North Manchurian Plague Prevention Service Reports (1927-1928) / edited by Wu Lien Teh.

Contributors

Dongbei fang yi chu.
Wu, Liande, 1879-1960.

Publication/Creation

[[Tientsin] : [Tientsin Press], [1928?]

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REPORTS
1927-1928

NORTH MANCHURIAN PLAGUE PREVENTION SERVICE.

EDITED BY

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Being Volume VI of the Series

PREFACE.

During the two years that have elapsed since the publication of Volume V of Manchurian Plague Prevention Service Reports, our staff has experienced a fairly quiet time owing to the absence of serious epidemics throughout Manchuria. However, towards the end of summer of 1928 news reached us of outbreaks of bubonic plague occurring simultaneously in Outer Mongolia, Shensi, Shansi and the newly opened-up regions of Fengtien Province around Tungliao district. To every one of these localities we were able to dispatch supplies of antiplague vaccine and serum.

In the case of Tungliao, our trained personnel was entrusted by the high Manchurian authorities with the complete management of the epidemic, and the necessary funds and cooperation with local officials and railways were available without difficulty or delay. As a result, what appeared at first a most alarming outbreak, threatening to assume pneumonic features with the approach of cold weather, was localised and speedily suppressed at a cost of only 500 lives and thirty thousand dollars. By adopting such prompt measures we managed to keep the adjoining South Manchurian Railway entirely free from the disease. From the time when our Dr. Winghan Chun obtained the first bacteriological proof of plague in this area on September 7 to the last case recorded on October 30, less than two months passed—a very creditable performance.

Since most of the material for this Volume VI has already been sent to the press in September, I am unable to publish at present more than a few preliminary notes on this epidemic. However, a full account, covering not only clinical and epidemiological aspects of the outbreak but also interesting researches made by our field laboratory staff, will appear early next year as a special Plague Supplement of the National Medical Journal of China.

From the forty odd articles and 70 illustrations comprising the present Volume, it will be found that our labours of the past two years have been mainly concentrated upon a study of the hibernation problem among tarabagans. We trust that these observations will also throw new light upon the question of perpetuation of plague among other species.

Likewise, I hope that the summaries upon pneumonic plague and plague in wild rodents, which supplement the material contained in my "Treatise on Pneumonic Plague", may assist fellow-workers in keeping abreast of the ever increasing literature dealing with these subjects.

My visit to India lasting from December 1927 till March 1928 enabled me among other things to study the plague problem in that vast endemic center, as well as to ascertain at first hand the views of numerous colleagues who had devoted their lives to this question.

A somewhat novel feature of the present Report will be found in the accounts of scientific expeditions undertaken by our expert staff to Mongolia and North Manchuria. This, I hope, will be the forerunner of joint undertakings which are now under consideration.

Turning now to public health as a whole, I am gratified to see that the Nationalist Government of Nanking has established in October 1928 a Ministry of Health with Mr. Hsueh Tu Pi (former Minister of Interior) as Minister and Dr. Liu Jui Heng (Superintendent of the P.U.M.C. Hospital, Peking) as Vice-Minister. The entire nation will greet this welcome innovation intended to promote the physical, mental and economic welfare of the people.

Our close connection with the League of Nations Health Bureaus at Geneva and Singapore has been fully maintained. Similarly, our happy associations with other health organisations of the world, such as, U. S. Public Health Service, South Russia Plague Prevention Service, etc., have been continued with mutual benefit.

As on former occasions, a Chinese edition is being published, for which Generals Chiang Kai Shek (Commander-in-Chief of the Nationalist forces) and Chang Hsueh Liang (Generalissimo of Manchuria) have kindly contributed forewords.

WU LIEN TEH.

Harbin, December, 1928.

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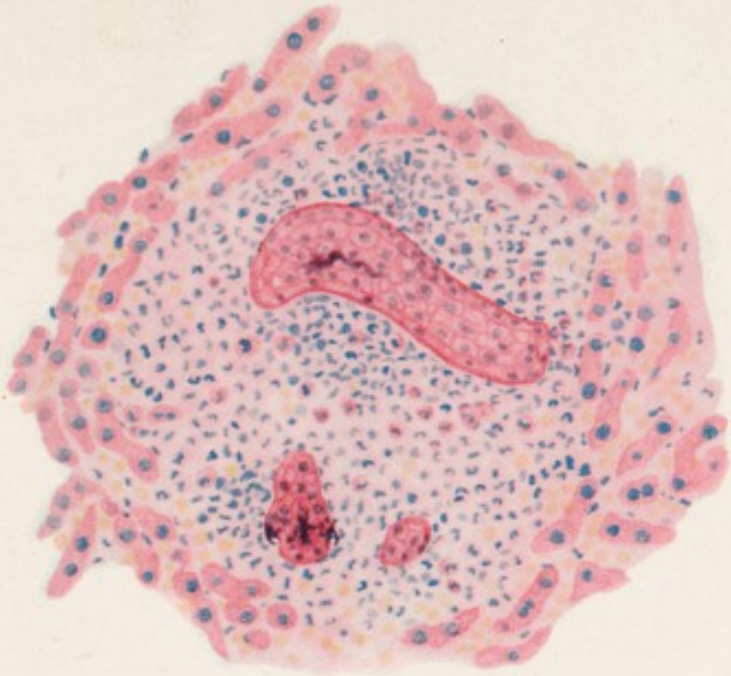

Fig. 1. Section of Liver from Tarab. 17. stained with haematoxylin and eosin. Magnif: x 350 diam. Showing node containing a new parasite cut longitudinally (one part) and transversely (two parts) In one transverse section (headpiece?) hooklets are seen.

（圖 一） 示旱獺十七號之肝組織用歇麻篤斯林及依奧神染色三百五十倍擴大結節部含有新原虫縱斷(一部)橫斷(二部)橫組織(頭部上)均著明

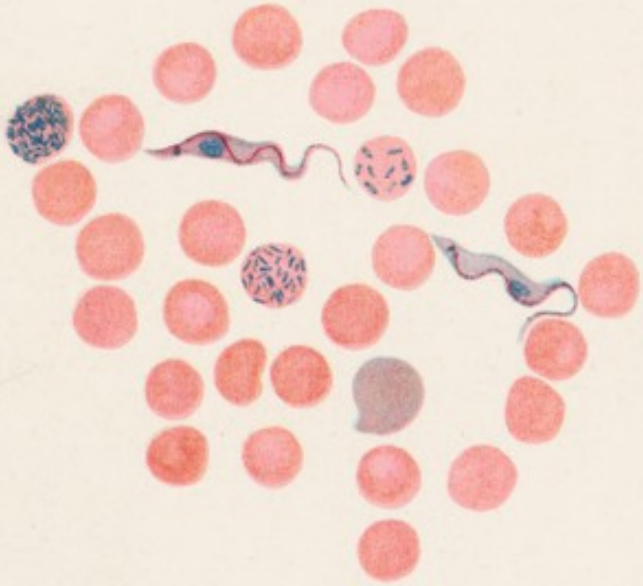

Fig. 2. Blood film from a big black vole of Sansing (town on the Sungari in North Manchuria). Stained with Giemsa. Magnif: x 980 diam. Among red cells three erythrocytes are seen containing Bartonella. Also two trypnosomes free-living. One large erythrocyte is seen with polychromatosis.

（圖 二） 示由三姓東省松花江之埠產大黑哈士特鼠血液標本用劍沙染色九百八十倍擴於赤血球中有三個內含巴唐尼拿及篤利巴羅蔬麻二條遊行血中又有一大赤球現過染

THE PERPETUATION OF PLAGUE AMONG WILD RODENTS.*

(First Communication).

(*Reprinted from American Jl. of Hygiene, 1928*).

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A. INTRODUCTION.

As in other epidemic diseases, one of the most fundamental and withal fascinating problems in plague is the question as to how infection is preserved and propagated from season to season, often without the presence of human cases and manifest rodent disease.

In this report I intend to deal with wild rodents, but for the sake of comparison it may be worth while to touch at first upon the situation as it affects domestic rats.

In their Summary on the Etiology and Epidemiology of Plague (1) the Indian Plague Commissioners come to the conclusion that the periods between the epidemics are bridged over by cases of *acute* rat plague, the epizootic being kept in check by :

(*a*) a high mean temperature prevailing,

(*b*) a diminution in the total number of rats together with an increase in the proportion of immune to susceptible animals, and finally

(*c*) a diminution in the number of rat fleas.

In an earlier contribution (2) the Commissioners had already refuted the theory that cases of chronic (or better resolving) rat plague are important in the propagation of infection. Among the reasons given are :

(*a*) It is unlikely that rats might contract acute plague by feeding upon the carcases of chronic plague animals;

(*b*) There is no evidence that resolving plague changes might "light up", resulting in bacteremia and thus rendering the rats dangerous.

* In the preparation of this article I have received much assistance from Dr. Robert Pollitzer of our staff. To him I wish to accord my best thanks.

The Commissioners therefore conclude "that these chronic plague-rats, inasmuch as the bacilli are shut up in abscesses, where fleas cannot possibly get at them, are *per se* of no importance in spreading the infection" (1).

Since this statement was issued in 1908, we find again and again reference to a possible role played by chronic plague rats. To our knowledge no satisfactory evidence has been brought forward to support such claims and it may still be maintained that resolving rat-plague is a sign of past infection rather than an active factor in the propagation of the disease. On the other hand, it is probable that sporadic cases of acute rat-plague are not the only ones which keep the virus alive during the off-seasons. Recent investigations, especially by Williams (3), suggest that 'carriers' of plague exist among the rats which, though apparently surviving for a prolonged period, display bacteremia and may therefore spread infection through their fleas. A further possibility is that rat-fleas, which under suitable conditions are apt to remain alive and infective for a considerable time, may help to preserve the virus.

Turning now to the wild rodents, we find that more or less satisfactory evidence is forthcoming for a few species only. Among those hitherto investigated, blood-sucking ectoparasites, specially fleas, have been found to be the principal, if not the only, means of transmitting plague. Therefore, animals with bacteremia are an essential link in the propagation of this infection.

B. THE GROUND SQUIRREL OF CALIFORNIA (CITELLUS BEECHYI).

Two factors favouring the spread of plague among ground squirrels are :

(*a*) Absence of a seasonal prevalence, heavy infection being noted in winter as well as in summer (4);

(*b*) Restriction of aestivation or hibernation to adult animals principally. Though this tendency to aestivation or hibernation (or probably a combination of both) is noted in ground squirrels, adult animals are mostly affected (5). The young animals, suspected by Harrison (6) to be most susceptible to acute plague, keep awake throughout the year.

