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MEASUREMENTS OF THE CIRCULATION IN CONSTRICTIVE PERICARDITIS BEFORE AND AFTER RESECTION OF THE PERICARDIUM¹

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Experience is showing that chronic constrictive pericarditis is not an uncommon syndrome. Attention has been directed to it again in recent years. Its recognition has been facilitated by White's (1) historical resumé and analysis of its clinical features. It is of therapeutic importance to recognize this syndrome for it is a cardiac affection that lends itself to surgical treatment as the experience of Churchill (2), Beck and Griswold (3), Blalock (4), and of Stewart and Heuer (5), as well as others, has demonstrated. The clinical manifestations of this disease have been very well described; the pathological physiology of the circulation has not, however, been sufficiently explored.

In the last two and a half years, we have observed 9 patients suffering from chronic constrictive pericarditis, and in six of these part of the pericardium has been resected by Dr. George J. Heuer.² In them, studies of the circulation have been made before as well as after partial pericardiectomy. One clinical group does not have the opportunity to see large numbers of these patients in a short time, and for this reason this paper records our studies of the circulation in this situation, together with a statement of our experience with surgical treatment.

PLAN OF STUDY

All patients were admitted to the hospital and remained in bed. The daily fluid intake was limited to 1200 cc., and the salt to 2.0 grams. A high protein diet was given.

Studies of the circulation were made when the patients first came under observation and before we in-

stituted drug therapy. Diuretics were then administered and studies were repeated, this time when they were in the best state it was possible for them to attain. Resection of the pericardium was then done. As soon after operation as the patients were able to participate, observations were repeated, as well as later when changes in the clinical state occurred. The patients were discharged from the hospital free of excess fluid and followed in the Cardiac Outpatient Clinic, from which they were readmitted to the hospital at intervals for repetition of the studies of the circulation.

METHODS

All observations were made in the morning while the patients were in a basal metabolic state. Measurements of the cardiac output were made by the acetylene method, three samples of gas being taken as first recommended by Grollman (6), and by Grollman, Friedman, Clark, and Harrison (7). During this measurement the patients were sitting in a steamer chair (angle 135 degrees). They were trained beforehand to carry out the procedures. While the patient was at rest, the cardiac rate was counted at intervals of five minutes. At the end of one-half hour the acetylene-air-oxygen mixture was rebreathed. Three samples of gas were taken during each rebreathing period for estimation of the arteriovenous oxygen difference. The first sample was taken after rebreathing 10 to 12 times in 20 seconds, the second after 2 to 3 breaths more, and the third after 2 to 3 additional breaths. All three samples were usually obtained before the end of 30 seconds. Samples were taken during expiration. Two to three periods of rebreathing were carried out on each patient. Shortly afterward, the oxygen consumption was measured with a Benedict-Roth spirometer. After a short pause, the vital capacity was measured, and height and weight recorded. In succession, sufficient time being allowed between each procedure for the patient to return to a basal metabolic state, an electrocardiogram was taken, the arm to tongue circulation time recorded, the venous pressure estimated, and the blood pressure measured; finally an x-ray photograph of the heart was made at a distance of two meters.

The arm to tongue circulation time was estimated by the use of decholin (8). Five cc. of a 20 per cent solution were injected rapidly (1 to 2 seconds) through an 18-gauge needle into an antecubital vein while the patient was lying quietly in the supine position. This was re-

¹ An abstract of these studies was read before the Association of American Physicians, Atlantic City, May 5, 1937.

² Since this paper went to press the pericardium was resected from another patient, making seven cases in all (5).

peated in one and one-half minutes after the response to the first test had been elicited. The time was recorded from the beginning of the injection until the patient perceived the bitter taste.

The venous pressure was measured by the direct method (9), using a large antecubital vein, the arm being placed on a level with the right auricle. Normal pressures by this method range from 4.0 to 10.0 cm. of saline. The antecubital vein of one arm was reserved for the injection of decholin and of the other arm for the measurement of venous pressure. In subsequent measurements the vein was entered at the site first punctured.

X-ray photographs of the heart were taken with the patient in the standing position, in full inspiration, at a distance of two meters.³ Measurements of the cardiac area were carried out by the technique of Levy (10) and estimations of volume were made as recommended by Bardeen (11). Special x-ray exposures in the antero-posterior as well as in the lateral positions were taken for the detection of calcification. Examination under the fluoroscope was also carried out. In certain patients photographs of the eyegrounds were made for definition of the vessels. Infra-red photographs of the patients were taken to record the state of the peripheral veins. The patients assumed as nearly as possible exactly the same position for each observation in order to assure uniformity from this point of view. In addition, each procedure in the observation was carried out by the same investigator.

The six patients who were operated upon and in whom observations were made both before and after partial pericardiectomy form the subject of this paper.

