T waves was not prevented from recurring in eleven cases. Nineteen patients who had organically inverted T waves because of left ventricular strain and/or organic heart disease were given an intravenous injection of 0.5 mg. of either ergotamine tartrate of dihydroergotamine 45. Seven of these patients exhibited a normalization of the T waves. Five patients developed severe anginal pain which lasted from two to twelve hours, and one of these patients who had syphilitic aortitis with narrowing of the coronary ostia died twelve hours after the injection.

From these observations, it is concluded that ergotamine preparations will not invariably prevent the inversion of the T waves with a change of position of the patient. Ergotamine preparations can cause a normalization of organically inverted T waves and therefore cannot be used as a diagnostic test. Their use is dangerous in patients with coronary artery disease.

CLINICAL OBSERVATIONS WITH FAGARINE.—D. SCHERF, M.D., A. M. SILVER, M.D., AND L. D. WEINBERG, M.D., NEW YORK, N. Y.

Fourteen injections of alpha-fagarine hydrochloride were given to thirteen patients with various forms of tachycardias, auricular flutter, and auricular fibrillation. The dose varied between 0.05 and 0.12 gram. In six patients the existing arrhythmia disappeared promptly after the injection. In two patients, however, fatal ventricular fibrillation appeared; in three others dangerous multifocal ventricular extrasystoles were observed.

The observations and side reactions in all cases are discussed and the risk involved in the use of this drug is stressed.

ETIOLOGY OF AURICULAR FIBRILLATION AND THE MECHANISM OF ITS PERPETUATION.—J. G. SCHLICHTER, M.D., CHICAGO, ILL.

In this report, our experience in man and dog relating to the etiology of auricular fibrillation and the mechanism of its perpetuation is presented. Vagal stimulation and anoxia are the main etiological factors in the initiation and perpetuation of auricular fibrillation.

Vagal stimulation (mechanical and chemical) may induce auricular fibrillation. Acetylcholine injected directly into the blood stream was used in our experiments to produce chemical vagal stimulation. Moderate anoxia reduces the threshold of the auricles to the initiation of fibrillation, but does not induce this arrhythmia, per se; marked anoxia, on the other hand, increases the threshhold to fibrillation.

Anoxia of the auricles was found or produced: (1) by interference with or obstruction of its vascular supply, (2) by a decrease in the amount of oxygen carriers, (3) by a decreased oxygen content of the blood due to anoxic anoxemia, and (4) by interference with tissue respiration.

The relationship between vagal stimulation and anoxia can be plotted in a graph, and on this correlation, the cause of the perpetuation of auricular fibrillation can be demonstrated. The clinical implication of these findings and the therapeutic approach to this problem are illustrated and discussed.

CARDIOVASCULAR CHANGES IN PERNICIOUS ANEMIA BEFORE AND AFTER THERAPY.—STEVEN O. SCHWARTZ, M.D., AND VLADIMIR C. FLOWERS, M.D., CHICAGO, ILL.

Ninety-two patients with pernicious anemia, ranging in age from 35 to 81 years, and equally distributed between the sexes, were studied while in hematologic relapse. Of these sixty-nine were Caucasian and twenty-three were Negro. Fifty-five complained of dyspnea, thirty-one of edema, eight had angina

pectoris, and one had intermittent claudication. Fifty-nine had systolic murmurs, distributed as follows: apical, thirty; pulmonic, ten; aortic, six; diffuse, thirteen. One patient had an apical diastolic murmur which disappeared on liver therapy (this patient had hypertension). Only eighteen patients had blood pressures over 150/90 while in relapse. Seventy-two of the patients had electrocardiograms taken; of these, forty-four were interpreted as abnormal. Fifty-seven patients had their cardiac size determined by x-ray films, thirty being found normal.

Forty-four patients were re-examined when blood values had returned to normal (three months). Of these, eleven had systolic murmurs (five apical, five aortic, one pulmonic), while nineteen had hypertension (150/90 plus). Electrocardiograms revealed abnormalities in twenty of forty-one repeat studies. Cardiac size was found to be normal in twenty-five of thirty-seven patients re-examined.

Discussion will center on the interpretation of these findings as it relates to: (1) the criteria of diagnosing cardiovascular disease in the presence of severe anemia; (2) the significance of the findings when reinterpreted on the basis of the age group involved in the study; and (3) the influence of the rapidity of onset of the anemia on the symptoms and findings.

CARDIOVASCULAR SYPHILIS IN YOUNG WHITE MALES.—JOHN B. SCHWEDEL, M.D., AND KONA SIMON, M.D., NEW YORK, N. Y.

Five hundred ninety-five syphilitic men and 786 controls were examined for auscultatory and radiographic findings to determine the incidence of aortic dilatation, aortic insufficiency, and a combination of aortic dilatation plus aortic systolic murmur and/or accentuation of the second aortic sound. The average incidence of dilated aorta was 4.2 per cent in the controls and 18 per cent in the syphilitic group. Ausculatory findings were three times as frequent in the syphilitic group without aortic dilatation and twice as frequent when the aorta was dilated. Isolated aortic insufficiency was present in 7.2 per cent.

Criteria for the diagnosis of syphilitic aortitis are suggested consisting of the presence of dilated aorta combined with significant auscultatory findings. The presence of a dilated aorta in syphilitics below the age of 40 years in the absence of aortic insufficiency, plus aortic systolic murmur or accentuation A_2, is sufficiently frequent to warrant the presumptive diagnosis.

Radiographic and fluoroscopic criteria, consisting chiefly in increased arching in the posteroanterior and left anterior oblique views, are illustrated.

PARCHMENT HEART (OSLER).—HAROLD N. SEGALL, M.D., MONTREAL, CANADA.

In revising the sixth edition of his textbook, *The Principles and Practice of Medicine*, Osler introduced the following paragraph in the chapter on dilatation of the heart: "Dilatation may be chronic, in which case it is associated with hypertrophy. Not always, however; there is an extraordinary heart in the McGill College Museum showing a parchment-like thinning of the walls with uniform dilatation of all the chambers; in places in the right auricle and ventricle only the epicardium remains." Periodic long searches of medical literature were made in the past few years and no reference to a similar case of dilatation and generalized thinning of the myocardium could be found. This unique specimen represents a condition about which one can only speculate in considering the etiology and pathologic physiology. The records of the McGill Museum contain no clue to the patient's history in this case. It is significant that neither the heart valves nor the myocardium reveals any evidence of inflammatory disease