THE EFFECT OF TUBERCULOSIS ON THE HEART *

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A good heart is one of the best assets possessed by a patient suffering from pulmonary tuberculosis. From the earliest infection by the tubercle bacillus it is more or less disturbed in its action, and as the focus of infection grows larger the effect on the heart becomes greater. The deleterious influences which react on the heart are many and from varied sources. The heart problem increases in complexity as well as in importance as the disease advances in severity. While it would seem that, with the present-day knowledge of tuberculosis and the means at our command for making an early diagnosis, we should see a greatly diminished number of cases of advanced tuberculosis in the future, yet I fear that it will be a long time before tuberculosis will be generally diagnosed early; and I do not doubt that any knowledge that may be added bearing on the condition of the heart in advanced infections will be welcome and of clinical value.

There are many difficulties in the way of giving an opinion on the condition of the heart in individuals suffering from tuberculosis, especially those suffering from advanced tuberculosis. The position of the heart in the chest is altered, the pulmonary circulation is embarrassed, changes occur in the systemic arteries as well as the heart itself, blood pressure is altered, the relative intensity of the heart tones is changed, numerous murmurs appear at the various valve areas and numerable adventitious sounds are heard. Without bearing these facts in mind many erroneous opinions will be given.

The first change that occurs in the heart and circulatory system is noted as soon as the infection is of sufficient grade to cause clinical symptoms. This is an acceleration of the pulse. It is probably of toxic origin and is associated with a lowering of the blood pressure due to the action of the tubercle toxins on the vasodilators. This quickening of the pulse may be slight, in which case it would perhaps be noted only after exertion; or it may be marked and even show when the patient is at rest. The causes which operate to quicken the pulse during the course of pulmonary tuberculosis are many. The rapidity is in

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part doubtless a physiological response on the part of the heart to compensate for the lowering of the blood pressure caused by the dilatation of the arterioles; in part due to a lack of power on the part of the heart muscle itself, resulting from the action of toxins or because of overwork; in part due to a stimulation of the sympathetic fibers in the chest, and doubtless in part due to the interference with the inhibitory action of the vagus.

As the disease progresses and the action of the toxins becomes more manifest, general muscular wasting occurs in which the heart muscle bears its share. Then it is that we find the greatest changes in the blood pressure. Opposing the fall in pressure we find, early in the disease, hypertrophic changes in the right ventricle; and later, quite often, a thickening of the arterial walls.

Having noticed a thickening of the arterial walls in many cases of advanced tuberculosis, I decided to find out how general it is, with the result as noted in Table 1. In the first place, I found it most commonly in patients who had suffered from the disease for many years, consequently in those in whom the fibroid form of the disease predominated. I do not doubt that the cause is the prolonged action of weak toxins on the vessel walls. This opinion is supported by Table 1, which shows the comparative frequency of palpable arteries in those ill less than one year, from one to two years and more than two years:

TABLE 1.—SHOWING THE COMPARATIVE NUMBER OF PALPABLE RADIALS IN PATIENTS WHO HAVE BEEN ILL LESS THAN ONE YEAR, FROM ONE TO TWO, AND MORE THAN TWO YEARS. TOTAL NUMBER OF PATIENTS, 162

	Duration of Disease.		
Condition of Radials.	Less Than 1 Year.	1 to 2 Years.	More Than 2 Years.
Palpable	14	20	60
Non-palpable	14	21	33

I took as an index to the condition of the arterial walls the condition of the radials, and determined whether or not they were palpable either after stripping the vessel from below upward with the second finger of one hand and from above downward with the second finger of the other hand and then using the first finger to do the palpating of the empty vessel, or by palpating the vessel while the blood was shut off from the artery by the cuff of the blood pressure apparatus. Very inexact data will be obtained unless the column of blood is first expressed from the artery.

Analyzing the blood pressures[1] of the 162 cases here represented, we see that there were 11 first-stage cases with an average pressure of systolic

1. Stanton's sphygmomanometer was used in making the blood-pressure observations recorded in this report. The average blood pressure of 20 healthy adults taken under the same conditions showed systolic 130, diastolic 110.

108, diastolic 78; 21 second-stage cases with an average pressure of systolic 108, diastolic 81, and 130 third-stage cases with an average of systolic 103, diastolic 75. It must be remembered that these pressures were taken in Southern California, where climatic conditions favor a relatively low pressure. If we note the influence exerted by the palpable arteries (we include only the 130 cases in Stage III), we shall see that the average pressure in the 83 cases in which the arteries were palpable was: systolic 105, diastolic 76; while that of the 47 cases where they were not palpable was: systolic 102, diastolic 75. That this condition of the arteries is not influenced largely by the age of the patients is very evident from Table 2, for of those who had palpable arteries none were between the ages of 15 and 20; 21 between 20 and 30; 45 between 30 and 40; 20 between 40 and 50, and 7 between 50 and 65. And of those whose arteries were not palpable 11 were between the ages of 15 and 20; 35 between 20 and 30; 16 between 30 and 40; 6 between 40 and 50, and 1 between 50 and 65.

TABLE 2.—CONDITION OF THE ARTERIES ACCORDING TO AGE

Conditions of Arteries.	15 to 20 Years.	20 to 30 Years.	30 to 40 Years.	40 to 50 Years.	50 to 65 Years.
Radials Palpable	00	21	45	20	7
Radials not Palpable....	11	35	16	6	1

Now, if we note the effect exerted by myocarditis (in this class I placed only those who showed marked clinical signs), we shall note the opposite effect. The average pressure in the 62 cases in the third stage showing myocardial change was: systolic 97, diastolic 70; while the average pressure in the 68 which did not show this change was: systolic 110, diastolic 82.

