# Assessment of left ventricular function

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Obstructive coronary artery disease may produce abnormalities of left ventricular function which may affect its diastolic properties and/or its systolic function. Left ventricular function is an important determinant of survival and of functional capacity of patients with coronary artery disease and of those undergoing coronary bypass surgery. Thus, a proper assessment of left ventricular function is vital. It is important to evaluate left ventricular properties individually and not to interchange or confuse them. For example, left ventricular ejection fraction is a parameter of systolic function, left ventricular end-diastolic pressure is an indicator of the diastolic properties of the left ventricle, and the two will not necessarily correlate in an individual patient. Thus, the variables that are measured should relate directly to one's goals.

Precise evaluation of diastolic properties of the left ventricle is difficult in clinical practice because precise assessment of left ventricular compliance necessitates measurements of left ventricular volume frequently during diastole, simultaneous determination of high fidelity left ventricular diastolic pressures, and also of left ventricular wall mass. The clinical value of such detailed determinations is not known and the most helpful information can be obtained by an accurate assessment of left ventricular end-diastolic pressure and volume.

Left ventricular systolic function may be considered in terms of muscle function, regional wall motion function, and pump function. In intact man, assessment of left ventricular muscle function or contractile state is not easy. Velocity of circumferential fiber shortening at peak stress is perhaps the best currently available method for assessing the contractile state; however, the calculations are complex. Mean velocity of circumferential fiber shortening is easier to calculate, but is dependent on the loading conditions of the heart.

Three main kinds of wall motion abnormalities are recognized: akinesis, dyskinesis, and hypokinesis. To avoid subjectivity in assessing wall motion abnormalities, it is essential to use an external reference system that takes the movements of the patient's diaphragm and heart into account and eliminates the observer variation of drawing the long axis prior to superimposing the end-systolic silhouette on the end-diastolic image. With this system, visual recognition of akinesis and dyskinesis is easy and also is quantifiable. Quantification of hypokinesis requires definition of the normal range of wall motion. With the use of a variety of techniques, normal limits of wall motion can be described quantitatively. Unfortunately, all of these have limitations because of conceptual problems that have not as yet been overcome.

Many parameters can be used to assess left ventricular pump function. Cardiac output is not a sensitive indicator of left ventricular function, but is of value in some patients. Ejection fraction is a widely used and important parameter of left ventricular systolic function.

Perhaps the best method for assessing overall systolic ventricular performance is by construction of ventricular function curves (Starling curves). Starling curves have not found great clinical use as a measure of ventricular performance because of difficulties in obtaining several volume measurements and in controlling all hemodynamic parameters. Recently we have described a method that allows us to obtain four different end-diastolic volumes and stroke volumes from a single diagnostic ventriculogram. Four successive beats from one diagnostic left ventriculogram are analyzed – atrial pacing at 5 to 10 beats greater than sinus, atrial extrastimulus in the sinus node reset zone. postextrastimulus and sinus. Utilizing end-diastolic volumes and stroke volumes from three beats (the postextrastimulus beat is excluded), one can show that left ventricular stroke volume falls from 124 to 59 cc (p < 0.001) and ejection fraction from 0.67 to 0.52 (p < 0.001) as end-diastolic volume decreases from 188 to 123 cc (p < 0.001). However, mean rate of circumferential fiber shortening (mean Vcf) was independent of enddiastolic volume. The technique is safe and easy to use in man. It demonstrates that ejection fraction is dependent of end-diastolic volume with rapid changes of RR interval. Therefore, both ejection fraction and enddiastolic volume should be measured when ejection fraction is used as an index of left ventricular performance, particularly if ejection fractions obtained from different angiograms are being compared. Mean Vcf appears to be relatively independent of acute changes in preload. The postextrastimulus beat, not used in the construction of function curves, allows assessment of residual wall motion and pump functions.

Parameters employed to assess left ventricular pump function are influenced by changes in preload, afterload, heart rate, and contractility. Therefore, changes in these hemodynamic influences must be taken into account when evaluating changes in left ventricular pump function.

