Cortisone Therapy in Acute Rheumatic Carditis: Preliminary Observations

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Four patients with acute rheumatic carditis of moderate to severe degree were treated with cortisone. After a latent period of 5 to 15 days, there was fairly rapid subsidence in temperature, erythrocyte sedimentation rate and some other signs of inflammation. After cessation of the course of cortisone therapy, there was evidence of recrudescence of rheumatic activity in all four patients. In three of these the therapy was resumed, with quite unusually rapid decline in the fever and sedimentation rate. The cardiologic signs showed no improvement throughout the entire period of observation.

Following the dramatic results reported for cortisone therapy in rheumatoid arthritis by Hench, Kendall and co-workers,1 these authors applied this hormone to the treatment of 3 patients with acute rheumatic fever and reported a rapid subsidence of fever, tachycardia, polyarthritis, elevated sedimentation rates, and abnormal electrocardiographic changes.2 A later report by the group, which included studies on 8 additional patients (4 given cortisone and 4 given adrenocorticotropic hormone, confirmed the earlier findings.3 In addition, however, it was noticed that 2 of the patients treated with cortisone suffered recurrences of rheumatic activity when the administration of cortisone was discontinued. Resumption of the use of cortisone abolished the evidence of activity in both cases. Massell4 has reported favorable results in the treatment of a series of 14 cases of acute rheumatic fever with ACTH.

The obvious importance of these findings led us to institute the experimental treatment with cortisone of patients with acute rheumatic carditis who were under close clinical observation. It was decided to choose for the first phase of the study a group of patients whose degree of cardiac involvement was more severe than seemed to be the case in the patients studied by Hench and his associates. Although thus far only 4 patients have been included in this study, certain observations which were made seemed to warrant the reporting of these preliminary data.

Methods and Materials

The 4 patients included in this study all had definite carditis of moderate or severe degree. They were hospitalized in the two institutions from which this study is reported. They ranged in age from 4 to 22 years.

The patients were examined physically at least every two days by two or three of the authors. Laboratory examinations included the erythrocyte sedimentation rate (ESR), white blood cell count, and hemoglobin concentration. Electrocardiograms were taken as required. The sedimentation rate was determined by a method described elsewhere.5 It involved a series of readings of the erythrocyte level at five minute intervals, in order to determine the rate of free fall of the corpuscles, and a correction for the relative volume of erythrocytes.

Blood counts of eosinophilic polymorphonuclear leukocytes were done by the Thorn technic.6 Antistreptolysin and streptococcal antihyaluronidase titrations of serum were done as described elsewhere.7, 8

The dosage of cortisone employed was 112.5 mg. for the 3 older patients, whose weights were, in every case, approximately 90 pounds, and 75 mg. for the 4 year old patient. These doses were somewhat higher than those used by Hench. They were maintained at a level
which caused substantial decrease in the blood eosinophil count, but did not give rise to glycosuria or to concentrations of blood sugar over 120 mg. per 100 cc. in the fasting state.

**Results**

1. Effect on Fever. In 3 of the patients the temperatures promptly fell following the first administration of cortisone, reaching normal in 4 to 7 days. In the fourth, J. K., the temperature fell gradually, reaching a plateau in 17 days.

When cortisone was readministered to 3 patients the temperatures, which had risen on cessation of cortisone therapy, fell even more sharply, reaching normal in 1, 3, and 6 days respectively.

2. Effect on Polyarthritis. When cortisone was first administered to the 2 patients complaining of joint pains, both were free of pain within 4 days.

Three patients complained of joint pains on cessation of cortisone therapy. These patients were free of pain again within 4 days after resumption of the hormone.

3. Effect on Sedimentation Rate. In three cases the sedimentation rates fell to within the limits of normal in 9, 16 and 21 days, respectively, after the beginning of cortisone therapy. The sedimentation rate of the fourth patient (F. M. W.) reached a plateau after 28 days of such treatment.

When cortisone was instituted to combat exacerbations, the sedimentation rates fell to within normal limits in 7, 10 and 17 days.