Further analysis of these cases seems to indicate that these two factors, thickening of the arterial walls and degeneration of the heart muscle, are two very potent factors in modifying the blood pressure in tuberculous patients. Table 3 shows this very plainly for the cases which did not show myocarditis. Where myocarditis was not present, those patients who had palpable radials had a blood pressure of, systolic 113, diastolic 82, even higher than the pressure of the first- and second-stage cases. In those of the same class whose radials were not palpable the blood pressure was, systolic 105, diastolic 83, making a difference of about 8 points in the systolic pressure, which seems to be due to the condition of the arteries as determined by the radials. This difference, although present, is not apparent in the cases showing myocardial change as arranged in Table 3. If Table 4 is studied, however, it will be seen that the reason for those who have palpable radials and myocarditis having a lower blood pressure than those whose radials did not show

the change is that several of the former had such severe myocardial change that the tendency to raise the pressure owing to the changes in the arteries was overcome and the actual pressure was lowered. This might indicate also that this thickening of the arteries, while helping to maintain the blood pressure in an earlier stage of advanced tuberculosis, finally proves detrimental in that it causes too great a strain to be thrown on the heart, and the heart muscle, being unable to meet the demand, fails.

TABLE 3.—SHOWING THE EFFECT OF THE CONDITION OF THE ARTERIAL WALLS ON BLOOD PRESSURE WHEN ASSOCIATED WITH AND WITHOUT MYOCARDIAL CHANGE

		Myocarditis			Non-Myocarditis	
Radials.	No. Cases.	Systolic	Diastolic	No. Cases.	Systolic	Diastolic
Radials Palpable ...	41	97	70	42	113	82
Radials Non-palpable	21	97	69	26	105	83

Myocardial change is present in some degree in practically all cases of tuberculosis. In the earlier stages of the disease when the patient's general condition is good, this change is a hypertrophy of the right ventricle to enable it to do the extra work forced upon it by the diminution of the pulmonary vascular area. Later, through the action of toxins, the general malnutrition, overwork and other deleterious agencies, the muscle degenerates and weakens. The field of cardiac response becomes diminished and whereas, at first, the heart is able to perform its function by calling on its reserve energy and working at its maximum power as evidenced by the hypertrophy which takes place, it is now no longer able to do so, and as a result the circulation is carried on imperfectly and dyspnea and cyanosis appear.

In determining the presence of myocardial change, I took into consideration both clinical symptoms and signs on the part of the heart itself; and designated as cases of myocarditis only such hearts as showed marked change.

There are many difficulties in the way of giving an opinion on the heart in advanced tuberculosis. In the first place, we do not have the same anatomical relations in advanced tuberculosis as we do in the normal chest. Through the effect of pleurisies, contractions and compensatory emphysemas the heart is often displaced and the heart valves no longer bear their normal relation to the superficial landmarks. If the heart is uncovered by the retraction of a portion of the lung, sounds that are not at all intensified may appear much louder than they would under normal conditions, and, on the other hand, if a large portion of emphysematous lung be interposed between the heart and chest wall the sounds will appear much weaker than they really would with normal covering, and it would be very difficult to accurately judge their intensity.

The presence of infiltrations and cavities also alter the quality and apparent intensity of the sounds. It can readily be seen, then, that it is difficult to compare and determine the relative intensity of the sounds at the different valve areas.

In making the observations recorded here I was compelled to disregard entirely the usual location of the valve sounds on the surface of the chest because the hearts were displaced to one or the other side in many instances. My data on the various valves was taken at the point where it seemed that the valve in question would most probably be located when the shifting of the heart had been considered.

The following observations were made with the stethoscope:

Apex—
- Weak, 7 times.
- Strong, 5 times.
- Double, 89 times.
- Gallop rhythm, 17 times.
- Bruit, organic 6, functional 11 times.
- Triple sound, 1.
- Irregular, 2.

Aortic—
- First sound weak, 21 times.
- Second sound accentuated, 72 times.
- Second sound weak, 5 times.
- Bruit, systolic 6; diastolic, 4; organic, 5; functional, 5.

Pulmonic—
- First weak, 18 times.
- Second accentuated, 94 times.
- Second double, 35 times.
- Bruit, 6 times.

Tricuspid—
- Insufficiency, 4 times.

Adventitious Sounds—
- Probably pleuropericardial adhesions, 15 times.
- Pericarditis, 1 time.
- Pericarditis (adhesive) 3 times.

Some of the observations mentioned above require explanation because the personal element always comes into play in characterizing observations of this kind. Errors of judgment are prone to creep in, but a few words of explanation will doubtless make the meaning clear.

A double beat was observed at the apex in 89 of the 162 patients. I designated as double what I considered as a departure from the normally recognized firm tone of the apex. The degree of doubling varied a great deal from what might be considered as a slight impurity to two very distinct sounds. It is estimated that a double sound at the apex is heard in about 10 per cent. of normal hearts, but it will be seen that in the patients here reported it occurred in more than 50 per cent. of the cases.

The frequency with which accentuation of the second aortic was found also requires explanation. In some cases this might be produced by the thickening of the systemic arteries; in others, it is only apparent and not actually an accentuation, the apparent accentuation depending on the nearness to infiltrations and cavities; in some cases it is due to the fact that the valve is uncovered and consequently seems louder and more accentuated than it is.

Three of the bruits designated as organic occurred in one family.

The four cases of tricuspid insufficiency were due to advanced stages of degeneration and dilatation of the heart muscle.

During the course of this investigation my attention was called to the Bock stethoscope as offering help in determining the relative intensity of the heart sounds. I regret that I did not have the Bock record in all instances, but I have tried it in a sufficient number of cases to demonstrate its value. By studying Table 4 it will be seen that the Bock readings give a fair estimation of the condition of the heart muscle. In interpreting the Bock readings it must be remembered that the standard is an arbitrary one and the readings of each heart must be considered alone. Bock has determined that for the normal individual the aortic sound should be about one-third less than that heard at the apex, and the pulmonary sound from five to fifteen points below the aortic.

