

EXPERIMENTAL STUDIES ON CONSTRICTIVE PERICARDITIS: ELECTROCARDIOGRAMS AND MYOCARDIAL HISTOLOGIC CHANGES

by

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INTRODUCTION

Whether constrictive pericarditis causes myocardial atrophy or not has been a subject of hot disputes for many years, but at present the occurrence of myocardial atrophy in this disease is generally accepted; furthermore it is also noted that calcification and connective tissue invasion thereby occur in the myocardium. The existence in this disease of such myocardial changes as atrophy and fibrosis has been given as one of the explanations for the fact that sufficiently extensive pericardial resection does not always bring about a rapid postoperative improvement even in cases with no complications. But it is still a very difficult problem to predetermine precisely the degree of myocardial involvement preoperatively. This consideration has prompted our present electrocardiographic and pathohistologic investigations into this disease. The problems we are going to face are as follows:

(1) What changes are there in the myocardium, when heart failure develops after experimental production of constrictive pericarditis?

(2) To what degree are these myocardial changes reflected in the electrocardiograms?

(3) Conversely, what significance have these electrocardiographic abnormalities for deciding operability, and prognostication of clinical cases of constrictive pericarditis?

METHODS

As already described in detail by KUMA(1957) and MENJO(1959) of our laboratory, constrictive pericarditis was experimentally produced by inserting polyvinyl-formal sponges into pericardial cavities of dogs.

Electrocardiograms were taken by following manners. Common injection needles were used as electrodes, and placed under the skin, and the limb leads were taken in the traditional manner. For chest leads V (WILSON) and CR leads were employed. The position of the chest leads was determined by taking the anatomical antero-posterior elongation of the dog thorax into consideration; V_{SR} , V_a (=anterior) and V_s were placed at the level of the apex cordis respectively in the right anterior axillary, midsternal, and left anterior axillary lines. Unanesthetized dogs were used, and in order to avoid the effects of change in position the dog was kept on the belly during the whole period of recording. Experimental animals were serially studied at certain appropriate intervals after insertion of the sponges.

As to pathohistologic investigations, the polyvinylformal sponge was cautiously removed from the heart after the dog was sacrificed, then heart weight was calculated. Thickness of the ventricular wall was measured halfway between basis and apex cordis. The heart was fixed in a neutral 10% formalin solution, and blocks were then taken from various regions of the heart mostly after the method of Gross (1930), and embedded in paraffin. To stain them, hematoxylin-eosin, VAN GIESON's and WEIGERT's elastic tissue stains were used.

RESULTS

1. Constiriction of the Whole Heart

In constriction of the whole heart, as KUMA, one of our associates, pointed out, the symptoms of right heart failure predominated, while those of left heart failure were almost latent (Fig. 1).

It is very difficult to infer the state of the myocardium from ECG alone in the early stage of the disease, for the configuration of the ST and T waves ascribable to the influences of the epicardium and subepicardial muscle layers are very liable to variations, irrespective of the presence or otherwise of myocardial lesions, being very sensitive to slight inflammation and irritation in the pericardial cavity; the ECG first acquires its significance when the labile variation in the T wave has gradually been stabilized into what HOLZMANN(1935) called the inversion of T wave. This fact being taken into consideration, clear differences electrocardiographic as well as pathohistologic were noted between normal dogs and those with symptoms of congestive heart failure (Table I). Comparisons were further instituted between them in the ratio of heart to bodily weight, and in thickness of the ventricular wall, but on these points no definite differences were noted perhaps because of possible alterations in the amount of extracellular fluid or interstitial tissue (Table II). Also no parallel

Table I PATHOHISTOLOGIC AND ELECTROCARDIOGRAPHIC COMPARISONS INSTITUTED AFTER EXPERIMENTAL CONSTRICTION OF THE WHOLE HEART BETWEEN DOGS WITH AND WITHOUT SYMPTOMS OF CONGESTIVE HEART FAILURE

			DOGS WITH SYMPTOMS	DOGS WITHOUT SYMPTOMS
PATHOHISTOLOGIC CHANGES	MYOCARDIAL CHANGES	ATROPHY	+	—
		DEGENERATION	+	—
		CONNECTIVE TISSUE PROLIFERATION	÷	—
	INTERSTITIAL CHANGES*		+	—
ELECTROCARDIOGRAPHIC ABNORMALITIES	LOW VOLTAGE		+	— (+)
	INVERTED T WAVE		+	— (+)

* Interstitial inflammation consisting of interstitial edema, cell infiltration and congestion.

Table II COMPARISONS IN RATIO OF HEART TO BODILY WEIGHT AND IN THICKNESS OF VENTRICULAR WALL BETWEEN THREE GROUPS OF DOGS*

*(One group was composed of dogs with constriction of the whole heart subdivided into those with and those without symptoms of congestive heart failure; another group of those with adherent non-constrictive pericarditis; and the third of normal dogs.)

		BODILY WEIGHT (kg)	HEART WEIGHT (gr)	H.W. / B.W. (%) #	THICKNESS OF WALL (mm)		
					LEFT VENT.	VENT. SEPT.	RIGHT VENT.
CONSTRICTION OF WHOLE HEART	No. 203	14.1	112.0	7.94	9.5	9.0	5.7
	DOGS WITH SYMPTOMS	215	16.6	112.0	6.75	12.0	9.3
	OF CONGESTIVE HEART FAILURE	212	7.9	58.5	7.41	7.0	6.5
		144	11.2	89.5	7.99	9.9	7.3
	MEAN		12.45	93.0	7.52	9.60	8.03
	No. 214	10.7	79.0	7.38	8.0	7.8	5.0
	DOGS WITHOUT SYMPTOMS	180			9.5	8.3	3.8
ADHERENT NON-CONSTRICTIVE PERICARDITIS	OF CONGESTIVE HEART FAILURE	204	10.8	111.0	10.28	10.3	9.5
	MEAN		10.75	95.0	8.83	9.27	8.53
	No. 1	8.4	75.5	8.99	11.0	10.4	4.5
	2	10.8	85.3	8.28	10.1	10.8	4.6
	3	9.1	100.0	10.99	10.5	9.6	4.8
NORMAL DOGS (MEAN OF 4 DOGS)	4	9.3	100.0	10.75	10.5	10.0	5.2
	MEAN		9.30	90.3	9.75	10.53	10.20
							4.78
							5.18

HEART WEIGHT/BODILY WEIGHT

relationship was established between the symptoms of the right heart failure and the degree of myocardial damage of the right ventricle to be mentioned later (Table III). This observation is in agreement with Iguchi's statement (1955) that though

Table III COMPARISONS OF THE SYMPTOMS OF THE RIGHT HEART FAILURE WITH THEIR CORRESPONDING PATHOHISTOLOGIC CHANGES

(Dogs included here are those which developed the symptoms of the right heart failure after experimental constriction of the whole heart.)

DOGS	SYMPTOMS OF THE RIGHT HEART FAILURE				PATHOHISTOLOGIC CHANGES							
	VENOUS PRESSURE (mmH ₂ O)	EDEMA	ASCITES	LIVER CONGESTION #	RIGHT VENTRICLE				LEFT VENTRICLE			
					MYOCARDIAL CHANGES			INTERSTITIAL CHANGES*	MYOCARDIAL CHANGES			INTERSTITIAL CHANGES*
					ATROPHY	DEGENERATION	CONNECTIVE TISSUE PROLIFERATION		ATROPHY	DEGENERATION	CONNECTIVE TISSUE PROLIFERATION	
No. 124		-	+	+	-	-	-	-	-	-	-	-
215	100	-	+	+	-	-	-	+	+	-	-	+
125	105	-	+	+	-	-	-	-	-	+	+	+
194	90~175	-	+	+	+	+	+	+	+	+	+	+
165		-	+	+	+	-	-	+	+	+	-	+
212	150	-	+	+	+	-	-	+	-	-	+	+
203	120	+	+	+	-	-	+	-	-	+	-	-
161	200	+	+	+	-	+	-	-	-	-	-	-
154	225	+	+	+	-	+	-	+	+	+	-	+

Confirmed at postmortem examination.

* Interstitial inflammation consisting of interstitial edema and cell infiltration; and interstitial fat proliferation.

microscopic study reveals various myocardial, valvular or vascular abnormalities in subjects with congestive heart failure, none of them is specific for this disease, nor can they explain its development.

Next, myocardial changes were pathohistologically classified into the following three stages (Table IV) :

First Phase: Edema, distended capillaries, and cell infiltration composed chiefly of neutrophils and small round cells in the epicardium; slight cell infiltration and capillary congestion and hemorrhage, but no other marked changes in the muscle layers; striking foreign-body reaction, namely, infiltration of neutrophils, round cells (monocytes and histiocytes) and foreign-body giant cells in the pericardium and around a sponge. This phase lasted about a month.

Second Phase: Appearance of acute interstitial myocarditis; interstitial edema, cell infiltration and congestion, but no other marked changes in the muscle layers (Fig. 2, A). Foreign-body reaction and connective tissue proliferation composed of

Table IV PATHOHISTOLOGIC CHANGES IN THE MYOCARDIUM CLASSIFIED INTO THREE STAGES AND THE CORRESPONDING ELECTROCARDIOGRAPHIC ABNORMALITIES

(Dogs included here are those which developed the symptoms of the right heart failure after experimental constriction of the whole heart.)

PHASES	DOGS	PATHOHISTOLGIC CHANGES								ELECTROCARDIO- GRAPHIC ABNORMALITIES	
		RIGHT VENTRICLE				LEFT VENTRICLE					
		MYOCARDIAL CHANGES			INTERSTITIAL CHANGES*	MYOCARDIAL CHNGES			INTERSTITIAL CHANGES*		
		ATROPHY	DEGENERATION	CONNECTIVE TISSUE PROLIFERATION		ATROPHY	DEGENERATION	CONNECTIVE TISSUE PROLIFERATION			
										LOW VOLTAGE	INVERTED T WAVE
FIRST PHASE	No. 165	+	-	-	+	÷	÷	-	+		
	149									+	-
	124	-	-	-	-	-	-	-	-		
	203	-	-	÷	-	-	+	-	-	÷	-
	215	-	-	-	+	÷	-	-	÷	+	-
	125	-	-	-	-	-	+	+	÷		
SECOND PHASE	221	#								+	+
	161	-	+	-	-	-	-	-	-	#	-
	154	-	+	-	+	+	÷	-	÷	#	-
THIRD PHASE	212	÷	-	-	+	-	-	÷	+	#	#
	194	#	÷	÷	#	+	÷	÷	+	#	÷
	163	#								-	#
	140	#								-	÷

* Interstitial inflammation consisting chiefly of interstitial edema and cell infiltration; and interstitial fat proliferation.

Pericardiectomized cases (to be mentioned later).

fibroblasts and fibrocytes in the pericardium and around a sponge. This phase lasted about 100 days.

Third Phase: Commencement of myocardial atrophy and degeneration (chiefly vacuolar), besides findings of acute interstitial myocarditis in the middle layers of cardiac musculature. Occasional occurrence of connective tissue invasion and marked myocardial atrophy in the subepicardial muscle layers; proliferation of interstitial fat in the subendocardial region (Fig. 3, A, B, C). Decrease in number of functional cells, and replacement of them by proliferating connective tissue in the pericardium and around a sponge.

