine (0.015 mg/kg) IM and ethrane (0.5%-2%) in oxygen was used in all patients. Following intubation of the trachea, an esophageal echo transducer was inserted per os into the esophagus with the aid of McGill forceps. An echocardiogram of the left atrium and the mitral valve was obtained. The transducer was then manipulated to obtain a clear reflection from the left ventricular posterior wall and interventricular septum at the level of the chordae tendinae of the mitral valve. This was the site of measurements of ventricular dimensions. Echocardiography, dye dilution cardiac output, atrial pressure with simultaneous ECG recording were performed at six stages; (1) 30 minutes after induction of anesthesia; (2) after the chest was opened, (3) after pericardial opening; (4) after bypass and before pericardial closure; (5) after pericardial closure; (6) after chest wall closure during surgery. Transesophageal M-mode echocardiograms were taken using a conventional echo machine. Left ventricular end-diastolic diameter (Dd) was measured at the summit of the R-wave of the ECG. End systolic diameter (Ds) was measured at the peak excursion of the posterior left ventricular wall. The thickness of the posterior left ventricular wall and interventricular septum and duration of minor axis shortening were also measured. An average of five consecutive cycles was used in all calculations. From those data, left ventricular mean velocity of circumferential fiber shortening (Vcf), ejection fraction (EF) ventricular volume (V), dp/dv, stiffness constant (KA) were all calculated by appropriate formulas. Thirty-six dye cardiac output measurements in 13 patients were compared to that by echo. Students t-test for paired data or the analysis of variance were used for statistical comparison.

Results. (1) In 21 cases, satisfactory TEE recordings of the left ventricle were obtained but in three patients, the recordings were not successful because of extreme horizontal location of the heart. (2) CO— a reasonable correlation was observed between cardiac output by dye and echo in 36 simultaneous measurements in 13 patients ($Y= 0.85 \times +101$, $\gamma= 0.72$). (3) Ventricular dimension and other indices - a statistically significant ($P<0.05$) decrease in Dd was found after sternal closure, which was associated with significant increases in dp/dv (75%, $P<0.05$) and in KA (13%, $P<0.05$).

Discussion. It has long been questioned whether the pericardium plays a role in limiting the dimension of the left ventricle. Recently Shabetai R[1], Ross J. Jr.[2], reported the function of the pericardium in changing the diastolic pressure volume relationships of the ventricle. To our knowledge there have been no human studies relating the influence of the chest wall on ventricular function. In this study we demonstrated that both the pericardium and chest wall play a role in limiting the diastolic size of the heart. The study also suggest that these events are due simply to a mechanical restriction of the left ventricular expansion. An increased KA and unchanged Vcf tend to confirm this view. In the heart already severely compromised due to intrinsic heart disease and surgery, this increased stiffness appeared to have a measurable effect on cardiac performance.

References.

1. Shabetai R: American Journal of Cardiology 42:1036-1043, 1978.
2. Ross J Jr: Circulation 59:32-37, 1979