Another very important topic for consideration in discussing the heart in its relation to advanced tuberculosis is the effect produced on it by the displacement which occurs on account of the changes in the lung and pleura. That this has received too little consideration in the past is self-evident.

It is natural to suppose that organs perform their functions best when in their natural positions. The heart in its normal position lies on the diaphragm and swings from the great vessels which serve as points of fixation at the base. It is surrounded and limited in its motion by the pericardium, and the amount of motion allowed the heart is dependent, to a certain extent, on the size of the pericardium. The pericardium is fixed at five points: to the under surface of the sternum, to the diaphragm, to the right and left pleuræ and to the great vessels of the chest. The heart is allowed slight motion within the pericardium under normal circumstances, as is noted on the change of position from lying on one side to lying on the other. Changes of this character in no wise interfere with the heart in the performance of its function because the position and size of the pericardium is not changed, the large vessels are lying free and easy in their normal place and no impediment is offered to the free and natural movements of the heart itself or to the

TABLE 4.—EFFECT OF TUBERCULOSIS ON THE HEART

Sex.	Stage; Turban.	Age.	Duration of Illness. Yrs.	Involvement.†	Blood Pressure.		Radials.*	Position of Heart.			Heart Tones.							Bock.		
					Systolic.	Diastolic.		Fourth Interspace. R. Cm.	Fourth Interspace. L. Cm.	Diameter.	Mitral.	Aortic.	Pulmonic.	Tricuspid.	Adventitious Sounds.	Heart Muscle.	Remarks.	Apex.	Aortic.	Pulmonic.
F.	I.	26	3	R. > L.	115	82	—	3.75	6.25	10	1st double.							85	62	62
F.	"	18	1½	L. > R.	115	84	—	3.5	4.5	8	Slight gallop rhythm.							82	75	80
M.	"	32	1½	L. > R.	100	74	+	3.0	7.0	10		2d accent.	2d accent.							
M.	"	18	5½	R. > L.	95	65	—	3.0	6.25	9.25		2d accent.	2d accent.							
F.	"	30	½	L. > R.	110	72	—	2.25	6.75	9		Systolic murmur organic.								
M.	"	26	2½	L. > R.	125	100	—	3.75	8.0	11.75										
M.	"	13	1	L. > R.	88	55	—	2.5	5.5	8.0										
M.	"	21	3	L. > R.	132	74	—	3.75	7.0	10.75										
F.	"	22	13	R. > L.	98	70	—	2.75	7.0	9.75								80	55	55
M.	"	29	¼	L. > R.	124	100	+	3.25	10.5	13.75		2d accent.						80	70	65
F.	"	34	1	L. > R.	112	77	—	4.0	7.0	11.0								72	60	64
M.	II.	24	2	L. > R.	96	69	—	4.0	7.87	11.87								70	55	65
M.	"	31	7	R. > L.	130	96	+	3.75	5.5	9.25		2d accent.	1st weak.					65	55	35
M.	"	30	⅓	L. > R.	113	80	+	3.75	7.0	10.75								85	70	65
F.	"	29	3	L. > R.	114	76	—	2.75	8.0	10.75	Double.							85	75	70
M.	"	22	1/12	R. > L.	108	88	—	3.5	7.0	10.5	Systolic bruit (functional).		2d accent.					90	72	70
M.	"	21	1/6	L. > R.	120	86	—	3.0	8.0	11.0	Systolic bruit (functional).		Systolic bruit (functional).					75	60	67
F.	"	17	½	R. > L.	112	83	—	3.5	5.75	9.25										
F.	"	14	½	R. > L.	98	69	—	4.5	4.25	8.75	Systolic bruit (functional).		2d accent.		Pleuropericardial adhesions.					

* In this column + stands for "palpable;" — for "non-palpable."
† R. > L. signifies right lung involvement greater than left; L. > R., left lung involvement greater than right.

M.	II	32	5/6	R. > L.	114	94	—	2.75	7.5	10.25	.....	.....	2d sl. accent...							
F.	"	25	5/12	L. > R.	92	67	—	3.0	5.5	8.5	1st double.....									
M.	"	23	1	L. > R.	109	78	—	2.0	9.25	11.25	.....	.....								
M.	"	32	1½	R. > L.	136	98	+	3.0	7.25	10 25	Systolic murmur (org).	1st weak.....	1st weak.							
M.	"	50	4	L. > R.	100	68	+	3.25	7.75	11.0	1st double.....	2d accent double.	2d accent.							
F.	"	20	1	R. > L.	110	83	—	3.5	8.5	12.0	Double murmur; systolic and diastolic (organic.)	Double murmur (org'ic).	.....	.....	.....	.....	.....	82	20	80
M.	"	29	1	L. > R.	98	67	+	3.25	8.0	11.25	1st double.....	2d sl. accent...								
M.	"	51	1	R. > L.	118	98	+	4.0	6.0	10.0	1st double.....	2d accent (metallic).								
M.	"	38	4	R. > L.	85	60	—	4.5	6.75	11.25	Double.....	2d accent.....	2d accent.....	.....	Pleuropericardial adhesions.	.....	Pericarditis.			
M.	"	30	½	R. > L.	88	65	—	5.0	7.5	12.5	Double.....	Systolic bruit (functional).	2d sl. accent...	.....	.....	.....	.....	88	80	82
F.	"	49	4	L. > R.	140	122	—	3.25	7.75	11.0	Double, irregular.	.....	Bruit; 2d accent.	.....	.....	.....	.....	80	75	85
M.	"	39	1	R. > L.	120	104	—	3.75	6.75	10.5	1st double.....	.....	.....	.....	.....	.....	.....	95	90	80
F.	"	20	6	L. > R.	122	98	+	3.5	7.0	10.5	1st double.....	.....	2d sl. accent; double.	.....	.....	.....	.....	80	70	75
M.	III.	25	1.	L. > R.	116	80	—	3.0	8.5	11.5	1st double bruit functional; gallop rhythm.	.....	2d accent.	.....	.....	Myocarditis.				
F.	"	25	1½	R. > L.	108	83	—	3.0	7.25	10.25	Double.....	.....	Bruit.							
M.	"	31	1	L. > R.	82	57	+	4.75	8.5	13 25	Double; gallop rhythm.	1st weak; 2d accent.	1st weak; 2d accent.	.....	.....	Myocarditis.				
F.	"	27	1¼	L. > R.	100	75	—	6.0	8.5	14.5	1st weak; gallop rhythm.	1st weak.....	2d sl. accent...	.....	.....	Myocarditis.	.....	75	60	75
M.	"	52	2	L. > R.	100	76	—	1.5	8.5	10.0	1st double.....	2d sl. accent...	2d accent double.	.....	.....					
F.	"	19	2	L. > R.	94	68	—	4.0	8.0	12.0	1st double.....	2d accent.....	2d accent.....	.....	.....	.....	.....	75	70	92
F.	"	40	9	L. > R.	90	68	+	−2	12.0	10.0	1st double.....	1st / 2d } weak	1st weak.....	Insufficiency.	Pleuropericardial adhesions.	Myocarditis.	.....	70	65	75
F.	"	41	7	L. > R.	96	74	—	4.0	7.25	11.25	Systolic bruit functional.	1st weak; 2d accent	2d accent.....	.....	.....	Slight myocarditis.				
F.	"	23	2½	R. > L.	110	83	—	3.5	6.0	9.5	Triple sounds.	.....	2d accent.....							

