Staff Meeting Bulletin
Hospitals of the . . .
University of Minnesota

Pericarditis

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INDEX

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William A. O'Brien, M.D.

I. LAST WEEK

Date: November 12, 1936

Place: Recreation Room
Nurses' Hall

Time: 12:15 to 1:10 P.M.

Program: Movie: "Oxygen Therapy - Care of Apparatus"

Abstract: Thyroiditis

Case Reports: Thyroiditis

Present: 128

Discussion: C. E. Rea
C. O. Rice
J. C. McKinley
R. W. Koucky
A. L. Herman
R. M. Johnson
P. H. Fesler

Gertrude Gunn
Record Librarian

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II. MOVIE

Title: Master Will Shakespeare

Released by: M-G-M

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III. ABSTRACT

PERICARDITIS

R. E. Mattison

Chronic adhesive pericarditis is a disease which is comparatively rare (2 to 5% of all autopsies). However, interest has been stimulated in the condition because in many cases satisfactory therapeutic results may be obtained. A considerable amount of the following abstract is taken from Rothstein's articles.

Chronic adhesive pericarditis is the end result of an acute pericardial inflammation of rheumatic, tuberculous or unknown origin. The most common cause is rheumatic infection, and the next tuberculosis. Often the cause is unknown, the antecedent acute inflammation having escaped notice. In this third group, the etiologic factor may have been a pyogenic microorganism: pneumococcus, streptococcus or less commonly staphylococcus and rarely influenza bacillus (Taylor) or the gonococcus (Huner and Bubis). Trout points out that adhesions are the end results of a defensive process on the part of the body and therefore it would be unusual to find any definite organism late in the disease. Rarely, blood in the pericardial cavity may cause the adhesion.

It is interesting to note the causes of acute pericarditis are somewhat different. They are in order of frequency: pneumonia, rheumatic fever, nephritis (uremia), tuberculosis, sepsis, aneurysm, cardiac infarction and typhoid fever. The discrepancies may be explained by the fact the acute pericarditis is frequently seen only in the terminal stages of a number of these; namely, uremia, sepsis and aneurysm. Pneumonia may be more important as an etiological agent of chronic adhesive pericarditis than is listed above.

Age

The greatest incidence of the pathological condition is found in the middle age (35 to 45). However, chronic pericarditis may exist at any age.

Sex

Males are more often affected; in a ratio of about 3 to 1.

Pathology of Pericarditis

Acute fibrinous pericarditis consists

of an infiltration of the pericardium with mononuclear and polymorphonuclear cells and a more or less adherent layer of fibrin covering a part or whole of the pericardium. It starts on either the parietal or visceral pericardium, but, as a rule, eventually involves both surfaces. The exudate sometimes is very massive, a centimeter or more in thickness. An increase of fluid in the pericardial sac is commonly found. The exudate may be purulent, serous or hemorrhagic. When the inflammatory or irritative process undergoes resolution and repair, there are left varying degrees of thickening of the pericardium. When the acute process is at all marked in degree, or extensive, adhesions, partial or complete, between the visceral and parietal pericardial surfaces are the rule and if the inflammatory reaction has been deep, the heart may be firmly anchored to the diaphragm, chest wall, pleura or mediastinum, or it may be encased in a firm, thick, unyielding scarred and contracted pericardium with little or no serous sac left. Hence, chronic adhesive pericarditis may be divided pathologically into 5 groups:

1. Slight scarring with thickening and fibrosis of precordium in small areas without any clinical effect (milk spots may be included here).

2. Slight loose localized pericardial adhesions of fibrous tissue which have no effect on size or function of heart.

3. Complete adhesion between the visceral and parietal pericardial surfaces without firm fixation to chest wall, diaphragm or mediastinum. In such cases, the adhesions are often loose and the pericardium is but little thickened so that there is no handicap to the heart in its function and no cardiac enlargement.

4. The adhesions may be solid and the pericardium thickened, contracted and even calcified in places so that the heart function is impeded, the movements much limited and the inflow of blood from the great veins obstructed (concretio cordis). This is usually associated with mediastinal involvement but not necessarily.