4. Effect on the Clinical Cardiac Status. In 3 patients heart failure developed 4, 10 and 20 days after the institution of cortisone, as indicated by hepatic enlargement, pleural effusion, ascites and edema. This condition responded well to the usual therapeutic measures while the hormone therapy was continued. All murmurs present at the beginning of the period of observation were present at the end. In addition, 3 of the patients acquired a new murmur while under treatment. M. B. developed a blowing, early diastolic apical murmur in addition to the apical systolic blow she first presented. F. M. W. developed a decrescendo early diastolic murmur and a crescendo presystolic murmur at the apex during her first course on cortisone and these have persisted. Furthermore, the decrescendo diastolic murmur heard only at the second left intercostal space when she was first observed could be heard transmitted past the cardiac apex during the latter part of this period. In addition to the aortic and double apical murmurs initially present in J. K., an aortic systolic murmur appeared.

Roentgenographic studies in 3 patients over the period of this study have shown no change in cardiac size or contour of the heart. All were initially enlarged: slightly in B. D.; markedly in M. B.; and extremely in J. K. The early cardiac x-ray films of F. M. W. were normal. Later, however, definite left ventricular enlargement with some fullness of the pulmonary artery segment was demonstrable.

5. Effect on the Electrocardiograms. (a) M. B.: The electrocardiogram taken two days before cortisone was instituted (fig. 1, day 2) showed right axis deviation; slight elevation of the QRS-T segments in leads II, III, and aVF, and
inversion of the T waves in leads II, III, aVf, and all precordial leads. Eight days later, six days after cortisone was started, the T waves were upright in all leads except III and CR2. The QRS-T elevations were very slight (fig. 1, day 9). The T waves continued to improve and the tracing was within normal limits during the second course of cortisone. Twenty days later, six days after cortisone was finally discontinued, some minor changes in the T waves of the precordial leads were noted. Ten days later all T waves had decreased in amplitude and were again inverted in CR3-4 (fig. 1, day 74). A week later the T waves in the chest leads showed a definite tendency to become more inverted.

(b) B. D.: The initial electrocardiogram showed only notched P waves and a sinus tachycardia. Ten days later, after nine days of cortisone treatment, increased amplitudes of the QRS complexes and the T waves were noted. An electrocardiogram taken 22 days after cortisone was discontinued showed top-normal Q-T intervals, left axis deviation, low amplitudes of the QRS complexes, and persistently notched P waves.

(c) F. M. W.: The electrocardiographic tracings taken before and during her first course of cortisone were normal (fig. 2, day 28). A tracing taken six days before cortisone was stopped for the second time was normal, but the voltage of the T waves was lower.

Ten days after cortisone treatment was
stopped her electrocardiogram showed a prolonged Q-T interval and flattened or diphasic T waves in nearly all leads (fig. 2, day 77). Five and ten days later, repeated tracings were the same.

(d) J. K.: The initial electrocardiogram showed a markedly prolonged P-R interval (fig. 3, day 6). Daily tracings showed a rapid shortening of the P-R interval until it became normal three days after cortisone was first given. At the same time, however, the QRS-T segments became progressively more elevated as pericarditis developed (fig. 3, day 9). They reached a peak and began to descend four days after the drug was started, reaching the base line 16 days later. Auricular fibrillation was recorded on tracings taken 8, 20 and 21 days after the institution of therapy. On the tenth day of treatment the T waves began to be inverted in leads I, aVL, and CR4,6, and this trend has progressed and persisted. On the twenty-first day the T waves of CR3,4 also became inverted (fig. 3, day 26).

An electrocardiogram taken at the height of the exacerbation that followed the first cessation of cortisone therapy showed the reappearance of prolonged P-R intervals and notched P waves. The T waves were better but still deeply inverted in the left precordial leads (fig. 3, day 43).

A tracing taken three weeks later showed a shorter P-R interval and some improvement in the T waves. An electrocardiogram made one month later showed a normal P-R interval but the T waves were still flat. The patient was receiving digitalis as Jan. 8, 1950.

