

ADHERENT PERICARDIUM AND PICK'S SYNDROME.
AN AUTOPSY STUDY.

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In 1896 Friedel Pick reported three cases of mediastino-pericarditic pseudo-cirrhosis of the liver studied at autopsy, one of whom was correctly diagnosed before death. This syndrome of constricting pericarditis and recurrent ascites has been since then associated with his name, although he was by no means the first in its description. There has appeared in the thirty-five years since Pick's report an extensive literature on the subject in which the two chief problems discussed have been the etiology of the condition and the relationship between pericardial disease and the not uncommonly associated polyserositis and pseudo-cirrhosis.

Therapy of the disease has languished for part of this time and with it the diagnosis—a notoriously difficult problem. But with the recent experimental work in the production of pericardial disease and its relief by surgical resection of the pericardial scar there has been a renewal of interest in it. European surgeons, until the last few years, have been the leaders in the actual operative technique through the work of Brauer, Delorme, Rehn, von Schmieden, Sauerbruch, and others. Churchill in 1929 collected reports from the literature of 37 operations for decortication of the heart (Delorme) for pericardial adhesion. In this paper he noted a very successful operative result which he obtained in the case of a patient followed by us for several years at the Massachusetts General Hospital. Sprague and White in 1931 reported five cases, including this one, recently operated on with satisfactory relief of symptoms in three and a mortality of 40%. Among patients with cardiac disease no cure is so nearly complete as that following a successful operation for constricting pericarditis unless it be the relief afforded by surgery in some cases of thyrocardiac disease. Our surgical results from the operations of Dr. Edward Churchill and Dr. Wyman Whittemore

have led us to survey the autopsy material at the Massachusetts General Hospital since 1896 (including one case of 1893) in relation to the incidence of adhesive pericarditis and Pick's disease. It so happens that this period almost coincides with the history of pericardial surgery in this hospital, since it was on November 24, 1895, that Dr. C. B. Porter performed the first resection of the pericardium for sepsis.

Our special study included, first, the material from 1900 autopsies performed between January, 1921, and April, 1931. In 1913 William H. Smith reported on a similar study of 3053 necropsies from 1897 to 1913 and in 1926 Cabot reviewed the necropsy experience from 1896 to 1919. To complete the entire thirty-five years our series was extended to include the year 1920.

One purpose of this review was to discover the frequency of the Pick syndrome, and to find, if possible, cases for which surgery might have held our some hope of relief had the diagnosis been made. The immediate problem, however, was to discover what justification seemed obvious for recommending pericardial resection following the subsidence of acute rheumatic pericarditis to prevent the later development of crippling pericardial synechia. An analysis of the ten-year series from 1921 to 1931 is presented.

INCIDENCE OF PERICARDIAL ADHESIONS AT THE MASSACHUSETTS GENERAL HOSPITAL.

In the 1900 autopsies studied 43 cases of pericardial adhesion were found—twenty-five showed complete obliteration of the pericardial sac and eighteen showed partial obliteration or bands of adhesion between visceral and parietal layers. Two cases showed extensive parietal adhesions combined with complete obliteration of the pericardial cavity. Pleuro-pericardial adhesions were present in eleven cases, of which eight had complete, and three partial, obliteration of the pericardium. The incidence of chronic pericarditis was 2.26% of all autopsies. This is similar to Cabot's finding of 114 cases in 4000 autopsies at the Massachusetts General Hospital from 1896 to 1919 (2.8%). We are not including cases showing nothing but a few old adhesions between pleura and pericardium. In 100 serially autopsied cases we found that 14% had these fibrous bands.

