

STRAINS FROM PASTEURIZED MILK

B. coli communis	Thermal Death-Point, C.	
	15 Minutes	30 Minutes
Strain 30	+ 66-67	+ 62-63
Strain 6	+ 65-66	+ 63-64
Strain 11	+ 65-66	+ 63-64
Strain 12	+ 62-63	+ 60-62
Strain 26	+ 58-59	
Strain 2	+ 57-58	
Strain 14	+ 57-58	
B. coli communis immobilis		
Strain 1	+ 57-58	
B. coli communior		
Strain 29	+ 67-68	+ 64-65
Strain 16	+ 64-65	+ 62-63
Strain 34	+ 64-65	+ 62-63
Strain 7	+ 59-60	
Strain 13	+ 58-59	
B. coli communior immobilis		
Strain 33	+ 63-64	+ 62-63
B. aerogenes		
Strain 4	+ 59-60	
Strain 8	+ 59-60	
Strain 19	+ 59-60	
Strain 20	+ 59-60	
Strain 27	+ 59-60	
Strain 31	+ 59-60	
Strain 28	+ 58-59	
B. cloacae		
Strain 18	+ 63-64	+ 60-62
Strain 10	+ 60-61	- 60
Strain 9	+ 59-60	
Strain 17	+ 59-60	
Strain 15	+ 57-58	
Strain 32	+ 57-58	
Strain 5	+ 54-55	
Strain 21	+ 53-54	
Strain 25	+ 53-54	
B. coli liquifaciens		
Strain 22	+ 60-61	- 60
From Unheated Milk		
B. coli communior		
Strain 109	+ 58-59	
Strain 112	+ 56-57	
Strain 104	+ 54-55	
Strain 114	+ 54-55	
B. aerogenes		
Strain 105	+ 59-60	
Strain 115	+ 59-60	
Strain 108	+ 58-59	
Strain 107	+ 57-58	
Strain 103	+ 56-57	
Strain 111	+ 56-57	
Strain 113	+ 56-57	
Strain 106	+ 55-54	
Strain 110	+ 55-54	
Control		
Stock B. typhosus	+ 54-55	

SUMMARY

Of the thirty-one strains of various gas producing aerobes isolated from pasteurized milk, eleven possessed a thermal death-point above 60 C. for fifteen minutes. Three of these survived 63 C. for thirty minutes and three others survived 62 C. for the same length of time. One strain, identified as *B. coli communior*, possessed a thermal death-point of 68 C. at

fifteen minutes and 65 C. at thirty minutes. This was the most resistant strain of *B. coli* recovered. The least resistant in this group were two cultures obtained from unheated milk which were killed by an exposure to 55 C. for fifteen minutes. The highest thermal death-point of *B. cloacae* was found to be 64 C. for 15 minutes, 62 C. for thirty minutes. The lowest belonged to two strains which originally failed to ferment lactose broth but which later acquired this power. These cultures were killed on being subjected to 54 C. for fifteen minutes. No culture of *Bact. aerogenes* isolated survived 60 C. for fifteen minutes. Different strains of this organism varied comparatively little in regard to their thermal death-points. All those recovered from pasteurized milk were killed by 59 or 60 C. for fifteen minutes. Two strains from unheated milk failed to survive 56 C. for the same length of time.

CONCLUSIONS

1. As observed by De Graef and De Jong, certain strains of *B. coli* are not killed by a temperature exceeding that commonly used in pasteurization.
2. The thermal death-point of this and similar organisms is not a constant quantity but varies for different strains of the same bacterium. In the case of *B. coli communior* this variation was found to be as great as thirteen degrees.
3. The presence of *B. coli* in pasteurized milk cannot be taken as an index of its improper pasteurization or subsequent contamination.

THE SMALL HEART IN TUBERCULOSIS

A SUGGESTED PHYSIOLOGIC EXPLANATION

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The small heart, often found in tuberculosis, has long been looked on as being a predisposing factor in this disease. That small hearts and hearts smaller than normal are common in tuberculosis, and this, in early as well as late tuberculosis, seems to be well-established. Ordinary methods of examination, as well as orthodiagraphic records of excellent observers,¹ prove this to be a fact.

It will be recalled that Brehmer,² the founder of modern phthisiotherapy, being convinced by the researches of Louis, Rokitsky and Beneke that the small heart was a predisposing cause of tuberculosis, founded his method of treatment on the idea that the cure of the disease could best be furthered by strengthening and increasing the capacity of the small heart. Exercise, particularly graduated hill climbing, was a very important part in his program.

The small heart in tuberculosis has been accounted for by Martius³ as being due to constitutional weakness and as being associated with narrowing of the arteries. Grödel¹ says that it is an expression of malnutrition. He also says that the small heart is found in those suffering from enteroptosis and particularly those having narrow chests. If the small heart is found in tuberculosis, enteroptosis and those having narrow chests, it is well to make inquiry in order to find out whether or not there are factors affecting the

1. Grödel: Lehman's Med. Atlanten., München, 1909, vii.

2. Brehmer: Die Etiologie der chronischen Lungenschwindsucht, 1885.

3. Martius: Pathogenese innere Krankheiten.

circulation which are common to these three conditions, and to see if such factors could in any way alter the size of the heart.