5. The adhesive pericarditis may be complicated by an extension of the process to the diaphragm or chest wall so that the heart itself is anchored to one or other of these structures or both by firm adhesions.

In the last two groups, obstruction of the great veins of the heart and hepatic veins may occur by kinking and compression. This leads to hepatic congestion, enlargement and eventually cirrhosis which is the mediastinopericarditic pseudocirrhosis originally described by Pick in 1896. This may or may not be associated with polyserositis and perihepatitis, icing or frosting of the peritoneum over the liver (Zuckergussleber) due to chronic peritonitis. It should be added that in his recent work, Beck has noted that similar changes were found on the liver of dogs whose pericardial cavities had been injected with Dakin's. Occasionally, lime and rarely even bone is deposited in chronic pericardial adhesions when they are especially thick and massive.

Beck and Griswold have also proved experimentally in dogs that this syndrome may occur without the formation of adhesions between the heart and percardium. They have shown that the presence of generalized intrapericardial adhesions is not necessarily associated with the Pick syndrome since the essential feature for the development of the latter condition is not the mere presence of adhesions but the compression effect exerted on the heart by the contraction of the scar tissue. Obliteration of the pericardial cavity by adhesions, therefore, of itself may not necessarily produce cardiac disability unless the pericardium is contracted so that the expansion of the heart is hindered.

Abnormal Physiology

Signs and Symptoms

Clinically, cases of chronic ad-

hesive pericarditis may be grouped into two main divisions according to the type and location of the adhesions.

First: There is the group in which the adhesions are between the parietal pericardium and either the mediastinal structures or the diaphragm, or both. The mediastinal disease may bind the pericardium to the chest wall and in this event the systolic contraction of the heart will be hindered, for in every systole the cardiac muscle must contract against and overcome the resistance of the rigid, bony and cartilaginous framework of the anterior chest wall. The cardiac decompensation occurring under such circumstances may be attributed to the strain on the heart caused by this abnormal fixation, particularly since the heart has probably suffered some degree of myocardial damage from the infection originally causing the pericarditis.

Second: Either the adhesions are between the two layers of the pericardium and the heart lies encased in an inelastic, firm envelope bound by a thickened and scarred pericardium (concretio cordis).

In these cases, the heart eventually becomes so throttled by the unyielding wall of scar tissue that its chambers can no longer expand in diastole to receive the inflowing blood and a marked degree of stasis, particularly of the caval system, is produced with gradual dilation of the veins. This results in Beck's clinical triad of chronic intrapericardial pressure, namely: high venous pressure, ascites, and small quiet heart. Volhard has termed this "inflow stasis" because of the picture of lack of overfilling of the right side of heart (which is held undilated by constriction of scarred pericardium) even in the presence of high degree of venous stasis.

Obviously, variations in degree and extent of adhesions in these two general groups must exist and the degree of interference with the proper function of the heart and the degree of severity of the clinical symptoms will depend upon the resultant contraction of the scarred pericardium. Furthermore, the two types may be present in the same case and varying forms of transition between the two may occur. In general, the patients in the first group with adhesions between the pericardium and the anterior chest wall progress until they show a more or less high degree of decompensation with characteristic signs and symptoms of chronic cardiac passive congestion. Since the heart is bound to the thorax by adhesions, systolic retraction of the precordial area of the chest wall and a "bulging back" (diastolic shock) during systole may occur in the second type of case where the heart is throttled by a contracted fibrous pericardium and the clinical picture is characterized by a striking disproportion between the slight or even absent objective cardiac signs and the marked degree of signs and symptoms of cardiac passive congestion.

In either type, the onset is insidious. The course is chronic sometimes over a period of years. A careful history is an important aid in making a diagnosis, for careful questioning may bring to light the details of a previous acute pericarditis or of repeated rheumatic or tuberculous manifestations.

When in the case of the last group, the heart is exhausted by the strain of tugging on the chest wall or of pulling up the diaphragm with each contraction. The usual symptoms of congestive failure appear of which the chief and earliest is dyspnea. When the heart is so compressed by encasing external adhesions, thickened fibrous pericardium or intramyocardial scarring that there is an insufficient filling and emptying of the heart, dyspnea, weakness and marked venous stasis of the abdominal viscera occur.