TABLE 4.—EFFECT OF TUBERCULOSIS ON THE HEART (Continued)

Sex.	Stage; Turban.	Age.	Duration of Illness. Yrs.	Involvement.	Blood Pressure. Systolic.	Blood Pressure. Diastolic.	Radials.	Position of Heart. Fourth Interspace. R. Cm.	Position of Heart. Fourth Interspace. L. Cm.	Position of Heart. Diameter.	Heart Tones. Mitral.	Heart Tones. Aortic.	Heart Tones. Pulmonic.	Heart Tones. Tricuspid.	Heart Tones. Adventitious Sounds.	Heart Tones. Heart Muscle.	Remarks.	Bock. Apex.	Bock. Aortic.	Bock. Pulmonic.
F.	III	36	2	L. > R.	90	68	+	2.75	8.0	10.75	1st double.....	1st weak; 2d accent.	1st weak; 2d accent.	.....	.....	My ocardi-tis.				
F.	"	47	9	R. > L.	95	58	—	4.0	6.5	10.5	.....	2d accent.....	2d accent.....	.....	.....	.....	.....	85	75	65
M.	"	32	1	L. > R.	108	76	+	2.75	10.0	12.75	1st double.....	2d accent.....	2d accent metallic.	.....	.....	.....	.....	75	65	45
F.	"	37	4	L. > R.	100	70	+	3.75	3.75	12.5	1st double.....	.....	2d double.....	.....	Pleuropericardial adhesions.	My ocardi-tis.	.....	92	70	75
M.	"	38	4	L. > R.	98	72	+	2.75	8.0	10.75	Double.....	.....	2d double accent.	.....	.....	My ocardi-tis.	.....	65	63	70
M.	"	30	2	R. > L.	108	76	+	5.0	4.5	9.5	1st double.....	2d accent.....	2d accent.....	.....	.....	.....	.....	83	75	77
F.	"	36	2	R. > L.	115	78	—	4.5	6.5	11.0	1st double.....	2d sl. accent...	.....	.....	.....	.....	.....	80	75	75
M.	"	24	1½	L. > R.	88	54	+	3.5	8.0	11.5	1st double.....	2d accent.....	1st weak.....	.....	.....	My ocardi-tis.	.....	67	50	30
M.	"	36	⅓	L. > R.	122	88	+	3.75	5.75	9.5	.....	2d accent.....	2d accent.....	.....	.....	.....	.....	65	70	55
F.	"	39	2	L. > R.	87	68	+	1.5	11.0	12.5	Double.....	1st weak.....	1st weak.....	.....	.....	My ocardi-tis.				
F.	"	36	2	L. > R.	116	88	—	0.5	8.5	9.0	1st double.....	2d accent.....	2d accent.....	.....	.....	.....	.....	80	70	70
M.	"	34	1	R. > L.	100	72	+	6.5	7.75	14.25	1st weak.....	1st very weak..	1st very weak..	.....	.....	My ocardi-tis.				
F.	"	29	4	R. > L.	80	58	—	5.25	4.25	9.5	Double.....	2d double accent.	.....	Insufficiency.	.....	My ocardi-tis.				
F.	"	34	1½	L. > R.	100	75	+	2.0	9.0	11.0	Gallop rhythm.	2d sl. accent...	2d accent.....	.....	.....	My ocardi-tis.				
M.	"	29	⅔	R. > L.	122	86	+	3.75	7.5	11.25	Bruit systolic; organic.	2d metallic accent.	.....	.....	.....	.....	Nephritis..	80	75	72
F.	"	30	½	L. > R.	95	77	+	2.25	6.75	9.0	1st double; systolic murmur (functional).	Systolic murmur (functional).								
M.	"	20	7/12	L. > R.	104	74	—	4.0	6.75	10.75	1st double.....	.....	.....	.....	.....	My ocardi tis.				
M.	"	37	2	R. > L.	106	80	+	4.5	5.5	10.0	1st double.....	1st weak; 2d weak.	2d double.....	.....	.....	My ocardi-tis.	.....	82	72	60

