Report on the Typhus Epidemic in Upper Silesia

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(Archiv. für Patholog. Anatomie u. Physiologie u. für klin. Medicin, 1848, Vol. II, Nos. 1 and 2)

CHAPTER 3. THE DISEASE

A. SYMPTOMS

The following communication relies to a large extent on my own findings. These I obtained in part by observations made at the military hospital in Sohrau, which is under the direction of Dr. Sobeczko, a man as well versed as he is experienced, and in part by visits to patients in Rybnik, Sohrau, Pless, Ratibor, Loslau, Lonkau, Radlin, Geikowitz and Smollna. The primary basis for this report, as well as for later control, was the information furnished by the local physicians and by those foreign physicians who had been residing in the area for some time. Whenever my remarks are based solely on such information furnished, I shall expressly point it out. The great number of patients occurring in a relatively small area allowed me to form a general view of the different stages of the disease in a short time, so that by combining the conditions observed in different individuals, a picture of the course of the disease could soon be constructed. I have later attempted to confirm the accuracy of this picture by sustained observation of individual cases at the military hospital. Results were further consolidated by the fact that the symptoms as well as the course of the disease showed excellent agreement in the different patients. Nevertheless, it is possible that, even so, my presentation of the facts might be incomplete in some points. This I fear mainly because my observations were made at a relatively favorable season and because the course of the disease might have been less benign a little before or after. Correction of such points also must be left to other observers.

In the normal course of the disease I believe it convenient to discern four stages: a prodromal stage of early symptoms, one of peak manifestation, one in which the disease abates, and lastly the stage of convalescence.

The symptoms, even the existence, of the first stage could only very rarely be recognized among the rural population, as most of the members of that "class of society" pay too little attention to slight symptoms to be able to give any information about them. A few, however, stated definitely that they had felt unwell and weak, had had pain in the joints, chills, headaches, nausea, etc., before the disease had broken out. This prodromal stage was more clearly apparent among foreigners, in particular among the physicians who fell ill under endemic influences. Dr. Beifel first felt so unwell on the 27th of February that he took the emetic he was commonly prescribing at the beginning of the disease; he then recovered to the extent that he resumed visiting his patients; on the 5th of March, however, the disease reappeared so pronouncedly that he had to return to Breslau. Prof. Kuh, whose definitely ascertained illness seems to date from the 19th of February, had symptoms before that date which he himself had considered to be due to catarrhal rheumatic fever. In others, the symptoms between the inception of the disease and the appearance of

Originally published in *Archiv. für Patholog. Anatomie u. Physiologie u. für klin. Medicin, 1848*. This English translation is from Volume 1 of Virchow, Rudolf, *Collected Essays in Public Health and Epidemiology*, edited and with a foreword by L. J. Rather, published in1985 by Watson Publishing International, Sagamore Beach, MA. All endnotes are from the original translation

clearcut manifestations were so indefinite that all were deceived with regard to them. Prince Biron of Courland had a violent headache after a restless night on the 24th of February and felt so unwell that his personal physician, Dr. Altmann, urged him to return to Breslau. However, these symptoms receded, so that on the next day he continued to Pless. I there saw him on the 27th, at a meeting of the local committee, most active on behalf of the sick, showing the eagerness and circumspection which made his presence in the districts so uncommonly beneficial. Later, at dinner, he ate with a good appetite and conversed in a most lively fashion. The next morning he returned to Breslau, where he busied himself in the interests of the Upper Silesians. When I came to Breslau on the 9th of March, I already found him on his sickbed, which a few days later was to become his deathbed. My friend, Dr. von Frantzius, while returning from Vienna to Berlin, visited me in Pless and accompanied me to Sohrau. There on the 5th of March he complained for the first time of feeling indisposed. After a restless night he got up with a headache and sore limbs; he had a slight fever and a fit of shivering, with a dry, hot skin. Towards noon he had an exceedingly abundant outbreak of perspiration, which greatly relieved him. During the following days he was quite well; his appetite was even very good; he only felt somewhat tired; sometimes he complained of slight pains in the limbs and he was rather quiet and listless. On the 10th, together with me, he arrived in Berlin pursuing his business in a sober-minded way; he helped in building the barricades on the 18th. The malady first struck with greater intensity on the evening of the 21st.

It is easily understandable that such light and transient symptoms followed by almost complete relief from all discomfort are not noted by the great majority of patients and that there is a tendency to post-date the invasion of the disease to a much later time than the facts of the case would warrant. I am drawing special attention to this point in particular, as I shall have to repeatedly come back to it so as to prove that information pointing to such later dates are doubtful. The majority of patients of all social strata reckoned the start of their illness from the moment when it had reached so high a pitch that they had to take to bed. From the cases mentioned it is plain that the disease might have already been present for at least 14 days, even though its manifestations were so mild and vague that no physician was in a position to assert its presence with certainty.

After the prodromal stage, the duration and symptoms of which do not seem to be constant, has persisted for some time, there begins the second stage of the disease, which usually reaches its peak very rapidly. In analogy to older authors it could be designated as the inflammatory stage, because it presents a number of symptoms of exaltation. However, the mainly depressive character of the affection, which will soon come to the fore, is also simultaneously apparent. The beginning of this stage is frequently marked by the occurrence of chills and shivers, which the patients describe as similar to those of the ague, which they know so well. Their duration is not constant; sometimes these attacks lasted for 1-2 hours and were then followed by fever without sweating, or they lasted for a short time only and were repeated after a few hours, or after a day or two. A few patients mentioned only a slight feeling of chilliness lasting for a while; others denied any feelings of chill whatsoever.

From then onward the rise of the skin temperature was permanent. In general, the skin was dry and the heat very soon became burning *(calor mordax),* and that to a higher degree than I have ever seen in other diseases. When I placed my fingertips on the forearm of a patient for half a minute to count his pulse, a disagreeable prickling sensation sometimes persisted for as long as 10-15 minutes. It lasted longest in the lobes of my ears when I once auscultated a patient by placing my bare ear against his back. Sometimes the skin was covered with a slight sweat, which was not always sticky, but in that case the stinging

sensation of the *calor mordax* became even more repellent. Skin turgor was then always increased, the face usually reddened, and the eyes somewhat injected and shining.

The intensity of the fever, inasmuch as it was directly apparent from the circulatory system, differed according to the nutritional condition of the individual. In strong and well-fed people the fever exhibited a somewhat inflammatory character during the first days. The contractions of the heart were frequent and energetic; counts of 96, 100, 104 and more rather large and full beats were soon reached; the tension of the arterial walls, however, was rarely strong enough to offer a noticeable resistance to the pressure of the fingers. In some patients the number of contractions of the heart barely reached the number quoted in the first days. In all undernourished persons the character of the pulse differed, the artery was easily compressible, the impulse of the blood wave was weak, the number of heart beats was frequently 110-120 per minute.

At the same time the movements of the intestine were slowed, defecation was rare; if it occurred, the feces were formed and hard. During this period the urine was reduced in quantity, acidic, clear, dark yellow and grainy. The tongue was moist, the finer papillae bore a whitish epidermoid coat, while the coarser papillae *(papillae fungiformes)* frequently were yet visible between them as red bodies, so that the surface of the tongue presented a piebald, spotted appearance. The patients felt relatively little thirst; their appetite was moderate, directed to sour dishes. In rare cases the tongue was heavily coated, sticky to the touch, whitish or yellowish, and the appetite gone completely, with nausea, and a tendency to vomiting or to diarrhea. The abdomen was always soft, usually full, but not swollen; greater accumulation of gases and flatulence were not common. I could not find any particular tenderness in any particular part of the body as a constant symptom. At first I thought that pain in the hypochondrium, especially on the right side, belonged among the peculiarities of the disease. But it soon appeared that this pain occurred only in the muscles, and that its apparently greater intensity in the places mentioned only stemmed from their being more thoroughly examined. Enlargement of the spleen, sometimes very extensive, was frequently diagnosed by palpitation and percussion; however, all these patients had previously suffered from malaria, which they had treated themselves. In those who had remained free from intermittent fever, there was no tumor of the spleen.

A moderate bronchial catarrh with a frequent urge to cough was almost universal during this period; the cough was tormenting, producing a somewhat viscous, frothy, glossy mucus that was sometimes mixed with a little blood and sometimes a little painful. The respiratory movements were slightly increased, the respiratory sounds were accompanied by whizzes and whistles. I have practically never seen the common cold in the nose, but I did see catarrh of the conjunctiva of a moderate degree.

The prostration of the patients was extreme. Not that they would have been unable to move, to stand up or even to walk, but they felt so fatigued and exhausted that they usually were apathetically lying on their backs. It therefore was not an actual, true weakness, but rather a feeling of weakness, an inhibition of the stimulation for movement; not the muscles *per se* or the conductive apparatus of the stream of stimuli (nervous system) must be considered as being affected, but the excitatory centers (ganglionic nodes). At the same time many patients suffered from so-called 'muscular pains.' These were most frequently located in the muscles of the lower extremities, the soles of the feet, the legs and also the thigh; not uncommonly pain was felt in the trunk, especially in the abdominal and dorsal muscles; I saw it rarely in the upper extremities. Sometimes it was exceedingly severe, tearing or pulling in nature, or involving a state of tension and increasing upon external pressure and on movement. The effect of exhaustion combined

with the pain in the pharyngeal muscle was peculiar: the patients usually said they could not swallow, and when they were prompted to do so they complained of pain. But at most a moderate venous hyperaemia was present in the mucous membranes of the pharynx and throat.

As regards central nervous manifestations, they were insignificant during that stage. Few patients complained of headache or dazedness. All were well able to describe their condition, reasonable, and in accordance with their national character even able to do so in a lively manner. The majority, however, were restless, particularly at night, and talking to themselves all along in a low voice. Almost all of them, and this was one of the most constant symptoms, complained, from the start, of buzzing and whizzing *(huczy)* in the ears, which they compared with the sound of flowing, foaming, water. Later, when the sensation intensified, which usually was the case, they described it as a knocking, like the clatter of a mill wheel. To the extent that these subjective manifestations intensified, the power of hearing diminished and the majority became acutely hard of hearing. It does not seem likely to me that the cause of these manifestations would lie in an affection of the nervous system or in the nerves themselves; the course of development of this deafness, combined with the complete lack of affection of the other sense organs as well as of the head, rather speak in favor of a catarrh of the mucous membranes, which, analogous to the catarrh of the aerial ducts in the respiratory tract, extends from the pharynx to the Eustachian tubes and the tympanic cavity. (Compare Pappenheim, in the *Zeitschr.f. rat. Med.* 1844, Vol. I. p. 335). Unfortunately, I have neglected to pay attention to this point in the post-mortem examinations so that I must now regard this condition as merely probable.

In such a manner, sometimes more and sometimes less severe, the symptoms usually manifest themselves during the first 3-4 days of this stage. It is seen that the most important among them pertain to the nervous system, and indeed it is not the brain alone that is affected but also the spinal and sympathetic apparatus. Usually after a severe shivering fit we meet with the peculiar burning heat of the skin, which even if we cannot trace it with certainty in the nervous system, never occurs without simultaneous significant changes of such a nature. At the same time we have a most important excitatory inhibition (i.e. probably changes in the ganglionic nodes), characterized by a great weariness of the limbs and a reduction in the peristaltic movement, in secretion, etc.; increased stimulation is only found in the nerves of the blood vessels and the muscles. The second significant group of symptoms is represented by the catarrhal inflammation of the mucous membranes, especially those coating the respiratory tract.

On the 3rd and 4th day, sometimes a little later, a new change used to appear, namely, exanthema of the skin. Since special importance has been attached to this symptom, we must deal with it in some detail. The exanthema preferentially appeared in the two forms that have been known for a long time in the history of typhous diseases.

The first form is usually designated as measles-like *(exanthema morbilliforme; rubeolous eruption).* It forms spots averaging 2-3 lines in diameter, but also smaller ones, which are mostly flat, rarely a little raised, of a pale bluish-red color, fading away at their borders; they disappear completely on pressure by the finger, and reappear very rapidly when pressure is released; they do not start solely or primarily from the vascular system of the hair follicles, since they are frequently seen to extend over several hair follicles at the same time; sometimes the hair follicles occupy an eccentric position and sometimes none are involved. Their shape is rarely perfectly round, but usually a little deformed, irregular, even slightly serrated. This exanthema first appears, almost without exception, on both sides of the lower part of the chest, in the area of the false ribs and in the epigastrium; from there it rapidly spreads to the

chest, abdomen and back, appears on the arms and hands and reaches the extremities; it is very rarely seen in the face, more frequently on the neck. It usually remains but for a short time: in some cases it begins to fade one day after its appearance, in others after 3-4 days, or more. The spots then become pale, so that, when looking at a larger area of skin, it is merely mottled; this slight mottling is easier to recognize from a little distance away than when the eyes are too close to the body. I have never observed subsequent desquamation. Whenever true flaking of the epidermis was seen it occurred also in parts that had shown no exanthema. Even where the spots had already disappeared, they reappeared with heat or with mild irritation of the skin, e.g. after washing with vinegar. Summarizing these facts, it will be easily concluded that we here have nothing but a compound capillary hyperemia of the skin. To determine the onset of this exanthema exactly is not possible, in particular because, as we have seen earlier, it was too difficult to determine the onset of the disease, or of its second stage in every individual case. I can best reconstruct the situation by the example of the physicians who caught the disease. Mr. von Frantzius had had prodromal symptoms of the disease since the 5th of March; the stage of culmination began on the 21st, and on the evening of the 23rd the first exanthema spots were observed. Mr. Beifel fell sick on the 27th of February; the second stage of the disease began on the 5th of March and when I visited him in Breslau on the evening of the 9th I found the exanthema developed on the upper part of the abdomen, the chest and the forearms; nothing of the kind had been observed a short time previously. Mr. Kuh felt seriously ill on the 19th of February; the first spots of exanthema were noticed in the morning of the 24th. These cases would seem to indicate that the exanthema appeared from the 3rd to the 5th day of the second (or peak) stage. I could further support this evidence with numerous other cases, the validity of which, however, is based entirely on the statement of the patient. Other cases, in contrast, seemed to indicate a much later onset of the outbreak. Thus I found this exanthema in a woman from the Klischtuwka near Sohrau who, according to her statement, had been ill for 7 days, and in whom Dr. Wachsmann, who had taken me to see her, had not observed anything on the previous day. But not all such cases had been under medical observation from the beginning; although the patients had been very tired, weary and dazed during the first days, they had only sought the physician's help when the intensity of the disease had increased. It might therefore be possible that in these cases too the above-mentioned timing (3^{rd}-5^{th} day) applies; however, I cannot assert this with certainty.

Another question, as difficult to decide, is the constancy of the exanthema. Whenever I had the opportunity to follow up the development of the disease from the first days onward under somewhat favorable conditions, I always observed the exanthema. This, of course, was not always possible in the cottages of the poor, as they were so dark and the beds stood in so distant a corner of the room, that none but the grossest of observations could be made. Frequently the skin was so covered with dirt that it was not possible to gain clarity as to the presence of exanthema. Some of the local physicians asserted emphatically that they had seen cases that had proceeded without exanthema; others did not wish to commit themselves, as they had not been able to make consistent observations in view of the imperfect manner in which they visited the excessive number of patients. In fact, it is easy to understand that a physician can rarely form a reliable opinion when he is only able to see a rural patient every 2nd or 3rd day, and that only under the most difficult of conditions, when he has at his disposal only very unreliable information on the case histories.

Lastly, there arises the question of differentiating between this exanthema and other similar eruptions. A conflict had arisen in the matter among the physicians at Ratibor, which has been publicized in the periodical political

press. Dr. van Decken, judging from the exanthema, definitely considered the disease to be measles, while Dr. Polkow denied this with equal emphasis. It now happened that, simultaneously with the typhus fever epidemic, there occurred in the districts an epidemic of measles that spread rather rapidly, in particular in the orphanages. However, this very co-existence most clearly showed that the exanthema of measles and the measles-like exanthema could indeed be distinguished from one another. Whereas the former regularly started on the face or at least affected it preferentially, the latter first appeared on the midriff, while the face was not affected in the majority of cases; the former formed more intensely colored, more rounded, slightly raised and densely crowded spots which rather constantly arose from the hair follicles, the latter exanthema remained paler, was more irregular in shape, flat, and mostly quite dispersed. Taking into consideration the other symptoms of the disease, confusion is hardly possible. I must, however, assert the total identity of this exanthema with the common typhus roseola. This is also sufficiently documented by the older descriptions of war typhus. Ign. Rud. Bischoff *(Beobachtungen über den Typhus und die Nervenfieber,* Prague, 1814, p. 8) described the *exanthema morbilliforme typhosum* which broke out between the 3rd and 5th day on the chest and the forearms and sometimes even on the face, as consisting of rose-red spots the size of a grain of wheat, variously shaped, often oval and one end tapering off like a flame; they did not change on being pressed with a finger, but differed from the red petechiae which were of a darker red, round, and like flea bites without the central dots. The only conflicting item in this description is the fact that the spot did not change under finger pressure. This, however, seems to find its explanation in a publication by Wedemeyer *(Űber die Erkenntniss und Behandlung des Typhus,* Halberstadt, 1814, p. 69), for that author stated that the primary petechiae, the name given to the roseola, almost all disappeared upon pressure, whereas later on they could not be made to disappear on pressure. In respect to this particularly, the war typhus thus approaches the *typhus fever* of the English (compare Valleix, *Arch. Gener.,* 1839, Sept.-Nov.), while the exanthema of the Upper Silesian typhus is exactly similar in its appearance, as well as in the areas of eruption, to the well known *tâches roses lenticulaires* of the *fiévre typhoide* and to the roseola of the German ileotyphus. The only difference from the abdominal typhus observed in Berlin concerns the number of the areas of eruption, these usually being very limited in Berlin. From the Paris typhoid fever might differ by the timing of its outbreak, which was observed from the 4th to the 35th day of the disease (Louis, *Recherches sur la fièvre typhoide,* 1841, Vol. II, p. 96). In relation to the first of these points, I must, however, point out that in Upper Silesia I have seen quite a number of cases in which the eruptions were not more numerous than in our abdominal typhus.

The second form of exanthema is the purpura-like or the petechial form. On their first appearance the petechiae are always small dots, at most the size of a pinhead, almost perfectly round, fiat and of a uniformly bright red color which does not change under finger pressure, i.e. they are not capillaries filled with blood corpuscles, but extravasations of blood. They appeared randomly on different parts of the body, sometimes on the trunk, sometimes on the extremities, without any discernible rules. I have never seen them on the face, though I did on the neck. Of this petechial exanthema I can assert with certainty that the onset of eruption is very variable. In Lonkau, Dr. Babel showed me 2 persons, a man and his wife, who were lying in a bed and had allegedly fallen ill simultaneously 4 days ago; both had petechiae on the most varied parts of their body. The first petechiae had appeared on the body of Mr. von Frantzius towards the end of the first week after the beginning of the acute stage. This time might generally be considered as the average for the

appearance of petechiae. This eruption, likewise, is not a constant manifestation in all cases. I have observed a number of patients up to the indubitable onset of convalescence without seeing petechiae at any time. When I came to the district, most of the physicians had not observed the petechial exanthema. After I had subsequently found several cases in Rybnik, several physicians told me that they had now also seen the petechiae, and that the character of the epidermis must thus have changed a little. Such a change would not be without analogy. Thus Kennedy observed the purpuralike exanthema to predominate in the Dublin typhus epidemic during the last months of 1836 *(Medical Report of the Fever Hospital, Cork Street, Dublin,* 1839, p. 17), whereas the rubeola-like exanthema had been more frequent in 1837. I could not find a definite relationship between the petechiae and the roseola in relation to either localization or time of eruption. A. Anderson *(Observations on typhus. Glasgow, 1840,* p. 20) mentioned that the usual eruption (the roseola) in the Scottish typhus sometimes changed to the petechial form, since the red color at first disappeared upon pressure, while it did not later on. I, however, wish to state that the petechial nature of the exanthema, the presence of a true extravasate, does not follow from non-disappearance under finger pressure. Wedemeyer (loc. cit., p. 71) objects with justice to such an identity with the petechiae of a roseola that cannot be suppressed by pressure, or, in his terminology, the primary and secondary petechiae; the former being merely caused by a congestion of the blood in the vessels together with a sort of exudation of the blood. I have autopsied a patient in Rybnik who in life had appeared to exhibit most numerous extravasations of the skin and on autopsy the still reddened parts showed only venous hyperemia with a surrounding imbibition of the hematin (compare *Archiv,* I, p. 442). I myself have never seen a real transition from roseola to petechiae. Where the petechiae had developed while roseola was still present, the petechial spots always appeared between the roseola spots; when the latter had already disappeared, the relationship could naturally no longer be ascertained. But the circumstances that the petechiae usually developed very abundantly in those areas in which the roseola exanthema developed least or not at all, e.g. on the feet and calves, do speak against any such transition. In the majority of cases I saw the petechiae appearing when the roseola had already disappeared or begun to fade. However, I have also repeatedly seen both side by side and, in one case in the Hospital at Sohrau, roseola was present on the second day only after admission to the hospital, while petechiae were already present on the first day. Finally, O'Reardon *(Medical Report of the Fever Hospital, Cork Street, Dublin, 1840,* p. 6) observed the petechiae more frequently among the poorer typhus patients than among those of the middle class and the wealthy. This too was not the case in Upper Silesia. The above-mentioned married couple in Lonkau were well-to-do farmers; they still possessed potatoes and stated that they had not yet suffered want. Among the roughly 40 other patients whom I saw in that village I found petechiae only twice; the majority of these patients were in the most miserable of condition, since the distribution even of flour had been organized only very recently.

This much on the exanthemata. I have here discussed both in detail, as the various questions involved could thus be most clearly exposed. Let us now return to the course of the disease.

From the moment when the exanthema first appeared the course of the disease varied in different patients. In general, let us distinguish a severe and a mild form, as a more detailed discussion of the individual cases would only serve to confuse the general picture.

