Ten patients with ischemic cardiomyopathy and with marked cardiomegaly and "intractable" congestive heart failure were studied to determine their response to prolonged complete bed rest therapy. Ischemic cardiomyopathy is similar to other types of cardiomyopathy with severe congestive heart failure, cardiomegaly and electrocardiographic abnormalities, but is distinguished by a history of myocardial infarction or angina pectoris. The prognosis is poor since patients are subject to all the complications of cardiomyopathy, as well as to the sequelae of ischemic heart disease. A proper therapeutic approach encompasses treatment of the heart muscle disease, as well as of the ischemic basis. Management of patients with ischemic cardiomyopathy by prolonged, complete bed rest resulted in initial clinical improvement in all patients. Four patients experienced partial reduction in heart size. Two of these patients lived four and eight years, respectively, after discharge and two are presently living seven and eight months after completing bed rest therapy.

It is recognized that the most common type of heart disease in the United States today is ischemic heart disease. Clinicians acknowledge the spectrum of angina, myocardial infarction, rhythm disturbances and alterations in conduction due to myocardial ischemia. Less well appreciated, however, is the cardiomyopathy that can arise from degenerative changes in the myocardium as a result of inadequate blood supply. This syndrome, due to myocardial ischemia, offers challenge in differential diagnosis since it resembles the conventionally accepted cardiomyopathies. Ischemic cardiomyopathy also responds extremely poorly to conventional therapy, whereas with proper therapy the cardiomyopathies in general have a much better prognosis, especially when treated early. In fact, it is most important to exercise every effort to prevent ischemic cardiomyopathy, for when it once develops, prognosis is poor. Difficulties and confusion in diagnosis and management are well illustrated in a recent report.

A characteristic picture of intractable congestive heart failure and a large, dilated heart may evolve with or without preceding clues such as angina or classic clinical myocardial infarction. Patients present with a protodiastolic gallop rhythm with low blood pressure (low systolic and relatively high diastolic pressure), pallor, weakness, dyspnea, distended neck veins and edema of the tissue supplied by the systemic and pulmonary circulations so characteristic of chronic congestive heart failure involving both ventricles. These patients characteristically respond poorly to the usual therapeutic measures for congestive heart failure.

We have been interested in all types of cardiomyopathy for many years. Since 1958, however, we have studied their management with prolonged bed rest in a special research unit designed for this purpose. Because ischemic cardiomyopathy poses difficulties in diagnosis and management, clinical observations on ten patients with ischemic cardiomyopathy with large hearts, intractable congestive heart failure and clinical characteristics of classic advanced cardiomyopathy who were managed with prolonged rest in bed are summarized here.

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MATERIALS AND METHODS

Patients were referred from Tulane Medical Service of Charity Hospital and the Veterans Administration Hospital of New Orleans from 1959 to 1968 and were admitted to the special ward of the U.S. Public Health Service Hospital in New Orleans for prolonged bed rest. Although it was not planned to include ischemic heart disease among the other classic cardiomyopathies studied, it was decided, nevertheless, to study ten patients with ischemic cardiomyopathy for comparison because of its stubborn resistance to all forms of therapy and in order to learn if prolonged bed rest could benefit these unfortunate people. For admission to the study, these patients had to meet the following criteria: 1) the appearance of signs and symptoms of cardiomyopathy with persistent cardiomegaly, and 2) clinical findings of ischemic heart disease as evidenced by angina and/or myocardial infarction.

The operation of our special unit for prolonged bed rest has been previously described. Because of the deleterious effects of a hot and humid environment on myocardial function, the unit is air-conditioned.

The initial period of bed rest for these ten patients varied from 4 to 552 days (mean, 327 days) depending on the clinical response and cooperation of the patient. Ambulation was begun when no additional reduction in heart size had occurred after a six-month interval of further rest. Ambulation was graded over three months. In addition to prolonged bed rest, all patients received conventional medical therapy for ischemic heart disease and congestive heart failure, including sodium restriction, digitalis and diuretics, as well as nitroglycerin and other coronary vasodilators. A coordinated approach on the part of nursing personnel, the diet service, social service, and occupational therapy achieved optimal patient cooperation and sustained a high level of morale, so important during periods of prolonged hospitalization.