The most common sign of chronic adhesive pericarditis, especially in the first group is cardiac enlargement, but this is inconstant and not diagnostic. The venous enlargement when present is due to decompensation. White states that some of the largest hearts ever noted (over 1,000 grams) have been found associated with chronic mediastinopericarditis. On the other hand, when the heart is encased in a contracted

fibrous pericardium, the heart may be small due to constricting effect of scar tissue.

Another important and distinctive physical finding in cases of chronic adhesive pericarditis is that of hepatic engorgement (and cirrhosis) with ascites alone or out of proportion to the degree of dependent edema in the lower extremities. This occurs especially in cases of chronic constrictive pericarditis. The presence of the marked ascites with little or no extremital edema is a point of great interest.

Rolleston explains this phenomenon as follows: The pericardial adhesions by contracting lead to rigidity and dilation of the right auricle, inferior vena cava and hepatic veins, and by this means free regurgitation of blood into the liver is rendered permanent. It is possible that at the time of the primary pericarditis, inflammation spreads to the mouth of the hepatic veins and by weakening their walls leads to dilation and so to a freer entry of blood into them. When once brought about, this dilation of the hepatic veins becomes permanent. The brunt of the backward pressure thus falls on the liver, while other branches of the inferior vena cava, the renal and iliac veins, suffer less than in ordinary cases of chronic engorgement of cardiac origin. Thus, since the iliac veins are not apt to be markedly involved, edema of the extremity does not result. When the ascites has accumulated to a marked extent, ascitic fluid may press on the iliac veins and edema of the extremities may then result.

A sign which occurs in about 8% of cases and which has been said to be a confirmatory sign of extensive pericardial adhesions is that described by William Broadbent and his son, Walter. It consists of a systolic retraction of one or more intercostal spaces to the left of the sternum (more commonly of the 3d, 4th or 5th), particularly in the left axillary region or in the posterior intercostal spaces in the region of the 11th and 12th ribs. Although this sign is sometimes also seen when the heart, especially the right ventricle, is very large without adhesions, it is best seen in cases of adherent pericardium. Kegelsberger considers this sufficient to make the diagnosis in doubtful cases. This sign of course is not present in the constricting type listed above.

The paradoxical pulse (Kussmaul's sign), an inspiratory distention of the veins without forced breathing, may be present. Should a tricuspid regurgitation develop in a case with dilated cervical veins, pulsation of the cervical veins will be observed. A sudden collapse of the veins of the neck during systole is often present (Friedrich's sign) and when associated with a systolic retraction of the intercostal spaces is a very valuable sign.

The blood pressure is low if the blood flow is much reduced by the hampered action of the heart. It may be high if a marked myocardial hypertrophy with no signs of decompensation is present. The venous pressure is elevated. It is determined, according to the method of Eyster, by inserting an aspirating needle into the cephalic vein of the arm at the level of the heart and then measuring the pressure against a column of physiological saline. In Beck's case, the venous pressure had risen to 35 cm. of physiological saline. Usually 25 cm. of physiological saline is reported. Eyster gave the normal limits as 4 to 6 cm. of water with an upper limit of 11 cm. of water. Beck regards the increased venous pressure as the most reliable index of a contracted and restraining pericardium.

Friction rubs may occasionally be heard. De Teyssier reported a case in which a rather small collection of pericardial fluid produced friction rubs. This observation was confirmed at autopsy and tuberculous pericarditis was found. In Richter's case of adherent pericardium, friction sounds were heard during life; at necropsy, some old blood clots were found in the loose connective tissue of the pericardial adhesions. Reiss reported an interesting sign: that of loud metallic heart sounds heard over the stomach. He interpreted them as being caused by the adherence of the pericardium to the diaphragm.

