The Natural History of Rheumatic Heart Disease in the Third, Fourth, and Fifth Decades of Life

II. Prognosis with Special Reference to Morbidity

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This report is intended to contribute to an understanding of the natural morbidity of patients with chronic rheumatic heart disease in the third, fourth, and fifth decades of life. The data should prove useful in a consideration of the criteria for surgical intervention.

The recent emphasis on early surgical intervention for patients with chronic rheumatic valvular disease has made the need for basic data concerning the natural history of this condition of practical importance.

Data regarding the natural morbidity of patients with chronic rheumatic heart disease in the third, fourth, and fifth decades of life are presented. This information should prove useful in evaluating the effects of surgical intervention on the natural morbidity and mortality of the disease. 1–3

Methods and Materials

Of 757 adult patients with rheumatic heart disease analyzed in the previous study on survivorship, 385 patients were examined and followed by the authors personally during 1953 to 1955. All of these patients have been under medical supervision in the same cardiac clinic since childhood as part of a long-term follow-up study. They were therefore not comparable with a group of adults attending a cardiac clinic because of cardiopulmonary symptoms.

There were 160 males and 225 females, and this sex ratio held true for the subgroups studied. All patients were examined an average of 6 times in the 3-year period. Patients with cardiopulmonary symptoms were seen more frequently. On each visit they were questioned concerning their tolerance to physical stress in home, employment, leisure, and sport activities. Physical examination was amplified by fluoroscopic examination, electrocardiogram, and determination of vital capacity. Fluoroscopic examination included 3 standard positions and the use of barium.

In addition, the records of 78 patients who were under observation during the 40-year period and who died after the age of 20 were included for analysis of the morbidity experienced prior to death and the postmortem findings in 25 patients. The diagnosis of the type of valvular deformity was made according to the criteria of the New York Heart Association. 4 The anatomic diagnosis was established in the majority of patients by the twentieth year. The persistence of murmurs and their regression for 2 or more years were noted. There were 173 patients with mitral insufficiency alone, and 161 with mitral insufficiency and stenosis of whom 1 also had tricuspid insufficiency; 51 patients had combined mitral and aortic valvular disease including 11 with aortic stenosis and 1 with tricuspid insufficiency.

Cardiac enlargement was classified as minimal, moderate, or marked, fluoroscopically. Heart size was determined according to the shape of the cardiac silhouette in the posteroanterior projection, the angle of clearance of the left ventricle in the left anterior oblique projection, prominence of the pulmonary artery segment and atrial displacement of the barium-filled esophagus in the right anterior oblique projection (graded 1+, 2+, 3+), and enlargement of the right ventricle in the oblique projection.

Minimally enlarged hearts included those with a normal cardiac silhouette in the posteroanterior projection, an angle of clearance of the left ventricle of 55 degrees, and 1+ retrodisplacement of the esophagus in the right anterior oblique projection. Moderately enlarged hearts included those with minimal or moderate increase in the cardiac silhouette in the posteroanterior projection, an angle of clearance of 60 degrees or 60+ degrees, 2+ retrodisplacement of the esophagus in the right anterior oblique projection, and slight right ventricular enlargement. Markedly enlarged hearts included all those in which the dimensions exceeded the above in all projections.

Twelve-lead electrocardiograms were recorded with standard, unipolar extremity, and precordial leads.
Vital capacities were measured with a water displacement, cylindrical spirometer. Each patient's prior maximum reading served as the basis for comparison with subsequent recordings. Acute respiratory infections excluded, the normal range referred to 90 per cent or more of the previous maximum. Moderate reduction is defined as 75 to 89 per cent of maximum. Less than 75 per cent is defined as marked reduction.

For purposes of analysis all the patients were grouped arbitrarily on the basis of their cardiopulmonary symptoms. This modified functional cardiac classification corresponds with that most commonly used in the selection of such patients for valvular surgery. Functional class I, including 340 patients, consists of those patients with no or minimal symptoms of cardiopulmonary disability. Class II, including 20 patients, contains those patients who had symptoms and disability of but moderate degree that were not progressive in nature. Class III, including 13 patients, contains those who were clearly and progressively ill with advancing symptoms of cardiopulmonary disability. Class IV, including 12 patients, contains those with extreme disability.

