Atlas of
HEART-LUNG TRANSPLANTATION

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Preface

This color atlas of heart and lung transplantation provides a pictorial description of the operations currently performed for end-stage heart and lung failure. It will provide for the cardiothoracic surgeon a detailed account of the steps carried out for each procedure and for the cardiologist an overall understanding of the complex operations by some of the recognized authorities in their field.

Implanting left ventricular assist devices, performing cardiomyoplasty, and heart and lung transplantation are supersurgical operations that require photographic memory of the procedures, exquisite know-how, and the delicate art of finger calisthenics. We hope this artistic atlas will be a useful and enjoyable guide to enhance the technical skills of the surgeon.

This atlas is also a companion to our textbook *Cardiomyopathies and Heart–Lung Transplantation*, McGraw-Hill, Inc. The text in the atlas is kept to a bare minimum and emphasis is on the practical and technical aspects of the various procedures. Tables, photographs, graphics, and line drawings are generously employed to highlight the operative techniques.

The atlas is divided into six parts. Part I provides an overview of cardiomyopathies and heart transplantation evaluation. It also includes the techniques of implanting left ventricular assist devices and dynamic cardiomyoplasty. Part II deals with thoracic organ donor procurement, management, and surgical operation. In Part III techniques of heart transplantation and endomyocardial biopsy are discussed. Heart–lung transplantation that has become technically feasible is illustrated in Part IV. Part V illustrates lung transplantation and bronchial anastomosis. In Part VI, complications, histopathology, xenotransplantation, and results of heart and lung transplantation are presented in a pictorial format.

We thank McGraw-Hill for allowing us to reproduce illustrations published in *Cardiomyopathies and Heart–Lung Transplantation*. We thank the medical illustrators who were involved in the production of this atlas; we especially thank Genevieve Wilson for her masterful artwork. Dr. Jane Pennington of McGraw-Hill deserves special thanks for her perseverance and her editorial ability in seeing this atlas to completion.

We are indebted to all the talented contributors, who are among the recognized leaders in their field and who shared their skill and innovations with our readers.

AMAR S. KAPOOR
HILLEL LAKS

Contents

Part I

Dilated Cardiomyopathies

Chapter 1

End-Stage Heart Disease

Amar S. Kapoor

The spectrum of cardiomyopathies is diverse, but the dominant feature is abnormality of heart muscle. Dilated cardiomyopathy (DCM) is a multicausal syndrome characterized by severe myocardial damage resulting in ventricular enlargement, systolic contractile dysfunction, and often myocardial failure with symptoms of congestion. DCM represents a final common pathway of many conditions that produce myocardial damage.[1,2] In some of the secondary dilated cardiomyopathies, the cause of myocardial abnormality may include alcohol abuse; viral infections; immunologic disorders; a variety of toxic, chemical, and physical agents; systemic arterial hypertension; pregnancy; and disease of the small arteries. DCM is by far the most common type of cardiomyopathy. Some potentially reversible causes of DCM are shown in Table 1-1.

TABLE 1-1 Potential reversible causes of DCMs

Alcoholic cardiomyopathy
Hypocalcemia
Hypokalemia
Hypophosphatemia
Pheochromocytoma
Myocarditis
Sarcoid heart disease
Lead poisoning
Selenium deficiency
Uremic cardiomyopathy

ETIOLOGY

There is an elaborate list of causes of secondary cardiomyopathies. A very common etiology encountered in patients referred for heart transplantation is ischemic cardiomyopathy, which is really end-stage ischemic heart disease. End-stage ischemic heart disease may result from fixed obstructive coronary artery disease and multiple ischemic insults and myocardial infarcts. An ischemic insult will set up a sequence of pathophysiologic events resulting in a whole spectrum of ischemic syndromes, as shown in Fig. 1-1. Myocardial ischemic episodes may have a cummulative effect resulting in ventricular dilation, even in the absence of myocardial necrosis. A frequently encountered entity is the so-called hibernating myocardium, a state of chronic ongoing ischemia with imbalance between supply and demand. Once left ventricular dilation manifests itself, a progressive vicious cycle ensues, with progressive left ventricular dilation and increasing stiffness of the ventricle.

It has been speculated that viral myocarditis is a possible cause of idiopathic dilated cardiomyopathy because many patients may have histologic evidence of an inflammatory infiltrate. An inflammatory infiltrate and myocyte damage are required for the unequivocal diagnosis of myocarditis, as seen in Fig. 1-2. There is a moderate lymphocytic infiltrate. The morphologic and histopathologic criteria for the working diagnosis of myocarditis, formulated by a panel of cardiac pathologists in Dallas, are called the Dallas criteria (Table 1-2).

DCM is characterized by dilation of the ventricular cavities and atria and increased weight (Fig. 1-3 and 1-4). In the end stage of the myopathic process, there is marked attenuation of the fibers, which is a morphologic expression of excessive dilation. Ultrastructural abnormalities include increased sarcoplasmic reticulum, prominent Golgi apparatus, and coarse endoplasmic reticulum.