Murmurs may be present due to valvular disease caused by the same disease process responsible for the pericarditis. Thirty-four per cent of Sears' series of adhesive pericarditis were accompanied by valvular disease of one form or another. On the other hand, Horst found that 30 cases of 61 of adherent pericardium had no valvular defects. In Smith and Liggetts series of 107 cases, valvular disease was noted in 29%. Rothstein found 29.4% of 34 cases in his series with cardiac murmurs. However, the apical systolic murmur found with cardiac enlargement may not be caused by actual valvular disease but may be due to functional mitral insufficiency. Very rarely, there may also be a mitral diastolic murmur at the apex without actual mitral or aortic valvular disease due simply to the marked cardiac dilatation secondary to the pull of mediastinopericardial adhesions.

According to White, auricular fibrillation is not uncommon in the more marked cases. Fixation of the heart when present is distinctive of a severe degree of chronic adhesive pericarditis. However, this is hard to determine. In some cases, physical examination, roentgen-ray examination and electrocardiography all show little or no change in the position of the heart with change in body position. When the heart is fixed to the sternum, the heart rises rather than descends with inspiration, and retraction of the lower end of the sternum occurs at the same time the fixation of the heart and pleural edges by pleuro-pericardial adhesions may prevent any change in the percussion of the pulmonary resonance over the heart between full inspiration and full expiration. The area of "absolute" cardiac dulness may be increased since the pleural edges are usually somewhat retracted from over the heart. When definite evidence of fixation of the heart is present, it is diagnostic of chronic adhesive pericarditis. When the signs are doubtful, as is usual, they cannot be relied upon.

Electrocardiography

In cases of adherent pericarditis, electrocardiography is of questionable value: It was thought that normally there is a change in the form of the electrocardiogram on shifting the patient to the right or left side due to rotation of the heart about a longitudinal axis. The most noticeable change is in the height and conformity of the QRS wave in the different leads, although all the waves are affected to a certain degree. Furthermore, although all of the three leads are affected, usually only leads I and III show marked variation. Dienarde found that this normal change or shift of the electrical axis was absent in the electrocardiography of cases of chronic adhesive pericardiomediastinitis and believed that this fixation of the electrical axis of the heart was diagnostic of the condition. However, Einthoven in a series of normals finds a shift of 5 to 55% in the electrical axis. He also found 3 to 42% shift in a series of cases with mediastinopericardial adhesions. He also points out that effusion into the pericardial cavity produces at times a similar picture. He concludes therefore that normal rotation occurs in the presence of disease and that fixation of the electrical axis is not pathognomonic. He states that physical examination and x-ray are more valuable. Other abnormalities in the electrocardiogram are seen, such as abnormalities in the T waves, low voltage and notching, and slurring of the QRS complex but these may occur in other conditions.

Roentgenography

Roentgenological findings are among the most important in establishing a diagnosis of chronic adhesive pericarditis. When calcification of the pericardium is observed on x-ray examination, the finding is pathognomonic of adhesive pericarditis, according to White. Only 10% show calcification however and these are not all demonstrable. Roentgen examination also infrequently shows actual irregularities in the contour of the cardiac shadow, due to the pull on the heart of pericardial adhesions attached to the diaphragm, pleura or strictures of the mediastinum.

Lukin describes a fluoroscopic sign of a partially adherent pericardium--

that of a rhythmic tug of a part of the left dome of the diaphragm near its center with each systole of the heart, with the diaphragm fixed in inspiration. He explained this on the basis of an adherence of fisceral and parietal pericardium. E. S. Smith noted the same sign in one of his cases.

Holmes states that in the presence of pericarditis, the pulsation of the heart becomes diffused over the entire cardiac area and that it is impossible to distinguish the auricular beat from that of the ventricle, although this can ordinarily be done under fluoroscopy in the normal heart. This, he states, is present both with adhesions and effusion. He also noted that Rotch's sign of obliteration or change in the cardiohepatic angle was more likely to occur in pericarditis with adhesions rather than in cases of effusion. In his experience, other findings, such as retraction of the diaphragm with the heart beat and fixation of the heart within the chest as shown by failure to be displaced on turning the patient from side to side, were subject to so much error in observation by x-ray that he found them of little value. Holmes also presents another sign. In the lateral view, particularly if the mediastinum is involved, the heart may rise with the chest wall instead of dropping with the diaphragm during forced breathing. This is a very definite and easily interpreted sign.