Observations

Mitral Insufficiency

There were 173 patients with mitral insufficiency, of whom 85 were in the third decade of life, 78 in the fourth, and 10 in the fifth (table 1). In over 70 per cent there was regression of a previously long-standing apical systolic murmur. Patients with persistent murmurs had a higher incidence of moderately enlarged hearts. Sixty per cent of the patients had minimal cardiac enlargement, and the remainder had moderate enlargement. None had very large atrial chambers or detectable right ventricular hypertrophy. Progressive cardiac chamber enlargement was not noted with advancing age. Active carditis was not observed in any patient after age 20. These patients were asymptomatic or had minimal complaints of dubious significance. They were apparently unimpaired functionally and leading normal, active lives. Pregnancies were well tolerated as were other intercurrent stresses and illnesses. Ten per cent of the patients had significantly reduced vital capacities not correlating with the persistence or regression of murmurs, or the degree of cardiac enlargement; these few patients presumably were not aware that their reactions to exertion were not normal. All 173 patients, included in functional class I, would not be considered to warrant surgical intervention (table 2, fig. 1).

Mitral Stenosis and Insufficiency

There were 161 patients with mitral stenosis and insufficiency; 52 were in the third decade of life, 79 in the fourth, and 30 in the fifth (table 1). One hundred thirty-four patients were included in functional classification I, 13 in class II, 10 in class III, and 4 patients in class IV (table 2, fig. 1).

Functional Classification I. One hundred thirty-four patients had no or minimal cardiopulmonary symptoms; 47 were in the third decade of life, 67 in the fourth, and 20 in the fifth. In 36 patients the diastolic and systolic murmur persisted, in 44 both murmurs regressed, and in 54 only the diastolic murmur regressed. There were no instances of so-called “pure” mitral stenosis in this group. It will be recalled that the valvular diagnosis was not changed when murmurs regressed. After the age of 20 years, 4 patients experienced active carditis, including 1 patient with 3 episodes. Cardiac enlargement was minimal in 39 per cent, moderate in 55 per cent, and marked in 6 per cent. There were only 8 patients in whom right ventricular enlargement was detected fluoroscopically. No significant progression in cardiac chamber enlargement was observed with advancing age. Multiple electrocardiograms of 117 patients were available. Normal records were noted in 76 patients (65 per cent), broad notched P waves in 24 per cent, evidence of left ventricular hypertrophy in 9 per cent, and right ventricular hypertrophy in only 2 patients.
TABLE 2.—Distribution According to Cardiopulmonary Symptoms

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<th>Decade</th>
<th>MI heart enlargement</th>
<th>MI, MS heart enlargement</th>
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* Eleven of these patients also had aortic stenosis.
† These patients also had tricuspid insufficiency.

Vital capacity was within normal limits in 75 per cent of this group; 19 per cent had moderate, and 6 per cent marked reduction. Nearly all of the patients in the third decade, but only about half those in the fifth decade, had normal vital capacities. Marked reduction was limited to the fourth and fifth decades. Vital capacity did not correlate with heart size or with the persistence or regression of the murmurs, and it is likely that the reduction in vital capacity in the older asymptomatic patient may be related, at least in part, to loss of pulmonary elasticity secondary to aging. As in the mitral insufficiency group, they apparently were unaware of any abnormal reaction to effort. Fifty-five of the 88 women in this group had been pregnant. The total number of pregnancies was 130. Among these asymptomatic patients, pregnancy did not appear to influence adversely the cardiopulmonary status. This group, at the present time, would probably not be considered candidates for surgical consideration.

Functional Classification II. Thirteen patients are included in functional classification II because of persistent but nonprogressive cardiopulmonary symptoms of mild to moderate degree. The murmurs were persistent in all. There were 4 patients in the third decade of life, 5 in the fourth, and 4 in the fifth. All but 1 of the patients had moderately or markedly enlarged hearts that did not appear to increase with advancing age. Six had fluoroscopic evidence of right ventricular enlargement; all had left atrial and left ventricular enlargement. Electrocardiograms were normal in 5 patients. Changes indicative of right ventricular hypertrophy were noted in 3 patients, of left in 4, and 8 patients had abnormal atrial complexes.

