EXPERIMENTAL STUDIES AND CLINICAL EXPERIENCE IN CONSTRICTIVE PERICARDITIS: VENOUS PRESSURE AND LIVER FUNCTION

by

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INTRODUCTION

Chronic constrictive pericarditis was first described by RICHARD LOWER in 1669, and in 1896 PICK pointed out that pseudocirrhosis of the liver was a usual complication of this disease. As for surgical treatment of this disease, HALLOPEAN first performed pericardiectomy in 1910, acting on the suggestion DELORME made in 1898. Since

then many clinical reports concerning this disease have been published in Europe and America. Especially since 1949 experimental research into the pathology of this disease has been pursued with increasing vigour. And our knowledge of this disease has been greatly widened by the papers of PARSON, HOLMAN, ISAACS and HALLER. In Japan, too, MINAKAWA has published excellent papers.

Since 1952 we also have instituted experimental research into this disease, and KUMA, one of our associates, has recently published a report which chiefly analysed pressure curves in the atrium and ventricle of cases of this disease. In previous experimental research, however, investigation of hemodynamics has always occupied a prominent place, while no serious attention has been paid to ascites, hypoproteinemia, and hepatic dysfunction, which are characteristic of this disease.

Slight malleolar edema in contrast to the early marked collection of ascites in this disease still demands an explanation. Also unsettled is the cause of hypoproteinemia. And again is there really a correlation between manifestation of symptoms and the height of venous pressure, or the degree of hepatic dysfunction? What curative effects has pericardiectomy on hepatic dysfunction, and raised venous pressure?

The author carried out experimental investigations to solve these problems, the results of which are published here, together with information on four clinical cases which had operation at our hospital.

PART I EXPERIMENTAL CASES

METHODS

1) Production of Constrictive Pericarditis

About three hundred adult mongrel dogs were used as experimental animals. Under intravenous thiopental sodium (Ravonal) anesthesia and endotracheal positive pressure respiration the heart was exposed, and into the pericardial cavity was inserted a polyvinylformal sponge devised by Prof. NAGAISHI of the Kyoto University Tuberculosis Research Institute in order to produce constriction of the whole heart. As to production of segmental pericarditis, the pericardium was sutured to the right and left sides of the myocardium, and into the pocket thus formed the above-mentioned sponge was inserted. In some cases sponges coated with various chemical irritants were used.

2) Recordings of Venous and Right Heart Pressure

With no anesthesia, and while the dog was kept in a prone position, venous pressure recordings were done direct puncture of the lower leg, and in some cases, of the jugular vein. The zeropoint was set at the height of the right atrium. The venous pressure in the course of pericardiectomy was recorded by direct canulation of the femoral vein, the right heart pressure was determined through direct heart puncture and recorded by electromanometer.

3) Liver Function Tests

i) Bromsulphalein Test

The experimental animals were intravenously injected with 5mg. per kg of

0.5% solution of Bromsulphalein (Hepatosulphalein). Samples of blood were drawn 10 and 20 minutes after the injection, and their bromsulphalein concentrations were read by tintometry. As MILLS and others stated elsewhere, the excretion of this pigment is very rapid in dogs, and even in dogs with hepatic dysfunction the excretion is nearly always completed within 30 minutes after the injection. So the readings were done on samples drawn 10 and 20 minutes after the injection.

ii) Cobalt and Cadmium Reaction Tests

These tests were done according to the method of INOUE. As the left side reaction is commonly strong in dogs, the tubes to the left of No. 1 tube were marked with a minus sign; and this tube number was taken as the reaction value. In normal dogs the cabalt reaction showed values between R-3 and R-5, and the cadmium reaction between R-14 and R-18.

iii) Serum and Ascites Protein Values and Patterns

The total amount of protein was determined with a HIDACHI refractometer. Paper electrophoresis with GRASSMAN'S apparatus was applied to the determination of the protein quotient. The buffer used was a veronal solution (pH 8.6, M-0.1, 100V, 0.3mA/cm). B. P. B. was used as the staining material, and a densitometer was employed for quantitative determination. Clinical cases were investigated with a TISELIUS apparatus of the HIDACHI HT-A model.

iv) Other Liver Function Tests

Besides the above-mentioned tests, TAKATA'S and GROSS' reaction tests, the corresive sublimate reaction test and the zinc sulfates test were made. The results of these tests, however, have not been included in this paper, as the dogs showed too striking individual disparities in reaction, which rendered the evalution of results nearly impossible. Moreover, in paper electrophoresis with dogs the distinct separation of protein fractions was not achieved, and therefore, generally speaking, the serum colloid reaction yielded no reliable results.

4) Pathologic Examinations

In addition to macroscopical examination, various organs were histologically examined, using hematoxylin-eosin, WEIGERT'S and Van GIESON'S stains.

RESULTS

Chapter I Constriction of the Whole Heart

Cases of induced pericarditis did not always develop the same symptoms. This was of course due to differences in thickness and width of the sponges used as well as to individual differences in the resistance of dogs. But on the whole transient right heart failure, raised venous pressure, ascites and edema appeared about 10 days after production of experimental pericarditis. This period may be considered to be the acute inflammatory stage. In severe cases this acute stage was gradually aggravated by collection of ascites, and was followed by death in one or two months.

Chronic cases followed the course very similar to that of clinical cases, and survived, no better, now worse, for more than one year till they underwent pericardiectomy. In our cases of induced constriction of the whole heart symptoms

of right heart failure were chiefly noted, but such symptoms of left heart failure as coughing and crepitations rales were latent.

Section I Venous Pressure

Peripheral venous pressure took a sudden rise about one week after the production of experimental pericarditis, but then fell slightly and stayed at the level of about 100mm of water. In normal dogs the venous pressure of the lower leg was between 30 and 50mm of water.

1) Relations between Peripheral Venous Pressure, and Ascites and Edema

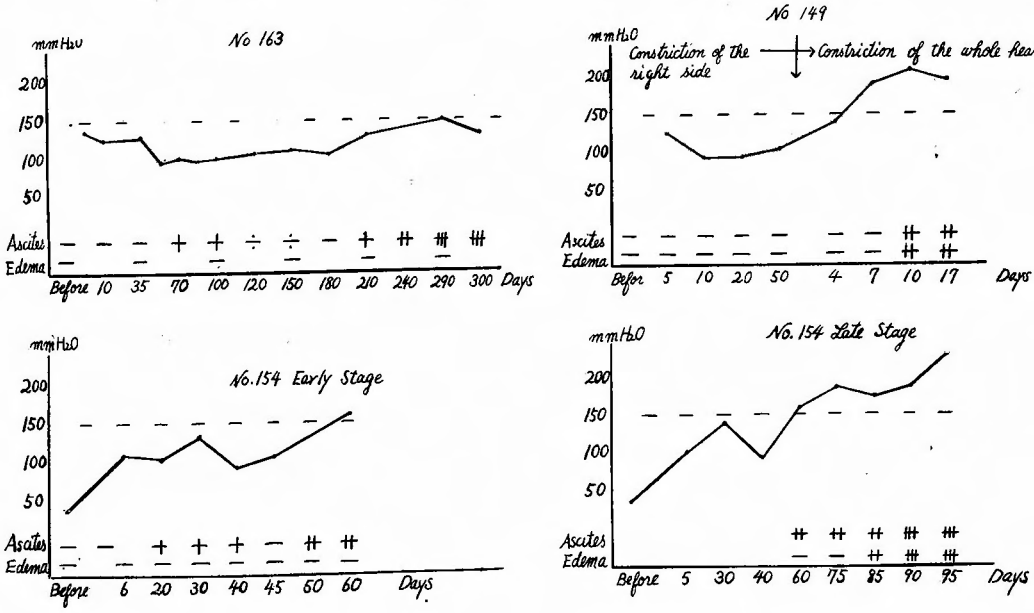
No exact parallel was established between venous pressure and the amount of ascitic fluid, but when ascites collected, venous pressure was kept above 100mm of water. When only collection of ascites was noted with no edema, the venous pressure was always two or three times normal, that is between 100 and 150mm of water, as was the case with No. 163 and the early stage of No. 154 (Figs. 1 and 2).

When the condition worsened, or in the cases which were serious from the bignning, the pressure reached values of 150～200mm of water or more; and edema appeared all over the body, especially in the abdominal wall and lower leg. These cases went from bad to worse, and soon died.

2) Effects of Tetraethylammonium-bromide on Venous Pressure

Increased venous tone is generally considered to be a factor in venous pressure rise. To pursue this problem further, TEAB (Japanese trade name: Tebron) was

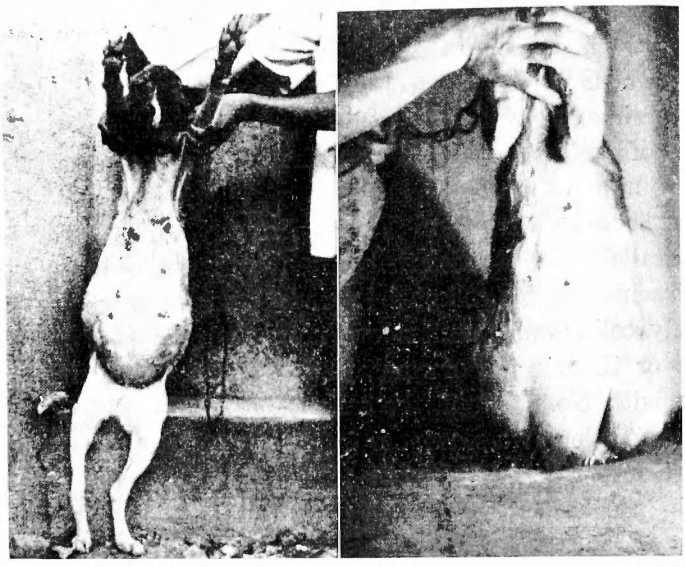
Fig. 1 Relations between peripheral Venous pressure, ascites and edema



The cases which only collection of ascites without edema.

The cases which ascites accompanied with generalized edema

Fig. 2 Constriction of the whole heart No.154



60 days postoperative.
Venous pressure 110～150mm H_2O.
Only collection of ascites was noted without edema.