PATHOPHYSIOLOGY OF HEART FAILURE

Most patients with DCM eventually develop the clinical syndrome of heart failure. A series of compensatory adaptive mechanisms is initiated with the onset of heart failure (Table 1-3). Several compensatory mechanisms are activated in response to inadequate effective circulating volume. The Frank-Starling mechanism is brought into play to compensate for the depressed cardiac output (Fig. 1-5).

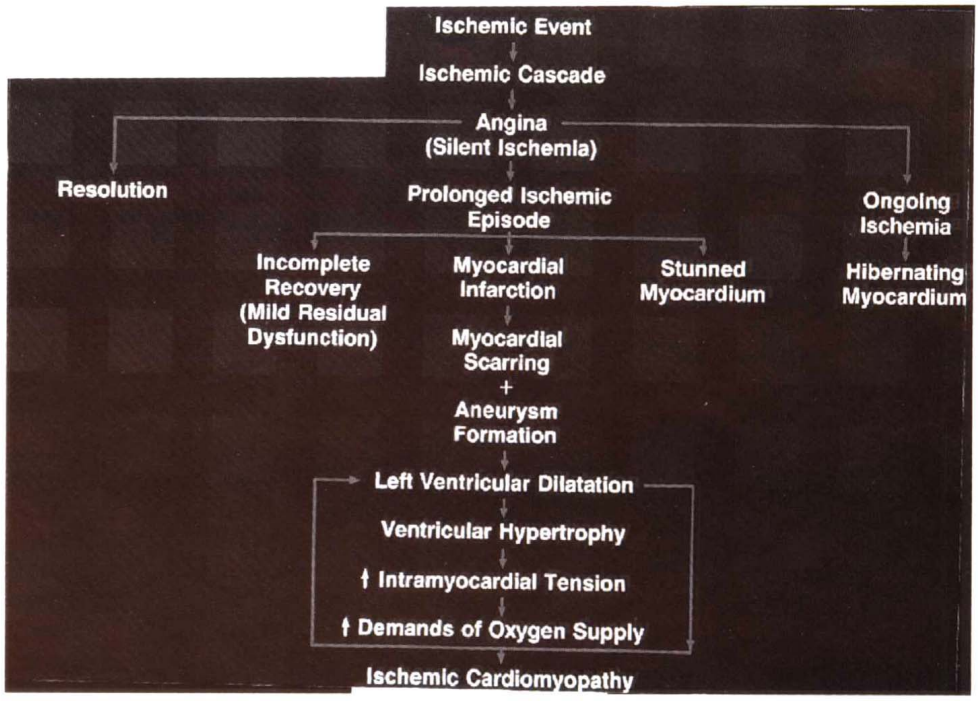

FIGURE 1-1 Pathophysiologic factors that lead to ischemic cardiomyopathy. This is a conceptual representation of possible mechanisms leading to ischemic cardiomyopathy.

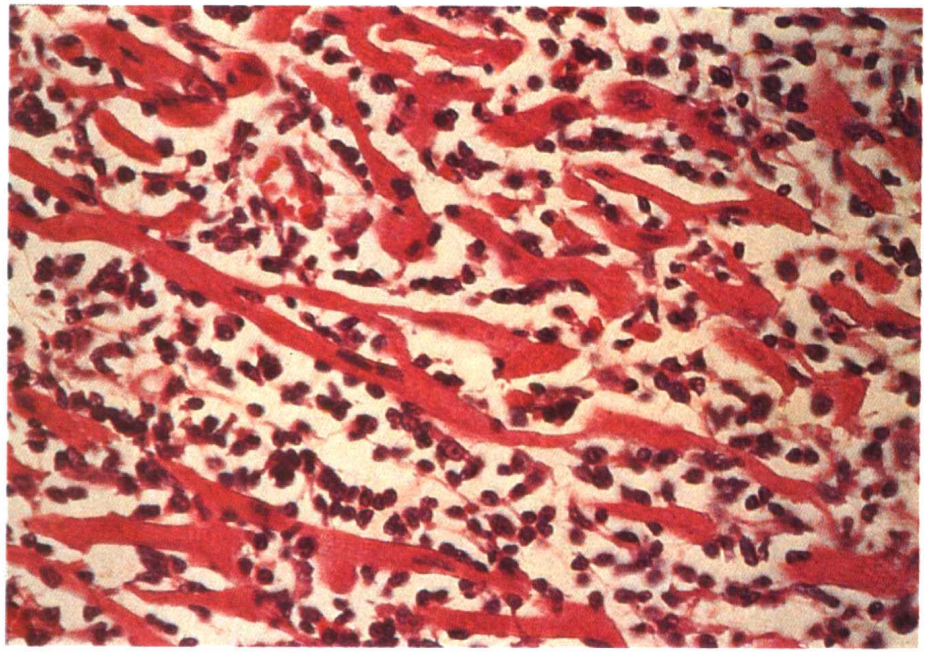

FIGURE 1-2 Histologic findings in acute myocarditis include moderate lymphocytic infiltrate with myocyte damage.

