

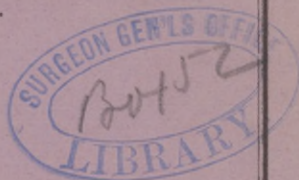
*Gleitsmann (Wm.)*

ON THE  
NATURE AND CURABILITY  
OF  
PULMONARY PHTHISIS.

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BY  
WM. GLEITSMANN, M. D.,  
OF  
BALTIMORE.

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1874.



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PULMONARY PHTHISIS.

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TRANSLATION OF A LECTURE DELIVERED BEFORE THE  
GERMAN MEDICAL ASSOCIATION OF BALTIMORE.

BY

WM. GLEITSMANN, M. D.,

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PROF. E. S. GAILLARD, EDITOR.

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But few other departments of internal pathology have undergone so great, and we may say so beneficial a change during the last ten years as the doctrines of pulmonary phthisis, which I purpose this evening briefly to sketch. Views that up to that time had been considered infallible dogmas, were overturned, the number of their adherents growing less from day to day. In the field of pathological anatomy the new theory has been generally accepted, chiefly through Virchow's labors. Notwithstanding the stern antagonism with old, deep-rooted prejudice, it has gained strength continually by the force of its own merit, so that now the truth of its principals is acknowledged by almost all authorities.

I have endeavored in so far as the literature was accessible to me, to do justice to all views of importance and to classify them. To this end I will commence by briefly reviewing the old theory, and after relating the facts and investigations, which induced its overthrow, proceed to explain the new doctrine in accordance with the conceptions of various scientists. As the views of the etiology of the disease have changed with the new pathology, I must touch upon this point also, before passing to the sympomatology. I beg to remark at the outset, that it is not my intention to give an exhaustive description of the symptoms and course of the disease, but to discuss these only in so far as it may be requisite to distinguish the differ-

ent morbid processes and phenomena. Nor shall I in therapeutics enter into details of the various remedies generally recommended and adopted, but merely enlarge on those primary principles, which alone can serve as a basis for a cure of the disease.

When you take up an old hand-book of the practice of medicine and turn to the chapter on pulmonary phthisis, you will generally find two paragraphs upon the subject, the one treating of chronic, the other of acute tuberculosis of the lungs, the former being sometimes styled chronic consumption, the latter acute miliary tuberculosis. The first disease commences by a deposit of miliary growths, called tubercles, which, according to their metamorphosis, as well as their slow or rapid development, produce the well-known symptoms. It was Laënnec, the estimable father of percussion and auscultation, who represented the formation of tubercle to be the primary pathological symptom in a lung diseased by phthisis, considering all further alterations as secondary consequences of tubercle. According to his idea, consumption and tuberculosis were one and the same disease, having always a constitutional basis, but never being developed from other local disorders. A chronic catarrh presenting the symptoms of phthisis in its further development, was according to his view, caused by secondary irritation due to deposit of tubercles. It was in like manner his opinion, that in all cases where the first traces of consumption closely succeeded a bronchial hæmorrhage, tubercles were developed simultaneously or even previous to the hæmorrhage. Laënnec regarded, as indeed we still do at the present day, the tubercle-granule to be a new growth, but he attributed to it special qualities, which do not in reality pertain to it. The most frequent alteration to which the tubercle is subject, is the caseous metamorphosis. The fresh, grayish, transparent tubercle does not possess any blood-vessels of its own; as it is therefore nourished with difficulty, it becomes all the more readily a prey to caseation and anæmic destruction, when its nourishment is impeded by pressure or the close proximity of other tuberculous deposit.



As it was the belief in former times, that tubercle alone possessed the capability of undergoing this caseous metamorphosis, the logical deduction was drawn from this assertion, that all such caseous deposits were the result of former tubercular formations. It was this opinion, however, that, by presenting the point of attack, gave the first impulse to reform and total subversion of the doctrines of Laënnec.

Virchow has proved, that not only tubercle has the property of assuming the caseous metamorphosis, but that other growths of entirely different nature, as for instance old cancer-knots, swelled lymphatic glands, hæmorrhagical infarctus, etc., can show quite the same changes,—that this is therefore no specific quality of the tubercle. The assertion, that every such caseous alteration was a tubercular infiltration, a tuberculization, became obsolete; Niemeyer having for some time argued in opposition to it.