98 days postoperative.
Venous pressure 220mm H_2O.
Ascites were accompanied with generalized edema.

intravenously injected 1mg per kg, into dogs with induced pericarditis. After this intravenous injection the venous pressure dropped immediately, reached its lowest level in about 5 minutes, but gradually regained its former value. The higher the venous pressure, the more marked was the effect of this drug. In normal dogs a temporary rise is noted immediately after injection, followed by a slight fall. In this case the restoration of the former value is extremely slow.

After the above experiment, normal dogs were intravenously injected with a large amount of isotonic saline solution. After this injection had raised the venous pressure, these dogs were again given an injection of TEAB. But as shown in Fig. 4, no fall occurred (Figs. 3 and 4).

3) Summary and Discussion

As mentioned in the introduction, slight malleolar edema in contrast to early collection of ascites is characteristic of this disease. To provide a sufficient explanation for this symptom, observations were made on venous pressure and clinical condition after the production of experimental pericarditis. And it was ascertained that if venous pressure remained lower than 100～150mm of water, ascites alone was present, but that if venous pressure rose to more 200mm of water, malleolar edema made its appearance. In view of the so-called backward failure theory derived from the hemodynamics of this disease, this increase in peripheral venous pressure may be largely attributable to the elevated central venous pressure. It is quite possible on account of anatomic connections for such a rise in pressure to have a powerful effect upon hepatic function. That is considering from the point of hydrostatic pressure

Fig. 3 The effects of TEAB on venous pressure in the dogs with constrictive pericarditis

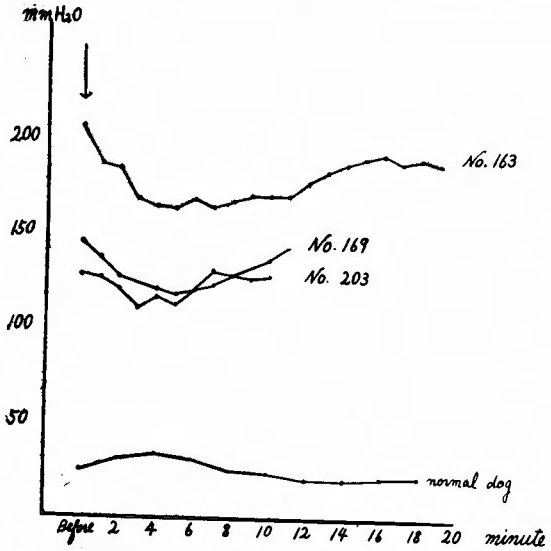
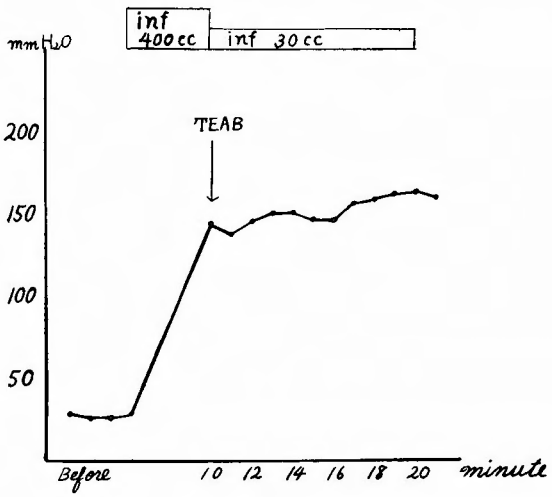


Fig. 4 The effect of TEAB on the mechanically elevated venous pressure on the dog.
The arrows represent the intravenous administration of 1mg/kg body weight of TEAB.



alone, the escape of lymph from the hepatic surfaces may be accelerated in dogs with the peripheral venous pressure between 100～150mm of water, while on the other hand elevation of the central venous pressure may cause a pressure rise in lymphatics also, and thus by increasing the difficulty of absorption of filtering fluid from the capillaries facilitate formation of ascites.

Moreover, as the liver is an intraabdominal organ, less outside pressure weighs on its capillaries than on the four extremities. Naturally this fact has something to do with the abundant outflow of lymph from the liver.

As for the mechanism of venous pressure rise in constrictive pericarditis, the backward congestion which is explainable by the backward failure theory seems to play the most important role in it.

Further (1) Increase of blood volume, (2) elevation of venous tone and (3) extraneous pressure on the vessels may be taken into consideration as extracardiac factors.

With regard to venous tone, HALMAGYI, BRAWN and KELLY noted that in patients with common heart failure sleep and the vasodilatory and the autonomic blocking drugs caused lowering of venous pressure. HALMAGYI reported that in dogs the venous pressure rise due to intravenous injection of a large amount of saline solution could not be decreased by TEAB.

The present author clearly observed a marked depression of venous pressure caused by the administration of TEAB in his dogs with pericarditis, and ascertained that venous tone was involved in venous pressure rise in constrictive pericarditis.

Clinical cases showed thickening and sclerosis of peripheral veins, but in the present experimental cases these changes were not noted perhaps because of shortness

of the survival period (Fig. 5). But such sclerosis and thickening of peripheral vessels may be considered as one of the factors influencing venous pressure rise in the late stage.

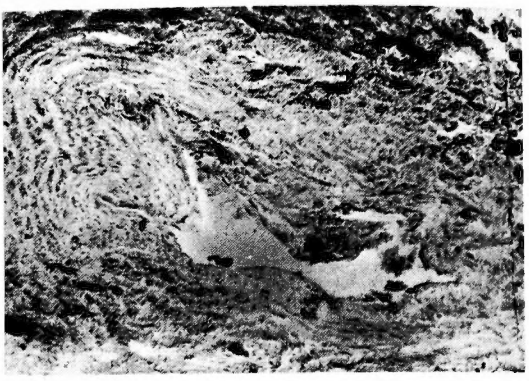


Fig. 5 Case 1. Postmortem section show thickening and sclerosis of cubital vein.

Section II Liver Function and Pathology

Pathologic changes in the liver due to experimental constrictive pericarditis are chiefly caused by congestion. In cases of constriction of the whole heart, or of the right side of the heart, the same pathologic changes, though different in degree, were observed in all cases which presented the symptoms of right heart failure.

1) Cobalt and Cadmium Reaction Tests

Cobalt and Cadmium reaction tests showed a strong left side reaction immediately

Fig. 6 Functions of the liver

Constriction of the whole heart

No. 134

Postoperative Days.	CoR	CdR	BSP
Before	8	10	2.5 %
3	8	26	17.5%
7	-12	18	10 %
10	died		

No. 163

Postoperative Months	CoR	CdR	BSP
Before	-5	12	2.5%
1M	-6	18	2.5%
3M	-4	16	5 %
5M	-4	18	
8M	-6	14	5~7.5%
10M	-6	16	5 %

Constriction of the left side

No. 24

Postoperative Months	CoR	CdR	BSP
4M	-3	24	2.5%
5M	-4	16	2.5%
7M	-5	16	2.5%
10M	-5	16	2.5%
12M	-5	12	2.5%

Constriction of the right heart

No. 112

Postoperative Days	CoR	CdR	BSP
Before	8	16	2.5%
4	- 8	20	7.5%
15	-10	20	7.5~10%
20	-10	14	10 %
25	-14	18	20 %
30	died		

No. 115

Postoperative Days	CoR	CdR	BSP
Before	- 4	14	2.5%
10	-10	17	5~2.5%
15	-10	20	7.5~5%
20	- 8	18	10 %
30	-10	20	10 %

Constriction of the left side

No. 21

Postoperative Months	CoR	CdR	BSP
1M	-4	18	2.5%
4M	-5	16	2.5%
5M	-4	16	2.5%

after the production of pericarditis, but after the acute stage was over, they returned to nearly normal in cases with no heart failure, while in cases with accumulated ascites they showed a weak left side reaction. But the results of these tests were not so significant as to provide a prognostic standard (Fig. 6).

2) Bromsulphalein Test and Pathologic Findings in the Liver

The BSP test yielded the most significant results of all the liver function tests (Fig. 7).

Fig. 7 Retention of Bromsulphalein at 10 minute

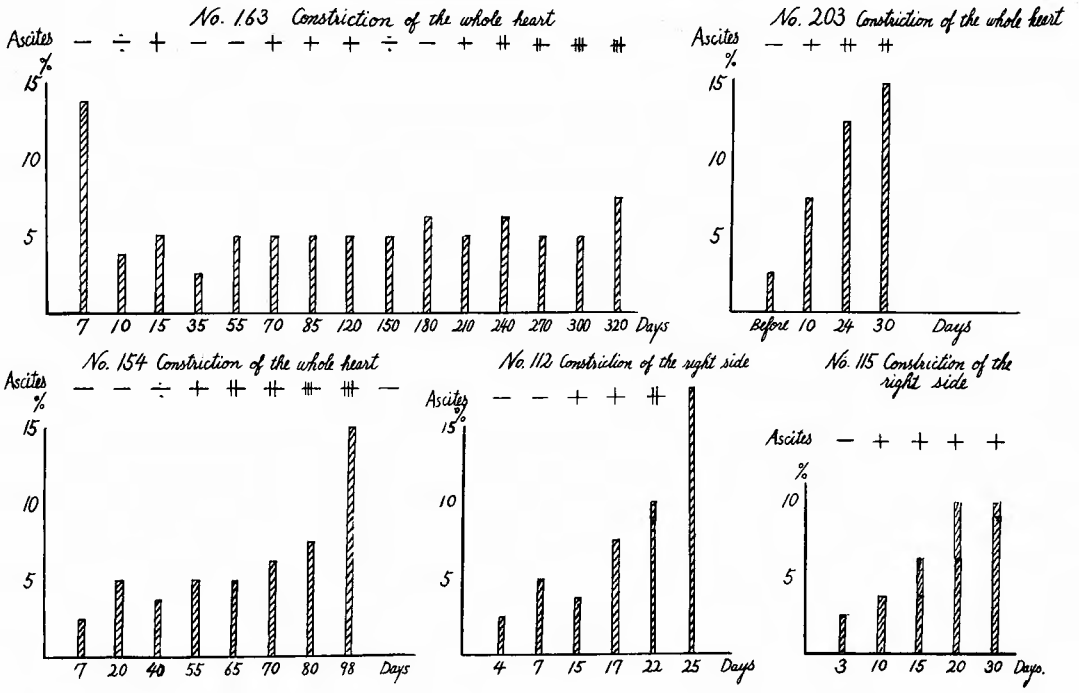
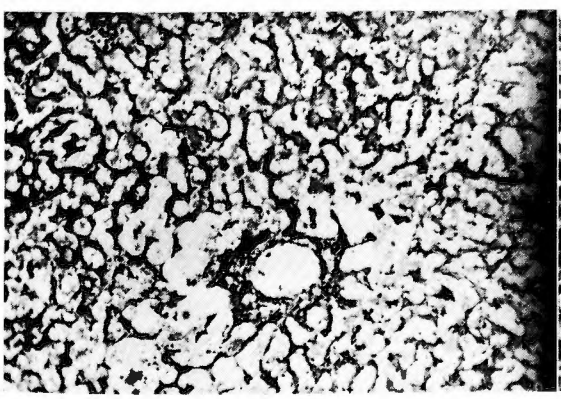
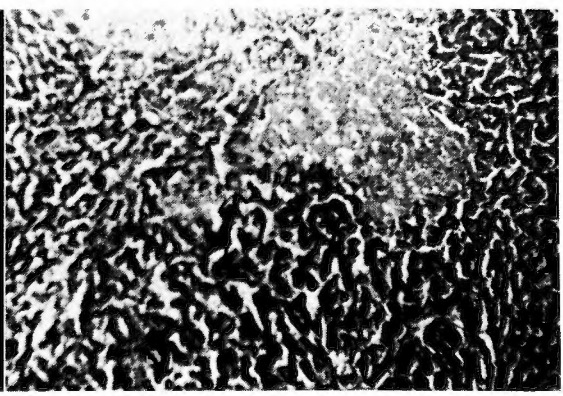