During the development of the roseola in the mild cases, the various other manifestations tended to intensify. The buzzing in the ears and hardness of hearing, as well as the congestion in the head, increased; the face was more reddened, the patient felt somewhat dazed and became

mildly delirious, at least at night. In a few cases nosebleed was also observed. The skin was burning hot, mostly dry, and a little reddened all over; the tongue began to get dry, but did not acquire a heavier coating, so that we must assume the reason for this to lie merely in the high temperatures and in breathing with an open mouth. Neither the tongue nor the teeth exhibited the well known sooty efflorescence of abdominal typhus; the tongue merely turned yellowish, or yellowish brown, and presented a more flattened and dry aspect. At the same time respiration became frequently more superficial, the cough rarer and drier. In some cases there occurred at that time a slight diarrhea lasting for a few days, which usually overtook the patient so rapidly that the evacuations soiled the bed. The pulse now speeded up so that the majority of patients had above 100, more frequently above 110 pulsations per minute; its quality took a turn for the worse: though the flow of the blood stream remained sufficiently strong, the tension of the arterial walls diminished day by day. The muscle pain receded into the background, while the languor and weakness of the limbs increased markedly.

In contrast, very early the pulse frequency in the severe cases increased to 120 and even 140 per minute; the pulse was strong but easy to compress, the wave of blood was not sharply delimited but showed some fluctuation; breathing became more frequent and uneasy. After the tongue had become dry, yellowish brown and cracked on its surface, it became stiff and could be moved only with difficulty. In some cases (e.g. Mr. von Frantzius, and in a municipal functionary in Sohrau, at the beginning of the second week) a fuliginous coat appeared on the teeth, and on the tongue a more brownish, thicker coat. In all patients the skin retained the *calor mordax,* in some despite the rather abundant perspiration of a sticky and stinking sweat. The petechiae gradually increased and the whole surface of the body assumed a spotted, hyperemic, rash-like appearance. In some patients impairment of hearing increased to actual deafness. Weakness was extreme, the lower jaws were so slack that the patients found it very difficult to take up liquids with their lips and to swallow them. The circular muscles of the eyelids were not completely closed during sleep, the eyes being mostly rotated upwards and inwards. When making an effort to move the eyes there resulted a fixed and yet somewhat uncertain stare. Some patients exhibited twitching of varying violence, sometimes in the form of a twitching tendon, sometimes in the form of extensive cramp-like fits. In Sohrau, in the clinic of Dr. Wachsmann, I saw a woman in this stage who, according to the very expressive and lively description of her daughter, had had a generalized convulsive attack. She nevertheless recovered. Her face, in general, was very red and hot. Some persons in this condition jumped out of their beds and ran off for a certain distance; most of these were slightly delirious by day and by night. The delirium sometimes definitely bore a character of exaltation, even of frenzy, but usually it was of a depressive nature, alternating with a soporific state. Thus, a man was constantly repeating-most revealingly-that he saw calamity sitting in all four corners of the room. In some cases there occurred a more marked catarrhal diarrhea, without, however, showing intensification of the symptoms or reaching great severity. Thereafter meteorism was very rare.

According to the intensity of these manifestations their duration as well as that of the peak stage varied greatly. In the lighter cases the climax of the disease passed quickly. The exanthema appeared on the 3rd, 4th or 5th day after the overt outbreak of the disease, the symptoms persisted with some in tensity up to the end of the week or at most into the following week for a few days, the disease then passing into the third stage. Most of the patients whom I saw were of this form, and as I had the opportunity of observing them accurately in the hospital at Sohrau, I shall later report on some of these cases in detail. In the more severe cases,

however, the highest peak of the symptoms used to appear on about the 9th-10th day, in a few cases a little later, for instance on about the 14th day. But in these cases the acute peak stage always continued beyond the beginning of the first week and almost always included the second week completely. Most of the patients who exhibited this form were strong, well nourished people, so that one could not escape perceiving a definite relationship between the intensity of the disease and the bodily strength of the individual, a combination that finds a certain confirmation in the great predisposition of younger and stronger persons to typhus fever. In contrast, there was no noticeable relationship between the appearance of the roseola exanthema and the severity of the disease; it did persist somewhat longer than usual in some patients of the severe form, but in others it faded away as it did in mild cases. In cases of the former nature it did happen, however, that it was visible up to the 10th or even the 14th day. The extent of the petechiae also did not correspond to the peril of the particular case. I have seen mild cases with extensive petechiae and severe cases with very few petechiae; other physicians saw fatal cases without petechiae. I can, therefore, only vent my opinion that neither the roseola nor the petechiae had any definite prognostic significance.

In those patients who did not succumb to the disease by that time (I shall deal with the others in the next section) the severity of the disease was now broken and there began the third stage, which we have called the abating stage of the disease. It corresponds partly to the nervous stage of other authors, inasmuch as in this stage the symptoms of weakness, exhaustion, and depression are apparent most markedly and clearly. In the milder cases, as is evident from the above data, this stage already began in the second week of the disease, in the severer cases its onset could be reckoned to occur from the third week onward, i.e., in such a manner that the beginning of this stage can be taken to lie between the 7th and 14th day, though I am unwilling to give preference to anyone day.

Usually the so-called critical manifestations marked the transition. In all patients the skin lost the burning, stinging sensation of the *calor mordax,* a light sweat broke out in many, or the body surface grew at least soft and moist. In a few cases there occurred a vesicular miliary rash, particularly on the trunk. The urine of most was altered; in the majority of cases it was still acidic and produced heavy sediments of ammonium urate which, however, were not accompanied by the excessive excretion of coloring matter, such as in malaria and during convalescence from inflammations with great exudates, etc. *(sedimentum latericium, roseum),* but had a more loamy, dirty gray brown or whitish brown color. In some patients the urine was decidedly alkaline, with a rapid and abundant separation of large crystals of ammoniated magnesium phosphate (triple phosphate). These changes in the urine lasted for several consecutive days; then the urine became clear, with an orange or straw-yellow color. Then, starting from its borders, the tongue began to grow moist, the dry areas in the middle softening or detaching in brown crusts. The coughing increased again and more easily yielded a foamy sputum, of a whitish slimy, purulent aspect.

As the symptoms of skin congestion subsided, the face rapidly paled and the patients, often in the course of a single day, presented a collapsed, exhausted aspect, while up to that time they had still presented the picture of a well nourished person (this naturally does not apply to those who were already exhausted as a result of starvation). Moreover, the advanced muscular debility only now first entered the subjective feelings of the patients, for now it was not their earlier false weakness, a sluggishness of movement in which movement was hindered by suppression of stimulation, but they developed true weakness, an impairment in their power to move in spite of proper stimulation. At the same time the muscle pains became more prominent in many patients, with the difference, however,

that various muscle areas were designated as being painful in alternation.

The number of heart contractions increased in all patients. In the milder cases, though, they dropped rather rapidly, so that in a few days their frequency was reduced from 100-96 to 84-76; a large number of the more severe cases, however, continued with a fast pulse. I have seen patients where 100-120 contractions were counted. In all of them the pulse was exceedingly poor; the impulse of the blood wave was very weak, the tension of the arterial walls was insignificant and the pulse could be very easily compressed. I have not seen any central nervous (i.e., brain) trouble apart from a moderate weakness of memory and a tendency, in the more severe cases, toward troubled dreams.

During this stage the petechiae disappeared in most patients by gradually growing fainter at the margins, thus being effaced, sometimes assuming a light yellowish color, and lastly being resorbed without residue. In some individuals, however, they still increased during this stage, becoming denser, particularly in the peripheral areas, i.e. at the extremities of the upper and lower limbs, partly becoming confluent and forming larger more irregular spots, which, however, as far as I have seen, never exceeded the size of a lentil. In these cases, too, regression occurred entirely in the above-mentioned manner, but then continued until convalescence.

The duration of this stage was very irregular and a definite assessment of its limits is not possible at all. The transition to the fourth stage, that of convalescence proper, took place so gradually; without any marked or characteristic events, that any fixed timing would be completely arbitrary. In general it might be said that the milder cases passed into a definite state of convalescence at the end of the second week and the severer cases at the beginning of the 4th week. Convalescence, however, could be regarded as beginning at the moment when the pulse returned to its normal frequency. Prof. Kuh, who fell ill on the 19th of February was still in the third stage of the disease on the 9th of March, when I saw him again in Breslau[9], i.e. after 4 *[sic]* weeks, although towards its end. However, he had reached this stage under the worst of conditions. Dr. Neumann fell ill in Radlin on the evening of the 23rd of February and, after a restless night had himself moved to Loslau on the following morning, where I saw him. (He had a moderate fever, his head felt somewhat heavy; *calor mordax* with moist skin, light coating of the tongue.) He was then soon taken to Breslau, and when I visited him there on the 9th of March, i.e., at the beginning of the third week after catching the disease, I was most pleased to find him in full convalescence.

The symptoms during convalescence were mainly those that expressed the direct continuation of the conditions already in operation. At first there was a general weakness of the transversely striated muscle system. In many patients this was combined with severe pains in the muscles of the external parts. When these people were asked whether they had any complaint, one could be sure that they would mention pain in one part or another of the body. On further questioning, they would show a new place; upon examination, pressure, no matter where applied, would elicit the words *boli, boli,* it hurts! In the hospital at Sohrau there was a young girl who suffered from such severe pains in the soles of her feet that she could not sleep for 3 days and nights consecutively and was always weeping. The much lowered energy of the heart beat was sufficiently evident from the very feeble pulse, but there was added to it the frequent occurrence of a symptom whose dependency on such a cause I have shown earlier (compare: *Beiträge zur exper. Path. und Physiol., 1846,* No.2, p. 39), i.e. the spontaneous clotting of the blood in the veins. I had confirmed this by 2 autopsies. I have also seen, in the hospital at Sohrau, a third most interesting case of obstruction of the jugular vein during life, on which I shall report in detail.

The bronchial catarrh in some patients

developed into real pneumonia. In the third stage there occasionally occurred pains in the chest, especially upon coughing, while the respiratory sound in the lower and posterior parts of the lung became indeterminate; in the 4th stage a fine bubbly crackling was heard and the sputum showed an admixture of blood. However, I never met any case in which the degree or the course of these pneumonias could be considered as serious, had they been treated early. I shall later report on a case in which the pneumonia had become chronic and had finally caused death.

I have never seen decubitus in the sacral region. However, I have been shown several cases with gangrenous decay of the limbs. In some of them it was not evident from the case history whether this gangrene might not have been caused by freezing, an assumption which is not unlikely in persons who go barefoot on snow and ice. Have I not seen children with bare edematous feet walking on frozen roads and wading in snow water? In many cases, however, it could not be doubted that the typhus was the direct cause of the gangrene. Dr. Babel in Lonkau showed me a boy, 15-16 years old, whose right foot was mummified, black and dried up to the middle of the metatarsal bone; the toes of the left foot were in a similar state and a deep line of demarcation was beginning to form in all affected parts. This boy had developed typhus and gangrene before the onset of the severe cold. Mr. Zillmer, the regimental medical officer in Gleiwitz, informed me of another case, in which the leg had become gangrenous two thirds up the shank and had spontaneously separated (the bone having broken when the patient got out of bed).

Certain physicians claimed to have seen parotitis but such cases were rather sporadic. Self-evidently, atrophies and edema were very frequent in poorly nourished and neglected persons, in particular as the various previous bouts of malaria had a predisposing effect. I shall later report on one such case. In stronger individuals, in whom the disease had not reached a high intensity, the loss of weight was quite insignificant, and in this respect recuperation contrasted most markedly from our abdominal typhus. But in very severe forms of the disease there occurred extreme emaciation.

Finally, I must mention those irregularities of recovery that were due to gross dietetic errors. These disorders, whenever I heard about them, always were of an alarming character. I saw the first such case in Loslau in the clinic of Dr. Türk. A man who was in the 6th week of the disease and on his way to recovery suddenly was most violently ill after eating liver cooked in sour sauce. When I saw him his death seemed imminent: sunken in, pale face, very emaciated body, a pulse of 130, very feeble pulsations, respiration frequent, superficial and stertorous, diarrhea, vomiting, severe tenderness in the right hypochondrium, which, over most of its area gave a muted tone. The second case was Mr. Preiss, a surgeon in Rybnik, who in the 5th week of his disease also was on the way to complete recovery. Having on the 23rd of February too abundantly partaken of buttermilk, which at that season, always, is not very wholesome, he suddenly felt greatly agitated and experienced violent chills, followed by an intense sensation of heat with very high fever and a pulse of 160 heats/minute, as well as by abundant bilious vomiting. This condition persisted up to the morning of the 24th, when he again entered a new stage of severe chills, developing into a renewed stage of heat, fever and vomiting. On the evening of that day, when I saw the patient, no local affection could be detected; the vomit was strongly bilious. On the morning of the 25th a new attack of shivering fits. Mr. Kunz, the district physician, gave him quinine; the shivers disappeared, but the vomiting only became more persistent. Champagne seemed at first to inhibit it, but it soon resumed, turning into hiccups; the patient died on the 5th of March. An anatomical examination was not made, as I only arrived in Rybnik on the following day. The suspicion that a liver affection might have developed, as in the first mentioned case, can thus not be further discussed.

I never saw any extensive scaling.

I conclude this section by reporting some case histories from the hospital at Sohrau that, in general, illustrate the prevailing mild form of the disease. I have so selected them that every case presents some characteristic feature, but shall abstain from any further comment as the above data convey my opinions in sufficient detail.

Case I. Alois Waligura, a robust young man of about 20 years, was in hospital at the same time as his father (compare case VI) and his sister. The sister had first fallen ill, then the father and lastly the son. Without having had overt chills he had felt tired, slightly dull and a little chilly 4 days earlier and had taken to his bed. When I saw him in the hospital on the 29th of February, he gave completely reasonable indications, complained of a slight headache and buzzing in his ears, together with some difficulties of hearing; his face was somewhat red. He felt a pervading lassitude. His lower extremities, from the feet up to the lower ends of the thighs, hurt a great deal, the pain intensifying with movement or pressure, while no outward changes could be recognized in these parts. His skin was soft, somewhat odorous, there was severe *calor mordax;* roseola was moderately distributed on the lower part of the chest, on the upper part of the abdomen and on the back, the spots being slightly raised, somewhat darker in the middle and disappearing for a moment upon pressure by the finger. The pulse consisted of 96 rather large and full beats; the urine was somewhat saturated and acidic. Even now the patient continuously felt chilly, with frequent impulse to cough, and with some pain in the chest on coughing. On auscultation a whistling noise was heard when inhaling, in particular in the back, in the area of the larger bronchial branches. At the back and below, partially indistinct breathing. The tongue was red, with a light white coat only at the center, the abdomen soft, some pain in both hypochondria, but no changes to be detected on palpation or percussion in the liver or spleen. Stools were normal and solid. The night passed quite calmly; on the next day the buzzing in the ears had intensified, the exanthema had further spread on the chest. The feet were still exceedingly painful; he felt very weak. Pulse 92, large, rather strong. Tongue moist, slightly whitish; he still had some appetite. On the 2nd of March the exanthema had faded almost completely; the skin was soft, not very hot, less burning; pulse 96, moderately large, easy to compress. The face was pale, somewhat sunken in. Tongue moist, with a slightly whitish coating. Abdomen soft, not painful. Strong tingling. Increased hardness of hearing. Cough frequent, moist, easily releasing a formed white sputum; undefined breathing with strong wheezing was heard at the back on the right side; on the left side undefined breathing; in front, above, there was marked whistling on both sides; below, indistinct breathing with a buzzing sound. Severe pain in the soles of the feet and the shanks up to the knees. On the 3rd of March the exanthema had disappeared completely; the patient felt better and only complained of dizziness, pain in the feet and legs, and great weakness. The tongue was moist and clean. The pulse consisted of 80 somewhat weak beats. The urine was clear, straw yellow, neutral. The respiratory sounds were purer, the thoracic pain had totally disappeared, expectoration was moderate, slightly mucous and purulent. On the 4th of March, the head had somewhat cleared, and the hardness of hearing had somewhat abated. Respiration had become more free, expectoration was rather abundant; whistling was present on the lower right side, on inspiration. The fever was gradually subsiding. Same situation on the 5th, 6th and 7th of March; the hardness of hearing was almost entirely gone; the head remained completely free. The cough eased up. The appetite was good, and the pain in the feet had lessened. Only the weakness still persisted.

Case II. Halbhauss, clerk from Loslau, 20 years old, a vigorous well nourished man from a non-infected house. According to the report by Dr. Sobeczko, who had already seen him there,

he fell ill on the 26th of February (probably the disease had merely intensified at that time, but had been present for some time earlier). When I saw him at the military hospital on the 29th his head was heavy, his cheeks had a strong bluish red tint (venous hyperemia); he was hot, his eyes shiny and with a fixed stare. The hearing much impaired. While the patient was lying in bed, and without any external stimulus, he soon began to mumble incomprehensibly in delirium. His skin was soft, somewhat moist; severe *calor mordax;* slight exanthema on the chest. A pulse of 132 moderately large and strong pulsations per minute. His tongue was moist, covered with a light whitish coat; the abdomen soft, no pain anywhere; there had occurred several thin, involuntary defecations. Rare, rather dry cough. The following night the patient was restless, constantly talking to himself. On the morning of the 2nd of March, the patient showed great agitation, he groaned a lot, wanted to get out of bed. His appearance had markedly changed, he looked much more alert. The skin was warm but not burningly hot and was covered by quite profuse sweat; his tongue was moist, covered with a thick yellowish coat peeling off like plaster. In the morning the pulse consisted of 100 much stronger pulsations which were not completely suppressible. The exanthema still was present. Defecations were pasty and occurred voluntarily; the abdomen soft. According to Dr. Sobeczko, a strong exacerbation of the fever took place in the evening, the frequency of the pulse increasing, the skin becoming drier and hotter. At night some restful sleep. In the morning of the 3rd great lassitude, severe pains in the feet, much agitation, very pronounced deafness. The skin was soft and warm, the exanthema had disappeared. The tongue moist, with a light whitish coat. The pulse made 104 small, easily compressible pulsations. The urine was clear, saturated and acidic. Some soft defecations. Little cough. At night again some sleep. On the morning of the 4th great weakness, face pale and exhausted; much buzzing in the ears, difficulty in hearing; the feet painful. The skin moist, a little sweaty; the urine dark, acidic with an abundant flocculent dirty brownish sediment that consisted mostly of urates and mucus. In the morning a pulse of 100 easily compressible pulsations, its frequency increasing later on. In the following night, the patient again slept. On the morning of the 5th, the hardness of hearing had somewhat subsided, but the ears were still buzzing; great agitation. The skin soft, but not moist, the urine was flecked, dark brownish, alkaline, with an abundant sediment and a surface film, both consisting of triple phosphate crystals. The pulse consisted of 116 small and weak pulsations. A light erosion which was very painful had appeared on the sacrum. The tongue was moist and clean, the abdomen soft, not painful, no diarrhea. On the 6th of March, after a restful night the patient felt better. The soreness of his limbs and the cough had completely subsided, while the buzzing still persisted. The tongue was moist, the appetite was returning. The urine continued to contain triple phosphate, the pulse went up to 112. On the 7th of March the subjective condition was well, the pulse quieter, the urine turbid, with a copious, slimy, flocculent sediment.

Case III. Johann Klimezar, 34 years old, a Goralle from Hungary[10], a very strong, splendidly built man. He had first felt ill 10 days ago in Tarnowitz and had already spent 4 days in bed in Beuthen. At that time he had complained of great weakness; the physician had administered an emetic, whereupon he developed diarrhea. On the 29th of February he was admitted to the hospital at Sohrau. He complained of insomnia, headache; particularly in the frontal region and severe buzzing in the ears *(huczy);* his face was much reddened, his forehead hot, his eyes shiny. His skin was strongly turgescent all over, hot, although somewhat sweaty. Exanthema was quite widespread on chest and abdomen, but in view of the general redness of the skin looked almost pale. The pulse consisted of 104 large and strong pulsations per minute. The tongue was red and

moist. Thirst was moderate; the abdomen soft and not painful. Diarrhea had been present for 2 days. He had started out with a cough, but had lost it. His feet hurt; they were, however, neither swollen nor changed in any way. The night was somewhat agitated, but there was no delirium. On the first of March in the morning no further headache; his thinking was completely clear, he had buzzing in his ears as if a mill were working in his head. His feet too no longer hurt. His skin was moderately hot, the exanthema pale; the pulse now consisted of 104 moderately strong, somewhat smaller and softer pulsations. The tongue was dry, brownish and cracked at the center. Stools thin. After eating, the tongue very soon became moist and then showed a whitish coating. In the afternoon I first noted some petechiae on the abdomen. The night was somewhat restless, the patient occasionally mumbling to himself. On the morning of the 2nd, he was very weak, much buzzing in the head, pronounced hardness of hearing. He coughed much, and ejected a sticky, foamy and glossy sputum; when coughing he had a headache, but no pains in the chest. At the back of the thorax on both sides of the vertebral column bubbly râles were to be heard. The skin was moist and soft, the pulse consisted of 128 feeble and forceless pulsations. The tongue was moist, lightly whitish; urine and feces, the latter was liquid, were voided in the bed. The roseola disappeared while the petechiae increased, especially on the abdomen. No more pain in the feet. The night was a little quieter. On the 3rd, deafness was most pronounced; vertigo and great debility. The skin was soft, no longer burning; the congestion in the head had now disappeared. His face was pale; the petechiae rather numerous. The pulse showed 92 small and weak pulsations; the urine was clear, straw-yellow and strongly acidic. The tongue was moist, with a light whitish coat; no diarrhea. Coughing was frequent, the sputum came easily, forming large, balled, slimy, almost whitish lumps. On the 4th of March the patient was better. Defecation voluntary. Appetite was returning. The petechiae began to disappear. The pulse consisted of 100 smallish pulsations. On the 5th sediments of ammonium urate were noted in the urine; this continued for a few days, after which the urine became clear and straw-yellow. The condition of the patient remained good. The pulse frequency gradually decreased, as did the deafness; the patient's weakness, however, persisted.