A recently utilized development in the x-ray study of the heart which seems to be of extreme value in cases of chronic adhesive pericarditis is the roentgenkymograph, as described by Hirsch and as used by Johnson at Louisville. The roentgenkymograph is an instrument which records by x-ray the amplitude of the excursion of the heart borders during one or more cardiac cycles as desired. Simply, it records permanently what may be seen on fluoroscopy. There are few reports as yet but the results are extremely favorable.

Diagnosis

The diagnosis of adherent pericardium may be very difficult. An adequate and painstaking history regarding a previous pericarditis or evidences of either a rheumatic infection or tuberculosis may afford a clue. The patient's attention may first be attracted by a progressive enlargement of the abdomen, accompanying weakness, dyspnea and cyanosis. Edema of the legs and hydrothorax may or may not be present.

Physical examination of the heart may reveal a striking paucity or even absence of signs when compared with the signs of circulatory failure present. In such a case with an enlarged liver, ascites and proportionately little or no edema of the extremities, the diagnosis of adhesive pericarditis is to be strongly considered. Percussion may show definite fixation of the heart on shift of position, the fluoroscopic, x-ray and electrocardiographic examinations may help to confirm the presence of adherent pericardium.

Differential Diagnosis

When chronic adhesive pericarditis produces any signs at all, it must be differentiated from any other cause of cardiac enlargement or failure. This can be done when other etiologic factors are present, such as hypertension or volvular disease. However, it must be remembered that valvular disease may be associated with adherent pericardium (about 30% of cases).

Chronic adhesive pericarditis should be suspected more often than it is as a possible etiologic factor in cases with cardiac enlargement of unknown origin, especially in young people or children.

Chronic mediastinopericarditic pseudocirrhosis of the liver and concretio cordis must be differentiated from cirrhosis of the liver due to other causes. This may be very difficult and even impossible.

Treatment

Treatment of chronic adhesive pericarditis may be divided into medical and surgical treatment.

1. Conservative treatment consists of measures for the support of the heart and failing circulation. These usually include restriction of activity, digitalization and the use of diuretics. Usually abdominal paracentesis and thoracentesis must be done to relieve the ascites and pleural effusions when and if they occur. Usually relief if obtained is only temporary and the reaccumulation of fluid in the serous cavities necessitates repeated aspirations. Ultimately, the patient succumbs after a varying interval of months or years from progressive cardiac decompensation and exhaustion or is carried off by an intercurrent infection which of itself might be of little moment but when superimposed on a failing heart leads to death. The use of digitalis certainly can be of no conceivable value in cases of concretio cordis and constricting mediastinopericardial adhesions. If improvement follows digitalis therapy, then probably myocardial insufficiency has been playing an important role in the case under question; if improvement does not follow rest and digitalization, then the mechanical factors may be considered paramount but still an underlying myocardial weakness cannot be excluded. The sole hope of relief in these cases and of cure in others rests in surgical therapy directed against the release of the heart from its constricting bonds, the adhesions or the scarred pericardium.

2. Surgical treatment may be divided into 2 important surgical procedures:

a. Operations freeing the heart from its adhesions to the chest wall by resection of the ribs and cartilaginous portions of the sternum (Brauer type of operation).

b. Operations in which division of the adhesions to the surrounding structures and resection of the pericardium are done in order to liberate the heart from the constrictive effect of unyielding fibrous scar tissue.

Both of these operative procedures should be called cardiolysis but usage has confined this term to the former. Pericardiectomy is the term usually applied to the latter.

The chest wall operation was first described by Brauer in 1902. It consists chiefly in the removal of several ribs and costal cartilages on the left side over the precordium for a distance of 10 to 15 cm. from the sternum. This releases the heart from the rigid unyielding adhesions to the anterior chest wall and relieves it of the extra work which it has to do during the systolic contractions.