An important means of staying the spread of plague among squirrels is the immunity usually developing in regions where the disease has prevailed for several years. McCoy (7), in an early contribution on this subject, says that this evolution "may mean a gradual extinction of the disease or it may indicate that this partially resistant race of rodents will, if not vigorously attacked, perpetuate the disease for many years." Now there is no doubt that the infection tends to perpetuate rather than disappear. The immunity prevailing in endemic localities is not absolute as has been shown by the instructive experiments of McCoy (4) upon squirrels from plague-affected and plague-free zones respectively :

		Died.		*Killed.*	
	Total used.	Acute plague.	Subacute plague.	Residual bubo.	No lesions.
Squirrels from plague zone	16	1	3	3	9
Squirrels from plague-free zone	19	8	9	—	2

It can thus be seen that subacute and even acute plague, though rare, were not entirely absent among squirrels of the first category. A similar variation in the lesions is also seen in naturally infected squirrels. Not only those with acute disease but also some of the subacutely affected animals show bacteremia and are therefore infective. Moreover, it is claimed by Rucker (8) that even in regions where the disease has existed for a few years and the majority of animals present non-acute plague, small areas will be found, e.g. isolated valleys, where the acute form preponderates. For all these reasons it is not difficult to understand why plague persists among the Californian wild rodents.

C. THE SOUTH AFRICAN WILD RODENTS.

A very rapid type of infection is noted among the wild rodents involved in South African epizootics. As far as our present knowledge goes, chronic plague does not exist among either gerbilles or multimammate mice. The evidence in regard to carriers is scanty, only one Namaqua gerbille and one Karroo rat respectively found in one and the same locality suggesting such a condition. It is evident therefore that, once plague infection is introduced in a locality, many animals will succumb quickly, little fuel remaining to keep the virus alive. A satisfactory explanation for this situation has been given by Pirie (9), who proved that plague infection could be maintained in a scanty rodent population by flea transmission. In a series of well-planned experiments he demonstrated that the fleas could carry over the infection for a period of three or perhaps four months. He reasons thus:

"The mechanism of the persistence of plague in the wild rodent reservoir is therefore not too difficult of explanation even when the rodent population is scanty. When the rodents are few in number the disease would of necessity be of a quiet, smouldering type, through frequent failure of the necessary contacts to be established, but a rodent infection every here and there at two-monthly intervals would be sufficient to keep the disease alive. As the number of rodents increase and the fleas presumably increase *pari passu*, so would contacts be more easily made and the number of cases increase.

Whether increase in numbers to a high level is in itself sufficient to bring about the recurrence of definite epizootics every 3-4 years, and still more markedly every 10-11 years, in accordance with Elton's theory, or whether some extra factor, possibly a widespread climatic one, must also come into play to allow of a flare-up from the smouldering stage to that of the raging fire must be left for future observations to settle."

D. THE SOUTH-RUSSIAN RODENTS.

As in South Africa, so in South Russia, several species are known to suffer from plague. Only a few are of fundamental epidemiological importance, namely, the small susliks (sisels) on one hand and domestic and wild mice on the other. An interesting point is that no regular transition of the infection from the susliks to the mice, or *vice-versa*, exists so that the spizootics run an independent course in each group (10).

As far as we know the problem of how plague is perpetuated among the *mice* has not been studied in detail. Chronic affections seem to be present in them (11), so it may be assumed that some immunity does develop in the course of the epizootics. Among the domestic mice 'carriers' have been observed as well. *B. pestis* growing in abundance though no macroscopic signs are noticeable (12) No doubt plague is constantly transmitted among the mice and not caused by periodical reinfections from the gnawing of human plague corpses (13). It seems safe to assume that the epizootics among these non-hibernating animals occur in the same way as among domestic rats.

The *susliks* lie in another category since prolonged hibernation is an obligatory feature among them. As in the case of mice it has been assumed in the past that plague is not constantly present in susliks, infection being contracted after the hibernation period from plague corpses. But the systematic work of Nikanoroff and his school has lately shown that such an unusual assumption is unnecessary.

Nikanoroff (10) has suggested an influence of the seasons upon experimental suslik plague :

Tests were made in this direction by infecting fortnightly from the middle of June batches of 30 susliks with cultures of the same origin and uniform virulence. Four series were made with subcutaneous injection. In the first most animals succumbed quickly to acute plague while in the other three series the disease displayed more and more a slow evolution. Thirty days after infection there still lived : in the first group 0; in the second group, 5; in the third group, 5; in the fourth group, 18.

These results have been supplemented by observations of Gaiski (14) undertaken throughout a whole year. He infected subcutaneously 242 susliks in 27 batches, using for each successive series a strain isolated from the preceding. Seasonal differences were noted in two directions :

(a) The mean length of illness varied, reaching its minimum (3 days) in June and maximum (25 days) in winter.

(b) While in June and July 100% of the animals displayed bacteremia, only 60% were so found in winter and 40% in March. The other animals suffered either from pure local plague with bacilli confined to the site of infection, or from a transitory form (bacilli in the organs but not in the blood). The incidence of local plague was highest in winter (30%).

Of special interest are Gaiski's results with hibernating animals. Of 30 such susliks :—

awoke and succumbed after 2-22 days (average 8 days)	21
were killed after 15 & 35 days after infection respect.	3
succumbed after 45-138 days	6
Total	30

Interesting details of the above experiments are :

(*a*) Of the three animals killed, two (15 and 35 days after infection respectively) were well nourished and showed plague bacilli at the site of infection but not in the blood or organs. In the third (killed after 15 days) bacteremia was present.

(*b*) Of the three animals dying after 138, 120 and 96 days respectively at the physiological end of hibernation, at least one had passed through a stage of bacteremia. At *postmortem* plague bacilli were present both in the internal organs and at the site of infection; in the other two animals only in the local abscesses.

Later researches by Golov and Joff (15) indicate that the suslik fleas have probably a considerable share in keeping the virus alive :

They prove that the feces of infected fleas may harbour plague bacilli for a considerable time (observed up to 79 days at a temperature of 7-10° C. and 92% humidity), though they were allowed to bite an infected animal only once, and afterwards healthy ones.

Infected fleas which were afterwards kept starving at temperatures corresponding to those of the burrows in winter, survived up to 206 days and yielded at death living and virulent plague bacilli. Golov and Joff also showed that suslik fleas kept in test tubes at such temperatures remain active and able to feed upon hibernating susliks. Similar observations were also made under natural conditions.

Suslik fleas can stand low temperatures (down to—25° C.) and prolonged starvation in empty burrows (up to 10 months).

It is thus seen that conditions for preserving the plague virus among the rodent population are almost ideal. On the other hand, as pointed out by Gaiski, the seasonal changes of susceptibility may protect the species from extinction through acute plague even though no specific immunity developes.

E. THE ALPINE MARMOT (A. MARMOTTA).

These rodents, though perhaps in former centuries playing some part in the spread of European plague, have now to be classed among the experimentally susceptible ones. They are however of considerable interest to us in that, beside being close relatives of the Siberian marmot, successful hibernation experiments have been performed upon them. Dujardin-Beaumetz and Mosny (16) injected *B. pestis* subcutaneously

into three European marmots. One non-hibernating animal died in 2½ days. The other two, which hibernated, succumbed after 61 and 115 days respectively, showing no local reaction or buboes, but foci of chronic pneumonia, in which plague bacilli were present in enormous numbers. These French observers consider this as sufficient proof that the tarabagan of Siberia is a "reservoir" for plague, the virus being held in abeyance during winter. Though this conclusion looks at first radical and rash, it seems to be quite justified by our latest experiments upon the true Siberian marmot.

F. THE TARABAGAN OR SIBERIAN MARMOT (A. BOBAC).

1. INTRODUCTORY REMARKS.

The Siberian marmot, like the susliks of South-Russia, undergoes prolonged hibernation. Most authors agree that plague-stricken animals do not seek shelter in the burrows but stay out and die in the fields. Such rodents have been found on the surface long after the onset of the hibernation period, having either not slept at all or got up again with the development of symptoms.

For these reasons it is doubted by several observers if the virus is permanently kept alive in the tarabagan. They contend that in autumn a thorough separation takes place between healthy animals which retire to the holes and sick ones which remain outside to die. Different theories are conceived to explain how the tarabagan population becomes again infected in spring. As in South-Russia it was thought that the animals might contract the disease by gnawing human plague corpses. This hypothesis is no only intrinsically weak but disproved by the fact that no importation of human plague takes place at all as was presupposed. It seems also unlikely that infection could be preserved in the dark and moist burrows of the tarabagan and propagated through animals searching for mates or otherwise chancing into such holes. A theory maintained by Sukneff (17) deserves attention. According to him the reservoirs of plague are not the tarabagans but certain species of small rodents. Among his contentions are :—

(*a*) the tarabagan does not suffer from chronic plague;
(*b*) Only healthy animals hibernate, whereas sick ones remain outside and die off.

For many reasons we have never agreed with Sukneff (18). Though severely sick animals may no doubt stay out, we have been able to show experimentally that those developing the disease hibernate in much the same way as healthy ones. Regarding chronic plague in tarabagans we have supplied evidence of its existence in laboratory animals. The question whether chronic plague exists in naturally infected tarabagans does not seem fully established. Some of the morbid changes seen by us in 1923 appeared suspicious in this regard but our latest investigations show that it is not always easy to definitely interpret such findings. However, as already mentioned in the foregoing pages, the presence of chronic plague is certainly not a *sine qua non* for the perpetuation of plague in

a rodent species. One is almost tempted to reason that the presence of chronically affected animals, in other words the development of some immunity in that species, complicates the question as to how the virus is kept alive. Be this as it may, it is easy to explain how plague is propagated among the tarabagans during the *warm* season without their being wiped out. For these rodents, living in families, as a rule do not stray far away from their burrow but keep within its reach so as to escape danger. It seems probable therefore that the disease among them bears usually a familial character. On the other hand, the infection certainly creeps slowly from burrow to burrow and then settlement to settlement. In this an important role is played by very sick animals which are seen to stagger about aimlessly and may shed some of their parasites on the way. The latter will seek new hosts as their old ones die off. Chance-meetings may also take place among animals of different families, e.g. during the mating season, in case of danger, etc.

2. EARLIER HIBERNATION EXPERIMENTS.

The pivotal point of our problem is therefore what happens to the virus during the long *winter*. Dujardin-Beaumetz' and Mosny's experience, suggestive as it is, cannot be taken as a valid proof that the *B. pestis* is preserved in the Siberian marmot throughout the hibernation period. Likewise our former (1922-23) winter experiments upon tarabagans (19), though showing that infected animals may continue to sleep and succumb to infection considerably later than those infected in summer, were not wholly satisfactory. For, in order to obtain as much preliminary experience on hibernation as possible, we adopted the following lines in tackling the problem :

(*a*) Different methods of infection were chosen, so that comparisons could be made among small sub-groups only;

(*b*) The animals were daily handled in order to have their temperatures taken. In all probability this often, perhaps inevitably, hastened their death.

3. PLAN FOR NEW HIBERNATION EXPERIMENTS.

To eliminate errors, we approached the hibernation problem again, and after careful consideration formulated the following plan :

(*a*) To extend our program over two winters :

i. In 1926-27 we waited for the spontaneous death of the test animals, handling them as little as possible.

ii. In the winter 1927-28 we propose to kill some of the infected animals at regular intervals in order to study in more detail the manner in which the virus is preserved.