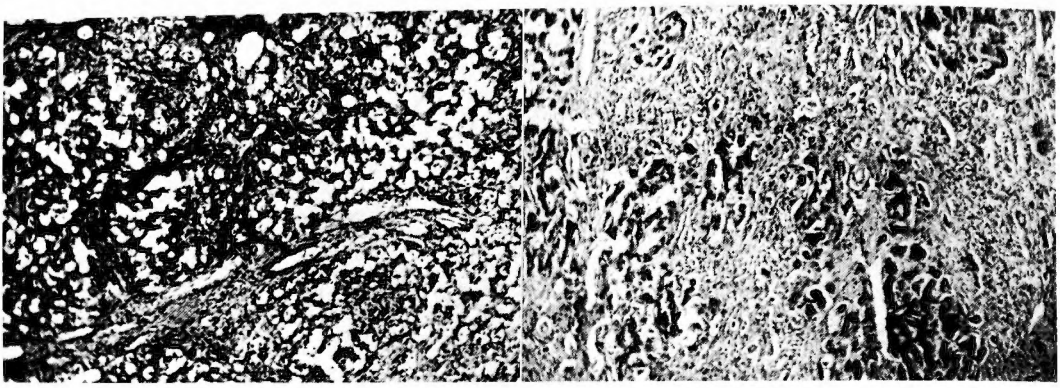
Fig. 8 No.154. 100 days after production of pericarditis. BSP retention 20% at 10 minutes.



Postmortem section show distention of sinusoid and centrilobular necrosis.



Postmortem section show intense congestion and hemorrhage around the V. centralis.



No.163. 300 days after production of pericarditis. BSP 5～7.5% retention. Atrophy of liver cells and great increase in connective tissue.

Case 1. K.H.
BSP 35% retention at 30 minutes. Postmortem section show fully developed cardiac cirrhosis.

In severe cases (No. 203, No. 112, No. 115 and the late stage of No. 154) which developed marked right heart failure immediately after production of pericarditis, the BSP test gave stepwise increasingly poor results, and all of these cases died nearly one month later. In these cases were noted pathologically distended sinusoids, intense congestion, centrolobular necrosis, hemorrhage around the central veins and severe atrophy of hepatic cells (Fig. 8). On the other hand, in such cases as No. 163 and No. 154 which followed a chronic course, the BSP test gave rather good results in spite of accumulated ascites.

In one case (No. 163) which survived more than one year, the congestion of the early stage was no longer observable, and though atrophied hepatic cells and distended sinusoids were present, no blood cells were detected. Instead connective tissue proliferated in GLISSON's capsule and around the central vein, presenting the picture of advanced fibrosis of the liver. But even in this late stage impairment of the excretory function was comparatively slight, as revealed by the BSP test.

3) Serum Protein

Generally speaking, the amount of serum protein decreased, and in the protein ratio a decrease of albumin and an increase of β- and γ-globulin were noted. Details of serum protein will be given later.

4) Summary and Discussion

According to INOUE, the cobalt and the cadmium reaction tests have a normal or weak right side reaction in common cases of heart failure. In the present experiment these tests were normal or, in cases with ascites, had weak left side reaction, but when the liver develop cirrhosis, these reactions shift to the left.

The BSP test chiefly indicates functional capability of the parenchymal cells of the liver. In chronic cases comparatively slight excretory dysfunction is accompanied by proliferation of connective tissue, and thickening of vessels, but at the same time regeneration of hepatic cells takes place with the gradual disappearance of the congestion of the early stage. The excretory dysfunction, as revealed by the BSP

test, is therefore comparatively slight.

Generally speaking, excretory dysfunction is not so serious when fibrosis is the only pathologic change in the liver. And so when serious excretory dysfunction is shown by the BSP test to be present in clinical cases which follow a chronic course, it must be considered that hepatic fibrosis has been aggravated into cirrhosis. Further, stepwise and rapidly increasing excretory dysfunction, as witnessed in short-lived cases and in the late stage of chronic cases, means that decompensation has set in, and also histologically that centrolobular necrosis, hemorrhage, and advanced atrophy of hepatic cells have occurred. Prognosis for such cases is rather hopeless.

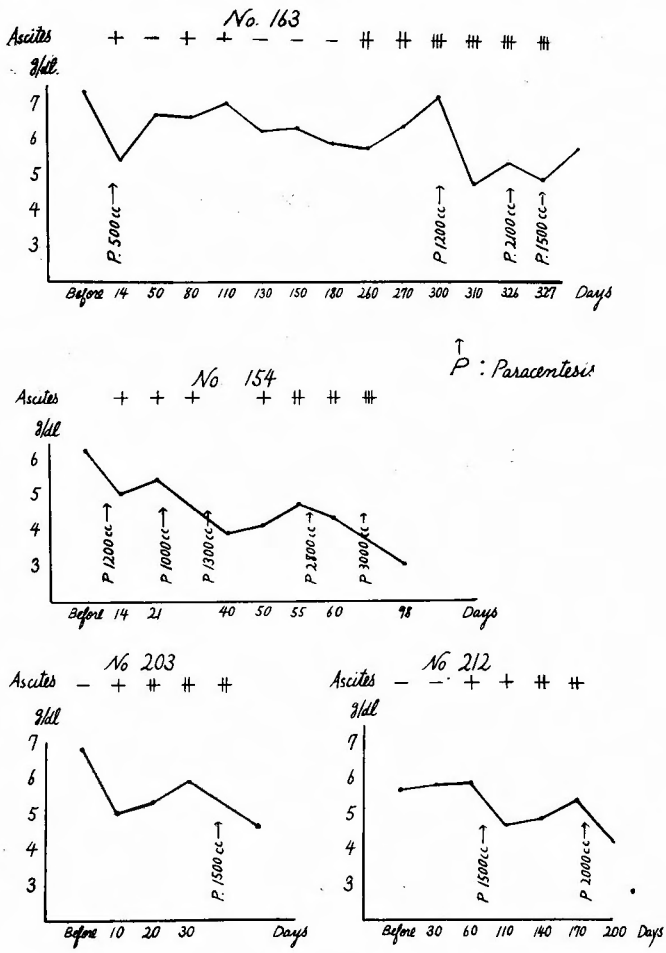
Section III Ascites in Constrictive Pericarditis

One of the preceding sections descrived the relations between venous pressure, ascites and edema, which are characteristic of this disease. In this section they will be cohsidered from the point of view of serum proteins and hepatic function.

1) Amount and Pattern of Serum Proteins

Production of pericarditis commonly causes a decrease in the amount of serum

Fig. 9 Relation between serum protein and collection of ascites.



protein, but the presence or degree of ascites has no appreciable effect on it. For example, No. 163 had a rather large amount of serum protein, when it had ascites, and in No. 212 the volume increased after the production of pericarditis. The protein pattern showed that the production of pericarditis causes a decrease of albumin, and an increase of β- and γ-globulin; in long-surviving cases γ-globulin tends gradually to increase (Figs. 9 and 10).