It is especially noteworthy that such regressive changes as atrophy and degeneration predominated in pathohistologic aspect of these myocardial lesions, and that the right ventricle was more severely affected than the left. But neither calcification nor fibrosis were noted in the myocardium, and the endocardium and papillary muscles,

too, showed no marked changes. No correlationship was seen between the degree of myocardial damage and thickness of the epi- and pericardium.

In order stereoscopically to investigate vascular changes at this stage of atrophy and degeneration bismuth-gelatin were injected into the right and left coronary arteries, and roentgenograms were taken. In dogs with symptoms of congestive heart failure the coronary arteries gave the impression of being rigid and stiff, and looked very constricted, when compared with those of normal dogs (Fig. 4, A, B). But pathohistologically no changes answering to these roentgenographic findings were discovered in the vessels.

Next, these pathohistologic changes in the myocardium classified into three phases were contrasted with the ECG of the corresponding stages. A low-voltage appeared in the second phase, and in the third phase, in addition to it, the inversion of T waves tended to occur (Fig. 2, B) (Fig. 3, D). It is very interesting to note that electrocardiographic abnormalities did not appear until pathohistologic changes in the myocardium reached a certain advanced stage, and that pathohistologic changes became more prominent corresponding to the progress of the electrocardiographic changes.

2. Constriction of the Left and Right Side of the Heart

To clarify the mechanism of development of heart failure, right and left pericardial cavities were each inserted into with sponges (Table V).

Table V PATHOHISTOLOGIC CHANGES AND ELECTROCARDIOGRAPHIC ABNORMALITIES IN CONSTRICTIVE PERICARDITIS OF THE RIGHT AND LEFT SIDE OF THE HEART

			CONSTRICTIVE PERICARDITIS OF LEFT SIDE OF THE HEART		CONSTRICTIVE PERICARDITIS OF RIGHT SIDE OF THE HEART	
			LEFT VENTR.	RIGHT VENTR.	LEFT VENTR.	RIGHT VENTR.
PATHO-HISTOLOGIC CHANGES	MYOCARDIAL CHANGES	ATROPHY	+	+	—	÷
		DEGENERATION	÷	++	—	++
		CONNECTIVE TISSUE PROLIFERATION	÷	—	—	—
	INTERSTITIAL CHANGES*		÷	+	—	—
ELECTROCARDIOGRAPHIC ABNORMALITIES			LOW VOLTAGE		÷	
			INVERTED T WAVE		+	

* Interstitial inflammation consisting chiefly of interstitial edema, cell infiltration, and congestion; and interstitial fat proliferation.

In long-surviving dogs with constrictive pericarditis of the left side, pathohisto-

logic changes and electrocardiographic abnormalities resembled those of the above-mentioned third phase rather than of the second, but dogs whose right side was constricted showed signs and symptoms very similar to those of the second (Fig. 5, A, B). However, when one side of the pericardium was thus constricted, the chest leads over the same side did not reveal any specific variations.

The present pathohistologic investigation has made it clear that such myocardial damage as atrophy and degeneration is more apt to develop in the right ventricle than in the left not only in constriction of the whole heart, but of the left side as well. But no specific variations were noted in the chest leads over the right side.

3. Constriction of the Inferior, and of the Superior and Inferior Venae Cavae

When the inferior vena cava was constricted to one-half or one-third its outside diameter in three dogs, a marked low-voltage appeared, sometimes accompanied by the inversion of T waves. Similar findings were obtained when in four dogs the superior and inferior venae cavae were constricted to one-half or one-third their respective diameters, but with no ligation of the azygos vein (Fig. 6, A, B). Boucek et al. (1952), too, noted the reduction of amplitude of the QRS-complex of the electrocardiogram during the progressive circulatory failure in dogs with constriction of the superior and inferior venae cavae and ligation of the azygos vein. This fact teaches that it is a great mistake to ascribe a low-voltage solely to myocardial damage. The same experience was obtained from a case of constriction of the whole heart. When this case developed congestive heart failure after experimental induction of pericarditis, all the limb and chest leads showed the progressive reduction of amplitude, besides tachycardia and ventricular extrasystole (Fig. 7, A). And at postmortem examination hydropericardium of unknown etiology (about 300-400 cc) and thickening (about 1.5 mm) of the peri- and epicardium were noted, but this case was pathohistologically shown as pancarditis, and no definite findings of myocardial atrophy were obtained (Fig. 7 B). Such being the case, low voltage in this case is undisputably regarded as due to the hydropericardium. As it is now generally agreed, it is quite dangerous to rely on electrocardiographic findings for ascertainment of myocardial damage.

4. Pericardial Resection

Partial pericardiectomy was conducted on three cases of constriction of the whole heart with congestive heart failure (Table VI). In one case (No. 221) which preoperatively showed no electrocardiographic abnormalities but the inversion of T waves congestive heart failure was remarkably improved by partial pericardial resection of the right side. Its postoperative ECG was free from marked deviations, and also pathohistologically no serious myocardial damage was demonstrated (Fig. 8). A second case (No. 163) preoperatively showed the progressive reduction of amplitude in the chest leads of the right side, besides the inversion of T waves, and its symptoms of right heart failure were rather aggravated by partial resection of the left ventricular pericardium. As had been expected, histologic study revealed advanced myocardial damage (Fig. 9, A, B, C). The last case (No. 140) in which the progressive diminution of voltage of QRS was preoperatively noted, showed no tendency of improving the symptoms of right heart failure after partial pericardial resection

Table VI COMPARISONS OF PRE- AND POSTOPERATIVE SYMPTOMS OF THE RIGHT HEART FAILURE AND PATHOHISTOLOGIC MYOCARDIAL CHANGES IN THREE PERICARDIECTOMIZED CASES OF EXPERIMENTAL CONSTRICTION OF THE WHOLE HEART

DOGS	SYMPTOMS OF THE RIGHT HEART FAILURE							
	PREOPERATIVE				POSTOPERATIVE			
	VENOUS PRESSURE mm H ₂ O	EDEMA	ASCITES	LIVER CONGESTION #	VENOUS PRESSURE mm H ₂ O	EDEMA	ASCITES	LIVER CONGESTION +
No. 221	130	—	+	+	80	—	—	+
163	125	+	+	+	110	—	+	+
140	180	+	+	+	150	+	+	+

DOGS	EXTENT OF PERICARDIAL RESECTION	PATHOHISTOLOGIC CHANGES							
		RIGHT VENTRICLE				LEFT VENTRICLE			
		MYOCARDIAL CHANGES				MYOCARDIAL CHANGES			
		ATROPHY	DEGEN- ERATION	CONNECTIVE TISSUE PROLIFERATION	INTERSTITIAL CHANGES*	ATROPHY	DEGENERATION	CONNECTIVE TISSUE PROLIFERATION	INTERSTITIAL CHANGES*
No. 221	right ventricular pericardium	—	—	—	—	—	—	—	—
163	1/2 left ventr. pericardium	+	+	—	+	+	+	—	+
140	4/5 right ventr. pericardium	—	—	—	+	+	—	+	+

During the operation of pericardiectomy laparotomy was simultaneously conducted, and specimens of hepatic tissue were taken out, and afterwards examined by MENDO of our laboratory.

+ Confirmed at postmortum examination by MENDO of our laboratory.

* Interstitial inflammation consisting chiefly of interstitial edema and cell infiltration; and interstitial fat proliferation.

of the right ventricle, and on pathohistologic examination rather intense myocardial involvement was demonstrated (Fig. 10, A, B, C, D, E).

Applying these cases to the avobementioned classification of myocardial changes into three phases, the first case may be considered to have been in the second phase at time of operation, and the latter two in the third. All these three cases died about two weeks after partial pericardiectomy before marked abnormalities appeared in the postoperative ECG.

As electrocardiographic changes are produced through very complex mechanism, it is almost impossible to determine the degree of myocardial involvement by the configuration alone, but in the above three cases the electrocardiographic findings may be said to have given rather reliable results.

CLINICAL OBSERVATION

Four patients who underwent pericardiectomy for constrictive pericarditis in our hospital will be briefly observed here from the standpoint of the present study (Table VII).

Table VII FOUR CLINICAL CASES OF CONSTRICTIVE PERICARDITIS

Here presented are pre- and postoperative symptoms of the right heart failure, history, extent of pericardial resection and pathohistological findings of excised pericardium.

CASES	SYMPTOMS OF THE RIGHT HEART FAILURE							
	PREOPERATIVE				POSTOPERATIVE			
	VENOUS PRESSURE mm H ₂ O	EDEMA	ASCITES	HEPATIC ENLARGEMENT (from costal margin)	VENOUS PRESSURE mm H ₂ O	EDEMA	ASCITES	HEPATIC ENLARGEMENT (from costal margin)
1. K. H.	220	+	—	6 finger-bredth	* 230	* +	* +	* 5 finger-bredth
2. H. T.	323-400	+	+	4½	200	—	—	1
3. I. F.	250-320	— (÷)	—	2½-3	100	—	—	not palpable
4. Y. U.	240-300	÷	+	3-4	160	—	—	1½

CASES	AGE AT TIME OF OPERATION	SEX	HISTORY		EXTENT OF PERICARDIAL RESECTION	EXCISED PERICARDIUM (PATHOLOGIC FINDINGS)
			RHEUMATOID DISEASE	TUBERCULOUS DISEASE		
1. K. H.	17	M	—	—	left ventricle	unspecific inflammation #
2. H. T.	24	M	—	At 6 tuberculous hilitis At 21 tuberculous peri carditis	both ventricles	unspecific inflammation #
3. I. F.	49	M	+ ?	At 34 right lung infiltration	both ventricles	unspecific inflammation #
4. Y. U.	24	M	—	—	both ventricles	unspecific inflammation #

* This patient was still in such a bad condition 11 months after operation that he had to be operated on once again.

All traces of specific inflammation seem to have disappeared during a protracted period of cicatrization.

Case 1, K.H. Preoperative electrocardiographic findings: low voltage; flattening or inversion of T waves in all limb and chest leads; and notching of P waves in leads II and CR₁ (Fig. 11, A). Resection of left ventricular pericardium temporarily brought about increase in voltage, and the patient made uneventful postoperative recovery for about two months, but after then, amplitude of the QRS-complex seemed to decrease progressively, and the T wave remained unchangeably negative. Eleven months later the patient had a relapse of edema and dyspnea, and was operated on second time, but this time died on the operating table due to the development of ventricular fibrillation. Postmortem findings revealed extreme thinning of the right

ventricular wall, thickness in some places being reduced to 1 mm. The heart as a whole presented a gross appearance of brown atrophy. Pathohistologic findings: severe myocardial atrophy, nuclear swelling, and occasional pyknosis; focal necrosis in myocardium; vacuolar and fatty degeneration in subendocardial muscle layers; hyaline and fibrinoid degeneration of papillary muscles; interstitial edema, fat proliferation, hemorrhage, separation of muscle groups, and thickening of blood vessels. These changes were more conspicuous in the right ventricle (Fig. 11, B, C, D, E, F).

Case 2, H.T. Preoperative electrocardiographic findings: Low voltage; flattening or inversion of T waves in all leads; prolonged peak interval of P wave in I and II; Notching of P waves in V₁, V₂, and sinus rhythm (Fig. 12, A). Postoperative recovery from congestive heart failure took nearly a year. In postoperative ECG were sinus rhythm, gradual increase of QRS waves and continued definite inversion of T waves noted (Fig. 12, B, C, D).

