Valvular heart disease promises to be another area where radionuclide ventriculography (RNV) can be extremely useful in patient management. Although radionuclide studies can often define valvular heart disease, they are not cost-effective tests for diagnostic screening, yielding to the physical examination and ultrasound studies. RNV is helpful, however, in left ventricular (LV) assessment and in evaluation for associated coronary artery disease (CAD). The positive decision, and timing for surgical intervention and valve replacement, are often difficult for the clinician. The optimal time of replacement, particularly for volume overload lesions, should be as late as possible, but before irreversible myocardial damage occurs. The situation in valvular stenosis is better defined with valve area measurements, e.g., changes, and the clinical picture major determinants for surgical intervention. Two-dimensional echocardiography is a useful non-invasive test in this group. This chapter will briefly discuss application of RNV in valvular heart disease and give case examples.

LEFT VENTRICULAR VOLUME OVERLOAD

Both aortic and mitral regurgitation (AR and MR) are usually well tolerated clinically, but these lesions lead to significant degenerative myocardial changes which may occur even before the patient develops clinical symptoms (Boucher et al., 1981). Johnson et al. (1983), suggest that LV volume determination by RNV studies yields more useful information about myocardial functional reserve than the ejection fraction (EF) alone. LV volume can be approximated accurately from first pass RNV studies (Nickel et al., 1982). However, functional imaging adds a new dimension in patient management in that characteristic wall motion changes often are seen even before global parameters become abnormal. RNV studies have been shown to be reproducible (Upton et al., 1980), but it is essential that serial studies be performed with the same patient position (i.e. supine vs erect) as illustrated by Manyari and Kostuk (1983). Exercise tolerance is usually preserved in patients with regurgitant lesions (Levinson et al., 1970) and for this reason there is need for a good non-invasive study which can detect myocardial dysfunction before irreversible symptoms develop.

AORTIC REGURGITATION

Borer et al. (1978) and Wexler et al. (1981) suggest that surgical intervention for aortic insufficiency should be performed at a time when resting function is normal, but exercise response is diminished. Becoming somewhat disenchanted with EF response to exercise as a reliable indicator of dysfunction, Steingart et al. (1983) developed a regurgitant index (LV stroke counts/RV stroke counts) and found this number significantly higher in patients with AR. This index usually decreased with exercise in these patients and may be the mechanism for preservation of exercise tolerance in patients with AR. However, Nicod et al. (1982) could not accurately differentiate between 2+, 3+, and 4+ aortic regurgitation using this index. This leaves timing of aortic valve replacement controversial (Selzer et al., 1976; Rahintoola, 1977; O’Rourke and Crawford, 1980).

Wall motion abnormalities are seen by contrast ventriculography in patients with chronic volume overload from either AR or MR with a measurable decrease in velocity of fibre shortening (Osbakken et al., 1981). In all patients, however, global function (EF) was below normal. Wall motion evaluation with functional imaging using RNV reveals characteristic abnormalities which can be followed non-invasively.

Case 1: Figures 13.1 and 13.2

This 58-year-old female presented with palpitations and back pain. RNV shows typical wall motion changes seen in association with AR. There is a central, cone-shaped abnormality seen best in the RFI and mean transit time images which does not extend to the cardiac apex but increases with exercise. End-diastolic volume decreases from 220 ml at rest to 196 ml at exercise and ejection fraction decreases from 49% to 37%. Cardiac output and cardiac index both increase with exercise, accounting for the lack of symptomatology. Associated ischaemia cannot be totally excluded in the presence of valvular heart disease as both conditions lead to similar wall motion changes (Hecht and Hopkins, 1981); however, with ischaemia, function-
al images usually are less symmetrical and the abnormality may disrupt the apical perimeter during early systole (see Chapters 4 and 5).