Fig. 10 Serum protein pattern in cases of ascites collection

No. 221

Postoperative Days	Al	α-gl	β-gl	γ-gl
Before	34 %	20.1 %	32.5%	13.4 %
10	23.6%	23.23%	36.4%	16.77%
20	24.3%	13.1 %	46 %	16.6 %

No. 203

Postoperative Days	Al	α-gl	β-gl	γ-gl
Before	40.3%	16.6%	22.8%	10.8%
7	30.4%	18.2%	36.2%	15.2%
20	20.8%	21.8%	28.4%	19.1%
30	20.2%	21.4%	40.0%	18.1%

No. 163

Postoperative Months	Al	α-gl	β-gl	γ-gl
2	20 %	15 %	43.0%	18.0 %
3	18.4%	17.7 %	49.5%	11.2 %
5	28.2%	13.47%	48.1%	16.23%
6	31.8%	15.2 %	25.4%	27.6 %
8	25.5%	11.5 %	37.1%	25.9 %
9	31.5%	15.2 %	25.1%	27.1 %
10	20.4%	13.9 %	37.5%	28.2 %

No. 154

Postoperative Days	Al	α-gl	β-gl	γ-gl
20	27.0%	10.0%	42.0%	21.0%
45	20.4%	17.7%	47.5%	14.2%
60	20.8%	18.1%	42.2%	18.9%
98	19.6%	17.7%	43.4%	20.9%

Fig. 11 Relation between amount of serum protein and ascites protein

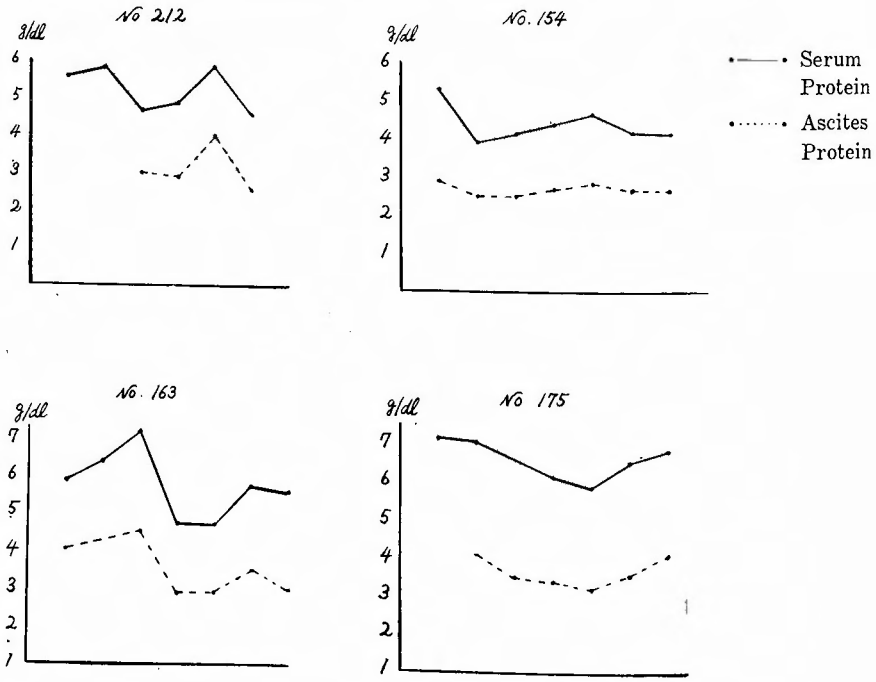


Fig. 12 Relation between pattern of serumprotein and ascites protein

No. 154

	Albumin %	α-globulin %	β-globulin %	γ-globulin %
Serum	27	10	42	21
Ascites	28.4	13.8	36.3	21.2
Serum	19.6	17.7	43.4	19.4
Ascites	20.3	18.4	46.5	14.2

No. 163

	Albumin %	α-gl %	β-gl %	γ-gl %
Serum	31.7	24.1	31.4	12.8
Ascites	33.3	21.1	32.5	13.1
Serum	23.7	11.1	37.5	27.5
Ascites	25.5	11.5	37.1	25.9

Case 2

	Albumin %	α-gl %	β-gl %	γ-gl %
Serum	48.8	10.9	17.1	23.2
Ascites	53.4	13.2	15.2	18.1
Serum	37	11.0	25	17
Ascites	44.2	12	20	25.8
Serum	46.8	9.6	14.9	28.7
Ascites	49.6	8.8	11.5	24.8

2) Relations between Serum Protein and Ascites Protein

When ascites occurred, samples of serum and ascitic fluid were taken at the same time, and their protein compared.

The amount of ascites protein reached as high as 65% of serum protein, and its increase or decrease paralleled that of serum protein. Their protein ratios, too, were nearly alike, only ascitic fluid contained a little more albumin than serum. (Figs. 11 and 12)

3) Function and Pathology of the Liver in Cases of Ascites Collection.

As mentioned above, there existed no exact parallel relation between hepatic dysfunction and the developed of ascites. In severe cases hemorrhagic ascites developed even in the early stage, and hepatic dysfunction became mone and more serious, and in such cases the surface of the liver was found to be covered with fibrinous membrane; in some there were fibrinous masses as big as a first.

In chronic cases the ascitic fluid looked clear, but in the sediment a large number of red cells were seen. The surface of the liver was covered with fibrinous membrane, and histologically thickening of the hepatic capsule, proliferation of endothelial cells, enlargement of subcapsular lymphatic spaces, and engorgement

with lymph in intra-GLISSON's capsular spaces were noted. A histologic picture suggestive of fluid extrusion from the hepatic surface was also noted (Fig. 13).

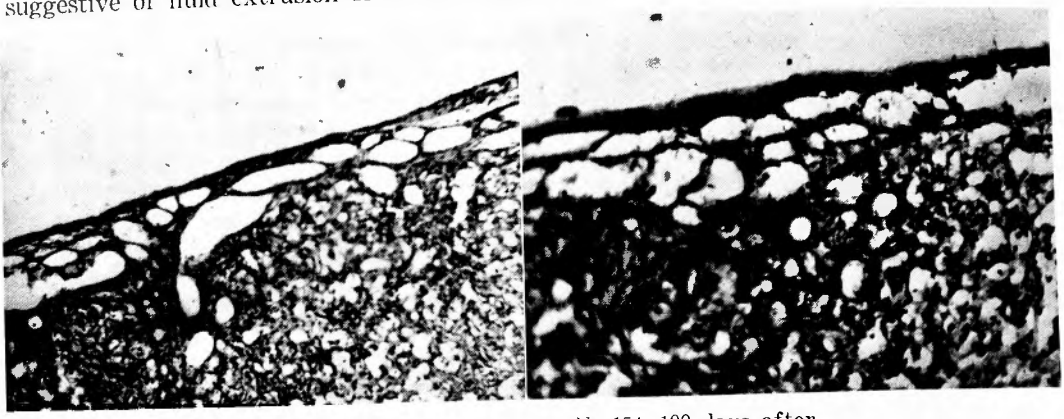


Fig. 13 No. 163. 300 days after production of pericarditis.
Thickened capsule, dilated subcapsular lymphatics and dilated sinusoid.

No. 154. 100 days after production of pericarditis.
Thickened capsule aud dilated subcapsular lymphatics and proliferation of endothelial cells.

Surface of liver were coverd with fibrious membranes.
This picture suggestive of fluid extrusion from the hepatic surfaces.

4) Summary and Discussion

The cause of the early collection of ascites in this disease was attributed by ALTSCHULE to the early appearance of hepatic dysfunction. Pressure rise in thoracic lymphatics was further added as a factor by BLALOCK and BURWELL. BOUCEK et al. stated after experimenting with dogs with pericarditis created by the DAKIN solution techinc that the escape of lymph from the liver into the abdominal cavity might play a role in the genesis of ascites.

Led by BOLTON, Mc KEE and VOLWILER, many researchers have investigated the cause of common ascites. This problem has been studied by experimental production of ascites through constriction of thoracic inferior vena cava, and at present the idea that escaping lymph from the liver is the source of ascites is most widely held.

In experiments with constriction of the vena cava DRINKER et al. noted that the amount of ascitic fluid protein nearly equaled that of hepatic lymph, and asserted that ascites originated in the lymph of the liver. MC KEE, BOLTON and BARNARD found enlargement of lymphatics of the hepatic surface, and proliferation of capsular endothelial cells, and took these findings as evidence of lymph extrusion from the liver. BOLTON, observing red cells in ascites, held that changes in the vessel wall might also be responsible. LUETSCH and TAKAHASHI noted similarity in protein quotient between ascites and edema.

The present author's results correspond perfectly with these. That is, he noted in ascites a large volume of protein which might be supposed to have originated in the lymph of the liver. He also ascertained that a large number of red cells were present in ascites. This fact suggests rupture of some of the blood vessels. It was also pathologically noted that the surface of the liver was adherent with bloody fibrious masses due to rupture of blood vessels, and covered with fibrinous membranes. These findings suggest extrusion of lymph from the hepatic surfaces. Moreover, capsular thickening caused by proliferation of capsular endothelial cells was present together with enlargement of subcapsular lymphatic spaces, and engorgement of lymph. These findings, too, lend support to the lymphatic origin of ascites.

As in TAKAHASHI'S and LUETSCH'S experiments, similarity in protein quotient was noted between ascitic fluid and serum.

This results suggest that extrusion of lymph from the liver because of hepatic engorgement is an important factor in the formation of ascites in dogs with constrictive pericarditis, just as in the case of dogs with a constricted thoracic vena cava.

In regard to the amount of serum protein and albumin which have the most important connection with colloidal osmotic pressure, the production of pericarditis generally brings about hypoproteinemia, but a large volume of protein is sometimes demonstrated even when ascites has developed. Thus a parallel relation dose not always exist between the amount of protein and the degree of ascites. Albumin generally decrease, whenever ascites develops, but here too, there is no exact quantitative parallel.

As stated above, ascites dose not always parallel hepatic dysfunction, hypoproteinemia and hypoalbuminemia, which nevertheless play an important role in the genesis of ascites.

Such being the case, it is reasonable to consider that extrusion of lymph caused by hepatic engorgement due to increased central venous pressure is the most powerful factor in the genesis of ascites in this disease, just as in the case of constricted vena cava, though hepatic dysfunction, hypoproteinemia and other various factors also play assisting roles.

The cause of hypoproteinemia will now be discussed. As shown in Fig. 9, if paracentesis was not done, the amount of serum protein did not change, and continued to be comparatively large, but if ascitic fluid was removed, the amount of serum protein immediately began to decrease. But a fall in intra-abdominal pessure

facilitated accumulation of ascitic fluid, and ascites reached the pre-paracentesis two or three days later (Fig. 9).

In the case of No. 163, however, no ascitic fluid collected after paracentesis in the early stage. But in the late stage when decompensation appeared, ascites production, paracentesis and hypoproteinemia followed one another in a vicious circle, and more and more serious hypoproteinemia resulted.

To sum up, in the early stage with no decompensation, hypoproteinemia caused by paracentesis is well compensated by the disappearance of ascites due to decrease of systemic venous pressure, and serum protein easily regains its former balance with improvement of the general condition, while in the period of decompensation paracentesis seem to aggravate hypoproteinemia.