Case 3, I. F. Preoperative electrocardiographic findings: auricular fibrillation; inversion of T waves in leads II and III, but no other abnormalities (Fig. 13, A). Improvement of congestive heart failure occurred comparatively early after operation. In postoperative ECG auricular fibrillation and amplitude of QRS waves remained unchanged; in all leads inversion of T waves became more marked; and in leads V₅ and V₆ slight elevation of ST segment was noted (Fig. 13, B, C, D).

Case 4, Y.U. Preoperative electrocardiographic findings; sinus rhythm and low voltage; prolonged peak interval of P wave in lead II; inversion of T waves in II, III and all chest leads. Postoperative findings: sinus rhythm, gradual increase in amplitude of complexes, and intensified inversion of T waves in all leads. Improvement of congestive heart failure and of electrocardiographic abnormalities occurred nine months after operation (Fig. 14, A, B, C).

It may be known from the above clinical observations that low voltage and the inversion of T waves in preoperative ECG mean a poor, discouraging postoperative prognosis. In case 1 it was seen that serious myocardial damage in the right ventricle underlay marked electrocardiographic deviations. It is indeed a very risky venture to perform an operation on such a case as this. Case 3 with preoperative minor electrocardiographic abnormalities, on the contrary, took a speedy favorable turn after operation. Pericardiectomy is therefore best indicated only in the early stage of the disease before marked abnormalities appear in ECG.

DISCUSSION

1. What mechanism is responsible for myocardial damage in constrictive pericarditis?

It has been traditionally considered that inactivity atrophy of the myocardium is caused by hard cicatrization of the pericardium. It has also been held that it is ascribable to nutritional disturbance caused by the compression brought on the blood vessels by the cicatrized pericardium. Though no pathohistologic findings confirming these views are obtainable from small vessel walls, data of roentgenography with bismuth-gelatin do not conflict with them. Recently it has been asserted that dys-

proteinemia leads to myocardial damage, but the author could not find a definite relationship between value of serum protein measured by MENJO of our laboratory and amplitude of the complexes measured on the same time (Table VIII).

Table VIII LOW VOLTAGE CONTRASTED WITH VALUE OF SERUM PROTEIN AFTER EXPERIMENTAL CONSTRICTION OF THE WHOLE HEART IN DOGS WITH AND WITHOUT SYMPTOMS OF RIGHT HEART FAILURE

	DOGS No.	LOW VOLTAGE	VALUE OF SERUM PROTEIN (g/dl) ※
NORMAL DOGS			7-8
DOGS WITHOUT SYMPTOMS	148	—	7.5
	180	+	7.5
DOGS WITH SYMPTOMS OF THE RIGHT HEART FAILURE	221	—	5.6
	203	—	4.8
	163	—	5.4
	140	+	4.7
	194	++	6.4
	154	++	5.0
	161	++	5.7
	149	++	4.8
	212	++	5.4

※ Measured by MENJO of our laboratory.

Polyvinylformal sponges used in the present study possess only a mild irritating action, and the foreign-body reaction caused by these sponges was limited to the inside and just around themselves. Accordingly their action on the myocardium may be presumed to have been of purely mechanical nature. But in case of continued exacerbation of right heart failure there is a possibility of a secondary development of myocardial damage as a result of what WUHRMANN (1950) called a vicious circle between right heart failure and hepatic dysfunction. It is, moreover, quite thinkable that residual inflammation, relapse and other complex and unknown factors may contribute to production of myocardial damage.

DINES et al. (1958) stated the following effect after a pathologic study of necropsied clinical cases that prolonged pericardial compression was presumably the cause of uniform appearance of atrophy throughout the myocardium of both ventricles. But to the author it seems too hasty to lay on this compression alone the whole responsibility for myocardial atrophy. The author feels necessary to emphasize here the complex mechanism of myocardial atrophy.

Again DINES et al., noting that there was no necessary correlation between the duration of this disease and the amount of myocardial atrophy, stated that this atrophy would make its appearance early in the course of the disease, and show little further progress. But it was noted in the present study that relatively simple mechanical compression alone was capable of progressively increasing myocardial damage. In the light of the fact, and the abovementioned complex factors, it does

not seem advisable to employ solely conservative treatment for this disease, which may in the meantime reach the incurable stage. On this point the present author agrees with HOLMAN et al. (1949), VAN NIEUWENHUIZEN (1951), SAWYER et al. (1952) and BURWELL (1957).

The reason for the weaker resistance of the myocardium of the right ventricle is not yet adequately explained, but it may be guessed that this is in part due to commonly larger thickness of the right-side pericardium, and the thinner wall of the right ventricle.

2. To what degree is myocardial damage reflected in electrocardiograms?

It may be said from results of the present study that minor myocardial changes are not always reflected in ECG, but any considerable change necessarily produces its corresponding electrocardiographic abnormality.

Low voltage, inversion of T waves, auricular fibrillation, notching of P waves, and slurring or notching of QRS complexes are generally regarded as characteristic signs of constrictive pericarditis, but in the author's opinion the most important of these are the first two. As a matter of fact, low voltage and the inversion of T waves are observed also in anasarca, myxedema, diffuse myocardial fibrosis, hydropericardium and constriction of the inferior, and of the inferior and superior venae cavae, but if noted in cases of constrictive pericarditis, they may be taken as sure signs of serious myocardial damage. These signs are of course expressive of myocardial damage of the whole heart, but as stated above, the myocardium of the right ventricle is more easily affected than that of the left, and so it is possible from these signs to know the existence of serious myocardial damage in the right ventricle. Chest leads over the right side, however, could not give corroborating findings. This may be explained by unstable fixation of the mediastinal septum of the dog, and inappropriate position of the exploring electrodes.

3. Of what clinical use are the above considerations in deciding operability, and prognostication of cases of constrictive pericarditis?

If the abovementioned diseases are excluded, marked low voltage and the inversion of T waves in the ECG of constrictive pericarditis may safely be interpreted as signs of serious myocardial damage. And so it is very desirable to establish the diagnosis of this disease by other ordinary diagnostic means in its early stage when significant electrocardiographic findings are still unavailable; this will make early surgical approach possible, and greatly enhance the chance of success in operation.

When the above signs become more and more pronounced in serially studied ECG, the utmost caution must be exercised in indicating pericardiectomy, for operative procedure on the severely damaged myocardium will easily result in lacerations of the thinned wall, and in acute overdilatation of the attenuated and atrophied right ventricle. Under such circumstances zonular excision of the pericardium is to be preferred, but after all, even when an operative procedure is possible, satisfying results are not to be expected. Pericardiectomy should therefore be contraindicated under such circumstances.

As already pointed out, similar signs are observed also in hydropericardium,

constriction of the inferior, and of the superior and inferior venae, and other diseases. And so it must specially be remembered that we cannot pass any reasonable judgment upon the state of the myocardium from electrocardiographic findings alone; enough attention must be paid to examination of clinical symptoms and other medical tests.

SUMMARY AND CONCLUSION

1) Myocardial lesions in cases of constrictive pericarditis which presented the symptoms of congestive heart failure were histologically classified into three phases. It was thereby noted that such regressive changes as atrophy and degeneration were the predominant features of these lesions, and that the right ventricle was more severely affected than the left. Some consideration was given at the same time to the causative mechanism of these changes. It was also ascertained that electrocardiographic abnormalities corresponding to these pathohistologic changes consisted chiefly of low voltage and the inversion of T waves. It was further recognized that serious myocardial damage underlay marked electrocardiographic abnormalities.

2) Cases with congestive heart failure due to constrictive pericarditis of the left and right sides were compared respectively, and it was found that in cases of left-side pericarditis both myocardial changes and electrocardiographic abnormalities were more conspicuous than right side, and the right ventricle was involved at the same time. No matter which ventricle might have been constricted, however, the chest leads over the diseased side did not give specific changes.

3) Nearly similar electrocardiographic abnormalities, above described, were observed in hydropericardium, and constriction of the inferior, and of the inferior and superior venae cavae. It is therefore simply absurd to try to make a correct estimate of myocardial damage from electrocardiographic findings alone.

4) Pre- and postoperative serial clinical as well as experimental studies of ECG were conducted together along with pathohistologic investigation of the myocardium, and as a result it was found that the appearance of low voltage and the negative T wave, other diseases being excluded, was a factor not to be ignored in deciding operability or otherwise of cases of this disease.

Positive use of electrocardiography was urged, for constrictive pericarditis must be operated on in its early stage before the characteristic changes appear in ECG.

5) The correlationship between the progressive abnormalities in serially studied ECG and aggravating myocardial lesions was established. Therefore, if such electrocardiographic findings are obtained, pericardiectomy should be contraindicated.

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和 文 抄 録

実験的収縮性心膜炎に於ける心筋の病理組織学的所見と心電図の変化について

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収縮性心膜炎の手術的予後を左右する因子として従来心筋傷害が重視されているが、その程度を術前に心電図を以て的確に知ることが可能でないだろうかと考え、この点を解明すべく、試験として犬を用いて実験的に各種の収縮性心膜炎を作製し、心電図学的並びに病理組織学的に検索して次の結論を得た。

1) 全心の収縮性心膜炎例で心不全症状が出現した場合の心筋変化は組織学的に3期に分類したが、萎縮、変性等の退行性変化が主として認められ、左室よりも右室の方にそれら変化の強い事が観察された。而もこのような心筋変化は、血管圧迫による心筋の栄養傷害、右心不全症状と肝傷害の間に形成される悪循環によつて起る2次的な心筋傷害、或いはその他病巣の存続乃至再発等種々の複雑不明な因子が関係して発生するものと推察されたが、他方、これに対応する心電図変化としては低電位差に陰性T波を合併する傾向がみられ、この著しい心電図変化の出現したものでは心筋変化が高度であることが認められた。

2) 左心並びに右心の収縮性心膜炎例で、心不全症状が発現した場合には、後者よりも前者の方が心電図並びに心筋変化が強く、且つ右心室にも変化がみられ

た。しかしこれら両者はいずれの場合にも、同側の胸部誘導に於て特にその指標と目すべき心電図所見は得られなかった。

3) 単なる下大静脈及び上下大静脈の収縮並びに心臓水腫によつても、略々同様の心電図変化が得られた。従つて、ただ心電図所見のみから心筋の状態を論ずることは出来ない。

4) 収縮性心膜炎について、実験的、臨床的に、心臓切除術前後の心電図継続的観察と心筋の病理組織学的検索を併わせ行つた結果、その際臨床例で上述の下大静脈及び上下大静脈の狭窄、心臓水腫の外に全身浮腫、粘液水腫、びまん性心筋線維症等が除外されるならば、これを収縮性心膜炎による低電位差と陰性T波の出現と考え、このような症例にはむしろ積極的に心電図判断を下してこれら変化が典型的とならない時期に早期手術を施行すべきである。

5) 心電図の継続的観察に於て、上述の変化が高度に進行性である場合には、特に右室心筋の変化が極めて高度になつている事が予想されるので、むしろ手術禁忌であると推断すべきである。



Fig. 1 Constriction of the Whole Heart.