RESULTS

The data are recorded in Tables I and II and certain of the data are summarized in Figure 1.

The arteriovenous oxygen difference before operation was increased in all except one (Case 6, 59.7), the range being from 71.5 to 88.6 cc. After operation when the patients were in their best state, it decreased in all patients and only one fell outside of the normal range, which was then 51.4 to 68.7 cc.

The cardiac output per minute and cardiac index⁴ were decreased in all except one patient (Case 6, 2.16 liters), the range of the index being 1.35 to 1.82 liters. After operation it increased and ranged between 1.80 to 2.72 liters, and was below normal in only one (Case 4).

³ The authors are deeply indebted to the X-ray Department of the New York Hospital for their cooperation in this investigation.

⁴ Cardiac index = liters per square meter per minute.

The stroke volume was decreased and the range was from 20 to 42 cc. per beat. After operation it increased and ranged from 33 to 50 cc. per beat.

The venous pressure was elevated in every case, the range being 17.9 to 24.0 cm. After operation it fell and when the patients were in their best state the range was 8.3 to 16.7 cm.

The arm to tongue circulation time ranged from 13.5 to 29.8 seconds before operation; in short, it was prolonged. After operation the range was 7.3 to 17.1 seconds when the patients were in their best state.

There was no consistent behavior of the heart rate. In certain patients it was elevated before operation and slowed afterward, and in others the reverse happened. The basal metabolic rate was not altered significantly in this syndrome, nor was it changed by operation.

The vital capacity before operation was not lowered if the pleural cavities were free of fluid. In certain patients it decreased and in others it increased after operation. Decrease after operation was in part due to the flexible thoracic cage resulting from removal of the ribs.

Infra-red photographs revealed marked distention of and increase in the number and caliber of the venous channels before operation. As improvement occurred after operation there was progressive decrease in their number and caliber (Figure 2).

CLINICAL COURSE OF PATIENTS

After operation there were 3 trends: (1) Clinical improvement was rapid and striking, and associated with this were changes toward normal of the measurements of the circulation (Tables I and II, Figure 1). In two patients "cure" was a matter of months (Cases 1 and 2). (2) In one (Case 3), clinical improvement was slow and gradual, to cure in approximately 1 year after operation (Tables I and II); in her, the measurements of the circulation showed gradual changes. (3) Three patients improved gradually after operation; their condition has now become stationary (Cases 4, 5, and 6). They are better than before the operation and are ambulatory, and there have been changes in the circulation toward normal (Tables I and II).

TABLE I
Data relating to 6 patients suffering from chronic constrictive pericarditis