Chapter II Segmental Constrictive Pericarditis

In inducing segmental pericarditis, the pericardium was fixed to the myocardium with the coronary artery as a mark in order to make pockets on the right and left sides. The sponge was inserted into either one of these pockets.

1) Constriction of Right Side of the Heart

In this constriction the experimental animals developed nearly the same symptoms as in constriction of the whole heart. In severe cases venous pressure rose to about 100mm of water, and hepatic dysfunction appeared together with ascites.

Pathologic changes in the liver were the same as in the cases of constriction of the whole heart. The cobalt and cadmium reaction tests showed a shift to the left, and the BSP test revealed ever increasing excretory difficulty (Fig. 6).

Severe cases died in about a month. Pulmonary congestion was noted in the early stage, but soon disappeared.

In light cases the venous pressure rise was slight, and no symptoms of right heart failure were noted. They continued to live for a long time. If constriction of the left side of the heart was added to these light cases, rapid development of right heart failure occurred. This interesting fact, as KUMA, one of our associates, has already reported, shows that the ventricular septum has the capability of automatically controlling the outputs of both ventricles.

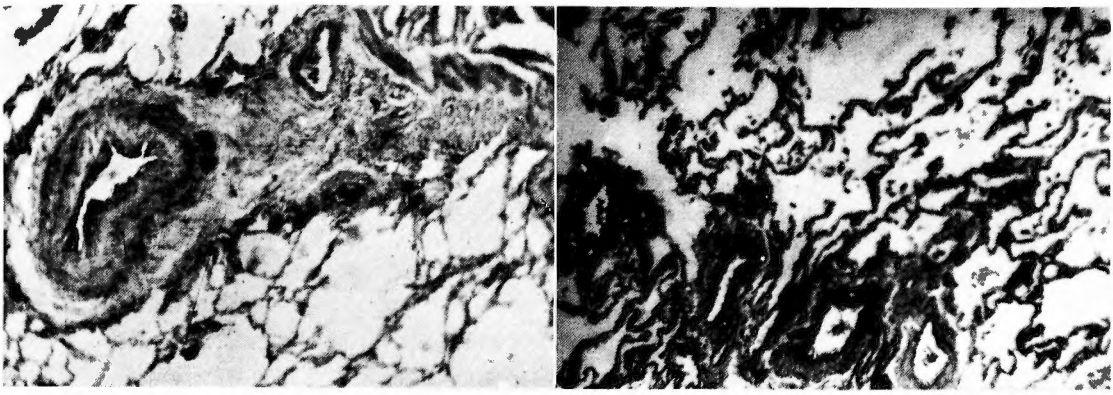
2) Constriction of Left Side of the Heart

Induced constriction of the left side of the heart temporarily raises venous pressure, and at the same time causes congestion in the lungs as well as in the liver. These symptoms, however, vanish with the passing away of the acute stage. Intense pulmonary congestion was the cause of early death in severe cases.

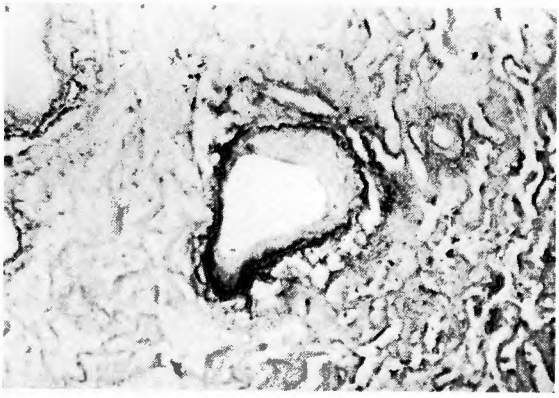
In chronic cases venous pressure returns to normal when the acute stage is over. These cases survived for a long period of time apparently with no symptoms. No hepatic dysfunction was noted.

Histologic examination of these cases, however, showed thickening of vessel walls in the lungs due to the increased pulmonary arterial pressure. Further, in some of these cases were noted congestion around the central veins, and proliferation and infiltration of the connective tissue of GLISSON's capsule. But it is hard to decide whether or not these changes were brought about by anoxemia due to left heart

Fig. 14 No. 24 No. 21



Constriction of the left side of the heart. Thickening of pulmonary vein.



Case 1 Postmortem section show thickening of pulmonary vein.

failure (Fig. 14).

3) Summary and Discussion

Induced constriction of the right side of the heart brings about venous pressure rise, and right heart failure in severe cases, but in light cases produces only a mild increase in venous pressure, and no right heart failure. Symptoms of right heart failure produced by this constriction are less severe than those in constriction of the whole heart.

Induced constriction of the left side of the heart produces no venous pressure rise. Symptoms of left heart failure appear only in severe cases; light cases apparently regress without symptoms. But even in the latter, thickening of vessel walls of the lungs ensues if the arterial pressure of the lungs continues to be high for a long time.

In some long-surviving cases hepatic dysfunction was observed. It may be too bold to hold anoxemia responsible for this, as many points still remain unclarified.

From the above experimental results, effects of partial pericardiectomy in cases with the whole heart constricted may be inferred to be as follows:

Depression of venous pressure may be expected from pericardiectomy of the right

side of the heart, but if the output of the left ventricle does not increase in corespondence with the increased right ventricular output, pulmonary congestion, or acute dilatation of the right ventricle will occur.

Pericardiectomy of the left side of the heart will decrease the arterial pressure of the lungs, and the function of the right ventricle will be improved somewhat, as the ventricular septum, as KUMA stated, controls ventricular output. But this sort of pericardiectomy will not be effective enough for such symptoms of right heart failure as ascites and edema.

The following pericardiectomy was conducted to meet the above considerations.

Chapter III Pericardiectomy

The effects of partial pericardiectomy in cases of constriction of the whole heart were studied, and comparison was made between these cases and those with segmental pericarditis. Also some speculation was done as to how much of the pericardium should be excised.

1) Methods

A few cases of constriction of the whole heart which had suffered from continuous collection of ascites, but were sufficiently well to endure the operation were chosen for partial pericardiectomy through right or left thoracotomy. They included those cases which survived for one year, and one year and a half. The venous pressure and hepatic function of these animals

Fig. 15 The venous pressure in the course of pericardiectomy.

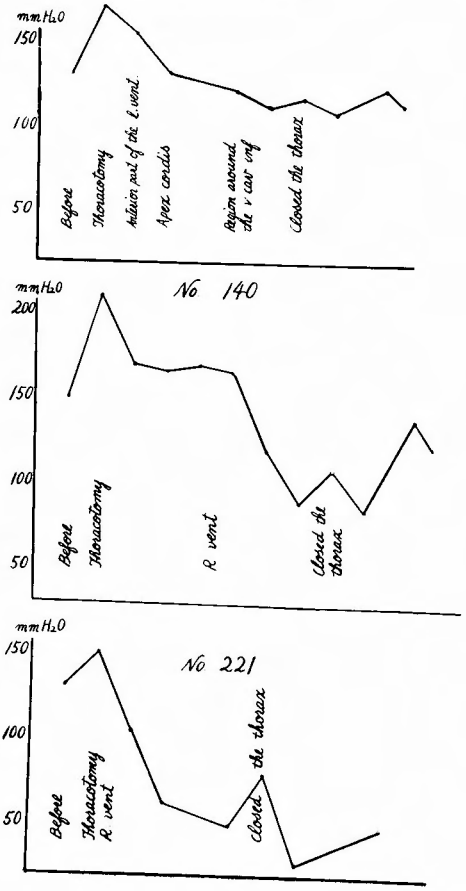
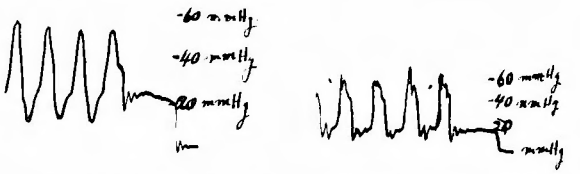
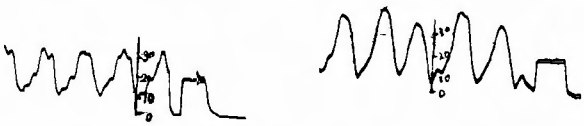


Fig. 16 The right heart pressure in the course of pericardiectomy. No. 221



Before Pericardiectomy After pericardiectomy
Systolic pressure 42mmHg→52mmHg
Diastolic pressure 3mmHg→0mmHg
Enddiastolic pressure 11mmHg→6mmHg

No. 140



Before Pericardiectomy After pericardiectomy
Systolic pressure 32mmHg→40mmHg
Diastolic pressure 10mmHg→8mmHg
Enddiastolic pressure 18mmHg→11mmHg

were examined before, during and after the operation (Figs. 15 and 16).

2) Case Report

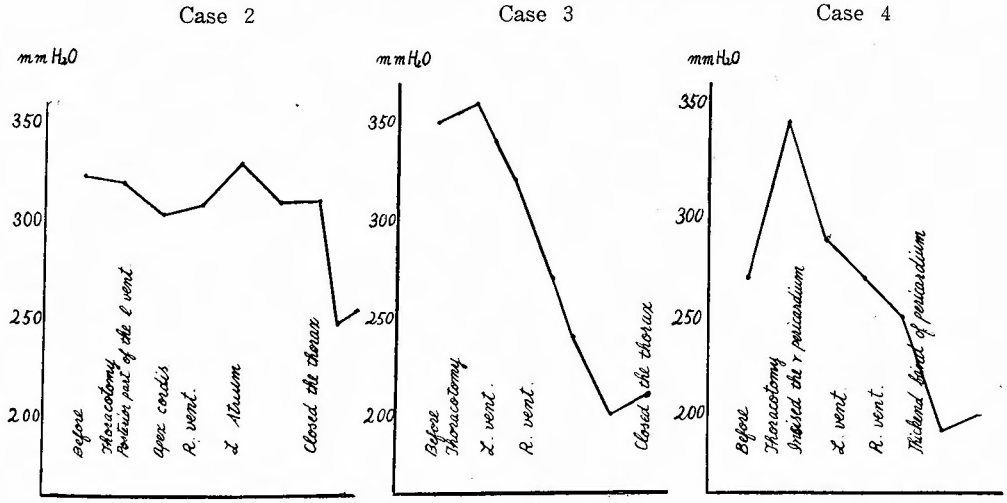
Case 1, No. 163, female, weight 15.3kg.