Case IV. Trautvetter, 27 years old; smith; born in Tepliwoda near Münsterberg, a very strong and well nourished man, had fallen ill on the 25th of February in a house where no one else had the disease. Severe chills followed by fever, headache and lassitude. These symptoms very rapidly increased in intensity, so that he had to stop working and took to bed on the 27th of February. When I saw him in the hospital on the 2nd of March, he complained mainly of exhaustion and lassitude. His head was free, his hearing almost unimpaired. His skin was moist and warm; the area of the upper abdomen and the hypochondria presented sporadic pale bluish-red roseola spots, the urine was dark, turbid, like bad brown beer, acidic, the pulse consisted of 96 weak pulsations. His tongue was moist, with a slight whitish coating, the abdomen soft, defecation rare, but liquid. A moderate cough. Some sleep at night. On the morning of the 3rd, slight headache and great lassitude. The skin was moderately hot, soft; some isolated exanthema spots on the trunk. The urine was dark, acidic, with an abundant, flocculent, whitish sediment of urates. The pulse 76, with rather strong and full beats. The tongue was moist, plastered with solitary epithelial masses; the abdomen was soft; stools retained. Catarrh of the conjunctiva. Cough somewhat more frequent and moist. The night was interrupted by much coughing. On the 4th of March the patient complained of great muscular weakness and headache, but not of buzzing in the ears. The skin was moderately warm and soft, the exanthema had disappeared. The pulse made 96 rather small but strong beats. Frequent moist cough; sputa whitish, balled and slimy. The

tongue unchanged, covered with whitish fragments; 3 thin, very copious stools; body soft, not painful. On the 5th, the skin was warm and dry; the urine yellowish brown, a little turbid, acidic; pulse 88, small, easily compressible. Great weakness, much restlessness and groaning; moderate deafness. Catarrh of the conjunctiva more severe, the eyelids very adherent. Tongue moist, with a whitish fragmented coat, as if torn; no stools. Cough moist, sputum copious, whitish and rather purulent; on the 6th less frequent; tongue still somewhat streakily coated; urine dark, turbid, brownish, acidic. Pulse: 80 rather large and strong beats. Greater turgor of the skin. On the 7th condition much the same, the bronchial catarrh was decreasing and the catarrh of the conjunctiva had almost disappeared; there remained great weakness.

Case V. Marianna Kubiczkowa, 50 years old, a very weak and emaciated person, came from a house where a man had contracted typhus 14 days previously. She had first felt unwell 4 days earlier, had the shivers, felt dazed, had aching limbs and was overcome by great lassitude. On the 1st of March she was admitted to the hospital; she still suffered from aching feet, dizziness and buzzing in the ears. Her appearance was relatively good. Her skin was warm, a little moist; on the trunk, neck and the extremities numerous red petechiae the size of a pin head in the upper layers of the skin; roseola was not seen. The pulse: 80 moderately large but not strong beats. Her tongue had a whitish coat, was dry, smooth and brownish in the center but remained moist for a long time after eating, drinking etc. Lips and teeth were normal; there had been no defecation for 2 days; the abdomen was soft, tender only in the left hypochondrium. Percussion showed a dull tone over a large area above the false ribs to the left (she had last had a bout of malaria 5 years ago). Cough rare and moist. On the 2nd of March, roseola appeared on the dry but not hot skin of the chest and abdomen, the spots were pale red not raised and measured 2 lines in diameter. The buzzing in her ears had increased; moderate deafness. Strong pain in the feet. Catarrh of the conjunctiva. Pulse 72, moderately large, soft. On the 3rd of March, after having slept a little during the night, great lassitude. Head free, hearing almost cleared up, but there was a rushing noise in the ears. Skin dry, a little burning; the roseola had disappeared again; the petechiae were still as red as before. Urine sparse, brownish, acidic. Pulse 92, small and weak. Cough moderate, slightly moist. Tongue dry, brownish and cracked; 2 thin stools. Feet very painful. On the 4th of March deafness increased; cough more frequent and more moist. Tongue rough in the middle with a somewhat whitish coat. Pulse 116, easily compressible. Great weakness. On the 5th pulse 116, large and empty; enormous weakness. The cough let up a little. Skin dry, but not hot, the petechiae slightly more bluish-red in tint. The diarrhea did not return. Abundant, sticky, viscous mucus excreted in the throat, and at the root of the tongue. On the 6th of March, pulse 140, small pulsations; the tongue brown, dry, somewhat scabby in the center. Rare cough, moderate deafness, enormous prostration. Petechiae still persisting.

Case VI. Bernhard Waligura, 53 years old, baker, father of Alois W. (case I). A rather emaciated and ill-nourished man. Having suffered from weakness and pain in the legs for 7 weeks, he first had chills 10 days ago, then soon developed a severe headache, buzzing, a loss of appetite, great weakness and a tormenting cough. After admittance to the military hospital, his condition speedily became so alarming that he received the last sacraments on the 26th of February. But on the 29th I found him in the following condition: pronounced hardness of hearing, pain in the forehead, weakness; but completely lucid. The skin moderately hot; on the abdomen, the chest and the arms rather small, pale bluish-red roseola spots with somewhat darker centers, very rapidly returning after pressure. The pulse: 88 moderately large, easily compressible beats. Respiration a little speeded up. A rare dry

cough. Tongue moist, with a slightly whitish coat, abdomen soft, the area of the spleen not altered; no defecation. On the 1st of March the condition was essentially unchanged; exanthema was still present, some spots were even slightly raised. The fever was moderate, the cough somewhat more frequent with a sometimes somewhat slimy sputum. Catarrh of the conjunctiva. On the 2nd of March the patient was feeling better, but there still prevailed great deafness. Exanthema was still present on the abdomen, but the spots had become very small; practically the only part visible was the pale central point. On the 3rd some dispersed petechiae appeared at different points; the exanthema had almost disappeared. The pulse was quite steady. Skin soft; buzzing in the ears; insomnia; a somewhat heavy head; the urine was clear, acidic, orange yellow. In the following days the patient's condition steadily improved; his appetite returned; the petechiae did not develop in large numbers and soon disappeared, so that on the 6th of March the patient was regarded as recuperating.

Case VII. Eduard Gettler, 18 years old, shoemaker; came from a severely infected house. In that same house there had been 13 deaths as follows: 4 members of the Gettler family, 6 of the Kotrimba family, 2 of the Wyusna and one of the Wilczek family. From the Gettler family the father, the mother, a sister and a brother had died. The remaining 4 brothers and sisters also all contracted the disease; two of them still were in another hospital, and the other two (the patient in question and his sister) were in the military hospital. The father and mother had died before the new year; the last of the inhabitants of the house, a man by the name of Wyusna, had died 8 weeks ago. Soon after his death the farmer Wilczek moved into Wyusna's lodgings, fell ill and died 14 days later. One of the Gettler sisters, Marianne, nursed him and then also fell ill.

Eduard G. claimed to be in the 3rd week of the disease. In the winter of 1846-47 he had had quartan malaria for 4 weeks and had cured himself with milk. At the beginning of the present infection, when he already had swollen feet, he had been lying on the cold floor for 4 days.

At the time of examination, on the 29th of February, he had a pale, puffy appearance. The exanthema was no longer clearly visible; also he was not hard of hearing. The pulse consisted of 100 moderately strong, though small pulsations. His skin was dry, but not hot. His tongue moist, slightly whitish. He complained of a heavy head, and of restlessness. The right half of his face, especially the eyelids, showed a marked edematous swelling. He complained of severe pain on the right side of the neck which he found impossible to move. The area from the sternal attachment of the sternocleidomastoid muscle to the angle of the lower jaw was very painful, spontaneously as well as on palpation; it was swollen; in the direction of the internal jugular vein under the sternocleidomastoid up to the jugular foramen a hard cord could be felt, the thickness of the little finger, which could clearly be moved to and fro (occlusion of the vein). This pain had started two days ago, the swelling of the face one day ago. The abdomen was tender everywhere, particularly in the region of the left hypochondrium. Above the false ribs, in an area of ¾’ in length and ½’ in the breadth, percussion revealed a dull tone. On the 1st of March the pain in the neck was still present, the one-sided edema of the face had increased. The hard cord was still clearly palpable. In addition, the patient complained of pain in various other parts of the body. All muscles (extremities, back, chest) turned out to be sensitive. Otherwise his condition was passable. On the 2nd of March, the edema and the pain had somewhat receded but the cord was still palpable (collateral circulation having developed). The soreness of the muscles also had diminished. Some nose-bleeding. On the 3rd, the edema had yet regressed, the urine was clear, straw-yellow, neutral. From then on improvement was continuous.

Case VIII. Marianne Gettler, 20 years old;

sister of the previous patient, in the 5th week of the disease. A solidly built, well-formed girl, not too thin. On the 29th of February she complained of pain on the right side of the chest and of a tormenting cough; these symptoms were much aggravated on the 1st of March. She in particular named the lower right part of the chest as the site of affection. Percussion there yielded a moderately subdued tone up to 1" under the armpit, and during inhalation a delicately bubbling crepitation could be heard in the whole lower lobe. The cough was very frequent, distressing and persisting; the sputum was foamy, viscous and whitish; there were 44 inspirations per minute. The pulse consisted of 120 small relatively strong pulsations. Her face was somewhat bloated, with a livid redness on the cheeks (venous congestion). Her tongue was clean and moist; appetite good. Severe pain in the feet. On the 2nd of March the same complaint with regard to the chest; percussion unchanged, but the respiratory sounds with a large bubbling; in the back they were rather undefined with a purring sound during expiration. Severe pain in the soles of the feet and from there up to the knees. Skin dry, not hot. Pulse 80, hard to feel. On the 3rd, severe muscle pain, spontaneously as well as upon movement, in the extremities as well as in the trunk. Cough frequent, with a viscous and whitish sputum; respiratory sound on the right lower side still rattling. The face quite pale and sunken in. Pulse of 80, very faint. Urine clear, straw-yellow, slightly alkaline. On the 4th of March an especially tearing pain in the hands while the pain in the trunk had lessened. Cough frequent, still tormenting. Pulse 80, very weak. The patient continued in this manner. The thoracic symptoms abated very slowly. The remaining, manifestations still persist.

Case IX. Johann Victor, 11 years old; around Christmas of last year he had contracted malaria in Baranowitz, where he worked as a cowherd. The malaria had at first assumed the tertian, then the quartan form, and had disappeared after 4 weeks without medical treatment. This very pale, thin and weak boy said he had been ill for 3 weeks. His head was unaffected, there was a slight buzzing in his ears, his eyes were clear and fresh, his lips pale, otherwise normal, his tongue dry, but not scabby. His skin was warm and dry, slightly flecked and desquamating on the chest. The pulse consisted of 100 small but strong beats. Respiration was short and shallow and, because of the great debility of the boy, laborious; 56 inspirations were counted per minute. He coughed frequently, with little expectoration; the percussion tone was normal everywhere; auscultation revealed a slightly rattling sound on the lower left side, and, on the right side, over the entire region of the lower lobe, a partly slight, partly pronounced bubbly râles. The patient moreover complained of pain in the abdomen, which was large and full, somewhat swollen up by gases, and almost everywhere gave a tympanitic tone, which was dull only in the cecal area. In addition, the spleen was easily palpable and percussion yielded a dull and empty tone over an extent of ½'. The condition of the patient did not change in the following days, for which reason I confine my remarks to the foregoing.

In the above case histories, I have assembled partly fresh, partly old, and convalescent cases such as constituted the majority of the Upper Silesian patients. From these case histories it will be easier to get a picture on the course of the disease than from long descriptions. The course of the disease is so simple that there is no need to discuss it any further. I would have liked to clear up certain details more exactly. But the impossibility of making an examination without an interpreter, with whom it was sometimes also difficult to communicate, coupled with the imperfections of a military hospital which had just been newly established and equipped, and lastly the very short time at my disposal must all serve to excuse me.

B. Death and the State of the Corpse

Death occurred either at the peak of the

disease, in the second stage, or at a much later time as a result of consequent diseases or of the above mentioned interruption of convalescence due to gross dietitic errors. I shall for the time being limit myself to a detailed account of the former cases, since that of the latter is, in part already given and, in part, self-evident.

When death occurred in the acute stage of the disease it nearly always took place between the 9th and 14th day, with a steady increase of the symptoms, that I have described in the more severe cases. The frequency of the pulse increased until it could no longer be counted, the individual pulsations were mostly large, but very easy to compress and frequently not clearly separated from each other. At last only indistinct undulations could be felt. The head which, as a rule, conspicuously hung backwards, remained constantly hot, the cheeks were intensely red, eyelids and lips always remained open, the eyeballs always being rotated upwards and to the inside. When addressing the patients in a loud voice to overcome their deafness, they always attempted to answer, but tongue and lips failed them. Left to themselves they soon became delirious, in most cases softly mumbling incomprehensible words. The skin retained its burning character; although sticky sweats were not rare; in most of them the petechiae multiplied, or the skin as a whole was covered with a sprinkle of red dots, or seemed to be covered with a rash, as if it were suffused with blood, and yet this coloration was due only to hyperemia of the venous system. Respiration was very frequent, shallow and gasping. There occurred partial muscular contractions, spasmodic jumping of the tendons and, to a lesser extent also floccillation.

In such a manner did the symptoms appear until their increasing severity caused the death of the patient. Comparing the manifestations of the last hours of life with the result of the autopsy, as I shall presently do with respect to a pertinent case, it could be said with certainty that a venous hyperemia of the brain had been present at the end, which had developed from the congestion of the blood in the vessels of the neck as a result of incomplete inspiration and insufficient heart beat. But the true cause of death must be sought in the very same cause that had produced fever and the above mentioned derangement in the respiratory and circulatory organs. As already stated, most of the deaths of this sort occurred between the 9th and 14th day of the disease. I myself have never seen a case in which death occurred earlier, and most physicians in the districts agreed with me. Laymen quite generally named considerably earlier dates, such as the 3rd day, and usually linked these cases in particular with infection. As I shall have to return to this point, I shall here only say that in every case in which I was able to check more closely, no matter how convincing the original information had sounded, it was found that the persons concerned had already been ill before that time, but had probably not taken to bed before, or had only then called a physician.

Before my arrival in the two districts, only 4 cadavers had been examined anatomically: one by Prof. Kuh, in Sohrau, together with his colleagues there; one in Rybnik by Drs. Altmann and Biefel; one in Loslau by Drs. Raschkow and Turk; one in Pilchowitz by Mr. Willim. All agreed that there were no significant changes, in particular no ulcerations, in the intestine; at most they mentioned anemia, the spleen was usually reported to be enlarged and soft; in some patients there was reduction in the consistency of the brain, in one other edema of the glottis. I myself was able to perform 4 autopsies, and I am reporting on these here in detail.

Case X. Johann Soyka, a vigorous young man; died in the evening of the 7th of March in the military hospital at Rybnik, towards the 14th day of the disease, with symptoms as described above. The fever had been unusually severe, the skin burningly hot, intensively reddened throughout, and as spotty in appearance as if it had been sprinkled all over with petechiae. The autopsy took place 12 hours later on the 8th around 10 in the morning, in the presence of Dr. Heinke and Dr. Samostz from Breslau, and also

of Dr. von Frantzius.

Well built body, moderately nourished, considerable rigor mortis. The skin a light yellowish tint everywhere; intense bluish-red blotches on the lower abdomen, the upper arms and hands, the thighs and legs, the individual spots irregular, large, looking like ecchymoses. But on dissection the skin was quite free and only the vessels, in particular those of the superficial layer, were congested with blood. Both feet were bluish-red up to the ankles, with only a few whitish islands of skin remaining. On incision only venous hyperemia was met up to the superficial fascia, without extravasation. Wherever incised, the muscles were dark red, resembling smoked meat.

The scalp was rich in blood, the bones normal, the brain sinuses contained much blood with a buffy coat. Strong venous hyperemia of the membranes, extensive edema of the pia mater. The brain substance was of good consistency, the white matter very rich in blood. A moderate amount of fluid in the ventricles.

Thyroid gland normal. Larynx and trachea normal, the latter with viscous whitish mucus in its lower part. In the anterior mediastinum the veins were strongly congested with blood; lungs not retracted. Except for some reddish fluid, the pericardium was clear. Heart of normal size, the left ventricle strongly contracted, filled with rather large clots having a buffy coat that extended far into the aorta; the fibrous material of the buffy coat moderately cohesive, turbid, yellowish white; the endocardium normal. In the right ventricle, the buffy coat was even more extensive; at the same time it was strongly granulated by the accumulation of white blood corpuscles; endocardium normal here also. The heart muscles were somewhat pale, otherwise normal. Some old adhesions of the connective tissues of the left lung; strong venous hyperemia of the costal pleura; radiating connective tissue scar on the lung surface. The lungs contained air throughout; venous hyperemia pronounced in the lower lobes and moderate in the upper lobes; the bronchial mucous membrane was highly hyperemic, dark red, and covered by a viscous very abundant whitish mucus that almost blocked the bronchia. The right lung had even more numerous connective tissue adhesions; it was completely filled with air, highly hyperemic, dark red on the face of the cut; the bronchial mucous membrane was less reddened and its mucous coating less thick. The diaphragm was covered by a thick venous plexus.

The veins of the omentum were surcharged with reddish-black blood. Both lobes of the liver extended far beyond the edge of the false ribs; the spleen was also visible beneath them. The liver, in particular the left lobe, somewhat enlarged; structure and consistency normal, the veins filled with greasy dark blood. The gall bladder quite abundantly filled with dark yellow, slimy, tacky bile. The spleen much enlarged, adhering to the diaphragm with its upper end by masses of old connective tissue about 1 foot in length, ½’ across in width, 2" in thickness, very firm, but somewhat limp to the touch. The cut surface dark red, very firm, homogeneous, looking like goose-meat; the parenchyma, granular at a broken off surface, did not show the white bodies very clearly, but in one place showed a firm hyperemic wedge.

The stomach much contracted, containing a little fluid stained with bile; mucous membrane slightly thickened, here and there containing a somewhat altered extravasate in its upper layers. Duodenum quite normal; rather abundant secretion of mucus. The small intestine moderately extended by gas and fluids, hard fecal matter in the rectum. On opening the small intestine much mucus stained with bile was found in its upper part while in its lower parts there was a greenish yellow fluid mixed with numerous flakes of mucus; in the cecum a rather dry and hard fecal coating on the intestinal surface which increased in the large intestine and which was finally succeeded by brownish feculent masses. The mucous membrane of the jejunum was pale and generously covered with mucus, with slate-gray points on some parts of

the villi. From the beginning of the ileum onward the solitary glands were swelled up to the size of a small pin head; the condition was most marked above the valve, where the follicles of the patches of Peyer were, also slightly enlarged. The whole mucous membrane in the cecum and the upper part of the colon was of a dark bluish-red hue, the venous plexuses were abundantly filled with blood, the glands normal. Further in the colon the solitary glands were again somewhat enlarged, appearing as very small whitish points; between them, venous hyperemia in spotty distribution. In the rectum, the glands slightly enlarged, with a slate-gray point at their apex; in between, pale spots of venous hyperemia. The mesenteric glands not enlarged, containing a little more blood than usual.

Kidneys normal; slight catarrh of the renal pelvis; urinary bladder strongly contracted, its mucous membrane slightly hyperemic; the urine collected in it flocculent, turbidly whitish. In the inferior vena cava and the crural veins there was abundant blood, without a buffy coat, dark, clotted in lumps, almost tar-like.

Case XI. On the 26th of February, at 4.30 p.m., I sectioned a man in a suburb of Sohrau. Dr. Sobeczko had seen him only once; when he already lay unconscious. But his condition clearly indicated typhus, and a daughter of his lay stricken in the acute stage of the disease. The man had died in the morning of the same day. The autopsy was done rather hastily and under most unsuitable conditions on the lid of his coffin. Dr. Wachsmann from Sohrau and Dr. Eichholtz, head physician in Potsdam, were present.

Moderate rigor mortis. Body emaciated; man in the middle years of life. Body still slightly warm. Fatty tissues had disappeared, muscles dark, saturatedly colored. Intestines strongly distended with gas. Abdominal cavity free. Mesenteric glands normal. The cecum with some yellowish feces and *Trichocephalus dispar;* its mucous membrane of a slightly slate-gray tint; of the small intestine about 2' were examined and found to be completely normal, somewhat anemic, the glands hardly visible. Spleen greatly enlarged, I' in length, 1/2' in width at its center, tapering downwards; bluish-red, limp and shriveled, showing a few hard nodules of a darker blue; on the cut surface the white bodies were normal; the pulp of medium consistency, grayish red, the harder nodules dark red and dry. Liver enlarged, pale brownish, somewhat dirty, flaccid, shriveled, but not friable; bile plentiful, light yellow. Kidneys normal, in the calices a turbid, thick, slimy mass. Urinary bladder empty, normal.

The cartilages of the ribs were ossified. Numerous adhesions of the pleura, especially on the lower lobes. Pericardium normal. The size of the heart normal, the left ventricle was firmly contracted, the right one flaccid; valves, endocardium and muscles normal. Almost no blood, in the left heart, some clots with a slight buffy coating, greatly friable. On the right side much blood, mostly lumpy, dark red, but also clots with a buffy coat, extending into the arteries of the lungs. Lungs without tubercles; both lungs edematous, the posterior and lower parts hyperemic. The bronchial mucous membrane intensively reddened, bloody mucus. Bronchial glands black, not enlarged.

Larynx and trachea ossified. Mucous membrane intensively red with very bloody mucus.

Heavy stench, which clung to the fingers for a long time and could not be eliminated even after washing with vinegar. An ointment on a lead white base (Empl. de cerussa) which I had on my finger was not stained black.

Case XII. On the 25th of February, at 9.30 a.m. I sectioned a man in the miner's hospital in Rybnik, who had been in the care of Dr. Goldmann. (The latter had just traveled to Breslau with Prof. Kuh and had himself fallen ill during the journey). At the time of his admission to the hospital the man was already unconscious. The only thing known was that he had been ill for several weeks previously, with violent fever, much coughing and diarrhea. Death had

occurred on the previous evening. The following were present at the autopsy: Dr. Altmann, personal physician to Prince Biron of Courland, from Polish Wartenberg; Wehowsky, army physician for disabled veterans in Rybnik; Dr. Eichholtz, chief physician in Potsdam, and Dr. Biefel, chief physician in Breslau.

Sturdily built man in the middle years of life, moderately thin, military vesicles on the trunk, on the extremities numerous dark red post-mortem spots. Severe rigor mortis. Muscles all hard, dark red, resembling smoked meat. Very little fatty tissue.

Top of cranium very small, round, quite thick, moderately rich in blood, rather adherent to the dura mater. Dura mater moderately rich in blood, otherwise unchanged. Pia mater thickened in some places at the surface, especially along the longitudinal sinus; quite pronounced edema especially at the basis. The sinuses very full with dark, liquid unclotted blood. The veins of the pia mater and of the brain substance rather congested. The brain substance normal, of good consistency throughout; the gray substance (cortical layer, corp. str. and thalamus, pons) very red. The surface of the brain not separable from pia mater. The ventricles filled with a little yellowish serum. Hypophysis normal.