As a further proof against the identity of consumption and tuberculosis of the lungs, it has been frequently demonstrated upon minute examination, that, what had been formerly considered tubercles, showed themselves to be the smallest bronchi with caseous contents or condensed alveoli with caseous infiltrations. In numerous post-mortems of consumptives, the remarkable fact was established, that not a single tubercle could be found in the lungs, but all changes found were the results of other processes, chronic and indurating pneumonia, on which I will touch later. In such cases, where besides condensation and destruction of the lungs, tubercular deposits are to be found, an unprejudiced examination will frequently elicit the fact, that the tubercles, when accompanied by those pathological changes alluded to, have been developed at a later stage. The tubercle is to be most frequently met with and in closest proximity at those places, where the destruction is most advanced, that is to say, where the original disease had its commencement and first seat. In like manner caseous deposits are found almost without exception in other organs, when tuberculosis appears in lungs previously sound and free from caverns, this being surely a proof

of the dependence of tubercle on other products of caseous metamorphosis.

That the miliary tubercle finally can not be considered an exclusive characteristic of individual disease, has been proved by the inoculations of various experimenters. Villemin in the year 1866 published the results of his attempts at inoculation in the "*Gazette Hebdomadaire*"; he injected the matter of tubercle as well as caseous substance. Others, and amongst them Lebert, confirmed and extended these researches. According to Villemin the mere injection of caseous substance was sufficient to produce miliary tubercles in rabbits and guinea-pigs. He obtained the same result with tubercle-substance. It was afterwards shown that by injection of matter, as Lebert did, or by embodying blotting-paper or gutta percha (Cohnheim and Fränkel), sponge or cork (Ruge), mercury and coal (Lebert, Wyss,) granular anilin (Waldenburg) miliary bodies can be produced. These experiments positively teach us the non-specific qualities of the tuberculosis, and the local production of tubercle in consequence of various interferences. The former assumption of a constant constitutional basis of tuberculosis must be therefore relinquished.

In these words the main principle of the new doctrine is expressed. Whilst Laënnec traces all the caseous knots and caverns in the lungs back to previous tubercles, the conviction has been obtained by reason of the above data, that in most cases the basis of consumption does not in the least consist in tuberculosis, but nearly always originates from various pneumonic processes.

Although scientists without exception adopt the opinion that consumption seldom or in very rare cases commences with tubercular deposits, their views nevertheless diverge in regard to the pneumonic processes giving rise to consumption; nor has an agreement so far been arrived at. The greater majority, with Virchow in the lead, declare, that during pneumonic changes the primary exudation is deposited in the free spaces of the smallest bronchioles and alveoli, and is observed in croupy, acute and chronic catarrhal pneumonia. On the other



hand, two investigators of our day, Rindfleisch and Buhl, look upon the inflammation as parenchymatous and interstitial. Last of all I must mention, that Niemeyer, although he coincides with Virchow in all other respects, believes bronchial hæmorrhage, by irritation of the alveolar walls to be productive of inflammation and consequent caseation.

If we proceed to regard the various forms a little closer, it will be found, that amongst the abnormal terminations of croupy pneumonia, tuberculization, or to designate it more correctly, caseation was known long ago. The conditions requisite to its appearance are not yet elicited, since healthy persons, as well as those already possessing casaceous deposits and caverns, are subject to this termination of pneumonia, which, however, takes place but seldom. Emphysematous patients are more liable to it than others.

In acute catarrhal pneumonia, where we have an extension of the catarrhal process from the minutest bronchi into the alveoli, these latter become crowded with young, newly-grown cells; the tissue of the lungs condenses, assuming at first a brownish-red color on account of the hyperæmia; and later growing paler. The diseased parts can, as in croupy pneumonia, again be made accessible to air and restored to soundness through changes in the exudation leading to the formation of mucus, through fatty degeneration and resorption of the deposited cellular exudation. In unfavorable cases, however, the cells crowd together still closer, the fatty metamorphosis remains incomplete, and in turn the tissue of the lungs changes to a dull, yellow, caseous mass. Catarrhal pneumonia, appearing mostly during childhood as a companion of measles, whooping-cough, croup, rachitis and scrofulosis reveals, according to the careful researches of Ziemssen, its complication with catarrhal bronchitis by an increase of temperature up to 104° F. and of the fever-symptoms generally. Not only children are liable to it, but particularly weak, delicate adults, with whom there is a tendency to repetition. Strong, healthy persons can likewise become subject to its attacks, running the risk of consumption by the change of the

profuse cellular exudation into caseous substance. Such cases, taking a very rapid course, are vulgarly termed galloping consumption.