That not all persons with small hearts develop tuberculosis is evident. Then it is necessary, if we are to maintain the theory that the small heart is a predisposing factor to tuberculosis, to explain why it is predisposing in one individual and not in another.

My conception of clinical tuberculosis is that it is a pure accident. Practically every one is infected with tubercle bacillus, as is shown by both clinical observation and pathologic examination. Whether the disease shall go on to an immediate active disease or assume a quiescence, at least temporarily, is largely a matter of suitability of the soil (whatever that may mean) and the size of the dose of inoculating bacilli. Whether the disease, which quiets for a time, shall ever become active again, or remain quiescent, or heal, depends on factors which we do not understand, although the number of bacilli in the inoculation is probably a factor. That the bacilli do multiply and form metastases in and about the apices of the lungs of a large portion of adults is now well known. This amounts to 63.4 per cent. of Hart's series of cases and 71.2 per cent. of Naegeli's. Whether this metastatic infection shall take on itself activity and produce clinical pulmonary tuberculosis does not necessarily depend on an undernourished condition, for the well-nourished and those of stocky build are often victims of clinical tuberculosis. The small heart theory of the cause of tuberculosis was propounded long before the bacillus was discovered, but its disappearance has been exceedingly slow even though we now know the disease to be an infection. We know the infection is not a result of the small heart, for practically all have it, and it would be exceedingly difficult for us to show a definite causative relationship between the active clinical disease and the small heart. It does not seem rational that the size of the heart should be the determining factor in these cases.

It seems to me that a more rational explanation of the small heart in tuberculosis is that it is a result of compensatory circulatory changes; and that this is probably correct is suggested by the fact that the same small heart is found in enteroptotic individuals.

This explanation is based on well-known physiologic facts. The most important direct forces in the circulation of the blood are the impetus given the column of blood by the heart during systole and the elasticity of the vessel walls. The most important accessory mechanism is the pumping effect on the returning blood exerted by the inspiratory enlargement of the thoracic cavity. Every inspiration increases the negative pressure within the thoracic cavity, dilating the large veins and heart chambers. The amount of dilatation depends on the length of the inspiration and the degree of expansion of the lungs. There is no other mechanism that can take the place of this accessory action of the inspiratory act in its effect on the circulation. If inspiration is interfered with, the suction action on the intrathoracic vessels will be lessened and a diminished amount of blood will be delivered to the heart in a given time. The result of this is a consequent decrease in the total content of blood in the chambers of the heart and a decrease in the output of blood at each systole, which results in a relatively small amount of blood in the arteries and a damming back of blood in the systemic veins.

This condition is found in tuberculosis, and also in enteroptosis and other conditions which interfere with the contraction of the diaphragm and consequently with the freedom of the inspiratory act. Such patients have a relative arterial anemia and appear pale in consequence of it. On the other hand, they have an excessive amount of blood in the veins, particularly the splanchnics and the liver.

The heart usually delivers from 50 to 100 c.c. of blood at each contraction. In order for a given heart to deliver the normal amount of blood at each systole, the circulatory system must be in perfect functioning condition. The heart and arteries must be in a state of perfect compensation, and an adequate amount of blood must be delivered into the right auricle.

Inasmuch as this delivery of blood to the heart depends on a normal inspiratory expansion of the chest, any disturbance in the inspiratory act will decrease the amount of blood sucked into the heart, and if the heart and vessels are not able to compensate for this in some manner, there can be but one result, a lessened amount of blood in the chambers of the heart and a lessened amount delivered at each contraction.

Since the conditions which interfere with inspiration in tuberculosis are constant, at least extending over a long period of time, the heart must adapt itself to a smaller intake, a smaller content and a smaller output of blood for a like period. If the circulation is properly balanced, each chamber of the heart contains the same amount of blood, and the result is that by adapting itself to this smaller quantity of blood the heart must, of necessity, become smaller itself.

That the heart is an organ which adapts itself to various conditions there can be no doubt. Moritz⁴ has shown that there is a decided reduction in the size of the heart in the standing as compared with the reclining position. He has shown this in animals as well as man. Common observation shows the heart as enlarging and decreasing in size under pathologic conditions. We are justified in believing that any constant factor or any factor extending over a prolonged period of time which decreases the amount of blood delivered to and consequently the total amount of blood contained within the cavities of the heart at any one time would be met by a compensatory contraction of the organ as a whole. This is the condition in tuberculosis and enteroptosis. This may also account for the reduction in size of the arteries in the same conditions, as noted by certain pathologists.

The factors which interfere with the full expansion of the thoracic cavity come early in tuberculosis. They may be either the changes in the pulmonary tissue itself or the pleural adhesions which are often present (these are more potent factors as the disease progresses) or the reflex motor disturbance of the muscles of inspiration, particularly the diaphragm. Later these factors are exaggerated and others enter.

It seems logical that these hindrances to inspiration might produce:

1. A diminution in the size of the heart.
2. A diminution in the size of the arteries.
3. A relative arterial anemia with its consequent (a) lowering of blood pressure; (b) apparent anemia, which examination fails to confirm, and (c) congestion of systemic veins, particularly those of the abdominal organs.

4. Moritz: Ueber funktionelle Verkleinerung des Herzens, München. med. Wehnschr., 1908, No. 14.