The hemodynamic adjustments are operative for maintenance of pump performance. The pathophysiologic derangements that follow in DCM are shown in Table 1-4.

Another fundamental compensatory mechanism is a reflex increase in autonomic nervous system activation. In the early stages of heart failure, activation of the adrenergic limb of the autonomic nervous system increases myocardial contractility and heart rate, which maintain cardiac output. In the later stage of severe heart failure, vasoconstriction mediated by the sympathetic nervous system and angiotensin II maintain systemic blood pressure and redistribute the peripheral blood flow to the central, coronary, and cerebral beds.

There is activation of the renin-angiotensin-aldosterone axis when there is reduction of cardiac output. Figure 1-6 is a schematic representation of the renin-angiotensin-aldosterone system in patients with low cardiac output syndrome. In the advanced stages of heart failure there are

TABLE 1-2 Dallas classification for myocarditis

First biopsy
Myocarditis with/without fibrosis
Borderline myocarditis (rebiopsy may be indicated)
No myocarditis
Subsequent biopsies
Ongoing (persistent) myocarditis with/without fibrosis
Resolving (healing) myocarditis with/without fibrosis
Resolved (healed) myocarditis with/without fibrosis
Inflammatory infiltrate subclassified as
Lymphocytic (mild, moderate, severe, focal, confluent, or diffuse)
Eosinophilic
Neutrophilic
Giant cell
Granulomatous
Mixed

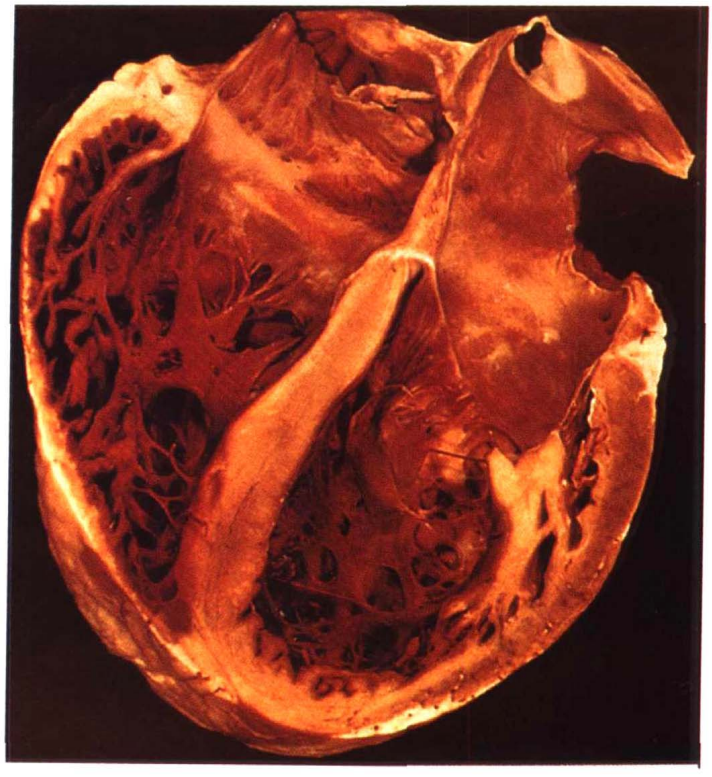

FIGURE 1-3 Pathology in DCM is characterized by dilation of ventricular and atrial chambers.

marked reduction in cardiac output, reduced flow to the kidneys, increased aldosterone levels, and active neurohumoral participation in the perpetuation of marked sodium and water retention. The degree of neurohormonal activation increases with advancing heart failure. High levels of a vasopressin cause constriction of arterial vessels to control blood pressure and also increase salt and water retention (Table 1-3). The concept of a parallel vasodilatory system as a countercompensatory mechanism that is mediated through atrial natriuretic peptide has recently been advanced (Fig. 1-7).

EVALUATION OF DCMs

The symptoms of heart failure—namely, progressive dyspnea, decreased exercise tolerance, and palpitations—can be quantified according to the New York Heart Association classification (Table 1-5). Gallops are frequently present