Cross section. (No. 154)

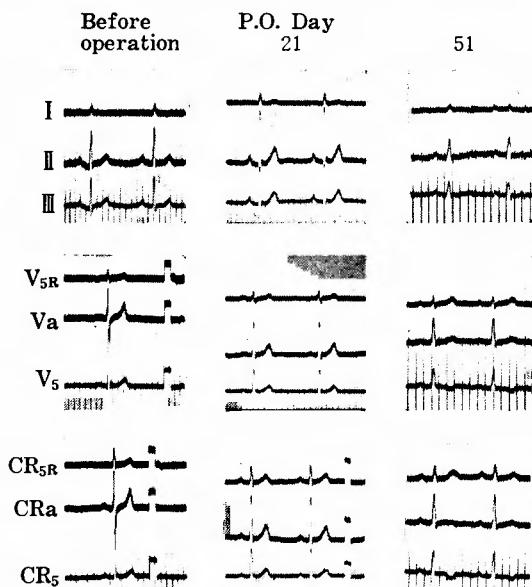


Fig. 2 Constriction of the Whole Heart.

Myocardial changes and electrocardiographic abnormalities in the second phase. (No. 161)

(A)

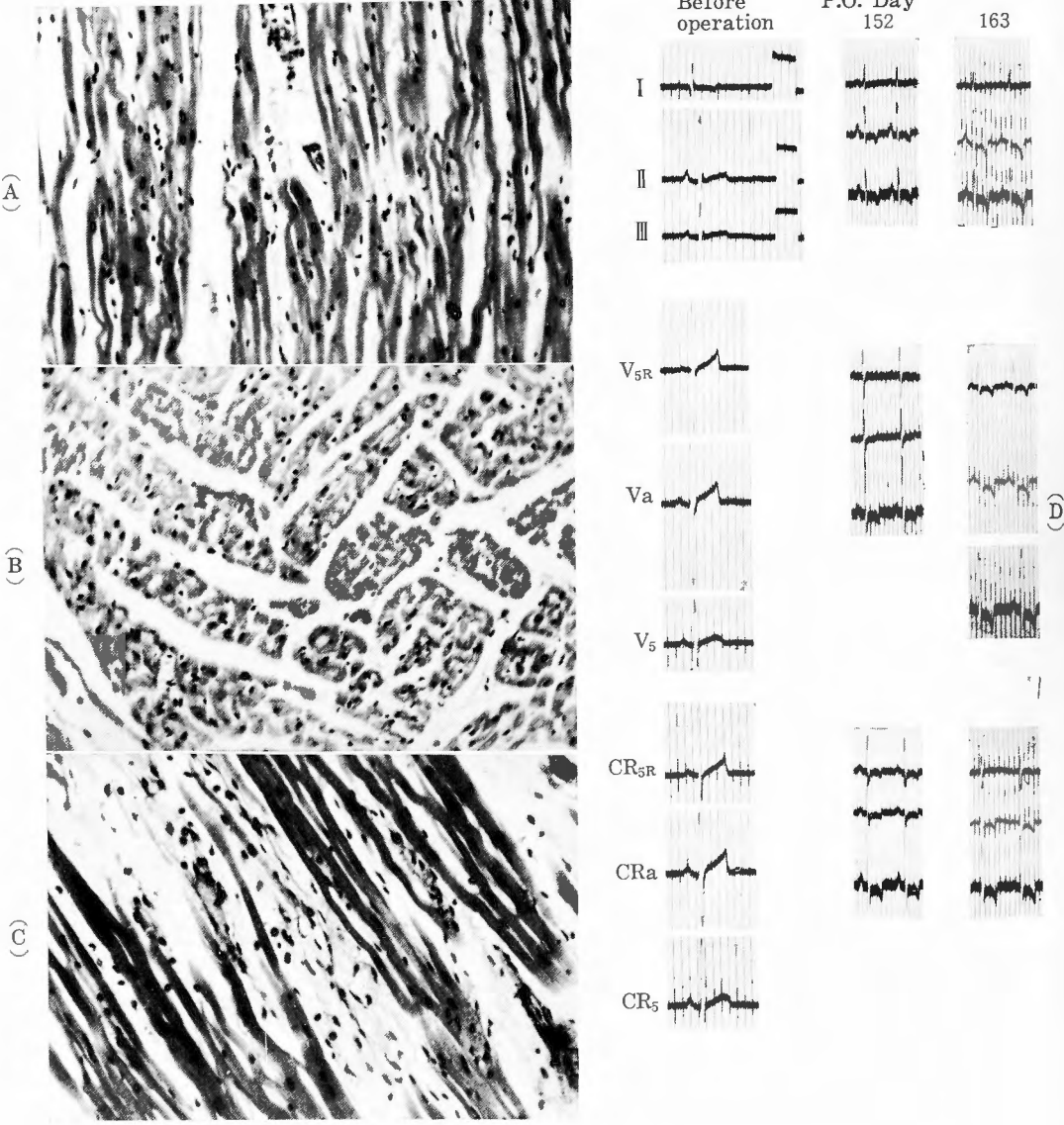
(A) : Interstitial edema, congestion and hemorrhage in the right ventricle. Hematoxylin and eosin; $\times 200$.



(B)

(B) : Note diminution of voltage.

Fig. 3 Constriction of the Whole Heart.
Myocardial changes and electrocardiographic abnormalities in
the third phase. (No. 194)



- (A) : Myocardial atrophy and interstitial inflammation in the left ventricle.
Hematoxylin and eosin; $\times 200$.
- (B) : Slight vacuolar degeneration in subendocardial muscle layers in left papillary muscle.
Hematoxylin and eosin; $\times 200$.
- (C) : Advanced myocardial atrophy and interstitial inflammation in the right ventricle.
Hematoxylin and eosin; $\times 200$.
- (D) : Note progressive diminution of voltage, and inversion of T waves.

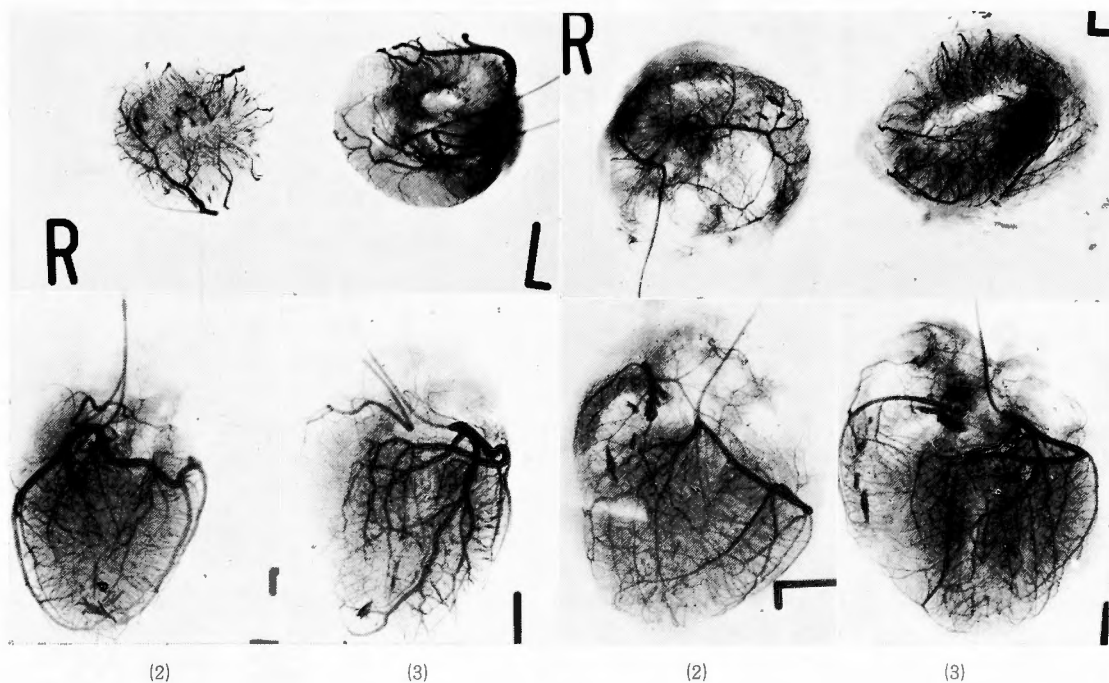
Fig. 4 Roentgenograms of coronary arteries filled with Bismuth-gelatin.

A: Control.

B: Constriction of the Whole Heart. (No. 215)

(1)

(1)

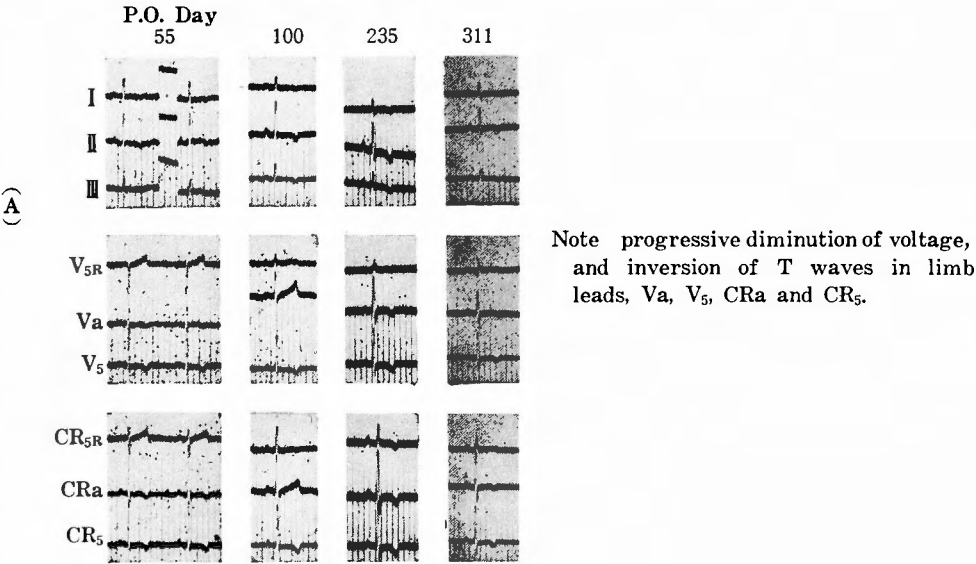


Direction of roentgen irradiation:

- (1) Basis→Apex. (The heart was intersected halfway between basis and apex cordis in all cases.)
- (2) Left→Right.
- (3) Front→Back.