6. Serologic Observations. Sera were prepared from each blood specimen drawn from these patients and examined for their content of antistreptolysin and streptococcal antihyaluronidase. The titers of these antibodies decreased during the period of observation, but whether the rate of this decrease is significantly greater in patients with rheumatic fever under cortisone treatment than in control patients can be decided only after a considerably larger number of patients has been studied.

Summary of Clinical Data

The sequence of events during these courses of treatment, then, followed this general pattern: For the first 5 to 15 days after the beginning of treatment with cortisone there was either no objective improvement or there was actual clinical evidence indicative of exacerbation or spread of the inflammatory process. Thereafter, there was fairly rapid subsidence of the signs of inflammation, per se, so that the temperature, pulse rate and erythrocyte sedimentation rates were within the normal range or, in one case, at a slightly elevated plateau, within three weeks of treatment, without a corresponding improvement in cardiac findings, other than some electrocardiographic changes. At this time, when the general signs of inflammation had subsided, and the cardiac signs had ceased to show change, cortisone was withdrawn. Within two to nine days after this there was in each case an exacerbation, with the same clinical picture as had characterized the beginning of that episode in that patient. In 3 of the 4 patients cortisone therapy was re-instituted, and within three to seven days all of these 3 showed a rapid improvement in the general signs of inflammation. The cardiac signs, however, showed little or no change during the few days following withdrawal or re-institution of cortisone therapy. Following the rapid improvement in general signs at the beginning of the second course of treatment, it was possible to reduce the dosage of the drug until the drug was withdrawn completely within two weeks. In 2 patients elevation of the temperature and sedimentation rate followed this withdrawal. These were probably due to terminal activity of the rheumatic inflammation, and were followed by unfavorable electrocardiographic changes within 6 to 10 days.

Discussion

These data are not presented as a basis for clinical evaluation of cortisone in the treatment of rheumatic carditis. Such evaluation would require the study of a far greater number of patients over a longer period of time, and the inclusion of such special groups as those patients with acute rheumatic carditis in whom the diagnosis can be made and treatment instituted within the first few days of the episode. However, these limited observations are being presented because they may suggest certain
generalizations or problems regarding the mechanisms involved.

First, the sequence of clinical events on cessation and resumption of cortisone therapy would seem to indicate strongly that one or more of the physiologic functions or pharmacodynamic effects of the hormone must be closely related to some mechanism involved in the pathogenesis of rheumatic inflammation. Thus it is probable, regardless of the ultimate role of cortisone therapy in rheumatic carditis, that the results of laboratory investigations of cortisone may have significant applications to the problem of the mechanisms involved in rheumatic infection.

Second, it is of interest to compare the effects of cortisone in these patients on the signs of infection of the body as a whole and on the evidences of inflammation of cardiac tissue. Although the response of these patients to cortisone was quite satisfactory in terms of the period of elevation of temperature, pulse rate and erythrocyte sedimentation rate during the first course of treatment, and quite dramatic during the second course, the cardiac signs did not correspondingly show changes which could be said to compare favorably with those seen in patients with acute rheumatic carditis treated symptomatically, since 3 of the 4 patients had auscultatory signs of more extensive cardiac involvement at the conclusion of the total course of cortisone therapy than at its inception, and only 1 patient had cardiac signs which were definitely as good at the end of the treatment. The fact that in this particular sampling of patients some showed changes of this sort, in spite of the generally favorable course of all 4 patients, suggests the possibility of a curious dichotomy between the effects of cortisone on the cardiac changes and those on the signs of inflammation of the body as a whole. These data also suggest the possibility of a difference between the clinical results in rheumatic carditis and those which the drug has been shown to have in rheumatoid arthritis. In the cases of the latter condition treated with cortisone, as described by Hench and his co-workers and by others, functional and even histologic improvement of the inflamed organ, the joints, accompanied the return of the sedimentation rate to normal limits and the general clinical improvement. In the 4 patients with acute rheumatic carditis treated with cortisone in the present study, not only did the chief seat of inflammation, the heart, fail to improve functionally as the general signs of inflammation subsided, but, as is mentioned above, in 3 out of the 4, there were clinical evidences of extension of the pathologic process. Thus, it would appear that no matter what the final evaluation of cortisone as a therapeutic agent in rheumatic carditis, there may be a difference between the effects of this drug in rheumatoid arthritis and those in rheumatic fever.