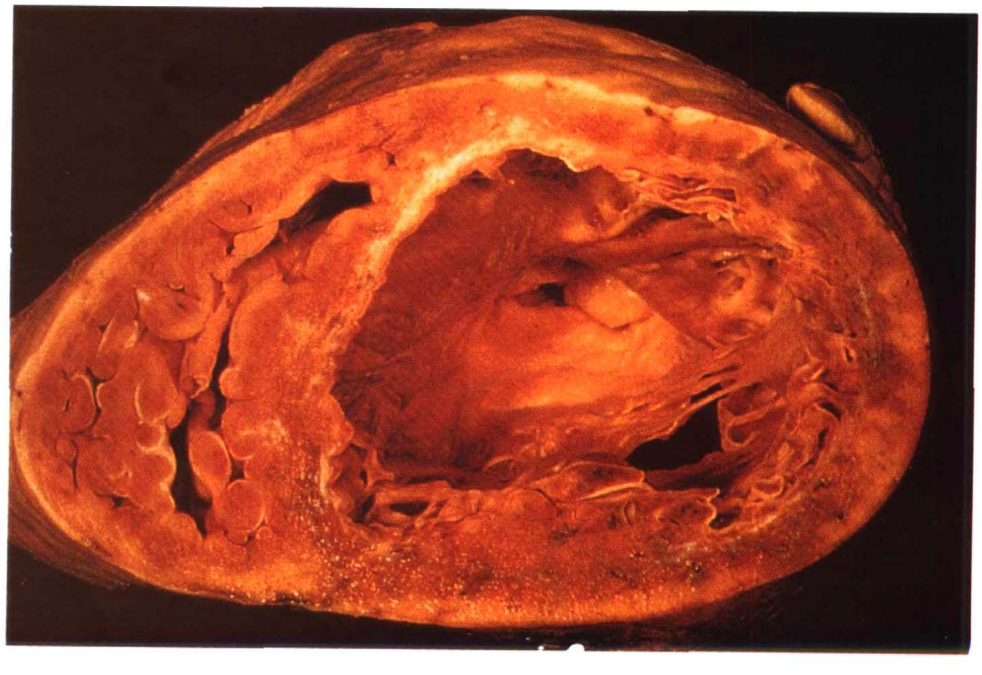

FIGURE 1-4 Cross-section of heart showing severe hypertrophy and dilation of the left ventricle.

TABLE 1-3 Compensatory mechanisms operative in heart failure

Frank-Starling law of the heart (increased preload acts to sustain cardiac output)
Myocardial hypertrophy with cardiac chamber dilation
Activation of autonomic nervous system
Augment cardiac contractility
Tachycardia
Arterial vasoconstriction
Venous vasoconstriction
Renin-angiotensin-aldosterone system activation
Arterial vasoconstriction
Sodium and water retention
Increased cardiac contractility
Redistribution of left ventricular output

TABLE 1-4 Pathophysiologic derangements in DCM

Pathologic changes
Total left ventricular mass increased
Ratio of left ventricular mass and cavity unchanged or decreased
Cavity size markedly increased
Left atrial enlargement accompanying left ventricular dilation
Right heart chamber hypertrophy and dilation
Functional derangement
Left ventricular ejection fraction profoundly reduced
Left ventricular end-diastolic and end-systolic volumes increased
Wall stress increased
Chambers stiffness increased
Myocardial stiffness increased
dP/dt decreased
Mitral and tricuspid regurgitation common

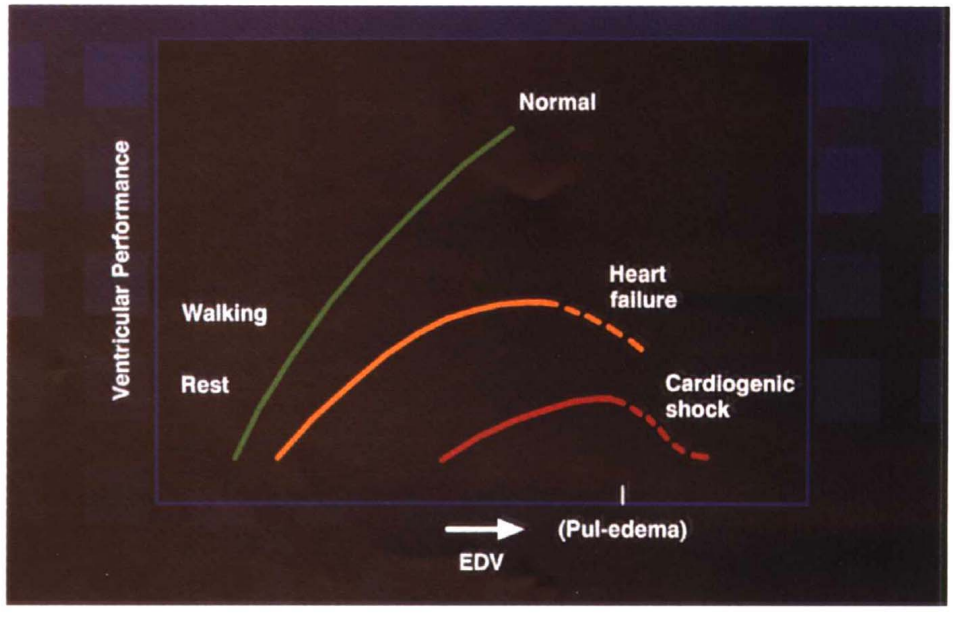

FIGURE 1-5 Diagram showing the relationship of ventricular end-diastolic volume (EDV) associated with filling pressures resulting in pulmonary edema. Ventricular performance (on the ordinate) of a normal person, the middle one with heart failure, and the lower one with cardiogenic shock. The dotted lines are the descending limbs of the ventricular performance curves.