Tongue with greatly elongated papillae, especially at the center; these were of a dirty yellowish-gray color, anemic and dry. The root of the tongue with congested veins; marked secretion of mucus. The upper part of the oesophagus was filled with much mucus, otherwise normal. Thyroid gland very large, pale, much colloidal substance in the dilated foIlicles. Larynx and trachea normal, containing a narrow dirty gray band of mucus.

On its parietal side the pericardium was beset with connective tissue (tendonous) nodules the size of a hempseed. In its cavity a little yellowish fluid. The heart was of normal size; the right heart, in which the cone of the pulmonary artery was a little dilated, had collapsed; the left heart was tightly contracted, its veins swelled with dark blood. The valves normal, the muscles somewhat dark and hyperemic, the endocardium normal, the subendocardial vessels of the left ventricle abundantly filled with blood. The blood in both halves of the heart was thinly fluid, unclotted, dark red, in the right part with some discreet gelatinous clumps of fibrin, to which were attached numerous whitish easily crushable nodules; in the left heart small white clumps were swimming in the red fluid. Microscopic examination showed the red corpuscles unchanged; but in every drop of blood examined there were small islands of white corpuscles. The white masses consisted almost entirely of white corpuscles, mostly of quite considerable size, granulated, usually showing a round, cloverleaf or horseshoe shaped nucleus on addition of acetic acid.

The pleura entirely normal. The lungs a little contracted, without adhesions or scars, with numerous black pigmented spots that were denser on the posterior parts, containing little air, bluish-red, the cut surface dark red (hyperemia). The bronchial mucous membrane hyperemic, especially at the bifurcation of the bronchi, mostly on the right side; somewhat swollen, covered with a thick, viscous, bloody mucus. On the left side, this condition disappeared lower down; on the right side, however, the hyperemia intensified below, becoming blackish red; the contents of the bronchi were rather cream-like, white, non transparent, purulent. When cutting through the tissues, white masses of pus were seen to be surrounded by a dark red halo consisting of the hyperemic bronchial mucous membrane and the surrounding parenchyma of the lung, which was equally hyperemic (beginning bronchopneu-monia). The bronchial glands rather large, intensively black in color, without any symptoms of recent change. Both pulmonary arteries were almost completely obstructed right after the bifurcation: at first there came a very large, relatively dry, blakish-red, sometimes grayish-red coagulated mass filling the whole

lumen and loosely adhering to the arterial wall; then came dry, yellowish-red sometimes buff colored masses, riding on the sites of bifurcation and tightly adhering to the wall, which were very friable, and usually were continued by a dry but red plug that extended rather far into the efferent branch. The walls of the pulmonary arteries normal.

Abdominal cavity. Position of the viscera normal; only the sigmoid bend very far to the right; the omentum was pushed almost completely to the left. The liver smaller than normal, of normal consistency, with a smooth, brownish-red surface occasionally showing slightly yellow blotches. The cut surface had a uniformly light brownish-red color, dark red blood oozing from the vessels. The gall bladder relatively large, the bile abundant, thinly fluid and light yellow. Spleen of normal size, consistency greater than normal, the white bodies not changed, the parenchyma (pulp) harder than normal, dry, pale buff colored. Kidneys somewhat rich in blood, otherwise normal. In the renal pelvis a strongly mucous whitish fluid. Urinary bladder normal, urine copious, clear, dark yellow. Pancreas normal.

The mesenteric glands somewhat enlarged, in the cecal area also intensely hyperemic, but not changed in consistency nor in the moisture content of the parenchyma. The stomach of normal size; bilious, thinly liquid content; fundus and cardiac portion normal; the pyloric part with a heavy coating of whitish mucus; the mucous membranes thickened, slightly tumid, a little hyperemic, in many places slate-gray; in various places there were slate-gray lentil-sized spots. Duodenum well filled with bile, the openings of the glands of Brunner near the pylorus beset with slate-gray dots. The whole of the small intestine contained much very thin fluid having a fecal smell, yellowish in color, with numerous yellowish flakes of mucus and an acidic reaction; no significant turbidity on boiling; under the microscope nothing to be seen but intestinal epithelia and flocculent granules of precipitated bile. The small intestine anemic, the villi somewhat thicker than normal in the upper part only, otherwise the mucous membrane very thin; the solitary glands were slightly enlarged almost everywhere; the patches of Peyer were heavily flecked in a slate gray hue and presented a wavy aspect. In the large intestine harder yellow fecal masses adhering firmly to the mucous membrane. The mucous membrane of the cecum intensely hyperemic; throughout the lower parts of the tract up to the anus, only the top of the folds were reddened; the solitary glands were enlarged everywhere, slightly raised, marked by a slate-gray spot and ring.

The inferior vena cava, the iliac and the crural veins were filled with thickly fluid, cherry-red, almost tar-like blood, without any clots. The left crural vein from Poupart's ligament to the veins of the shank obstructed by large, dry clots often showing pearl-like blotches that filled the whole lumen. They were driest in the somewhat varicose veins of the lower legs. Dry, yellow-red clots of small size adhering firmly to the wall of the vessels emerged from some of the muscular branches in the center of the thigh. The inner layers of the vessels were nowhere reddened. The internal surface of the aorta had scattered small fatty areas.

Case XIII. Josepha Malcha, 50years old, died on the 5th of March at the Sohrau military hospital after having contracted chronic pneumonia during her convalescence from typhus. In the course of the pneumonia she had suddenly coughed up a most evil smelling mass, an event which was not repeated. In the end she suffered from an exhausting diarrhea. The autopsy took place on the 6th of March towards 3 o'clock in the afternoon in the presence of Mr. von Frantzius.

Very emaciated body. Rigor mortis. Intense icteric staining of the skin. Considerable edema in the left lower extremity. Fat had vanished from all parts of the body.

Top of skull remarkably round and deep, its inner surface quite normal. In the longitudinal sinus a little buffy coated blood. Dura and pia mater anemic on the convex side, pia mater very

edematous. The brain small, but heavy, considerable consistency, almost sticky; cut surface very moist throughout, the gray matter very pale, but of normal cohesion. In the ventricles a small quantity of fluid; the right posterior horn obliterated. Both transverse and cavernous sinuses and the veins arising from them and running to the pia mater were completely obstructed, containing large, usually discolored, clots adhering to the walls.

Thyroid gland somewhat enlarged, pale, yellowish. The respiratory mucous membrane in the primary respiratory passages anemic. The heart with heavy cushions of fat and large epicardial patches. In the right ventricle strongly buffy coated clots, but the buffy coat somewhat wrinkled and friable. In the left heart similarly buffy-coated clots with many clumps of white blood corpuscles. Endocardium normal. The upper lobe of the left lung strongly adhering but separated below from the costal wall by a turbid exudate mixed with fibrous flakes. The parenchyma containing little air, nearly everywhere edematous; the upper lobe firmer, the cut surface airless, compact, gray, lead colored, smooth, releasing a whitish fluid upon pressure. No old or young miliary tubercles anywhere. A large cavern occupied the upper half of the upper lobe of the right lung; it was filled with a rather thin, stinking, grayish white fluid; the inner wall smooth throughout, partly formed by the thickened pleura and partly by consolidated lung parenchyma traversed by numerous new vessels. The remaining part of the upper lobes partly edematous, partly compact. This latter mass was smooth on the cut surface, its color was whitish in some parts, in others slate gray, at a few points it was white and nontransparent, like tuberculous infiltration. On pressing a whitish fluid was evacuated. The bronchia of both sides were filled with tough slightly purulent mucus.

At first glance these changes might have been regarded as tuberculous: tuberculous infiltration of the parenchyma, a tuberculous cavity. But apart from the absence of isolated tubercles (tubercular granulations) the fact that the compact exudate did not show the friable, dry, granulated character of infiltrated tubercle and that a turbid whitish fluid could be pressed out everywhere speak against such an assumption. Microscopic examination immediately dispelled any doubt. The masses always consisted of cells, whether one observed the evacuated fluid or the cut surface, cells which at many points were entirely similar to those occurring in pus, while at others they had largely undergone fatty metamorphosis. Therefore, the condition can only be regarded as a gray hepatisation resulting from chronic pneumonia: an old exudate in the lung vesicles had very slowly transformed into a perishable tissue consisting of cells. The sudden discharge of a large amount of stinking sputum evidently denotes the development of the cavity *en bloc,* by massive necrosis: the mortification of the exudate and the tissues, together with the decomposition of the mortified substance, i.e., a circumscribed gangrene of the lung. That cavities with absolutely smooth walls may result from such processes is well known.

Liver of normal size, very finely lobate, infiltrated with some cholepyrrhin, with slight fatty degeneration. The bile very slimy, dark brown, somewhat flocculent, with various small, brown pigment stones. The spleen enlarged, very limp, covered with patches of fresh, fibrous exudate, bluish-red, very firm to the touch, grayish-red on the cut surface, dry, offering considerable resistance to pressure, no fluids oozing out; white corpuscles were not seen. Kidneys anemic, otherwise not altered. Pancreas normal. Uterus also normal.

The stomach of normal size, with pronouncedly bilious contents, its mucous membrane somewhat thickened and papillary, the muscle layer somewhat thick. The intestine somewhat collapsed. In the small intestine great accumulations of mucus mixed with bile, the solitary glands in its lower parts slightly swollen, no change whatsoever in Peyer's patches, nor any scars seen, the mucous

membrane slightly hyperemic in some places. Dysenteric changes in the large intestine from the valve to the anus; the mucous membrane hyperemic in a pattern corresponding to the longitudinal and transverse folds in the lower parts of the intestine, eroded at the top of the folds, the submucous tissues edematous; no fresh exudates present. The mesenteric glands only slightly enlarged, slightly hyperemic.

The left crural vein formed a hard cord; it contained a large, blackish, dry, clotted mass, adhering to the walls; which extended to the inferior vena cava; in its upper part it was quite discolored, softened in the center and filled with a whitish, pus-like, pasty mass.

If we compare the above autopsy reports with those of autopsies done before my arrival as well as with the observations by Drs. Lemonius and Polkow already mentioned, one may consider it as an established fact that, in this epidemic, the characteristic anatomical modifications of abdominal typhus *(fièvre typhoïde)* were not present. As is well known these changes are manifest in the follicular apparatus of the intestinal mucous membrane, in the mesenteric glands and the spleen, and mainly consist in the following: in the first stage (I here mean the stages of the local process, not those of the disease) there appears, concomitantly with hyperemia, a slightly aqueous exudate which lends to the organs a swollen appearance; in the second stage the hyperemia subsides, while the aqueous exudate increases, and, by mixing with the elements present, and by admixture of the more solid constituents of the exudate, there arises the peculiar "medullary" appearance; and finally, in the third stage, the firm constituents of the exudate increase, whereby are formed, in some parts, dry tubercle-like, yellowish-white necrosed foci. But what did we have in this epidemic? Let us examine the various organs:

1. The mesenteric glands in some cases were somewhat enlarged and hyperemic. However, in patients who had died at the acme of the disease, they were not flaccid, not greatly enlarged, without the medullary infiltration which should have been characteristic, and, jn case XIII, where the typhus as such had terminated long ago, they were not in a degenerated condition, i.e. flaccid and slate gray, as they are found after typhous swelling in the stage of convalescence, but here also they were red.

2. The spleen was enlarged in 3 cases. However, in one case (XII) it was not only not enlarged, but showed no traces of previous tumescence. The other 3 cases evidently represented residual malarial tumor, as is sufficiently clear from simple description. Never was there the plump, firm, dark red tumor of (abdominal) typhus which on the cut surface shows enlarged white bodies (Malpighian vesicles) in a dark cherry red, greatly friable parenchyma.

3. Peyer's gland groups were entirely unchanged. In one case (X) the related follicles above the valve were somewhat enlarged, in another (XII) they showed slate gray spots and the entire plexus had a wave-like surface as a result of the raising of the interfollicular connective tissues above the level of the follicles. The solitary glands were somewhat swollen in 3 cases, and that not only in the small intestine, but also (case X and XII) in the large intestine and the rectum. All these changes indicate no more than the presence of a catarrhal affection. In one case (XII) the latter was certainly chronic, as indicated by the slate-gray coloration of the stomach, of the intestinal villi, and of the follicles themselves; in the other two cases the great amount of mucous excretion present in the intestinal cavity demonstrates the catarrhal state with sufficient clarity. As is now well established, intestinal catarrh is almost always accompanied by changes in the glandular system, and the above mentioned changes of the mesenteric glands can also be related to this condition. However, a simple swelling does not suffice for construing a typhus, as, for instance, William Davidson tried to do. Swelling of the solitary glands, such as described, may be seen in the course of the most various acute disease, i.e. in pneumonias, scarlet fever, and

rheumatism; no one will or can deduce from it anything warranting a diagnosis of typhus.

I would now like to examine the constitution of the blood, in the light of the anatomical findings. I have had no opportunity of obtaining blood during life and perhaps such an opportunity will be found by a later observer of the epidemic. In the cadavers, however, even in the acutest of cases, we never found broken down, decomposed putrified blood, but on the contrary only well clotted blood with a buffy coat, which serves to show by a further example that it is not the lack of fibrin which causes the typhuses (compare this *Archiv,* I, p. 572). In various cases, apart from the presence of fibrin, the multiplication of the white blood corpuscles had become so pronounced that the lower surface of the buffy coat had assumed a granular appearance (compare *Med. Vereinsztg.* 1847, No.4). I am drawing attention to this condition in particular because Allen Thomson observed the same in the closely related remittent fever that prevailed in Edinburgh in 1843. (John Rose Cormack, *Natural history of the epidemic fever at present prevailing in Edinburgh,* p. 113.)

In addition, we found acute and chronic affections of the bronchial mucous membrane, of the mucous membrane of the renal pelvis etc., venous hyperemia in different organs, especially in the most acute case (X); spontaneous clotting of the blood in various veins, in the more chronic cases (XII, XIII). I do not discuss this point further as it adds nothing essentially new to our presentation[11].

Finally, the mortality rates remain to be discussed. In view of the particular conditions prevailing in Upper Silesia, I can furnish only the scantiest information. When the epidemic was at its peak, not even all deaths were registered with the clergy in charge of the graveyard registers, and frequently several bodies were put into the same grave. Traditionally the family bonds of the Upper Silesian are rather loose and his feelings of kinship, except for the closest of relatives, are weak; he thus cares very little for his dead. There probably are few places in the world where the burial places are as unadorned, the various graves as unidentifiable as here. Only as long as the ground is freshly thrown up or dug can a grave be recognized. After a few years all is covered by the same smooth lawn. Consequently, when even the number of deceased cannot be obtained from official registers, the manner of death, the nature of the last disease, will be even more uncertain. At the very best one can obtain a rough idea by comparing present mortality figures with earlier averages, the picture obtained being, however, distorted, as the foregoing dysentery and simultaneous starvation contributed in raising mortality. Except for the most recent times, incidence figures for disease cannot be obtained at all, not even approximately, as nobody was ever free to take care of such tables until district physicians were made available. Moreover, the figures which could then be obtained do not allow any conclusions on earlier times when starvation in its most horrible form, a winter temperature as low as -23°R (= -28.8°C) and the disease were all affecting the populations simultaneously. In Sohrau, a city which before the emergency had a population of 4,000, according to the medical lists, the number of patients at the end of the week extending from the 20th to the 27th of February was 109; at the end of the following week (27th of February to the 5th of March) it was 161, 6 patients having died. Thus, although the epidemic was spreading in the area and the number of patients had increased by half, the intensity of the epidemic was so low that mortality was only 5.5%. Since I have already reported that, at the peak of the epidemic, 600-700 dead had been buried in the graveyard, it necessarily follows that at that time there must have prevailed a much higher mortality rate; or else the epidemic would have spared none of the inhabitants. In the single month of January, 106 corpses were registered. In a report by Canon Heide in Ratibor (compare *Die Hungerpest,* p. 52) we read: "In the parish of Benkowitz, Ratibor district, which comprises

2,100 souls living in several villages, the parson has administered the last holy sacraments to 170 persons severely stricken by typhus in the period from the 6th to the 18th of January. Of these only 42 persons died during that same period. Chaplain Bienacki, whom I sent for to help the overworked and almost collapsing parson, visited 60 typhus patients in the period from the 19th of January to the 1st of February, and 55 patients from the 2nd to the 9th of February, and prepared them for death; of these 33 died." Although this report naturally only included the more severe cases it does furnish some indications, i.e., in 5 weeks there were 185 severe cases of typhus among the 2,100 inhabitants, and 75 deaths, corresponding to an 8.8% incidence of typhus among the populations and to a mortality rate of 40.5% among the patients In a meeting of the physicians of the Rybnik district convoked by Mr. Barez on the 26th of February, district physician Dr. Kunze estimated the approximate number of patients in the district at 5-6,000, and the district president von Durant at 6-7,000, the number of deaths amounting to 1718 (compare the data provided by Dr. Kiinzer, priest of the monks-hospitallers, *Die Hungerpest,* pp. 57 and 58). According to his figures every third patient almost should have died. However, the mortality data furnished by the physicians do not tally with these indications. Dr. Türk in Loslau and Dr. Wachsmann in Sohrau conceded a mortality rate of 10%, Dr. Chwisteck of Sohrau a rate of 20%. From these diverging opinions the lack of reliable data can easily be gauged.

May I now add some further information: In the parish of Lubom, Ratibor district, with 3,000 souls, 276 persons died of starvation, dysentery and other diseases in the year 1847, i.e., about 9% of the population; in January 1848, 83 persons died of typhus, i.e., about 2.7% of the population in one month *(Die Hungerpest,* p. 52). In Staude, mortality usually amounted to 28 deaths yearly, but in January 1848 alone it amounted to 46 (ibid., p. 42). In the parish of Pless (7,083 inhabitants) 161 persons died in the period from the 1st of January to the 11th of February 1848 (ibid., p. 64). In Lonkau, the death register revealed an average of some 60 deaths yearly for the past 5 years: in 1847 there occurred about 230 deaths, and in the months of January and February 1848, 86 deaths; the births also decreased steadily, for in the previous years there had been about 70 births per year, in 1847 some 20; in the two months of the year 1848 only 4. The number of patients at the end of February was some 60; the population of the village numbered about 1,400. In Geikowitz, where 450 inhabitants lived in 38 houses, the number of deaths, up to the 25th of February 1848, was 26.

As regards the actual mortality from starvation, we have fuller information on it with respect to the Pless district in a small brochure, *The Upper Silesian Hunger Pest. With official figures. A question addressed to the Prussian Government,* Leipzig 1848. The district of Pless covers 19½ square miles and had a population of 69,000 inhabitants; it thus possessed a very dense population (3,538 persons per square mile). In 1846 there died 2,399 persons; in 1847, 6,877. 97 of these, the physicians stated after medico-legal examinations, had died of starvation. According to a table in the appendix of the brochure, based on the reports of the parsons of the 25 parishes of the district and compiled by the rural district council, 907 persons died of starvation, i.e., 1.3% of the population. In all, a tenth part of the inhabitants died; from starvation and epidemics, 6.48%.

C. The Patients

The epidemic spared no nationality: Slavs, Germans and Magyars were infected, and the alleged immunity of the Jews, which Boudin traced back to Fracastorius, was here not at all confirmed. Of those who escaped the epidemic, it could not be said that their national origin or their previous residence constituted an explanation. Some held the opinion that this typhus fever was practically a Slav typhus and had to be considered as a peculiarity of the

Slavonic tribes. However, their own example has partially disproved them: daily experience was witness against such a view. If the typhus occurring in Middle and Lower Silesia is of the abdominal form this proves nothing, for it should not be forgotten that the conditions of life of the inhabitants are different.

Concerning the age of the patients, the majority were in the flower of their years, youth always predisposing for typhus. Nevertheless, numerous examples of infection in older persons are not lacking (compare cases V and VI), and I am not able to set a limit as to age. As for the situation in children, it is different: in general I have seen only very few cases of typhus in children; the lower limit according to Mr. Zillmer, regimental medical officer in Gleiwitz, was 2½ years.

I could not find any difference based on sex. Among the women several were pregnant, without the typhus showing any deviation in form. These observations therefore disprove the purported "mutual exclusion of the typhous and puerperal conditions." Hamernik *(Prager Vierteljahresschr.,* 1846, Nr. 2) admitted compatibility in the case of "anomalous typhus with imperceptible changes of the blood;" I, however, cannot concede any validity to his whole argument about the normality and abnormality of typhus and about typhous blood, for it totally lacks scientific method.

I have no precise information on the influence exerted by mode of life and occupation on the infection. In general, however, there seems to have existed a certain difference between the villages in the open country and the suburbs, which are similarly constructed, on the one hand, and the inner city districts which can boast of better location and construction, on the other hand, the latter being less severely affected. The poor, who live in insalubrious dwellings under very crowded conditions, i.e., the rural population and the workers, suffered most severely.

Lastly, we have to consider the individuality, or perhaps rather the constitution, of the patient. In general, I cannot assert that any special corporeal feature would predispose more for the disease. It has already been mentioned that persons who were exhausted by previous diseases (e.g. malaria) most easily caught typhus; this apparently was likewise the case with those who had suffered severe starvation at the beginning of the epidemic. Later, however, I also saw confirmed that particularly the young and strong were affected rather severely. Previous infection did not absolutely protect against relapse. During my short stay I myself could not make direct observations on repeated contraction of the disease, but I have heard of it from physicians, whose reliability is beyond any doubt. Thus, Dr. Wachsmann in Sohrau took me to a mill n the suburb of Klischtuwka, where a man, his daughter, a servant and a little girl all lay ill. The man had had typhus fever, with very extensive exanthema, at Christmas time. He had then completely recovered, had fallen ill again at the beginning of March and now again showed roseola. He died a few days after our visit. Dr. Dümmler, from Berlin presented another very striking example. He had contracted typhus fever in Prague a few years earlier, had then dragged himself to Berlin, and there again gone through the course of the disease with very extensive eruptions. Now he had fallen ill in Chelm (Pless district) and had a very difficult and late convalescence.

D. Nature and Cause of the Disease

All physicians who have personally observed the disease (with the sole exception of the previously mentioned Dr. van Decken) hold, as far as I know, the opinion that the disease is a typhous affection. About its closer appellation, opinions differed to some extent. Initially, the disease was given the name of hunger typhus; Mr. Neumann at first declared it to be exanthematous (petechial) typhus (compare *Die Hungerpest,* p. 70); Mr. Kuh also agreed to the term typhus exanthematicus *(Med. Vereinsztg.* 1848, No.8); Mr. Barez, however, at the meeting of physicians in Rybnik, insisted on the identity

of the disease with the contagious typhus described by Hildenbrand.