(*b*) To infect the animals with doses of uniform size by pricking the paw, choosing the inner aspect of the left hinder leg.

(Prick in the tail would have been more satisfactory in certain respects but in that case we could not be so certain of the bubo localisation).

(*c*) To infect batches of two tarabagans fortnightly. With each batch a guinea-pig was percutaneously infected with the same material in order to confirm its nature and virulence.

(*d*) To begin the experiments with our strain "V"* and to pass it as far as possible directly from tarabagan to tarabagan. (Failing this a culture from the guinea-pig of the preceding batch was used).

(*e*) To house the infected tarabagans in the unheated outbuilding used in our former hibernation experiments.

(This plan was modified in the course of the work because a few animals seemed to die prematurely of unspecified lung or intestinal disease due perhaps to the extreme cold. Hence from the third experiment onwards only one animal of each batch was kept in the out-house, the other in the warm plague room of the laboratory. The evolution of infection did not seem to be affected by this difference in the temperature.°)

4. EXPERIMENTS IN WINTER 1926-1927.

In the winter of 1926-27 we infected altogether 9 guinea-pigs and 16 tarabagans (14 hibernating). Little need be said about the former. With one exception they all died within 3-7 days, mostly on the 5th day, of acute plague yielding typical cultures. The exception was the guinea-pig of the first batch, which succumbed to plague on the 11th day. That the initial culture was virulent is proved by the fact that one tarabagan of this batch, which did not continue to sleep, died after 6 days of acute plague. A guinea-pig infected with material from Gp. 1 also succumbed after 5 days to the disease. Hence the lengthy survival of Gp. 1 must have been due to individual resistance or some untoward incident.

Of the 14 tarabagans hibernating at the time of infection (winter 1926-27):

Number		*Condition after infection*	
Died after 2 days, no plague	1	Continued to hibernate.	
Died after 5-19 days with signs of manifest plague with bacteremia	6	Slept fairly well	1
		Slept interrupt.	4
		Awoke	1
Died after 22-60 days, no plague	3	Continued to hib.	2
		Slept interrupt.	1
Died after 28 and 48 days respect. with signs suggesting residual plague	2	Continued to hib.	
Died after 88 and 130 days resp., i.e. a few days after awakening at the normal end of hibernation with signs of local and bacteremic plague	2	See text.	
	14		

* This is a strain of tarabagan origin, which has since 1923 been repeatedly passed through laboratory animals. It has high and stable virulence.

° A table showing the temp. of the two rooms will be found in the Appendix.

One striking feature of the above summary is the great variation in the results obtained. It seems an open question if the same holds true in nature. Try as one would, one could not imitate the undisturbed quietness, low but perhaps only slight oscillating temperature, etc., which reign whenever the tarabagans sleep in their natural habitat. Hence it is more than probable that a certain percentage of our experimented animals died prematurely while others would have survived under natural conditions. Nevertheless, valuable conclusions can be safely drawn from the experiments. These results may now be discussed in detail.

a. ANIMALS SHOWING NO SIGNS OF PLAGUE AT POST MORTEM.

Of the four tarabagans composing this group one showed bronchopneumonic foci as confirmed by histological examination; two had subpleural petechiae and had presumably succumbed to a lung process; the fourth, dying two days after infection, displayed signs of an acute enteritis. In none of these animals were any macroscopic changes noted at the site of infection or in the inguinal glands. In one instance a few non-characteristic bacilli were seen in smears from the spleen, while in the pneumonic case such were present in preparations from both spleen and lung. All cultures were sterile.

It is possible that an exhausting experimental and histological examination would have yielded some traces of plague infection. Because of our plan to make a systematic search for such next winter we thought it wise not to spend too much time and energy upon chance findings. It is therefore difficult at present to draw any final conclusions in regard to this group. However, some of the hibernating tarabagans may escape infection or overcome it.

b. ANIMALS SHOWING SIGNS OF LOCAL (PROBABLY RESIDUAL) PLAGUE :

The findings in the two animals under this group point perhaps in the same direction. Their protocols are :

Tb. 3/a, infected Jan. 3, 1927, died Jan. 31, i.e. after 28 days, having hibernated well throughout.

P.M. Big well nourished animal.

No changes visible at site of infection. In the left inguinal region one gland hyperemic and increased to size of half-pea, another of about same size filled with caseous matter.

Lungs pink; nothing abnormal. Liver somewhat enlarged, darkbrown.

Spleen slightly enlarged, but firm. Bladder full (no pelvic bubo).

Bacteriological examination.

In smears from the inguinal glands a few suspicious bacteria. Smears from the heart-blood show a few faint bipolar-stained bacilli; from spleen doubtful. Cultures from internal organs negative. Gp. infected percutaneously with material from the glands survived.

*Histological examination.**

i. Caseating gland. Capsule much thickened with leucocytic infiltration. Extensive destruction of lymphatic tissue, only remnants of which are to be seen among necrotic masses. Bacteriological diagnosis difficult owing to presence of an enormous mass of granular debris; no definite bacilli seen, perhaps involution forms present.

ii. Hyperemic gland. Capsule thickened and infiltrated. Lymphatic tissue much engorged, numerous deposits of a brownish pigment. Single bipolar-stained bacilli in the medulla and near hilus.

Tb. 5/a, infected Feb. 1, 1927, died March 21, i.e. after 48 days, having hibernated well throughout.

P.M. Large congested area at site of infection. Inguinal glands on left side somewhat enlarged, but not congested. Lungs pink with small hyperemic areas on surface. Spleen not enlarged but slightly softer than normal. Bladder as in Tb. 3/a.

Bacteriological examination.

Cultures from internal organs sterile. Gp. infected subcutaneously with material from the site of infection survived.

Histological examination.

i. Area of local reaction. No marked congestion. In the subcutaneous tissue hemorrhages of varying extent noted with slight leucocytic infiltration at places. The latter under high power showed scanty bipolar-stained bacilli.

ii. Inguinal glands. No marked changes; if anything the trabeculae somewhat enlarged. Capsule of gland is undoubtedly thickened and shows cell proliferation but no leucocytic infiltration.

iii. Lung. At places groups of alveoli filled with a cellular exudate, mostly erythrocytes (red hepatisation). No plague bacilli or other microorganisms.

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* For our histological examinations we used, besides Hemalum-Eosin, mostly Kossel's method of staining:

10 c.c. conc. aq. sol. Meth. Blue (Hoechst) is diluted in 100 c.c. distilled water.

To this add 30 drops of 5% sol. of Sod. Carbonate (cryst.). 6 c.c. of 1% aqueous Eosin A extra (Hoechst) is now gently added while shaking, so as to prevent formation of sediment. Solution must be prepared and filtered immediately before use.

Stain sections in this solution for 10-30 m. Differentiate in dil. acetic acid (1 drop glacial in Petri dishful of dist. water) until pink colour appears in parts. Pass through 95, then absolute alcohol, until no more blue comes out. Then pass through Xylol and mount in cedar wood oil.

While there is little doubt that a few plague bacilli still persisted in these tarabagans (one at the site of infection and the other in a regionary lymph-gland) it is difficult to ascertain their true significance. We shall later bring evidence to show that plague bacilli introduced during the hibernation period may remain at the site of infection, cause certain changes there, and finally lead to general infection when the animal awakes. Naturally the question arises whether the findings in the two tarabagans above do not constitute such local deposits of bacteria which would have led to manifest plague had the animals survived until spring. This possibility will have to be affirmed or negatived by our investigations next winter. Up to the present we are inclined to believe that the changes seen in tarabagan 3/a and 5/a are the result rather of a successful struggle with the invaders, and that the few bacilli still remaining would have eventually disappeared had the animals not died prematurely.

c. ANIMALS DYING OF MANIFEST PLAGUE DURING THE HIBERNATION PERIOD.

Only one of the six animals of this group continued to hibernate fairly well after infection; four slept interruptedly, while the fifth was up throughout the six days of its illness. These observations are not easy to explain. Though there is undoubtedly much reason in the contention of authors like Gaiski that infected hibernating rodents wake up because symptoms of plague develop in them, we doubt if this always holds true. Possibly in some instances the disease runs a quick course because the animals are disturbed on account of the artificial conditions in which they are kept.

THE FOLLOWING TABLE SHOWS THE MACROSCOPIC FINDINGS OF THIS GROUP.

Tb. No.	Died days after inf.	Local reaction.	Bubo.	Liver.	Spleen.	Lungs.	Other organs.
1/a	6	None	None	Fat-infiltration. Subcapsular hemorrhages	Sl. enlarged & softer.	No marked changes.	Hemorrhages in mesenterium Serosa intestine congest.
4/b	12	Marked Suppuration at places.	Size bigger windsor-bean. Caseous matter on section at places.	Enlarged, congested.	Swollen & soft. Indistinct nodes.	Subpleural petechiae.	Kidneys congested.
5/b	10	Abscess size half-pea.	Size over windsor-bean. Some suppuration.	Small nodes & hemorrhages size lentil all over.	Much enlarged. Nodes up to size lentil.	Some areas of congestion.	Omentum adherent to spleen, congested. Stomach mucosa congested. Small intest at places congest. Mesenterial glds. enlarged and congested.
6/a	5	Hemorrhages and indistinct infiltration.	Small. Inguinal glds. rt. side enlarged and congested.	Congested, hemorrhages below caps.	Not markedly changed.	No marked changes.	Hemorrhages in omentum.
6/b	5	Infiltration size two peas.	Size two peas. Marked periadenitis with hemorrhages.	Fat-infiltration. Subcaps. hemorrhages.	Much enlarg., sl. softer.	Pale. Fine subpleural hemorrhages.	Tubes and ovaries congested.
7/b	19	Infiltration size two peas.	Size windsor-bean. Oedema of abdominal subcutis.	No marked changes.	No marked changes.	Pale.	Hemorrhages omentum and mesenterium.

N.B.—Diagnosis in case 6/b confirmed by smears and cultures; in all other instances also by experiment.

I proceed now to a detailed description of the more important changes :

i. *Local reaction and bubo.*

Even the few tarabagans in this series seem to fall into different groups. On one hand we had a case of purely 'septicemic' plague without local changes (Tb. 1/a); on the other, animals with a somewhat prolonged course of the disease and subacute changes characterised by the presence of suppuration (Tbs. 4/b & 5/b).

Tb. 7/b, succumbing on the 19th day after infection, though belonging to this group, did not show such marked gross changes. It resembled macroscopically the animals 6/a and 6/b which displayed signs of acute plague, thus standing between the two just mentioned groups.

With exception of Tb. 1/a (which was not investigated in this respect), positive *bacteriological* results were obtained in each instance from the site of infection and bubo.