M.	III	36	11/12	R. > L.	98	70	+	4.0	8.5	12.5	1st double.....	1st weak.....	1st weak; 2d dbl. accent.	.....	.....	My ocardi-tis.	.....	86	65	67
F.	"	43	4	R. > L.	90	67	+	5.0	6.5	11.5	1st weak; gal-lop rhythm.	1st weak; 2d weak.	.....	.....	.....	My ocardi-tis.	.....	65	45	35
F.	"	28	13	R. > L.	102	70	+	3.0	7.0	10.0	Systolic bruit (functional).	.....	Systolic bruit (functional).	.....	.....	.....	.....	82	65	65
F.	"	40	9	L. > R.	133	108	+	4.5	9.5	14.0	.....	.....	.....	.....	.....	.....	.....	90	75	65
M.	"	25	3	L. > R.	118	78	+	4.0	8.25	12.25	Double.....	Slight accent..	.....	.....	.....	.....	.....	85	65	75
M.	"	39	6	R. > L.	138	110	+	3.5	6.0	9.5	Double.....	Sl. accentuat-ed.	.....	.....	Pleuropericar-dial adhe-sions.	.....	.....	90	80	85
M.	"	45	3	L. > R.	104	80	+	3.75	9.0	13.75	Weak; 1st double.	1st weak; 2d accent.	1st weak; 2d accent.	.....	.....	My ocardi-tis.	Irregular..	90	65	70
M.	"	33	3½	L. > R.	94	66	+	3.0	10.5	13.5	1st double.....	2d accent.....	2d db'. accent. systolic bruit.	.....	.....	.....	.....	95	70	70
M.	"	37	2	L. > R	118	88	+	3.75	9.0	12.75	.....	.....	.....	.....	.....	.....	.....	80	75	65
M.	"	38	19	L. > R.	135	85	+	2.5	8.0	10.5	Strong,duoble.	2d weak.....	2d double ac-cent.							
M.	"	28	½	L. > R.	114	80	+	4.25	6.5	10.75	Weak.....	.....	2d sl. accent...							
M.	"	29	1½	L. > R.	100	70	—	4.75	7.0	11.75	.....	.....	2d double.....							
M.	"	25	1½	L. > R.	108	78	—	1.5	9.0	10.5	.....	2d sl. accent..	2d markedly accent.							
F.	"	22	1½	R. > L.	110	80	—	2.75	5.5	8.25	1st double.....	.....	2d accent double.							
M.	"	38	9	L. > R.	122	90	+	2.5	7.0	9.5	.....	Systolic bruit.								
F.	"	47	¾	L. > R.	80	58	+	1.0	7.75	8.75	1st double gal-lop rhythm.	2d sl. accent...	2d sl. accent...	.....	.....	My ocardi-tis.				
F.	"	27	4½	L. > R.	122	90	—	—5.5	+11 5	6.0	.....	2d accent.....	3d dbl. mark-edly accent.							
M.	"	41	2	R. > L.	108	78	+	3.25	6.75	10.0	1st double.....	2d metallic ac-cent.	2d double met-allic accent.							
M.	"	21	⅜	R. > L.	108	78	—	.....	.....	.....	1st double.....	2d double.....	2d double met-allic accent.	.....	.....	My ocardi-tis.				
M.	"	21	¼	L. > R.	110	80	—	2.0	8.0	10.0	1st double.....	.....	2d double ac-cent.	.....	.....	My ocardi-tis.				
M.	"	36	2½	L. > R.	90	72	+	2.0	12.75	14.75	Strong double.	1st weak.....	1st weak; 2d sl. accent.	.....	.....	My ocardi-tis.				
F.	"	43	15	L. > R.	112	84	+	—3.5	19.0	15.5	Strong.....	2d accent.....	2d accent double.							
F.	"	18	⅓	L. > R.	82	58	—	0.5	8.0	8.5	.....	.....	2d metallic sl. accent.							
M.	"	31	4	R. > L.	120	90	—	3.75	8.5	12.25	....	2d accent.....	1st weak; 2d accent.	.....	.....	Sl. myocar-ditis.				
M.	"	46	4½	L. > R.	105	67	+	0.75	10.5	11.25	.....									

TABLE 4.—EFFECT OF TUBERCULOSIS ON THE HEART (Continued)

Sex.	Stage; Turban.	Age.	Duration of Illness. Yrs.	Involvement.	Blood Pressure. Systolic.	Blood Pressure. Diastolic.	Radials.	Position of Heart. Fourth Interspace. R. Cm.	Position of Heart. Fourth Interspace. L. Cm.	Position of Heart. Diameter	Heart Tones. Mitral.	Heart Tones. Aortic.	Heart Tones. Pulmonic.	Heart Tones. Tric spid.	Heart Tones. Adventitious Sounds.	Heart Tones. Heart Muscle.	Remarks.	Bock. Apex.	Bock. Aortic.	Bock. Pulmonic.
M.	III	50	10	L. > R.	88	55	+	2.5	8.5	11.0	Weak; 1st double.	2d sl. accent...	2d double metallic.	Systolic bruit.	.....	Myocarditis.				
F.	"	25	1	R. > L.	112	80	—	2.5	6.0	8.5	1st double.....									
F.	"	40	3	L. > R.	85	63	—	2.0	10.0	12.0	Gallop rhythm.	Diastolic murmur (org'ic).	2d accent.....	.....	.....	Myocarditis.				
M.	"	26	2½	R. > L.	108	74	+	4.0	6.5	10.5	1st double.....	2d double accent.	2d accent.							
F.	"	35	1	L. > R.	96	66	+	3.75	8.75	12.5	1st double.....	2d accent.....								
M.	"	20	3	L. > R.	92	64	—	1.75	9.0	10.75	.....	.....	.....	Relative insufficiency.	.....	Myocarditis.	Adhesive pericarditis; nephritis.			
M.	"	15	1	L. > R.	94	69	—	2.0	7.25	9.25	1st double.....	2d accent metallic.	2d accent double.							
M.	"	63	4	R. > L.	95	75	+	2.0	8.0	10.0	1st double	2d accent metallic.	2d accent.							
F.	"	33	2	R. > L.	96	60	—	4.75	5.5	10.25	.....	.....	2d sl. accent...	.....	.....	Myocarditis.				
F.	"	44	2	R. > L.	98	58	+	2.0	7.5	9.5	1st double.....	2d accent.....	2d accent.....	.....	.....	Myocarditis.				
F.	"	36	4	L. > R.	92	62	+	3.5	6.5	10.0	Double.....	.....	2d double.....	.....	.....	Myocarditis.				
M.	"	29	¾	R. > L.	100	80	+	5.5	6.0	11.5	1st double.....	2d accent double.	2d accent double.	.....	.....	Myocarditis.				
M.	"	40	24	L. > R.	114	85	+	1.75	9.25	11.0	1st double.....	.....								
M.	"	65	9	R. > L.	135	100	+	0.0	12.5	12.5	.....	2d accent.....	2d accent.....	.....	.....	.....	Arteriosclerosis.			
M.	"	47	3	R. > L.	106	74	+	4.0	9.0	13.0	1st double....	1st weak; 2d double.	2d accent.							
M.	"	20	¼	L. > R.	100	70	—	4.0	7.0	11.0	1st double; gallop rhythm.	.....	.....	.....	Pleuropericardial adhesions.	Myocarditis.				
F.	"	38	7	R. > L.	106	72	+	0.75	10.5	11.25	.....	2d accent.....	2d accent.....	.....	.....	.....	Pericarditis.			
F.	"	38	1	L. > R.	92	56	—	0.75	8.75	9.5	1st double.....	2d accent.....	2d accent double.							