Vital capacity was within normal limits in 7 patients although these had subjective complaints. It was moderately reduced in 5, and markedly in 1. After the age of 20, 1 patient experienced an attack of active carditis, and 4 developed atrial fibrillation. Of the 11 women in this group, 9 had a history of 20 pregnancies. All but 3 patients tolerated their pregnancies
well. One woman developed congestive heart failure, and another developed chronic atrial fibrillation during the first pregnancy but withstood her second pregnancy well. In a third patient a mitral diastolic murmur that had temporarily regressed, reappeared with her second pregnancy and persisted. These 13 patients, according to most observers, would be considered probable candidates for surgery.

Functional Classification III. Ten patients had progressive cardiopulmonary symptoms. The majority were in the fourth and fifth decades of life. All had persistent murmurs. All but 1 had marked cardiac enlargement. Nearly every patient had electrocardiographic evidence of left ventricular and right ventricular hypertrophy, and atrial involvement. After the age of 20, 1 patient experienced active carditis. Five had atrial fibrillation. All patients had reduction in vital capacity, most of them marked. Four women experienced 10 pregnancies. One of these developed a concomitant subacute bacterial endocarditis and another 2 patients developed persistent hypertensive cardiovascular disease after pregnancy with a subsequent increase in cardiopulmonary symptoms. Several of these patients would be considered candidates by many surgeons.

Functional Classification IV. Four patients had extreme cardiopulmonary symptoms. There was 1 patient in the third decade of life, 2 in the fourth, and 1 in the fifth. Three patients had marked cardiac enlargement, marked reduction in vital capacity and intractable congestive heart failure; 1 of these had associated tricuspid insufficiency. The remaining patient had idiopathic pulmonary fibrosis and insufficiency. None had had active carditis after age 20; 2 patients developed atrial fibrillation. One patient had been pregnant 3 times. In all instances, electrocardiograms indicated ventricular hypertrophy and abnormal atrial com-

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**Fig. 1.** Comparison of functional classification and extent of cardiac enlargement for 212 patients with mitral insufficiency and mitral stenosis and combined aortic lesions.
plexes. At the present time most of these patients would not be considered suitable for surgical intervention.

**Combined Mitral Stenosis and Insufficiency, and Aortic Valvular Disease**

Fifty-one patients with mitral stenosis and insufficiency had associated aortic valvular lesions. All had aortic insufficiency; 11 had aortic stenosis. One had tricuspid insufficiency. Twenty patients were in the third decade of life, 19 in the fourth, and 12 in the fifth (table 1). There were 33 patients in functional classification I, 7 in class II, 3 in class III, and 8 patients in class IV (table 2, fig. 1).

**Functional Classification I.** Thirty-three patients were asymptomatic; 17 in the third decade of life, 11 in the fourth, and 5 in the fifth. There were 4 instances of aortic stenosis. In 25 patients the murmurs were persistent. Among the other 8 patients, the murmur of mitral insufficiency subsequently regressed in 4, that of mitral stenosis in 5, and that of aortic insufficiency in 6 patients. In 6 patients the murmurs of mitral stenosis and aortic insufficiency both regressed, but not simultaneously. Only 14 patients had a characteristic widened pulse pressure. It is possible that in some patients the basal diastolic murmur may have represented pulmonary incompetency. Twenty patients had moderate and 13 had marked cardiac enlargement. All of the patients had fluoroscopic evidence of left atrial and left ventricular enlargement, and none had demonstrable right ventricular enlargement. In 32 patients multiple electrocardiograms were available. Seventeen patients had normal tracings. All the remaining 15 had the abnormal P waves usually associated with mitral stenosis, and 11 of them had evidence of left ventricular hypertrophy; none had right ventricular hypertrophy.

Three patients had had an acute attack of active carditis after the age of 20. Five of the 12 women in this group had been pregnant a total of 8 times without untoward effect. Only one third of the patients had more than minimal reduction of vital capacity, and only 1 had marked reduction. Few of these 33 patients, according to commonly accepted criteria, would be considered candidates for surgery at the present time.

