Role of Stress Echocardiography in Ischaemic Heart Disease

In the presence of CAD reduced coronary blood flow will create a supply-demand mismatch resulting in myocardial ischaemia; the sequence of events that leads to the development of myocardial ischaemia is referred to as "the ischaemic cascade" (Fig. 5.17).

As mentioned, the echocardiographic identification of myocardial ischaemia due to CAD is based on the recognition of RWMA. However, patients with severe CAD may have normal LV function at rest. In these patients RWMA may be induced by increasing myocardial oxygen demand. This can be achieved by stress echocardiography which aims to increase myocardial oxygen demand in order to induce RWMA that are not present at rest.

The types of stressors used to increase myocardial oxygen demand include physical exercise (bicycle, treadmill or isometric), pharmacological stress (adrenergic stimulation [dobutamine] and vasodilation [dipyridamole and adenosine]) or rapid cardiac pacing. The normal and abnormal responses to stress are summarised in Table 5.4 and illustrated in figures 5.18-5.20.

Dobutamine stress echocardiography may also be used to differentiate between viable myocardium and irreversible myocardial necrosis (Table 5.5). Viable myocardium can be defined as dysfunctional myocardium that improves contractile function when an adequate coronary blood flow is restored. Viable myocardium can be classified as "stunned" or "hibernating". Stunned myocardium refers to the persistence of LV regional dysfunction after transient coronary occlusion; that is, recovery of wall motion abnormalities is delayed despite adequate reperfusion therapy or restoration of normal coronary flow. Hibernating myocardium refers to chronic LV regional dysfunction when there is severe CAD and chronic ischaemia; complete or partial recovery of function occurs following coronary revascularisation.

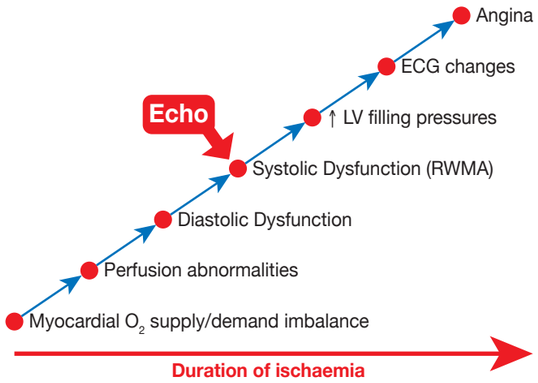

Figure 5.17 The ischaemic cascade is a series of pathophysiologic events caused by myocardial ischaemia. Coronary artery disease causes a reduction in myocardial blood flow which leads to a supply-demand imbalance. This is followed by perfusion abnormalities, diastolic dysfunction, systolic dysfunction (or regional wall motion abnormalities [RWMA]) and a subsequent rise in left ventricular (LV) filling pressures. This is then followed by ECG changes and angina. The primary role of echocardiography in this cascade is to identify RWMA.

Table 5.4 Normal and Abnormal Responses to Stress

	Following Stress
Normal Response	• Uniform increase in regional wall motion (becomes hyperdynamic) • Decrease in LV end-systolic cavity area • Increased LVEF
Inducible ischaemia	• Development of new RWMA • Deterioration of existing RWMA • Tardokinesia (delayed systolic contraction) • Failure to augment wall thickening • Increase in LV end-systolic cavity area • Decrease in overall LVEF

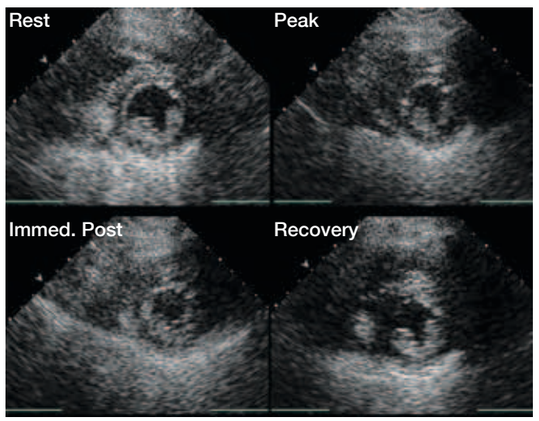

Figure 5.18 These end-systolic still frame images shown in a quad-screen format were recorded from the parasternal short axis view at the papillary muscle level. These images show a normal exercise response; observe that the LV cavity decreases in size and the walls become hyperdynamic at peak exercise (*top right*).

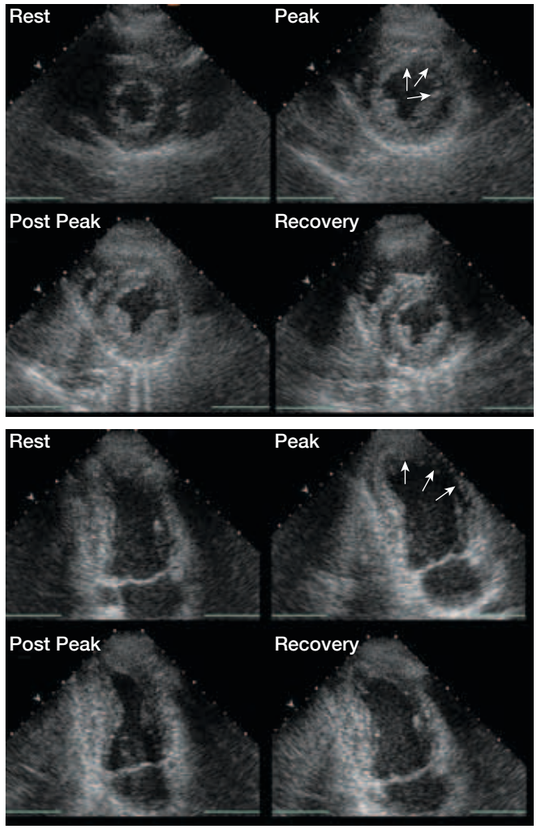

Figure 5.19 These end-systolic still frame images shown in a quad-screen format were recorded from the parasternal short axis view at the papillary muscle level *(top)* and the apical 2-chamber view *(bottom)*. These images show an abnormal exercise response; observe that at peak stress there was severe hypokinesis of the anterior wall *(arrows).*