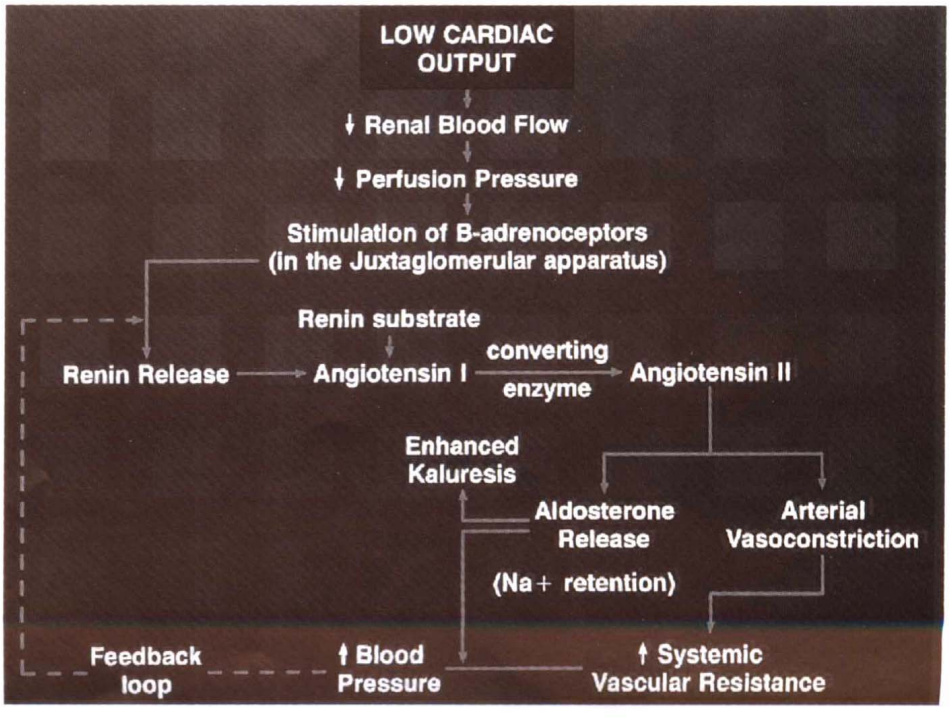

FIGURE 1-6 Schematic representation of the renin-angiotensin-aldosterone axis in patients with low cardiac output syndrome.

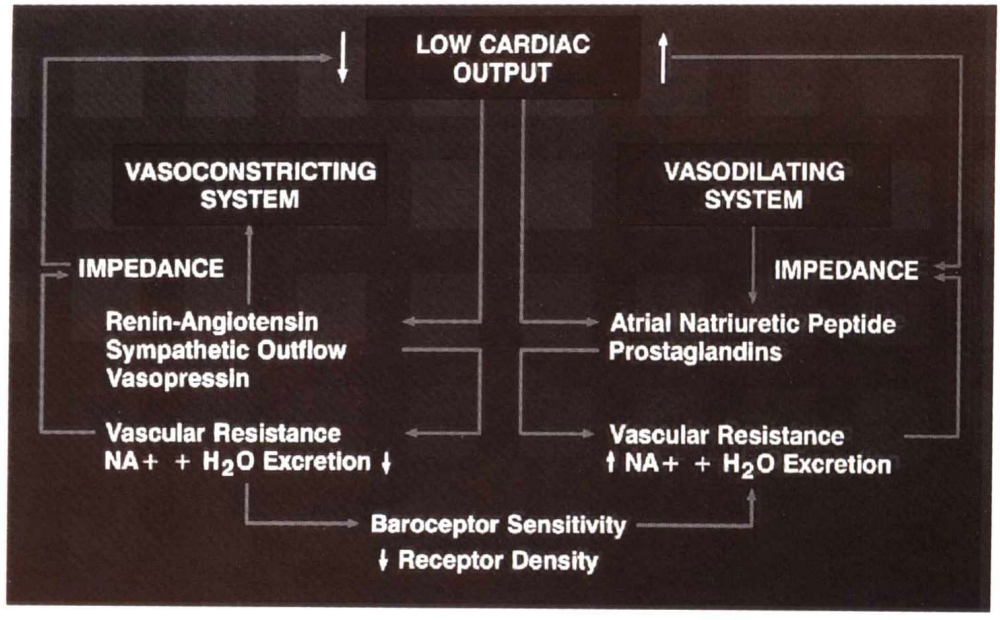

FIGURE 1-7 Diagram showing a parallel vasoconstricting and vasodilating system. A parallel vasodilatory system is activated to attenuate the deleterious effects from the overactivation of vasoconstrictive neurohumoral system (NA^+ = sodium ion, H_20 = water; ↑ = increase; ↓ = decrease).

TABLE 1-5 Functional classification of heart failure

Class	Description
I	No limitation in performing daily activities
II	Slight limitation: dyspnea or fatigue occurs with ordinary physical activity
III	Moderate limitation: minimal physical activity results in dyspnea or fatigue
IV	Severe limitation: symptoms of dyspnea are present at rest

with murmurs of mitral or tricuspid regurgitation. With biventricular failure, there is elevation of central venous pressure along with ascites and congestive hepatomegaly.