Case 2: Figures 13.3 and 13.4

This 24-year-old essentially asymptomatic male with known AR presumably due to rheumatic fever underwent RNV as part of a routine evaluation. Studies revealed severe enlargement of the left ventricle (EDV 393 ml) with a central wall motion abnormality which does extend to the apex and which can be confused with ischaemia. Following exercise, wall motion deteriorated further but cardiac output and index were preserved. Because of the severe abnormalities seen by RNV, the patient underwent cardiac catheterization where he was found to have a markedly dilated LV, 4+ AR, and normal coronary arteries. There was no MR. After valve replacement, function and wall motion have improved dramatically.

MITRAL REGURGITATION

Parameters for following patients with MR are more complex than for those with AR because the EF is usually normal and occasionally supernormal because of the decreased resistance created by the incompetent mitral valve (Bolen and Alderman, 1977; Miller et al., 1965; Kennedy et al., 1970). In actuality, EF in this situation is an additive of both forward (aortic) ejection as well as the regurgitant fraction (mitral) which passes back into the left atrium. A more accurate measure of function would be to calculate only the percentage ejected into the aorta with systole, but present methods of calculation (area/length and LV counts) are concerned only with what leaves the ventricle by either direction. Invariably, patients with MR show a significant decrease in EF following surgical valve replacement (Boucher et al., 1981). To date, no method of preoperative evaluation can accurately predict the postoperative EF (Phillips et al., 1981).

Using first pass RNV, a method for determining the ‘forward’ or true ejection fraction has been developed which in fact adds the regurgitant left atrial counts back to the LV counts before calculation is made. This corrected ejection fraction equals LV end diastolic counts − (LV end systolic counts + left atrial counts) ÷ end diastolic counts (see Figure 13.11). After surgery which closes the lower resistance regurgitant pathway, the overall ventricular residual resistance is increased, which results in a lower EF. However, with readjustments in volume (fibre length) in the postoperative period, some improvement in global function can be hopefully expected. In a series of five patients, the corrected ejection fraction correctly predicted the postoperative EF (Table 13.1).

Wall motion changes seen in association with MR are also common and similar to those described with AR, but MR usually produces abnormalities oriented more toward the LV septum.

| Case 3: Figure 13.5 |

This 74-year-old male with a cardiac murmur since childhood presented with a recent episode of mild congestive failure. Initial RNV study showed good global function (EF = 58% at rest and 52% with exercise) and mild diffuse wall motion changes most pronounced in the septal area. The corrected ejection fraction, however, was calculated to be 39% at rest. Subsequent cardiac catheterization revealed severe MR, trace AR, and normal coronary arteries. He underwent mitral valve replacement and the resting EF immediately after surgery was 33%. This increased to 52% 2 weeks later and at 1 year, function remains normal with EF 55% at rest and 58% with exercise. Wall motion has also returned to normal.

| Case 4: Figure 13.6 |

This 42-year-old female was seen for cardiac evaluation prior to eye surgery. RNV study revealed central wall motion changes similar to those described in association with AR. In this case, EDV was 258 ml, EF 43% at rest and exercise. Enlargement of the left ventricle with associated decrease in velocity of fibre shortening produces the central abnormality.

LEFT VENTRICULAR PRESSURE OVERLOAD

Pressure overload produces a much different effect upon the LV than volume overload and consequently different considerations regarding surgical intervention. Pichard et al. (1981) showed a reduction in coronary vascular reserve in patients with LV hypertrophy secondary to hypertension. Rabino­witz and Zak (1975) state that myocardial metabolism is almost exclusively aerobic with substantial quantities of adenosine triphosphate (ATP) supplied by mitochondrial oxidation of fatty acids and carbohydrate substrates. The ATP requirement increases significantly with stress and is usually met by increased mitochondrial synthesis. Attarian et al. (1981) have shown that LV hypertrophy results in lower endocardial blood flow, lower subendocardial high-energy phosphate stores, and depressed mitochondrial function. Therefore, the myocardium is more susceptible to ischaemia. Wangler et al. (1982) also demonstrated decreased coronary reserve in patients with hypertension and cardiac hyper-