## Tests to assess left ventricular func-

Clinically, left ventricular pressure and angiography are the most useful determinations in assessing basal left ventricular function. Ventriculography allows assessment of (1) left ventricular wall motion abnormalities, (2) left ventricular systolic pump function, (3) left ventricular end-diastolic volume, and (4) the amount of mitral regurgitation. Therefore, both of these should be of high quality and should be obtained under basal, steady-state conditions. Measurements of cardiac output, right heart pressures, coronary blood flow, and myocardial metabolism are of value in some patients. Interventions such as exercise, pacing tachycardia, nitroglycerin, and infusion of inotropic agents allow assessment of left ventricular function in the nonbasal state and are of value in some patients.

#### Effects of contrast medium

Contrast medium produces profound transient effects: (1) vasodilatation which occurs early, (2) myocardial depression, especially with coronary arteriography, (3) increase in circulating blood volume which occurs early, and (4) diuresis which occurs late. The latter two effects are the result of hyperosmolality of the contrast agents. For these reasons, left ventricular function should be assessed prior to coronary arteriography. If sequential left ventricular angiograms are utilized to assess left ventricular function, it is important that (1) the effects of the injected contrast medium from the first ventriculogram are dissipated before the second ventriculogram is performed, and (2) drugs such as nitroglycerin and additional amounts of contrast medium are not injected between the two ventriculograms.

### Spontaneous changes in left ventricular function

Left ventricular function has been assessed in sequential studies without therapeutic intervention. Ejection fraction is one of the least variable of the measurements of left ventricular systolic function (mean difference  $\pm$  1 SD: 6%  $\pm$  16% change or 0.04  $\pm$  0.10). The site and extent of wall motion abnormalities are essentially unchanged in sequential studies.

# Objective vs subjective evaluation of left ventricular angiograms

Objective evaluations of end-diastolic volume and ejection fraction by the same observer and by two different observers are highly reproducible. On the other hand, subjective evaluation, that is by visual inspection of angiograms, shows wide variability in assessing end-diastolic volume and ejection fraction.

Objective analysis of wall motion function shows it is highly reproducible (1% variance) and the incidence of interobserver variance is 8%. Subjective analysis of wall motion function shows a variance of 19% to 27%.

## Effects of coronary artery disease on left ventricular function

Coronary artery disease, per se, does not usually produce abnormalities of left ventricular function. Recent or past myocardial infarction and the presence of acute ischemia may affect left ventricular diastolic and systolic properties. The consequences of a change in the diastolic properties are important because any increase in left ventricular volume may increase the risk of pulmonary edema in the patient with a less compliant ventricle. It is possible to have abnormalities of left ventricular diastolic properties, i.e., a stiffer ventricle, without affectation of the systolic pump function of the heart. Although acute myocardial ischemia produces transient depression of left ventricular systolic pump function, the exact frequency and extent of this depression in the resting state due to ischemia are unclear. Loss of systolic pump function in the basal state is mainly related to myocardial infarction. The extent of reduction of ejection fraction can be related to the extent of wall motion abnormalities. It is possible to have segmental abnormalities of wall motion function, yet the extent of this may not be sufficient to decrease ejection fraction to the abnormal range. Patients with congestive heart failure usually have severe reduction of ejection fraction. The effects of a left ventricular aneurysm on ventricular function are complex.

#### Summary

Left ventricular performance is complex and several functions are involved. In the presence of coronary artery disease, there may be a considerable range of abnormalities of each of the left ventricular functions. One or two parameters will, therefore, not describe all properties of the left ventricle and several measurements are required to understand fully the derangements of left ventricular function that may be present in any patient. Left ventricular pressure and angiography are of the most clinical value and therefore high quality recordings of these measurements should be obtained under basal. steady-state conditions. It is important to use objective techniques and to measure values from these recordings rather than estimate them by visual inspection. Spontaneous variation in parameters of left ventricular function must be taken into account. Under resting conditions, abnormalities of left ventricular function usually result from past or recent infarction and are directly related to the amount of destroyed myocardium.