In spite of the number of cases of chronic pericarditis autopsied

in ten years it was of interest to us to discover that no case of Pick's disease was found among them. This at once impressed us with the rarity of the condition, at least at the autopsy table in a hospital not primarily treating chronic disease. A comparison with the discharge diagnoses of the hospital cases over a period from 1923 through 1930 showed, however, that the diagnosis of chronic mediastino-pericarditis had been made even clinically only five times (exclusive of the five cases reported by Sprague and White in 1931). Further analysis of this 1923 to 1930 clinical group showed no case proved at autopsy, but did reveal two living in which the diagnosis appeared correct. Two of the other three were probably incorrectly diagnosed and the fifth case was proved to have been wrongly diagnosed because in spite of recurrent ascites the pericardium, great veins, and liver were normal at necropsy. No case having a discharge diagnosis of "polyserositis" has been in the Massachusetts General Hospital since 1927.

ETIOLOGY. RHEUMATISM.

Eighty per cent of the 43 autopsied cases of adherent pericardium had associated heart disease. The most common of these was rheumatic heart disease. Twenty cases of this type occurred in the series and two others with complete obliteration of the pericardial cavity had a clear history of rheumatic infection, but no valvular deformity. In the rheumatic group, four had had influenza and two had had pneumonia.

In the presence of rheumatic valvular disease and the inevitable myocardial involvement of these cases the pericardial adhesion appears to play a secondary part in the picture of congestive failure and in no case could it be held to be a true constricting pericarditis. Of these 22 patients 13 died from cardiac causes, two from subacute bacterial endocarditis and in seven the heart was not primarily responsible for the death. Interest is attracted to the obliterative pericarditis of the two cases without valvulitis. One died at the age of 56 with pernicious anemia, and one at 21 with chronic nephritis and uremia. Adherent pericardium was not suspected in either and the pericarditis was "vestigial" in the term of Cabot.

OTHER ASSOCIATED CARDIAC DISEASE.

Thirteen patients are included in the remaining group with cardiac disease. Nine had significant hypertension, in four combined with coronary disease and in one with coronary disease and lues. One other case had lues combined with coronary disease. Coronary sclerosis was diagnosed in nine cases.

The cause of the adhesions in this group was undeterminable from the history or the pathological examination. Pneumonia appeared in the past history of three, influenza in two, and chronic nephritis in one. One had terminal miliary tuberculosis.

ASSOCIATED NON-CARDIAC DISEASE.

A group of eight patients was found with various causes of death, only one related to the heart. Seven of these were over 65 years of age and the eighth died of broncho-pneumonia at 41. One looks in vain in the histories of aged patients to find adequate cause for chronic pericardial disease which has never caused symptoms. The pericardium was completely adherent in five cases and calcified in one. The causes of death and the ages were chronic asthma and auricular fibrillation (76), terminal erysipelas from a varicose ulcer (70), nephrectomy for hypernephroma (65), lobar pneumonia (84), Addison's disease (66), broncho-pneumonia (41), amputation for gangrene and infection (77), and operation for ovarian tumor (68).

In this group a history of previous pneumonia occurred only in the patient who died of broncho-pneumonia. The only other probable etiologic factor unearthed in this group was puerperal sepsis which occurred thirty years before death in the woman dying after nephrectomy.

TUBERCULOSIS.

It is evident from our figures that tuberculosis can rarely, if ever, be proved responsible for the benign pericardial adhesions found in this series. One patient with rheumatic heart disease had chronic pulmonary tuberculosis and terminal miliary tuberculosis; one other rheumatic patient had terminal miliary tuberculosis and one man of 84 with hypertension and coronary disease had terminal miliary tuberculosis. There was nothing in any of the histories as recorded which revealed chronic or arrested tuberculosis of the lungs. Eight

cases showed microscopic evidence of obsolete tuberculosis in glands or lungs. Twenty-three had chronic pleuritis.

SEX.

Of the entire group 27 were males and 16 females. In the rheumatic group 14 were males and 8 females; of the non-rheumatic cases 13 were males and 8 females. This incidence of 63% of males is not quite so impressive as the 72% males in Cabot's series. However, in the group of eight cases over the age of 70 six were males (75%).

AGE.