As regards the causes of the disease, those who considered the disease to be hunger typhus believed either that it had been caused by starvation, or, as did the majority of the physicians, they declared it to be the usual endemic typhus which had only assumed such dimensions because of the conditions of privation. All, without exception, physicians and laymen, considered the disease to be contagious (compare Kuh); consequently some thought that the disease had been imported from Galicia and Austrian Silesia.

I may reply to these opinions with the words of Huxham: *Parum fortasse interest, num illam (febrem) putridam, malignam aut pestilentialem appellare velis;-quando petechiae erumpunt, quilibet illam exanthematicam aut petechialem vocat,-et quando a contagio exorta est, contagiosam.-Ego quidem de verbis contendam cum nemine, id tamen necesse est, ut aliquid habeamus, quo nostras ideas cum aliis communicare queamus, quae ubi bene sunt definitae, insignis quaedam rixae causa relinquitar nemini.* ["Perhaps there are small differences: you might wish, now, to call this fever putrid, malignant or pestilential. When petechiae break out, some one may call it exanthematous fever, and when it has arisen from contagion, another will call it contagious fever. I shall not argue about words with anyone. Nevertheless, it is necessary that our concepts be well defined when we wish to communicate our ideas to others, so as to arrive at good results. Then no one will find cause for dispute."] *(Opera physico-medica,* Lipsiae, 1773; Vol. II, p. 100). It is hard to reach a scientific conclusion on these different opinions, since the factors determining such a choice are only very imperfectly understood, and since science itself has not yet arrived at a definitive decision as to the character and the origin of this type of disease. What is typhus? and how does it originate and spread? These are questions which no one has yet answered satisfactorily, and which cannot be answered concisely at the present stage of our knowledge. If we want to gain some clarity, to arrive at even approximately correct results, we must attempt a short review of the historical development of the concept "typhus." As is well known the name already existed in the terminology of Kos, though only as a general designation for a series of diseases, whose connection I, at least, am not able to perceive. Hippocrates *(Opera,* ed. Kühn. Part II, p. 496-506), in the book on internal diseases, described four disease forms successively, naming each one of them τυφοδ. In my opinion the third of these descriptions could be interpreted as acute rheumatoid arthritis. That of the first form, however, nearly fits the later conception of typhus: it is represented as an acute disease of midsummer, which proceeds with violent fever, high temperature, heaviness, lassitude and severe weakness of the limbs, stomach trouble, meteorism and the voiding of foul smelling fecal masses, impairments in sight, in consciousness, etc. However, it is clear that, since there still are three other diseases classified under the name of "typhus" which do not fit this picture, there is a great uncertainty in the definition from the start. We therefore find in general that the name was very rarely used by subsequent writers up to the present century, and that our disease is listed under quite different names such as *febris ardens, maligna, putrida, synochus,* etc., because, to the extent that the doctrine of fever became more complete and more consequent from the ontological standpoint and from the point of view of its essential nature, it became split into the different categories of the nosological systems. How far this fragmentation has proceeded can be seen most clearly in Boisseau's book which was so long and so widely used in France *(Pyrétologie physiologique ou Trairé des fièvres. Paris,* 1831. *Ed. 4me)*

Among the most common designations under which the disease entity now recognized under the name of "typhus" was listed in the years before the establishment of pathological

anatomy and physiology there are in general three: *febris nervosa* (Willis), *febris mucosa* (Röderer and Wagler), and *typhus* (Sauvages, Cullen). As the one or the other of these schools predominated in different places, the one or the other name became established. Have we not experienced in most recent times that while long-winded discussions were going on in Southern Germany on the subject of "Schleimfieber" it was no longer remembered in Northern Germany what should be understood by such a term? However it was more or less agreed, since Cullen, that contagiousness was necessarily to be postulated in true typhus. Therefore, English physicians up to now were differentiating between typhus fever and persisting remittent fevers which the German doctors singled out as typhus from the reign of the natural history school onward. When we read the description of the West African remittent fevers, for instance the fever so accurately examined by M'William which wrecked the well known Niger expedition, these, according to the German point of view, can only be interpreted as typhous affections.

From the above it is easily seen how variable the meaning of the term typhus actually is, and how greatly the range of the designation varies in the different schools. Very characteristic in this connection is the official royal Prussian pronouncement on the subject. In supplement B to the regulation of the 28th of October 1835, on the methods to be applied in sanitary procedures against infectious diseases, it is stated under §29: "For a long time it has been customary to call nervous fever every fever that is prevalently combined with an affection of the brain and of the nervous system, and with great debility, and to call typhus a higher degree of the same condition. But we shall understand by typhus in the narrower sense only that type of nervous fever which, having initially originated from a peculiar corruption of the air, is then also transmitted and spread in its absence by way of infection, and because of this double condition assumes a more or less epidemic character." Here we thus find differentiation between nerve fever and typhus, between typhus in a narrower and a broader sense. The only criterion for true typhus mentioned is contagion, for primary development from a "peculiar air corruption" cannot possibly serve as a standard for any physician who has to follow the instructions of that regulation.

Only after anatomical studies had furnished new points of departure for the understanding of these affections did more accurate views arise. After Röderer and Wagler had directed attention to the changes occurring in the intestinal mucous membrane, in particular to those of the glandular system, Serres and Petit demonstrated, by exact studies, the constancy of the affection of the intestinal mucous membrane and of the mesenteric glands in certain fevers, which up to that time had been described as "essential" fevers. These observations initiated new confusion. On account of the localized affection, Serres and Petit had named the fever *"fièvre entéro-mésentérique,"* still considering the fever to be the principal factor; Broussais, who opposed such entities on principle, went a step further; he declared the local affection to constitute the main factor, and the fever to be its consequence and therefore named the disease *"gastro-entérite."* Bretonneau, Cruveilhier and Bouillaud only more sharply defined these views, the first giving the disease the name of *dothienentérie,* the second introducing the term *entérite folliculaire* and the third that of *entéro-mésenterite typhoide.*

While fever entities were rejected, inflammation-entities were created. In the long run such a point of view could not be maintained. To the extent to which, under the influence of experimentalists, the humoral view again began to find a place in medicine, and to which horizons were enlarged by experiments on putrid infection of the blood, the necessity of distancing oneself from local affections as a primary cause and of seeking a general cause again moved to the foreground. H. Boerhaave pointed out the resemblance between the

feverish nerve diseases and certain poisonings, for instance that with belladonna, and German physicians such as Horn already referred to the war typhus of 1813 as an "animal poisoning of the brain and nervous system" (Horn, [Observations on the treatment of the infectious nerve and hospital fever.] Berlin, 1814, p. 14). From the rather cautiously expressed views of Andral, Louis and Littré, the opinion that there occurred a change in the blood by the absorption of noxious substances gradually gained acceptance in France, until, in most recent times, Rayer practically derived the local affection from a general putrid infection and Piorry named the disease *entérite septicémique.*

In Germany, where, owing to the influence of Marcus Senior, of Wedemeyer and of others, the opinion that typhus consisted in an inflammation of the brain had been maintained for some time, an opinion which has also found partial confirmation in the observations of Hildenbrand, another view had in the meanwhile gained increasing acceptance, due to the Natural History School of thought, i.e., that typhus was based on a primary change in the blood and that the intestinal affection should be considered as a secondary local affection. The supporters of Cullen, Brown, and Broussais were increasingly relegated to the background, especially, since the young Vienna School – a somewhat impatient heir of the Natural Historians.-.began to exploit its legacy in a distinctly craseological sense, with the result that in the end a large family of different disease entities was created, which were all called "typhus." For a time chemico-physical examination of the blood seemed to indicate that certain changes in the blood (reduction of the fibrous elements, presence of ammonia, etc.) were characteristic for such a family, and a typhous blood mixture was frequently spoken of. Unfortunately, recent investigations have shown the fallacy of such a view, and, with respect to the blood, we are now just as far from our goal as we were at any time (compare this *Archiv* I, pp. 572, 563) [12].

If we honestly want to clarify our position with respect to typhus doctrine, we must admit that we still do not know anything definite about the cardinal point of the matter and that our arguments on this subject are nothing but more or less probable hypotheses. But so as not to be unjust we must point out that in so difficult a problem our knowledge has nevertheless advanced significantly and we have more accurately understood three particularly important points which had caused a great confusion in the old doctrine. These are the following:

1. The difference between typhous diseases and typhous conditions. Before, by anatomical studies and by the improvement of techniques, diagnosis reached the level of exactitude at which it finds itself at present, those febrile diseases were very frequently identified with one another whose phenomenology showed the presence of some particular affection of the nervous system, mostly of a depressive character (asthenia, adynamia, debility). Every febrile disease could be assessed as nervous or as typhous, if the affection of the nervous system assumed such a character. It was thus possible, for example, at first to call a gastric fever nervous and then to let it develop into a typhous fever (compare with the above cited regulation). In such a case the name typhus was conferred on a disease in which merely the depression of the nervous manifestations had reached a certain extent. This view collapsed as soon as it was recognized that the fever itself was only an expression of the general participation of the nervous system in the disease, the expression of the generalization of the disease, and that this participation may assume a character of depression under the most varied conditions. If we now include all those changes in the nervous system which simultaneously cause fever and nervous symptoms of a depressive character under the collective name of typhous state, it is evident that a large number of diseases must be capable of causing it, as, for instance, acute miliary tuberculosis, the major pneumonias, or putrid infection of the blood. In contrast to such

diseases with typhous conditions are the actual typhous diseases, in which the symptoms of depression of the nervous system necessarily belong to the essential symptoms of the disease and are not merely accidental, as in a pneumonia that has become typhous or nervous, those diseases in which great perturbations of the nervous system, such as shivering fits, usually accompany the initial stages of the disease, then allowing it to run a course that is regular, although not bound to sharply defined critical days, while in genuinely putrid infection violent disorders erupt over and again in an irregular manner, and in acute miliary tuberculosis the nervous system is not affected suddenly, but is gradually involved in the disease. We consequently limit the name typhus to a disease process which is always and essentially accompanied with the typhous condition, to a febrile process which runs a regular course coupled with depressive manifestations of the nervous system. From this difference there partly follows another, namely:

2. The difference between acute catarrhs that appear together with the typhous condition and typhuses that are accompanied by acute catarrh. All typhous affections have the peculiarity of affecting one or more mucous membrane systems in which there develops acute hyperemia, with changes in their nutrition and secretion. As is well known, the respiratory and intestinal mucous membranes in particular are the sites of such catarrhs, and I have already pointed out (this *Archiv,* I, p. 249) or that the diarrheas met in iIeotyphus do not stem from the affection of the follicles or from the ulcerations, but from the simultaneous intestinal catarrh. The Upper Silesian epidemic has furnished the best examples. But these catarrhs are not typhous; typhus fever with a catarrh of the bronchial mucous membrane is not a broncho-typhus, the catarrh being only a co-effect of the same cause that produces the actual typhous symptoms. The fact that it is associated with typhus, that it is a link in the phenomenology of the typhous process, does not prevent us from considering it in its particular significance. On the other hand we know that the catarrhal inflammation of the mucous membrane is *a priori* linked, more than any local affection with profound changes in nervous activity[13]. These changes other are so strongly apparent, particularly in the catarrhs of the upper part of the digestive canal, in the inflammation of the mucous membrane of the stomach, of the duodenum and of the bile ducts, i.e., in the so-called *febres gastricae* and *gastrico-biliosae,* in *gastroduodenitis* (Stokes), that they constitute the typhous condition, and that we see forms corresponding to the old concepts of the *febres gastrico-nervosae* and *biliosae putridae.* But if we consider the course of the disease, a differentiation from typhus is quite possible in the majority of cases.

3. The exanthematous nature of typhus. I do not know exactly how far back recognition of this tenet can be traced in the literature, but that it has already been emphasized with regard to war typhus can be clearly seen from Bischoff (loc. cit., p. 3). As is well known, the Natural History School has created one of its most brilliant paradoxes in that it differentiated between two main forms of typhus: the exanthematic and the enanthematic the petechial and the abdominal, i.e. that it granted equal status to the typhous changes in the follicular system of the intestine and to the exanthema of the skin. Perhaps there is no other maxim which more clearly shows the superficiality with which this school expressed its ontological analogies, and the self-complacency with which it presented such ideas to the world in monographic form. Let petechial typhus be studied from the writings of as many authors and on as many patients as you please, no difference will be found between the type of exanthema existing in it and in abdominal typhus. Roseola is present here as well as there, and if true petechiae purpural spots, etc., occur in the latter, real petechiae nevertheless occur sometimes, or in some epidemics even frequently, in abdominal typhus, as I myself have seen. The only difference lies in the spread of the

exanthema, in the number of the spots; it is, therefore, only quantitative, and abdominal typhus must therefore be included in the exanthematic disease as well as petechial typhus. But that the modifications in the follicular system of the intestine have been contrasted from the exanthema of the skin as internal exanthema is pure frivolity. The processes which can be observed in the intestine do not have the least resemblance to those taking place in the skin, while they are completely identical with those which are found in the mesenteric glands. The roseola is a circumscribed capillary hyperemia, the petechiae are circumscribed extravasations in the skin; the changes in the intestine and in the mesenteric glands consist of a process which, though it begins with hyperemia, ends with abundant and often necrotizing exudates. I therefore in all earnestness reject any games of analogies, while I most definitely support the view of the exanthematic nature of the typhous affections recently suggested by Roupell *(Treatise on typhus fever,* Lond., 1839), Andrew Anderson *(Observ. on typhus,* Glasg., 1840), Eustace *(Medical report on the fever hospital, Cork Street,* Dublin, 1841), William Davidson ("On Typhus in Great Britain and Ireland," translated by Rosenkranz, Kassel, 1843) and Dagincourt *(Gaz. Méd.* 1848, Febr., No.8). Eustace in particular has correctly pointed out that the recognition of the disease as having a regular periodic course that constitutes an interconnected process, directly arises from such a recognition.

Having underlined these three points, we can now proceed with some assurance to answer the confused question of the nature of typhus. I define typhus as an acute disease, which generally starts with a considerable upheaval of the nervous system and with severe fever, very soon develops a roseola exanthema[14], very soon also produces symptoms of depression of the nervous system, is accompanied by an acute catarrhal inflammation of the mucous membrane, and takes a regular, although not sharply typical course. I regard it as probable that the basic cause of the typhus is a change in the blood, its poisoning by noxious substances; I deny, however, that examinations of the blood so far have provided any support for this view. It is a simple consequence of this view, or more exactly, this view is the simple consequence of the observation that the changes so far demonstrated in the quantitative relation of the blood constituents to each other are not primary changes, but related only to the development of the disease. Whoever assumes a miasmatic or contagious origin of the typhous affections and does not perhaps enthuse about the theory of botanical or zoological parasites (Henle, or Holland) should never doubt that if the changes in the blood are to be regarded as the cause of the disease, they must be qualitative. That up to this time such changes have not been found, proves as little against this view, as does the fact that no specific substances have been found in the blood of patients suffering from smallpox and measles that differ qualitatively from those found in healthy persons. It would also be quite arbitrary to expect such substances to be demonstrable in the blood during the entire course of the disease. Would anyone expect that in metal poisoning, the metal taken in should remain demonstrable in the blood for as long as the effects caused by it in the nervous system? It is not necessary to invent hypothetical qualitative changes in the fibrin, the protein, and the blood corpuscle, as is done in the Viennese science of craseology; it is indeed more than unscientific to do so, because, as I have already shown previously, such changes in certain chemical substances would not fit in with any of the known groups of chemical bodies, neither under the isomeric, nor under the isomorphic bodies. It is possible that the substance which qualitatively deviates from the normal blood constituents and which causes the typhus, i.e., the typhous miasma, may remain in the blood for a certain time, but it is just as possible that it disappears easily, while the symptoms caused by it in the nervous and nutritional systems

continue their course.

The last difficulty we have to discuss concerns the significance of the local specific products of typhus, or, in accordance with our terminology, the products of the local and specifically disturbed nutritional processes. I have already mentioned that French medicine differentiates between typhoid fever and typhus fever to such an extent that it considers them to be definitely different diseases, whereas German medicine considers the petechial and abdominal typhus as two different manifestations of the same basic disease. The differentiating character between the two forms of the disease is the specific change in the intestinal follicles, in the mesenteric glands and partly in the spleen in the abdominal, typhoid, form. Davidson (loc. cit., p. 124) who asserts the complete identity of typhus and typhoid fever, believes that it would lead to an endless and very unphilosophical classification if simple swellings of the intestinal glands, such as are very frequently seen in Scottish typhus, were to be differentiated from the changes occurring in the typhoid fever of the continent. This, however, is not the case. The swellings described by Davison occur in pneumonia as well as in the typhous affections; they are, in the majority of cases, only the expression of a catarrhal affection, and pathological anatomy differentiates them in the most precise fashion from the specifically typhous changes. If we admit the latter and consider their absence in British typhus, we shall have to decide, in the main, what rank we want to assign to these changes. I have already mentioned above the opinion of the French authors (Broussais, Cruveilhier, Bouillaud), who assume an inflammation of the intestine or of its glands and consider it as the very essence of the whole process. Some German writers have gone even further and have considered the ulceration of the follicles, the intestinal ulcers, as the main factor. This is entirely wrong for the reason that the ulcer does not actually pertain to the typhous process, but falls under its results, just as an abscess or an ulcer of the skin does not pertain to the inflammatory process of the skin. It indeed quite frequently occurs that there is no ulceration in typhus, as a resorption of the exudate might take place during the stage of medullary infiltration and as ulceration depends solely upon the occurrence of the necrotizing (diphtheritic) exudate. If, thus, I cannot conclude that the intestinal affection is the essence itself of the process, I must nevertheless speak out with equal certainty against the opinion that this peculiar type of nutritional disturbance also pertains to the essence of the process. Typhus, with its changes in the intestinal and mesenteric glands and in the spleen, i.e., the entero-mesenteric, abdominal or ileotyphus, includes the entire phenomenology of petechial or exanthematic typhus proceeding without specific anatomical changes, as was very well shown by Davidson, and in addition a few other symptoms that are absent in the latter. If we consider petechial typhus as the simple form, then something more has been added to it, for abdominal typhus is a complicated form. What then is this complication? Anderson (loc. cit., p. 54) dealt with the matter too lightly. He considered abdominal typhus, typhoid fever, as a purely local disease of the intestinal follicles, which, as are other local affections, is frequently accompanied by a feverish condition and sometimes combines with typhus to constitute a compound disease having the symptoms of both. In a very similar manner does Stokes speak of a combination of the (Irish) typhus with ileitis. I cannot agree with this opinion because no one has yet seen such an ileitis without typhus and because the same reasons which can be adduced against the French view on the significance of local affection also apply here. If we observe the parts affected in abdominal typhus we find that they are more or less closely related to blood formation. The intestinal follicles and mesenteric glands are the main organs for the absorption and formation of the chyle fluid, which, in its turn is the building material of the blood; the spleen has a connection with the cellular building of the blood (compare *Med.*

Ztg., 1847, No.4; this *Archiv,* I, p. 571). Thus, in abdominal typhus there are added to the symptoms of the simple typhus further affections of certain organs connected with the formation of blood.

Should these diseases now be considered to be dependent upon the already altered blood, nervous system, etc., or should the changes in the blood rather be considered to depend on these diseases? Are these diseases, thus, primary or secondary? If one considers well founded what I have described above as constituting the essence of typhus, then there will be no question that the affections of the intestinal follicles, of the mesenteric glands, as well as of the spleen must also be considered as secondary, as depending upon the primary changes in the blood or in the nervous system, or whatever else is regarded as the primary site. An example will illustrate this point more convincingly.

No one can doubt that bubonic plague, the pest, is a generalized (not local) disease, whether it be explained according to the humoral or to the cellular theory; moreover, no one can deny that the diseased lymph glands (buboes) are a secondary manifestation depending on the general cause of the disease. Many writers, such as Jos. Frank and the entire Natural History School have attempted, by arguments of analogy, to represent bubonic plague as practically typhus, equating the affection of peripheral glands in bubonic plague with the affection of the internal glands in typhus. Even though a resemblance between plague and typhus cannot be denied, it does not follow that both belong to the same "disease family" and that a third form should be added to the two above mentioned forms of typhus, i.e., a plague-typhus. Otherwise I would certainly admit that my definition of typhus was too narrow; but in plague the petechiae appear only in the very late stages and I cannot consider either the petechiae or the carbuncles as equivalents of roseola. Pruner *(Die Krankheiten des Orients,* Erlangen, 1845, p. 380) described the Egyptian typhus in broad detail and mentioned its presence during plague epidemics. Possibly later investigations uncovering the real nature and cause of these diseases may reveal a closer relation of these two affections. For the present, however, let us not proceed further than the methods of natural science permit. Plague may only serve as an example of an acute disease in which the generalized disease causes a local affection of the glands, as an argument for the dependence of the gland affection in typhus on the general ailment, and no more[15]. Accordingly, we distinguish a simple and an abdominal typhus, conceding to the latter a secondary affection of organs that are in direct relation to blood formation as a characteristic peculiarity.

Other writers have previously raised the question as to what constituted the cause of this complication of typhus. Davidson discussed in a somewhat imprecise manner place, season, epidemic and other yet unknown influences, and stated in particular that in Glasgow 1/3 of the typhus patients exhibited the abdominal form, that the latter was rarer in Dublin, and that it befell 1/4 of the patients in London, but that incidence varied considerably at different seasons. Seitz *(Bemerkungen über epidemische und endemische Krankheitsverhältnisse,* 1848, p. 86) stated that if the place of residence and way of life had an influence on the formation and manifestation of the fevers, their form, inasmuch as it was the expression of prevalent local lesions, nevertheless seemed to depend upon atmospheric conditions. He based this view in particular on the observation that, in 1846, there simultaneously prevailed in Paris, London and Munich a typhus with very little involvement of the intestine. However, this observation, which, moreover, is not based on sufficient data, does not meet the case. Although the general character of the weather conditions in Berlin in the summer of 1846 showed no deviation from the picture sketched by Seitz, we have nevertheless constantly observed the most extensive intestinal modifications in typhus patients. And even though it is correct that abdominal typhus occurs frequently enough in

England (compare the illustrations in Bright's *Reports of Medical Cases.* London, 1827. Plates 13-15), it should not be overlooked that, as a rule, it is quite rare as compared to simple typhus, and that in Ireland especially the latter is almost endemic. Most probably local conditions of society determine the form of the disease, and we may suggest as an almost generally valid result, so far, that the simple form is the more frequent the poorer and more one-sided the food and the worse the living quarters. Albers *(Die Darmgeschwöre,* 1831, p. 101, 302 ff.) argued that scrofulosis was a predisposing factor for intestinal affection; although I cannot agree with his in part rather strange arguments, it does seem correct to me that abdominal typhus occurs more frequently wherever scrofulosis and tuberculosis most profusely develop, i.e., when, on account of the mode of living, there is a certain proneness for the affection of certain glandular organs. A precise answer to these questions will depend on the further development of medical statistics, which at the present time is one of our most urgent requirements.