**Functional Classification II.** There were 7 patients who had persistent but not progressive cardiopulmonary symptoms of mild or moderate degree. All but 1 of them were in the fourth and fifth decades of life. One patient had aortic stenosis. In 2 patients the murmur of aortic insufficiency had regressed. Three patients had wide pulse pressures. Two had moderate and 5 had marked cardiac enlargement fluoroscopically, with left atrial and left ventricular enlargement in all, and detectable right ventricular enlargement in 5. Electrocardiograms were available for each patient; only 1 tracing was a normal record. Three patients had abnormal atrial complexes and 4 had left ventricular hypertrophy. None showed evidence of right ventricular hypertrophy. Three episodes of active carditis were experienced by each of 2 patients after the age of 20. Four of the 5 women had been pregnant a total of 5 times without exaggeration of disability. Two patients had minimal, 4 moderate, and 1 marked reduction of vital capacity. Some of these patients might be considered suitable for cardiac surgery at the present time.

**Functional Classification III.** Three patients, all in the fourth decade, had progressive cardiopulmonary symptoms. One had aortic stenosis. The murmur of aortic insufficiency had regressed in 1 patient. None had a wide pulse pressure. All had marked cardiac enlargement fluoroscopically, including the left atrium and both ventricles. Electrocardiograms disclosed abnormal atrial complexes in each patient, with evidence of left ventricular hypertrophy in 2 and of right ventricular hypertrophy in none. One patient had had active carditis after age 20. All 3 patients had a moderately reduced vital capacity. All had been in congestive heart failure on one or more occasions, and 2 had chronic atrial fibrillation. These 3 patients might be considered likely candidates for cardiac surgery by some surgeons.

**Functional Classification IV.** Eight patients with mitral and aortic valvular disease had extreme symptoms of cardiopulmonary disability; 2 were in the third decade of life, and 3 in each of the fourth and fifth decades. Two of the older
patients had advanced portal cirrhosis and coronary arteriosclerosis respectively, with moderate cardiac enlargement. The remaining 6 patients had severe aortic valvular disease and marked over-all cardiac enlargement. Five of them had wide pulse pressures. Five had the murmur and thrill of aortic stenosis, and one of them had tricuspid insufficiency as well. Three of them were in chronic congestive heart failure, and 1 had atrial fibrillation.

Only the patient with portal cirrhosis had a normal electrocardiogram. The others had records showing left ventricular hypertrophy and abnormal P waves but not right ventricular hypertrophy. There were 2 instances of carditis after age 20. One woman experienced 2 pregnancies without complications. Nearly every patient had markedly reduced vital capacities. None of these 8 patients would be considered suitable for surgical intervention.

**Additional Observations**

Of the total 385 patients, only 45 had cardiopulmonary symptoms. There were 28 patients with mitral stenosis and insufficiency alone, and 17 with associated aortic disease. Possible factors that may have been responsible for the morbidity after age 20 were considered. All but 1 of these patients were in functional classification I for 2 to 24 years, averaging 10.5 years, and progressed variously to functional classification II, III, and IV. There were 71 instances of such a change among the 45 patients with single or multiple shifts in symptomatic status. Forty-one patients progressed from an asymptomatic state to functional classification II. Eighteen developed progressive symptoms and entered class III. Twelve eventually developed extreme cardiorespiratory symptoms and were included in functional classification IV.

In 60 instances of a change in symptomatic status, factors were identified that might have been causally related to the development of subjective complaints of dyspnea associated with the objective findings of decreased vital capacity, increased heart size, or congestive heart failure. Among these 60 instances of changed morbidity, decreasing vital capacity, further cardiac chamber enlargement, and congestive heart failure either as an episode or continuous and intractable, characterized the course. In 8 instances active carditis was responsible; in an additional 2 instances, active carditis may have occurred when these patients were not under our medical supervision. In 14 instances the onset of atrial fibrillation attended the changed symptomatic status. In 6 instances there was progressive deterioration from an asymptomatic state to functional classification IV, due to cirrhosis in one, chronic pulmonary disease in a second, and the development of hypertensive or arteriosclerotic heart disease in 4. In 22, other factors were identified with a change in functional classification. These included pneumonia in 15, healed subacute bacterial endocarditis in 3, and thromboembolism in 4. Whether there was an associated subacute carditis in some of these patients could not be ascertained. In 8 instances pregnancy was associated with a change in morbidity.