M.	III	31	2	L. > R.	98	54	+	0.0	9.0	9.0	1st double.....	2d accent.....	.....	.....	.....	My ocardi-tis.				
F.	"	25	4	R. > L.	95	73	—	3.5	6.25	9.75	1st double; systolic bruit functional,	.....	2d accent.....	.....	.....	My ocardi-tis.				
F.	"	28	2	L. > R.	130	105	+	0.0	8.5	8.5	1st double.....	2d sl. accent...	2d accent.							
M.	"	27	1½	L. > R.	94	56	+	0.0	10.5	10.5	.....	2d accent.....	2d accent.							
M.	"	32	3½	L. > R.	85	56	+	4.25	6.0	10.25	1st double.....	.....	.....	.....	.....	My ocardi-tis.				
M.	"	29	2	L. > R.	80	52	+	3.25	9.0	12.25	Gallop rhythm.	Sl. diastolic murmur(functional). 2d sl. accent.	2d accent double.	.....	.....	My ocardi-tis. My ocardi-tis.				
F.	"	48	1½	R. > L.	85	60	+	5.25	10.5	15.75	Systolic bruit (organic).	2d accent double.	2d accent double.	Insufficiency.	.....	My ocar i-tis.				
M.	"	49	4	L. > R.	108	86	+	2.5	8.25	10.75	1st double....	2d accent.....	2d weak.....	.....	.....	My ocardi-tis.				
M.	"	33	5	L. > R.	128	95	—	4.25	7.5	11.75	.....	2d accent.....	2d accent double.	.....	.....	.....	Nephritis.			
F.	"	27	1	L. > R	95	72	—	—1.5	13.0	11.5	Double	Systolic murmur (func.).	2d accent.....	.....	.....	My ocardi-tis.	Nephritis.			
M.	"	38	3	L. > R.	100	75	+	—2	11.5	9.5	.....	2d accent double.	2d accent.....	.....	.....	My ocardi-tis.	Nephritis.			
M.	"	23	2	R. > L.	94	58	—	5.0	8.5	13.5	.....	2d accent metallic.	2d sl. accent..	.....	.....	My ocardi-tis.				
F.	"	16	1½	L. > R.	112	78	—	1.5	9.5	11.0	.....	.....	2d accent.....	.....	.....	My ocardi-tis.				
M.	'	37	4	L. < R.	108	84	+	3.5	8.0	11.5	.....	2d accent.....	.....	.....	.....	My ocardi-tis.				
M.	"	26	1½	R. > L.	110	62	+	2.0	8.5	10.5	1st double.....	2d accent.....	2d accent.....	.....						
M.	"	30	1	R. > L.	86	64	—	5.0	7.0	12.0	1st double; gallop rhythm.	1st weak; 2d double metallic.	1st weak; 2d accent.	.....	.....	My ocardi-tis.				
F.	"	34	4	L. > R.	114	68	+	4.5	6.5	11.0	Weak.....	Weak.....	Weak.....	.....	.....	My ocardi-tis.	Adhesive pericardit s.			
F.	"	24	½	L. > R.	114	76	—	1.5	9.5	11.0	.....	.....	2d accent.....	.....	Pleuropericardial adhesions.					
F.	"	25	1	L. > R.	105	80	—	3.25	7.5	10.75	.....	.....	2d accent double.							
F.	"	33	1½	L. > R.	86	54	+	2.75	8.5	11.25	.....	.....	2d sl. accent..	.....	.....	My ocardi-tis.				
F.	"	15	2	L. > R.	88	60	—	1.5	10.0	11.5	Gallop rhythm.	Diastolic murmur; funct.	.....	.....	.....	My ocardi-tis.				
F.	"	30	10	R. > L.	98	62	+	3.75	6.0	9.75	Systolic murmur organic.	1st weak; 2d accent.	2d accent.....	.....	.....	.....	.....	70	50	

TABLE 4.—EFFECT OF TUBERCULOSIS ON THE HEART (Continued)