Case number; Initials; History number; Age; and Sex	Date	Height cm.	Weight kym.	Oxygen consumption cc. per min- ute	Arteriovenous oxygen difference cc.	Cardiac output liters per sq. m. per minute	Cardiac output liters per sq. m. per minute	Heart rate per min- ute	Stroke volume cc.	Arterial pressure mm. Hg	Circulation time seconds	Venous pressure cm.	Vital capacity cc.	Digitalis grains	Other drugs	Evidence of congestive heart failure ^a							Hemoglobin per cent	Red blood count mil- lions	Paradoxical pulse ^b		
																Dyspnea	Cyanosis	Rales	Liver	Ascites	Edema	Pleural effusion					
Case 1 A. B., No. 28323 ^a 16 years ♀	May 10, 1936	150.5	40.9	160	71.7	2.23	1.70	112	20	93/84	15.4	24.0	1900	0	0	Aminophyllin 0.1 gm. t.i.d.	0	0	0	++	++	0	0	0	76	3.8	++
	June 2, 1936	151.0	37.0	141	66.5	2.12	1.70	78	27	82/70	15.5	18.3	1850	0	0		0	0	0	++	++	0	0	0	80	4.3	++
	Operation on June 5, 1936																										
	June 8, 1936	150.7	34.7	139	53.1	2.67	2.20	84	32	85/60	10.4	9.6	1350	0	0		0	0	0	++	++	0	0	0	80	4.8	0
	June 23, 1936	151.0	34.7	143	48.5	3.00	2.42	88	34	86/55	10.4	11.4	1500	0	0		0	0	0	++	++	0	0	0	92	4.1	++
	Oct. 20, 1936	151.0	39.7	153	43.3	3.53	2.70	90	39	90/68	10.1	7.8	1800	0	0		0	0	0	++	++	0	0	0	100	4.9	++
Case 2 W. M., No. 103699 36 years ♂	Apr. 6, 1937	151.0	44.9	193	51.1	3.78	2.72	93	41	100/60	7.3	8.3	1600	0	0		0	0	0	++	++	0	0	0	112	5.8	++
	Sept. 10, 1935	172.5	56.7	199	51.9	3.53	2.28	96	40	104/70	21.3	12.0	3200	0	0	Theocalcin 1.5 gm. t.i.d.	0	0	0	++	++	0	0	0	112	5.8	++
	Sept. 17, 1935	172.5	58.2	198	62.5	3.18	1.87	88	36	94/64	26.3	14.3	3100	0	0		0	0	0	++	++	0	0	0	112	5.8	++
	Sept. 21, 1935	172.5	58.7	198	66.6	3.00	1.72	88	34	94/60	22.0	14.8	2900	0	0	Theocalcin 1.5 gm. t.i.d.	0	0	0	++	++	0	0	0	112	5.8	++
	Sept. 26, 1935	172.5	58.0	209	53.9	3.88	2.28	86	45	99/68	20.0	11.0	3500	0	0	Theocalcin 1.5 gm. t.i.d.	0	0	0	++	++	0	0	0	112	5.8	++
	Sept. 30, 1935	172.5	57.2	214	54.5	3.93	2.38	84	47	102/64	19.4	7.3	3800	0	0	Theocalcin 1.5 gm. t.i.d.	0	0	0	++	++	0	0	0	112	5.8	++
	Oct. 7, 1935																										
	Oct. 13, 1935																										
	Nov. 1, 1935	172.4	60.3	222	71.0	3.13	1.82	86	36	99/68	22.3	15.7	3200	0	0	Theocalcin 1.0 gm. t.i.d.	0	0	0	++	++	0	0	0	92	5.0	++
	Nov. 11, 1936	172.8	59.5	203	74.2	2.74	1.60	70	39	115/90	18.4	21.0	1800	0	0	Theocalcin 1.0 gm. t.i.d.	0	0	0	++	++	0	0	0	92	5.0	++
Case 3 A. R., No. 91648 30 years ♀	Operation on Nov. 25, 1936																										
	Dec. 5, 1936																										
	Dec. 11, 1936	172.0	54.7	187	56.3	3.51	2.14	72	49	95/68	20.2	13.8	1800	0	0		0	0	0	++	++	0	0	0	102	5.1	++
	Dec. 29, 1936	172.5	57.2	199	53.4	3.73	2.25	72	52	105/78	21.8	14.8	2500	0	0		0	0	0	++	++	0	0	0	106	5.3	++
	Apr. 1, 1937	172.0	53.8	193	53.8	3.59	2.19	80	45	95/75	16.7	7.1	1900	0	0		0	0	0	++	++	0	0	0	106	5.3	++
	Jan. 28, 1938																										
	Dec. 20, 1935	154.7	42.7	189	88.6	2.14	1.55	74	29	92/66	20.7	20.8	1500	0	0	A.C. 1.0 gm. t.i.d.	0	0	0	++	++	0	0	0	80	4.3	++
	Jan. 27, 1936	154.0	45.9	201	81.2	2.48	1.76	100	25	92/70	15.0	21.5	1500	0	0	A.C. 1.0 gm. t.i.d.	0	0	0	++	++	0	0	0	80	4.3	++
	Operation on Jan. 28, 1936																										
	Jan. 29, 1936																										
Case 4 J. McC., No. 128763 20 years ♂	Feb. 4, 1936	153.0	47.7	186	85.0	2.19	1.52	120	20	110/82	13.7	20.3	1000	0	0		0	0	0	++	++	0	0	0	77	3.9	++
	Feb. 13, 1936	153.0	46.8	184	66.7	2.76	1.94	96	29	100/75	15.5	29.0	900	0	0		0	0	0	++	++	0	0	0	77	3.9	++
	Feb. 15, 1936	153.5	46.8	185	194	2.65	1.84	84	32	96/64	10.2	27.8	22.7	0	0		0	0	0	++	++	0	0	0	77	3.9	++
	Feb. 18, 1936	153.5	47.5	195	73.5	2.65	1.84	84	32	96/64	10.2	22.7	22.7	0	0		0	0	0	++	++	0	0	0	77	3.9	++
	Apr. 27, 1936	154.0	48.4	183	71.8	2.54	1.70	88	29	105/75	9.9	22.0	950	0	0		0	0	0	++	++	0	0	0	82	4.4	++
	Dec. 9, 1936	154.0	49.9	197	57.2	3.44	2.34	80	43	108/72	9.0	19.3	1400	0	0		0	0	0	++	++	0	0	0	84	4.3	++
	Mar. 9, 1937	154.5	51.5	184	54.9	3.35	2.25	80	42	105/75	14.4	9.8	2000	0	0		0	0	0	++	++	0	0	0	84	4.3	++
	Mar. 18, 1937	153.5	51.5	184	54.9	3.35	2.25	80	42	105/75	14.4	9.8	2000	0	0		0	0	0	++	++	0	0	0	84	4.3	++
	Nov. 30, 1937	154.5	50.3	176	70.3	2.70	1.84	88	31	100/60	17.6	6.0	2800	0	0		0	0	0	++	++	0	0	0	80	4.5	0
	Dec. 16, 1937	155.2	49.7	181	62.4	2.90	2.00	80	36	90/65	12.4	4.8	2850	0	0		0	0	0	++	++	0	0	0	87	4.1	0
Case 4 J. McC., No. 128763 20 years ♂	Nov. 19, 1936	173.0	61.3	228	81.2	2.81	1.63	96	29	98/70	24.0	14.5	1400	0	0		0	0	0	++	++	0	0	0	104	5.7	++
	Dec. 8, 1936	174.0	61.5	205	81.7	2.51	1.44	96	26	100/70	24.0	14.0	1400	0	0		0	0	0	++	++	0	0	0	100	5.2	++
	Operation on Dec. 21, 1936																										
	Feb. 3, 1937	173.5	61.4	218	76.6	2.84	1.57	102	28	105/80	20.0	20.5	1300	0	0	A.C. 1.0 gm. t.i.d.	0	0	0	++	++	0	0	0	100	5.0	++
	Feb. 23, 1937	173.5	59.4	207	68.7	3.01	1.80	90	33	90/70	22.1	22.0	2000	0	0	A.C. 1.0 gm. t.i.d.	0	0	0	++	++	0	0	0	110	5.6	++
	Mar. 29, 1937	173.5	59.4	207	68.7	3.01	1.80	90	33	90/70	22.1	22.0	2000	0	0	A.C. 1.0 gm. t.i.d.	0	0	0	++	++	0	0	0	110	5.6	++
Jan. 29, 1938 ^a	171.8	65.1	232					88		110/80	21.5	18.8	1700	0	0	Urea, 10.0 gm. t.i.d.	0	0	0	++	++	0	0	0	110	5.6	++