There were 11 instances of change in clinical status in which no causal factor could be identified to explain the change in functional classification. In 3, it represented a change from class I to class II; in 4, a change from class II to III, and in 4 a change from class II or III to class IV. All of these patients had marked cardiac enlargement with mitral stenosis and insufficiency and aortic stenosis and insufficiency that were present since age 20. It is possible that changes in symptomatic status were related to the progressive influence upon the circulation of chronic valvular disease. However, the extent of myocardial damage and the presence of unrecognized carditis must be considered.

Further information pertinent to the study of the natural morbidity of rheumatic heart disease was available from the records of 45 of 53 patients who died of cardiac causes after surviving to the age of 20 years or more.

There were 14 patients with mitral stenosis whose records were suitable for analysis; 7 were in the third decade of life, 4 in the fourth, and 3 in the fifth decade. Of those in the third decade, all were asymptomatic until within 1 year or less of death. Five had moderate cardiac enlargement. In 5, death was sudden, and in 2 of these pregnancy was a complicating feature. Two patients with marked cardiac enlargement became symptomatic with the onset of atrial
fibrillation. Of the 4 patients in the fourth decade of life, all had cardiopulmonary symptoms for a period of 2 to 7 years. All but 1 had marked cardiac enlargement. Two had had mitral valve surgery, one of whom survived 3 years but had no change in functional status, and the other remained in intractable congestive heart failure for the year following surgery. Of the 3 patients in the fifth decade of life, all had marked cardiac enlargement and atrial fibrillation with cardiopulmonary symptoms for 2 to 6 years before death. Two patients had had valvulotomy; one survived for 2 years without improvement, and the other succumbed after 4 years with pulmonary embolism.

There were 31 patients with aortic and mitral valve disease. Nine of these had demonstrable carditis as the terminal event; 4 of these were asymptomatic prior to their terminal event for a period of 2 to 9 years, 3 had atrial fibrillation, and 2 had severe intractable congestive heart failure for 3 and 5 years respectively before death. Of the remaining 22 patients who died, 9 were in the third decade of life, 12 in the fourth, and 1 in the fifth decade. In the third decade, all but 1 patient had marked cardiac enlargement, and 5 had atrial fibrillation. One patient sustained sudden death from pulmonary embolism, and 2 had a preceding subacute bacterial endocarditis. They had been asymptomatic for 1 to 9 years. In the fourth decade of life, all but 1 of the 12 patients had marked cardiac enlargement, and 10 had atrial fibrillation. The majority were asymptomatic for a period of 6 to 15 years. Five patients had a sudden death, presumably embolic in nature. The 1 patient in the fifth decade of life, with marked cardiac enlargement and atrial fibrillation, who had been asymptomatic for 19 years, had nonprogressive symptoms for an additional 4 years and then experienced pulmonary edema within 6 months of her terminal episode, which was associated with bronchopneumonia.

It is of interest that of the 45 patients whose death was attributed to cardiac causes, carditis, fibrillation, pulmonary embolism, and pneumonia were identified as common factors that appeared to induce the terminal event.

Autopsies were performed in 25 of the 78 deaths. Six autopsied patients had carditis clinically; it was confirmed in all. No other instances of carditis were found at autopsy. In 22, death was directly attributable to rheumatic heart disease or its complications. In all instances of mitral stenosis the clinical diagnosis was confirmed pathologically and no false positive diagnoses were made clinically. A failure in clinical diagnoses was noted in 2 instances of aortic insufficiency, 2 of aortic stenosis, and 4 of tricuspid stenosis. Two patients had mitral insufficiency alone, both of whom died of noncardiac causes. There were 9 patients with mitral stenosis and insufficiency alone, and 15 with mitral and aortic disease; 5 of the latter group had tricuspid insufficiency.

These autopsies were performed between the years 1937 to 1954, and only 19 were performed at the New York Hospital. In 22, data were available with reference to heart weight, and in 21 direct measurements were made of the thicknesses of both ventricles. Figure 2 shows a good correlation between the fluoroscopic estimate of over-all heart size and the per cent in excess of actual, over expected, heart weight when corrected for body weight and sex. The heart weights ranged between 270 and 1,010 Gm. Among 21 patients with left ventricular enlargement fluoroscopically, hypertrophy was noted in 16 at autopsy. In 11 patients enlargement of the right ventricle was demonstrated fluoroscopically and in 6 of these hypertrophy was reported pathologically.