Chest x-ray (Fig. 1-8) will reveal cardiomegely, with an increased cardiothoracic ratio greater than 0.50 with a small aortic knob. Chest x-ray will also reveal the presence of heart failure with upper lobe vascular redistribution, interstitial edema, and Kerley B lines.

Echocardiographic examination is the procedure of choice in the evaluation and diagnosis of DCM. Echocardiographic studies assist in the differential diagnosis of var-

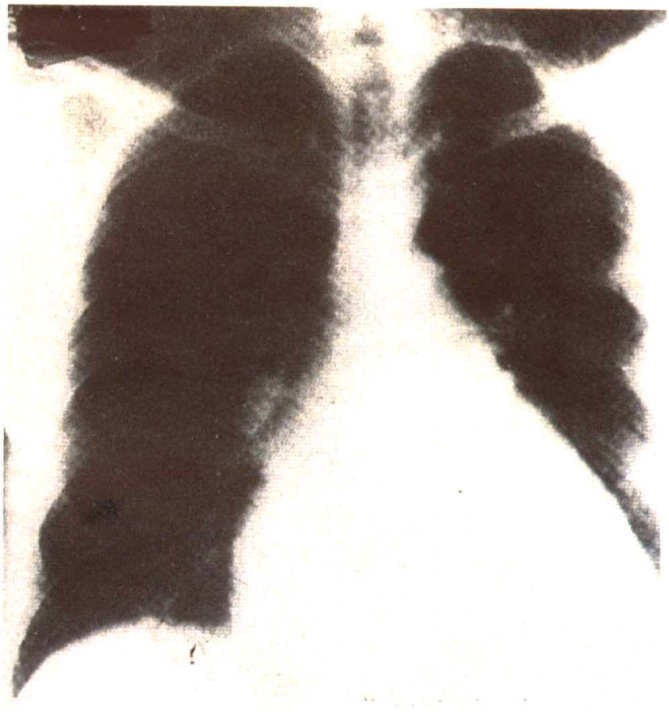

FIGURE 1-8 Chest x-ray in a patient with cardiomyopathy showing increased cardiothoracic ratio greater than 0.50, a small aortic knob, and upper lobe vascular redistribution.

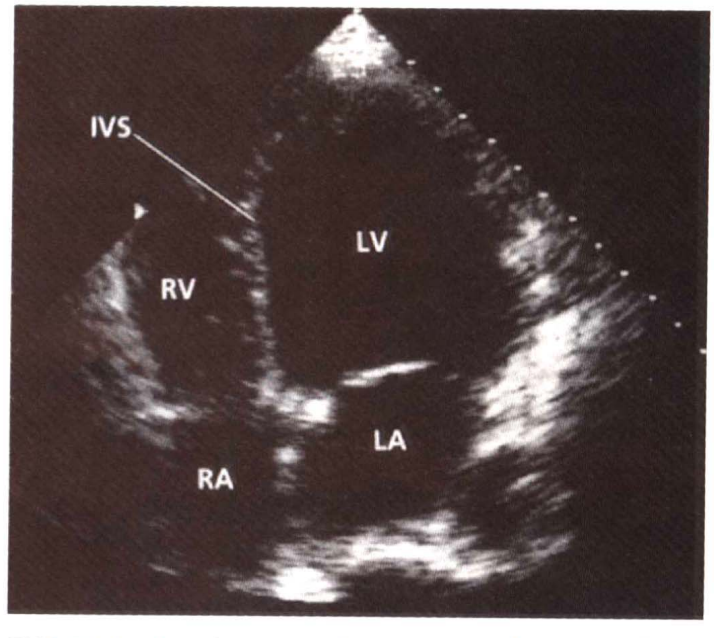

FIGURE 1-9 Two-dimensional echocardiographic study of a patient with dilated cardiomyopathy revealing four-chamber enlargement.

TABLE 1-6 Echocardiographic findings in DCM

M-mode echocardiography
Left ventricular end-diastolic dimension increased significantly
Diminished motion and thickening of septum and posterior wall
Mitral valve opening diminished
E to F slope of mitral valve closure possibly slow, with elevated filling pressures
Aortic excusion diminished
Left atrium enlarged
E-point septal separation significantly increased and characteristic of severe left ventricular dysfunction
Two-dimensional echocardiography
Hypocontractility of the left ventricle
Enlargement of cardiac chambers
Mural thrombus (when present)
Ejection fraction
Doppler echocardiography
Mitral regurgitation
Tricuspid regurgitation
Estimation of pulmonary artery pressure

TABLE 1-7 Hemodynamics in DCM

Cardiac output in normal range despite markedly decreased ejection fraction
In advanced disease state, cardiac output reduced and pulmonary capillary wedge pressure increased
Widened arteriovenous oxygen difference and increased tissue oxygen extraction
Arterial pressure maintained
Systemic vascular resistance increased
Resting tachycardia
Stroke volume or stroke work decreased

ious cardiomyopathies and primary or secondary valvular diseases and in the functional assessment of both ventricles (Fig. 1-9). In addition, one can estimate volumes, chamber dimensions, diastolic or systolic dysfunction, and the presence of mural thrombi. Table 1-6 lists echocardiographic findings in DCM. Doppler and color echocardiographic studies are sensitive methods of assessing ventricular filling dynamics and valvular regurgitation.