The ages were from 13 to 84 years, the average of the entire group being 46.6 years. The average age at death of those with rheumatic valvular disease was 27 years and of the others was 62 years. Twenty-three patients, or over half of the series, were over 50 years of age. The average age of death in the rheumatic group is changed to 23.6 years if we include only those dying from cardiac cause. It appears that the lowered age at death in this group merely indicates a more severe cardiac infection rather than showing an important burden upon the heart from the pericardial adhesion.

CAUSE OF DEATH.

Reference has been made to some specific causes of death. In the entire series 20 died from cardiac causes. This shows that over 50% died from a cause not related to the heart and suggests that the adherent pericardium was of no importance. In no case could the pericarditis be called the primary cause of death.

HEART WEIGHT.

Twenty-six of the hearts of the entire series weighed over 500 grams; the largest was 917 grams in a case with rheumatic mitral and aortic disease. In the nine cases without a definite association with heart disease the average heart weight was 431 grams. Weights of the heart and pericardium in cases without associated heart disease, but with complete obliteration of the cavity were 330, 470, 498, 500 and 660 grams.

DIAGNOSIS.

The notorious difficulty in the diagnosis of adherent pericardium is apparent from our figures. In six cases a diagnosis was made

before death and in six others it was suggested. The X-ray study of these cases was incomplete as is usually so in patients who are sick enough to die in the hospital. Four fluoroscopic examinations and 14 roentgenograms were made. The diagnosis was made once on fluoroscopy from fixation of heart and diaphragm.

As no case of Pick's syndrome was found in this series it is only natural that the diagnosis of adherent pericardium was not made more often clinically; the pericardial adhesions in these cases caused no signs or symptoms or were obscured by other disease, most frequently rheumatic valvular disease with congestive failure.

PICK'S SYNDROME AT THE MASSACHUSETTS GENERAL HOSPITAL.

The failure to find cases of mediastino-pericarditis in the autopsies of the past ten years led us to investigate the experience prior to our study. Smith in 1913 reported a study of 62 cases of chronic adherent pericarditis in 3053 necropsies (2.03%) at the Massachusetts General Hospital from 1897 to 1913. Three patients had an associated perihepatitis and perisplenitis. In one, a boy of seven years, the abdomen required frequent tapping. In the other two, women of 35 and 65 years, no ascites was present. In one patient ascites was found post mortem. Cabot's series of 4000 autopsies from 1896 to 1919 revealed only one case recognized as true Pick's disease, but he refers to one other which was autopsied at this hospital in 1893. It so happened, that in the short period between the end of Cabot's study and the beginning of ours one more case came to necropsy.

From 1893 to 1932, a span of 40 years, only three cases of Pick's disease have been proved at autopsy at the Massachusetts General Hospital and one of these died three years before Pick's description of the syndrome.* A short review of these cases follows: (1) a boy 18 years old died at the Massachusetts General Hospital, in March, 1893. He had no history of rheumatism or tuberculosis. For six years he had had enlargement of the abdomen and ascites for four years. The cause of death was pulmonary edema and exhaustion.

*Since this study was completed, one of the five cases of mediastino-pericarditis operated on and described in the series of Sprague and White has come to autopsy. This case had completely adherent constrictive calcified pericardium. Pathological examination gave evidence which was very suggestive of tuberculosis, as did the examination of the tissue removed at operation.

At autopsy a completely adherent pericardium was found. The heart was not enlarged and showed no valvular disease. There was no pulmonary tuberculosis, but a cheesy nodule was found in the pericardium. (2) A woman, 23 years old, died in March, 1912, with congestive heart failure. She had had painless ascites for eight years, following mumps and pertussis. Seven years before she had been operated on at the Boston City Hospital and the peritoneum was found studded with tubercles. At autopsy a normal sized heart with completely adherent pericardium was revealed. There was an infarct of the left lung, but no pulmonary or peritoneal tuberculosis was found. Cheesy putty-like material was found in the pericardium, from which it was not possible to culture tubercle bacilli. (3) The last case was a man of 29 years of age dying in June, 1920. He was said to have had pneumonia with pleurisy three years before. However, fever persisted for a year and tuberculosis seemed likely. For five weeks dyspnea and ascites had been present, although he had had edema of the ankles at intervals for three years. A diagnosis of adherent pericardium was made as the heart was fixed on fluoroscopic examination, and a Talma operation was performed. He died a few days later. Autopsy showed a normal heart and an almost completely adherent pericardium with adhesions to the pleura and diaphragm and constriction of the upper part of the inferior vena cava. There was also probable tuberculous pleuritis and perihepatitis.

