Angiographic assessment of left ventricular function

Michael V. Herman, M.D.
New York, New York

Asynergy

In patients with coronary heart disease, the ventriculogram provides important data about the size, shape, and ejection pattern of the left ventricle. Studies of the motion of individual segments of the myocardium can provide information about local disturbances in contraction (asynergy) as well as information about the residual contractile pattern of normal zones of the ventricle. Approximately 35% of patients referred for assessment of clinical coronary artery disease show a normal ventricular contraction pattern at rest. The remaining 65% demonstrate some type of resting left ventricular asynergy. Results of previous studies have indicated that these abnormalities most commonly occur in patients who had myocardial infarction, but asynergy can also occur without evidence of infarction. The patterns may range from the classic well-demarcated bulging left ventricular aneurysm to various types and degrees of abnormal motion of a segment of the heart without change in overall contour. Asynergic segments may be dyskinetic, akinetic, or hypokinetic, and the area may be composed of muscle or scar or both. A disturbed temporal sequence of contraction (asynchrony) may also be observed.

One might hypothesize that the pattern of ven-

tricular contraction that is normal at rest could be disrupted during acute ischemia, at which time segmental or global wall motion may abruptly become abnormal. This has been observed experimentally in animals. In man, wall motion may be studied during stress known to precipitate ischemia (such as tachycardia induced by rapid atrial pacing). The demonstration of a new asynergic zone on ventriculogram under such conditions suggests deficient perfusion of the myocardial segment. This can provide critical information about the severity or significance of a given local coronary arterial obstruction.

Conversely, when the resting ventriculogram is abnormal, the effect of an intervention that augments contraction might provide valuable information. Although the abnormal zone in some patients may appear isolated and surrounded by normally contracting muscle, in others poor contraction of ventricular wall may be extensive without clear-cut demarcation between normal tissue, viable but nonfunctioning ischemic muscle, and frank avascular scar. Furthermore, overall contractile activity may be depressed below its usual level for a variety of reasons. The differentiation of those segments of muscle potentially capable of being stimulated has great clinical importance. Two interventions during ventriculography have been employed: sublingual nitroglycerin and the potentiated postextrasystolic beat. Each of these interventions appears to unmask zones of previously noncontracting muscle that appear capable of shortening in certain patients with coronary artery disease. In other patients, these agents demonstrate zones that are apparently incapable of any contractile response and are presumably irreversibly damaged. The use of nitroglycerin requires a second ventriculogram that is performed after a control study. The postextrasystolic potentiation technique employs a pulse generator attached to a pacemaker catheter in the right ventricle and the introduction of an impulse approximately timed in the QRS cycle to create a ventricular premature beat followed by an augmented beat during the resting ventriculogram. Thus, the augmented beat can be compared with a control beat during the same ventriculogram. Both of the techniques described define potential contractile reserve not only in normal segments but also in marginal or even scarred zones.

Currently, the angiographic left ventricular ejection fraction is the most useful measure of left ventricular function. It is also a good predictor of long-term prognosis and response to both medical and surgical therapy. The ejection fraction is determined at rest and with any intervention (e.g., nitroglycerin, pacing, and handgrip).

Ventricular aneurysm

A ventricular aneurysm may be defined radiologically as an abnormal bulge or outpouching of the margin of the ventricle, or angiographically as a segment of ventricular wall that is noncontractile or expands during ventricular systole. Aneurysms are found at autopsy in 3.5% to 20% of patients with prior myocardial infarction in a male-female ratio of 4:1. Pathologically, there is fibrosis and calcification with atrophy or absence of ventricular muscle. Thrombus is common, and adherent pericardium may be found. Aneurysms are located at the apex in 35%, the anterior wall in 35%, the posterior wall in 20%, and the septum in 10% of patients.

Hemodynamically, a small aneurysm may have little effect. A large aneurysm behaves like mitral regurgitation during ventricular systole. Blood is forced into the aneurysm (systolic expansion) and increased left ventricular work is required to expel an adequate stroke volume. In diastole, it resembles aortic regurgitation: blood returns to the body of the ventricle during ventricular filling, over and above diastolic inflow from the left atrium.

Clinically, there is a history of myocardial infarction and >60% of patients have congestive heart failure or angina pectoris or both. Arrhythmias, dyspnea, and occasional embolic episodes occur. On physical examination, cardiomegaly is frequently present; the electrocardiogram shows an infarction pattern.

The chest x-ray film is normal if the aneurysm is small and shows cardiomegaly, angulation of the ventricular border, paradoxical, absent, or decreased pulsation on fluoroscopy, and pulmonary vascular redistribution and edema in about 40% of patients. Gross calcification is observed in 5% to 20% of the patients.

Angiographic studies in the right anterior oblique projection are valuable and demonstrate the aneurysmal sac, paradoxical pulsation, or diminished wall motion. There is persistent filling of the aneurysm by the contrast agent; filling defects representing thrombi may be observed. The ventricular wall is thin if no thrombus is present. The end-diastolic volume is increased, and the ejection fraction is decreased.

There is only a 20% 5-year survival of patients with aneurysm compared to 60% of those who survive the first myocardial infarction. The cause of death is usually heart failure, thromboembolism, or recurrent infarction.

Papillary muscle dysfunction and rupture

Normal systolic closure of the mitral valve is a complex process. It is dependent upon the functional integrity of not only the leaflets themselves but also their chordal attachments, the papillary muscles, and the myocardium surrounding the papillary muscles. Normal closure also depends upon the maintenance of the proper spatial relationship between the papillary muscle attachments and the valve leaflets and annulus.

If a papillary muscle fails to contract normally because of ischemia (with or without infarction), closure is impaired and mitral regurgitation may result. In such cases, left ventriculography may show an abnormal degree of posterior motion of a valve leaflet into the left atrium in systole, often with mitral regurgitation, commonly mild, with an eccentric regurgitant jet. Asynergy of the myocardium adjacent to the papillary muscle is frequently present. Papillary muscle rupture results in a flail leaflet, usually with severe regurgitation. If multiple heads or an entire papillary muscle trunk is involved, the patient's course may be rapidly downhill with pulmonary edema and even death.

If the normal spatial relationship between the papillary muscle and the mitral leaflets is altered (as may occur with left ventricular dilation or an aneurysm), mitral regurgitation may result even in the presence of intrinsically normal leaflets, chordae, and papillary muscles. In such cases the mitral annulus may be dilated, also contributing to this dysfunction.