Cardiac catheterization can be used to measure disordered hemodynamics, quantify valvular lesions, and document coronary anatomy. Hemodynamics in DCM are shown in Table 1-7.[3]

Endomyocardial biopsy is sometimes of assistance in determining the presence of a treatable or reversible cardiomyopathy. Endomyocardial biopsy can provide direct evidence in the diagnosis of myocarditis, which requires the presence of inflammatory infiltrate and myocyte necrosis. Endomyocardial biopsy is also diagnostic in evaluating hemachromatosis or amyloidosis. Sometimes cardiotoxic drugs such as Adriamycin can cause Adriamycin cardiotoxicity, which can be identified by histologic evaluation (Fig. 1-10).

Positron emission tomographic (PET) scanning is of value in assessing patients with ischemic cardiomyopathy to document whether there are viable areas of left ventricle that could be potentially salvaged by coronary revascularization. In Fig. 1-11, there are areas of mismatch, which represent viable myocardium that is probably suitable for revascularization. Some patients with ischemic cardiomyopathy with low ejection fractions can be considered for high-risk coronary artery bypass grafting.

Cardiopulmonary stress exercise testing (CPX) can be used to objectively measure cardiac function and reserve. CPX is an extremely valuable tool in evaluating exertional

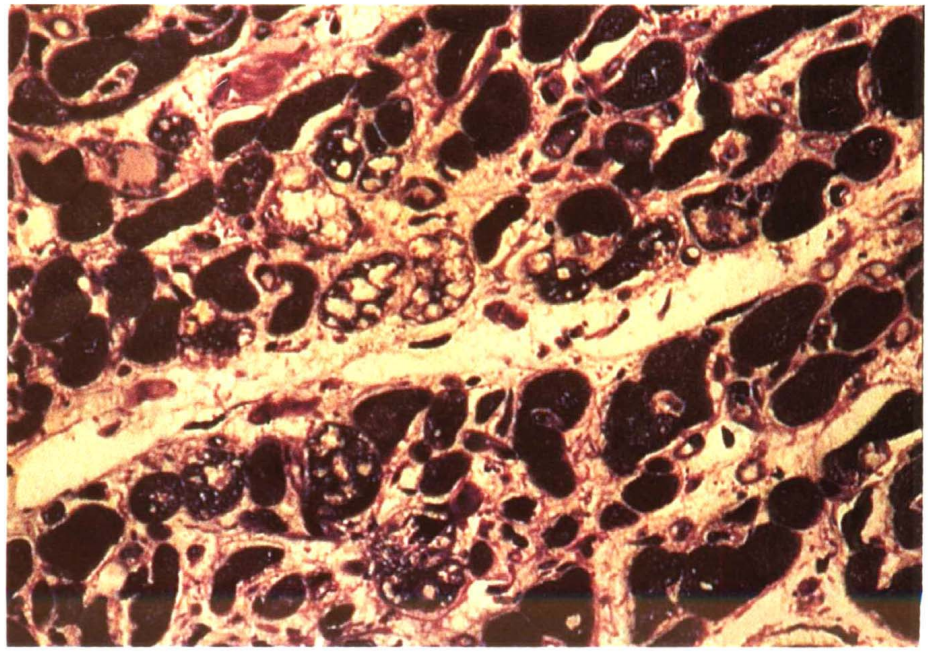

FIGURE 1-10 Electron micrograph illustrating features of adriamycin-induced cardiotoxicity with vacuolization (straight arrows). (*By permission of Springer-Verlag.*)