These three cases of Pick's syndrome are clearly related to constrictive pericarditis or mediastino-pericarditis. The pathology is consistent with tuberculosis.

In a recent review by Sprague and White of five cases of constricting pericarditis operated on at the Massachusetts General Hospital for relief of adhesions, it was shown that three cases were probably tuberculous in origin, one was found to have a probable subacute rheumatic pancarditis and one had no clear origin, although there was a family history of tuberculosis.

A critical survey of the cases discharged from the wards of the Massachusetts General Hospital from 1896 to 1931 showed in addition to the cases mentioned from 1923 to 1930, that only one other case without a rheumatic history or evidence of rheumatic heart disease could be considered suggestive of Pick's disease. This occurred

in a boy of 15 years of age who had a history of only four months of ascites apparently related to a "typhoid-pneumonia" and empyema with drainage a year before. This was not confirmed, as autopsy was not secured.

One other group should be noted and that consists of four cases autopsied prior to the series which we are reporting who had no chronic ascites and no rheumatic history nor valvulitis, but showed marked pericardial synechiae, and in three an additional mediastinitis.

In one, a man of 40, a pleurisy with effusion 18 years before suggests that tuberculosis was the etiology. In another, a man of 33, a history of chronic otitis media and erythema nodosum also indicates a possible tuberculous origin. The two others were more obscure—one occurred in a young woman dying of uremic coma a few months after delivery. She gave a story of weakness, pain in the chest and vague joint complaints. Pleural and pericardial cavities were obliterated and the sternum was bound down by adhesions. Chronic tuberculosis of a bronchial gland was found. The final case was that of a man of 49 who was well until one month before entrance to the hospital when a "boil" on the buttock was incised. Fever, aching joints and purpura followed and at autopsy the pericardium was almost obliterated, but contained a little sero-fibrinous exudate. There were adhesions to pleura and diaphragm and obsolete tuberculosis of a bronchial lymph node.

The respective weights of heart and pericardium in these four cases were 425, 403, 377 and 500 grams.

DISCUSSION.

RESULTS OF OPERATION.

Within the limitations of an autopsy study from a hospital treating mainly patients with acute disease it would seem justified to conclude that in over 35 years at the Massachusetts General Hospital only three cases of chronic constrictive mediastino-pericarditis have come to necropsy that might have been helped by surgical resection of the pericardium and those are the three with chronic ascites.

During the period in which these patients were in the hospital, 1893 to 1920, there was at intervals an attempt to relieve from

chronic ascites cases diagnosed adherent pericardium by means of surgery, but the only direct operation performed at the Massachusetts General Hospital was the Brauer cardiolysis or removal of ribs and cartilages overlying the heart to free adhesions to the chest wall. From 1908 to 1914 five such operations were done and the results seemed to justify a pessimistic evaluation of the method, but a survey of them shows that the fault may have lain largely in the indications for operation as dependent upon diagnosis. At operation in two cases no abnormality of the pericardium was discovered, and in two others there were no extrapericardial adhesions, but the pericardium was not described. In the other case, the only one in which the pericardium was opened, an extrapericardial adhesion was found posteriorly at the apex and the two layers of the pericardium were everywhere adherent. No attempt was made to divide the adhesions.

Three patients survived the operation, but were not relieved of symptoms.

In the same period two patients with chronic ascites and thought to have constricting pericarditis, but no valvular disease had Talma operations performed. One died (the case proved in 1920 at autopsy to have Pick's disease), and one apparently was somewhat helped and lived for four years. A third case was first thought to have tuberculous peritonitis, but the peritoneum was normal at laparotomy. There was improvement, however, for three years. The patient then returned and the abdomen was tapped with relief lasting while he was in the hospital. A fourth case, a child of eight years, had a sapheno-peritoneal anastomosis for ascites of one month in duration without improvement.