Fig. 5

(A) : Constriction of the Left Side of the Heart. (No. 114)



(B) : Constriction of the Right Side of the Heart. (No. 110)

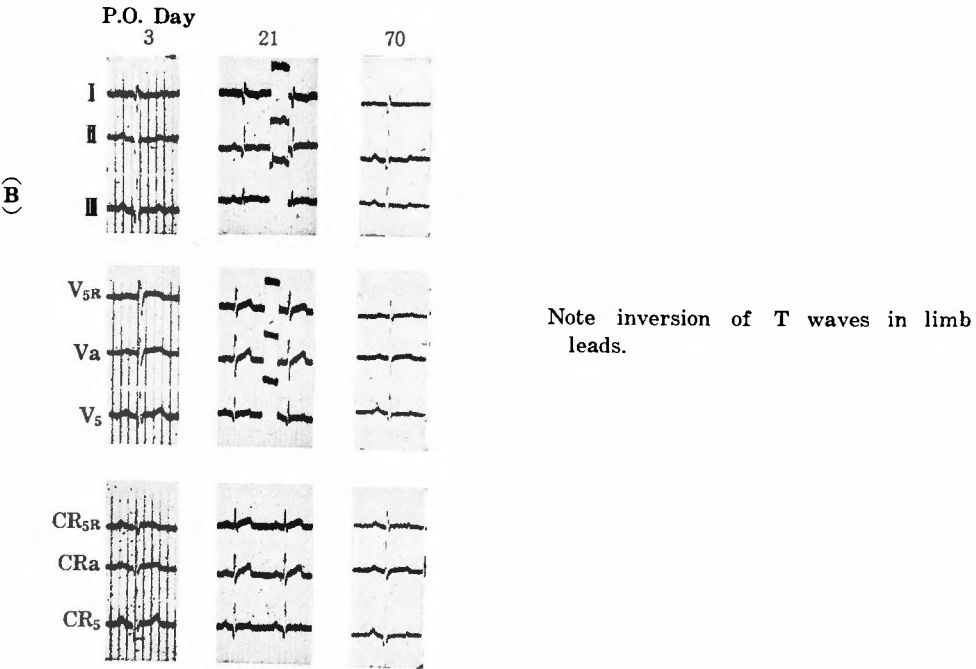
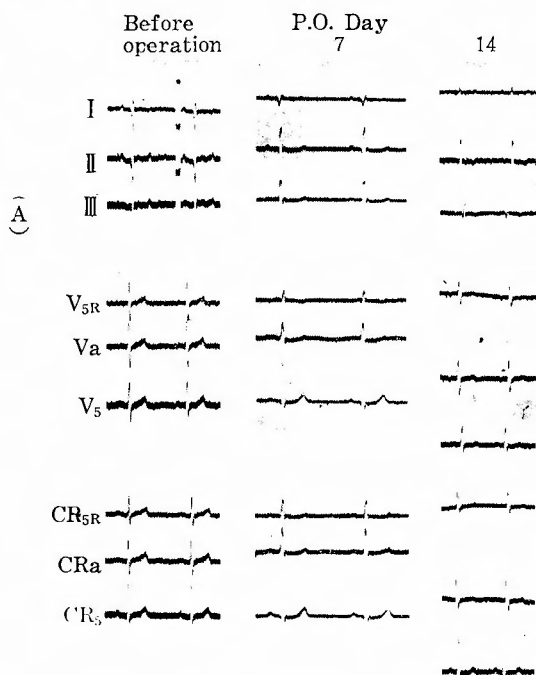


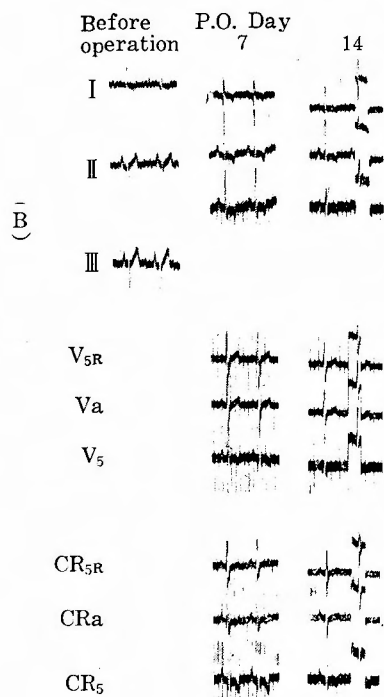
Fig. 6



(A) : Constriction of the Inferior Vena Cava (No. 181)

The inferior vena cava with outside diameter of 14 mm was constricted to 5 mm across.

Note reduction in amplitude of QRS waves, and appearance of inverted T waves in leads II and III.



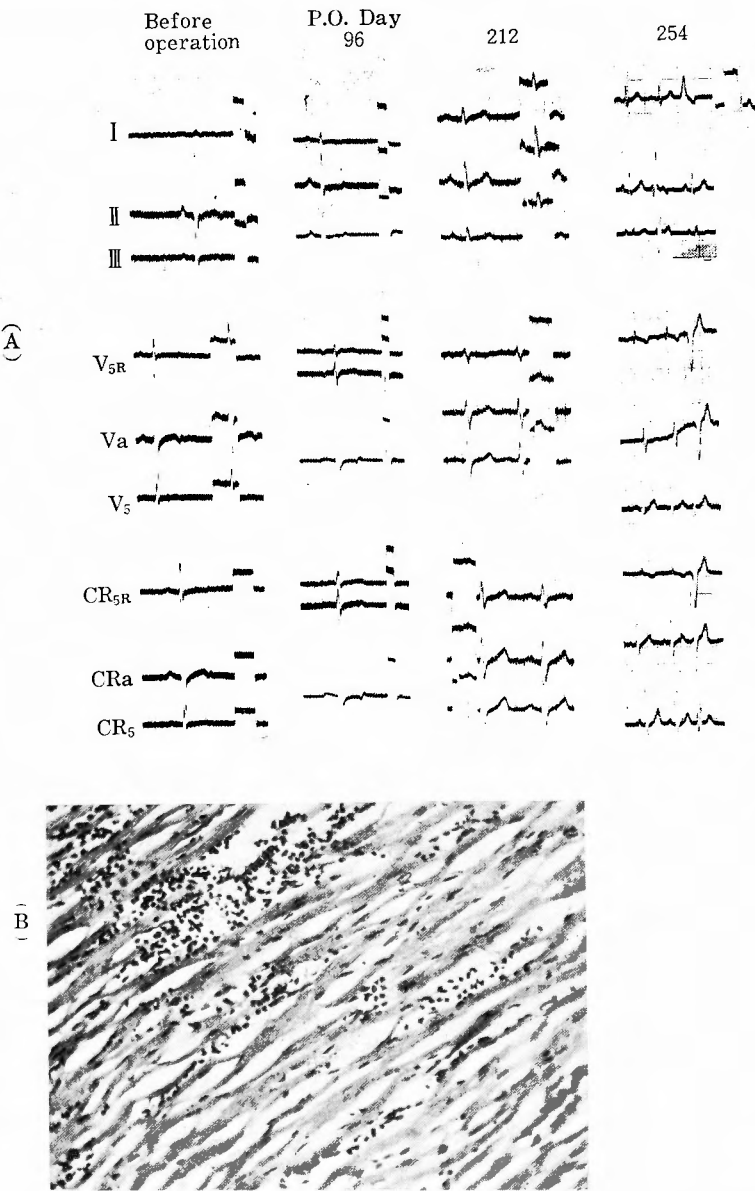
(B) : Constriction of the Inferior and Superior Venae Cavae. (No. 223)

Diameter of the superior vena cava was constricted from 15 mm to 10 mm, and that of the inferior from 10 mm to 6 mm, but the azygos vein was left free.

Note reduction in amplitude of QRS waves, and inversion of T waves in limb leads, V₅ and CR₅.

Fig. 7 Hydropericardium. (No. 152)

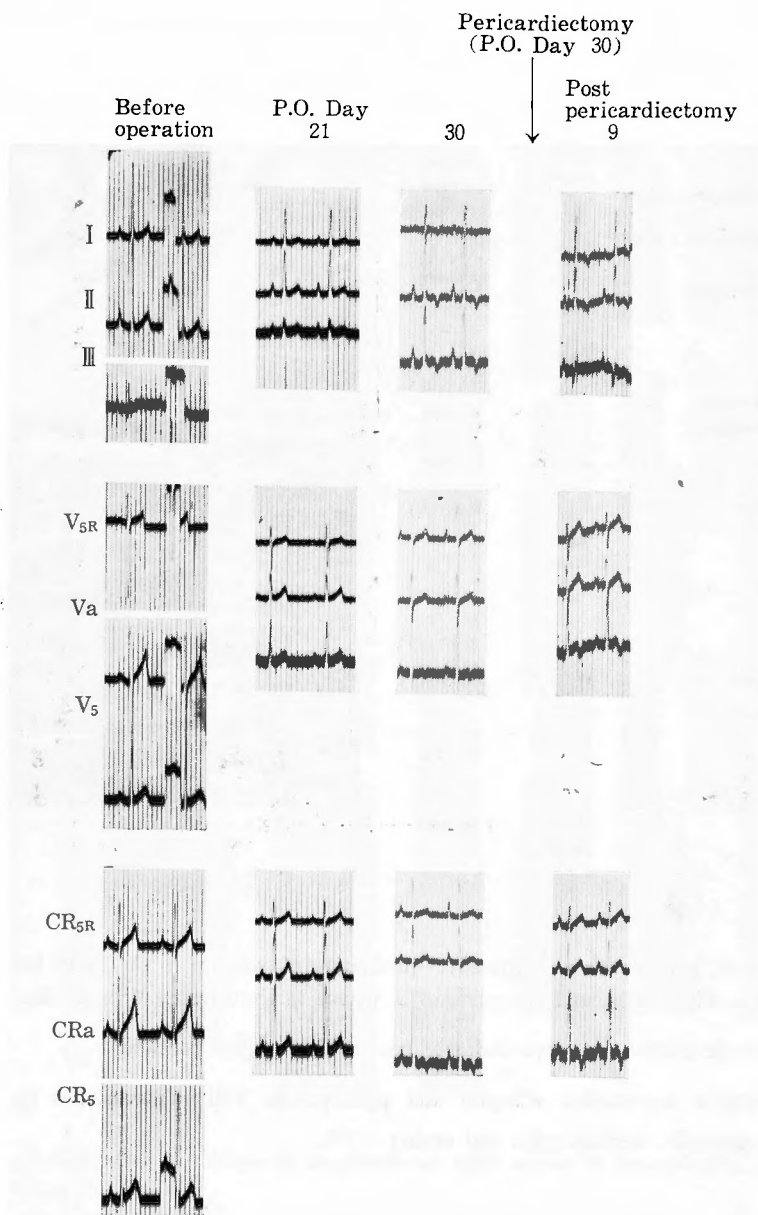
Entire pericardial cavity was inserted with polyvinylformal sponges ; at necropsy hydropericardium of unknown etiology (about 300-400cc) was noted.



(A) : Note progressive diminution of voltage in all limb and chest leads, and appearance of tachycardia and ventricular extrasystole 254 days after insertion of sponges.