Results of Brauer Operation

54 definite cases collected by Trout:

38 cases	70%	Excellent
2 "	3.7%	Successful
9 "	16.6%	Improvement
2 "	3.7%	No improvement
3 "	5.5%	Death

107 cases collected by Liggett and Smith:

20.6% mortality in 3 months; improvement noted in 84%; 29% of patients became self-supporting; valvular disease was present in 29% of these cases.

It is apparent that the Brauer operation while it gives relief in a high percentage of cases falls short in effecting complete or almost complete circulatory restoration. Recurrences of the symptoms of adhesive pericarditis may occur subsequently if the disease process has caused sufficient scar tissue formation in the pericardium or myocardium. Consequently, it is obvious that although the Brauer operation releases the heart from external adhesions, it does not free the heart of the intrapericardial part of the constriction and this is, therefore, its main disadvantage.

The other operation is that advocated by Delorme in 1898, called Delorme's decortication of the heart. This consists of precordial rib removal with resection of the left side of the sternum, whereby the pericardium and heart are exposed, followed by the actual cutting away of some of the thick constricting pericardium from the heart

surface, with resection also of the constricting bands about the great vessels, especially such as may involve the inferior vena cava under the sternum. This procedure is of course extensive and the surgeon must be expert and experienced. The younger and fitter the patient, the less risk and the more likely is marked improvement apt to follow.

Results of Delorme Operation

a. 37 cases collected by Churchill, supplemented with 7 cases by Trout:

8 operative deaths 18%
9 cases or . . . 20.4% were not completed because of accidents during operation.
6 cases or . . . 13.6% died in less than 1 month.
15 cases or . . . 34% showed relief for over 1 year, longest 11 years.
1 case (complete operation) - showed no relief.

b. 13 cases reported by Paul White:

10 cases operated upon, 5 were complete recovery.

c. 2 cases, recently reported, 1 by R. A. Griswold and 1 by T. E. H. Robert and A. T. Wilson in which pericardiectomy was done - both returned to active life.

Which procedure is to be done should depend on the extent and type of adhesions and whether the pericardium is sufficiently involved to cause constriction of the heart. Relief has been obtained by each procedure. The symptoms and signs of cardiac insufficiency and of hepatic congestion have disappeared and the patients have become symptom-free for years. Unfortunately, the underlying infection which was the etiologic factor in the development of the pericarditis, originally whether tuberculosis or rheumatic heart disease, may become so far advanced as to cause death quite unrelated to the problem of pericarditis. Consequently, even after an immediate "cure" is obtained by surgical means, general medical therapy must be resorted to and consistently directed against the basic constitutional disease process if the cure is to be more than temporary.

Conclusions

Chronic adhesive pericarditis is always preceded by a previous acute pericarditis which may be due to a number of causes. The pathology seems to be the same regardless of the etiological agent. The extent of the pathology depends on the severity of the acute infection. Clinically, chronic adhesive pericarditis may be divided into two general groups: (1) The case of large heart which is relatively immobile associated with gradual onset of congestive heart failure; (2) the case of small immobile heart with increased venous pressure and ascites. The most reliable finding in either case seems to be the evidence obtained with the roentgenkymograph. Treatment is supportive, medical and supportive (surgical). The surgical treatment of choice seems to be the Delorme operation or a modification of this whenever the general condition of the patient warrants it. However, there are good results in Brauer's type of operation. Finally, it must be remembered that, as a rule, chronic adhesive pericarditis is a local manifestation of a general infection and any surgical treatment would be little more than temporary relief if adequate medical treatment of the general infection is not carried out.

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IV. CASE REPORT

PROBABLE ADHESIVE PERICARDITIS

43 years of age,
white male.
Hospital number 650540

7-17-36 - Seen in Out-patient Department and admitted on 7-23-36, and discharged 11-12-36;

Complaints

1. Chronic discharge from ear, several years duration.
2. Dyspnea and orthopnea, 2 months duration.
3. Slight enlargement and soreness in the lower abdomen, 2 months duration.
4. Weakness, anorexia and loss of weight (8 lbs.), in 2 months.
5. Relatives state that there has been some mental retardation - amnesia and dis-orientation at times.

