Echocardiographic Evaluation of Disturbances of Cardiac Rhythm and Conduction

The effects upon mechanical ventricular performance of disturbances of cardiac rhythm and conduction have long been a topic of intense interest to cardiovascular investigators. Nevertheless, the precise mechanical consequences of many electrophysiologic disorders remain uncertain. Recently, echocardiography has provided a noninvasive diagnostic method capable of examination of intracardiac anatomy and ventricular contractile pattern in a manner which is, in some respects, superior to cineangiography. Therefore, it was inevitable that ultrasonic assessment of cardiac function would soon be applied to the evaluation of the mechanical effects of electrophysiologic cardiac disorders.

Initially, alterations in the pattern of the motion of the mitral leaflets on the echocardiogram were observed in the presence of supraventricular cardiac arrhythmias. Thus, rapid opening and closing oscillations of the mitral leaflets secondary to phasic atrioventricular flow produced by atrial contraction were recorded in the setting of atrial flutter and fibrillation. Subsequently, the echocardiogram of the mitral valve was utilized to demonstrate that atrial transport function returned promptly following direct-current cardioversion of supraventricular arrhythmias in nearly all patients. Ultrasound has also been utilized in conjunction with vectorcardiography in studying the mechanism of atrial flutter.

Perhaps the most important contribution of echocardiography to the evaluation of electrophysiologic disorders has been the study of asynchrony of left ventricular contraction secondary to abnormalities of cardiac conduction. Thus, left ventricular contraction has been found to be normal in the presence of right bundle-branch block. In contrast, an abnormality of interventricular septal motion consisting of a rapid peaked posterior motion of the septum at the onset of systole followed by inappropriate anterior or flat movement has been observed in nearly all patients with left bundle-branch block. Importantly, recognition of the prevalence of this asynchronous septal movement in left bundle-branch block was possible only by echocardiographic studies. In this issue of Chest (see page 463), Lebovitz and associates have turned their attention to the mechanical consequences of another abnormality of cardiac conduction, the Wolff-Parkinson-White syndrome. Although several prior reports have utilized echocardiographic studies to evaluate the left ventricular contractile pattern in patients with the Wolff-Parkinson-White syndrome, uniform agreement has not been reached in regard to all ultrasonic findings. Previously, it had been observed that many patients with type-A Wolff-Parkinson-White syndrome manifested premature contraction of a portion of the posterobasal left ventricular wall on the echocardiogram. The prematurely contracting area detected by ultrasound was highly localized and was found to be related to the proportion of ventricle activated by the accessory pathway, as represented by the prominence of the delta wave of the electrocardiogram. Although the contractile pattern of the interventricular septum was normal in patients with type-A Wolff-Parkinson-White syndrome, the premature posterobasal contraction appeared to produce an early systolic anterior movement of the septum, resulting in a delay in the onset of net posterior motion of this structure. Contractile abnormalities of the posterior wall were not observed in patients with type-A Wolff-Parkinson-White syndrome by Francis and associates or in the present study; however, the former investigators did not perform an exhaustive search of the posterobasal wall, and, thus, the subtle prematurely contracting area of myocardium may not have been recorded.

Patients with type-B Wolff-Parkinson-White syndrome, in contrast, have been observed to have normal posterior wall motion on the echocardiogram; however, abnormal interventricular septal contraction has been recorded in some, but not all, type-B patients. The abnormal septal motion in patients with type-B Wolff-Parkinson-White syndrome was found to resemble that observed in patients with left bundle-branch block, in that a peaking posterior movement of the interventricular septum occurred in early systole; however, Chandra and colleagues were unable to record such an early contraction in their study, and Francis and associates additionally observed a prominent late systolic notch in the systolic motion of the interventricular septum in patients with type-B Wolff-Parkinson-White syndrome. More detailed analysis of the electrophysiologic characteristics of the bypass tract in larger groups of patients has indicated that septal motion is abnormal in most patients with type-B Wolff-Parkinson-White syndrome in whom the accessory pathway results in early activation of the anterior right ventricular wall, but not those in whom initial activation occurs in the posterior wall of the right ventricle. In the present study, Lebovitz and colleagues incriminate another variable in the genesis of abnormal ventricular contraction in patients with Wolff-Parkinson-White syndrome in reporting that asynchronous movement was observed only in their patients in whom the
duration of the QRS complex was greater than 130 msec.

That disparities should exist in the echocardiographic findings in the Wolff-Parkinson-White syndrome in different studies is not surprising in view of the recent recognition of the multiplicity of atrioventricular pathways exhibited by patients with this disorder. Moreover "M" mode echocardiography is limited in the expanse of left ventricular circumference which is able to be examined, and any ultrasonic evaluation of the contractile pattern in patients with the Wolff-Parkinson-White syndrome must include high-quality tracings of as much of the ventricle as possible. Thus, the requisite conditions for the echocardiographic recording of asynchronous left ventricular contraction in patients with the Wolff-Parkinson-White syndrome include both sufficient size and adequate location of the mass of myocardium undergoing premature activation.

With these concepts in mind, therefore, certain generalizations regarding abnormalities of left ventricular contraction in the Wolff-Parkinson-White syndrome emerge. Thus, patients with type-A Wolff-Parkinson-White syndrome may be expected to manifest premature contraction of the left ventricular posterobasal wall on the echocardiogram when the accessory pathway results in premature activation of a substantial area of myocardium in the area of the left ventricular posterior wall. Patients with type-B Wolff-Parkinson-White syndrome may be expected to exhibit echocardiographically abnormal motion of the interventricular septum similar to that found with left bundle-branch block when electrical recordings indicate that the bypass tract depolarizes a substantial area of myocardium in the anterior wall of the right ventricle. Echocardiograms may be expected to be without abnormalities of left ventricular contraction in type-A patients with initial activation of the anterolateral left ventricular wall, type-B patients with accelerated conduction to the posterior right ventricular surface, or in either type-A or type-B patients in whom the QRS complex of the ECG is consistent with premature depolarization of only a small mass of myocardium.

Whether the echocardiogram or electrical records are more accurate in indicating the course of the accessory tract in those patients in whom the ultrasonic pattern anticipated from electrophysiologic data is not observed remains an important area for future investigation; however, it seems certain that echocardiography will continue to contribute to the understanding of the Wolff-Parkinson-White syndrome, as well as to other disturbances of cardiac rhythm and conduction.

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