TABLE I—Continued

Case number: Initials; History number; Age; and Sex	Date	Height; cm.	Weight; kgm.	Oxygen consumption; cc. per min.	Arteriovenous oxygen difference; cc.	Cardiac output; liters per min.	Cardiac output; cc. per min.	Heart rate; per min.	Stroke volume; cc.	Arterial pressure; mm. Hg	Circulation time; seconds	Venous pressure; cm.	Vital capacity; cc.	Digitalis; grains	Other drugs	Evidence of congestive heart failure ³	Red blood count; millions per cmm.	Hemoglobin; per cent ⁴	Paradoxical pulses ⁵
Case 5 P. A., No. 141237 44 years ♀	Aug. 21, 1936	156.0	64.0	184	83.1	2.22	1.35	66	34	122-116/78	18.6	17.9	1660	0.1 q.d.	None		5.1	122	++
	Sept. 22, 1936	156.0	57.0	191	71.5	2.09	1.71	78	34	126/72	18.4	13.8	2280	0.1 q.d.			5.3	104	++
	Operation on Sept. 28, 1936																		
	Nov. 12, 1936	154.7	43.9	172	61.8	2.78	2.01	64	43	110/70	13.3	16.1	1200	0.1 b.i.d.	A.C. 1.0 gm. t.i.d.		4.0	78	++
	Dec. 10, 1936	154.7	57.7	209	75.3	2.78	1.76	80	36	126/68	11.2	11.3	1400	0.1 b.i.d.	A.C. 1.0 gm. t.i.d.		4.3	96	++
	Mar. 31, 1937	155.5	57.7	203	64.3	3.16	2.02	84	38	100/80	16.7	15.3	760	0.1 b.i.d.	Ammonophyllin 0.1 gm. t.i.d. Urea, 15.0 gm. t.i.d.				
Case 6 J. S., No. 109168 38 years ♂	May 26, 1937	166.5	104.3	278	59.7	4.66	2.20	112	42	130/75	12.5	20.1	2000	0	Theocalcin 1.5 gm. t.i.d.		5.5	110	++
	June 15, 1937	166.0	92.3	251	58.3	4.31	2.16	88	49	118/70	13.5	12.7	2800	0			0		++
	Operation on June 17, 1937																		
	July 1, 1937	166.0	88.0	224				82		110/88	10.2	11.1	1800	0			4.8	90	+
	Aug. 6, 1937																		
	Aug. 27, 1937	164.5	72.0	224	61.0	3.67	2.05	78	47	90/65	16.7	12.9	3360	0	A.C. 1.0 gm. t.i.d.		0	86	++
	Nov. 19, 1937	165.3	72.4	222	65.5	3.39	1.88	74	46	120/80	18.0	10.7	3600	0	A.C. 1.0 gm. t.i.d. and theocalcin 1.0 gm. t.i.d.		4.9	96	++
	Nov. 26, 1937	164.3	73.8	220				74		80/68	19.5	8.7		0			5.9		++
	Dec. 1, 1937	165.3	86.9	220	56.5	3.39	2.00	78	50	100/75	21.4	8.3	2900	0					+
	Jan. 28, 1938																		

¹ The rhythm of the heart was normal sinus rhythm in all the patients except P. A., Case 5.