Ten patients had electrocardiograms demon-
stratifying atrial fibrillation, all of whom had fluoroscopic evidence and 7 of whom had autopsy evidence of left atrial enlargement. Inasmuch as the majority of these patients died prior to the use of unipolar leads, no statement can be made as to the relationship between electrocardiographic and postmortem evidence of specific chamber hypertrophy.

Discussion

It is significant that 89 per cent of the 385 patients with rheumatic heart disease, ranging in age from 20 to 49 years, who were under our medical supervision were asymptomatic. It is to be recalled that the anatomic diagnosis was established in the majority by age 20, and that these patients were under observation because of a long-term follow-up. Of interest is the observation that one half of these patients had simple mitral insufficiency without marked cardiac enlargement. None of these asymptomatic patients would, as of the present time, be considered candidates for cardiac surgery.

Of particular importance is the finding that only 45 patients with mitral stenosis and insufficiency alone or associated with aortic valvular disease had cardiopulmonary symptoms. This small percentage of the total would thus constitute a group comparable to the type of patients presenting themselves for surgical consideration. The 173 with mitral insufficiency were asymptomatic. Of the total 161 patients with mitral stenosis and insufficiency, 27 had symptoms. Eighteen of the 51 with mitral stenosis and insufficiency and aortic valvular disease were symptomatic. It is notable that the majority of these patients had marked cardiac enlargement, constituting two thirds of those with mitral stenosis alone, and three fourths of those with associated aortic disease. The greater proportion of these patients were in the fourth and fifth decades of life.

It should be emphasized that progressive cardiac enlargement was not observed with advancing age alone in either the symptomatic or asymptomatic group. There is thus no evidence from these data that valvular deformity per se was a major factor contributing to degree of cardiac enlargement. Morbidity, like mortality, would appear to be more closely related to degree of cardiac enlargement than to type of valvular lesions.

The observation that some patients in the asymptomatic group revealed a diminished vital capacity is worthy of comment. We suggest that these patients may have been unaware of abnormal symptoms. In some, decreased vital capacity may have been related to reduced pulmonary elasticity. It is of interest that the patients in functional class II who were symptomatic had vital capacities within their normal limits. It is likely that the subjective complaints may not have been organic in origin.

In the analysis of changes in morbidity, it was apparent that a common factor was the occurrence of demonstrable carditis in both the living and in those who died. Also of importance was the occurrence of atrial fibrillation, subacute bacterial endocarditis, and thromboembolism. In others, with associated illnesses such as pneumonia and the occurrence of atrial fibrillation, the presence of subacute carditis could not be excluded.

The small number of patients in this study who were symptomatic renders difficult any over-all statement as to indications for surgery. However, these patients are probably representative of those for whom surgical intervention is considered. An inspection of our data reveals that the outstanding factors inducing progressive disability are not amenable to surgical correction; specifically, carditis, atrial fibrillation, thromboembolism, and marked cardiac enlargement. The removal of thrombi from a dilated atrium would probably reduce morbidity and mortality consequent upon thromboembolism. It is possible in some cases that surgical reduction of interatrial pressure might decrease the incidence of fibrillation. Since the majority of symptomatic patients were in the fourth and fifth decades of life, one could speculate that asymptomatic patients may be considered for surgical prophylactic therapy early in the third decade.

Summary

The natural morbidity experienced by 385 children with rheumatic heart disease, reaching the ages of 29 to 49 years, is presented. One
hundred fifty-seven patients were in the third decade of life, 176 in the fourth, and 52 in the fifth decade. The anatomic diagnosis was established in the majority by the age of 20 years: mitral insufficiency in 173 patients, mitral stenosis and insufficiency in 161, and 51 patients had aortic and mitral valvular lesions. All of the 173 patients with mitral insufficiency were asymptomatic. Cardiac enlargement was minimal or moderate. Of 161 patients with mitral stenosis and insufficiency, 27 (17 per cent) had cardiopulmonary symptoms. About two thirds of these had marked cardiac enlargement. Of 51 patients with combined aortic and mitral valvular lesions, 18 or about one third had cardiopulmonary symptoms; 14 of these had marked cardiac enlargement. Three hundred forty (89 per cent) patients were asymptomatic, and 45 (11 per cent) experienced cardiopulmonary symptoms.