Underwent left pericardiectomy 11 months after production of pericarditis. In the early stage followed a chronic course, ascites sometimes developing, sometimes disappearing. From about the 200th day ascites was continuous. Before operation the venous pressure of the lower leg was 125mm of water. BSP retention 7.5% at 10 minutes. Serum protein 5.49 g/dl, Albumin 20.4%, α-globulin 13.9%, β-globulin 37.5%, γ-globulin 18.2%.

Before the operation as much ascitic fluid removed as possible. The operation was done through opening the left side of the thorax.

The polyvinylformal sponge adhered completely to the heart, and was of cartilaginoid solidity. Action of the heart was markedly restricted. Marking the course of the coronary artery, we excised the pericardium over the left ventricle, and the sponge together with it. The venous pressure dropped from 165mm to 110 mm of water. The pericardium was further decorticated from the regions around the inferior vena cava, but no change occurred in venous pressure. Loss of blood amounted to 100 cc during the operation. A postoperative transfusion was done. After closing the thorax specimens of the liver were taken through laparotomy. The liver was markedly enlarged and congested. On the 2nd day after operation ascites began to develop, and the venous pressure was 110mm of water. On the 10 th day 3500 cc of ascitic fluid was removed by paracentesis. But right heart failure went from bad to worse. On the 14th day 4000 cc of ascitic fluid was again removed. BSP retention was 12.5% after 10 minutes. On the 15th day this animal died of dyspnea after jumping down from a table one meter high. Findings at necropsy: Pleural effusion 150 cc, slight pulmonary congestion macroscopically. Ascitic fluid 500 cc. Intense hepatic congestion. The surface of the liver was coated with fibrinous masses.

Fig. 17 The venous pressure in the course of pericardiectomy.



Case 2, No. 140, male, black, weight 11. 4kg.

Underwent right pericardiectomy one year and a half after production of pericarditis. Before the operation the venous pressure was 118mm of water. BSP retention 7.5% at 10 minutes. On the day before the operation serum protein (T. P.) was 4.7g/dl, Albumin 34%, α-globulin 15.9%, β-globulin 45.5%, γ-globulin 15.4%. 1500 cc of ascitic fluid was removed. On the day of operation T. P. was 3.9 gr/dl.

The right side of the thorax was opened, and four fifths of the pericardium was excised, only the portion posterior to the right ventricle being left. Depression of the venous and the endocardiac pressure was noted. On the 2nd day after the operation ascites began to develop, and right heart failure gradually went from bad to worse, complicated by edema. This animal died on the 14th postoperative day. On the 10th day BSP retention was 10% after 10 minutes.

Findings at necropsy: slight pulmonary congestion. Ascitic fluid 1500 cc. Intense hepatic congestion.

Case 3, No. 221, male, black, weight 14.6kg.

Continuous presence of ascites was noted from the 10th day after production of pericarditis. Right pericardiectomy was performed in the fourth week. On the day before the operation 2000 cc of ascitic fluid was removed. Before the operation the venous pressure was 130mm of water. BSP retention was 15% after 10 minutes. T. P. was 5.9g/dl, Albumin 24.3%, α-globulin 13.1%, β-globulin 46%, γ-globulin 16.6%. During and following the operation the venous pressure commenced to decrease markedly, and the right endocardiac pressure also improved. The postoperative general condition was good with no ascites present. On about the 10th postoperative day the operation wound began to open, and 2 weeks later this animal died of spontaneous pneumothorax. Findings at necropsy: Pleural effusion 50 cc No ascites. Slight hepatic congestion. No fibrinous membrane noted on the surface of the liver.

3) Summary and Discussion

Isaac's and Minakawa's papers are at present the only reports on experimental pericardiectomy. To judge the effects of pericardiectomy these researchers took mid- and postoperative changes in the venous pressure as the standard.

The most important and knotty problem in the experimental production of pericarditis is how to produce chronic pericarditis very similar in symptoms to clinical cases. Many more difficulties must be overcome, if we want to enable the cases of induced pericarditis to live for a sufficiently long time to make significant observations on the effects of the operation. By overcoming these difficulties we fortunately succeeded in performing pericardiectomy on one case with ascites more than one year after the production of pericarditis. The performance of this operation on such a long-surviving case of induced pericarditis has not been recorded in medical literature.

i) Venous Pressure

After making an experimental study of pericardiectomy Isaacs stated that left pericardiectomy produced hemodynamics similar to those of constriction of the right

side of the heart, while from right pericardiectomy resulted hemodynamics similar to those of constriction of the left side, too, is able to cause a decrease in venous pressure.

In regard to changes in venous pressure due to the operation, these two researchers unaimously reported that venous pressure commenced a marked decrease already during the operation, and dropped to normal in the immediate postoperative period. In their experiments, however, pericardiectomy was usually done about a month after the production of pericarditis, in the acute or subacute stage when myocardial failure was still latent.

No. 221 which underwent right pericardiectomy about one month after production of pericarditis showed a marked decrease in venous pressure already in the course of operation like the cases reported by the above researchers, and had no ascites after the operation. But pericardiectomy on the long-surviving cases which showed symptoms similar to those of clinical cases proved to be not so effective. Namely, though excision of the right side of the pericardium produced a slight fall in venous pressure in No. 140, the venous pressure was, two or three days later, as high as before the operation. Moreover, right heart failure took a sudden turn for the worse, and a few days later a large collection of fresh ascites and edema brought death to this animal. Pathologic examination of this case revealed atrophy and degeneration of the myocardium, as will be shortly reported by SAITO, one of our associates. But in the former case no such pathologic changes were noted in the heart.

From the above data it is considered that normalization of the venous pressure depends very much on the condition of the myocardium at the time of pericardiectomy; in the stage when myocardial failures has occurred, many other factors which prevent depression of the venous pressure, such as change in venous tone and vessel walls, must be considered to be present in more advanced degrees.

In No. 163 the venous pressure decreased while the left side of the pericardium was being excised, but after the operation rose again to its preoperative height. This animal died of postoperative exacerbation of right heart failure. In this case, too, myocardial failure was pathologically demonstrated.

As shown above, and as was inferred from experimental results on segmental pericarditis, pericardiectomy, whether right or left, is capable of decreasing the venous pressure. It is desirable for complete normalization of venous pressure that the pericardium should be removed as extensively as possible.

When severe myocardial failure is present, no essential decrease in venous pressure can be expected from pericardiectomy; a slight midoperative depression is soon followed by a postoperative rise. Therefore, the operation should be done in the early stage before myocardial failure becomes serious.

ii) Liver Function

The influences of pericardiectomy upon the liver will be discussed here.

In No. 221 preoperative biopsy should intense congestion of the liver accompanied by hemorrhage in places. BSP retention was as high as 15%, intense congestion

must have disturbed the function of hepatic cells. But after operation decrease in venous pressure, and disappearance of ascites were noted. The BSP test, too, gave a better result (5%). At necropsy comparatively mild atrophy of hepatic cells was noted. This fact shows that hepatic dysfunction brought about by intense congestion is easily recoverable through pericardiectomy in the early stage when myocardial failure is absent.

In such chronic cases as No. 163 and No. 140 fibrosis and atrophy of hepatic cells were histologically more predominant than congestion. Disturbance of the excretory function, too, was rather mild (BSP retention 7.5～10%). But in spite of all this, the operation exacerbated right heart failure, and postoperative BSP retention became high. It is of course very unreasonable to expect that a long-standing hepatic dysfunction should show instant improvement with pericardiectomy. Moreover, the decrease in venous pressure brought about by pericardiectomy will be offset by various injurious factors attending surgery which tend to increase hepatic dysfunction, hypoproteinemia, ascites and edema. Therefore in such chronic cases pericardiectomy has an ominous possiblity of causing untimely death by giving rise to a temporary postoperative aggravation of right heart failure. Such being the case, if mid- and postoperative decrease in venous pressure is slight, and if severe excretory difficulty is present in chronic cases, hepatic dysfunction and hypoproteinemia must be given adequate pre- and postoperative treatment.

PART II STUDIES ON CLINICAL CASES

The present author studied four clinical cases which underwent pericardiectomy at our hospital, making use of the experimental results of part I, and compared them with the experimental cases.

1. Case Report

Case 1, H. K., aged 17, male, unemployed.

Chief complaint: tachycardia and dyspnea. History of the disease: Participated in a Marathon eight months ago; one week after the race developed tachycardia and dyspnea; then edema, oliguria and abdominal swelling (Ascites) made their appearance. Admitted after repeated episodes. Clinical Findings at the time of admission: Ascites and edema absent. Area of cardiac dulness enlarged. Pericardial friction rub heard. Rales present in both lungs. Pericarditis pattern and right ventricular hypertrophy noted in electrocardiograms. Before operation the liver was enlarged by five finger-breadths. The pressure of the cubital vein was 220mm of water. BSP retention was 35% at 30 minutes. Co.R R3, CdR R7, T. P. was 5.4g/dl, albumin 53.2%, α-globulin 8.0%, β-globulin 16.8%, γ-giobulin 20.0%, A/G 1. 14. Total serum choresterol was 159.48 mg/dl, chlesterol ester 138.7 mg/dl.

The thorax was opened through a left dorsal incision. The lungs were adherent to the pleural and the diaphragma. No heart action was noted, but it appeared with decortication of the pericardium from the regions adjoining the right and left ventricles. The patient had a favourable postoperative course, and left the hospital. He

was readmitted ten months later, as he had a relapse of edema and dyspnea. Clinical findings at the time of readmission: Venous pressure 230mm of water, Gross' reaction (±). Urinary urobilinogen (++). Serum Na 34.5mg/dl, K 15.2mg/dl, T. P. 5.4g/dl, Albumin, α-globulin, 13.9%; β-globulin, 20.9%; γ-globulin, 19.7%; CoR R1, CdR, R10. Reoperation: The thorax was opened through median sternotomy. Immediately after thoracotomy paroxysmal tachycardia (300/minute) appeared, which as a paroxysm again occurred upon removal of a 3cm. square of pericardium, the operation was discontinued, and the thorax was closed. Ten minutes after closing of the thorax, the patient stopped breathing. Though massaging of the heart produced temporary recovery, he died three hours later. Necropsy: The liver showed hypertrophy, and cirrhosis in places. Pulmonary congestion was of a mild degree, but the vessel walls showed thickening, and heart failure cells were present. Degeneration of the myocardium was noted. The cabital vein also thickening and sclerosis.