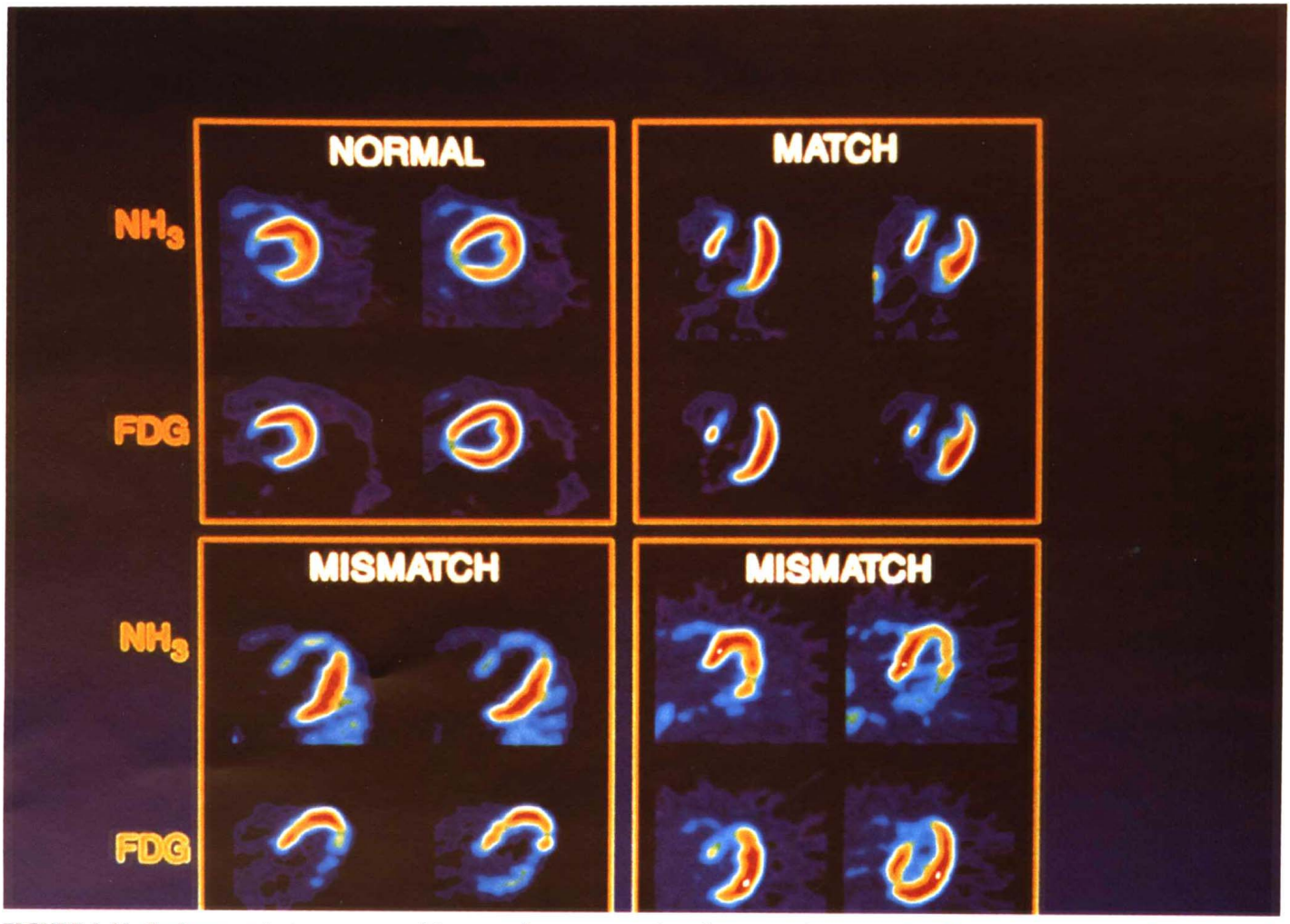

FIGURE 1-11 Positron emission tomographic scan of a patient with ischemic cardiomyopathy. The areas of mismatch show viable myocardium.

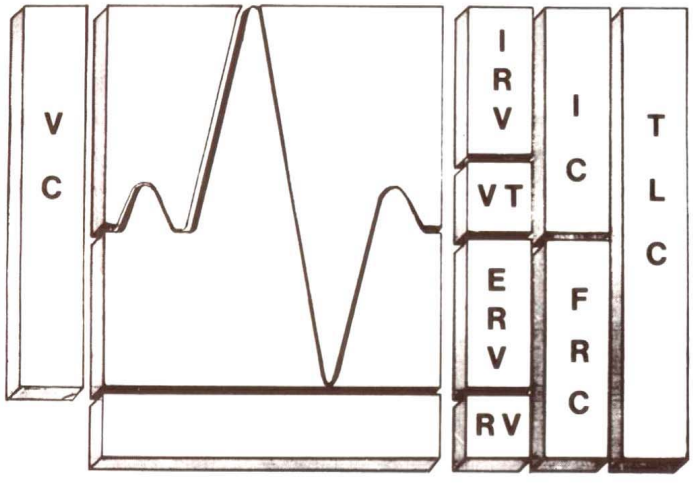

FIGURE 1-12 Lung volumes and capacities. The vital capacity (VC) includes the inspiratory (IRV) and expiratory (ERV) reserve volumes from which tidal volume (VT) is derived at rest or during exercise.

dyspnea and exercise capacity. CPX involves exercising a patient while monitoring heart rate, airflow, and gas exchange and determining maximal oxygen consumption (V_{O_2max}; Fig. 1-12). Resting oxygen consumption is 3.5 mL/min per kilogram and is equivalent to 1 metabolic unit (met). Weber and associates[4] devised a functional impairment table based on V_{O_2max}, or the anaerobic threshold, to predict cardiac reserve and the severity of myocardial failure (Table 1-8). The anaerobic threshold is determined by a disproportionate rise in carbon dioxide production (V_{CO_2}) or ventilatory equivalent (VE) for oxygen relative to V_{O_2} (Fig. 1-13).

PROGNOSTIC FACTORS IN DCM

The reported incidence of cardiomyopathy varies from 4 to 23 percent. A significant number of such patients develop heart failure. In patients who develop heart failure, the