Upon discharge, patients were seen in a special cardiomyopathy clinic. They were readmitted for further periods of bed rest when necessary for recurrence of congestive heart failure, increase in cardiomegaly and other medical problems. Follow-up periods varied from two months to eight years (mean follow-up, two years).

CLINICAL DATA

Clinical information on the ten patients is presented in Table 1. The age when initially admitted to the study varied from 42 to 74 years (mean age, 58 years). All patients were men; five were Negro.

All patients had congestive heart failure. Ischemic heart disease was indicated by myocardial infarction in six patients and by angina pectoris in five patients. Other features indicative of arteriosclerotic vascular disease included transient cerebral ischemic attacks in one patient, intermittent claudication in another patient and cerebrovascular accidents in two patients.

Radiographic examinations disclosed generalized cardiomegaly with a tendency toward left ventricular prominence (Fig 1, 2, and 3). On fluoroscopy, the magnitude and vigor of the ventricular contractions were diminished.

All patients had abnormal electrocardiograms

Table 1—Clinical Data on 10 Patients with Ischemic Cardiomyopathy Treated with Prolonged Bed Rest

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age on Admission (years)</th>
<th>Onset of heart disease before bed rest (months)</th>
<th>Bed Rest Period (days)</th>
<th>Response to Bed Rest</th>
<th>Follow-up after Bed Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>51</td>
<td>84</td>
<td>519</td>
<td>Improved clinically; partial decrease in heart size</td>
<td>Living 7 months after discharge</td>
</tr>
<tr>
<td>2</td>
<td>57</td>
<td>96</td>
<td>369</td>
<td>Improved clinically; partial decrease in heart size</td>
<td>One readmission for coronary insuff. Living 8 mo after discharge</td>
</tr>
<tr>
<td>3</td>
<td>66</td>
<td>12</td>
<td>381</td>
<td>Improved clinically; partial decrease in heart size</td>
<td>Four later admissions for CHF and cerebrovascular insuff. Died 8 yrs later</td>
</tr>
<tr>
<td>4</td>
<td>55</td>
<td>72</td>
<td>552</td>
<td>Improved clinically; partial decrease in heart size</td>
<td>Four later admissions for CHF and coronary insuff. Died 4 yrs later</td>
</tr>
<tr>
<td>5</td>
<td>74</td>
<td>24</td>
<td>346</td>
<td>Improved clinically; no decrease in heart size</td>
<td>Died suddenly one month after discharge</td>
</tr>
<tr>
<td>6</td>
<td>52</td>
<td>108</td>
<td>174</td>
<td>Improved clinically; left hospital AWOL</td>
<td>Died suddenly 3 weeks after leaving hospital</td>
</tr>
<tr>
<td>7</td>
<td>49</td>
<td>72</td>
<td>329</td>
<td>Improved clinically; no decrease in heart size</td>
<td>Recurrent CHF following upper respiratory infection 2 mo after discharge. Died 2 mo later</td>
</tr>
<tr>
<td>8</td>
<td>42</td>
<td>72</td>
<td>411</td>
<td>Progressive CHF and death in hospital</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>58</td>
<td>84</td>
<td>149</td>
<td>Improved clinically; then developed pneumonia and died</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>69</td>
<td>4</td>
<td>46</td>
<td>Died suddenly in sleep</td>
<td></td>
</tr>
</tbody>
</table>

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A.) On admission
B.) After 359 days bed rest

9/13/68
9/11/69

Figure 1. Telescopic radiograms showing reduction in heart size in a 50-year-old man with ischemic cardiomyopathy after 359 days of bed rest.