After having dealt, if only cursorily, with these preliminary questions, it will now be possible to give an opinion on several of the views which have been quoted at the beginning of this section. In any case, the disease occurring epidemically in Upper Silesia must be interpreted as being typhus, since, as appears from our previous reports, it is an acute exanthematic disease, bound to a regular course, that presents from the very beginning severe, feverish perturbations of the nervous system, very soon develops nervous symptoms of a depressive character-especially with respect to the muscular system- and is accompanied by a catarrh of the mucous membrane covering the air passages, and an exanthema which appears as a roseola. We can moreover declare that we had simple typhus, not accompanied by any affection of the organs related to the formation of blood. From what has been expounded so far it follows that we reject the name "exanthematic typhus," because we have assigned an exanthematic nature to every typhus. We regard such a designation as no less pleonastic than to speak of exanthematic measles or of exanthematic glanders. The name petechial typhus might be acceptable because of its historical significance, but we prefer to propose that it be eliminated altogether from medical nomenclature, because it no longer corresponds to present views. The designation petechiae, in the present medical nomenclature, is not used in the same sense as previously. Fracastorius, who gave the first exact description of this disease *(Opera.* Lugd. 1591. *Pars I. De morbis contagiosis Lib. II. cap. 6*) used the words *lenticulae, puncticula, peticulae* as synonyms and from then on the name petechiae was used for a long time as a general designation for all flat macular exanthemas, whether caused by simple capillary hyperemia or by extravasation of blood. Wedemeyer, as I already mentioned, still speaks of primary and secondary petechiae and understands by the former the simple hyperemic spots on the skin and by the latter the spots of extravasate. The relationship between these spots and the disease, i.e., whether the petechiae should be regarded as a separate disease, or only as a symptom of a disease, and as the expression of certain changes in the body, has been controversial for a long time, as we can read in at length in Sarcone *(Geschichte der Krankheiten, die 1764 in Neapel sind beobachtet worden,* Zörich, 1772, Vol. III, p. 140). Authors did increasingly agree that the dark red spots passing into a leaden or blackish hue, (i.e., the true extravasations) are caused by a decomposition, a putrid corruption of the blood. The disease called petechial typhus frequently exhibits this type of spot, as we have also seen in .the Upper Silesian epidemic; however, they are only a secondary phenomenon, since almost all writers agree in contesting any regular relationship with the course of the disease. It is not permissible to name a disease after a symptom which is only an accidental, even if a frequent, consequence of certain conditions arising in the course of the disease. But the

petechiae from which the petechial typhus originally received its name were not spots of extravasate, but clearly roseola spots, *taches roses lenticulaires, lenticulae.* It fitted the mentality of the population to call the whole of the disease by the name of the exanthema. Measles, smallpox and scarlet fever are expressions for the whole of the disease process and also simultaneously for the exanthema, although it was initially valid only for the latter. Fracastorius precisely formulated it thus: *(febres illas) vulgus lenticulas aut puncticula appellat, quod maculas proferunt lenticulis aut puncturis pulicum similes; quidam mutatis literis peticulas dicunt* [The people call (these fevers) "lenticles" or "puncticles," because the spots look like lentils or flea bites. Some, corrupting the word, call the spots "peticles."] i.e., the people simply called the whole disease "petechiae" (spotted fever). Had this name been retained, the later nosology would have been less exposed to misinterpretation, and in particular the exanthematic nature of the disease would have been retained. Now the situation is simply that typhus, according to my definition, means the same as the condition which in the 16th century was designated as "petechiae." I do not regard as feasible the restitution of this old term as this would cause general confusion. Nevertheless, it seems important to remember that by the rash erection of nosological systems, each of which has again engendered dozens of new ones, we have gradually come to understand by the term petechiae, which originally designated that acute exanthematic disease which we now call typhus, the local spots of extravasate on the skin. And while we have previously shown that the term "exanthematic typhus" is a pleonasm, we believe to have now proved that "petechial typhus" expresses no more and no less than a tautology.

The next question which we are now able to tackle is the identity of the Upper Silesian typhus with the contagious typhus described by Hildenbrand. This question has no great general importance as the identity of the former disease with many other typhous affections quoted in the literature on epidemics could be discussed with equal justification; I am only dealing with it because special emphasis has been placed on this point. If, for the time being, we put aside the matter of contagiosity, a point to which we must return very soon, and restrict ourselves to the phenomenology of Hildenbrand's epidemic, I believe I must energetically reject any question of identity. I must, though, add that I also do not share the view that Hildenbrand's typhus was a simple, or, as we say, a petechial typhus. For, according to Hildenbrand *(Ueber den ansteckenden Typhus,* Vienna, 1810), in the 5th stage of the disease which, according to his classification, falls in the second week after the start of the chills, there appeared pains in the bowels which he considered to be inflammatory, which were always coupled with a tendency for frequent liquid, very foul smelling feces; meteorism was an "invariable" symptom during that time. These indications definitely denote intestinal affection, and even if one should be inclined to grant that it might perhaps have been a simple catarrhal affection, we should not overlook that our epidemic showed intestinal catarrh only exceptionally, while meteorism was completely absent. However, it would not be justified, in view of the imperfect state of pathology in Hildenbrand's time, to entirely disallow the possibility that truly typhous affections of the intestinal follicles, the mesenteric glands, etc., were present (which need not necessarily be ulcerations). Let us keep in mind that knowledge of the anatomical changes which typhus causes in every organ had only just become available in Germany through the publication of von Pommer's work *(Beiträge zur näheren Kenntniss des sporadischen Typhus,* Tűbingen, 1821). Hildenbrand, moreover stated (p. 72), that a "stupor quite similar to inebriation had to a varying degree been the most prominent and constant symptom during the whole period of the disease." In Upper Silesia this symptom was among the great exceptions. It was in particular the lower degree of brain affection, the complete clarity of thinking that was

characteristic for the epidemic. Typhomania, a state of apathy toward one's surroundings, of dreaming in a half-awake state, a thinking modulated by internal stimulus only, but in such a way that the reaction to strong external stimulation resembled that of a person just roused from sleep and only partly awake, such symptoms were almost always absent in Upper Silesia, even though they sometimes occurred. In Hildenbrand's account these manifestations were the most essential and constant. These quotations will suffice to show the great difference between the Upper Silesian typhus and the epidemic described by Hildenbrand.

From the various views ,on the character of the Upper Silesian typhus quoted above only two, now, remain to be discussed, i.e., whether it was a contagious typhus, or hunger typhus. Any conclusions on this point will coincide with the etiological questions which have also been treated at the beginning of this chapter, and we shall directly turn to their discussion.

* * *

All local physicians, as I have mentioned, were agreed that in the form of the disease the present epidemic did not differ from the endemic typhus of Upper Silesia. We cannot enter into a detailed discussion of this view, as all premises for such a debate are lacking. Yet I believe that, in so simple a comparison, the general and unanimous observations have a high degree of credibility. Nevertheless, I shall also discuss the other, divergent views, especially as the absence of pathological anatomical examinations creates a very great gap. Naturally, if the identity of the epidemic and of endemic typhus is granted, the opinion which derives the typhus from the famine, as well as that which considers it imported from the outside by infection, will have to be modified; the former in such a way that the endemic typhus had gained epidemic development on account of the famine, the latter, that this had occurred by mutual infection. It must be remembered that one opinion does not exclude the other. The local physicians also believed the endemic typhus to be infectious, and it could therefore be thought that starvation might have caused the disease to have become epidemic merely by raising the degree of contagiousness, or, on the contrary, that starvation simultaneously increased the number of cases.

Let us begin with contagion. When I came to the districts and saw one physician after another catching the disease, while all observers, physicians as well as laymen, regarded contagion as a common and everyday experience, I at first did not think of looking for proof. But I then recalled the discussions which had taken place in recent years on the contagiousness of yellow fever and bubonic plague at the *Académie de Médecine* of Paris, discussions which shook to their very foundations views on the contagiousness of infectious diseases thought to be immutably fixed by centuries of observation, and which paved the way for a complete revolution in quarantine legislation, I remembered the dispute on the contagiousness of cholera, and I took the liberty of posing the question to the physicians attending a conference in Rybnik to solicit closer proofs. It turned out that two facts were advanced as being particularly convincing. Mr. Durant, the district president, was later kind enough to provide greater detail, and I shall presently quote this information. But a few remarks must precede it.

If it is true that simple typhus is endemic in the Upper Silesian districts, then it is logical that the causes and the requirements of the disease must also be local and endemic. Everyone who will expose himself to these conditions will thereby place himself in a position to contract the disease. All people living under these conditions, i.e. a greater or lesser part of the population, is in this situation from the start; everyone who comes from the outside, every stranger, who is subjected to endemic conditions, is liable to become infected. Let us give some examples. In Rome, malaria is endemic; the inhabitants suffer from it very

much; every foreigner who neglects the necessary precautions, especially at night, can contract malaria. In New Orleans, yellow fever develops in certain seasons; everyone who moves to northern areas at the right time remains free; everyone who comes to the city can fall ill. It is thus with cholera in India, with the plague in Egypt, with the tropical fevers on the coasts of Guinea.

Now it is a fact that many physicians, clergymen, and monks hospitaller contracted the disease while carrying out their professional obligations in Upper Silesia. I here list the names of those physicians that were affected in Upper Silesia who are known to me, as I feel that the medical press has the duty to maintain in public memory the great sacrifices which the medical profession makes to society in respect to health and life without any expectation of obtaining the laurels of the soldier or the medals of the diplomat.

A. Local physicians

l. Dr. Kries in Rybnik†
2. Dr. Raschkow in Loslau
3. Dr. Babel in Pless
4. Dr. Chwisteck in Sohrau
5. Dr. Boss in Sohrau
6. Surgeon Preiss in Rybnik†
7. Surgeon Haber in Rybnik
8. Dr. Goldmann in Rybnik
9. Dr. Krieger in Ratibor†
10. Dr. Lemonius in Beuthen

(11. Dr. Tűrk in Loslau, in the summer of 1847)

B. Foreign physicians

1. Dr. Kuh, Prof. of surgery at Breslau in Rybnik.
2. Dr. Neumann, Assistant at the provincial lunatic asylum at Leubus, in RadIin.
3. Dr. Biefel, military chief physician at Breslau, in Rybnik.
4. Dr. Borchardt from Breslau, in Pless.
5. Dr. von Frantzius from Danzig, in Sohrau.
6. Dr. Steinberg, military chief physician at Potsdam, in Rybnik.
7. Dr. Glum, pensioned physician from the medico-surgical Friedrich Wilhelm Institute at Berlin, in Rybnik.
8. Dr. Scholler, pensioned physician from the same Institute, in Loslau.
9. Dr. Dűmmler from Berlin, in Chelm.
10. Dr. Stich, government physician for the poor in Berlin, in Pless.
11. Dr. Tichy from Berlin, in Pless.
12. Dr. Mittmann, head military physician at Ohlau, in Rosenberg.†
13. Dr. Rűhle from Berlin, in Rybnik.

Further,

14. Prince Biron of Courland, delegate of the Breslau Committee, in Rybnik.†

Although this sad list is long, it proves nothing as to the contagiousness of the disease. And if every single local and foreign physician had fallen ill nothing whatsoever would follow from that fact alone. Could not anyone who visited the patients in their lodgings have been exposed to the same conditions to which the patients were exposed? What if a society had a spirit of sacrifice that would make every member put his hands into the fire; would one then conclude from the circumstance that everyone had suffered a burn, that the one first burnt had infected the others? Certainly not! The fire would be infectious, but not the people. Thus as long as it is possible to explain the illness of the physicians from the primary endemic cause, these facts cannot be regarded as an argument for the contagiousness of the disease.

The same holds true for the clergymen and the hospitallers. Nothing that I have been told in this respect is valid. I am giving some examples as they were told to me: The parson of Deutsch-Weichsel had died of typhus, and his colleague Franz Grossek from Staude undertook to put his estate in order and to take over his ministrations. The daily and nightly efforts arising from the assumption of these duties exhausted him in the extreme; he was already feeling very ill and had to be carried to his sledge while still attempting

to bring religious comforts to these patients. He took to bed on the following day and died 3 days later (compare *Die Hungerpest,* p. 75). Two hospitallers who had nursed him, and the parson of Czwiklitz, a close friend of his who had again visited him, contracted typhus soon thereafter. A man from the parish of Boguschowitz, who worked in the Teschen area near Freistadt, where the disease had become epidemic, fell ill after his return and died very quickly, after "his face had become black." The parson of B., who had administered the sacraments to him was stricken immediately afterwards and died within a few days, with petechiae. Surgeon Preiss from Rybnik, who treated him, thereupon fell ill and died of the disease. Archpriest Ruska from Rybnik, who gave him religious consolation, was stricken twice, consecutively, with a gastric fever. The archpriest of Sohrau who had given the sacraments to many patients fell ill and died 4 days later. Many people attended the funeral. One of these, who had carried his coffin, died 3 days later. Among those who attended the funeral were the smith of Baranowitz and his wife. The latter who had been quite well up to then became ill, insisting that she had caught the malady from the corpse of the dean, and died 3 days later; the smith also fell ill and recovered only with difficulty.

I was told this by reliable but uncritical persons. It was no longer possible to check exactly the various facts in all cases; but whenever it was possible to do so inexactitudes were soon apparent. Thus, for instance, I had found it very remarkable that in the last case death had occurred so early, since I had gained the conviction that death only occurred between the 9th and 14th day. Dr. Sobeczko, who had treated the archpriest, accidentally remembered that he had already felt ill on the Wednesday, had first taken to bed on Sunday and died on Tuesday. Assuredly such established inaccuracies significantly diminish the reliability of the other points. Moreover, how could it even remotely be taken for sure that the infection of the physicians, clergymen and the hospitaller monks occurred from exactly that particular patient, since they had dealt with so many patients? True, one generally hears, not only from epidemiographers but also from practitioners who had fallen ill during the epidemic that, from a peculiar shock to the nervous system and a sudden horror or disgust during the examination of a certain patient, they had all of a sudden realized that they had become infected. Nevertheless, I cannot admit such a feeling to constitute scientific proof. I am not aware of having felt any petty fear when facing infectious patients, but I have never scoffed at the possibility of infection. However, I remember very well, particularly during the initial period of studying the Upper Silesian epidemic, that I was overcome several times by such a dread or distaste during the direct and close examination and palpation of patients. As in addition I suffered from quite acute gastric troubles, headaches, etc., for several days, I did suspect infection; had I really contracted the disease I would probably have been inclined to date contraction from the moment of overt distaste. If thus the infection of physicians and clergymen can hardly ever be traced back to a certain patient, we may assume only the general possibility of infection and the most powerful argument against this is the endemicity of the causes.

In Sohrau, public opinion indicated importation of the disease from the comitate of Wadowicz in Galicia through a Jewish trader by the name of Prager. His infection was said to have been kept secret and to have been propagated to other Jewish families by infection, its presence becoming known only when it extended to other inhabitants. Mr. Kunze, the district physician, had immediately checked on these rumors and had found that at the time when the Jew fell ill, the inhabitants of many houses of Sohrau had already died out. This is the extent to which one may rely on such tales.

Let us now consider the two examples which are regarded as especially conclusive. I quote them verbatim from Mr. Durant's report:

"On the 8th of December 1847, after typhus had already been rampant in many houses of the villages of Brodek and Roy, in particular in those of the latter village, a cow was stolen from the village of Paruschowitz, with clues leading to those two villages and raising hopes of finding the thieves there. Constable Dolezych followed the clues to Brodek and there arrested Joh. Machel, a man under police observation, and then, in Roy village, Franz Chylla, a man also under police observation (the wives and children of both men were abed with typhus), and brought the two arrested men to the guard room of the municipal council in Rybnik. In the following night he repeated his search in the village of Roy and arrested Martin Machel, Franz Motzka and Joseph Strongek, who had taken flight and escaped the previous night, and also brought them to the same prison. Then three of these prisoners were transferred to the prison of the Royal County and Municipal Court of Justice at Rybnik, and there fell ill with typhus, infecting all inmates, of whom 6 died in short succession. Typhus had also broken out at the city prison, and on the 19th of December Constable Dolezych fell ill, and a few days later privates Paczek and Swientek and municipal sergeant Walter who had all been present in the guard room. Moreover, the night watchmen Franz Sollonz, Carl Leschnig, Koch, and Jacob Rarzellick also fell ill, of whom the last named died. All had been present in the guard room. I ordered the disinfection and cleaning of the guard room, after the prisoners had been taken out. Of the 2 women who did the cleaning, Antonie Koch fell ill on the second day and Marianne Sollonz on the 4th day, to which I may add that they were the wives of the night watchmen and had already had typhus previously."

"Smith Pozimorski lives in the colony of Paulsdorf. An aunt of his had died of typhus in Sohrau and he was her nearest heir. His inheritance consisted of the bedding on which the deceased had died. Pozimorski took the bedding to Paulsdorf and hung it up in the attic. As he feared it might be stolen there he brought it into his room a few days later and placed it behind the stove. One night his son used the bedding and very soon fell ill with typhus. Within 3 days 7 houses became infected in the colony, which up to that time had been perfectly healthy."

These cases, which at first sight appear conclusive, do not stand up to critical examination. In the first group we see that five persons from villages where typhus was prevalent were taken away from their families which were also sick, and brought to Rybnik, partly to the municipal guard room and partly to the jail of the court of justice. In the former they came in contact with nightwatchmen, municipal soldiers, and so forth; in the latter with other prisoners. They themselves, as well as these other persons, fell ill. Afterwards the prison was cleaned by the wives of the affected night watchmen, and they in turn contracted the disease. These are the facts. The conclusion drawn from them was that these five persons had already become infected at home or had brought along with them a contagion which they transmitted to all persons who came into contact with them, and that the contagion had established itself In the jail so well that the women engaged in cleaning it had in turn acquired the infection. This conclusion is quite arbitrary. It is not proved that those five persons had brought the disease with them, and it is quite possible that they, as well as the remaining prisoners, only contracted the infection in prison. Had typhus not been present in Rybnik at that time it would be easier to disregard this possibility. However, according to the inquiries which I made, a chimney-sweep had the disease, and in a printer's house all inmates were ill with typhus; in this latter family the wife and the daughter had first fallen ill, later the father who took care of them had taken to bed and died. It is

equally hard to see why the wives of the night watchmen should have contracted the infection in the prison, as they had the best of opportunities of contracting it at home from their sick husbands. Moreover, the speed with which the infection and diseases are supposed to have succeeded one another contradicts all other observations. That lastly the night watchmen and the municipal soldiers should have contracted the disease in prison and from the prisoners is very doubtful, since they could as well have contracted it without such contact, as had been the case with the other inhabitants in Rybnik. The second case is even less convincing. The smith had not contracted typhus, although he had brought the bedding from Sohrau; his family had remained free from the disease, although the bedding was in the space behind the stove. From the fact that the son fell ill soon after having slept on the bedding for one night and that then seven houses had become infected within 3 days, it does not follow that the son was infected from the bedding and that the other inhabitants of the colony contracted the disease from the son in so short an incubation period. None of this can be proved in any way.

Let us now turn to the other criterion of contagiousness. In nearly all contagions, especially in the exanthematic diseases, there is an incubation stage of generally constant duration. The question now arose whether such a period of latency of the disease could be demonstrated in Upper Silesian typhus. My inquiries of the physicians at first seemed to show that this, actually, was the case and that the incubation period lasted from about 14 days up to 3 weeks. If this was correct we should have had a state of affairs similar to that so fortunately detected by Panum (this *Archiv,* I, p. 492) in respect to the measles epidemic on the Faro Islands, even though one could not expect connections to appear in such purity, as the conditions of traffic are much more diverse and infections would definitely cross. In general one might have expected that, with an incubation period of 14 days, the disease should always have occurred in the different families or houses at an interval of 14 days, provided that one did not regard the disease to be infectious during its whole course, in which case no conclusion was to be expected, and no investigation was possible at all. In the houses I could not confirm an assumed 14 day interval between infections; observations on the infection of foreign physicians directly contradicted this, as Dr. Biefel, for instance, was in the district for 9 days only (19th-27th of February) when he fell ill.

Lastly, we must list the facts which directly speak against contagiousness. As I have already said, the local physicians believed that the Upper Silesian typhus of the previous years was also contagious. Mr. Haber, who had treated patients in the mining district of Rybnik, in the military hospital as well as in their homes for 11 years, rejected the possibility of an infection from the cadaver, of transmission of the disease through clothes, etc., and only accepted transmission of the disease from man to man in the living quarters of the people, as he had never witnessed direct infection in the military hospital, where typhus patients in all stages of the disease shared common rooms with other patients. Up to my departure from Sohrau the last mentioned fact still applied to the military hospital. A few other patients lay among the typhus patients in common wards, for instance one with frozen feet. But the disease befell none of them.[16] However, it is clear that the assumption of contagion taking place solely inside the home is quite arbitrary. It is but the ontological formula for the fact that transmission from man to man has not been observed, whereas residence in a house where people had fallen ill was observed to be the cause of the disease. But this fact rather constitutes an argument against contagion and for the endemicity of the cause. Several of the physicians affected traveled to other places at an early stage of the disease, going through its course in Breslau, Liegnitz and Berlin. To date I have not heard of any infection starting from them in these places.