The macroscopic findings were generally confirmed by *histological* examination :

Tbs. 6/a and 6/b show very acute changes at the site of infection and in the bubo. At the former leucocytic infiltration and hemorrhages are noted in addition to much congestion. Plague bacilli are present in enormous numbers, often forming large clusters and bizarre nets in the areas of infiltration and hemorrhage. The affected lymph-glands show in both cases severe congestion and hemorrhage in the adenoid tissue (in case 6/b also early caseation); *B. pestis* are very numerous, again often in big clusters. The capsule of the glands appears at places thickened and infiltrated with leucocytes. Periadenitis, which was not well marked macroscopically, is made evident by the microscope, being characterised by much congestion, leucocytic infiltration and even hemorrhage at places.

The local changes in animals 4/b and 5/b are different from those just described. Hemorrhage is absent and instead of the more diffuse infiltration with leucocytes one sees more or less well-defined agglomerations of such. In Tb. 4/b some reaction seems present in the surrounding connective tissue, where at places marked congestion and cell proliferation are noted. Plague bacilli are pretty numerous, often forming clusters in the abscess-like formations. In the buboes there is no hemorrhage and less marked congestion than in the foregoing cases. Smaller and larger caseating areas are present. Plague bacilli are quite plentiful in bubo 5/b, were they are mainly arranged in clusters; in Tb. 4/b they do not appear so numerous and involution forms are met with. Periadenitis is quite marked in this case though no hemorrhage is noted; the capsule of the gland is apparently involved. In animal 5/b changes round the gland are not conspicuous; some alteration of the capsule is noted.

That the gross appearances in Tb. 7/b were not so typical is explained by the fact that in this case hemorrhages and some diffuse leucocytic infiltration are present at the site of infection. But here also one notes under the microscope abscess-like formations of leucocytes. Again reaction on the part of the connective tissue has taken place; it

would seem that the latter tries at places to encapsulate the abscess-like formations or to penetrate into them. Plague bacilli are quite numerous but not often arranged in clusters. In the bubo there is marked congestion, and hemorrhages are noted at places in addition to some caseation. Plague bacilli are numerous, occasionally in groups, but on the whole do not seem so plentiful as in the acute cases. The capsule is moderately thickened with some leucocytic infiltration and cell proliferation. The tissues round the gland are congested and show leucocytic infiltration which is perhaps not so marked as in the animals succumbing quickly.

ii. *Liver and spleen.*

In every animal of this group positive bacteriological findings were obtained from liver and spleen. The morbid changes correspond in general to those observed in bacteremic diseases, especially plague; sometimes the absence of marked lesions was conspicuous (see tabulation). Only two cases with peculiar features deserve special discussion :

(*a*) *Tb. 4/b.* The spleen of this animal showed indistinct nodes besides much acute swelling. Histological examination confirmed the presence of large and small areas where lymphocytes and leucocytes, at places mixed with red blood corpuscles, are embedded in uniformly contrast-stained, necrotic tissue. Plague bacilli (usually single) are fairly numerous within such areas. At other parts, especially the periphery of the necrotic nodes, they are seen in enormous numbers, forming clusters and nets. Even within the Malpighian bodies some *B. pestis* are met with, especially at spots where a little hemorrhage seems to have taken place.

(*b*) *Tb. 5/b.* Here marked appearances of "nodose" plague were noted in both liver and spleen. Histological investigation reveals severe alterations in the former consisting of marked congestion and infiltration of the liver cells with fat globules; at places more or less extensive hemorrhage is seen, while at others the liver tissue seems more or less destroyed, leucocytes alone or mixed with red corpuscles abounding in the damaged tissue. Plague bacilli occur in moderate clusters, being often situated at the periphery of the necrotic areas. Similar but larger nodes are encountered in the spleen, bacilli in groups occurring near their circumference.

Tarabagans suffering from such "nodose" plague have been recorded in the past. Thus :

(*a*) A few animals, including that shot in the fields by Barykin in the year 1907 (20) showed small greyish nodes in the spleen;

(*b*) One naturally infected animal found by Sukneff in 1923 had some bulging nodes in the lungs, many nodules and hemorrhagic spots in the liver; the spleen of this animal was partly eaten by eagles so that its nature could not be ascertained (19). Histologically the condition in the liver, though further advanced, was similar to that observed in Tb. 5/b (21);

(c) One tarabagan infected conjunctivally in the course of our former winter experiments (1922-23) and succumbing on the 17th day after infection showed numerous pin-head nodules of yellowish-white colour in the liver and larger white nodes (size lentil) in the spleen.

Cultures from the cases b and c were somewhat impaired in virulence. For this and other reasons we were inclined to consider such nodose changes as the result of a subacute or even chronic stage of the disease. As shown by our recent experience, such alterations may develop comparatively quickly, so that one must be chary of hasty conclusions. We believe that in the tarabagan as in other rodents such forms as acute, subacute and chronic ought not to be separated by any sharp arbitrary line. In all probability transitory forms between these also exist.

d. ANIMALS SUCCUMBING TO PLAGUE AFTER AWAKENING FROM HIBERNATION.

The two animals belonging to this group may now be described:

i. Tb. 3/b, infected on January 3, 1927, was up to Jan. 10 kept in the outhouse, later in the laboratory stable. Slept well with short interruptions up to March 8. From then onwards it was mostly in a drowsy condition, though eating a little food at times. When stirred, it did not bark, was not shy of human beings, and generally remained listless. When occasionally taken out of the cage it did not resist or attempt to run away; sometimes the hinder legs looked paralysed. On the whole it gave the impression of being affected by a chronic disease (plague?) rather than in a state of hibernation. That the latter condition prevailed, became evident early in May (2nd) when the animal began to react better, displaying its teeth when approached, but not barking. A week later (May 9) it was wide-awake, greedily feeding when a carrot was thrown in. This condition remained the same up to the morning of May 12, when it was last seen alive. Next morning (May 13) it was found dead, i.e. 130 days after infection.

P.M. Little fat, though not emaciated.

No marked local reaction. Small but markedly congested bubo in left inguinal region; some hemorrhages in the fascia nearby. Right inguinal glands slightly enlarged and congested; cervical glands somewhat congested but not enlarged.

Lungs oedematous; right shows large areas of congestion.

Numerous petechiae on epicardium.

Liver not enlarged, brownish-yellow in colour; some perihepatitis in form of white linear thickening of the tissue.

Spleen softer than normal though not enlarged; congestion at places.

Retroperitoneal hemorrhages.

No other conspicuous changes.

Bacteriological examination.

Smears from bubo, heart, lung and spleen positive. Cultures from bubo no growth; from heart, lung, liver and spleen positive though somewhat contaminated. Two guinea-pigs infected immedately at *postmortem* succumbed to plague. The first, which was pricked with a needle dipped into the bubo, died on the third day, while the second, rubbed into the shaved skin with material from bubo, heart and lung, succumbed on the fourth.

Histological examination.

Sections from the *bubo* show, as far as the gland tissue is concerned appearances similar to the acute cases. Severe hemorrhage is present, leading at places to a disintegration of the structure. Plague bacilli are very numerous, often arranged in big clusters at the periphery of the gland. The capsule, however, shows at places marked thickening and cell proliferation, but leucocytic infiltration is absent. The tissues near the bubo are much congested; hemorrhages are occasionally met with but no leucocytic infiltration.

A slightly enlarged *lymph-gland from the right groin* shows much congestion but no hemorrhage. Plague bacilli are numerous in the larger blood vessels, but scanty outside them. The capsule is not perceptibly changed. The surrounding connective tissue is less congested than in the case of the bubo; no leucocytic infiltration could be seen, only small hemorrhages.

The *liver* shows at places thickening of the capsule. Congestion and parenchymatous degeneration are present. Fairly numerous plague bacilli are noted in both vessels and capillaries, sometimes in small clusters.

The *spleen* is much congested. The Malpighian bodies seem comparatively small, the trabeculae prominent. Plague bacilli occur in large numbers but are more evenly distributed than in the foregoing cases, so that no big clusters are met with.

The *lungs* are congested and show foci of broncho-pneumonia; the exudate is mostly cellular, red blood corpuscles being more numerous than white ones. *B. pestis* are plentiful, rarely in groups.

The *kidneys* show congestion and parenchymatous degeneration, plague bacilli being noted within the vessels only.

In the *retroperitoneal tissue* one sees large hemorrhages; here the bacilli are mostly grouped together in small clusters or loose nets.

ii. Tb. 4/a was infected on January 17, 1927 and kept throughout in the outhouse. Slept well with almost no interruption up to the beginning of April. Being occasionally up from April 4 the animal was wide-awake on April 11 : on this day it was seen to sit on the straw in its cage but to hide itself immediately when approached. Was then well and feeding up to April 13 when seen for the last time before death. Found dead on the morning of April 15, i.e. 88 days after infection.

P.M. Medium-sized animal, still moderately fat.

Some reaction is noted at the site of infection (superficial layer of musculature) where congestion and perhaps some infiltration are present. Left inguinal glands slightly enlarged but not markedly congested.

Lungs are oedematous, anterior parts pale; areas of congestion in dorsal parts of both lower lobes.

Liver is congested and shows some indistinct subcapsular hemorrhages. Some perihepatitis over left lobe in form of a net of white, thickened tissue.

Spleen not markedly changed.

Stomach shows petechiae below mucosa, is full of bile-stained liquid. Duodenum and upper part of jejunum much congested, their contents bloody. Subserous hemorrhages on duodenum.

Bacteriological examination.

Smears from spleen show numerous bipolarstained, gram-negative bacilli; from lung somewhat atypical, gram-negative bacilli. In films from bloody intestinal contents, bacilli similar to *B. coli* besides larger gram-positive bacilli.

Cultures from heart, liver, spleen are typically positive, those from the lung somewhat atypical but suspicious. Culture from intestine is negative for *B. pestis*.

Altogether five guinea-pigs were inoculated. One, receiving a dose of culture from the intestine into the shaved skin, survived; the others all succumbed to plague, thus :

(*a*) Pricked with material from the site of infection, died on 3rd day;
(*b*) Rubbed with material from lung and intestine, died on 7th day;
(*c*) Rubbed with liver culture .....died on 7th day;
(*d*) Rubbed with lung culture .....died on 9th day.

Histological examination.

After prolonged search a small abscess is found at the *site of infection*. The cells at the periphery stain fairly well; those in the center have undergone necrotic changes, so that a diffusely stained mass is present showing at places indistinct nuclei. The connective tissue near the abscess is markedly changed; rich in cells and blood vessels it has the aspect and arrangement of granulation tissue. Small hemorrhages are seen at places but no agglomerations of leucocytes. No bacilli may be distinguished in the center of the abscess, though they are fairly numerous at the margin, occurring both in typical and involution forms, the former occasionally in clusters. Round the abscess plague bacilli are plentiful in the vessels, but scarce in the tissue, with the exception of the hemorrhagic areas; even here no large clusters of bacteria are seen.

One enlarged *left inguinal gland* shows under the microscope some congestion and small hemorrhages. The connective tissue seems increased in volume at the cost of the parenchyma. The arrangement of the bacilli resembles that in the connective tissue of the site of infection. The capsule of the gland is thickened with cell proliferation at places. Some congestion exists around the gland but no marked periadenitis.