Case 2, A. T., aged 24, male, student.

Chief complaint: Abdominal swelling. History of the disease: Anorexia and general malaise in the summer of his 21st year without any special cause. In May of his 22nd year felt a pulsating pain at the back of the breast bone. In June of the same year suffered from dyspnea, abdominal swelling and fever of 38~39°C. Bloody exudate was with drawn by pericardial puncture. These symptoms were alleviated by the administration of SM, PAS, and INAH. In October facial edema appeared, and hepatic swelling was noted. In January of his 24th year abdominal swelling became very marked, and 4600 cc of ascitic fluid was removed. Clinical findings at the time of admission: Marked engorgement of the jugular vein, abdominal swelling, and signs of developing ascites. Edema absent in the lower extremities. Enlargement of area of cardiac dulness not very remarked. Heart sounds faint; murmur not distinct. The liver enlarged two finger-breadths. Operation: Median sternotomy. Decortication was done from the apex to the left ventricle, and lastly the pericardium over the right ventricle was excised, after which the heart beat gained strength. The heart axis lay horizontally.

Postoperative fall in the venous pressure was slight, and ascites did not disappear.

Effects of the operation were noted one month later in i) improvement of hepatic function, ii) appearance of the apex beat of the heart, iii) increased oxygen saturation and cardiac output, iv) increased urinary output, and v) improved appetite. But no improvement was noted in i) arterial and venous pressure, ii) the amount of ascites, and iii) QRS pattern of the electrocardiogram.

Five months after the operation the venous pressure remained still unimproved, but subjective symptoms were much better. From the 7th postoperative months ascites began to abate, and the general condition improved markedly. Ten months later the liver was enlarged only one finger breadth, and the mean urinary output was 1500 cc. A year after the operation the patient returned to school.

Case 3, aged 51, male, dealer in printed mousseline.

Chief complaint: Edema and abdominal swelling. History of the disease: Struck hard on the chest about twenty years ago, contracted right pulmonary tuberculosis.

In February of his 48th year complained of general malaise and low fever, while edema appeared on his face and lower extremities. Diagnosed as pericarditis, but took no decisive turn for better or worse. In June of his 51th year suffered from coughing, abdominal swelling, and edema of the face and lower extremities. Clinical findings at the time of admission: Marked engorgement of the jugular vein was noted. The liver was palpable three finger breadths below the costal margin. Sign of developing ascites were noted. The area of cardiac dulness was enlarged towards the left. Mucous and dry rales were heard. Biopsy after hospitalization should be hepatic cirrhosis. Operation: The thorax was opened through median sternotomy. The heart action was scarcely discernible on the anterior part of the right side, but slightly noted on the left side. Decortication was conducted from the region adjoining the left ventricle towards the right ventricle. The right side of the pericardium was thickened, and contained the residue of the cavum pericardii, in which neither pus nor caseous masses were present. On the right side excision extended to the right auricle. While excision was being done, calcium deposits were detected in several places.

About twenty days after the operation i) dyspnea, ii) abdominal swelling and iii) engorgement of the jugular vein disappeared. About three months later the venous pressure fell to 100mm of water accompanied by marked improvement in the general condition.

Case 4, Y. T., aged 26, male.

Chief complaint: Edema of the lower extremities, and abdominal swelling. History of the disease: In April of 1942 had a chill, and fever of 39°C without any exciting cause. Under the diagnosis of tuberculous pericarditis he received SM, PAS, and INAH. The fever abated but it was replaced by systemic edema, abdominal swelling, and coughing. Digitalis and diuretic drugs had no decisive effect on these symptoms. Clinical findings at the time of admission: Abdominal swelling was present. The liver edge was palpable three finger breadths. Signs of developing ascites noted. Polycythemia was present. Operation: The thorax was opened through median sternotomy. Heart action was rather strong. The pericardium showed a white beltlike thickening over the heart, the left side of the right ventricle, and right atrium. The pericardium over the right ventricle was first incised, and decorticated, when the venous pressure showed small decrease. Then decortication proceeded to the left ventricle and the posterior side of the right ventricle. The thickened band was noted a little lower than the middle part of the right and left ventricles. Constricted by this band, the lower part reaching the apex showed a feeble beat. Upon incision of the band the venous pressure showed a sudden, marked drop, and the beat, too, was suddenly intensified. After the operation pleural effusion collected, and decrease in serum protein due perhaps to surgical injury was noted. Development of ascites, and rise in venous pressure were also noted. But these symptoms gradually subsided.

2. Discussion

1) Peripheral Venous Pressure

Increase in venous pressure is characteristic of this disease: all our clinical cases

had pressures of 200～400mm of water. But a correlation does not necessarily exist between the height of venous pressure and the degree of hepatic dysfunction, ascites and edema. For sample, case 1 was clinically most severe, but venous pressure rise was rather low, while case 2 and 3 which had mild symptoms had rather high venous pressure. With regard to the venous pressure changes caused by pericardiectomy, excision of the left ventricular pericardium alone produced a considerable fall, but if that of the pericardium over the right ventricle was added, a far sharper drop was noted. The most remarkable drop resulted from excision of the thickened pericardium encircling the heart like a belt. In the relation of the site of excision to the decrease in venous pressure a perfect correspondence was noted between the clinical cases and the experimental ones; removal of the right side of the pericardium was more effective than that of the left side.

Papers concerning postoperative normalization of the venous pressure have been published by ZENKER, SAWYERS, SCANNEL, LINDSKOG and MOUNSEY among others, but there is some disparity of opinion on this point. Some assert that the venous pressure returns to normal within a short time after the operation, while others say that the normalization takes 10～20 week. In our case 2 and 3 in which a considerable decrease of venous pressure was noted already during the operation rapid postoperative normalization took place. Case 2 in which only a slight depression was noted during the operation showed normalization of the venous pressure and improvement of the condition only after one year and six months observation period.

MINAKAWA emphasised the correlationship between postoperative decrease of the venous pressure and pathology of the pericardium. The present author stated in Part 1 that normalization of the venous pressure depended upon the degree of myocardial failure. This fact proved to hold true in clinical cases. Namely, case 1 with advanced myocardial failure followed a very unhappy course.

Long-term observation is necessary if the venous pressure decreases only slightly during the operation. And pericardiectomy should be carried out before myocardial failure has reached the advanced stage. As much pericardium should be removed as possible.

2) Liver Function and Pathologic Physiology

i) Hepatomegaly

Though in constrictive pericarditis hepatic swelling always appears together with increase in venous pressure, a parallel relation is not always established between the height of venous pressure and the degree of hepatic swelling. But in individual cases are concerned, a parallel is noted (Fig. 19).

ii) Liver Function (Fig. 18)

SCHERLOCK once reported on the relation between the pathologic findings and the hepatic function of common cases of heart failure, and in this report he stated that constrictive pericarditis developed a higher grade of hepatic dysfunction than any other kind of heart failure. In all our cases, too, we noted hepatic dysfunction, prolongation of BSP excretion, decrease in serum cholesterol, hypoproteinemia, and abnormality in differential serum protein.

Fig 18 Liver functions

Case 2

	T. P. g/dl	Al %	α-gl %	β-gl %	γ-gl %	A/G	B. S. P. %	Co, Cd
11/Ⅱ	7.7	48.8	9.3	15.1	26.8	0.95		
20/Ⅲ	7.1	46.8	9.6	14.9	28.7	0.88	27.0	R_3 R_{12}
22/Ⅳ	6.1	37.0	11.0	25.0	27.0	0.56	25.0	R_5 R_7
8/Ⅴ	Pericardiectomy							
16/Ⅴ	5.9	40.3	18.5	19.6	21.6	0.67		
27/Ⅴ	5.8	43.3	9.3	26.6	20.8	0.76	13.0	R_2 R_{12}
7/Ⅵ	6.3						15.0	
11/Ⅵ	6.6	47.2	11.1	16.7	25.0	0.89	17.0	
12/Ⅶ	6.8	48.8	10.9	17.1	23.2	0.95		R_3 R_{10}
12/Ⅻ	8.9	49.0	9.8	12.7	28.5	0.96	13.0	

Case 3

	T. P. g/dl	Al %	α-gl %	β-gl %	γ-gl %	A/G	B. S. P %	Co	Cd
22/Ⅻ	8.4	46.9	6.1	14.6	32.4	0.88		R_6	R_8
22/Ⅰ	8.0	47.1	4.6	13.2	35.0	0.89			
21/Ⅱ	8.2	41.7	8.3	16.7	33.3	0.73	10.0		
28/Ⅳ	8.0	51.6	4.9	14.5	29.0	1.06			
20/Ⅴ	8.0	54.7	5.9	12.9	26.5	1.21		R_6	R_7
22/Ⅸ	7.9	51.0	6.4	12.1	30.5	1.04			
24/Ⅹ							17.5	R_4	R_6
13/Ⅺ	Pericardiectomy								
3/Ⅺ	8.2	42.6	7.0	18.3	32.0	0.74	7.0	R_4	R_8
21/Ⅰ	8.8	40.6	8.8	15.7	35.3	0.67	7.5	R_2	R_8

a) Cobalt and Cadmium Reaction Tests

In experimental cases with ascites a weak left side reaction was noted, but in the four clinical cases there was no uniformity in reaction, that is to say, some of them had a left side reaction, and others a right side one.