Sex,	Stage; Turban.	Age.	Duration of Illness, Yrs.	Involvement.	Blood Pressure,		Radials.	Position of Heart.			Heart Tones.							Bock.		
					Systolic.	Diastolic		Fourth Interspace.		Diameter.	Mitral.	Aortic.	Pulmonic.	Tricuspid.	Adventitious Sounds.	Heart Muscle.	Remarks.	Apex.	Aortic.	Pulmonic.
								R. Cm.	L. Cm.											
M.	III	49	9	L. > R.	92	60	+	—1.5	12.0	10.5	1st double; 2d accent	2d double accent.	.....	.....	.....	My ocarditis.	Arteriosclerosis.			
M.	"	36	18	L. > R.	92	60	+	1.0	12.0	13.0	1st double.....	1st accent metallic.	.....	.....	.....	My ocarditis.				
F.	"	41	2	R. > L.	105	82	+	4.0	6.0	10.0	.....	Systolic murmur (org.).	2d accent.							
M.	"	38	3	R. > L.	122	95	+	3.75	7.5	11.25	Systolic bruit (organic).	2d accent.....	2d accent.							
M.	"	32	¼	R. > L.	102	77	+	5.0	7.5	12.5	.....	.....	2d sl. accent.							
F.	"	33	1	R. > L.	96	64	—	5.0	7.0	12.0	Gallop rhythm.	1st weak; 2d accent.	2d double and accent.	.....	.....	My ocarditis.				
M.	"	41	1½	L. > R.	115	85	+	4.25	7.25	11.5	....	2d accent.....	.....	.....	.....	.....	Nephritis.			
F.	"	40	1	L. > R.	110	74	+	1.0	10.0	11.0	1st double.....	2d accent.....	2d accent.							
F.	"	40	3	R. > L.	110	80	—	4.5	5.5	10.0	1st double.....									
M.	"	32	⅓	R. > L.	120	85	+	5.0	6.5	11.5	1st double.....	1st weak; 2d accent.	1st weak; 2d accent.	.....	Pleuropericardial adhesions.	My ocarditis, dilatation.	.....	82	70	75
M.	"	42	2	L. > R.	108	80	+	4.5	10.5	15 0	1st strong double.	.....	2d double.....	.....	.....	My ocarditis.	.....	85	40	50
M.	"	24	7	R. > L.	90	66	+	4.0	9.0	13 0	.....	.....	2d sl accent...	.....	.....	.....	.....	88	60	65
F.	"	31	7	L. > R.	97	68	—	2.75	7.75	10.5	1st double.....	.....	.....	.....	.....	.....	.....	77	75	75
F.	"	25	3	L. > R.	87	62	—	1.75	9 5	11.25	1st double gallop rhythm.	1st weak; 2d accent.	1st weak; 2d double.	.....	.....	My ocarditis.	.....	65	65	70
M.	"	33	4	R. > L.	108	78	+	4.5	7.5	12.0	1st double	2d metallic....	Systolic bruit; 2d double.	.....	.....	My ocarditis.	.....	70	62	55
F.	"	40	2	R. > L.	106	74	—	2.0	6.5	8.5	1st double.....	.....	.....	.....	.....	.....	.....	85	75	75
M.	"	37	1½	L. > R.	107	75	+	3.0	10.25	13.25	1st double gallop rhythm.	2d accent.....	1st weak; 2d dbl. accent.	.....	.....	My ocarditis, dilatation.	.....	75	65	65
M.	"	17	⅓	L. > R.	96	66	—	4.5	6.5	11.0	1st double.....	.....	1st weak; 2d accent.	.....	.....	.....	.....	77	75	75

M.	III	40	5	R. > L.	110	75	+	5.75	9.5	15.25	1st double.....	.....	2d sl. accent...	.....	.....	My ocardi-tis.	.....	72	80	65
F.	"	21	5	L. > R.	93	67	—	3.75	10.75	14.5	1st double.....	.....	2 d accent double.	.....	Pleuropericar-dial adhe-sions.	My ocardi-tis, dila-t on.	.....	77	87	75
F.	"	30	7	R. > L.	135	94	+	7.25	4.75	12.0	1st double gal-lop rhythm.	.....	2d accent.....	.....	.....	.....	.....	77	60	55
M.	"	46	10	L. > R.	84	68	+	4.0	9.5	13.5	1st double.....	1st accent.....	1st accent.	.....	Pleuropericar-dial adhe-sions.	My ocardi-tis.	.....	85	77	80
M.	"	26	4	L. > R.	78	54	+	3.75	7.0	10.75	1st double.....	.....	2d accent.....	.....	.....	My ocardi-tis.	.....	75	70	70
M.	"	27	2	L. > R.	138	120	+	4.0	12.0	16.0	1st double.....	2d accent.....	2d accent.....	.....	Pleuropericar-dial adhe-sions.	My ocardi-tis, dila-tation.	Athlete....	92	85	85
M.	"	25	1¼	R. > L.	102	84	—	5.25	9.0	14.25	1st double....	2d accent.....	2d accent.....	.....	.....	.....	Athlete....	80	80	77
M.	"	35	15	R. > L.	122	96	+	5.5	8.5	14.0	1st double.....	2d accent.....	2d accent.....	.....	Pleuropericar-dial adhe-sions	.....	.....	90	70	85
M.	"	26	2	R. > L.	120	102	+	5.5	9.0	14.5	1st double.....	2d accent.....	2 d accent double.	.....	.....	Hy pertro-phy.	Athlete....	87	70	80
M.	"	37	1	R. > L.	120	105	+	3 75	8.5	12.25	1st double.....	.....	.....	.....	.....	.....	.....	80	75	68
M.	"	22	2	R. > L.	112	95	—	4.5	10.0	14.5	1st double.....	2d accent.....	2d accent.....	.....	.....	.....	.....	85	80	80
M.	"	61	37	R. > L.	130	78	+	3.75	9.0	12.75	Systolic bruit (functional).	2d accent.....	2d accent.....	.....	Pleuropericar-dial adhe-sions.	.....	Arterio-sclerosis.	92	88	70
M.	"	35	2	L. > R.	104	88	+	4.25	8.75	13.0	1st double ir-regular.	1st weak.....	1st weak; 2d dbl. accent.	.....	.....	.....	.....	75	62	75
M.	"	25	2	R. > L.	100	78	—	4.75	6.75	11.5	1st double	.....	2 d accent double.	.....	.....	.....	.....	80	67	70
F.	"	34	5	L. > R.	94	74	+	3.5	8.5	12.0	Gallop rhythm.	.....	2d accent.....	.....	.....	My ocardi-tis.	.....	75	75	87
M.	"	28	7	L. > R.	74	60	+	3.5	8.25	11.75	Bruit function-al, double.	2d accent.....	2d accent.....	.....	Pleuropericar-dial adhe-sions.	My ocardi-tis.	.....	78	85	90
M.	"	22	2	L. > R.	122	98	+	4.5	9.0	13.5	Bruit func-tional.	2d accent.....	2d accent.....	.....	.....	Sl. myocar-ditis.	.....	82	75	77
M.	"	32	4	L. > R.	120	95	+	2.75	10.0	12.75	1st double.....	.....	2d accent.....	.....	Pleuropericar-dial ad he-sions.	.....	.....	80	70	65
F.	"	45	3	L. > R.	112	85	—	2.25	10.0	12.25	Double.....	1st weak; 2d double.	sl. accent; 2d double.	.....	.....	.....	.....	80	65	85