Past History

Essentially negative except for questionable history of rheumatis, 2 years before. No history of pericarditis

Examination

Dyspnea and slight cyanosis; serous drainage from left ear; distention but no pulsation of neck veins and arm veins (noted somewhat later); right hydrothorax; heart slightly enlarged to left but this is questionable, tones faint, no murmurs, blood pressure 90/70, pulse 88 and regular, no apex beat felt; liver enlarged, palpable 4 cm. below costal margin, slight amount of ascites, spleen not palpable; edema of both lower extremities, especially of left, varicose veins of left leg.

Laboratory

Routine laboratory procedures have been constant during hospital stay. Urine - negative. Blood - hemoglobin 85%, white blood cells 6,500, 78% pmn's, 20 lymphocytes, 1 monocytes, 1 eosino-

phil. Icterus index - 7. Wassermann - negative. Mantoux - negative. No free hydrochloric acid in gastric contents after histamine. Stools - negative for occult blood. P.S.P. - 60% return of dye in 2 hrs. Sedimentation rate - 27 and 46 mm. in 1 and 2 hours. Serum protein ratio - 3.8 albumen, 1.9 globulin.

Cirrhosis of liver and gastro-intestinal malignancies considered.

X-rays

Chest - pleural effusion, right. Abdomen - enlarged liver, spleen not enlarged. Barium enema - negative colon. Gastro-intestinal study - probably negative. Stomach and duodenum - There is downward displacement of the hepatic flexure of the colon.

Paracentesis

Paracentesis done - 250 c.c. fluid specific gravity 1.018; 2,220 cells, mostly red blood cells; 475 white blood cells per cubic millimeter, mostly lymphocytes; 16 grams protein per liter. Neurology consultation: Neurological examination negative. Diagnosis of mental changes--functional; suggest lumbar puncture and skull rays. Lumbar puncture reveals 20 mm. of mercury pressure and 75.9 mgs. protein, otherwise normal spinal fluid. X-ray of skull - negative; negative sella tursica.

Ear consultation: Diagnosis of chronic serous otitis media made; treatment conservative. Liver function - apparently normal. Urine urobilinogen .5 and 1.6 mg. per day; after urobilin injection .75 mgs. per day. Brom-sulfalein shows 5% retention in blood; galactose tolerance .72 gm. excreted in 6 hours.

8-7-36 - It is pointed out that all symptoms and signs can be explained on the basis of adhesive pericarditis. Electrocardiogram shows fixation of the electrical axis. Low amplitude QRS, all leads; slurring and notching of QRS, all leads; negative T_2 and T_3; iso-electric T. Venous pressure, 26.5 cm. saline. Patient has developed more ascites and edema of extremities. X-ray and fluoroscopy - extensive adherent pericardium with marked pleural effusion, right side. Thoracentesis, 1250 cc. fluid removed; specific gravity 1.014, white blood cells 1,750, all lymphocytes. Injected into guinea pig. Reported negative on 9-29-36.

Treatment

Strict bed rest. Fluids restricted to 1,000 cc. daily. High protein salt free diet. Salyrgan intravenously. Thoracentesis and abdominal paracentesis. However, there is only slight effect from salyrgan. Repeated thoracentesis necessary for dyspnea and cyanosis. Weight varies only slightly, 125½ to 132½ lbs., depending on effect of salyrgan. Weight 8-13-36, 127½ lbs.; 10-13-36, 131½ lbs.

Surgical consultation on 8-27-36 - concurred in diagnosis but wanted maximum improvement possible before surgery.

From 10-13-36 to 10-20-36, patient digitalized; has been on maintenance dose since; no evident effect.

10-17-36 - Venous pressure determinations, 10.5 cm. saline; on deep inspiration, 14 cm. saline. Electrocardiogram essentially the same.

10-20-36 to 11-12-36 - Continued above conservative treatment. Weight at discharge, 131½ lbs.

11-12-36 - Discharged to rest home where conservative treatment will be continued for 6 weeks. Patient to return then for observation and consideration of surgery.