Chronic catarrhal pneumonia is succeeded by symptoms that were formerly considered tubercular infiltrations and gelatinous infiltrations. The disease is quite as frequent as was formerly asserted of tuberculosis of the lungs. The pathological action is the same as described above. It can be readily understood, that, during the tedious progress of the disease and the massing of cells in the alveoli, these deteriorate by adjacent pressure, and shrinking become subject to the caseous infiltration. Here fatty metamorphosis and resorption of the exudations are possible, but it is apparent that this takes place with less ease and frequency. In such cases condensation of the lungs would again disappear and the patient recover. All that has been said of the acute form applies as well to attacks of chronic catarrhal pneumonia, to which, however, grown persons are more disposed. While in previously healthy persons this form of pneumonia forms the foundation of all those disturbances of nutrition that lead to consumption, in already diseased constitutions it becomes the means of extending and increasing degeneration and condensation.

Before I proceed to investigate the views of Rindfleisch and Buhl respecting the pneumonic processes favoring consumption, permit me shortly to review all that has been treated of.

1. We see that a neglected, protracted catarrh can very easily extend into the alveoli of the lungs, inducing caseous infiltrations and consequent consumption by deficient fatty change or through pressure exerted by the cellular exudation.

2. That as a rule the condensation and destruction of the lungs of consumptives are in the majority of cases the results of pneumonic processes.

3. That the patients are more subject to the danger of such disease and deleterious products in proportion as they are weak and badly nourished, inasmuch as the inflammatory processes have a decided tendency to cell-proliferation.

In passing over to the views of Rindfleisch and Buhl, I beg



to request you not to forget, that, however they, and especially the former of them, again approach the old opinion, neither of them believe in the dependency of consumption on the primary appearance of miliary tubercle in the tissue of the lungs, according to Laënnec. In the Transactions of the Medical Society at Bonn and at the meeting of German Physicians and Professors of Natural Science, held at Wiesbaden in September last, Rindfleisch has expressed his opinions, maintaining that those forms of inflammation causing phthisis and the primary caseous deposits, are occasioned by a parenchymatous inflammation, to which he ascribes a tubercular nature from the beginning. He argues this by reason of the similar histological construction of the miliary tubercle and caseous deposits, which both exhibit great accumulation of the lymphoid cells and granules in connection with giant cells. The process is said to have its rise in the bronchial walls, extending over the alveolar septa. As the miliary tubercle has its favorite seat in the walls of the blood-vessels, the vessels are likewise the starting point of the inflammation, which he consequently designates as perivascularitis or tubercular inflammation. The change from infiltration to caseation he assumes to be caused not so much by pressure, as by chemical alteration of the protoplasm and the nucleus of the cell.

Whilst Buhl also looks upon the inflammation in question as parenchymatous, he denies the possibility of a transition of croupy or catarrhal pneumonia, of chronic bronchial catarrh and bronchial hæmorrhage into caseation; the latter, according to his representation, always having a parenchymatous inflammation as its foundation. He holds, that this inflammation does not consist of superficial secondary disturbances, as in catarrhal and croupy pneumonia, but of a gelatino-albuminous infiltration and alteration of the alveolar walls and interstitial tissue. He calls this desquamative pneumonia, because it is accompanied by generation and shedding of bronchial and alveolar epithelium, the exudation consisting of desquamative epithelium. Buhl here further describes an inflammatory process of the finest bronchi, called peribronchitis, which, with a preponderating development of connective tissue corpuscles, has a



tendency to terminate in scaly induration, cirrhosis of the lungs, but which in connection with cellular development in the adventitia of the minutest vessels leads by the interruption of the circulation to tissue-death and caseation. The desquamative inflammation is often connected with peribronchitis.

In opposition to the views of both Rindfleisch and Buhl, Virchow maintains his standpoint with decision, insisting that the parenchymatous processes they speak of, if they exist at all, are of secondary character. The histological criterion which Rindfleisch finds for the tubercle, he declares untenable, and he warns earnestly against the introduction of old expressions into terminology, as this can only result in confusion.