A.) On admission
B.) After 380 days bed rest, C.) On second admission

9/8/59
12/21/60
4/13/62

D.) On third admission
E.) After 56 days bed rest
F.) On fourth admission
G.) On day of death

6/13/66
8/5/67
5/5/67
6/5/67

Died: June 5, 1967
Autopsy: Cardiomyopathy with hypertrophy and dilatation, Pulmonary congestion and edema

Figure 2. Serial telescopic radiograms showing variations in heart size of a 75-year-old man with ischemic cardiomyopathy following four admissions with varying periods of bed rest. Cardiac size was essentially static for eight years except for slight reduction following 380 days of bed rest during the initial hospitalization. The heart size increased rapidly during the last month of life after surviving eight years with ischemic cardiomyopathy.
indictive of ventricular septal fibrosis or myocardial degeneration. Q waves compatible with scars of previous myocardial infarction were observed in the electrocardiograms of six patients. All patients had depressed S-T segments in the standard and precordial leads with associated abnormal T waves.

In eight patients, the electrical alterations were further disclosed by spatial vectorcardiograms recorded with the equilateral tetrahedral reference system (Fig 4). Marked deformities of the QRS sE loops were observed. The QRS sE-loop was posteriorly oriented in all eight patients and superiorly oriented in seven patients. These changes were indicative of large electrically silent areas over the anterior and inferior surfaces of the heart.

All patients had normal hemoglobin and hematocrit values and hemoglobin electrophoretic patterns. Serum cholesterol values were greater than 250 mg percent in two patients. BUN was initially normal in all patients, but elevations occurred terminally in five. Four patients exhibited alterations in hepatic function as evidenced by abnormal retention of bromsulphalein dye and elevation in serum transaminase and bilirubin values which paralleled right ventricular congestive heart failure.

Hospital Course

The total duration of bed rest depended upon the clinical progress and degree of cooperation of each patient and exceeded 11 months for seven patients. According to response to the therapeutic program of bed rest, the ten patients were divided into four groups. A decrease in heart size was considered the most important sign of improvement.

Group 1: Four (40 percent) patients in whom the symptoms of congestive heart failure improved and the heart size on x-ray examination decreased.

Group 2: Two (20 percent) patients in whom the symptoms of congestive heart failure improved, but in whom there was no reduction in heart size.

Group 3: Three (30 percent) patients who died during initial hospitalization.

Group 4: One (10 percent) patient who was not able to remain hospitalized for the prescribed period of bed rest.

Initially, all patients displayed manifestations of severe intractable congestive heart failure. An unusual sensitivity to even very small doses of digitalis was reflected by frequent unifocal and multifocal premature ventricular contractions. Digitalis dosage was individualized on a daily basis. Mercurial diuretics were preferred to oral (kaliuretic) diuretics because of their greater safety in respect to arrhythmias. Clinical improvement with bed rest was slow even in those patients who responded

Figure 3. Serial televisiograms in a 51-year-old man with ischemic cardiomyopathy who failed to experience a reduction in heart size with prolonged bed rest.

(1946). Six patients were in normal sinus rhythm; two patients had chronic atrial fibrillation; two were initially in normal sinus rhythm and later developed atrial fibrillation. All patients had frequent ventricular ectopic beats. Of the eight patients presenting in sinus rhythm, three had a P-R interval greater than 0.20 sec. The mean electric QRS axis was left of 0° in the frontal plane projection in nine patients. In seven patients it was left of -30°. The electrocardiograms of six patients exhibited QRS durations of 0.10 sec or greater; one patient had a marked defect in intraventricular conduction with a QRS interval of 0.13 sec. The recordings of all patients showed slurred, notched and deformed R waves indicative of diffuse myocardial disease. Septal Q waves were absent in leads I, V₅, and V₆ of eight patients,
favorably to therapy. Reduction in heart size was not apparent on x-ray film until after six months of complete bed rest (Group 1). With improvement in congestive heart failure, the ability to tolerate digitalis increased, appetite improved and morale brightened.

Of the nine (90 percent) patients who completed therapy, four (44 percent) patients improved clinically and experienced a decrease in heart size, two (22 percent) patients experienced symptomatic improvement, but had no decrease in heart size, and three (33 percent) patients died during initial hospitalization. All three patients who died experienced clinical improvement initially, but had no decrease in heart size. One died in intractable congestive heart failure following a prolonged course of illness, one died from pneumonia and one expired unexpectedly in his sleep, presumably from a ventricular arrhythmia.