The Brauer operation has not been reinstated at the Massachusetts General Hospital, although reports in the literature suggest that it has a place in decompression of enlarged hearts with or without thoracic adhesions. The recent work of Churchill has created more interest and hope in the results of pericardial resection as suggested by Delorme and reported first by Rehn. In this operation the decortication of the heart is performed whether it is constricted by visceral or parietal pericardial scar. These results have been summarized by Sprague and White.

ETIOLOGY.

This hospital is situated in a rheumatic part of the world and it should be possible to form some conclusion about the probable responsibility of rheumatism in Pick's disease. Our figures give little support to the belief that in the absence of valvular disease rheumatism can be convicted. We have not observed recurrent ascites clinically in which rheumatic mediastino-pericarditis was a likely cause with the heart small and free from gross valvular defect. Cases have been reported in childhood in which recurrent ascites with constrictive pericarditis due to rheumatic infection has been present without valvular disease as in the two cases of Osler (1896), but the hearts in these cases were enormous and death obviously occurred in one of these during a subacute rheumatism with subcutaneous nodules, although the child had been in bed in the hospital almost continuously for three and one-half years and had been tapped one hundred and twenty-one times.

Recent evidence of the gravity of pericarditis in rheumatism has been presented by Holt, who studied fifty-one cases, thirty-nine of whom had a definite history of pericarditis in the past. Twenty-one had died and there had been five autopsies. In all but two cases (of the entire series) who had been "lost" so far as follow-up study was concerned, a clinical diagnosis of valvular disease was made. Valvular disease was present in all of the autopsied cases. In twenty-six cases acute pericarditis was the turning point in the disease and only one of this group was known to be alive three years after the attack. Twelve of the patients who died had ascites and enlarged liver, but only two required tapping. In only one case did it seem out of proportion to other signs of heart failure. She points out that ascites in rheumatic children is a very bad sign as distinguished from the ascites of Pick's disease; such children have large hearts and valvular disease.

Our autopsy experience confirms this view. So far as we can discover, from 1896 to 1931 at the Massachusetts General Hospital only six cases of adherent pericardium and a history of rheumatic infection have failed at autopsy to reveal also rheumatic valvular disease and in none of these was Pick's syndrome present. One other case not studied at autopsy had one year before entrance to the hos-

pital, at the age of 33, an attack of polyarthritis followed by ascites. There was cardiac enlargement, but no demonstrable valvular disease.

These six cases deserve some note since they represent the only direct evidence we possess of the occurrence of rheumatic pericarditis with adhesions without valvular disease, except for a case with this combination recently autopsied at the House of the Good Samaritan in Boston.

1. A man (1896), 26 years old. He had three attacks of rheumatic fever, eight, six and four years before. Fluid was removed from the pleura at the time of the last attack. For five weeks he had had shooting pain in chest and abdomen with some joint pain. Heart sounds were very poor and a precordial rub was heard. At autopsy the heart was enormous and weighed 1328 grams. Slight verrucose vegetations were found on otherwise undeformed valves. The pericardium was completely adherent and attached to the diaphragm and pleurae.

2. Boy (1900), 15 years old. Rheumatism began five months before and progressed to congestive failure, ascites and death. The pericardium was completely adherent by a loose vascularized reticulum. The valves were normal.

3. Girl (1902), 11 years old. (Case mentioned in series of W. H. Smith). She had had rheumatism four years before, and five months before admission had swelling and pain of all her joints followed by congestive failure, which increased. The heart was enlarged and there was a loud blowing apical systolic murmur, but at autopsy the valves were not deformed or thickened. The pericardial cavity was obliterated by old adhesions.

