Atherosclerotic coronary artery disease remains one of the leading causes of death in western civilization. The severity of coronary atherosclerosis however found in patients who die suddenly shows a remarkable variability suggesting that other factors superimposed on a variable degree of atherosclerosis may substantially contribute to sudden death [1]. Acute myocardial ischaemia is by itself responsible for the fatal event, whereas the anatomic lesions of the coronary arteries may only favour or predispose the development of such ischaemia [2].

Angina is only one of the possible manifestations of acute myocardial ischaemia, which can be totally asymptomatic or appear as sudden and unexplained acute left ventricular failure or as arrhythmias. Yet it is the symptom that constitutes the ‘alarm’ signal of myocardial ischaemia obliging the patient to undertake action and stop the pain and therefore the myocardial ischaemia.

In order to determine the anatomical distribution of obstructive coronary arterial lesion, coronary angiography is required. However, the latter is insufficient as it provides no information regarding the amount of blood supplied and therefore oxygen upon the myocardium. To ascertain the physiologic significance of coronary arterial narrowing, namely the presence and distribution of ischaemia, the use of a stress test is necessary. The current traditional modes of stress testing utilise graded treadmill or bicycle protocols with measurement of heart rate, blood pressure and electrocardiographic parameters, aiming to reproduce the patients clinical symptoms, namely the anginal pain. However, observations during exercise stress testing occasionally show the development of typical ischaemic electrocardiographic changes in the absence of anginal pain or long before its appearance (see chapters 6 & 8). Therefore these superficial measurements only constitute the ‘tip of the iceberg’ of myocardial ischaemia.

Echocardiography is perhaps the most readily available technique to

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provide the clinician with a complete assessment of left ventricular systolic performance by directly imaging the myocardial contractility. Furthermore, a rapidly developing area of cardiac ultrasound that is of great interest, is the use of ultrasound contrast techniques to determine myocardial perfusion and coronary risk areas.

**Exercise echocardiography**

Exercise stress testing is widely used in the diagnosis and assessment of patients with known or suspected coronary artery disease. The haemodynamic response to a maximal symptom-limited treadmill exercise stress test in a normal subject includes a one-and-a-half-fold increase in stroke volume and a three-fold increase in heart rate. The systolic arterial pressure increases on average by 60–80 mmHg and the diastolic arterial pressure decreases by about 10–20 mmHg [3].

Stress or exercise two-dimensional echocardiography has been demonstrated to be effective in eliciting new regional wall motion abnormalities by multiple investigators. These wall motion abnormalities correlate well with corresponding coronary artery atherosclerotic lesions. A wide variety of stress tests have been utilised. Handgrip, cold pressor test, supine bicycle exercise, upright bicycle exercise, treadmill exercise as well as pharmacologic interventions and pacing have all been used. Handgrip exercise did not produce ischaemic changes with sufficient frequency to be practical. Cold pressor tests are extremely painful and are not tolerated well by the patient. In addition, it too does not produce ischaemia with sufficient frequency. Supine bicycle exercise offers a problem with leg fatigue which limits the ability of the patient to generate a sufficient heart rate to produce ischaemia. Upright bicycle exercise is an excellent technique for performing echocardiograms during exercise. This examination is perhaps best done if the patient’s upper body is supported in some fashion so that it remains immobile during cycling.

An increasingly popular technique is obtaining a two-dimensional echocardiogram immediately post-treadmill exercise. There was however an initial concern that the wall motion abnormalities would revert back to normal too rapidly to permit clinically useful information. However, subsequent experience demonstrated that such concerns were unfounded; several investigators including ourselves, have now demonstrated that this is not the case. Wall motion abnormalities persist for several minutes so that there is plenty of time for the patient to lie on a couch still connected with the epicardial 12 lead electrocardiogram and obtain optimal two-dimensional echocardiographic images.