The capsule of the *liver* is thickened; cell proliferation is not noticeable. Marked congestion is present throughout the organ with occasional hemorrhages. The liver cells show signs of fatty degeneration. Plague bacilli are plentiful in vessels and capillaries; at and near the hemorrhages they also occur outside the vessels, now and then forming small clusters.

The *spleen* is very rich in blood. Capsule is somewhat thickened and shows signs of cell proliferation. Smallness of the Malpighian bodies and prominence of the trabeculae may be noted but this is not so conspicuous as in the foregoing case. Plague bacilli are very numerous though big clusters are absent.

The *lungs* show general congestion and foci of broncho-pneumonia especially near the surface. Here the alveoli are filled with a serous, less often a cellular, exudate: in the latter red blood corpuscles prevail. Bacilli are moderately numerous in the foci, often in small groups; in other parts of the lung they are present in the vessels and capillaries only.

The *kidneys* are congested. Parenchymatous degeneration is present. Bacilli are quite numerous in vessels and capillaries.

Sections from the *stomach* show extensive submucous hemorrhage containing bacilli in groups. The wall of the *small intestine* is much congested with hemorrhages at places extending to the surface of the mucosa. Big clusters of bacilli occur in and around the hemorrhages.

It can thus be seen that two of our 14 tarabagans, infected while sleeping, continued to hibernate. They succumbed to plague with bacteremia in the spring after they had been up and apparently well for a few days; in one of these two the hibernation period appeared prolonged.*

The bacteria present at the time of death in the blood and organs were in one animal (Tb. 4/a) but little impaired in virulence if at all; in the other they may almost be said to have increased in virulence.

The question as to where the plague organisms are preserved in such animals until the disease becomes manifest, is not yet fully established. There is little doubt that in Tb. 4/a they remained at the site of infection. In Tb. 3/b, however, no local reaction could be detected and, though some older change may be present in the inguinal glands, we cannot affirm if the bacilli reached them at an early stage of the infection. Some chronic lesions were noticed in liver and spleen of both animals, but it is difficult to gauge their significance Possibly they were caused by toxins circulating in the blood. We hope to elucidate all these questions during the next winter.

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* It is hardly necessary to mention that an accidental infection of the animals during their observation is out of question. Our tarabagans, kept at Harbin since summer, 1926, were free from parasites. No experiments with rats or other flea-bearing animals were performed throughout the whole time. The few infected guinea-pigs in the laboratory stable were placed in buckets away from the tarabagans. The latter were kept in individual cages and every possible precaution was taken against any unlikely contingency of infection through food and the like.

Fig. 2. Section of inoculated site, Tarab 4b, showing abscess-like agglomeration of leucocytes, haemorrhages being absent. High magnif.

旱獺乙四號示接種部膿瘍樣之白血球團聚未見出血强擴大

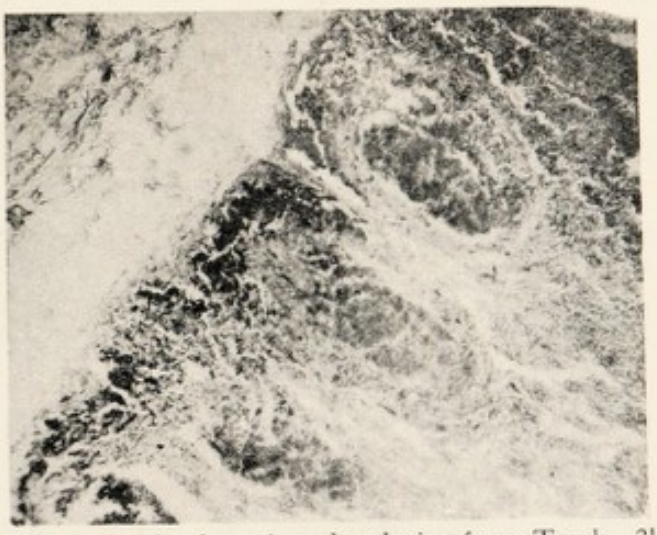

Fig. 3. Section of lymph gland on inoculated site from Torab. 3b, showing gland tissue to have undergone changes similar to acute cases (severe haem., etc.) Capsule at places shows marked thickening and cell prolif. but no leucocytic infiltration.

旱獺乙三號示接種部淋巴腺腫之組織變化似屬急性例（如廣出血等）夾膜部示著明的厚及細胞新生但未見白血球浸潤弱擴大

Fig 4. Section from liver, Tarab 11 (1928 series) showing *nodose plague*. Liver nodules are surrounded by thick layer of conn. tissue. Low magnif.

旱獺十一號（一九二八年組內）示肝臟結節鼠疫肝結節被厚層結締織圍繞弱擴大

Fig. 5. Section of site of inoculation, Tarab. 6a, showing diffuse leucocytic infiltration and haemorrhage in addition to much congestion. Low magnif.

旱獺甲六號示接種部蔓延性白血球浸潤
及出血且有多處充血弱擴大

Fig. 6. Section of site of inoculation, Tarab. 4a, showing old abscess with necrotic changes in the center. Low magnif.

旱獺甲四號示接種部慢性膿瘍中央部見壞死病變弱擴大

Fig. 7. Section of regional bubo from Tarab. 7b, showing marked congestion and haemorrhage and numerous plague bacilli, often in clusters. High magnif.

旱獺乙七號示接種部橫痃著充血及出血
又腐桿菌甚富且常團結成羣强擴大

G. SUMMARY AND CONCLUSIONS.

Evaluating the results of our latest investigations, we must admit that the two animals surviving up to spring had contracted infection *during and not before* the onset of hibernation, while all the twelve others died during winter, six of acute and subacute plague. As stated above, conditions are certainly much more favourable in nature than under artificial laboratory conditions. It seems therefore improbable that the percentage of rapidly evolving plague cases can be as high as we have witnessed in our experiments. Also it is probable that in some of the naturally infected animals plague does not develop at all or remains localised, resulting in recovery. Be this as it may, there is little doubt that the tarabagan fleas, like those of the susliks, play an important part in spreading the disease in winter as well as in summer and are able to preserve the virus especially during the cold season for lengthy periods. Supplementing the knowledge attained by our experiments with the above considerations we can see how plague is propagated among the tarabagans from year to year.

Summarising our knowledge as obtained from the tarabagan together with that of wild rodents elsewhere, and comparing the results with facts established in regard to the domestic rats, we may draw the following conclusions :

(1) The occurrence of rodent plague with bacteremia is a *sine qua non* for the propagation of the disease in the wild as well as in the domestic species.

(2) Cases with chronic plague (in the strict sense) therefore do not play any important role in the preservation of the virus.

(3) Besides cases of acute and subacute plague, carriers with bacteremia may intervene; their significance is, however, not yet fully established.

(4) The hibernation period to which some of the wild rodents suffering from natural plague are subjected, is not a hindrance to the perpetuation of the disease, but on the contrary an indispensable link for the preservation of both the virus and the species.

(5) In order not to confound the issues, no mention has been made in the text regarding migration. However, this may be important in two ways :

   a. *Immigration* of healthy animals into an infected locality;
   b. *Emigration* of infected animals or carriers of infected fleas into a healthy region.

It is evident that in the first instance an impetus would be given to any enzootic present, while in the second case ample fuel would be provided for the virus regardless of its fate at the place of origin.

H. APPENDIX.

I. TABULATION OF TEMPERATURES.

(*a*) in the unheated out-house, (*b*) in the basement of the laboratory, where the animals of the series (winter 1926-1927) were kept.

Date	*Temperature in centigrade*		*Date*	*Temperature in centigrade*	
	(*a*)	(*b*)		(*a*)	(*b*)
Dec. 2	— 7	—	Feb. 19	—10	—
,, 3	— 7	—	,, 21	— 9	—
,, 6	— 9	—	,, 22	— 9	—
,, 8	—10	—	,, 24	—10	11
,, 9	—12	—	,, 26	— 9 *Mean* :	— *Mean* :
,, 11	—12	—	,, 28	—10—11.25	10+10.9
,, 13	—12	—			
,, 15	—13	—	Mar. 4	— 9	12
,, 18	—13	—	,, 5	—	10
,, 20	—12	—	,, 7	— 8	13
,, 22	—11	—	,, 9	— 8	16
,, 24	—12.5	—	,, 11	— 7	—
,, 26	—13	—	,, 14	— 7	11
,, 28	—14 *Mean* :	—	,, 16	— 8	10
,, 30	—12—11.3	—	,, 18	— 6	11
			,, 19	— 6	—
Jan. 2	—10	—	,, 21	— 4	—
,, 3	— 9	—	,, 23	— 3	11
,, 4	— 8	—	,, 25	— 1	12
,, 6	— 8	—	,, 28	1 *Mean* :	12 *Mean* :
,, 8	— 6	—	,, 30	4—4.8	—+11.8
,, 10	— 8	—			
,, 12	— 9	—	Apr. 1	4	—
,, 14	—12	—	,, 4	2	12.5
,, 17	—16	—	,, 6	2 *Mean* :	—
,, 19	—16	—	,, 13	8+4.0	11.5
,, 21	—17	—	,, 19		13
,, 24	—17	—	,, 20		11
,, 26	—16	—	,, 21		15
,, 28	—16 *Mean* :	—	,, 22		12
,, 31	—15—12.5	12	,, 23		14
			,, 25		12
Feb. 3	—14	—	,, 28		13.5
,, 5	—13	11	,, 29		15 *Mean* :
,, 7	—13	12	,, 30		15+13.1
,, 8	—	10.5			
,, 9	—14	—	May 3		12
,, 10	—	11	,, 4		13
,, 11	—13	—	,, 5		15
,, 14	—11	11	,, 9		14 *Mean* :
,, 18	—	10.5	,, 11		14+13.6

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THE PERPETUATION OF PLAGUE AMONG WILD RODENTS, WITH SPECIAL REFERENCE TO THE SIBERIAN MARMOT.

(SECOND COMMUNICATION).

(*This article is being published for the first time*).

TABLE OF CONTENTS.

A. INTRODUCTION.

In a previous paper (The Perpetuation of Plague among Wild Rodents) we described the first stages of a new series of plague experiments upon hibernating tarabagans commencing 1926-7. Of the 14 animals then infected six succumbed more or less quickly to manifest plague. Four demonstrated at autopsy no trace of infection; these were however, not exhaustively examined. Two of our test animals showed postmortem lesions suggestive of residual plague, while the remaining two, succumbing 88 and 130 days respectively after inoculation, i.e. a few days after awakening at the normal end of hibernation, displayed signs of generalised plague together with older changes at (a) the inoculated site and (b) the regional lymph glands.

Satisfactory as this evidence appeared, we had realised from the beginning that a second set of observations would be desirable in which some at least of the test animals should be killed at varying intervals so as to ascertain from another viewpoint how the virus is preserved. We have now to report upon this second series of experiments.

During the winter of 1927-28 we inoculated altogether twenty tarabagans, using the same technique and the same strain as in the year before. Certain animals succumbed spontaneously to plague or died of intercurrent disease (with some evidence of plague). The rest were killed at various intervals after infection. Our results are summarised as follows :—

TABLE NO. I.