**SUMMARY**

Four patients with acute rheumatic carditis of fairly severe degree were treated with cortisone. After three weeks, when the temperature, erythrocyte sedimentation rate and pulse rate had returned to approximately normal limits and cardiac signs had ceased to show change, the cortisone therapy was terminated. Within a few days thereafter each of the patients showed a sharp exacerbation of signs of rheumatic infection, and on resumption of cortisone therapy these signs disappeared quite rapidly. The general signs of inflammation thus seemed to respond fairly well to this treatment.

However, the cardiac signs were worse in 2 of the patients and questionably so in a third patient of the group at the conclusion of the course of treatment in comparison with the findings before cortisone therapy was instituted.

**CASE REPORTS**

1. M. B., a 22 year old Negro woman, was admitted to the hospital on Dec. 26, 1949 in her first known attack of rheumatic fever, complaining of dyspnea, ankle edema, sore throat, and pain in the left knee of six days' duration. Her temperature was 102 F. orally and her pulse rate 90. She was dyspneic and orthopneic, and complained of severe chest pain. A low-pitched blowing systolic murmur was heard at the apex and a to-and-fro friction rub was heard at the base of the heart. Rales were audible over the bases of the lungs. The liver edge was felt 6 cm. below the right costal margin. Studies on admission showed the following: Chest x-ray films showed: pericardial effusion and small bilateral pleural effusion; the ECG showed inverted T waves in leads II and III, aVp and all precordial leads, and slight S-T elevation in II, III and aVp. Laboratory studies: (for brevity, the corrected erythrocyte sedi-
mentation rate, in mm. per hour, will be indicated by ESR; white blood cell count per cu. mm. by WBC; hemoglobin concentration, in grams per 100 cc. of blood, by Hb; electrocardiogram by ECG; dates will be conventionally abbreviated.) ESR: 87; Hb: 8.1; WBC: 12,500.

Cortisone therapy was instituted on 12/29/49, nine days after the onset of the illness. She had been receiving aspirin from the time of admission to 12/28/49. The patient continued to become worse. On 1/1/50 the respiratory rate was 60, and the dyspnea was so distressing as to require the administration of oxygen. She was severely ill for the next two days, and then began to show a number of signs of improvement.

In the ECG of 1/3/50 all the abnormally inverted T waves had become upright. Her temperature fell to normal by 1/4/50, and the ESR fell from over 110 on 12/30/49 to 18 on 1/13/50. The friction rub was no longer audible on 1/9, at which time the dyspnea was considerably decreased, and an x-ray film on 1/13 showed cardiac enlargement but no pericardial effusion. The liver could not be palpated or percussed on 1/19. Cortisone was discontinued on 1/19, at which time her disease process was subsiding but not inactive. Her apical systolic murmur had become more harsh and blowing in character, and was now transmitted to the midaxillary line. A diastolic third heart sound was occasionally heard at the apex. The Hb had risen to 10.1 Gm., and the WBC had fallen to 10,700.

Her convalescence continued uneventfully until 1/23, when she complained of precordial pain. Dyspnea reappeared and the pulse rose from 100 to 130. On 1/24, her temperature was 101 F. Bilateral pleural effusion reappeared and increased until 1/27 when it began to resolve. She complained of pain in the shoulders on 1/26 and pain in the right knee on 1/27. Oxygen therapy was necessary from 1/24 to 1/27. The ESR rose from 23 on 1/23 to 90 on 1/25 and 110 on 1/28. Her liver edge descended to 5 cm. below the right costal margin. The ECG showed only a sinus tachycardia.