LONGTERM FOLLOW-UP

After discharge from hospital, the patients were seen at weekly or biweekly intervals in our cardiomyopathy clinic. The present status of the ten patients is indicated in Table I.

Of the four patients who initially experienced a decrease in heart size with bed rest, two are presently living. In addition to the three patients who died during hospitalization, five died subsequently. Two deaths occurred among the four patients who experienced an initial decrease in heart size with bed rest. Both of the patients of Group 2 who experienced no decrease in heart size with bed rest expired shortly after discharge from hospital. The fifth fatality occurred in a patient who did not complete the prescribed course of bed rest.

Thus, of the seven patients who were discharged after the initial period of bed rest, five subsequently succumbed to their disease and only two are presently living. Of the four patients who experienced a decrease in heart size with bed rest, two survived four and eight years after initial admission, respectively, and two are living seven and eight months following discharge. The two patients who experienced no decrease in cardiomegaly and the patient who left hospital against medical advice died shortly following discharge from hospital. There were ten subsequent hospital admissions among four of the patients for treatment of exacerbation of congestive heart failure, three (two patients) following upper respiratory tract infections. Two admissions were for management of coronary insufficiency and one was for evaluation of cerebrovascular insufficiency.

POSTMORTEM FINDINGS

Postmortem examinations were performed in five of the eight patients who died. Three patients had

FIGURE 4. Spatial vectorcardiogram and electrocardiogram of a 53-year-old man with ischemic cardiomyopathy showing right and left ventricular enlargement and diffuse myocardial disease.
large pleural effusions, two of them with associated pericardial effusion. There were no epicardial or pericardial lesions. The myocardium was generally pale and of flabby consistency. All patients had diffuse and extensive myocardial pathology.

The heart weights varied from 550 to 790 grams (mean, 658 grams). The chambers were markedly dilated with mean wall thickness of 16 mm for the left ventricle and 5 mm for the right ventricle. Mitral and tricuspid valve circumferences were increased in proportion to overall chamber dilation.

The endocardium was diffusely thickened to varying extent in all patients. In four hearts distinct fibrous endocardial plaques were encountered. These changes were most common in the left ventricle, but there was involvement of the right ventricle and both atria also. Mural thrombi were present in three patients.

The coronary arteries were generally atheromatous with reduction of the luminal diameter in multiple sites. Adjacent areas of myocardial fibrosis corresponded with arterial narrowing. There was localized extensive myocardial fibrosis of healed myocardial infarction in four patients. Involvement extended over the posterior surface of the ventricles and septum in two patients. It included the anterior wall of the left ventricle in one patient, and the apex was involved in the other.

One patient had multiple pulmonary emboli. Three patients had infarcts of the spleen or kidneys. Four patients had old pyelonephritis and two had terminal pneumonia. All patients exhibited severe visceral changes characteristic of chronic congestive heart failure.

**Discussion**

Precedent has precluded the use of the term cardiomyopathy in relation to ischemic heart disease. However, these two terms are too frequently erroneously assumed to be mutually exclusive. The most common cause of heart muscle degeneration is myocardial ischemia secondary to coronary artery disease in this country. Ischemic cardiomyopathy is similar in its clinical manifestations to the other cardiomyopathies. Clues such as angina pectoris or myocardial infarction usually point to the ischemic basis of the disease, but not infrequently these features are absent. In such cases careful evaluation and a high index of suspicion are essential in arriving at the correct diagnosis in order to avoid confusion with the other cardiomyopathies.

In the ten patients presented in this report, the onset of congestive heart failure and protodiastolic gallop rhythm with a large dilated heart led to the diagnosis of ischemic cardiomyopathy. Past medical history in eight of the patients disclosed either angina pectoris or myocardial infarction or both. Furthermore, the mean age of 58 years was above that usually associated with other types of cardiomyopathy, and manifestations of arteriosclerotic disease of the clinically readily accessible vessels could often be elicited.