4. Woman (1905), 55 years old. She had had rheumatic fever at 15 years of age. For ten years there had been variable edema of the legs and abdominal swelling for six months. Auricular fibrillation was probably present at the time of death. At necropsy a band of adhesion was attached to the left ventricle and the pericardium and pleurae were adherent. Arteriosclerosis of the aorta, fatty infiltration of the myocardium of the right ventricle and thrombosis of the left innominate vein were present. The valves were undamaged.

5 and 6. These cases were reported in our series above—two

men aged 21 and 56—with a story of rheumatism, adherent pericardium and no valvular disease.

These six cases show that silent pericardial adhesions of rheumatic origin occur in rare instances in adult life and apparently play no part in cardiac failure. In younger people (Cases 1, 2 and 3) the pericardial involvement is usually part of an active or subacute rheumatism and attended with cardiac enlargement from rheumatic myocardial disease.

We have observed a few children with acute rheumatic pericarditis who completely recovered and show no definite evidence of valvulitis. This group will be important to follow up in relation to delayed signs of pericardial adhesions.

An uncompleted follow-up study by Dr. T. D. Jones and Dr. E. F. Bland of one thousand cases of rheumatic infection seen in the past ten years at the House of the Good Samaritan in Boston has furnished us with a series of 62 cases of definite acute rheumatic pericarditis. Thirty-seven are dead and in 28 the pericarditis was a part of a severe terminal infection. There were six autopsies. Of those who survived but died later, the average length of life was 3.3 years. Twenty-five are living—ten in good condition after a period of 6.5 years; five are in fair condition; eight in poor condition; and two have not been seen since leaving the hospital. In all but two living cases valvular disease is present and the heart is enlarged. One of these is a boy of 17 years of age who had severe rheumatic fever with congestive failure and pericarditis 10 years ago and now has no murmurs diagnostic of valvular disease and an apparently normal sized heart. The other is a boy who had two attacks of rheumatic fever and acute pericarditis nine years ago. No cardiac disease is now demonstrable. One autopsied case showed pericardial adhesion, but no valvular disease. In none of the 62 cases has Pick's syndrome developed in spite of practically certain pericardial adhesions.

So far as chronic symptomless pericardial adhesion of old age is concerned it need not concern us from the standpoint of therapy and its etiology is unsolved. It stands as a landmark of either defeated infection of unknown and probably variable type or as evidence of healing over minor myocardial infarctions. It apparently is no more significant than areas of chronic pleuritis.

The greater problem still remains, namely, the role of tuberculosis. The probability of this cause in the three proved cases of Pick's syndrome here reported suggests strongly to us that tuberculosis is the usual, if not the universal cause. It is usually secondary to tuberculosis elsewhere. Clarke recently could collect from his experience and the literature only thirteen cases of primary tuberculous pericarditis. The point about clinically proved tuberculous pericarditis, however, is that it is so virulent a manifestation of the disease that the patient usually dies. Of five proved cases at the Massachusetts General Hospital since 1921 four are dead and one still alive one year after pericardial resection. In other words, it is very difficult to trace the history of Pick's disease from an acute to a healed stage with the delayed appearance of signs of constrictive mediastino-pericarditis. Tuberculous pleurisy and peritonitis may go through this evolution of healing and so rarely may pericarditis. The crucial study involves watching it in one individual, as has been done following drainage of the pericardium for sepsis as in a case reported in a discussion by Danach.

As Broadbent pointed out in 1898 the key to the solution of adherent pericardium lies in watching cases of acute pericarditis as they go on to the formation of adhesions. Recent animal experimentation in this field has helped to complete the history of the Pick syndrome, as, for example, the demonstration by Beck of the production of mediastino-pericarditis followed by pleural effusion, edema and ascites, in the dog, by the injection of Dakin's solution into the pericardial sac. One other point of importance appeared in this work and that was the occurrence of fibrinous deposit between the lobes of the liver secondary to the pericardial inflammation without reference to any intra-abdominal manipulation, showing that perihepatitis may be secondary to pericardial disease and not an expression of so-called polyserositis.