Summarizing all these facts we can only say

that up to now there are no facts available which prove contagion, and that, on the contrary, certain observations are against it, and that nearly all processes that have been adduced could be explained by the endemicity of the cause of the disease. Unfortunately, physicians are as yet not well accustomed to scientific methods; they are too submissive to instructions by the authorities, too devoted to dubious considerations of probabilities, to make an exact analysis of proofs. In natural science it is the custom, and the only logical way, not to consider anything that has not been proved. In medicine it is the custom to consider everything that has not been disproved as being correct and meriting consideration. Nowhere has this principle become more solidly established that in the doctrine of contagious diseases, and it takes the most tenacious of fights to move the opponents step by step from their traditional ground. Do not the medical historians go so far in the defense of old traditional opinions as to place the arbitrary assumption of writers dating back three or more centuries above the painstaking observations of new researchers! As if the authority of physicians who believed phthisis, malaria and cancer to be contagious could adduce the slightest of proofs for the contagiousness of petechial typhus, plague and yellow fever! This genteel manner of criticizing the living experience of daily observation from behind the green tops of desks and out of old books must at last stop!

If I thus leave open the question of contagiousness of the Upper Silesian typhus until the day when we shall have direct evidence, I would like to stress that I do not wish to deny it categorically. That typhous affections can become contagious is indubitable. According to unquestionable observations, the war typhus, the English typhus, even abdominal typhus (compare Louis, II, p. 368) are transmitted from man to man under suitable conditions. Riecke *(Der Kriegs- und Friedenstyphus in der Armeen,* Potsdam, 1848, p. 63) has tried to dispose of the controversy, which is always cropping up in regard to the contagiousness of abdominal typhus, by stating that the typhus contagion consisted in a miasma newly developed from the typhus patient, whereby the difference in opinion becomes rather one of expression than one pertaining to the subject itself. This seems to me the most fruitful remark contained in his book which has been diligently assembled but has become rather unpalatable due to its hodge podge of hypotheses, strange ontologies and facts. Experience indeed shows that the incitement of disease in a new individual only takes place at a certain intensity (concentration of the substance causing the disease) and duration of action, and that the degree of the affection stands in a direct relation to the degree of incitement, and that the efficiency of the contagion is reduced in the measure of its dissemination (dilution). As regards the last point in particular we have available most interesting detailed data in Bertog's description of a typhus epidemic which was rampant in Halle in the spring of 1844 *(Diss. inaug. de typho Halis vere anni 1844 observato. Halis,* 1844). The observations made show most unquestionably that the typhus contagion differs essentially from the contagents of the true eruptive diseases (smallpox, scarlet fever, measles), of syphilis, etc., in that in these latter there is no blunting in contagiousness by dissemination, no increase of contagiousness when increasing the amount of transmitted substance, transmission taking place by even the smallest particle of the causative substance. Nevertheless, I would not separate the contagion of typhus from contagia in general, as Riecke has done, but I would on the contrary, point out that the contagious diseases have been treated from too one-sided a point of view. These latter should be divided into at least three large groups. The first includes those diseases which always and without exception propagate only by transmission from one individual to the other, such as, in particular, syphilis and the acute exanthemas, in which the changes in the skin have a critical significance. The second group

includes those diseases which are caused by the transmission and propagation of animals and plants from one individual to the other, i.e. the helminthiases, scabies, perhaps thrush and probably favus. The third group, finally, includes diseases which develop in animal bodies under certain conditions but are then transmitted to other bodies from the first foci of development. Under these I classify the catarrhal and the diphtheritic inflammations of the mucous membranes, of which the former in its intensive forms consists of blennorrheas (gonorrhea, blennorrhea of the eyes, leukorrhea), also nosocomial gangrene *(typhus traumatique),* the various typhous affections, puerperal fever, glanders, anthrax and rabies. This group of affections is generally counted among the miasmatic contagions but not rightly so, as blennorrhea, glanders and rabies can only be traced to miasmas by force. The various members of this group show a number of differences from one another, but they are all alike in that the disease can develop spontaneously. Concerning their contagiousness, it would be wrong simply to transfer all observations made on infectivity in the first group to this group. I remind the reader of Magendie's successful inoculation of a dog with material from a man infected with rabies, the dog then biting two other dogs which became similarly rabid *(Journ. de Physiol.* Vol. I, p. 42). But the efficiency of the poison then ended. This observation is directly connected with the fact that, in typhus, the infection cannot be continued endlessly. Rabies and typhus (also glanders?) thereby differ significantly from syphilis and smallpox, in which transmissibility has no limit. In the former the causative substance, the contagion, is exhausted quantitatively, in the latter every particle, even the smallest, remains fully infective. Liebig's well known theory of contagion thus would completely fit rabies and the typhous affections. For, if contagion consists in the transmission of the internal movement in the atoms of the causative bodies to the atoms of another susceptible body by the impulses of the moving atoms, then it would befit such a mechanical view that the extent of the internal movement should correspond to the impulse, that the force of the latter would be gradually weakened by the resistance and the inactivity of matter and, by chemical attractions, and that the extent of the movement caused should thus stand in an inverse relation to the distance, to the extent of the movement. Such a simple theory definitely would not apply to syphilis and to smallpox (compare my essay on puerperal diseases in the *Verhandl. der Ges.f. Geburtshölfe,* Vol. III, p. 162). Accordingly, if anyone spontaneously and miasmatically contracts typhus, and others contract the disease from him by infection, and others again from the latter in such a manner that the intensity of the disease decreases with every member in the chain, one should not conclude, as does Riecke, that typhus does not develop a contagious agent, but only that typhus is contagious in another way than are syphilis, smallpox, etc.

Some have thought that the contagiousness of the disease was in direct proportion to the extension of the skin eruptions, and believed that petechial typhus was distinguished from abdominal typhus by these two features. Thus Hecker *(Geschichte der neueren Heilkunde,* 1839, p. 161), writing from the historical and ontological standpoint, stated: "The critical significance of the spotted rash and an almost plague-like (!) infectivity are the essential characters of petechial fever, and only when these characters occur together it is justifiable to assign the putrid fever of our recent times to this form of typhus." According to these criteria the Upper Silesian typhus would hardly be classified as petechial in the historical sense, for we have seen that the skin eruptions coincide with the exacerbation of the symptoms of the disease and have usually already disappeared before the crisis, and that contagiousness is not established with certainty. Nevertheless it can hardly be doubted that this epidemic entirely corresponds with the concept of petechial typhus as understood in the past decades, i.e., with the

morbus peticularum of earlier authors. As regards the relationship between exanthema and contagiousness, I would like to add that when I raised this question at the meeting of physicians in Rybnik, Mr. Kunze, the district physician, considered the cases with exanthema to be more dangerous and more infectious, whereas Dr. Raschkow stated that he had seen cases as severe and infectious without exanthema.

Enough on the question of contagiousness. I have not dwelt on some of the information I was given, such as the dissemination of the disease by beggars, or the contamination of whole households and families, because it was too vague to prove anything, and, in part, could be interpreted in a sense opposite to that given. Thus, only the relation of the epidemic to the famine now remains to be discussed.

If by famine we understand a condition of the body social whereby there arises a mere reduction in food, though to any assumed degree of severity, then the development of typhus can in no way be derived therefrom. Simple reduction in food can only cause emaciation, weakness or finally a typhous condition. But starvation can occur in such a way only in single individuals or in a small group of persons. A real famine, apart from the reduction in the usual foodstuffs available, always entails the consumption of more or less indigestible or noxious surrogates. To the extent to which wholesome food becomes rare, people attempt to replace it by unwholesome foods. Now, can such unsuitable foods cause typhus? This indeed seems to be the case. In the literature we find a number of examples in which the consumption of putrid meat, the drinking of water mixed with decomposing animal substances (e.g. where the wells and latrines were too close to each other and their contents had been mixing) caused smaller epidemics of "typhus." However, all these examples only indicate the possibility that animal substances in the process of chemical modification are capable of causing the typhous process. Wherever mainly vegetable substances have served as substitutes, we rather find scurvy developing, if not dysentery, as has been recently observed in Ireland and in Scotland. Now we have explained earlier that the afflicted Upper Silesian peoples primarily took their substitute food from the vegetable kingdom, and we would, therefore, rather have expected to find scurvy among them to be more prevalent than typhus. These are the reasons that seem to speak against the view that typhus can be directly derived from the famine.

One could, however, suppose that a famine in which mainly vegetable substitutes are used might cause typhus under certain climatic conditions. Such a possibility should not be rejected off-hand, as typhus epidemics are definitely connected with climatic conditions and in certain places almost regularly recur at certain seasons; and also because at the very time when typhus became epidemic in Upper Silesia, it also had reached epidemic development elsewhere. I have already mentioned above that this was the case in all neighboring Austrian provinces. How similar that typhus was to the form flourishing in Upper Silesia is evident from the descriptions of the epidemic seen in Prague. In the general hospital there the dysentery prevalent up to August 1847 receded, typhus gaining the ascendency in September-October (when 113 new patients were taken in and mortality was 1 : 14.6) and in November, by which time dysentery had disappeared altogether. All cases exhibited profuse exanthema, usually a moderate delirium, constipation and in general only a slight involvement of the abdomen, the disease taking a rapid and mild course. Sometimes convalescence was prolonged. The fatal cases usually came to a close in a few days, the cadavers showing no changes in the intestinal canal. The disease could be recognized only from the character of the blood (?), acute tumor of the spleen, and in certain cases by lobular pneumonia; in some of the cases that occurred in August an added dysentery was the cause of death *(Prager Vierteljahresschrift,* 1848, V, Vol. II, analecta, p. 119). The numbers of deaths in

Prague from the 16th of December 1846 up to that same date in 1847 was 5,192, which, in a population of 120,000 inhabitants, corresponds to a ratio of I : 23 *(Gaz. des Hôp.,* 1848. Febr., No. 15). Dr. Götz, head of the municipal hospital at Danzig, wrote to me on the 14th of April of this year: "Since February, when the catarrhal diseases (influenza, measles) declined, we too have been kept .busy here by a gastric fever of a moderately epidemic character befalling a population rather similar to the Silesian in its poverty and distress; in its course this affection increasingly assumes the purely typhous form (always with roseola, rarely with petechiae), and has the peculiarity that beside the morbid blood crasis there occur practically only local affections of the respiratory organs (catarrhs and pneumonic infiltrations of the lung tissues). Only in such cases is our fever fatal. The intestinal mucous membrane is always healthy." At the same time the region of Flanders is being decimated by typhus, and the deprivations of the population were regarded as the cause of this "terrible" disease *(Gaz. Méd.,* 1848*,* Febr., No.6). Even in Brussels the disease prevailed to such an extent since the beginning of this year that the mortality was as high as that in the cholera epidemic of 1832 (ibid., Jan. No.4); nearly 60 physicians have already died in Flanders as victims of their zeal and their devotion *(Gaz. des Hôp.* 1848. Febr. No. 13). The situation is quite similar in Ireland.[17] In the province of Ulster alone 27 hospital physicians contracted the disease of whom 14 died *(The Lancet,* 1848, Febr. I, 8) Mr. Rodier and Mr. Guénau de Mussy, who had been sent by the French Government to study the epidemic, both fell ill and the latter died. In the autopsies no changes were found in the intestines; only rarely was the spleen enlarged or softened. The most frequent condition found was venous hyperemia of the pia mater, sometimes accompanied by edema. The disease usually ran a course of 9-11 days, with frequent relapses; characteristic symptoms were especially red or blackish petechial spots, which were found most constantly on chest and abdomen, great prostration, high fever and severe affection of the brain *(Gaz. méd. de Paris,* 1847, Oct., No. 42). In Scotland too the epidemic was of huge extent. In Edinburgh, in the period from June to October 1847, 887 cases were admitted to the fever hospital by way of the municipal offices for the indigent alone; of these 485 died, corresponding to a mortality of 54.6% *(Lond. Med. Gaz.,* 1847, Oct.). Thus everywhere we find the same emergency and distress, the same neglected and, for the most part, bigoted Catholic population, the same typhus. Further information is not available to me at the present time.

In my opinion, the influence of the climatic conditions on health can no longer be interpreted as it was in the past by medical meteorology. If we consider the subject according to the present state of science, we can theoretically admit three possibilities as to how the weather may affect the human body:

1. Indirectly, by affecting the conditions on the surface of the earth, the water content of the soil, vegetation, the chemical transformation of vegetables and animal substances present in and on the soil, etc.

2. Directly, in that the temperature of the air, its water content, pressure, and perhaps the aerial electricity influence the processes of animal economy (metabolism).

3. Also directly, the air currents carrying with them foreign substances, foreign in their mixture.

Those authors who have discussed the relation of disease to change in the weather have usually considered only the second class of effects; the first one has been mentioned only in passing, while the third one as far as I know has been neglected altogether. For this reason I propose to give it some attention now, so as to make clear my views.

The air currents which pass over the surface of the sea take away with them not only water vapor but also carry off salt particles, as is the case, on a small scale, in the salt works

(compare Rhenius, *Diss. inaug. de atmolutro muriatico apud salinas. Berol.,* 1841). When the air currents come into contact with large surfaces of loose sands, particles are lifted and carried to great distances, over many degrees of latitude, as has been demonstrated by the studies of Ehrenberg on the remains of siliceous shells of infusoria in the dust deposited. If, finally, gaseous products are present from the start, it is self-evident that they are mixed in the atmosphere and follow its movements. The fine work of Dove on the law of rotation according to the points of the compass has shown that the movements of the atmosphere do not only consist of waves or vibrations of otherwise stationary layers of air but also that there always are two sorts of current in more or less regular succession, a lower one which carries cold and dry air from the poles to the equator and a higher one which brings warm and humid air from the tropics to the poles. The winds from the north to the east are polar, those from the south to the west are equatorial, those lying in between are more or less deflected by the collision of these two currents. Accordingly, the polar currents can convey to us products from northern countries and the equatorial currents products of tropical areas. The latter are more important, as the tropical sky is the actual site of chemical decompositions. Thus, if for example, one does not wish to regard cholera as a contagious disease it seems to me that there is no other alternative than to explain its progressive dissemination from east to west by such equatorial air currents charged with tropical products. Thus it could easily be explained how the cold of the last winter has hindered the progress of cholera in Russia and ultimately caused its extinction, for there exist similar and even more striking phenomena in other places too. Mr. Halleur, missionary in the Gold Coast, told me that in Ashantee the "climate fever" stopped at once with the advent of a peculiar wind, the Harmattan. We observe a similar state of affairs, on a smaller scale, with malaria that arises on high rocky terrain, when the wind is blowing from the direction of swampy areas, and it seems to me that great epidemics of malaria, such as the epidemic of 1847, which extended from Holland deep into Poland over the plains of Northern Germany also denote analogous conditions.

If we approach our typhus epidemic from such a viewpoint we cannot assign a direct influence upon the disease to any type of air current. As we have seen, the epidemic already began in the summer of 1847 and soon reached a great intensity in various places during a season in which western winds were prevailing. In Loslau, however, the disease broke out with eastern winds and reached its peak during the severest cold. In Sohrau the epidemic began to subside in January and the first half of February, and it was hoped that it would stop in milder weather. But exactly the opposite occurred. As the air, in the presence of equatorial currents, reached a relatively high temperature the number of patients again increased. The simultaneous epidemic occurrence among the Czech and Polish populations in the Upper basins of the Elbe, the Oder and the Vistula, among the Cassubes on the lower Vistula, among the poorer population of Flanders, Ireland and Scotland prove nothing since the country in between these areas remained free. Although on my return from Upper Silesia I was shown a few patients with alleged petechial typhus at the Charité Hospital in Berlin, these definitely were cases of abdominal typhus, partly with petechiae (extravasates), and partly with very extensive roseola; of two dead patients which had been so diagnosed, one, on autopsy, showed an acute miliary tuberculosis and the other extensive pneumonia.

Both occurrence and intensification of the epidemic have been observed in different places, and under varied climatic conditions. Thus the whole question hinges upon the point of the contagiousness of the disease, which unfortunately has not been settled. For if one could definitely assert that the disease was not contagious, one could also directly conclude that

it had not been caused by climatic conditions of any sort; but if, on the contrary, it was contagious, then its first development might have depended on atmospheric changes, while its further dissemination and increase could have been independent of them and could have been determined by the general conditions of contagiousness only. As I am not in a position to decide on this dilemma, I limit myself to attempt a judgment on the indirect or direct influence of the atmosphere.

Up to now we have no reason to assume that modifications in atmospheric conditions (i.e. its content of water vapor or water droplets, temperature, pressure, electrical potential) can directly create typhus, and even though one hears frequently enough that someone has contracted the disease by exposure to cold or getting soaked, it is well enough known what such ananmestic information is worth. But we must evaluate quite differently the indirect effect of atmospheric changes on the body, and we need only refer to our earlier communications (p. 340, 348) to select for the purposes of the present discussion that meaning which in our opinion it merits.

If, however, one grants such an atmospheric influence on the production of the typhus miasma, one thereby admits that starvation, whose relation to the epidemic has particularly concerned us, will assume a lesser importance. That the question of the connection between famine and typhus could have assumed the prominence it now has assumed has a historical reason. From the position taken up by the medical historians such a connection would have to be answered in the affirmative, since terms like *"hunger fever," "hunger typhus," "typhus famelicus," "famine fever"* are most common appellations among them. But this is but another of the traditions which has not stood the test of contemporary critique. Henry Kennedy *(Gaz. méd.,* 1847*,* Oct., No. 42), by his compilation of all fever epidemics which have stricken Ireland for over a century, has provided certain proof of its untenability. The years 1725, 1726 and 1727 were years of famine, but they did not bring epidemic fevers which, however, raged in the years 1728-31, although 1729 was so rich a year, that a writer at that time could incriminate the intemperate consumption of food products of animal origin as the cause of the disease. In the great epidemic of 1740-41 the fever was already very widely spread before want became pronounced. The severe epidemic of 1817-18 was already most prominent in 1815 when food was still abundant. In 1815 the fever hospital in Cork Street, Dublin admitted more patients than in 1817, despite the fact that the preceding year had had a poor crop, the crop of 1817 being better, and that of 1818 excellent; nevertheless, the epidemic persisted with increasing violence. The epidemic of 1826 subsided in the middle of a famine, that of 1836-37 appeared before the beginning of famine. The diarrheas and dysenteries which preceded the last epidemic (1847) and first began in the autumn of 1846 markedly diminished in frequency during the winter, as starvation reached its peak, to return in the spring of 1847 without any perceptible cause. The critique applied by the medical historians themselves not infrequently turned out to be too indulgent on account of their desire to place their information on a broad and general basis. See Hecker on diseases prevalent in 1770. After a very bad harvest in that year, epidemic disease broke out in a great part of Europe which bore the character of simple typhus (petechial typhus, spotted fever). Because of its topographical interest for our epidemic, I emphasize from among these an epidemic that broke out in Bohemia and which was described by Sagar *(Historia morbi epid. in circulo Iglaviensi et adjacentibus regni Bohemiae plagis observati annis* 1771-72). In Bengal, too, after repeated bad rice harvests, a famine broke out at the end of that year (1770), the like of which is not known in recorded history, and which, together with a devastating smallpox epidemic, wiped out 3 million people, i.e. 1/3 of the total population, within a few months. At that time cholera was not present anywhere in Bengal.

The only epidemic of which we have heard was that of smallpox, already mentioned. Hecker *(Geschichte der neueren Heilkunde,* p. 120) puts it as follows: "It is certain that, in the course of the famine, epidemics of common diseases were raging among the people; and we may assume with equal certainty that, as a result of drought and putrefaction, there prevailed not only malignant swamp fevers but also typhus with liver affections, as well as dysentery in its most virulent form." If this, however, had actually been the case we would have had information on it as surely as we have on the smallpox epidemic; and Hecker's assertion that his assumptions are well founded is the more uncritical as it probably arose only from an inclination to generalize, which, though understandable in a historian, is nevertheless inexcusable.

Although it can be demonstrated that famine and typhus do not necessarily stand in a causal relation, I would not like to support Kennedy's opinion that the simultaneous occurrence of both was an accidental coincidence. The logical mistake that was made in my opinion merely consists in that reflections on these causality relationships have been limited to the two factors mentioned, without taking into consideration that both might be coeffects of the same basic cause. Hecker in his views on the epidemic has, however, implied this possibility by paying great attention to the conditions of the atmosphere and the soil. But this was not very fruitful, because his exposé was more artificial than natural, more imbued with the sense of natural history than that of science, more historical than medical, and because with somewhat uncritical erudition, he evidently threw together a number of phenomena which have nothing to do with one another, finally arriving at a confused mysticism regarding an "impulse pervading all life."