Cortisone was restored on 1/25 at a dosage of 112.5 mg. per day, and was later briefly raised to 150 mg. per day. Her temperature immediately began to fall, reaching normal in three days. Other symptoms and signs which were on the upgrade when cortisone was restored continued to become worse for three more days and then fell off rapidly. The ESR fell from 110 on 1/25 to 20 on 2/1, and her dyspnea had disappeared by this day. She had no joint or precordial pains after 1/27. The ECG of 1/30 was within normal limits. The pulse rate fell from 120 on 1/20 to 84 on 1/30. The dosage of cortisone was gradually reduced. The drug was discontinued on 2/13, at which time the dosage was 3 mg. per day.

Three days after the cortisone was stopped, she complained of coryza. The ESR rose to 30, then, four days later, to 75. Her temperature rose to 100 F. orally. Without further treatment she again became afebrile in three days and her ESR became normal within a few days. The Hb rose to 10.8

At this writing the patient feels well. The signs of inflammation are gone, but there is cardiac enlargement, mitral insufficiency with transmission of the murmur to the posterior axillary line, and an occasional early diastolic apical blowing murmur. Inversion of the T waves in her ECG has recurred. Some of the clinical features of this case history are presented in figure 1.

In view of the greater sensitivity of the elevated ESR than leukocytosis as evidence of activity of the rheumatic process, and of the recent observation of leukocytosis in children under treatment with ACTH, by Hain and Wilson, it is very probable that the leukocytosis noted in the case history and chart is indicative of effective levels of cortisone and not of activity of the rheumatic infection.

2. B. D., a 4 year old Negro girl, was admitted to the hospital on Jan. 12, 1950 complaining of severe epistaxis, fever, malaise and anorexia of one day’s duration. On admission the child was acutely ill, with a rectal temperature of 104 F. and a cardiac rate of 160. A blowing systolic murmur (which had been present before this episode) was heard, with maximal intensity at the apex and transmission to the midaxillary line. Initial laboratory studies showed the following: ESR: 85; WBC: 16,500; Hb: 8.0; ECG: sinus tachycardia; chest x-ray films: globular heart with prominence of the upper right border and suggestive mitralization of the left border; cardiothoracic ratio: 0.62.

A diagnosis of acute rheumatic fever was made and cortisone therapy was instituted three days after the onset of the illness at a dosage of 75 mg. per day. Her temperature began to descend on the second day of treatment and was normal by the fourth. Similarly her pulse rate fell to 100 to 110. The ESR, which was 100 on the first day of treatment, fell to 60 on the sixth day and to 15 on the tenth day.

The patient, however, did not reflect the favorable appearance of her clinical chart. She remained listless and anorexic. When her heart rate became slower, a blowing early-diastolic apical murmur became audible. On 1/23, 10 days after cortisone was started, she suddenly developed edema of the face and legs, ascites, hepatomegaly, further cardiac enlargement (her apex beat moved down and to the left), and a sleeping pulse rate of 148. Serum sodium and potassium concentrations were within normal limits. An x-ray film of the chest showed evidence of early congestive changes. To combat the acute decompensation, oxygen therapy, Theocain, and a salt free diet were employed. Penicillin and aureomycin were administered because of the fear of a complicating pneumonia. On this regimen her edema melted away, so that by 1/31 she had returned to a state
similar to that prior to the acute heart failure. All the special measures except Theocalcin were discontinued. The ESR at this time was 12. The next few days were characterized by notable subjective improvement. On 2/1 the dose of cortisone was reduced to 22.5 mg. per day, and on 2/2 it was stopped.

On 2/4 it was noticed she was lethargic and had slight but definite ankle edema. When questioned she complained of a sore throat. Her temperature was 104 F., rectally, and her pulse rate 168. The ESR rose from 6 on 2/4 to 22 on 2/8. Penicillin was administered. The patient improved gradually until 2/7 when she suffered three severe nosebleeds. Her temperature, which had been falling gradually, reached normal on 2/9, five days after its sudden rise. Her pulse rate fell to a range of 100 to 140. Penicillin was stopped on 2/9. Her course since has been one of steady subjective improvement.

Studies during this period showed the following: the Hb rose from 7.0 to 9.4; the WBC fell from 18,600 to 5,850; two ECG tracings were within normal limits. However, a tracing taken on 2/23 showed a top-normal Q-T interval, QRS complexes of low amplitude, and left axis deviation.