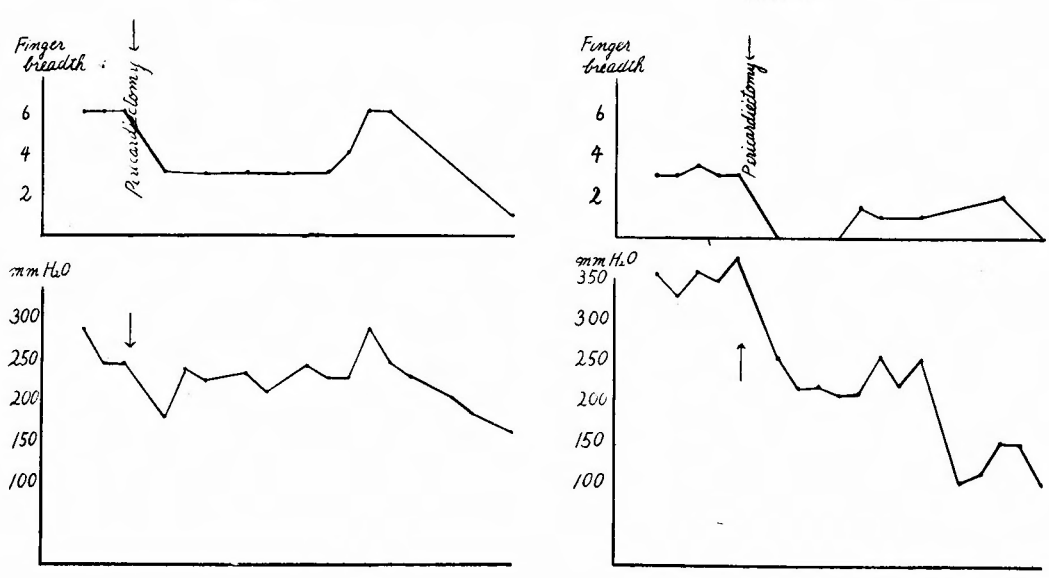
b) BSP Test and Liver Pathology

In chronic long-surviving experimental cases BSP retention showed comparatively small values in spite of hepatic fibrosis. In our clinical cases, however, the BSP test revealed a rather high degree of excretory difficulty; in case 1 BSP retention was 35% after 30 minutes, and at necropsy hepatic cirrhosis was noted. In case 3, rather intense fibrosis was noted at biopsy when BSP retention was 7% after 30 minutes. As already inferred from experimental cases, these facts should be understood as indicating that rather intense hepatic fibrosis or cirrhosis is present when BSP retention shows high values. Especially when BSP retention amounts to more than 20%, it is to be considered that hepatic cirrhosis has set in.

c) Serum Protein

Clinical cases commonly tended to be hypoproteinemia, and as ARAKI once

Fig. 19 Relation between the height of venous pressure and the degree of hepatic swelling



reported, the pronounced the hypoproteinemia, the worse the postoperstive prognosis.

Araki attributed the cause of hypoproteinemia to deterioration of the protein-producing function of the body. Paul noted hypoproteinemia in 20 of 38 cases, while Mukshik et al. held that patients before the age of ten had a peculiar pattern of serum proteins. Experimenting on dogs, we found that development of ascites did not cause severe hypoproteinemia, and had no appreciable influence upon the amount of serum protein, but that removed of ascitic fluid had much more to do with hypoproteinemia. It is receivable that in clinical cases, also paracentesis is a chief causative agent of hypoproteinemia, but also other factors such as decrease in protein intake due to abdominal swelling, hepatic dysfunction, and deterioration of the protein forming function seem to play important roles in its development.

With redard to the protein quotient, a decrease in albumin was always noted, but β-globulins with an increase of γ-globulin may be taken as signs of developing hepatic cirrhosis.

d) Effects of Pericardiectomy upon Hepatic Dysfunction

Scherlock stated that the presence of cardiac liver cirrhosis did not contraindicate pericardiectomy, as hepatic cirrhosis would become latent through the operation. In our case 4 BSP retention temporarily increased because of continuous postoperative accumulation of pleural effusion, but in all other cases the BSP test gave better results after the operation. The higher the BSP retention was before the operation, the more developed was the postoperative improvement. As stated above, a high BSP retention means the presence of hepatic cirrhosis caused by long-standing right heart failure. And it may be supposed that in such a case the myocardium also suffers from histologic damage proportional to the degree of cirrhosis.

It is therefore only natural that improvement in results of BSP test should take a considerably long time in cases with hepatic cirrhosis.

In case 2 the BSP test gave markedly better results before the venous pressure showed any sign of decrease. Cardiac catheterization showed a pronounced increase in thus showing that increase in oxygen supply (Fig. 20) can improve liver function to a certain extent in spite of high venous pressure. SCHERLOCK et al. held that anoxemia was chiefly responsible for cardiac liver cirrhosis and centrilobular necrosis in heart failure. Accordingly, even if we take the liver into consideration, the

Fig 20 Cardiac cathetelization
Case 2

Pressure mmHg	Before Pericardiectomy	After Pericardiectomy
1) P. C. m.	22	26
2) P. A. m.	30	39
3) R. V. m.	30	32
4) R. A. m.	20	19
5) I. V. C. m.	22	19.5

Blood Gass	Before Pericardiectomy CO_2	Before Pericardiectomy O_2	After Pericardiectomy CO_2	After Pericardiectomy O_2
1) P. A.	43.31	11.37	44.36	11.91
2) F. A.	39.29	18.01 (89%)	39.56	18.53 (98%)
3) Satur		19.23		19.01
4) O_2 Consumption		248cc/min		294cc/min
5) Minuten Volume		3.886*l*		4.41*l*
6) Stroke Volume		40.91cc		45.78cc

operation must be carried out in the early stage when hepatic cirrhosis has not set in. And with cases with high BSP retention attention should be paid to protection of the liver before as well as after the operation.

CONCLUDING SUMMARY

The pathologic physiology of experimentally induced chronic constrictive pericarditis was studied chiefly from the stand point of venous pressure and hepatic function. In addition four clinical cases were studied in the same way, and the following information was obtained.

1) Increased venous tone and sclerosis of venous walls are involved in the peripheral venous pressure rise characteristic of this disease.

2) Of all the liver function tests the BSP test is the best method of ascertaining hepatic function. Difficulty of BSP excretion is generally rather mild in this disease. The cases which show more than 20% retention should be considered as suffering hepatic cirrhosis. Pericardiectomy has no curative effects upon these cases.

3) The early development of ascites characteristic of this disease is not be considered as one of the symptoms of hepatic dysfunction, or polyserositis. Raised venous pressure is chiefly responsible for it. Just as in the case of experimental constriction of the thoracic inferior vena cava, the escape of lymph from the surface

of the liver due to hepatic congestion, which is in turn caused by raised central venous pressure, plays the most important role in the genesis of ascites. Much higher venous pressure is needed for edema to appear in the extremities.

4) Hypoproteinemia which has been regarded as one of the symptoms of this disease is not an essential characteristic of this disease. It was experimentally proved that its chief causative factor is loss of protein through removal of ascitic fluid which contains high concentration of protein. As a higher degree of hypoproteinemia means a worse prognosis, cases which need paracentesis should be considered for with no lose of time.

5) A considerable decrease in venous pressure results from excision of the right side of the pericardium alone, but the excision should include the left side also. If a complete decrease is desired, the excision should be as extensive as possible.

6) If pericardiectomy is done when organic myocardial failure is already present, right heart failure is often apt to get worse temporarily after the operation. This is due to decrease in serum protein, exacerbation of hepatic dysfunction, and other edema-developing factors which accompany surgery. Therefore adequate pre- and postoperative care should be directed towards the maintenance of serum protein.

Some of the clinical data given in Part II were derived from the in-patient record of the Third Medical Division (Director: Prof. M. Maekawa) of our University Hospital.

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和文抄録

収縮性心膜炎に於ける実験的並びに臨床的研究

特に静脈圧，肝機能に就いて

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毛 受 武 重

実験的慢性収縮性心膜炎を作製し，主として肝機能，静脈圧の面からその病態生理に関して検討を加え併せて4例の臨床手術例について，同じ観点から検討を加えた結果次のような知見を得た．

1) TEAB投与による静脈圧の変動並びに組織学的所見からすれば，本疾患の特徴である末梢静脈圧の上昇には，静脈壁緊張度(Venous Tone)の亢進，並びに静脈壁の硬化という因子の関与していることが考えられる．

2) 本疾患の肝機能検査中ブロームサルファレン排泄試験は肝障害の程度を知る最もよい指標であつた．

実験例並びに臨床例の成績から，本疾患の経過中，BSP 排泄障害は一般に比較的軽度であることが判つた．

従つて臨床例に於て20%以上の障害を示すものは，肝に相当高度の肝硬変症の存在するものと考えるべきである．

そしてかゝる肝障害を有する例に於ては，心膜切除術の成績も不良であつた．

3) 本疾患の特徴とされる腹水の早期貯溜は Polyserositis の部分症状，或いは肝機能障害のみによるものとは考えられず，胸腔内下空静脈狭窄実験による腹水発生に於けると同様に，中心静脈圧の上昇による肝鬱血によつての肝表面からのリンパの洩出が最も重要な腹水生成因子をなすものである．

また本疾患に於て腹水と共に四肢の浮腫が形成される為には，腹水のみの貯溜時よりも更に高い静脈圧を必要とする．

4) 本疾患の低蛋白血症は本疾患特有のものとは考えられない．

実験的には，高蛋白濃度の腹水の排除による蛋白の喪失が最も重要な因子であつた．

又低蛋白血症の著しいもの程予後は不良であり，従つて腹水穿刺を必要とする時期には速かに外科的療法を施行すべきである．

5) 心膜切除術に際しては，右心膜のみの切除でも可成りの程度まで静脈圧の下降をみるが，更に完全な低下を期待するには左心膜をも含めた広範囲の切除が望ましい．

6) 心筋障害を有する時期に於ては，心膜切除による効果も少なく，又術後手術侵襲による，血清蛋白の低下，肝機能障害，その他浮腫形成因子の加わることによつて，一時的に右心不全症状の悪化する危険がある．

従つてかゝる例に於ては，術前後の血清蛋白の維持に充分な注意を要する．