In regard to Niemeyer's opinion, that bronchial hæmorrhage leads to inflammatory irritation and caseation, many contrary views have been advanced. After the injection of blood into the air-passages of animals (by Sommerbrodt), cellular elements were nevertheless found in the alveoli as a sign of catarrhal pneumonia. Niemeyer's view might consequently appear correct, and the more so, as clinical experience often shows perfectly healthy and sound persons to have been seized with phthisis immediately after a bronchial hæmorrhage and dying of it after a few months.

When caseous deposits have formed in the lungs through one agency or another, they can become cretaceous during rapid formation of connective tissue, or can exist for some space of time before they break down and give occasion to the formation of caverns. By far the greatest danger threatens the patient from the fact, that the existence of these caseous deposits favors the appearance of tubercle, thus tuberculosis being added to consumption. Tubercles so frequently appear in lungs containing the residues of chronic inflammation, that their frequent appearance leads us to conclude on a casual connection conditional on the caseous metamorphosis of the pneumonic processes. The reason why tubercles have a greater tendency to form in the lungs than in other organs, is explained by the fact, that lungs are much more easily inclined to diseases followed by caseous deposits.

Acute miliary tuberculosis, which I have not mentioned so far, being based upon the formation of numerous minute tubercular bodies in the lungs and other organs, is explained by Buhl as a disease of infection through preëxisting caseous deposits, with which view the clinical results might agree. Nevertheless, many observations have proved its appearance without the existence of such deposits, and a constant dependency on these latter can not always be traced.

Having detained your attention by such lengthy pathological explanations, I crave your kind indulgence on the ground, that on this basis I can more freely explain all remaining etiological, clinical and therapeutic deductions.

As to etiology, I shall only touch on the hereditary tendency, on the connection of scrofula with consumption, the influence of want of pure air and the inhalation of various dusty products,—passing over all other points, as we have already seen that delicate constitutions and all influences that materially impede the nutritive process, predispose to those diseases, which, by inducing a profuse formation of cells, give rise to consumption.

According to the views I have developed, you will concede, that we may speak of an hereditary tendency to phthisis, but not to tuberculosis. Those parents who were consumptive or suffering from other exhaustive diseases at the time of generation, can beget children who possess only small power of resistance. On reaching the age, when diseases of the meninges, the skin, the bowels, etc., are replaced by pneumonic processes, such children become more readily a prey to these diseases, than healthy children, being more subject to such dangerous influences on account of their constitutional anomaly. Hereditary tuberculosis can only be assumed in those cases where the children acquire tubercular meningitis from diseased parents; but even in such cases we shall seldom fail to find degenerated bronchial or lymphatic glands. I add the observation, that the statistic data represent about ten per cent. of hereditary tendency.

As scrofulosis has a tendency to copious accumulation of



cells in lymphatic and bronchial glands, which are with difficulty absorbed, those persons will be open to greater danger, who are at an age, when former scrofulous products have not yet been absorbed and lung diseases become more frequent, than healthy persons or such as have entirely recovered from previous scrofula.

The pernicious influence of impure air may be daily gathered from the reports of prisons, barracks, factories, foundling and orphan asylums. I will give you a few proofs in figures, which I have gleaned from Hirsch and Brehmer. Although mental depression has great weight with prisoners, its influence is nevertheless acknowledged to fade from day to day during confinement. Accordingly Baly states, that in the Millbank Penitentiary in London, amongst 1000 prisoners, there died from phthisis and scrofula during the—

First year of imprisonment.....	6
Second year of       "       .....	31
Third year of       "       .....	49
Fourth year of       "       .....	52
Fifth year of       "       .....	63

To prove that incarceration is the cause, it is shown, that of 3,249 prisoners admitted in 1844, only 15—viz., 4.6 (per thousand) were already subject to the disease. In the prison of Petonville there died on an average up to the year 1844, 11.14 persons, and the disease disappeared almost entirely as soon as better ventilation was introduced. Even animals are influenced by privation of fresh air. In Paris, for instance, those monkeys which were kept in magnificent but close cages, died of phthisis, whilst others, which were subjected to all the inclemency of an out-door habitation, enjoyed comparative immunity. I could easily increase these examples, but will only add one point more, which shall, at the same time, lead us to the last subject still to be discussed. In the whole State of Massachusetts the mortality by consumption averaged annually between 1841-49 3.0 of the entire population, whilst at Boston and the manufacturing city of Lowell alone it reached 3.8. We can