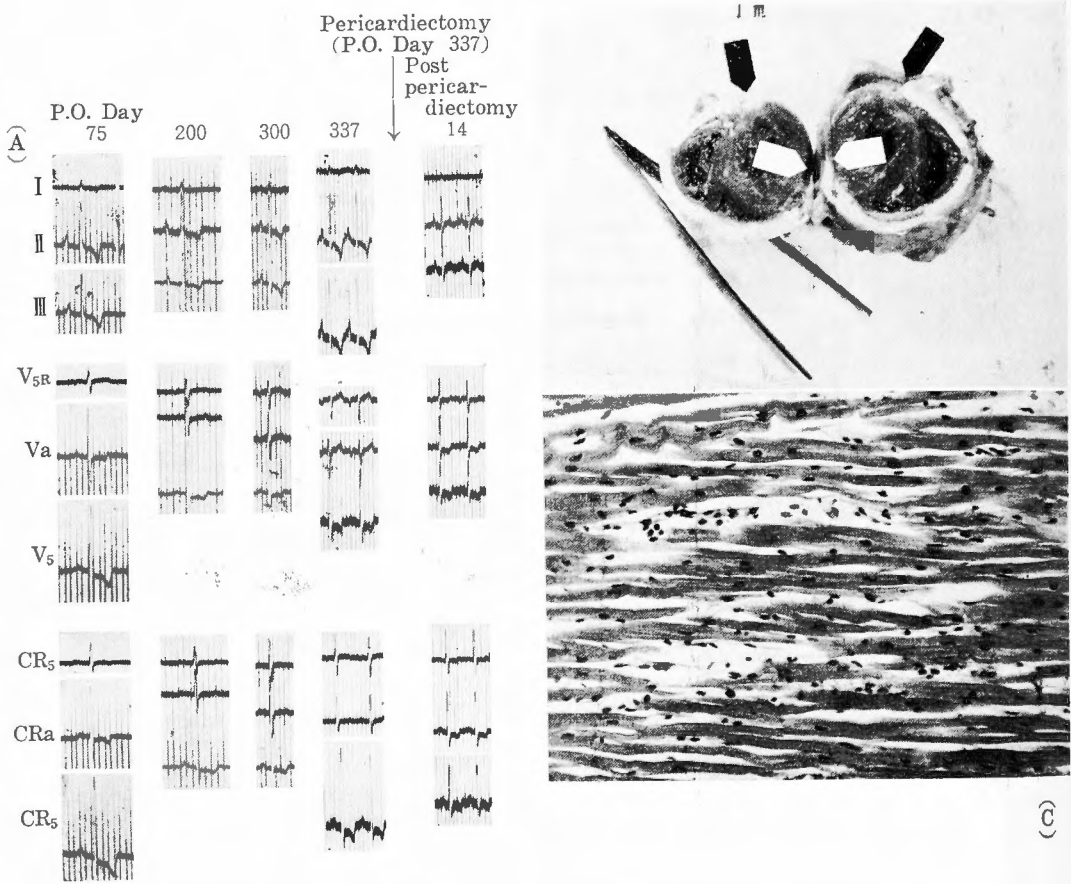
(B) . Pancarditis in the right ventricle. Hematoxylin and eosin ; $\times 200$.

Fig. 8 The First Case of Constriction of the Whole Heart which underwent Pericardiectomy. (No. 221)



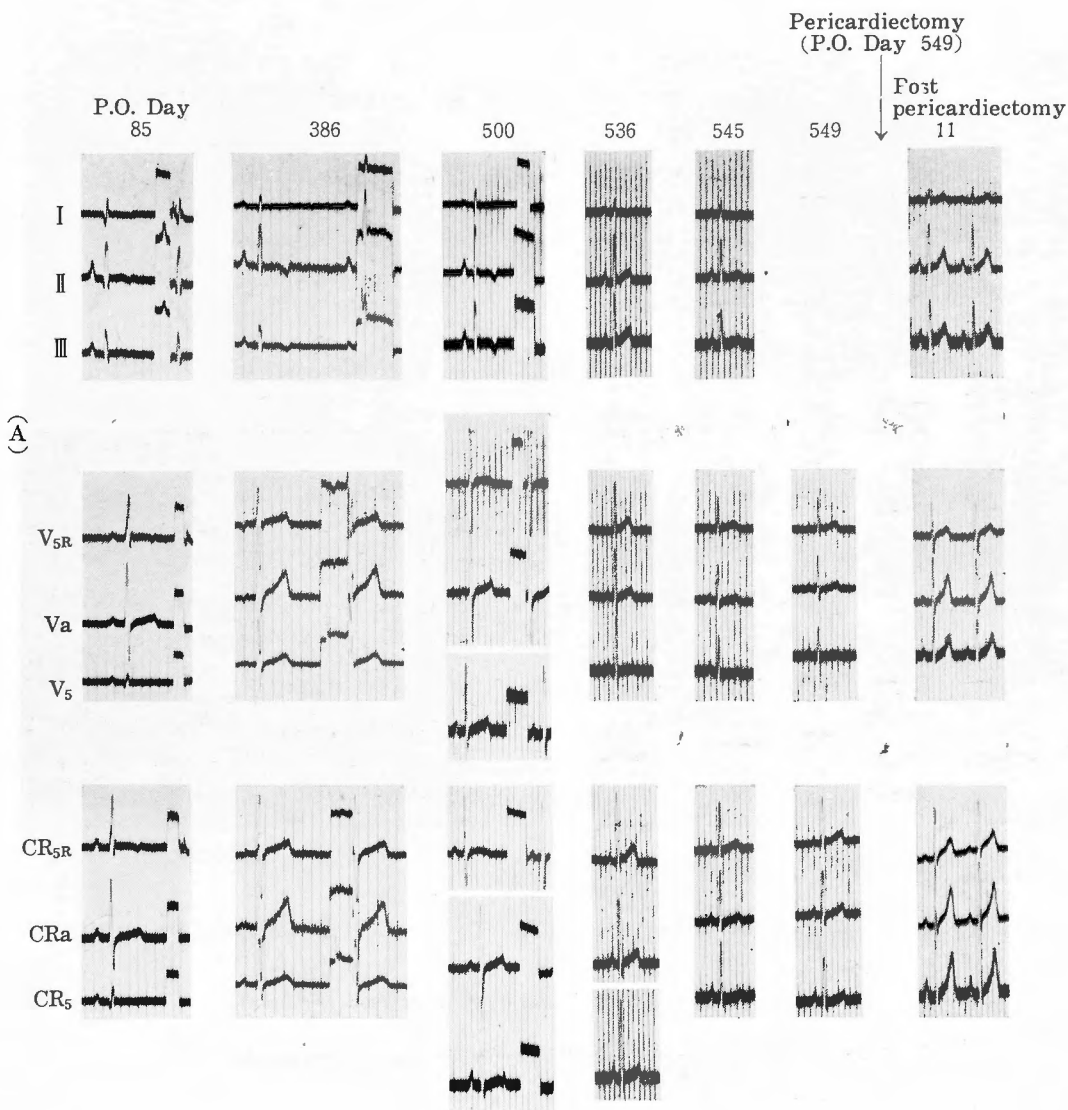
No abnormal signs except for inversion of T waves were noted in limb leads, V₅ and CR₅ before pericardiectomy.

Fig. 9 The Second Case of Constriction of the Whole Heart which underwent Pericardiectomy. (No. 163)



- (A) : Note preoperative progressive diminution of voltage in the chest leads of the right side, and inversion of T waves in limb leads, V₅ and CR₅.
- (B) : Cross section. Arrows indicate pericardiectomized portion.
- (C) : Slight myocardial atrophy and perivascular cell infiltration in the left ventricle. Hematoxylin and eosin ; ×200.

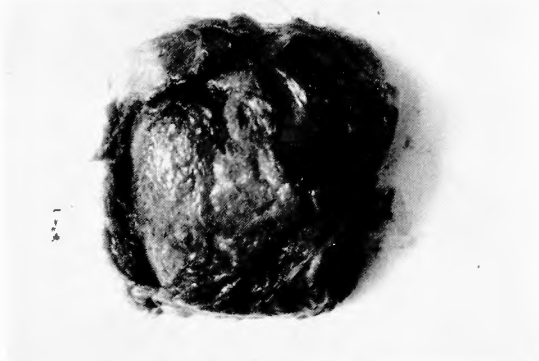
Fig. 10 The Third Case of Constriction of the Whole Heart which underwent Pericardiectomy. (No. 140)



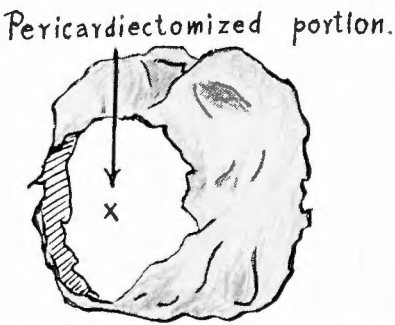
(A): Note progressive reduction in amplitude of QRS waves in preoperative period.

Fig. 10 The Third Case of Constriction of the Whole Heart which underwent Pericardiectomy. (No. 140)

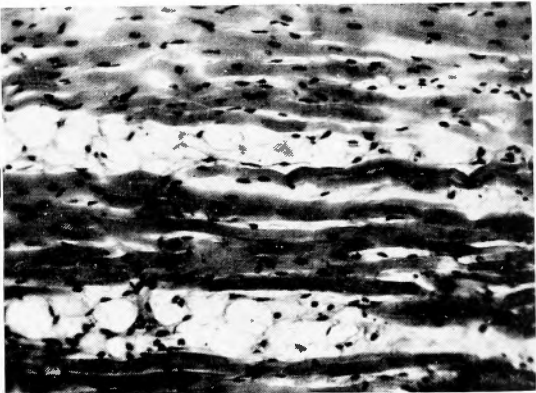
(B)



(C)



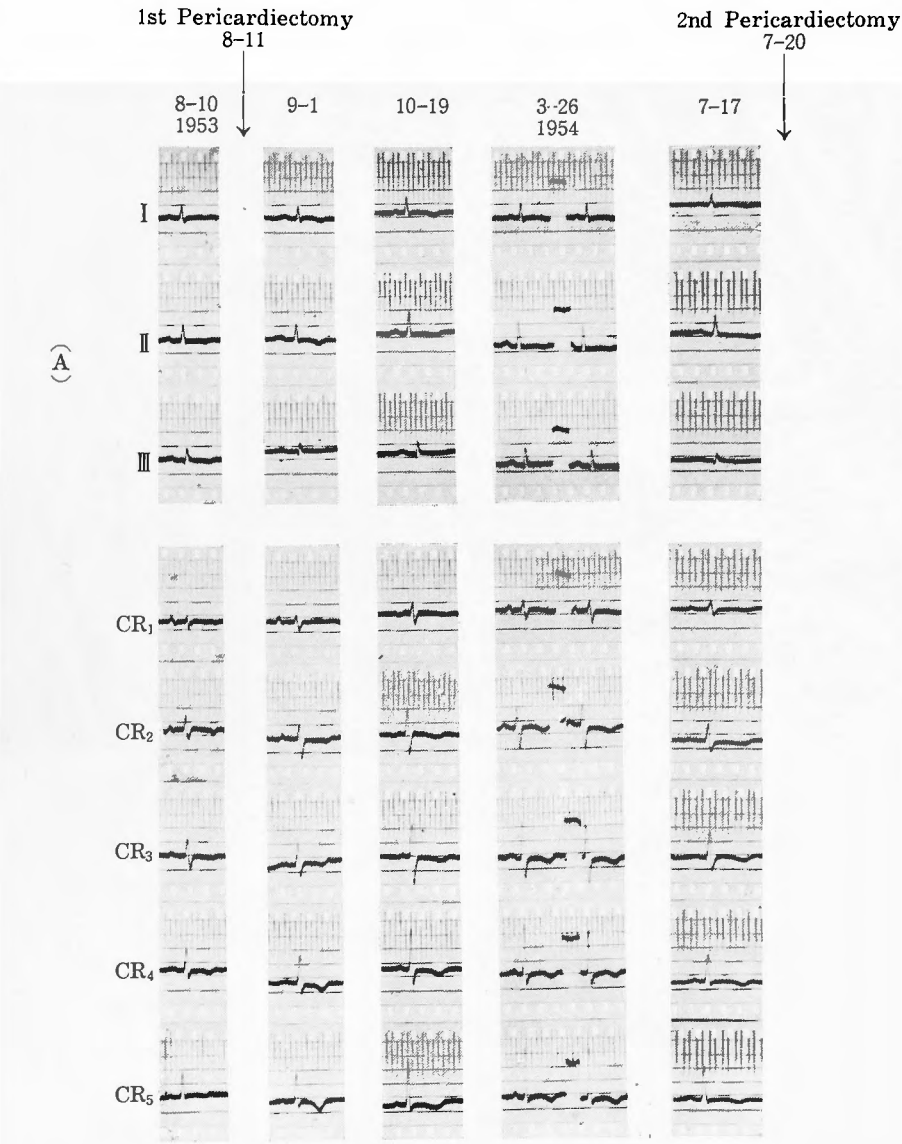
(D)