² The serum protein values were repeatedly shown to lie within normal limits in all the patients except P. A., Case 5, and J. S., Case 6, in whom the total protein repeatedly remained between 4.0 and 5.0 grams per cent.

³ 0, +, ±, ↓, = absent, present, doubtful, increased, decreased, respectively.

⁴ 14.5 grams hemoglobin equivalent to 100 per cent.

⁵ +, 0, ± = present, absent, questionable, respectively.

⁶ Mercupurin, 1.0 cc., was given intravenously on May 17, 1936.

⁷ No theocalcin had been given since September 10, 1935.

⁸ No theocalcin had been given since November 6, 1935.

⁹ On March 18, 1937, the patient was pregnant.

¹⁰ On the days when special studies of the circulation were made, maintenance doses of digitalis, or doses of other drugs, were not given to any of the patients until after the studies had been completed.

¹¹ A.C. = ammonium chloride.

¹² No digitalis had been given since December 28, 1935.

¹³ Mercupurin, 2.0 cc., was given intravenously on January 26, 1936.

¹⁴ No digitalis had been given since March 5, 1936.

¹⁵ "Resting" and not "basal" measurements.

¹⁶ Mercupurin, 2.0 cc., was given intravenously on February 1, 1937.

¹⁷ Mercupurin, 2.0 cc., was given intravenously on September 21, 1936.

¹⁸ Mercupurin, 2.0 cc., was given intravenously on November 17, 1937.

Theocalcin, 1.5 grams t.i.d., and ammonium chloride, 1.0 gram t.i.d., were also being given.

Theocalcin, 1.0 gram t.i.d., and ammonium chloride, 1.0 gram t.i.d., were also being given.

TABLE II
Additional data relating to 6 cases of chronic constrictive pericarditis *

Case number; Initials; and History number	Time	Duration of disease†	Etiology	Heart size	Calcification of pericardium	Fluoroscopy of heart	Electrocardiogram	Result	Time since operation‡
Case 1 A. B., Number 28323	Before operation	5 years 6 months	unk.	Very small	Present	Very small pulsations	Sl. R.A.D. QRS _{1,2,3} and T _{1,2,3} low ampl. T _{1,2} cove shaped. Axis shift 37°		
	After operation			Larger	Present	Very good pulsations	Axis shift 22°. No other change	Cured	1 year 9 months
Case 2 W. M., Number 103699	Before operation	1 year 4 months	unk.	Large	0	Sl. of lt. side, none of rt. side. No downward shift, sl. lt. shift	R.A.D. QRS _{1,2,3} low ampl. T _{1,2} neg. Axis shift 0°		
	After operation			Smaller	0	Good pulsations	R.A.D. QRS _{1,2,3} still low ampl. T _{1,2,3} incr. ampl. Axis shift 9°	Cured	1 year 4 months
Case 3 A. R., Number 91648	Before operation	4 months	tbc.	Sl. enlarged	0	Decr. pulsations of rt. aur. and rt. vent.	Sl. R.A.D. QRS _{1,2,3} low ampl. T _{1,2,3} low ampl. Axis shift 11°		
	After operation			Smaller	0	Excellent pulsations	No change. Axis shift 6°	Cured	2 years, 2 months
Case 4 J. McC., Number 128763	Before operation	7 months	unk.	Large	0	No motion lower ½ of heart	Sl. L.A.D. QRS _{1,2,3} low ampl. T _{1,2} cove shaped. Axis shift 11°		
	After operation			No change in size, but shape changed	0	Incr. motion but none of rt. vent.	No axis deviation. Incr. ampl. of QRS _{1,2,3} . Axis shift 18°	Improved	1 year, 3 months
Case 5 P. A., Number 141257	Before operation	§	unk.	Large	Present	Decr. pulsations of rt. aur.	Sl. R.A.D. QRS _{1,2,3} low ampl. T _{1,2,3} diphasic. Axis shift 35°		
	After operation			Not much change	Present	Incr. along lt. but almost none of rt. aur. and rt. vent.	No change. Axis shift 30°	Improved	1 year, 6 months
Case 6 J. S., Number 169168	Before operation	6 years	unk.	Small	Not seen in x-ray	Sl pulsations of lt. vent.	Sl. L.A.D. QRS _{1,2,3} low ampl. Low ampl. of T ₂ . Axis shift 0°		
	After operation			Larger	Present in micro. sec. of pericard.	Incr. pulsations	Sl. L.A.D. QRS _{1,2,3} sl. incr. in ampl. T _{1,2} incr. in ampl. Axis shift 30°	Improved	8 months

* In this Table the following abbreviations are used:

unk. = unknown

tbc. = tuberculosis

sl. = slight

lt., rt. = left, right, respectively

incr., decr. = increased, decreased, respectively

ampl. = amplitude

neg. = negative

R.A.D., L.A.D. = right and left axis deviation respectively

aur., vent. = auricle, ventricle, respectively

micro. sec. of pericard. = microscopic sections of pericardium

† As estimated from evaluation of patients' history.