therefore recognize the unwholesome influence of factories, the air of which is burdened with dusty particles. I add the remark, that according to an assertion of L. Hirt, amongst 100 sick laboring men, who have dusty work, 22.5 suffer from phthisis, and amongst 100 sick workmen, who are otherwise engaged, only 11.1. The changes in the parenchyma of the lungs have been designated by various names, according to the quality of the dust that has penetrated, thus, for instance, coal-dust produces anthrakosis, gravel-dust chalicosis, iron-dust siderosis, or as Zenker also calls it, pneumonokoniosis siderotica, designating by these names the various forms of scaly induration and cirrhosis of the lungs. Hirt again has shown the connection of dust with development of consumption. Metallic as well as cotton-dust has a specially detrimental influence on phthisical pneumonias, on account of its mechanical irritation. Coal-dust acts less unfavorably and produces mostly bronchial catarrh.

As a discussion of the symptomatology of phthisis does not belong to the tendency of my lecture, I will confine myself to proving the existing difference in the phenomena of a simple consumption and the disease, when complicated by tubercular deposits, although the latter may have accompanied the caseous deposits from the commencement, or made their appearance secondarily. I shall, in like manner, add a few remarks on the acute miliary tuberculosis.

In these explanations I make use of a small comparative table which I have constructed. Although possessing no claims to exhaustive accuracy, it may, nevertheless, serve to throw some light on the subject, as the differences are thrown out in stronger relief here than at the sick-bed.

*Differential Diagnosis Between*

PHTHISIS.	PHTHISIS with CONSEQUENT TUBERCULOSIS	ACUTE MILIARY TUBERCULOSIS.
<p>The frequency of respiration we know to be accounted for by reduction of the breathing surface, by accompanying catarrh, by the painfulness of respiration and chiefly by the fever.</p> <p>Cough and expectoration frequently precede the disease, and are succeeded by fever, emaciation and pale skin.</p>	<p>Intense frequency of respiration and distressing dyspnoea succeeding previous slight shortness of breath, without evidence of increased condensation of the lungs are the most important symptoms.</p> <p>If cough and shortness of breath have, from the beginning, been accompanied by fever and emaciation, before the expectoration grows profuse, we may suspect tubercular phthisis.</p>	<p>Increased frequency of respiration without dullness on percussion and without bronchial respiration.</p> <p>Cough and expectoration begin only after the patient has rapidly grown weak, pale and thin.</p>
<p>Sputa are numerous, being clotted and sinking to the bottom of a vessel. Elastic fibres in the expectoration always point to destructive processes in the lung.</p> <p>Intestinal and laryngeal symptoms are rare.</p> <p>Fever sets in when catarrh extends from the bronchial mucous membrane to the alveoli. The fever is of remittent type, the difference between morning and evening temperature <math>1^{\circ}</math> to <math>1.5^{\circ}</math> Cels.</p>	<p>Hoarse, dull-sounding cough is always suspicious in advanced stages of phthisis. Sputa consist purely of phlegm, being tough and transparent.</p> <p>Intestinal and laryngeal symptoms appear in the course of the disease.</p> <p>Fever differences of much less account at morning and night, therefore a continued type of fever gives a much worse prognosis.</p>	<p>Hoarse, dull-sounding cough from its commencement, with tough, transparent sputa, without marked physical signs in the chest.</p> <p>Intestinal and laryngeal symptoms show themselves at an early stage.</p> <p>Fever and emaciation begin before expectoration.</p>
<p>Dullness, bronchial respiration, coarse rales; increase of these symptoms keep pace with the advance of the disease.</p>	<p>The percussion sound not dull but empty; rapid respiration and scanty expectoration point to a decrease in the air capacity of the lungs through tubercle.</p> <p>High fever and emaciation without corresponding progress of dullness.</p>	<p>At first, negative physical results with unusual shortness of breath, fever; later symptoms of condensation.</p>



In order to guard against misconception, I once more repeat, that the symptoms rarely appear in the decided and unmixed manner set forth above. In practice, the boundary will rarely be so clearly defined, especially between the two first diseases. I believe, however, that a consideration of the above remarks may assist in a decision in doubtful cases, which may be of importance to the physician as well as the patient; therefore I request that my remarks may be looked upon in this light only.