At this writing her illness has been apparently quiescent for three weeks and her convalescence is continuing. Physical examination shows the apical systolic and diastolic murmurs have persisted. A chest x-ray film made on 2/23 shows the heart to be the same size and shape as at the onset of her illness.

3. F. M. W., a 13 year old Negro girl, was admitted to the hospital on December 15, 1949 in her first known episode of rheumatic fever, complaining of fever, sore throat, pain and redness of one knee, nosebleeds, and abdominal pain of four days' duration.

In the hospital the fever and abdominal pain were soon overshadowed by excruciating pain, swelling, and tenderness of the elbows, knees and ankles. Physical examination showed, in addition to the arthritis, a blowing, deesendo, diastolic murmur confined to the second intercostal space to the left of the sternum, and a soft, blowing, systolic, apical murmur transmitted only to the anterior axillary line. Other studies during this initial period yielded these average values: WBC: 18,150; Hb: 9.2; and ESR: 105.

A diagnosis of acute rheumatic fever was made and cortisone was started on 12/29 at a daily dose of 150 mg., which was reduced to 112.5 mg. after two days. On the day cortisone was started she presented the following findings: temperature: 104 F.; ESR: 105; WBC: 17,300; and Hb: 7.2.

The temperature immediately began to fall, reaching normal after three days of treatment. The joint pains were slightly eased for two days, then rapidly improved until 1/2/50, when, after four days of cortisone, she was free of pain. Her ESR continued to rise for four days, when it exceeded 110 mm. in one hour, then decreased during the next 10 days to a value of 32 on 1/14. In addition to the aortic diastolic and mitral systolic murmurs heard previously, a blowing, early diastolic apical murmur had appeared, as well as a presystolic apical murmur.

Cortisone, which had been reduced in dosage to 75 mg. a day, was discontinued on 1/19. Four days later her temperature began to rise, reaching 100 F. on 1/25. The evening of 1/24 she developed acute pain, swelling, heat and tenderness of the right wrist.

Cortisone was restarted on 1/25 at a dosage of 112.5 mg. per day. The next day pain and tenderness of both knees appeared. The ESR, which had been ranging from 40 to 49, rose to 72 on 1/25. The day after cortisone therapy was reinstituted her temperature had returned to normal. All joint manifestations had disappeared by 1/28. Her ESR continued to rise, however, to a value of 105 on 1/28. It then began to decrease at the same rate as when cortisone was first given, this time, however, reaching a lower value.

Her course since then has been one of steady improvement except for a mild "tightness" in her throat and enlarged sublingual glands which persisted from 2/3 to 2/15 in spite of penicillin therapy. She has been afebrile. Cortisone was gradually reduced in dosage until 2/20, when it was stopped.

Studies during this period included the following: three ECG tracings within normal limits; no glycosuria since cortisone was started; blood sugar levels normal on six occasions, and one at 121 mg. per 100 cc.; WBC gradually decreasing from 25,100 on 12/21/49 to 6,700 on 2/23/50; Hb rising from 7.2 Gm. on 12/29/49 to 11.3 Gm. on 2/14/50.

At this writing her convalescence is progressing satisfactorily. Physical examination shows the persistence of the apical systolic, diastolic, and presystolic murmurs and the aortic diastolic murmur. Ten days after cortisone was discontinued an abnormal ECG was recorded for the first time. The Q-T interval was prolonged and the T waves became flattened or diphasic.

4. J. K., a 14 year old white boy, was completing a five months' convalescence from a second episode of rheumatic fever when he contracted pharyngitis in late November, 1949. A week later there was a recurrence of rheumatic fever, characterized by repeated epistaxes, fever, dyspnea, and migratory joint pains. A screeching component appeared in the pre-existing apical systolic murmur, and the ESR rose from 12 to 90 mm. per hour. Because of the progressive anemia due to the profuse epistaxis, and his general downhill course, the patient was transferred from his convalescent home to a hospital, where blood transfusions were given.