The factors found responsible for morbidity and mortality after the age of 20 were active carditis, atrial fibrillation, bacterial endocarditis, pregnancy, pneumonia, and embolic phenomena.

Patients who experienced cardiopulmonary symptoms had been asymptomatic for periods of 2 to 24 years after age 20, with an average of 10.5 years. Patients who died of cardiac causes had been asymptomatic for 1 to 20 years before the terminal event.

Cardiac chamber enlargement did not appear to progress with advancing age per se, irrespective of the type of valvular deformity. Confirmation of fluoroscopic estimate of over-all heart and chamber enlargement was obtained on postmortem examination.

The majority of patients with rheumatic heart disease who survived to the age of 20 to 49 years were in functional classification I. The majority of patients with mitral stenosis and insufficiency alone or with associated aortic disease who had markedly enlarged hearts were in functional classification II, III, or IV.

The residual cardiac damage sustained in the first 2 decades of life, particularly the extent of cardiac enlargement, appears to be the major factor influencing morbidity and mortality in the third, fourth, and fifth decades.

**Summario in Interlingua**

Es presentate datos in re le morbiditate natural experienciate per 385 patientes pediastric con rheumatic morbo cardiac qui attingeva un etate de inter 29 e 49 annos. Cento cinquanta-septe del patientes eseva in le tertie decemnio de lor vitas al tempore del studio; 176 eseva in le quarte; e 52 eseva in le quinte. Le diagnose anatomic eseva establiste in le majoritate del casos ante le etate de 20 annos: Insufficentia mitral in 173 patientes, stenosis e insufficientia mitral in 161, e lesiones del valvulas aortie e mitral in 51. Omne le 173 patientes con insufficientia mitral eseva asymptomatic. Le allargamento cardiac eseva minimal o moderate. Ex le 161 patientes con stenosis e insufficientia mitral, 27 habeva symptomas cardiopulmonar (17 pro cento). Cira duo tertios de istes habeva marcate grados de allargamento cardiac. Ex le 51 patientes con combine lesions del valvulas aortie e mitral, 18 o circa un terto habeva symptomas cardiopulmonar. Decé-quatro de istes habeva marcate grados de allargamento cardiac. Tres centos quaranta patientes (89 pro cento del serie total) eseva asymptomatic, e 45 (11 pro cento) experiencia symptomas cardiopulmonar.

Le factores recognoscite como responsabile pro morbiditate e mortalitate post le etate de 20 annos eseva carditis active, fibrillation atrial, endocarditis bacterial, pregnancy, pneumonia, e phenomenos embolic.

Patientes qui experiencia symptomas cardiopulmonar habeva essite asymptomatic durante periodos de inter 2 e 24 annos post passar le etate de 20 annos. Le duration medie de iste periodo asymptomatic eseva 10,5 annos. Patientes qui moriva ab causas cardiac habeva essite asymptomatic durante inter 1 e 20 annos ante le evento terminal.

Allargamento de camera cardiac non pareva progreder con le progresso del etate per se, sin reguardo al typo de deformitate valvular. Le estimation fluoroscopic del allargamento del corde in general e del cameras individual eseva confirmate per examines necroptic.

Le majoritate del patientes con rheumatic morbo cardiac attingente etates de inter 20 e
49 annos esserà funzionalmente in le classificazioni I. Le majoritate dei pazienti con stenosi e insufficienza mitral sin o con asso- ciate morbo aortic sed con marcate gradi de allargamento cardiac esserà funzionalmente in le classificazioni II, III, o IV.

Le residue insulto cardiac esperienciate durante le 2 prime decennios del vita—specialmente le grado del allargamento cardiac—es apparentemente le major factor de influentia super le morbidity e mortalitate durante le terti, quarte, e quinte decennios.

REFERENCES


6 Smith, H. L.: The relation of the weight of the heart to the weight of the body and of the weight of the heart to age. Am. Heart J. 4: 79, 1928.

FORTHCOMING ARTICLES


Katz, L. N.: Rehabilitation of the Cardiac Patient.

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