there was no difference in the trauma caused by either procedure, as reflected by a similar incidence of sore throat or hoarseness. Further study should be done to document movement of the cervical spine as well as changes in cardiovascular and intracranial pressure measurements occurring during both techniques.

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Transeosophageal Echocardiographic Observations in a Patient Undergoing Closed-chest Massage

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The mechanism by which chest compression generates forward blood flow has not been clarified. Some authors believe that ventricular compression with normal valvular competence is responsible,7 and others believe that only a generalized increase in intrathoracic pressure is necessary, with the heart acting merely as a passive conduit.2,3 A few cineangiographic4 and echocardiographic5 studies of human subjects undergoing cardiopulmonary resuscitation (CPR) generally support the belief that the mitral valve does not move in response to chest compression; quantitative analysis of wall motion has not been described.

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REPORT OF A CASE

We report the echocardiographic and hemodynamic findings using transeosophageal 2-D echocardiography (TEE) in a patient undergoing CPR. As part of an ongoing investigation of TEE as an intraoperative monitor of myocardial function, this patient gave informed consent for the use of TEE during surgery for coronary artery bypass grafting (CABG). Prior to surgery, a Diasonics® echoscope incorporating a 3.5 MHz phased-array transducer was positioned in the esophagus immediately posterior to the left ventricle (LV) to provide a short-axis view of the LV at the mitral valve level. At several stages of the operation images were recorded on videotape and analyzed later for short-axis fractional area change (FAC) and segmental wall motion, using a center of mass model for wall motion analysis with the Franklin Quantic 2000 computer. This analysis corrects for translational movement of the heart by superimposing the cavity centers of mass in end-diastolic and end-systolic frames. The TEE transducer provided good quality, high-resolution images of the LV endocardial outline and mitral valve motion while CPR was completely unimpeded. Concomitant systemic and pulmonary arterial pressures and electrocardiogram were continuously recorded on a strip chart at a paper speed of 5 mm/s during CPR.

The patient, a 71-yr-old man with a 15-yr history of angina pectoris, had suffered three previous myocardial infarctions. Resting ejection fraction by mitigated radionuclide angiography was 31% and by cardiac catheterization, 26%. He was considered New York Heart Association (NYHA) angina Class IV. There was generalized, moderate hypokinesia of the LV. The mitral valve appeared normal and competent. After uneventful induction of anesthesia and a stable course before cardiopulmonary bypass, the patient underwent saphenous vein grafting to the left anterior descending, circumflex marginal, and right coronary
arteries and was separated from cardiopulmonary bypass without difficulty while receiving infusions of lidocaine and nitroglycerin. The pericardium was left open and the chest was closed. The thermodilution cardiac output was 4.2 L/min with sinus rhythm at 90 beats/min (fig. 1A). However, the patient's condition later deteriorated, with a falling cardiac output and a rising pulmonary artery diastolic pressure (fig. 2A). By TEE the lateral wall of the LV was observed to become markedly hypokinetic (fig. 1B). The nitroglycerin infusion rate was increased and nifedipine was given sublingually, but inotropic support with dopamine and epinephrine in increasing doses became necessary to support the failing ventricle. Subsequently ventricular tachycardia developed. External chest compression was begun at a rate of 90–120 beats/min (figs. 1C and 2B). Chest compression was performed by a physician who judged his effectiveness by the magnitude of the arterial waveform. Following defibrillation the patient converted to a sinus tachycardia with aberrant conduction which, with continued chest compression, resulted in improved ejection characteristics and more adequate hemodynamics (figs. 1D and 2C). Chest compression was discontinued with adequate maintenance of blood pressure (fig. 2D) but the patient later went on to develop intractable ventricular tachycardia and died. In addition to evidence of old infarctions, autopsy revealed acute myocardial necrosis of the lateral region of the LV below the level of graft anastomosis to the circumflex marginal artery, although all grafts were patent.

**DISCUSSION**

TEE recordings from the operation were reviewed and qualitatively scored by all authors to evaluate wall motion and examine the motion of the mitral valve during episodes of ventricular tachycardia (134 beats/min) and sinus tachycardia (90–120 beats/min), with and without CPR. Qualitative evaluation of the videotape agreed well with computerized analyses.

During ventricular tachycardia only the interventricular septum was moved inward by chest compression, producing a small FAC (fig. 2B). This septal motion, occurring synchronously with each chest compression, was clearly visible both in real time and by off-line frame-by-frame analysis of the videotape. The FAC has correlated well with ejection fraction in CABG patients. During sinus tachycardia each heart beat generated some lateral and anterior wall motion that, when occurring synchronously with the septal movement created by chest compression, resulted in improved systemic blood pressure, FAC, and, presumably, ejection fraction. These findings are illustrated in fig. 1; although some images omitted a small segment of endocardial border as shown, quantitative analyses were done with images including the entire endocardial outline. Similarly, only views that clearly showed the mitral valve leaflets were used for evaluation of mitral valvular motion. Normally, wall motion is best evaluated at a midpapillary muscle level where regional contraction patterns are almost symmetrical. At a
mitral valve level, septal motion may normally appear paradoxical; the inward movement is therefore all the more significant. Although septal thickening cannot be evaluated from these images, the effects of external chest compression on septal movement and on the LV minor axis area change at the mitral valve level are clearly shown.

Although the force delivered to the chest with each compression was not measured, the pressure transmitted to the pulmonary artery was found to be fairly constant with both ventricular and sinus tachycardia. Systemic pressures generated by CPR, though, were lower during ventricular tachycardia than with sinus tachycardia, again suggesting improved ejection when CPR is performed in conjunction with an underlying rhythm. The mitral valve was seen to open and close normally during sinus tachycardia. With CPR it was disturbed by chest compression but continued to move in synchrony with the underlying rhythm. However, during ventricular tachycardia there was no predictable motion, with or without CPR.

From our observations with this patient, we conclude that external chest compression moves only the septal wall of the left ventricle, and the mitral valve is not a competent valve in the absence of a stable rhythm. Performance of external chest compression in the presence of a stable rhythm resulted in a larger FAC and, presumably, ejection fraction than with the intrinsic rhythm alone. Improved hemodynamics during CPR with a stable rhythm may result partly from the myocardial contraction generated by an intrinsic depolarization and partly from the activity of a competent mitral valve. Synchrony of external compression with an intrinsic myocardial contraction would presumably result in the optimum cardiac output.

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