Such a clear sequence of events, however, seems not to be the usual history in chronic mediastino-pericarditis. Its onset is insidious. Rheumatic involvement is acute and attended with enlargement of the heart, valvular disease and usually death coming within a very few years. Tuberculosis of the pericardium as clinically proved is usually fatal. Sepsis of the pericardium is very serious, pericardiot-

omy, for it has a high mortality and in our experience has never resulted in delayed Pick's disease. Unfortunately for the suggestion of Broadbent in the study of this syndrome the progression is rarely from the acute to the chronic pericardial disease, but the onset more commonly remains obscure.

SUMMARY AND CONCLUSIONS.

A study of 1900 autopsied cases at the Massachusetts General Hospital from 1921 to 1931 in relation to pericardial adhesion is here presented with special reference to Pick's disease (chronic constricting mediastino-pericarditis). This was undertaken in an analysis of the possible opportunities for surgical relief of the condition as related to various etiologic factors.

A survey of the autopsy material at this hospital from 1893 to 1931 as regards the incidence of Pick's disease reveals only three cases discovered at necropsy during this period.

In 35 years in 6100 autopsies only six cases of adherent pericardium with a rheumatic history came to autopsy in which valvular disease was not present. None of these cases had Pick's disease.

Tuberculosis of an insidious type is the most probable cause of the constricting pericarditis of Pick.

Adhesive pericarditis in middle and old age, even when calcified, in the absence of valvular disease is an unimportant cause of symptoms and is very rarely a contributory cause of death.

It appears unjustifiable to consider pericardial resection following acute rheumatic pericarditis to prevent the later effects of pericardial symphysis, since valvular disease is practically always present and the prognosis following acute rheumatic pericarditis is usually bad, except for rare cases without proved valvular deformity in whom we have never seen Pick's syndrome develop.

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DISCUSSION.

DR. WILLIAM D. STROUD: Apparently Dr. Sprague has done a marvelous amount of work in going over these autopsies. I think we all agree that this type of pericarditis is rare, but like everything else in medicine the more you look for it, the more apt you are to find it.

I feel that in this discussion the most important part is first of all what to look for clinically, and secondly an estimate as to whether operation will help a patient when such a diagnosis is made.

It seems to me that the work being done in Cleveland by Dr. Beck and Dr. Cutler would suggest that those patients with large livers and cyanosis and with relatively little cardiac damage, etc., were the ones that would do the best, and they often appear clinically the sickest.

They showed us a case in Cleveland last December of a young boy whose liver at the time he was admitted to the hospital was at least the width of the hand below the right costal margin, who was intensely cyanotic, and this diagnosis was made. They operated, operating under negative pressure, which they believe is a most important part of the technique, keeping the normal negative pressure in relation to the heart during the entire operation, and removed these adhesions, which produce this symptom of enlarged liver and cyanosis, and which are usually around the inferior vena cava and the hepatic veins where they come through the diaphragm. They had to raise the heart up to relieve these adhesions, and immediately they were released they were found to be constricting the inferior vena cava, and the liver apparently began to decrease in size and the cyanosis disappeared at once. They brought this boy into the clinic and we examined him. He was selling newspapers on the street, and although a little breathless yet, he apparently was getting along perfectly all right. His liver was just palpable, and his cyanosis had entirely cleared up.

So I think the importance of this work of Dr. Sprague's is to bring to our mind that although these cases are rare, they do occur, and that when they occur and you feel that they are interfering with the return of the blood to the heart, involving the inferior vena cava and the hepatic veins, one should be reminded that there are a number of cases that have been operated on very successfully.

They did show us one case that was sent from Baltimore to Cleveland, in which the operation was unsuccessful in spite of having been done under negative pressure, because they went a little bit too far in slipping the pericardium off the auricle, caused a hole with a rupture, such as Dr. Gorham has been working on in dogs, and the patient died. It is an interesting fact that they had the pericardium there that had been stripped off, and the pathologists and all of them could not say what was the etiology of the pathological changes. They passed this around and discussed it, and just as Dr. Sprague has emphasized, it is difficult to know in the majority of these cases what the etiological factor is. (Applause.)