A famine is usually caused by bad harvests, which in turn are caused by too great and persisting drought or humidity, whether the weather has such a character during the whole of the year or whether changes take place during that same year. (Expressed differently, for the temperate zones this would mean that the development of plants stands in a certain relation to the amount of warmer and more humid air carried to them by the equatorial currents, and to the precipitation of the water vapor they contain by their collision with cold polar currents). If, accordingly, a famine can be ultimately traced to the type of air currents, then the development of a typhus epidemic, if it really were a coeffect of the same cause, could be reduced to the same conditions that produce the famine; and if those conditions are manifest by excessive dryness or excessive moisture, we must ask whether the former or the latter or both are capable of causing typhus. From the considerations I have discussed in the chapter on endemic diseases, it follows that too much moisture may be regarded as an indirect source of typhus, and the relationship of famine and epidemic typhus would then consist in that both can be direct consequences of the prevalence of certain air currents but that both coincide only, i.e. are simultaneous, when the air currents carry with them a relatively large amount of moisture. In this way one could explain that in 1770, in Bengal, typhus did not break out, since the failure of the crop that had caused the famine was due to persisting heat, while in Europe typhous diseases broke out everywhere simultaneously with the famine after steady rains had been pouring down in torrents. Davidson (loc. cit., p. 77) assembled a large number of facts from which it would appear that the typhous affections used to reach their widest distribution in Great Britain and Ireland when there was much rain. He did, however, also provide statistical evidence, from Glasgow, against the assertion that in the cities typhus developed mainly on the banks of the rivers. But I cannot regard the question as settled, since, especially for Halle, facts just as definite speak in favor of it. Hartung *(Diss. inaug. de typho Halae autumno anni* 1841 *observato. Hal.,* 1842, p. 9) as well as Bertog (loc. cit., p. 13)

reported that, in Halle, typhus first appeared on the banks of the Saabe in two epidemics, with mention of the crucial circumstance that the disease broke out in houses with the lowest location. The manner in which climatic conditions had shaped up in Upper Silesia in 1847 has already been indicated above, in particular how extensive and long-lasting rainfall had followed on a period of intensive heat and how, accordingly, the food crops had failed to mature (the potato blight being especially widespread). Similarly, Fracastorius reported that the epidemic of 1528 was preceded by so warm and rainy a winter that many streams flooded their banks; mist exerted so noxious an action on plants that the buds spoiled, especially those of the olive trees.[18]

The question of the causal relationship of the typhus epidemic to the famine would thereby be settled, were it not for a further objection. English authors, in particular Alison and Cowan have stressed the influence exerted by economic slumps on the dissemination and severity of typhus. In 1836, 10,092 persons contracted typhus in Glasgow; in 1837, after the outbreak of the trade crisis, 21,800 persons fell sick; the greatest mortality in that year occurred in May, after 8,000 persons had become breadless in April as a result of the suspension of work in the cotton mills (Davidson, loc. cit., p. 87). After the slump of 1842, 1/6 of the poor in Scotland were affected by the fever, without any involvement of the middle and higher classes of society. In 2 months more patients had contracted the disease than in the preceding 12 years. In Glasgow, in 1843, 32,000 persons were stricken by the fever, i.e.12% of the population, of whom 32% died. (Engels, *Die Lage der arbeitenden Klasse in England,* 1848, p. 126). These facts are so striking that they cannot be countered, but it would be mistaken to conclude from them that starvation directly causes typhus. It can be related to the increase in mortality, but the production of typhus and its dissemination could only be traced back to starvation if it could be shown that the food and food surrogates, the housing, in short, the changed mode of life were not the main causes. Davidson has suggested this for the epidemic of 1837, and I would like to add that the coincidence of peak infection with the lay-off of the working men might possibly be accidental. For in Dublin, which, as I know, is not an industrial city proper, conditions were quite similar. In the fever hospital in Cork Street, 9,508 patients were registered in 1837, while in 1836 the number had been only 7,658; the largest number of registrations (1,105) occurred in May. Mortality reached its peak (14 ¾ %) in the same month, when prevailing winds, according to meteorological tables (G.A. Kennedy, *Med. Rep.,* pp. 3,40,87) blew principally from the west.

One might further ask in which way the typhus miasma that we have assumed is produced by the humidity of the atmosphere or of the soil. Some years ago a very similar question was raised in France which, as far as I know, has never been answered. Assalon, a physician in Dieuze, reported to the Academy *(Gaz. méd.* 1845, No. 29, 32) that in the years from 1829 to 1843 there occurred in the area where he lived alternating epidemics of intermittent fever, typhoid fever and carbuncles, in a regular 3 year sequence, and that depending on the periodical clearing out *(exploitation)* of a swampy pool which was formed by the lower Indre river. In the year when it was dry, there prevailed carbuncles; in the second year, in which the water again collected, malaria; and in the third year, when it was again filled with water, typhous affections. These indications were contested by Gabriel (ibid., No. 36) and to my knowledge, the controversy was never settled. The contradiction between the law of Boudin and the observations of Mr. Haber as to the relationship between the amount of water and the formation of typhus I have already mentioned above (p. 369-370), and this important point must therefore be left open for the time being. Theoretically, however, it is more probable that only a certain moderate degree of humidity, known to best stimulate

decomposition, would be suitable for the formation of the typhus miasma, since in the last resort we must derive it from products of chemical decomposition.

From the sum total of experience that we have gained with regard to the development of typhus, it is not probable that the typhus miasma builds up everywhere on the surface of the earth or even everywhere on cultivated areas in the way that the malarial miasma develops in swampy areas. The formation of the typhus miasma may be observed most surely and definitely in closed rooms, in overcrowded military hospitals, in prisons, work-houses, on ships; and, since we have shown above, how greatly the habitations of the majority of the Upper Silesian population resemble such localities and how they comprise all factors making for the deterioration of the air, the only item left to be discussed is to what extent the weather or the housing is to be considered responsible for the disease.

The great influence of housing on the state of health of the inhabitants is shown in the statistical tables. Holland, who was officially entrusted with investigating conditions in Chortton-on-Medlock, a suburb of Manchester, found that mortality in streets of the second class was greater by 18%, and in those of the third class greater by 68%, than it was in those of the first class; further, that in the houses of the second class mortality was greater by 31%, and in those of the 3rd class by 78%, than in those of the first class; and that mortality in the inferior streets which had been improved, was reduced by about 25% (Engels, loc. cit., p. 134). The two main diseases causing this mortality were tuberculosis and typhus. With respect to the latter, most of the English reports, in particular, show quite definitely that the disease was most prevalent among that population which is poorly lodged. Nearly half of the patients taken in at the fever hospital in Glasgow came from the outskirts of the town, 4/10 from the city proper, 1/11 from its surroundings. The actual foci of the disease within the city are those localities which are most crowded and where there prevails the greatest poverty (Anderson, loc. cit., p. 13). Riecke (loc. cit., p. 37) reported many instances of typhus epidemics which broke out in circumscribed localities and can only be ascribed to the type of lodging. Davidson, on the other hand, has assembled many facts meant to show that typhus is caused neither by putrid effluvia, nor by the exhalations of the human body, nor by dirt and impure air in closed rooms, but that these are only conditions of its dissemination; he does not seem disinclined always to attribute the original development of such local epidemics to the importation of the contagious agent. However, the only conclusion that can be drawn from all these facts is that putrid effluvia, exhalations by persons, etc., do not suffice to cause typhus under all conditions and those who have built the entire etiology of typhus on this basis have been in error. Davidson quotes, among others, the authority of the famous and most meritorious Howard, who did not believe that prison typhus should be attributed to a lack of fresh air and cleanliness. Everyone will gladly concede that these are not the absolute conditions for the formation of the typhus miasma, since nearly every pathologist has further postulated an unfavorable relation between the space available and the number of persons living in it, i.e., a collection of many persons confined in a relatively small room. Howard *(Nachrichten von den vorzüglichsten Kranken-und Pesthäusern,* Leipzig, 1791, p. 411) sees the true cause of prison fever in the sudden changes in feeding regime and type of lodging. But even if one might quote in favor of such a view the general experience that in the hospitals of cities, a significant number of typhus patients are those very persons who have only recently changed their place of residence and have moved to the city, one cannot, on the other hand, deny that the change as such explains nothing, but that only the peculiar type of the new conditions, the character of the new food and the new dwellings should be regarded as significant. For not every sudden change in

food and lodging is capable of causing typhus and only if the new food is bad and the new dwellings are unsuitable can those factors be incriminated as the cause of the disease. Change may have a certain significance, but at any rate the poor quality of the items exchanged is the main thing. Basically, Howard's data express nothing else but what I have stated in the chapter on endemic diseases of Upper Silesia. If, with Davidson, all epidemically occurring typhous affections were attributed to importation, to contagion, or, more exactly, to the direct transmission of an already existing disease, we would ultimately have to deny the spontaneous development of sporadic typhus. But the annals of medicine have preserved a sufficient number of examples of the latter. Of these I only wish to point out one from the English literature itself, which Davidson will not be able to refute. The typhus epidemic which broke out on the ships of the Niger expedition and which was described by MacWilliam and Prichard surely had a spontaneous origin. This expedition, whose real purpose was completely thwarted by the outbreak of that disastrous epidemic, has now acquired a great importance for the very reason that it has provided a precise experiment for the spontaneous development of typhus. As soon as the ships had passed the mouth of the Niger the epidemic broke out, striking man after man, and ending with the total infection of the crew. What further proof do we need? A large number of persons are placed under the same conditions simultaneously and all fall prey to the same disease, epidemic typhus, the nature of which, on the basis of autopsies, cannot be doubted. Having convinced ourselves that not only typhus as such, but even an epidemic of it, can develop spontaneously without direct transmission of a contagion from the outside, it must also be conceded that it would be most arbitrary always to object by raising possible importation in every epidemic, when the absolute isolation of the patients cannot be unquestionably proved. If we further find that putrid effluvia and the like do not produce typhus under all conditions, we must investigate under which conditions they do. Since we have previously discussed in detail the influence of humid weather, since we have pointed out the influence of poor and especially of overcrowded lodgings, we may now be bold enough to conclude that the combination of these two factors in conjunction with bad food, unsuitable clothes and lack of cleanliness are able to create the typhus miasma.

When many persons are crowded into a relatively small space the air will gradually be changed by their breath, the effluvia of their skin, etc. Exact studies on this point are as yet lacking. Some observations by Robert Smith *(Philos. Magaz.,* 1847, *Jan.,* Dingler's *Polytechnisches Journal* 1847, July, p. 106) may serve as a preliminary introduction. In the fluid which had condensed on the windows of a hall during a concert, Smith found 1% of dry residue; on evaporation, the liquid emitted a strong odor of human sweat and, on heating the residue there arose an odor like burnt meat, from which one may conclude that it contained a nitrogenous substance. If, as is mostly the case in Upper Silesia, there are added to these substances the exhalations of the animals which void their excrement into the room and whose feed covers the floor, when to these are added the gases rising up from the fermenting food stuffs (Zur, sauerkraut), and the vapors of cooking, etc., the degree to which the air is fouled will be easily realized. Nevertheless, we are not justified in considering these conditions sufficient to cause typhus. Assuredly a second factor is needed. Such a factor I am seeking in the warm and moist air which sustains and stimulates the chemical decomposition already begun.

Such warm and humid air can be led to Northern areas by an equatorial air current and we then shall witness the outbreak of typhus under the prevalence of Southern and Western winds in a relatively warm season. Such a relationship will best be able to develop at the height of summer, when the sun is in its northernmost position, and when the shores of the Mediterranean Sea fall within an enlarged

zone of tradewinds (Dove, in *Poggendorf's Annalen,* 1846, No.2, p. 259) and when, in our country, their encounter with the northern current produces a rainy season with variable character. In this manner we could explain the typhuses of summer and of the beginning of autumn, which would find their most conclusive analogy in the typhus that breaks out under tropical skies (e.g. the Niger expedition). The case must differ in winter typhus, when it is not the natural warmth and humidity of the air nor that reflected by the ground which acts, but the artificial conditions in the lodgings that result from excessive heating, cooking in the room, the cohabitation of many persons, the humidity of the floors and walls, etc. This, then, would constitute a view resembling that held for certain epidemics of plague by earlier authors, for instance by Samoilowitz with regard to the epidemic in Moscow, which has recently been taken up again by Pruner.

It therefore lies within the limits of permissible speculation to assume that the basis for the formation of the typhus miasma is given when warm and humid air is maintained under such living conditions. By this we do not intend to say that the miasma can develop in no other way. On the contrary, from the view that all conditions mentioned should only be considered as particularly suitable conditions for supporting chemical decomposition of a special nature, it follows that this same decomposition can occur under the most various conditions, but that the basic conditions, which will later be understood from a further analysis of these situations, must always be the same. I also do not wish to assert that the question has been exhausted by the factors enumerated and that there might not possibly exist a third or fourth factor in order to complete the sequence of conditions. This much, however, seems to be certain: the development of typhus cannot be derived solely from the nature of the dwelling, as might be illustrated by a comparison of the observations made with cholera and plague. As is well known, it is in particular the poorer classes which in both diseases are affected first and most severely. Closer investigations have shown that especially those worst housed suffered most. According to Engels (loc. cit., p. 85), Senior described a street in Manchester that followed the course of a ditch so as to obtain deeper basements without the costs of digging (these cellars to be used as living quarters for people), and on this street not a single house escaped cholera. In regard to the plague epidemic of 1841 Pruner (loc. cit., p. 390) reported that, in the Nile Delta, the epidemic moved from east to west, bypassing some villages or groups of villages and being most severe in areas which were low and lying along the Nile. In Cairo, strikingly, the low lying humid and crowded houses of the Jews and Christians were those particularly affected, especially if they were exposed to the South wind and shut off from the North wind. In Upper Egypt the plague was observed only in certain very unsuitably situated places, among poor ilI-nourished people. We thus see that under certain general conditions, which we can only consider as depending on the climate, the quality of the housing does indeed constitute a determining factor for the development of these diseases, while the dwellings in themselves would not have produced such diseases. The history of the typhus epidemics themselves provides the most useful data in the matter. I shall only refer to the recent Torgau epidemic of 1843. In it the disease first erupted in the barracks among the military who were housed in inexcusably poor quarters. 50 days later it broke out among the remaining population, 21% of the soldiers falling ill, but only 4.3% of the civilian inhabitants of Torgau, while from among the farmers and people working in the open air no one was affected (Köppe, *Der Abdominaltyphus in Torgau in 1843*, p. 23). Riecke (loc. cit., p. 160) has tried to explain this state of affairs solely by a miasma developed from latrines, but he forgot to explain why the epidemic broke out among the soldiers in Hartenfels Castle at that very time, since the incriminated latrine had always been there. He might perhaps have found such an explanation,

directly or indirectly, in the climatic conditions; If we transpose Köppe's report (p. 19) into the language of recent meteorology, we see that during April, when the epidemic reached its peak among the soldiers, the Torgau area was the site of a struggle of polar and equatorial air currents which met one another with violent electrical discharges and precipitation of water and which alternatively displaced each other, so that in the first half of the month there prevailed south-western currents and in the second half north-eastern currents. It is quite possible that even such knowledge of meteorological conditions is not yet exhaustive, that a few factors must yet be added to the sources of that typhus. Later study must elucidate this point. In the meanwhile we emphasize only that we have recognized two different factors as important.

Applying these observations to our epidemic, in case later studies should demonstrate the contagiosity of the Upper Silesian typhus as a fact, the course of the disease could be understood to have run as follows: The first cases occurred when, at a relatively high temperature, and as a result of the collision of polar and equatorial air currents, rains fell in great quantity; these at the same time caused a famine by impairing the development of plants used for food; they also promoted various processes of chemical decomposition, exposing the inhabitants who were crowded in unhealthy dwellings to conditions that favored the contraction of the disease. Later in winter the epidemic developed to extreme intensity when, on account of the severe cold, the inhabitants stayed in their houses, which they overheated stiflingly, even more, retiring behind their stoves. From then on the disease might perhaps have spread to the wealthier classes by infection. Accordingly, starvation and typhus were only an indirect coeffect of the same cause (the weather); starvation might have increased the predisposition for the disease, reduced the resistance of the nervous system and increased the mortality, but starvation was not the real cause of the disease, as little as the latter depended solely and alone on the weather.

Since the disease is endemic, its cause must also be endemic, and the cause for its epidemic appearance cannot be an altogether new one, but only an aggravation of the old one. We have assumed a particular miasma to be the endemic cause, which was a product of chemical decomposition that occurred when the unwholesome circumstances resulting from the mode of life of the inhabitants in their dwellings are intensified under certain climatic conditions. Accordingly, we have always felt obliged to seek the cause of an epidemic in such an intensification of domestic insalubrity and to attempt a refutation of opposing views.

We, moreover, have recognized it as probable that such a miasma, i.e. a substance undergoing a chemical reaction (movement of atoms), a chemical agent, reaches the body, poisoning the blood and causing a series of secondary changes in the life processes, in the nutritive processes as well as in the nervous system. We, however, protest against the view that the infection of the blood must persist for the entire duration of the disease, and we have pointed out that the only demonstrable changes occurred in the blood cells (i.e. multiplication of the colorless corpuscles), changes that we have defined as a derangement in tissue formation.

In order to illustrate our opinions in a more concrete manner, we shall present yet another analogous example. It is known that gonorrhea patients using copaiba balsam will show various symptoms. Ricord (*Gaz. des. hōp.* 1846, May, No. 59; 1847, Oct., No. 128) distinguishes mainly three effects: a revulsive or purgative effect, a general effect depending upon changes in the blood, and lastly a direct antiblennorrheal effect, due to the passing of certain constituents of the drug into the urine. If we analyse this more exactly, we find that the balsam first acts as an irritant on the surface of the intestinal mucous membrane, causing increased secretion and movement. According to the suitability of the conditions for an emulsive dispersion of the

balsam, resorption by the chyle vessels will take place and the drug will then reach the blood. Whether it there causes other changes than those due to its mere presence we do not know, but we perceive, by the changes in the nervous system and in the processes of nutrition and secretion, that its presence becomes evident. Among the former processes Ricord particularly mentions severe congestion of the brain which can intensify up to hemiplegia; among the latter we must mention an exanthema of the skin in addition to the modification of kidney secretion. But the true classical and regular form of the copaiba exanthema is a roseola that appears preferentially at the joints, always on the side of the extensor, next to the ears and at the back of the neck, especially in spring and autumn when the weather is cold and humid, after the first few doses of the drug. According to Ricord the exanthema appears in particular when any direct antiblennorrheal action is absent. In his view, the aromatic principle then goes through the skin and is absent from the urine. But if it takes the latter path there sometimes occur severe lumbar pains starting in the kidney area, i.e., there is a manifestation of an inflammatory irritation of these parts. The exanthema commonly disappears on the 8th day after administration of the drug.

We here have a simple so-called drug disease. A balsam that contains a volatile etheric oil is introduced into the body and causes symptoms in the intestine, in the brain, on the skin, in the kidneys, partly directly and partly indirectly; we see in particular the appearance of an exanthema the phenomenology of which shows the greatest agreement with the exanthema of typhus. It surely cannot be disallowed that a part of these effects at least depends on the absorption of that etheric oil by the blood and on a poisoning of the blood. But it would be wrong to conclude therefrom that there existed a further modification in the consistency of the blood and especially a change that would persist for about those 8 days during which the exanthema is present. A relatively short and transient presence of etheric oil in the blood is quite sufficient to explain the symptoms, though not too short a presence, for we see that the more completely, and the earlier, the drug is excreted by the kidney, the less prominent are the other effects. These hints will suffice to illuminate clearly the analogy with typhus, which I intended to demonstrate so as to show in particular to what extent the assumption that typhus miasma is a (volatile) chemical substance can explain the manifestations of typhus and will allow integration of the positive observations of pathological physiology.

Endnotes

9. The physicians in Breslau had placed a black tablet with the inscription ‘typhus’ in front of his house.
10. The Goralles are inhabitants of mountainous regions (gora = mountain) travelling in foreign countries and trading with dried fruits and similar items. They are characterized by their peculiar costume (narrow trousers, large flat hats, brown raincoats) and by their fine figures.
11. The most recent issue of the Medicinishe Vereinszeitung (Nr. 16) publishes a letter by Dr. Adloff from Pless in which he states, with regard to the results of the autopsies: “In all cases there was a bloody infiltration in the whole intestine, though particularly so in the ileocecal area. I found ulcers only once, but not as large ones as I have often seen them in other instances.” This report which deviates from all previous experience is so obscure and so dubious in its brevity that I cannot take it into consideration. “Bloody infiltration” cannot mean anything but an infiltration of hematin in the tissues caused by decomposition, for no one has ever seen extravasate through the whole intestine andno one would call capillary hyperemia an infiltration. ‘Ulcers’ per se are not significant, as catarrhal ulcers are well known to exist. Reports of this type only have value if they are scientifically exact.
12. All authors who have written about “typhoid fever” have discussed the changes in the blood at a lesser or greater length; but they are generally agreed that, at the present level of science, we are not very advanced in this study (Jaquot, Gaz. Méd. 1845, August, No.33). (Transl. from the French).
13. Zeller, in Winnenthal (Zeitschr. F. Pyschiatrie, 1844, I. 1,p.61) stated: “It seems increasingly

probable that the conditions of the mucus membrane of the intestinal canal has a greater influence on the general feeling of well-being and indisposition, on the tonus of life and on exhaustion, than that of any other tissue, and that other pathological conditons of the brain and spinal cord are most easily reflected in it."

14. Anderson (loc. cit., p. 20) stated: "The eruption is necessary to constitute the disease; other symptoms may be absent." In Glasgow, among 2,852 cases he found exanthema 1,885 times = 80%. Roupell gave the ratio in London as = 70%. Cowan in Glasgow as = 73.99%. Chomel in Paris as = 77% (compare Davison, loc. cit., p. 32ff.). If the exanthema is frequently not seen, one should remember that patients very frequently only come under medical observation at a time when the exanthema has already faded.
15. I would here like to criticize an expression which we have heard from the followers of craseo-ontology to the point of nausea. It is said of a series of diseases, arbitrarily derived from a humoral basis, that, when no local symptoms were present, they were proceeding in the blood, and when local affections were present, that they had become localized. Thus, according to this view, if anybody swallowed hydrocyanic acid and died of it, the disease (i.e. poisoning) would have proceeded in the blood. But what would be happening in the blood, if anyone fell on his head and died (due to a concussion of the brain)? It should not be forgotten that the entire series of symptoms, for instance those of a typhus, might be initiated by a poisoning of the blood, but that all later processes might then be triggered from the nervous system affected by the substance produced, long after this substance has been excreted from the body and the blood has again become purified. Then, however, the disease mainly proceeds in the nervous system and not in the blood. Why all this localization? If it is to have any meaning whatsoever, one should only take into account critical localizations; since that which becomes localized, is necessarily removed from the blood. If this simple logical conclusion is disregarded, the expression loses all sense. A storm which rages in the upper layers of air, driving before it the clouds, is like the disease which is supposed to proceed in the blood, like the internal movement which forces the blood-constituents apart or together to form new substances and combinations. But when the storm comes nearer to the surface of the earth, uprooting trees, damaging houses and smashing ships to pieces-has it then become localized?-not at all; it blows over the surface, devastating everything that cannot resist its power, and only after a long time and in the far distance is its power broken.
16. Dr. Stich, who has just come back from Pless, tells me that at the military hospital in the Hechlowska near Pless a nun of the order of St. Elizabeth of Thuringia, who had come in contact with patients only, had contracted typhus. This would be the first fact supporting contagion with certainty, if the information as to her contacts were confirmed.
17. According to a compilation published by Cusak and Stokes in the Dublin Medical Quarterly there died, in 1847, 178 Irish physicians out of a total of 2,700, i.e., 6.74%, and among these were 64.51% who had died of typhus. From an average of 10 years, the mortality from typhus in Ireland was 1 : 10.59, but among the physicians it was 1:1.55 (The Lancet, 1848, Febr. 1, 7).
18. This agreement is particularly interesting, as this epidemic showed great similarities with the Upper Silesian one in another point also. As in the latter, a very long preliminary stage was observed, sometimes without any suspicious symptoms; Fracastorius relates that Andreas Naugerius, a representative of the Republic of Venice, came to France to the court of François Ist at Blois on the River Loire and there was snatched away by the disease, which was still quite unknown in that country, within a few days.