In turning to the last part of my subject, I shall make a few communications as to the spread of consumption, from which you will observe the existence of the disease in almost every country under the most diverse climatic conditions. In speaking of the regions that enjoy immunity from phthisis, we shall have to trace those conditions that are favorable to a cure of this disease.

If we take up the excellent historico-geographical pathology of Hirsch, and turn to this subject, we will find phthisis to be spread over all the five great divisions of the globe. The mortality amounts from 1.7 to 7.00 per thousand of the population, moving mostly between the figures 2.5 and 3.5. The last report of the Philadelphia Board of Health shows a similar proportion. In Philadelphia there died in the year 1872, of a population of 725,000 souls, the total number 18,987, of which 2,249 deaths resulted from phthisis, and accordingly 3.10 of its population. Baltimore, with 303,000 inhabitants, shows a total mortality of 8,851 in the year 1872 and 7,614 in the year 1873. The deaths by phthisis in 1872 amounted to 972, therefore 3.20, and in 1873 to 1,008, therefore 3.32 of the population. As other statistics correspond nearly to these figures with only slight variations, I will not further claim your attention by tedious iteration, but merely present you with a combination, showing, at a glance, the independency of phthisis from climatic influences. Hirsch gives three columns of figures representing the mortality by phthisis among 1,000 choosing those countries the temperature of which offers the greatest contrast to each other. The



degrees of temperature represent the average yearly standard, and are given according to Reaumur ( $4^{\circ} = 9^{\circ}$  Fahrenheit).

#### SCALE OF MORTALITY.

##### 1. Amongst entire population—

3.6—3.8 in Boston (7.3), London (8.4), St. Louis (10.3), Charleston (15.2).

3.3 in Copenhagen (6.0), at Malta (15.4).

##### 2. Amongst English troops—

3.5 in Newfoundland (2.8), Ionian Islands (13.0), Gibraltar (15.8).

3.9 in Canada (5.6), Mauritius (20.7).

4.3 in New Brunswick (3.6), Malta (15.4).

6.2 amongst European troops at Jamaica (20.5), and amongst the cavalry of the Guards in England.

##### 3. Amongst North American troops—

2.4 in the western inland States, West, (8.4); in the western inland States, South, (13.0), and on the southern boundary of Texas (17.9).

As these tables are constructed on older data, a few discrepancies with recent reports may exist, but they are not in any case of so much account as to deteriorate the present value of these figures. Hence you will gather that not only the latitude of places, but also dampness of air, as well as social conditions and mode of life, are without positive influence.

Of those places where immunity from phthisis exists, I have to mention the island of Iceland, the Faroë islands, the Kirghis-steppe, near Orenburgh, and elevated mountain altitudes. In the first three countries no conditions are to be met with that favor exemption from the disease. The long winter of Iceland and the Faroë islands, the severe storms, which are so intense as to upheave boulders and rocks, the air impregnated with salty particles, the intensely filthy habits of the people, who consume their meat and fish in a partially putrid state rather than when fresh—whilst the Faroëse washes his woolen clothes in urine—are surely influences that can in no wise be advantageous to patients. Nor can we apparently find any favorable conditions in the Kirghis-steppe, where the air is loaded with fine sand, and the coldest month shows  $-11.5^{\circ}$  R., the warmest  $+16.1^{\circ}$  R., on an average; in nine days,

from 17th to 26th December, 1839, a medium temperature of  $-24.8^{\circ}$  R. was observed, the lowest point of cold being  $-35^{\circ}$  R., and the greatest warmth in August  $+37^{\circ}$  R. in the shade. The endeavor has been made to explain the immunity of these three places by the mode of life of their inhabitants, as fat and fat-producing substances are consumed by the two former, and by the latter the koumiss or fermented mare's milk is consumed in enormous quantities. The dietary influences appear, however, to possess some intimate connection with the people's habits, as Icelanders, who have emigrated to Denmark, were attacked by phthisis. Many attempts have also been made to prepare the koumiss in other countries for the use of patients, but with little success. Only quite lately Dr. Simon in Berlin has advertised an establishment of this nature.