‡ As of March, 1938.

§ Edema of ankles was noted 10 years before admission. Symptoms were first noted 2 months before admission.

DISCUSSION

Our observations show that chronic constrictive pericarditis is characterized by decreased cardiac output per minute and per beat, rise in venous pressure, slowing of the velocity of blood flow, and engorgement of the venous vascular bed. Fluoroscopic examination shows decreased contraction of the heart chambers, and fixation of the heart may be observed. Clinical improvement after operation was associated with changes in all these functions toward normal. There appear to be two essential defects in this syndrome,

namely, (1) obstruction to the entrance of blood into the chambers of the heart resulting in decreased filling, and (2) interference with contraction of the heart. There is evidence for the first in (a) the decreased dilatation of the heart in diastole under fluoroscopic examination and at operation and (b) the observation at operation of the thickened pericardium which was not capable of much distention and may have been calcified. (c) Infra-red photographs revealed distention of the peripheral veins. (d) The elevated venous pressure is evidence that there is ample

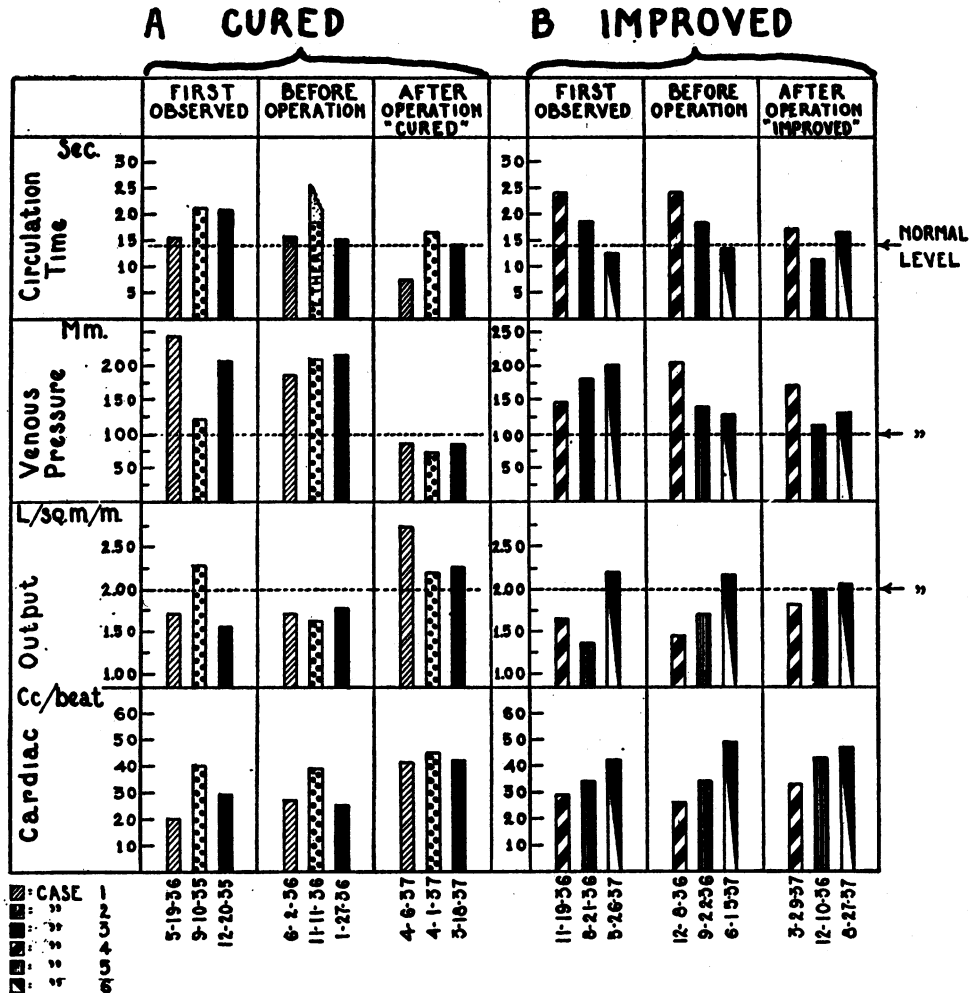


FIG. 1

Data relating to 3 patients "cured" by operation (A) and 3 "improved" by operation (B).

