

**ON THE TEMPERATURE OF
THE BODY AS A MEANS
OF DIAGNOSIS AND
PROGNOSIS IN PHTHISIS**

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On the temperature of the body as a means of diagnosis and prognosis in phthisis by Sydney Ringer

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BY

SYDNEY RINGER, M.D.,

PROFESSOR OF MATERIA MEDICA AND THERAPEUTICS AT UNIVERSITY COLLEGE;
PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL.

Second Edition.



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INTRODUCTION.

Two reasons influence the author to publish this second edition. First and chiefly because since the publication of the first edition our knowledge of phthisis has extended, so that it has become necessary to modify the phraseology to suit the altered pathological views. Thus, when the first edition was written, all forms of phthisis were considered to be due to two forms of tubercle, miliary or grey tubercle and yellow tubercle. At that time catarrhal or scrofulous pneumonia was not recognized. The lung induration forming that kind of phthisis now called fibroid lung was at that period attributed always to tubercle, and was supposed to indicate an effort to heal the destruction caused by it, indeed regarded as cicatrized tissue.

The second reason is that my little book has been curiously misread or perverted. It would seem indeed that some authors who have cited my views can hardly have read my book. Thus to select one instance from several:—Dr. Bathurst Woodman in his excellent translation of Wunderlich on Temperature says “I am loth to differ from Dr. Ringer but if I understand

him aright that there is an elevation of temperature in all cases of tubercular deposit, I am compelled to do so, if that statement be intended to apply at all times after the deposit of tubercle has once taken place." Now the main object of my work was to show the very reverse of what is here attributed to me. I had sought to prove that the temperature is raised only during the formation of the deposit, and that when this ceased then the temperature became normal; and that these thermometric indications afforded a delicate and a valuable test of the continuance, the amount, and the cessation of the tuberculization. A test, too, of scrofulous pneumonia, which when my book was issued had not been differentiated from tubercle. The very first proposition formulated in my former work says "there is probably a continued elevation of the temperature of the body in all cases in which a deposition of tubercle *is taking place* in any of its organs;" and further on it is said "In two of the remaining cases not only was there no elevation of the temperature but no increase could be detected in the physical signs, and on making the *post-mortem* examination the tubercle was found to have undergone retrograde changes and to have become obsolescent. No recent tubercle was found. Thus in the case of Cove, the cavities in both lungs were surrounded by thick tough fibrous walls and the grey granulations were shrunken, causing slight puckering of the surrounding lung tissue, extremely hard, and for the most part enclosed in their centre a small amount of cretaceous matter, they, moreover, contained and were immediately surrounded by much black pigmentary matter. The lung tissue also between them was tough and fibrous. Thus in all the cases observed in which the deposition of tubercle was going on, there was a continued elevation of the

temperature, whilst in those cases *in which the deposition of tubercle had ceased the temperature was normal.*"

At page 5 it is stated "it is probable that by means of the temperature we can conclude that the deposition of tubercle has ceased, and that any physical signs that are present are due to obsolescent tubercle and chronic thickening of the lung tissue between the tubercular deposits." Again a few pages further on we say that "in those cases in which the temperature becomes and remains normal the deposition of tubercle ceases." In fact on almost every page similar statements are to be found.

Clinically we recognize three forms of phthisis, catarrhal or scrofulous pneumonia, true tubercle, and the fibroid lung. These are the three forms of phthisis with which we have to do in this work. Scrofulous pneumonia affects the minute bronchial tubes and air vesicles. True tubercle the blood-vessels, lymphatic vessels and glands, and the connective tissue corpuscles. The fibroid lung is a hyperplasia of the connective tissue, the new tissue being imperfectly developed.

Thus in catarrhal or scrofulous pneumonia there is catarrh of the smaller bronchial tubes extending to the air vesicles. The thick tenacious mucus blocks up the minute bronchial tubes and produces collapse of the vesicles, and their vessels becoming bent and twisted the circulation is impeded, whence results œdema, and following this, induration and pigmentation. Through the catarrh of the air vesicles their cavities become filled with low formed cells, these accumulations either soften and rapidly lead to excavation, or becoming inspissated and undergoing fatty degeneration they form those cheesy masses which may become cretaceous, but more generally soften and

slowly form cavities. The cavities, cheesy masses, and obstructed bronchial tubes, act as irritants, causing hyperplasia of the contiguous connective tissue, thus producing one form of fibroid lung. In severe cases of phthisis with rapid formation of cavities and breaking down of the lungs, the air vesicles in very large numbers are mainly involved.

True tubercle consists of small rounded bodies varying in size from an almost imperceptible point to that of a large pin's head, generally pretty equally scattered through the lungs, often affecting many other structures, and in the disseminated form mostly occurring in children. Tubercles are probably produced by proliferation of the connective tissue corpuscles, or of the cells around small arteries and veins, situated most likely in the perivascular canals; or of the cells of the lymphatic vessels, or of those minute lymphatic glands which Dr. Sanderson has shown exist naturally in the lungs. According to the old views, still held by some excellent pathologists, these "grey granulations" may undergo fatty degeneration, and become opaque and then soften and lead to cavities. These bodies often become obsolescent.

The foregoing forms of lung disease are intimately associated, and pathologists now commonly teach that both forms may be generally found associated. Thus during the formation of tubercles the active cell growth involves the cells lining the air vesicles, thus producing catarrhal or scrofulous pneumonia. On the other hand the cheesy masses of catarrhal pneumonia, acting as centres of infection, induce a local and even general deposition of tubercle.

It is true that catarrhal pneumonia and tubercle are now differentiated into distinct diseases, but when the first edition of this book was issued they were considered identical. Now

as these diseases generally co-exist they will for the most part be spoken of together, so that only slight verbal alterations are necessary on account of our new pathological knowledge.

In fibroid phthisis portions of the lung become indurated by the formation of large quantities of low-formed connective tissue. It may originate in different ways, but is generally a consequence of catarrhal pneumonia. Thick tough walls surround the cavities, and indurations form between them and the cheesy masses. Fibroid phthisis extends often, slowly, generally by means of catarrhal pneumonia or the formation of small quantities of tubercle in the neighbourhood of the indurated lung. Sometimes fibroid phthisis spreads slowly, unhelped by these incidents. At other times the cavities dry up, the indurated tissue ceases to extend, and contracts when the lung may be considered healed. In a case like this the induration may truly be considered cicatricial.

In this edition the original number of cases, twenty-four, cited in the first edition is retained. A large number of cases, carefully observed during many subsequent years, serve but to confirm the author's original investigations.

In the following pages the terms fever, and elevation of temperature are used synonymously.