(E)

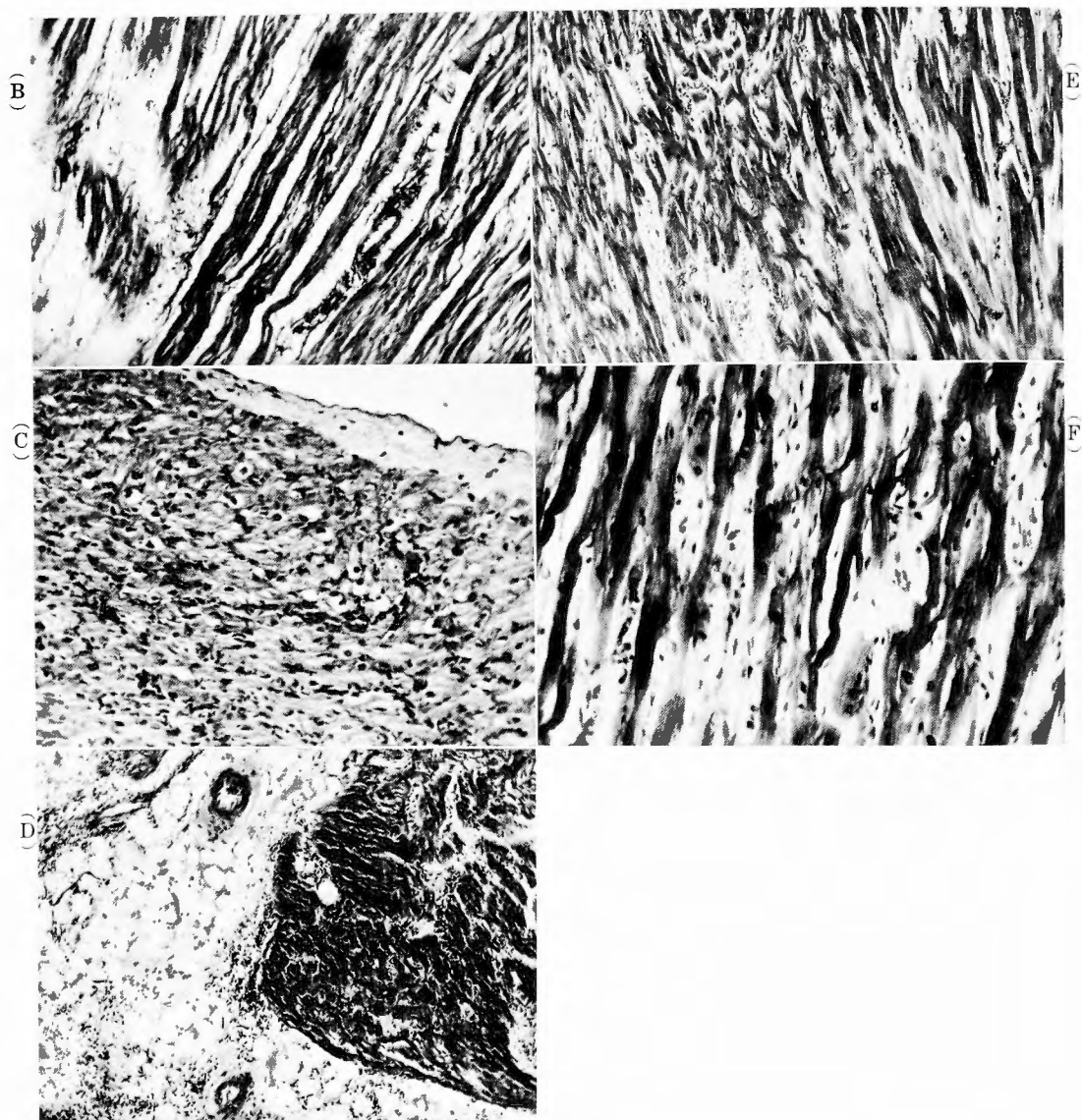
- (B) : Outside view.
- (D) : Cross section. Arrows indicate pericardiectomized portion.
- (E) Interstitial edema and fat proliferation in the right ventricle.
Hematoxylin and eosin; $\times 200$.

Fig. 11 Clinical Case 1, (K.H.)



(A) : Note low voltage and flattening or inversion of T waves in all limb and chest leads before first pericardiectomy.

Note that amplitude of QRS waves showed little or rather a tendency to decrease from Oct. 19th, 1953 onward.

Fig. 11 Clinical Case 1, (K.H.)

(B) : Myocardial atrophy and fibrinoid degeneration in the left ventricle.

Van Gieson's stain ; $\times 100$.

(C) : Vacuolar degeneration in subendocardial muscle layers in the right ventricle.

Hematoxylin and eosin ; $\times 200$.

(D) : Interstitial fat proliferation in the right ventricle. Hematoxylin and eosin ; $\times 100$.

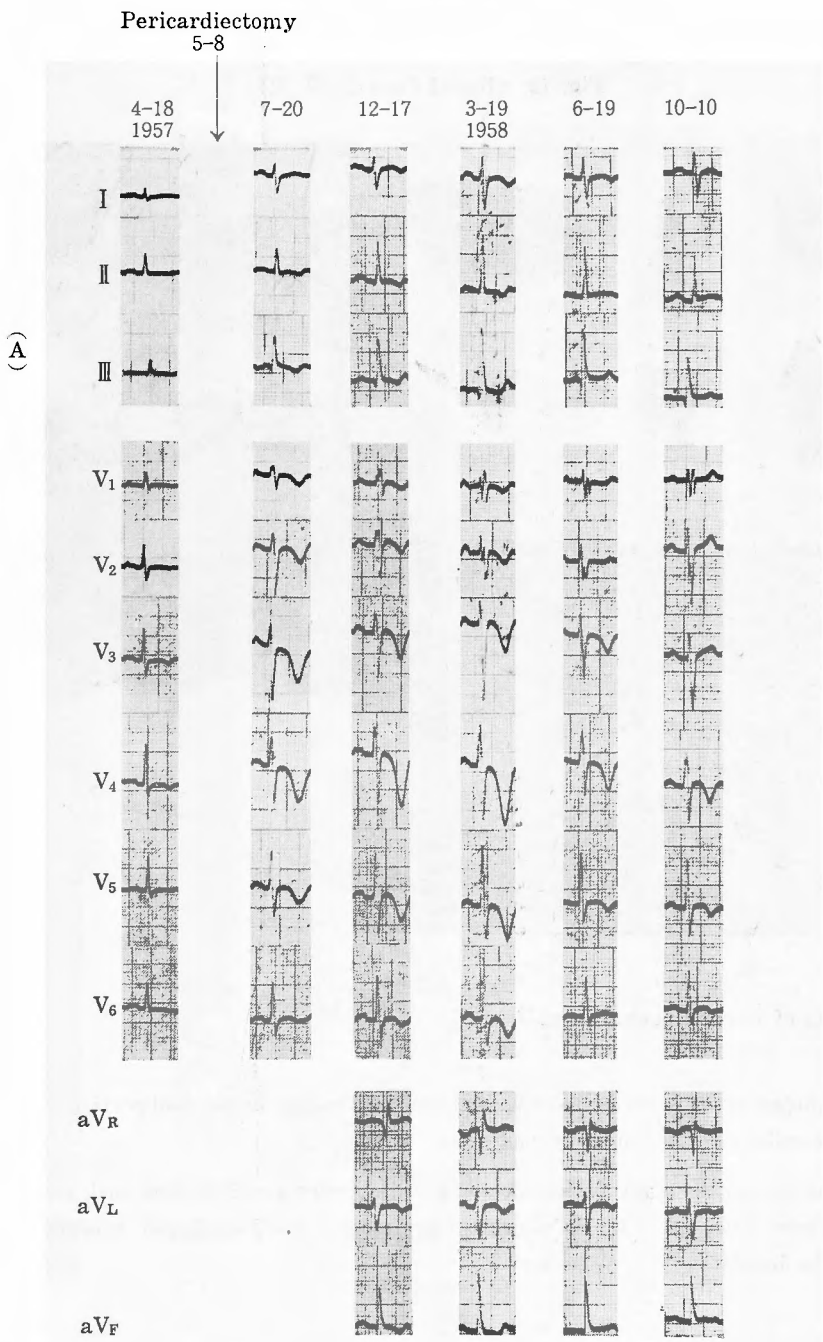
(E) : Hyaline and fibrinoid degeneration of left papillary muscle.

Hematoxylin and eosin ; $\times 200$.

(F) : Myocardial atrophy, and interstitial edema and cell infiltration in the left ventricle.

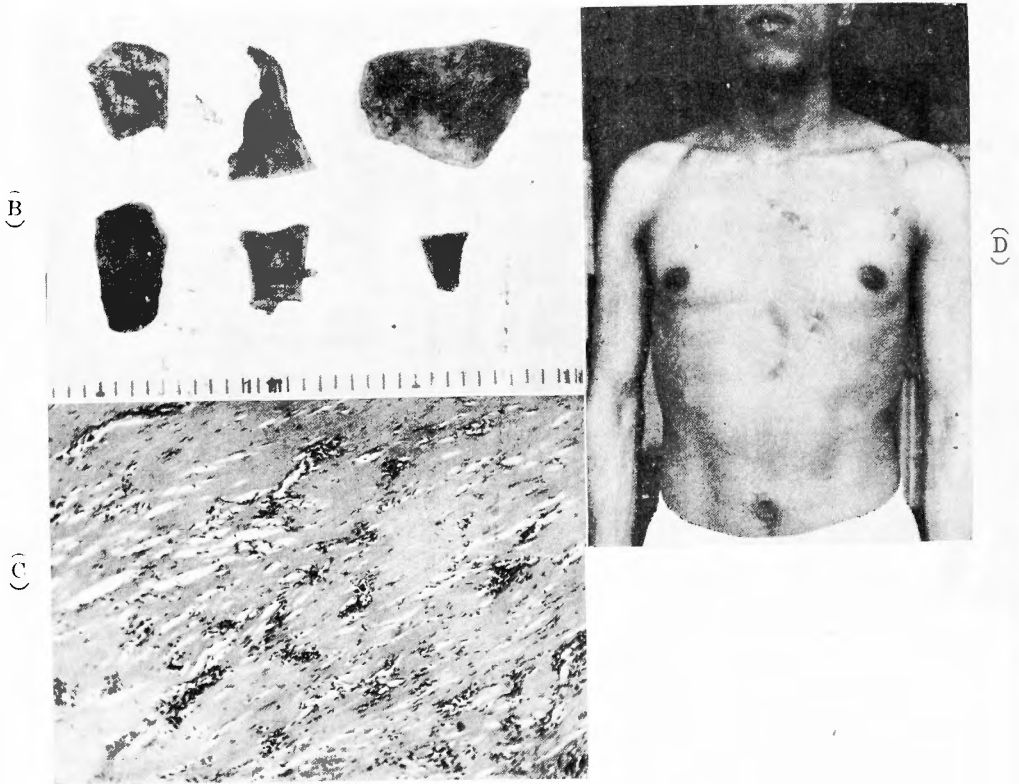
Hematoxylin and eosin ; $\times 200$.