P.M. findings :	Died spontaneously :	Were killed :	Total :	Remarks :
Early signs of plague infection in addition to unspecific pneumonia.	Tb. 21.	—	1	Died 3 days after inoculation.
Manifest plague with bacteremia.	Tbs. 12, 13, 14, 20, 22, 23, 27, 28.	—	8	Died 5-8 days after inoculation.
Residual plague.	—	Tbs. 11, 29.	2	Killed after 21 and 118 days respectively.
Latent plague.	Tbs. 17, 24.	Tbs. 15, 16, 18, 19, 25, 26, 30.	9	Tbs. 17 and 24 died after 12 and 14 days respectively, while the others were killed 16-40 days after inoculation
			Grand Total..... 20	

The above animals fall under 4 groups, which may now be discussed *seriatim*.

B. TB. 21, DYING AT AN EARLY STAGE OF THE DISEASE.

This animal was infected on January 21, 1928, together with three other tarabagans which all succumbed within 5 days to acute generalised plague. Tb. 21, which slept well at the time of infection was found dead on the morning of January 24. Post mortem findings were as follows :

Fairly big, female animal.

At the site of infection a large area of reaction in which hemorrhagic districts were interspersed with non-hemorrhagic ones; small abscesses seemed present in the latter. Microscopically one noted in the connective tissue between the muscle bundles as well as in the surrounding fascia hemorrhages and agglomeration of leucocytes which at places became so dense as to form small abscesses. Many gram-negative plague-like bacilli were seen within the abscesses, when they often formed clusters and small nets; round the abscesses bacilli were generally scanty except at some spots containing dense leucocytic infiltrations.

One of the regional inguinal glands was enlarged and hyperemic. Microscopically its capsule showed thickening and perhaps some cell proliferation in parts. Varying hemorrhages were noted within the gland while the surrounding tissues were congested. Only a few gram-negative, plague-like bacilli were noted within the gland.

The liver was markedly congested at autopsy; one indistinct node was noted. Microscopically distinct congestion was found together with numerous hemorrhages. In addition leucocytic agglomerations (both large

and small) were met with. The former, either round or irregular in shape, contained a few areas with scattered leucocytic infiltration, the cells being evidently embedded in a necrotic ground substance. The leucocytes composing the larger nodes were partly eosinophil. [1]). Some atypical, though suspicious gram-negative bacilli, were noted in the leucocytic infiltrations; away from these there were less.

The spleen apparently not much changed at post mortem was histologically found to be moderately rich in blood with fairly large follicles and prominent trabeculae. In Kossel-and gramstained specimens a few bacilli similar to those in the liver were found.

The lungs appeared very much congested. The microscope revealed also the presence of foci of confluent broncho-pneumonia with a cellular exudate in which red blood corpuscles greatly outnumbered the white ones. Very few gram-negative suspicious bacilli were seen in the peribronchial glands and alveolar walls, while occasionally small groups of gram-positive cocci similar to staphylococci were encountered in and near the pneumonic areas.

The kidneys presented no unusual macroscopic features. Histologically moderate congestion was noted in addition to some parenchymatous degeneration. Numerous small hemorrhages were seen in the medulla. Very few gram-negative, suspicious bacilli were found in and near these hemorrhages as well as in the glomerular capillaries.

We have no doubt that Tb. 21 died of acute pneumonia. Similar lesions have been noted by us during the past years among some of our infected as well healthy tarabagans, which succumbed during hibernation. It seems that such animals—though resistant in other respects—are specially liable to contract acute lung inflammation upon the slightest exposure. This is not surprising if we consider how well these marmots, when hibernating under natural conditions, are protected against any abrupt oscillations of temperature.

Some colonies of *staphylococcus albus* were cultivated from the heart, liver and spleen, but none from the lung. Plague bacilli could not be demonstrated with certainty in the cultures which, however, were taken from the internal organs only. That the animal was in an early stage of plague infection is amply proved by the local findings. In the bubo and some of the internal organs, notably the kidneys, changes were seen somewhat characteristic of this disease. Though a manifest bacteremia was absent, a few plague-like germs were found everywhere. It is difficult to decide whether these were the first harbingers of a general fatal infection or whether the animal, had it survived, would have kept the invaders at bay.

C. TARABAGANS SUCCUMBING QUICKLY TO ACUTE PLAGUE.

In addition to complete autopsies and cultural tests in this group proving that all eight animals had succumbed to bubonic plague with secondary septicemia, we resorted to histological examinations as well.

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[1] As will be discussed later on, these large nodes are in all probability not due to plague infection but to a parasitic invasion of the liver.

Our main object in doing so was to prove to our own satisfaction that our methods of fixing, embedding, cutting and staining the material would not hinder the demonstration of the *B. pestis* in the tissues. The results seen in these manifestly infected animals thus served as controls for the tarabagans of the other groups which were investigated simultaneously. There was *a priori* little hope that the histological examination might materially supplement the knowledge gathered in 1926-7 upon the animals succumbing to manifest plague. For all the tarabagans under discussion at present died rather quickly (within 5-8 days after infection) On the other hand, perhaps due to the unusually mild winter with intervening warm spells, many of the animals were likely to show more advanced changes than the corresponding ones which succumbed quickly in 1926-7.

We proceed now to a more detailed description :

1. Local reaction.

All eight animals showed a well-marked local reaction, more or less extended suppuration being macroscopically visible in a majority of the cases. Histologically more or less well defined abscess-like agglomerations of leucocytes were found in every case. In three animals (Tbs. 14, 23 and 27) these abscesses were not prominent as compared with the diffuse leucocytic infiltration around them, while in two (Tbs. 13 & 28) such diffuse infiltration was well marked in addition to the presence of clearly defined abscesses. In the three remaining tarabagans (Nos. 12, 20, 22) only slight or spot-like infiltration was found in the vicinity of the abscesses. More or less marked congestion of the surrounding tissues was the rule while hemorrhages, situated either around the abscesses or in addition some distance from them, were noted in practically all cases. Reparatory changes, as noted in some of the 1926-27 cases, were not evident.

Plague bacilli were in most cases numerous in the abscesses, but as a rule not uniformly distributed, being usually grouped together at the periphery of the leucocytic masses while scanty or even almost absent in the center. Round the abscesses such a clustering of the bacilli was the rule. Here they were most numerous within the diffuse leucocytic infiltrations, sometimes near the abscesses or within hemorrhages or occasionally near blood vessels (? in lymph-spaces). Within the blood vessels they were plentiful in two cases only (Tb. 12 & 13), moderately numerous in Nos. 14 & 20, while in the four other animals they were either scattered or practically absent.

2. Bubo.

The presence of a left inguinal bubo was in each instance proved by macroscopical examination. The glands in question, though as a rule but slightly enlarged, were more or less congested. Periadenitic changes, the presence of which was constantly seen under the microscope, were usually noticeable with the naked eye. In the bubo of Tb. 28 a caseating center was noticed at autopsy.

By an unfortunate mistake in the case of Tb. 23 a second piece of the local reaction was preserved instead of the bubo. Thus only seven of the buboes could be subjected to a histological examination. This showed either marked congestion or more or less advanced hemorrhage in the glands. Caseation was found in half the cases; this was conspicious in Tbs. 22 & 28. The capsule was usually involved, being at places thickened and infiltrated with leucocytes; in Tb. 14, which succumbed 8 days after infection, some cell proliferation was present as well. The changes round the glands usually consisted of hyperemia and spotwise leucocytic infiltration; sometimes hemorrhages were noted.

Plague bacilli were always plentiful within the buboes, often evenly distributed. Round the glands they were concentrated at places only, chiefly in the leucocytic infiltrations. Within the blood vessels bacilli abounded in three instances (Tbs. 13, 14 & 22), while in the others they were more or less scanty.

3. Liver.

The liver was in the majority of cases dark in colour; three showed a much lighter, yellowish hue. In Tb. 12 subcapsular hemorrhages could be noted, while Tb. 27 showed irregular, dark spots suspicious of hemorrhage throughout the organ. Tb. 28 had at places thickening of the liver capsule as confirmed by microscopical examination; no marked proliferation of cells could be noted.

Histological examination was undertaken in five cases. In the two instances where the liver had appeared hyperemic at autopsy, some hyperemia was found microscopically in addition to hemorrhage which not rarely led to destruction of limited areas of the liver tissue. In two of the three cases, which at autopsy suggested fatty changes, fatty infiltration was found under the microscope, while in Tb. 27 only parenchymatous degeneration seemed present besides plentiful hemorrhages. In most of the histologically examined material small areas of necrosis were noted. In such areas erythrocytes were not rarely present, sometimes quite plentiful. Leucocytes which were absent or rare within these foci could occasionally be found in small groups next to them. Such leucocytic agglomerations were commonly met with in apparently unchanged parts of the parenchyma. In Tbs. 13 & 14 evidence of *parasitic* invasion of the liver was found; a more detailed description of these changes will be given in a following paragraph.

In Tbs. 12, 13 & 14 bacteria were plentiful throughout the organ; they formed clusters at the periphery of the necrotic foci, but were less numerous in the center. In Tbs. 27 & 28 plague bacilli were quite plentiful in the parenchyma occurring almost all in the form of small clusters. Here only few bacilli could be detected within the larger blood vessels.

4. Spleen.

The spleen in all cases showed at autopsy acute swelling. Microscopically the organ was usually found rich in blood; hemorrhages were present in all five cases investigated. Tbs. 12, 13 & 14 showed in

addition small necrotic foci which sometimes were surrounded by hemorrhagic zones. Plague bacilli were plentiful in these cases, forming large clusters at the periphery of the necrotic areas. In Tbs. 27 & 28 no evidence of necrosis was found. Bacilli were here not so numerous or so evenly distributed as in the foregoing cases, being mainly clustered together in small groups.

5. *Lungs.*

The lungs showed at autopsy as a rule some congested areas.

In the five cases examined histologically, marked congestion was found in three (Tbs. 12, 13 and 14), while in two (Tbs. 27 and 28) this was less conspicuous. Bronchopneumonic foci of various sizes were found in every instance; sometimes these seemed to be situated mainly beneath the pleura. The exudate was partly fibrinous, partly cellular or merely cellular, the latter consisting of both leucocytes and erythrocytes in varying proportions.

In Tbs. 12, 13 and 14 fairly numerous bacteria were found in the vessels and capillaries as well as in the bronchopneumonic foci; within the fibrinous exudate they were as a rule absent, while groups of plague bacilli were seen occasionally around. In Tb. 27 the *B. pestis* were less numerous in the vessels, not evenly distributed throughout the foci, but occurred in clusters. In Tb. 28 the vessels seemed almost free of bacteria; in the foci only a few organisms were seen.

6. *Kidneys.*

The kidneys showed no marked changes at *post mortem* except slight signs of congestion and degeneration.

In the five cases which were further examined, parenchymatous degeneration was present as a rule. Congestion was usually much marked. Hemorrhages were invariably noted in the medulla. Rarely a little hemorrhage was noted in the cortex as well.