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V. GOSSIP

Because of a last minute change in plans for today's staff meeting, it was necessary to ask Internist R. E. Mattison to prepare his contribution ahead of schedule. Again he demonstrated his remarkable versatility by moving with lightning-like rapidity in grinding out his stint,

proving some old adage or other. We greatly appreciate his contribution and recommend it to you as one of our "different" meetings.....When the Mattisons purchased the family car frequently seen in front of the hospital, they were so pleased with its appearance and performance that they went on a trip. Hearing a loud noise coming down the road in Iowa, they pulled to one side to discover that a motorized circus was passing. In spite of their position far over on the parking strip, the lion cage skidded into them, smashing a front fender. After they had separated the cars, another car came along with more animals which smashed another fender. Waiting for the manager to appear, they discovered the circus had passed and that's that. .....She was from northern Minnesota and unaccustomed to the comforts of civilization. She was sent by Social Service to stay at one of our boarding homes near the hospital. The kindly landlady, noticing her weary expression, suggested a nice warm bath. She drew the tub, laid out the towels and soap, and invited our patient to enjoy herself. At 2 o'clock in the morning, the now distracted landlady called and asked for help to get our lady out of the tub. She had been laughing and splashing for over three hours and most of the other roomers were complaining of involuntary insomnia. She was not a neurological patient but just one of the great unwashed.....As next Thursday is Thanksgiving, there will be a holiday from this.....The Public Library Forum which runs for 24 weeks, each Wednesday night, is attracting a great deal of attention because of the advance notices. The majority of the speakers are from our faculty group and many of them would not recognize themselves from the titles and descriptions of their subjects. Most startling is Mally Nydahl's Sudden Death (Demonstrated). Other subjects: The Meaning of Disease, Inside Facts About Food, Science and Health, Secrets of the Heart (illustrated), Poison Hazards, The Skin Tells the Story (illustrated), One Jump Ahead of Disease, The Behavior of the Blood (illustrated), Invisible Man-Killers, Mystery of the Glands, The Art of Relaxation, Six Feet Up and Down (demonstrated), The Truth About Cancer (illustrated), The Breath of Life (illustrated), The Criminality of Contagion (illustrated), Borderline Mental Cases, Why Teeth Leave Home, Be Clean and Live!, Trouble on the Brain (illustrated), Syphilis and Gonorrhea (illustrated), Facing the Hazards of Surgery (illustrated), The Senses We Can't Keep (illustrated), Must We Die? (illustrated).....Try matching these names with the titles: Barron, Bell, Evans, Ahrens, McKinley, Scammon, Michelson, N. Johnson, White, Seham, Wright, Lyon, Newhart, S. Johnson, Visscher, O'Brien, Cottam, Ellis, Irvine, Myers, Larson, Harrington..... ....The physicians of Waterloo, Iowa purchased a country club for their own use from the County Medical Society earnings from the poor fund. Forty doctors and their wives enjoy this exclusive privilege in a most attractive setting.....Chief Pediatrician Irvine McQuarrie is busy in the East, examining applicants for registration in pediatrics and telling of his research. .....The University of Minnesota Hospitals will participate in the Fiftieth Anniversary Celebration of the Medical and Dental Schools next year. This building is not that old but prior to its construction, the University maintained a hospital in one of the campus houses. The old Dispensary Building, still owned by the University, is now a hotel at seven corners, Minneapolis, and can be seen just before the turn of Washington Avenue to go downtown... ...The opening ceremonies of the Center for Continuation Study (not the Adult Education Building) were quite an elaborate ceremony of the last weekend. Committees from all the schools of the University participated in a planning contest. I am sure that Director Benjamin, when he received their reports, must have felt very much like the President of Harvard who urged the super-specialized scientists to lay out a comprehensive plan for society based on their investigations.....Medicine is planning a four week's course in January and February in surgery, pediatrics, obstetrics and gynecology and medicine. A limited enrolment of 20 will be accepted for each week. It is planned to have the students live on the campus in the Center while they are taking their work.....Adios.

NO MEETING NEXT WEEK