> Stress echocardiography is a specialised technique and both the reporting physician and cardiac sonographer must be highly trained in image interpretation and image acquisition. This section on stress echocardiography covers only the very basic concepts of this technique. For a more comprehensive understanding of stress echocardiography readers are advised to review other sources as listed in the Further Reading section of this Chapter.

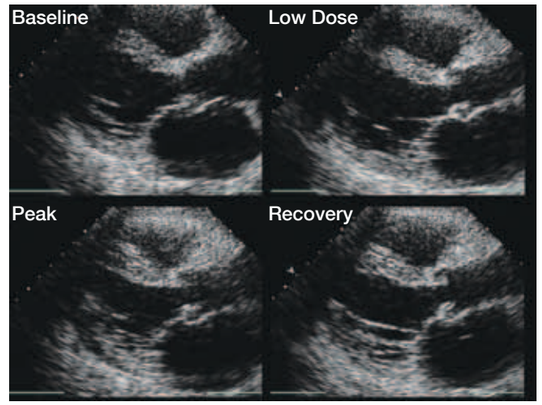

Figure 5.20 These end-systolic still frame images were recorded from a dobutamine stress echocardiogram. The quad-screen format shows images from the parasternal long axis view. These images show augmentation of all LV segments at peak dose *(bottom left)*.

Table 5.5 Viable versus Non-Viable Myocardium: Responses to Dobutamine

	At Rest	Low Dose Dobutamine	High Dose Dobutamine
Stunned myocardium	Impaired wall motion	Increased wall thickening	Further increase in wall thickening
Hibernating myocardium *	Impaired wall motion	Increased wall thickening	Reduction in wall thickening
Non-viable (infarcted) myocardium	Impaired wall motion	No change in wall thickening	No change in wall thickening

* The increase in wall thickening at low dose followed by a decrease in wall thickening at high dose dobutamine is referred to as a biphasic response. This worsening of regional function at high dose occurs due to the induction of ischaemia. This biphasic response differentiates stunned from hibernating myocardium.

Complications of Myocardial Infarction

In addition to identifying acute and prior MI, echocardiography can be used to identify the complications associated with MI. Importantly, complications can be anticipated based on the location of the MI and knowledge of the incidence, risk factors and timeframe for the development of each type of complication (Table 5.6). Complications following an AMI are now less commonly encountered due to the early reperfusion and thrombolytic therapies for STEMIs.

Left Ventricular Aneurysms

As previously mentioned an aneurysm can be described as abnormal outward bulging and deformation of a myocardial segment occurring in both systole and diastole. LV aneurysms result from the weakening of an infarcted (necrotic) wall and the pulsatile force of ventricular contractions. Therefore, aneurysms occur due to stretching and thinning of the infarcted region (infarct expansion). The wall of the aneurysm is lined by myocardium and the "neck" between the ventricle and the aneurysm is wide (Fig. 5.21). Over time, the aneurysmal wall becomes thin, fibrotic and focally calcified; therefore, LV aneurysms rarely rupture.

LV aneurysms are most commonly located at the LV apex but may also been seen in the anterior, inferolateral and inferior walls. The development of LV aneurysms usually occurs within 2 to 4 weeks following an AMI.

LV aneurysms are also referred to as "true aneurysms" as opposed to the pseudoaneurysm that develops following free wall rupture (discussed below).

By echocardiography, LV aneurysms have several characteristic features (Fig. 5.22-5.23). In particular, the neck of the aneurysm, defined as the junction between the aneurysm and the remainder of the LV, is wide with the ratio of the diameter at the neck to the maximum diameter of the aneurysm exceeding 0.5. The myocardium in the region of the aneurysm also appears thinned and echogenic; during systole the infarcted area bulges outward. Mural thrombus and/or spontaneous echo contrast (SEC) may also be seen within the aneurysm.

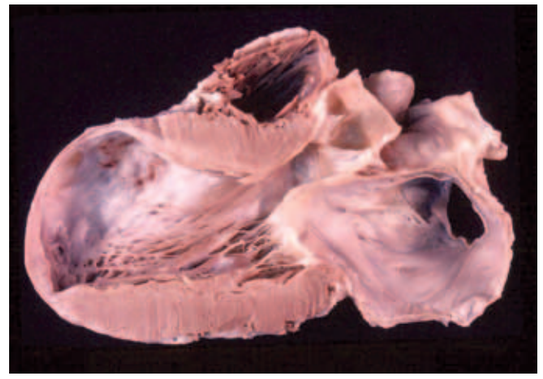

Figure 5.21 This gross pathological specimen shows the characteristic features of a post-infarction true aneurysm of the left ventricle (LV). Observe that the basal to mid septum and inferolateral LV wall are normal in thickness. The aneurysm is seen at the LV apex and apicoseptal regions. Note that the myocardium of the aneurysm is stretched and thinned and the "neck" between the ventricle and the aneurysm is wide.