The clinical data and past experience predicted a poor prognosis for all of these patients when treated by conventional methods. In view of this the patients were placed at prolonged bed rest in an effort to learn whether or not the prognosis could be improved. They represented a true cardiomyopathy with all of the inherent complications and tendency towards progression of the disease, but they were also subject to the other sequelae of extensive coronary artery disease and generalized arteriosclerosis in aging patients.

Previous studies have demonstrated the efficacy of bed rest in the management of postpartal cardiomyopathy, alcoholic cardiomyopathy, and idiopathic cardiomyopathy. The amount of tension that must be developed in the wall of the dilated heart is considerably greater than that required by a normal sized heart to achieve the same pressure. Time-course studies have shown that tension in the wall of the dilated heart continues to increase as systole progresses rather than to decrease as in a normal sized heart. If the added stress of hypoxia is imposed on a myocardium already under such an exaggerated mechanical load, further loss of function is to be expected. This will cause additional cardiac dilatation, establishing a cycle that progresses ultimately to death.

Resting the heart with prolonged complete bed rest can affect the cycle in several ways. With maximal reduction of the work load of the dilated heart, some degree of myocardial recovery can occur through healing mechanisms as yet almost entirely unknown and little studied. As cardiac size decreases, the mechanical disadvantage under which the heart worked also diminishes. The reduced work load plus any associated decrease in myocardial tension tends to reduce myocardial oxygen requirements and improve hypoxia. Furthermore, any increase in survival allows more time for the development of collateral circulation. The present data support this theoretic contention.

Of the nine patients who cooperated for bed rest, four (44 percent) experienced clinical improvement and a limited reduction in heart size. Following discharge two had reasonably long survivals of four and eight years, and two are presently living seven and eight months after discharge. The other five (56 percent) patients experienced varying degrees
of clinical improvement, but no reduction in heart size. Three died in hospital and two died shortly after discharge. Experience with this small group indicates that patients with ischemic cardiomyopathy achieve symptomatic benefit from prolonged bed rest, but enhanced survival is unlikely unless clinical improvement is accompanied by a decrease in cardiac size.

Postmortem studies revealed those changes usually encountered in cardiomyopathy, including cardiac dilatation, endocardial thickening, and myocar-dial degeneration and fibrosis. In addition, there was widespread coronary arteriosclerosis and localized regions of myocardial fibrosis consistent with old myocardial infarction. Three patients died suddenly, presumably from arrhythmia or acute myocardial infarction. Five patients died following courses of progressive worsening of congestive heart failure, one complicated by pneumo-nia and one with a pulmonary embolus. Ventricular arrhythmias and marked sensitivity to digitalis were commonly encountered.

These studies support the need to recognize ischemia as a cause of the classic clinical manifestations of cardiomyopathy. To accept the entity of ischemic cardiomyopathy is of assistance to the clinician in that he immediately realizes its extremely grave prognosis, resistance to therapy and the value of resting the heart even if it requires the patient to live the life of an invalid. If the diagnosis is not realized too late, patients with ischemic cardiomyopathy can live several years at bed rest. However, when the myocardial pathology is extensive, life is short even with bed rest. The myocardial cell is non-mitotic so that dead myocytes are never replaced and the surviving ones, even when in poor health, can only hypertrophy in an attempt to carry the load of work imposed upon the heart. Unfortunately, arteriosclerosis is a diffuse disease, so that even the living sarcomeres in general receive an inadequate supply of blood, certainly too little to meet the demands of an active person.

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Fellowship in Pulmonary Diseases

The Lung Station (Tufts) at Boston City Hospital will have an opening for one first-year Postdoctoral Fellow commencing January 1, 1972. American citizens only will be considered. Approved multiple-discipline training in pulmonary diseases with teaching and research opportunities. Further information and application forms may be obtained from Maurice S. Segal, M.D., Professor of Medicine (Tufts) and Director, Lung Station (Tufts) Boston City Hospital, 818 Harrison Avenue, Boston, Massachusetts 02118.

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