blood available for the heart. With respect to contraction, impairment is inferred from the (a) decreased extent of contraction on fluoroscopic examination and at operation, as well as from (b) the examination of the thickened unyielding pericardium incapable of much change during contraction. These two defects result in decrease in cardiac output, per beat and per minute, and piling up of blood on the venous side, accounting for increase in venous pressure and slowing of the velocity of blood flow. The heart may be unusually small or not much enlarged.⁵ Removal of the pericardium results in alteration of these

⁵ The cardiac silhouette is made up of cardiac shadow plus the shadow of the thickened pericardium.

two defects, in short, in removing obstruction to blood entering the heart, allowing the heart to stretch in diastole, and increase in extent of contraction; these account for changes in the circulation which have been recorded after operation. Parallel with improvement in circulation after operation, clinical improvement occurs.

These observations with respect to cardiac output and venous pressure are in accord with those reported by Burwell and his associates (12, 13), and by Beck and Cushing (14).

The delay in improvement of certain patients after operation may be due in part to dilatation of the region of the heart from which the pericardium has been resected, and in part to ob-

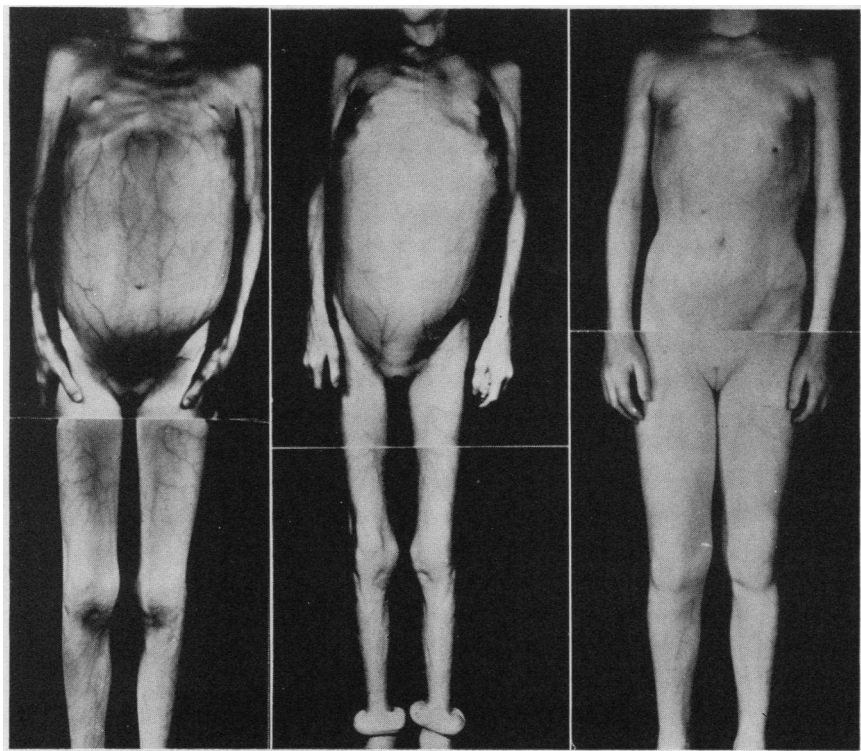


FIG. 2

Infra-red photographs of A.B. (Case 1), in whom operation was followed by "cure." Photograph A was made on May 20, 1936; B on June 29, 1936, 20 days after pericardiectomy, and C on April 4, 1937, approximately 10 months after operation. Attention is directed to the decrease in venous channels, to the patient's growth and development, and to change in flare of the ribs with the elimination of the chronic ascites from which the patient had suffered five and a half years.

struction not having been sufficiently relieved. At operation, the heart bulged through the window which was made in the pericardium and undue stretching of this muscle may have resulted. It may require time for this muscle to regain "tone."

Three patients (Cases 2, 3, and 4) were observed from the stage of pericarditis with effusion through the stage of constriction. In them, signs of obstruction disappeared with absorption of the pericardial fluid, but increased rapidly when formation of adhesions and constriction began and was associated with rapid parallel rise in venous pressure, and circulation time, and decrease in cardiac output.

The use of digitalis appears to be contraindicated except under certain circumstances. Stewart and his associates have shown that digitalis decreases cardiac size (15, 16, 17) and increases the extent of ventricular contraction (18, 19, 20).