outflow of blood in the vessels. When, owing to contraction of one or both lungs, traction is made on the pericardium through its attachments with the pleura, a very different condition is brought about and the heart is forced to work at a great disadvantage. This can be readily understood if we take an extreme case in which the heart has been drawn up and out until the apex is found in the left axilla owing to marked contraction of the left upper lobe and compensatory emphysema of the right lung. In such a case we must assume that the pericardial space is encroached upon, thus interfering with the free movements of the heart itself. The traction of the pericardium on the vessels not only changes them from their natural position, but bends them in their course and thus throws an extra amount of work on the heart. What is true in this extreme case is also true in those of less degree, but to a less extent. For the heart to perform its function under these difficulties would tax a heart muscle of normal power; but, in cases like these, the heart muscle is perhaps always the seat of degenerative change and consequently less able to meet the increased work thrown upon it.

In determining the deep cardiac dulness in order to estimate the size of the heart, I found considerable difficulty. In the first place, it is very difficult to percuss the deep borders with accuracy. The degree of error may be reduced to a minimum, however, by the employment of several different kinds of percussion to check each other. I used two or more of the following methods in each case: finger-finger percussion, Ebstein's percussion, rubber-tube percussion and auscultatory percussion.

There is some doubt as to what should be considered the normal limits of the normal heart. Of course, this differs according to the age and size of the individual and size and shape of the chest. Reiss[2] gives the following measurements from averages taken from a number of medium-sized individuals:

Distance of right border of heart from median line, third interspace, 2.75 cm.; fourth interspace, 3.75 cm.

Distance of left border of heart from median line, third interspace, 4.75 cm.; fourth interspace, 7.5 cm.

Sahli,[3] commenting on these measurements, says that they make the heart too small. If we take these measurements as a standard, however, it will be seen that a large percentage of the hearts in patients suffering from advanced tuberculosis are displaced. Of the 162 hearts examined, 58 were in practically the normal position, 27 were displaced to the right and 77 were displaced to the left. Of the Stage I cases one was displaced and 10 were in normal position. Of Stage II cases, 4 were

2. Reiss: Ztschr. f. klin. Med., 1888, xiv, 12.
3. Sahli: Diagnostic Methods, Philadelphia, 1905.

displaced and 17 were in normal position, and of Stage III cases, 99 were displaced and 31 were in normal position.

The question of the size of the heart has been one of great interest for many years. Of course, the measurements here given furnish no accurate data on this question because they are taken from patients in whom the disease has existed for some time and consequently are records of pathological hearts. Of the 162 hearts examined, 70 were under 11 cm. in diameter measured on a level with the fourth interspace, and 92 were more than 11 cm. Of course, it must be remembered that we are not always measuring the same cross-section of the heart, for in the displacement that occurs different portions of the heart are found on a line with the fourth interspace. It must also be understood that no effort was made to give the greatest cross-diameter of the heart in these measurements. It is very probable that these figures are somewhat in error as to the size of the heart, for signs of hypertrophy of the right ventricle were present in a large number of the cases, which, together with the traction of the pleura on the pericardium, as it often occurs in the advanced cases, would probably have a tendency in certain cases to force the heart backward. Thus a considerable degree of enlargement of the heart might be present without enlarging its borders as found on percussion. The same condition results from the fact that as the heart is pushed to the left it is forced to turn backward by the fact that the walls of the chest turn backward.

CONCLUSIONS

1. A relative low blood pressure is found in tuberculosis, especially in advanced tuberculosis.

2. The factors which favor low pressure are the effect of the toxins on the vasodilators, the weakness of the heart muscle and general wasting.

3. The factors which have a tendency to maintain pressure are hypertrophy of the heart muscle and thickening of the systemic arteries.

4. Thickening of the systemic arteries occurs perhaps as a result of the action of the toxins on the vessel wall and is found especially in patients who have had tuberculosis for some time.

5. Myocarditis is a condition very common in advanced tuberculosis and one which, if recognized, yields to appropriate treatment in many instances.

6. It is difficult to give an opinion on the heart tones in advanced tuberculosis because conditions surrounding the valves are changed by such things as infiltrations, cavities, emphysemas and contractions.

7. In the majority of advanced cases (99 out of 130) the heart is displaced and working at a disadvantage.

8. In estimating the size of the heart it must be remembered that as the heart pushes over to the left it pushes backward and consequently the lateral diameter as taken on a level with the fourth interspace does not give an adequate idea of the real or true size of the heart; also that the hypertrophy of the right heart often throws the left ventricle backward, producing the same result.