Fig. 12 Clinical Case 2, (H.T.)



(A): Note preoperative low voltage, and flattening or inversion of T waves, and also postoperative gradual increase of voltage.

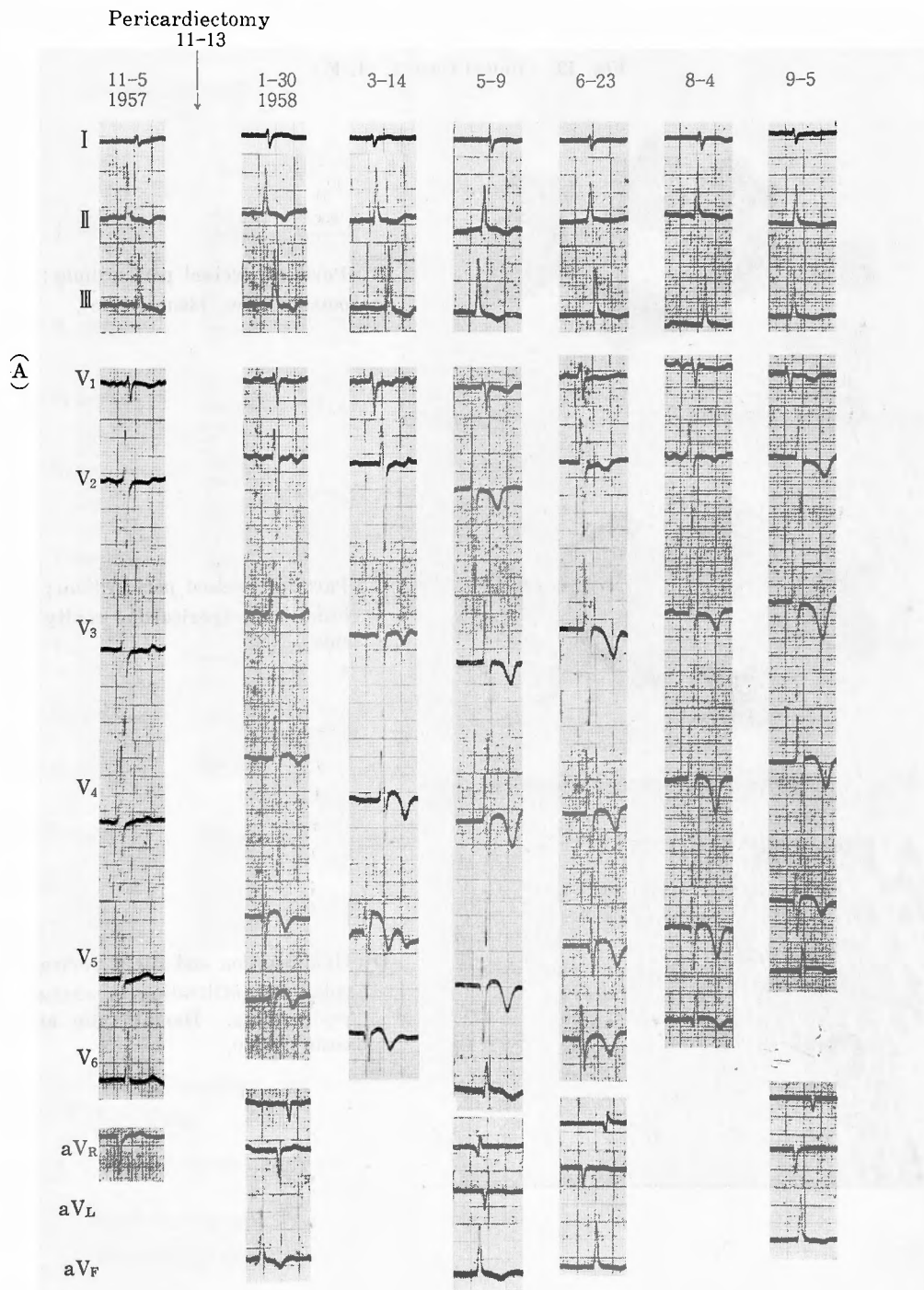
Fig. 12 Clinical Case 2. (H. T.)



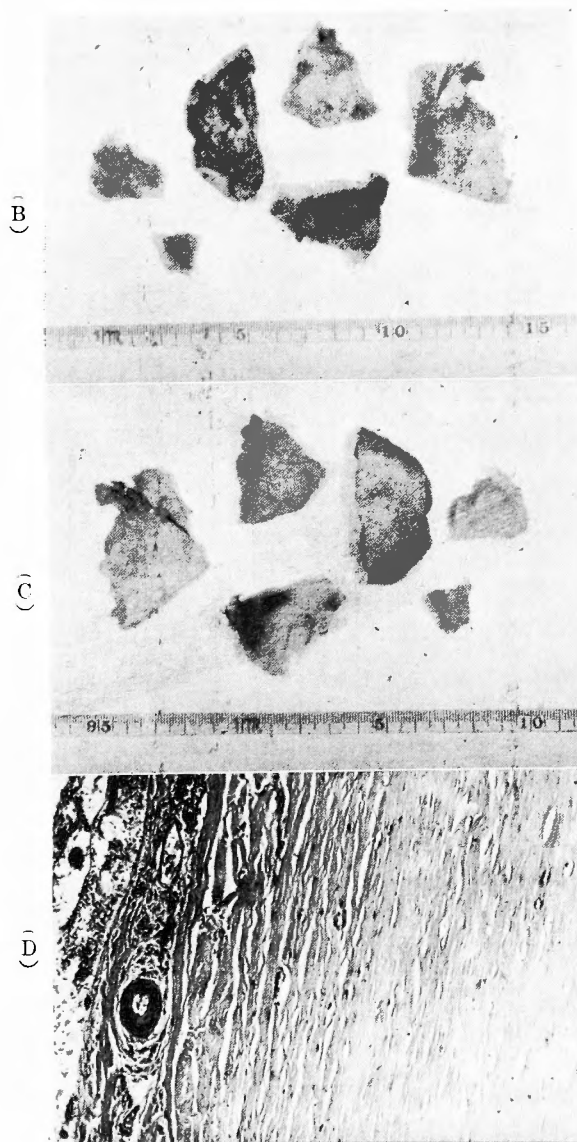
(B) : Parts of excised pericardium.

(C) : Hyalinization of connective tissue and cell infiltration in the mid-portion of excised pericardium. Hematoxylin and eosin; $\times 100$.

(D) : About 13 months after pericardiectomy: disappearance of ascites and abdominal swelling; liver not felt; no subjective symptoms; and bow-shaped operation scar on the breast.

Fig. 13 Clinical Case 3, (I.F.)

(A) : Note that no abnormal signs except for auricular fibrillation, and inversion of T waves in leads II and III, were preoperatively observed.

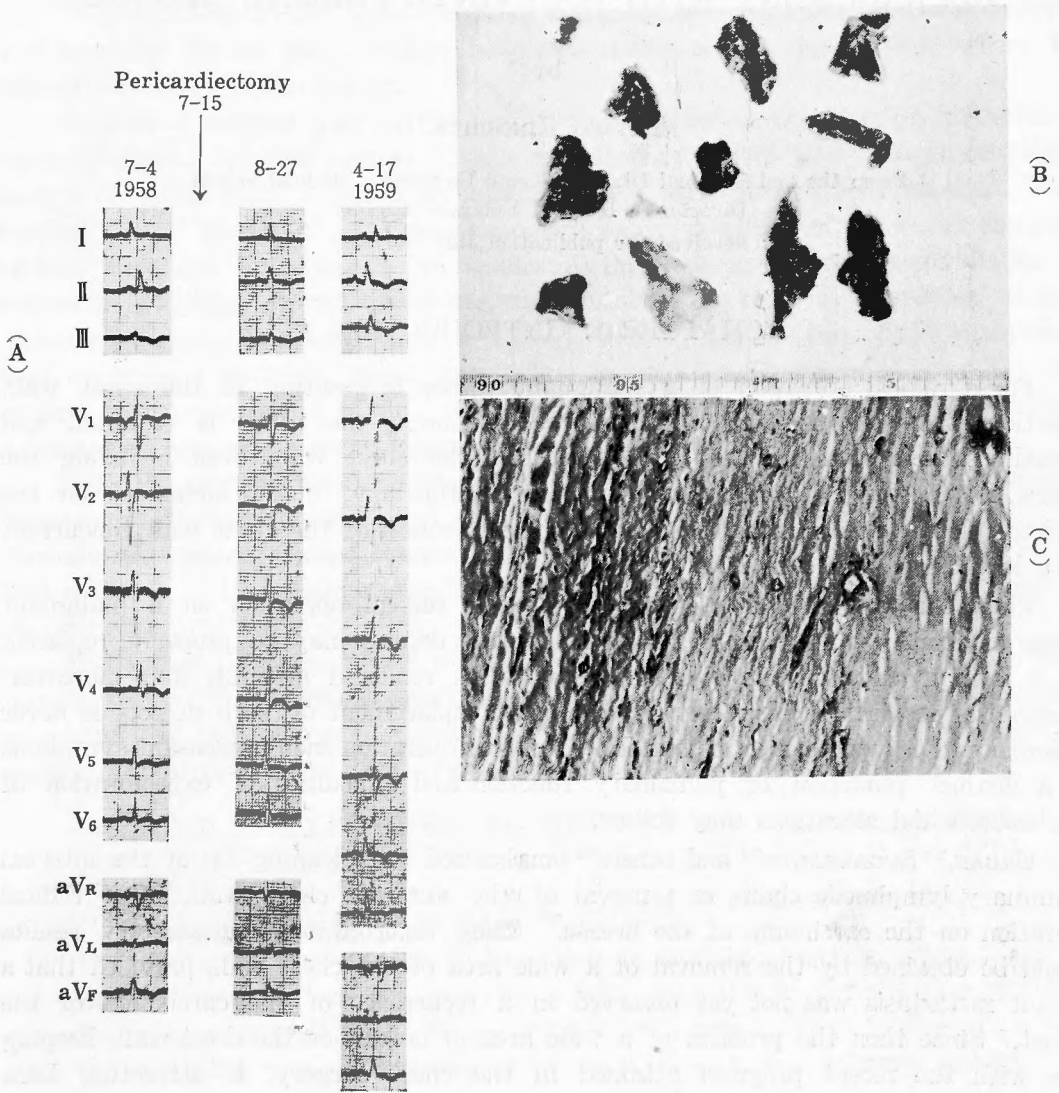
Fig. 13 Clinical Case 3, (I. F.)

(B) : Parts of excised pericardium ;
outside view (pleural side).

(C) : Parts of excised pericardium ;
inside view (pericardial cavity
side).

(D) : Hyalinization and slight perivas-
cular cell infiltration in excised
pericardium. Hematoxylin and
eosin ; $\times 100$.

Fig. 14 Clinical Case 4, (Y.U.)



(A) : Note preoperative low voltage and inversion of T waves in leads II, III, and all chest leads.

(B) : Parts of excised pericardium.

(C) : Hyalinization and slight cell infiltration in excised pericardium.
Hematoxylin and eosin; $\times 100$.