In the presence of constrictive pericarditis, the size of the heart may already be restricted and the cavities small, and further decrease in its size may not be beneficial but may increase the obstruction. On the other hand, the heart is probably contracting as fully as possible while it is attached to the unyielding thickened pericardium. One patient (Case 5) exhibiting auricular fibrillation was under the influence of digitalis when she came under observation. Its use seemed essential to maintain a slow heart rate, and it did not appear justifiable to withdraw the drug. To one patient (Case 3), exhibiting normal sinus rhythm, the drug was given *after* resection of the pericardium. In this instance, increase in cardiac output and fall in venous pressure occurred, effects which were to be expected (15, 16, 17), (Table I). Change in size of the heart could not be estimated because of the presence of fluid

in the right pleural cavity. Digitalis was given to another patient (Case 2) exhibiting normal sinus rhythm. We were unable to state the stage of the pericardial lesion at the time of exhibition of the drug; it was probably in the stage of absorption of pericardial fluid and early formation of adhesions. The patient was taking theocalcin at the time the first observations were made (Table I). When the drug was discontinued there was rise in venous pressure, slowing of circulation time and decrease in cardiac output. When it was given again the venous pressure fell, the circulation time decreased, and cardiac output increased, and all the measurements were in the normal range. We were unable to evaluate the effects of giving digitalis, 1.8 grams, on September 29, 1935. The control measurements were made 4 days beforehand and spontaneous changes which would invalidate the comparison may have occurred in the circulation in this interval. The effects of theocalcin can, however, be evaluated and were similar to those already recorded by Stewart and Cohn (15).

Case 6 calls for comment. Except for the elevated venous pressure, the measurements of the circulation were in the normal range before operation. She improved before operation by medical treatment and the use of drugs. She was able apparently to maintain at rest in bed a normal cardiac output per minute and cardiac index and approximately normal output per beat in spite of the very thickened adherent pericardium. Operation resulted only in moderate clinical improvement, and the venous pressure fell to normal. Subtotal thyroidectomy had been performed on this patient early in her illness 6 years before, when it was suspected that heart failure was caused by hyperthyroidism. Whether this accounted for the deviation of the patient from the pattern of the other patients, we are unable to state. The circulation was not maintained at the expense of an elevated heart rate, nor was there anemia (21). The plasma protein in this patient remained low although she was given a high protein diet. Before operation, diuretics were ineffectual in this patient. After operation, however, urine outputs of 5 liters a day resulted from 2.0 cc. injections of mercupurin, and 16 kgm. of weight were lost in 6 weeks (Table I).

The partition of the circulation time was studied

in 4 patients (Table III). The arm to tongue circulation time was measured by the use of decholin (D), the arm to lung time by the injection of ether (E) (22), and the lung to respiratory center by the inhalation of carbon dioxide (C) (23). D minus E should approximate the value of C, which was found to be the case when observations were made (Table III). The ether

TABLE III
Data relating to partition of circulation time in 4 cases

Patient	Date	Venous pressure	Circulation time			
			Arm to tongue. Decholin (D)	Arm to lung. Ether (E)	Lung to respiratory center. CO ₂ (C)	D-E should equal (C)
		cm.	seconds	seconds	seconds	seconds
Case 2	Jan. 28, 1938	9.2	19.2	5.5	15.0	13.7
Case 4	Jan. 29, 1938	18.8	21.5	13.5	9.5	8.0
Case 5	Feb. 1, 1938	15.3	19.2	6.0	13.5	13.2
Case 6	Feb. 28, 1938	15.5	21.4	8.3	12.0	13.1

arm to lung circulation time was in the normal range in Cases 2, 5, and 6, and prolonged in Case 4. The lung to respiratory center time was prolonged in Cases 2, 5, and 6, and normal in Case 4. In short, no uniformity was apparent.

The effect of pregnancy was observed in one patient (Case 3). Observations made on November 30, 1937 (Table I) when she was 3 months pregnant revealed increase in arteriovenous oxygen difference, decrease in cardiac output per minute and per beat, and in cardiac index, and lengthening of the circulation time, not only on comparison of them with the measurements on March 18, 1937, before pregnancy occurred, but also when restoration to normal was found on December 16, 1937, 13 days after therapeutic abortion.

SUMMARY AND CONCLUSION

Chronic constrictive pericarditis is usually associated with decrease in cardiac output per minute and per beat, and decrease in the cardiac index. The venous pressure is elevated and the circulation time prolonged, and there is increase in size and caliber of the peripheral venous channels. Rest in bed and medical therapy may occasion clinical improvement with disappearance

of the accumulations of fluid, and with changes of the circulation toward normal. After operation in those cured, the measurements assumed normal limits and in those "improved" the measurements of the circulation approached normal. In this syndrome the symptoms and signs appear to be a consequence of the defects in circulation which the constricting pericardium occasions. These defects appear to be two: (1) obstruction to entrance of blood into the chambers of the heart and (2) interference with contraction and emptying of the heart. These result in (1) decrease in cardiac output per minute and per beat and (2) piling up of blood on the venous side, which accounts for rise in venous pressure and slowing of the velocity of blood flow. Releasing the heart and removing obstruction by resection of part of the pericardium results in return of these functions toward or to normal levels.

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