Bacteriologically numerous bacilli were found in Tbs. 12, 13 & 14 in the vessels and capillaries as well as in the hemorrhages, while in animals No. 27 & 28 their number was limited everywhere.

7. *Intestine and mesenterium.*

Marked changes were noted in Tb. 28 only. Here a part of the small intestine was found to be much congested; large hemorrhages were seen beneath the serosa and a big mesenterial bubo seemed present.

Histological investigation showed the intestine to be filled with a fibrino-hemorrhagic exudate. The intestinal walls appeared much congested with hemorrhages in all layers.

Several gram-negative bacilli were found in the intestinal contents; the majority like *B. pestis.* Numerous gram-negative and plague-like bacilli were found in the intestinal walls as well. where they were often grouped together.

The mesenterial gland was much changed, in addition to congestion small foci of necrosis being present. The capsule appeared at places thickened and infiltrated with leucocytes. In the surrounding tissue there was congestion and at places also leucocytic agglomeration.

Plague bacilli were found in great numbers in the gland as well as in the infiltrated parts of the surrounding tissues and in some of the vessels. The bacilli were as a rule not evenly distributed, but clustered together.

It can thus be seen that—apart from the unusual feature of a specific intestinal affection in Tb. 28—the macroscopic and histological results obtained in the animals now under discussion were more or less uniform. The only striking difference lies in the quantity and distribution of the bacteria, according to which they may be divided in two groups. In one bacteria were numerous everywhere and as a rule rather evenly distributed. In the other they were on the whole less numerous and more grouped together in the tissues, while in the blood vessels they were as a rule less numerous or even scanty. It is perhaps not wrong to assume that the animals of the first group were absolutely overwhelmed by the infection, while in the others some attempt to cope with the invaders by localising them may be noticed.

D. TARABAGANS SHOWING RESIDUAL PLAGUE.

We think it best to deal now with Tbs. 11 & 29 which-though showing lesions different from those in the animals suspected in 1926-7 to suffer from residual plague—yet evidently belong to the same category.

Tb. 11 was infected simultaneously with Tb. 12 on November 25, 1927. While the latter succumbed after 6 days to acute bubonic plague with general septicemia, Tb. 11 continued to live and was finally killed on December 16, i.e. 21 days after infection.

The results of the autopsy were as follows:

At the site of infection a large abscess was present in the musculature. In its center caseation could be noted in addition to necrosis of the enclosed muscle fibers. The musculature and connective tissue round the abscess showed congestion, moderate infiltration with round cells and cell proliferation. Bacteriological diagnosis was not quite easy owing to the presence of plentiful debris. Undoubtedly, only a small number of atypically short, gram-negative bacilli were present in the caseating center of the abscess, while they were almost absent in the outer layer and only scanty in the surrounding tissues.

A bubo surrounded by fatty tissue seemed present at autopsy, the piece in question being removed and preserved *in toto*. Unfortunately after fixation no glands could be detected in it; apparently the regional glands were so little changed as to escape notice at *post mortem*. The other external lymphatic glands appeared normal.

The liver was congested and showed a few nodes of yellowish-white colour and the size of millet grains. Microscopical examination confirmed the presence of congestion. Smaller leucocytic infiltrations and agglomerations were noted in addition to larger nodes. The latter were round in shape and showed in their center uniformly stained necrotic tissue pervaded by round cells which were especially plentiful at the periphery of the necrotic masses. A thick layer of connective tissue was seen to surround the nodes outside of which a ring of liver tissue was

noted which seemed to have undergone fatty degeneration; here as well as in the normal tissue round the nodes small leucocytic agglomerations were present at places, while they were rare in the connective tissue. Some atypical suspicious bacteria were found in the necrotic areas. In the tissue round them they were met with especially in and near the leucocytic agglomerations. A few suspicious bacteria were present also in the small leucocytic infiltrations and agglomerations while they were very rare in the parenchyma.

The spleen showed several slightly prominent nodes analogous to those in the liver. Microscopically they were also seen to possess a similar structure; necrosis was present in their centers, leucocytes being embedded in the uniformly stained masses and specially gathered together at their periphery. The nodes were surrounded by thick layers of connective tissue; around this hemorrhage was frequently noted. A few suspicious bacteria were seen in the central parts of the nodes; very few atypically big bacteria were met with in the surrounding connective tissue. In the parenchyma suspicious bacilli were present in small numbers.

The lungs were congested and showed several nodes of a similar appearance as those described above. Microscopically the organ appeared much congested. At places small groups of alveoli were filled with an exudate consisting of erythrocytes and leucocytes. In the center of the large round nodes leucocytes were so densely massed that it was impossible to distinguish with certainty a necrotic ground substance. Towards the periphery small and large areas with less dense infiltration were seen where necrosis of the lung tissue was manifest. The nodes were surrounded by thick layers of connective tissue which were—at places rather densely—infiltrated with round cells. Some suspicious bacilli were found in the nodes, and very few in the connective tissue round them, the alveolar exudate and the peribronchial glands.

The kidneys showed no marked changes at autopsy. Microscopically there was congestion. Areas similar to those found in the tarabagans of the preceding series (C) were seen in the medulla showing in addition to congestion and occasional hemorrhage formation of connective tissue. Only a few atypical suspicious bacteria were seen, mainly confined to the changed areas of the medulla and the glomerular capillaries, rarely at other places (e.g. in lumina of tubuli).

Smears from the site of infection showed a few suspicious bacteria. Cultures from the internal organs remained sterile. Two guinea-pigs were inoculated subcutaneously, one with an emulsion of two loopfuls from the local pus, the other with a suspension of two loopfuls of heart blood Both animals survived for more than a month when—being reinfected with virulent plague strains—they succumbed quickly.

Summarising our knowledge obtained from the above tarabagan, we may say that—as far as the gross appearances are concerned—this case should be classed as one of "nodose" plague. It will be remembered that in the previous paper we dealt with this form saying that in the tarabagan (as well as in other rodents) the presence of nodes does not

necessarily indicate a subacute or chronic stage of the disease. However they may point to such varieties, as has been seen in Tb. 11. In this animal the endeavours to grapple with the infection and to encapsulate the foci can be clearly seen. And—contrary to the tarabagans with "nodose" plague referred to last year—only few bacilli (partly involution forms) were found (a) at the site of infection, (b) in the nodes as well as (c) in the organs in general. Comparing these findings together with the negative cultural and experimental results, there seems little doubt that the animal, if not prematurely killed, would have recovered from the infection showing later on signs not of *resolving* but of *resolved* plague.

A more advanced stage was evidently reached by Tb. 29, which was inoculated on February 11, 1928, together with Tbs. 28 and 30, one of which died in 6 days of acute generalised plague. Tb. 29 slept uninterruptedly up to March 21, after which date it was occasionally seen to stir slightly. From about middle of April it was up at times and then partook of a little food. It took the animal about a month to become fully awake. After that time (middle of May) we at first thought it to be ill because it never barked or resisted when approached. No further symptoms developed, however, and finally we resolved to kill the animal on June 8, i.e. 118 days after infection.

The principal findings at autopsy were as follows:

Tb. 29. No changes were detected at the site of infection except some indistinct thickenings of the subcutis. Histologically cell proliferation could be noted in parts of the subcutaneous tissue, and after prolonged searching a small but dense agglomeration of round cells was detected at one place in the immediate vicinity of an artery. In gram-stained preparations small groups of gram-positive cocci were observed in the horny layers over the skin. In the leucocytic area as well as in the subcutis a few gram-negative suspicious bacilli were found; rarely some gram-positive cocci in pairs also were present.

The regional as well as the other external lymph glands were macroscopically found to be slightly enlarged but otherwise unchanged. Some capsular thickening was noted in the inguinal glands only. Everywhere a little brownish pigment was found in the otherwise normal parenchyma. Gram-negative bacilli were scanty in the regional lymph glands, and were only after much search detected in the other glands as well.

The liver was dark-brown in colour showing some mottled capsular thickening, and at one place three small nodes were present together. Under the microscope no marked congestion was noted. The capsule was at places diffusely thickened without marked cell proliferation. Smaller leucocytic infiltrations were frequently noted; sometimes a thin layer of round cells was seen lying immediately beneath the capsule, particularly at places where the latter was thickened. In addition, large leucocytic nodes, some round and some irregularly-shaped were seen. Within these, areas with necrotic changes similar to those described in the case of Tb. 21 were met with. Eosinophils, though occurring, were not so plentiful as in Tb. 21; a few giant cells were occasionally observed in the center of the nodes. Few suspicious bacteria were found in and near the leucocytic agglomerations.

The spleen appeared both macro—and microscopically free though it contained a few suspicious bacilli.

The lungs were pale except some areas where evidently recent aspiration of blood had taken place. Histological examination showed, besides the presence of districts where the alveoli were filled with fresh blood, other areas where small groups of alveoli were filled with both erythro—and leucocytes. A few gram-negative suspicious bacteria were noted in the lymphatic tissue round the small bronchi, occasionally also in the alveolar walls. Now and then a few gram-positive cocci similar to *Diplococcus pneumoniae* were seen within the alveoli.

The kidneys appeared unchanged at autopsy. Microscopically, in addition to some congestion small leucocytic agglomerations were detected in the cortex, rarely in the medulla. A few atypical gram-negative bacilli were found in and around the agglomerations as well as in the glomerular capillaries.

Smears from the site of infection, the regional glands and the spleen were negative; cultures from the internal organs generally proved sterile; that from the spleen showed slight contamination. Three guinea-pigs were inoculated with emulsions from (a) the site of infection, (b) the regional glands and (c) the internal organs (lung, liver, spleen and kidney). The last mentioned animal (c) died after 2 weeks, yielding no evidence of plague. The other two survived for a month and then succumbed quickly when reinfected with virulent plague cultures.

In spite of these negative cultural and experimental results we may take it for granted that Tb. 29 had contracted plague which it had so successfully resisted that it might almost be said to have reached the point of recovery. Since it is more than probable that the lesions found in the liver were mainly caused by a parasitic invasion of the organ, we may claim that—though the animal had undoubtedly passed through a kind of bacteremic stage—marked changes due to the plague infection were present at the site of infection only. In other words, even when making due allowance for the considerably longer time this animal survived as compared with Tb. 11, we may safely say that a fundamental difference lies between the two. Tb. 11 displayed the residues of "nodose" plague, while in the case of Tb. 29 evidently no such nodes had formed. Thus Tb. 29 of this series may be classed in the same category as Tb. 5A[2] of the 1926-7 series, though it is undoubtedly in a more advanced stage. Of great interest is the undeniable resemblance between these two animals (particularly Tb. 29) and those tarabagans developing features of *latent* plague. This point will be discussed later on. For the present, when we try to supplement the experiences made in regard to residual plague of 1926-27 with those observed in 1927-28, we may not only emphasise the existence of this type in the tarabagan but also conclude that recovery from plague infection may be brought about in quite different ways.

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[2] It will be remembered that this animal died accidentally 48 days after infection.