In turning our attention to elevated countries, we find that von Tschudi was the first to point out the non-existence of consumption on the Andes of Peru. Muehry, in his *Climatological Researches*, and Fuchs, in his *Medical Geography*, followed the subject further, finding immunity in many places. Of these I will only mention the table-lands of the Rocky mountains and their southern continuation, the Cordilleras de los Andes, further in the tropics the mountainous boundary of the northern coast of South America, the towns Santa Fe de Bogota, 8,100', Quito, 8,970', Potosi about 12,000', and particularly the Puna region of the Peruvian Andes, 11,000'—14,000', with the towns Caxamarca, Micuipampa, 14,000', Cerro de Pasca, 13,228'. In Europe the line is lower, and can be found in many places at 2,000' already, as for instance, on the highest points of the Harz, the Erzgebirg, the Carpathians, the Spessart, many places in the Alps, that have become known on this account, as in Pinzgau, Styria, Carniola, on the western section of the Pyrenees, in Switzerland, at an elevation of 4,500', whilst in many places below the level of 2,000' in the Vosges, the Jura valleys, the Odenwald, phthisis very frequently occurs. In Africa immunity exists, on the plateaus of Abyssinia; in Asia, on the high plateaus



of Armenia and Persia; on the highest points on the western Ghats and Nilgherry mountains in Lower Bengal, 4,000'—7,000'; in less degree also in the mountain districts of Java and the table-land Nuwera Ellyia of the Island of Ceylon, 6,500.

From this combination you will conclude, that constancy of the element of altitude is exhibited in the most varied latitudes. Moreover you will observe, that the nearer we approach the equator, the higher the elevation at which immunity commences. Before concluding, allow me to impress upon you once more the fact, that altitude is the cause of immunity. The want of connection between phthisis and temperature is clearly proved by the tables quoted from Hirsch. The same frequency of consumption in the United States as in other countries show that the greater dryness of the atmosphere in high places is not the reason alone. (The dryness of the air in America has been shown by the American naturalist Desor in a brilliant lecture delivered before the Swiss Meeting of Naturalists in the year 1853). The purity of the mountain air can not be accepted as the reason either, as the same influence fails to exempt the lowlands from disease. Nor again can the ozone of the air be made accountable, as just in winter-time, when the ozone is considerably increased, consumption so frequently takes its commencement. We can therefore deduce only the one fact of altitude, or what is synonymous, rarefaction of the air, the influence of which must be explained as follows. As I have endeavored to prove, phthisis is based upon a caseation of inflammatory products, which disposition to caseation is most probably to be found in a certain dryness or relative bloodlessness of the lung-parenchyma. The rarefied air produces a greater flow of blood in the peripheric organs, and especially in the lungs and consequently in the heart also. Therefore it acts in direct opposition to those conditions, which engender a disposition to phthisis, as I have already indicated in my article, giving a description of Waldenburg's portable pneumatic apparatus.\*

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\* Baltimore Physician and Surgeon, March 1, 1874.



In addition, mountain life induces an increased exercise of the chest in consequence of the increased respiration produced by the rarefied air. It will not be needful to explain more fully, that rich, nutritious diet especially of fat-producing substances, the greatest possible amount of fresh air, antipyretics in the widest sense, amongst which I also count the use of cold water, are not to be left unheeded.

On these principles several climatic establishments have been founded, whose happy results have amply proved the soundness of these theories in practice. The oldest of these is Dr. Brehmer's Institute in Gœrbersdorf, Silicia, Prussia, founded by him in the year 1854 under great difficulties. He has, up to 1869, treated 958 consumptive patients. Of these 315 were already in the colliquative stage (these at Madeira are quite excluded from the statistics). Of the total number of 958, only 47 patients died, or 4.4-5 per cent.; if you deduct 18 patients who were subjects of tuberculosis from these 47, a percentage of 3 deaths by phthisis remains, whilst 20 per cent. were *permanently* cured during the comparatively short treatment of eighty-six days. Besides Gœrbersdorf, Davos in Switzerland has also gained a good reputation by its successful cures.

Believing that I have shown you the curability of phthisis by conclusive facts, I beg only to observe to the practitioner, that he will be much more quiet and successful at the sick-bed in knowing the greater number of cases to be no deleterious new-growths, but only pathological processes, which are capable of amelioration by careful treatment, Nature herself having provided a climatic means of cure.

NOTE.—A most conclusive, logical and satisfactory demonstration of the effects of locality upon the origin and curability of phthisis.—E. S. G.