In this hospital he was found to have, in addition to the cardiac findings mentioned above, an early diastolic apical murmur and basal rales. He was
suffering intensely from joint pains and was dyspneic enough to require oxygen therapy. Aspirin and penicillin were given without any improvement. The patient was transferred to the Philadelphia General Hospital on 12/19/49 for treatment with cortisone.

At this date the patient had been acutely ill for four weeks in his third attack of rheumatic fever. He was slightly dyspneic and orthopneic, and suffering repeated epistaxis, arthralgia and precardial pains. His temperature was 102.5 F. and his pulse rate was 120. He presented the murmurs characteristic of mitral insufficiency, mitral stenosis, and aortic insufficiency. His heart was enlarged. Laboratory studies were as follows: Hb: 8.0; WBC: 17,900; ESR: greater than 110; ECG: increased P-R interval.

Cortisone was started on 12/20/49 at a dose of 112.5 mg. per day. By 12/23 all joint manifestations had disappeared. However, the slowly descending temperature and ESR plus the relief of his arthritis were the only improvements noted at this time. On 12/22, two days after cortisone was initiated, his epistaxis recurred. On 12/23 his dyspnea and orthopnea became worse; he complained of severe precordial pain, a pericardial friction rub became audible, and an ECG confirmed the presence of pericarditis. Signs of heart failure appeared: his liver edge descended to 7 cm. below the costal margin and the rales at his lung bases became more obvious. Two days after cortisone therapy was begun the P-R interval was normal although the S-T segments were greatly elevated in the fashion characteristic of acute pericarditis. Auricular fibrillation was noted clinically and by ECG on one occasion (12/27).

By 1/4/50, 12 days after its onset, pericarditis had disappeared. At this date his ESR was 46 and his temperature 99 F. Premature beats were heard on 1/6, and on 1/7 auricular fibrillation recurred. He remained comfortable except for the dyspnea, however, until 1/8 when he developed acute pulmonary edema for which he was treated with oxygen, phlebotomy, and digitalization. After this episode he began a period of slow steady improvement. His heart reverted to a normal sinus rhythm, his ESR fell to 10, and there were no peripheral signs of heart failure. His cardiac findings were unchanged except for the appearance of a systolic thrill and murmur over the aortic area. The dosage of cortisone was reduced to 75 mg. a day on 1/11 and the drug was discontinued on 1/14 after 26 days. Digitalization was maintained.

The rheumatic process was considered to be subsiding in this patient until 1/23, nine days after cortisone was stopped, when he complained of pain in the left knee. During the next few days the other knee, one shoulder, and his chest became involved, the pain progressively becoming more severe. His temperature rose to 102 F., his pulse to 120 and his ESR to 70. His liver edge descended to 6 cm. below the right costal margin. The increased P-R interval reappeared.

Cortisone was restored on 1/24 at 75 mg. per day. The arthritic symptoms immediately began to recede, and the patient was free of pain by 1/27. His temperature reached its previous level by this day, and fell to normal three days later. His ESR fell from 70 on 1/28 to 21 on 2/1. The dyspnea improved sharply and by 2/3 had disappeared, although some orthopnea continued. An ECG on 2/14 showed a normal P-R interval, and, for the first time, an improvement in the T waves. The ESR fell to 7 on 2/8. Except for the improvement in cardiac compensation, there were no other cardiovascular changes. The dosage of cortisone was slowly decreased and the drug was discontinued on 2/20.

On 2/19, at which time the cortisone dosage was 1 mg. per day, the patient complained of a sore throat and his temperature was 100 F. orally. Headache, leg and shoulder pains, and earache developed over the next few days. The fever continued to rise, reaching 101.5 F. on 2/22. His ESR rose to 43 on the same day. A throat culture showed no recognized pathogens. Penicillin was given from 2/19 to 3/5. The temperature returned to normal after seven days of fever, the ESR reached normal limits in 14 days, and the Hb had risen to 13.8 on 2/14.

At this writing the disease process is apparently subsiding. The evidence or profound cardiac damage is still present, with the addition of an aortic systolic murmur and thrill. His electrocardiographic abnormalities are decreasing. He is continuing to receive digitalis.

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