GRAPHIC TECHNIQUES IN CARDIOLOGY

Simultaneous Left Atrial Echocardiography and Aortic Blood Velocity during Right Ventricular Pacing in Man*

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Atrial function during pacemaker-induced tachycardia has been studied in experimental animals and human subjects. It has been demonstrated that the atrioventricular interval, aberrant ventricular activation, and mitral regurgitation are important determinants of atrial and ventricular performance during right ventricular pacing.

Recent investigation has indicated that echographically assessed aortic root motion is an index of stroke volume. We describe here the use of simultaneous Doppler ultrasonographic studies (using an aortic catheter) and echocardiographic studies as a method to study hemodynamics during pacemaker-induced right ventricular tachycardia.

Figure 1 shows the simultaneously recorded right ventricular pressure, phasic velocity of the aortic flow, lead 3 of the electrocardiogram, the phonocardiogram of the mitral area, and the echocardiogram from a 28-year-old woman with mitral stenosis. A ventricular driving rate of 120 impulses per minute results in an increase of the left atrial internal dimension. At a rate of 180 impulses per minute, there is a marked reduction of the velocity of aortic flow in association with a return of the left atrial size toward control values. The configuration of the aortic wall remains normal at both rates of pacing, although the amplitude of deflection is diminished.

Figure 2 discloses similar measurements in a 47-year-old man with coronary arterial disease. Right ventricular pacing at rates of 120 and 160 impulses per minute produces progressive enlargement of the left atrial internal dimension. Electrical stimulation of the right ventricle is associated with flattening of aortic motion and is most evident at a rate of 180 impulses per minute.

Invasively studied left atrial pressures and volumes have been observed to increase during right ventricular pacing. The augmented left atrial size has been ascribed to systolic mitral regurgitation and incomplete diastolic atrial emptying. Underlying mechanisms for these changes include (1) loss of sequential atrial and ventricular activity, (2) inefficient mitral valvular closure due to depressed myocardial function, (3) atrial cannonading (ie, premature atrial contraction against a closed mitral valve), (4) weak atrial contraction secondary to hypoxia, and (5) an initial positive gradient across the mitral valve creating increasing mitral stenosis.

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FIGURE 1. Simultaneously recorded right ventricular pressure (RV Press), aortic flow velocity (AO FL VEL), lead 3 (LIII) of ECG, phonocardiogram of mitral area (MA), and echocardiogram from 28-year-old woman with mitral stenosis. Right ventricular (RV) pacing at rate of 120 impulses per minute results in increase of left atrial internal dimension. At 180 impulses per minute, there is marked reduction of velocity of aortic flow in association with return of left atrial internal dimension toward control values. Configuration of aortic wall remains normal at both rates of pacing. AO, aortic valve; AR, aortic root; LA, left atrium; and PI, pacing impulse.
assessing the influence of ventricular tachycardia on myocardial performance.

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Figure 2. Lead 3 (LIII) of ECG, aortic flow velocity (AO FL VEL), phonocardiogram of mitral area (MA), right ventricular pressure (RV PRESS), and echocardiogram from 47-year-old man with coronary arterial disease. Right ventricular (RV) pacing (PI, pacing impulse) at rates of 120 and 160 impulses per minute produces progressive enlargement of left atrial internal dimension. Pacing results in flattening of aortic wall that is most evident at 160 impulses per minute. AV, Aortic valve; AR, aortic root; and LA, left atrium.

Under these conditions the left atrium has a greater pressure and volume, thereby functioning as a conduit, rather than a booster pump, at a critical level of distension. The role of tachycardia-related atrial insufficiency in the genesis of an enlarged left atrial internal dimension is unknown. A decline in the left atrial size at a driving rate exceeding 160 impulses per minute was possibly due to a critical reduction of myocardial contractility, which resulted in poor venous return to the left atrium.

Combined Doppler and M-mode aortic echocardiographic studies in combination with an estimation of left atrial size indicates that the reduction in the velocity of blood through the aortic root occurs in conjunction with depressed oscillation of the aortic root and with left atrial enlargement at driving rates approaching 160 impulses per minute. Such study provides a multigraphic technique for assessing the influence of ventricular tachycardia on myocardial performance.