

Abnormal postoperative interventricular motion: New intraoperative transesophageal echocardiographic evidence supports a novel hypothesis*

[American Heart Journal](#)

[Volume 126, Issue 1](#), July 1993, Pages 161-167

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[https://doi.org/10.1016/S0002-8703\(07\)80024-X](https://doi.org/10.1016/S0002-8703(07)80024-X) [Get rights and content](#)

Abnormal interventricular septal motion is a frequent finding after cardiac surgery. However, the time course and underlying mechanisms are not well understood. Nineteen patients, mean age 54 years (range 20 to 82 years), were studied with intraoperative transesophageal echocardiography at five specific times: with the chest closed (baseline), with the chest open and the pericardium closed, with both chest and pericardium open, after cardiopulmonary bypass with the chest open, and after cardiopulmonary bypass with the chest closed. In each patient interventricular septal motion was recorded from the transgastric view; tricuspid annular motion and Doppler color flow mapping of tricuspid regurgitation were obtained from the four-chamber view. All the echocardiographic data were stored on videotape and were later viewed in random sequence by one investigator who was aware of the baseline stage but was blinded to the other stages. All patients had normal septal motion before cardiopulmonary bypass. After cardiopulmonary bypass, with the chest still open, 5 of 17 patients (29%) with adequate recordings had abnormal septal motion while 13 of 17 patients (76%) with adequate recordings had abnormal tricuspid annular motion. After chest closure, only three patients (14%) had normal septal motion and one patient (6%) had normal tricuspid annular motion. Significant tricuspid regurgitation was an infrequent finding in all cases. It is concluded that abnormal interventricular septal motion occurs after cardiopulmonary bypass and is related to abnormal tricuspid annular motion. We hypothesize that suboptimal right ventricular myocardial preservation impairs the motion pattern of the right ventricle, including the tricuspid annulus. This factor alone or with the addition of acute mechanical restriction of the edematous heart by the chest wall may impair right ventricular free wall motion sufficiently to cause recruitment of the interventricular septum toward the right ventricle to maintain right ventricular stroke volume.