E. TARABAGANS SHOWING *LATENT* PLAGUE.

Of the 9 animals now to be discussed seven were killed at varying intervals after infection. In five of these the existence of a peculiar, *latent* form of plague could be proved by experiment. In the remaining two such proof is missing; nevertheless there is strong reason to assume that they also developed *latent* plague. The same holds true of Tbs. 17 and 24 which succumbed spontaneously, their deaths being probably due to accidental causes.

The following table gives some details of the tarabagans under discussion :

TABLE II.

Tb. No.	*Died* days	*Killed* days	*Results in gps. infected with material fr.* :		
			Inocd. Site	Regional lymph-glands.	Internal organs.
17	12	—	—	—	—
24	14	—	—	Survived.	Survived.
16	—	16	Plague pos.	—	Dto.
25	—	18	—	Survived.	Dto.
26	—	27	Survived.	Plague pos.	Dto.
15	—	28	Plague pos.	—	Dto.
30	—	28	Dto.	Survived.	Dto.
18	—	33	—	Plague pos.	Dto.
19	—	40	—	—	Dto.

N.B.—Cultures from the internal organs were invariably negative. Rarely, positive or suspicious results were obtained in smears from the site of the inoculation, the bubo or the internal organs.

A detailed description is now appended :

1. Local reaction.

Autopsy revealed in every case more or less marked changes at the site of infection. Generally speaking these were more prominent in the animals killed or having succumbed at an early date than in the later ones. In the first group usually some evidence of suppuration was macroscopically evident, while in the second group as a rule some thickening of the skin and congestion or similar alterations of the subcutaneous tissue were observed.

Histologically, the presence of abscesses in varying sizes could be satisfactorily proved in all cases with exception of Tb. 19. In this animal it was only after prolonged search that some minute collections of leucocytes could be found between the superficial layers of the muscles.

Reaction was invariably present round the abscesses, consisting of a slight diffuse infiltration of leucocytes often accompanied by congestion and hemorrhage, occasionally by an increase of the connective tissue. In addition, intracutaneously situated abscesses were detected in animals No. 26, 30, and 18; possibly—though full proof is wanting—a similar process was present in Tb. 19. In the first three cases the leucocytes did not stain well and were embedded in diffusedly-stained masses; evidently these skin pustules were of long standing. It may seem strange at first glance that these pustules were found only in the animals killed long after infection. We must remember, however, that in the others conspicuous local changes were a rule at autopsy, thus leading perhaps to insufficient attention being paid to any slight alteration betraying such skin abscesses to the naked eye.

Suspicious plague-like bacilli were detected in every case. Even in animals, where positive experimental results were obtained with material from the site of infection, bacilli were scarce in the abscesses and still scarcer around them. In Tb. 18 fairly numerous gram-positive cocci—similar to staphylococci—were present in the skin abscess. These cocci in addition to gram-negative bacilli were met with in the deeper layers. In animals 30 and 19 a few gram-positive cocci were found side by side with gram-negative bacilli in the skin abscesses, or in the superficial layers of the subcutis, while only scanty plague-like bacilli were noted in the deeper layers. Probably the invasion of the staphylococci had occurred secondarily in both cases. We are inclined to think that a similar evolution took place in Tb. 18 as well, as proved by the far greater number of the cocci in the skin abscess than in the deeper layers. It must be noted also that in this case large nests of staphylococci were seen on the surface of the skin. In Tb. 26 only plague-like bacilli were found distributed in small numbers.

2. Regional lymphatic glands.

Enlargement of one or a few of the regional glands was seen in some cases, being on the whole more conspicuous in the tarabagans succumbing or killed early than in the later ones. The same seems to hold true of slight periadenitic changes (slight congestion or hemorrhage).

Microscopically few changes were found within the glands except the regular presence of some brownish pigment deposits. Some congestion was noted in Tb. 17, while animals 15 and 16 showed slight hemorrhage. The capsule of the glands was more or less thickened at places with some cell proliferation, occasionally some round-cell infiltration. Slight periadenitic changes were confirmed not only in most of the animals dissected early, but were present also in some of the later ones.

Plague-like bacilli, repeatedly proved to be gram-negative, were found in every instance. Even in the cases where positive results were obtained with material from the glands, the bacilli were few in number, being often detected after a prolonged search only. In cases 18 and 19 a few gram-positive cocci (evidently staphylococci) were found in addition.

3. Other external lymphatic glands.

Whenever any uncertainty regarding the other lymphatic glands arose at an autopsy, material from these was preserved for further study.

A matter deserving attention is that positive findings were only obtained in such tarabagans where a longer interval had elapsed between infection and death. The lesions found were analogous to, though as a rule lesser in degree, than those noted in the regional glands. Some suspicious bacilli were present in a majority of the cases, occurring in very small numbers and not always typical in appearance.

4. Liver.

The liver of the tarabagans under discussion, while never markedly enlarged, appeared always more or less congested. Whitish capsular thickenings-usually in the form of small specks—were not rarely seen at *post mortem;* sometimes small nodes also. In Tb. 16 one of these appeared calcified: a cyst with size of a pea, filled with a clear liquid and evidently of parasitic origin, was also present.

Congestion of a marked degree was confirmed by histological investigation; sometimes this was most conspicuous beneath the capsule. The latter showed—both in the cases where this had been noted at autopsy and in a few others——spotwise or more diffuse thickenings; cellular proliferation was not marked at such places. A peculiar feature in Tb. 26 was the presence of two small cysts filled with homogeneous, fibrin-like masses, which were situated near one another immediately below the capsule; the latter showed diffuse thickening over this area, while some agglomerations of leucocytes could be noted in the liver parenchyma round the cysts as well as immediately beneath the capsule near them.

Hemorrhages were frequently found. Often they were situated at the periphery of the leucocytic agglomerations or they were in their turn surrounded by a ring of round cells.

Smaller leucocytic agglomerations were found in every case; they were either round and dense, or diffuse and loose. Except in Tbs. 24, 25 and 30, larger leucocytic nodes were present in addition. Some were similar to the small round agglomerations, consisting of closely packed round cells. In everyone of the six tarabagans, either one or a few large agglomerations of leucocytes were seen to surround *parasites* of a peculiar structure. These parasites were evidently identical with those already referred to in Tbs. 13 and 14. They did not resemble the flukes seen in the livers of other species of animals but were apparently roundworms of a not inconsiderable size, the diameter of the transversal sections being 26-32 microns, while the longitudinal sections encountered —though obviously corresponding only to a smaller or larger part of the curled-up parasites, reached a length of 130 microns or more. In the

many specimens at our disposal, the parasites were sometimes well preserved, sometimes only broken up fragments were encountered.

In Tb. 24 a whole calcified parasite surrounded by thick layers of connective tissue seemed present. It is not possible to state whether this was identical to the above mentioned parasites or not.[3]

Besides the above described nodes containing parasites, others containing none were seen in the six tarabagans. In these leucocytes were less densely massed together, sometimes intermixed with red blood corpuscles. Both red and white cells were embedded in an uniformly stained necrotic mass while round these areas dense agglomerations of round cells were present in which thin layers of connective tissue could be distinguished. Such nodes were not rarely encountered in the immediate vicinity of the parasitic ones.

Eosinophil leucocytes were sometimes noted in the larger nodes. Even on these occasions they seemed practically absent from the smaller agglomerations and infiltrations.

In all nine animals of the series a few suspicious bacilli were found in and near the leucocytic agglomerations of the liver. They seemed as frequent in the large nodes with necrotic changes as in and round the small ones; in some—though not all—instances such bacilli could also be detected in the nodes round the parasites. The suspicious bacilli were repeatedly proved to be gram-negative and seemed usually present in pure culture. An exception was Tb. 18 where a few small groups of staphylococci were noted in addition to the plague-like bacilli.

It can thus be seen that—as compared with the regional lymphatic glands—marked lesions were found in the livers of many of the tarabagans with *latent* plague. It would be too far-fetched, however, to ascribe them all to plague infection. In fact it seems that only the congestion and the small leucocytic formations, commonly present both in the animals apparently free from parasites and these harbouring them, may be ascribed to the plague infection. Such formations are often seen in animals suffering from acute plague. The larger leucocytic nodes, including those with evidence of necrosis, seem principally due to the parasitic invasion. This assumption is not disproved by the fact that positive bacteriological findings were obtained in such nodes. For it is naturally as easy for the *Bacillus pestis* to invade already damaged tissues as to attack normal areas.

5. *The spleen.*

The spleen was usually enlarged and slightly softer than normal; only in a few cases was the swelling marked. Hemorrhage was rarely

[3] We do not propose to enter here into a detailed description of the structure of the parasites, as this will be dealt with in a special paper.

seen. The follicles appeared often smaller than normal, while the trabeculae were prominent. In a few cases small necrotic areas seemed present. Bacteriologically some suspicious bacilli were found in every case; often they were so scarce as to be detected only after a prolonged search. In Tb. 18 a few gram-positive cocci (arranged in pairs) were noted as well.

6. The lungs.

Tbs. 17 & 24 which died spontaneously, deserve special notice. In these two animals areas of congestion could be noted in the lungs at *post mortem*. Histologically bronchopneumonic foci with a partly fibrinous, partly hemorrhagic exudate were noted. A few gram-positive diplococci were seen in and around the pneumonic foci as well as in the peribronchial glands, in addition to some gram-negative plague-like bacilli. As in the case of Tb. 21 there seems little doubt that the death of animals No. 17 & 24 was due to a rapidly evolving lung process of an unspecific nature. In every other respect the two last mentioned animals displayed the same features as the killed ones.

The latter animals did not show signs of marked pneumonia. Some changes attributable to recent aspiration of blood from forceful killing were often present in the lungs. The congestion, more or less marked in every case, might also be due partly to the forceful death. Sometimes, under the microscope, alveoli singly or in small groups, appeared to be filled, not with fresh blood, but by an exudate partly fibrinous, partly cellular. In the latter case both erythrocytes and white blood cells were present.

In every instance a few gram-negative plague-like bacteria were found in the alveolar walls and in the peribronchial glands. In some gram-positive diplococci—often like *Diplococcus lanceolatus*—were noted as well.

7. The kidneys.

No marked changes were noted at autopsy.

Histologically congestion was always present. Parenchymatous degeneration was occasionally marked. Small areas with round cell infiltration were in a majority of the cases detected in the cortex. Usually, in the same animals small areas were noted in the medulla where formation of connective tissue was seen in addition to congestion, slight leucocytic infiltration and occasional hemorrhage.

A few suspicious gram-negative bacilli were invariably noted in the capillaries especially of the glomeruli. Such bacteria were almost always found in the foci of the medulla and cortex.

It can thus be seen that quite marked alterations were present in the kidneys. Emphasis may be placed upon the foci in the medulla which

as regards position and size correspond to the hemorrhages noted in animals succumbing to acute plague. One is tempted to believe that these lesions present in the animals with *latent* plague were originally of an analogous character and have undergone changes of a reparatory nature.

F. DISCUSSION OF *latent* PLAGUE.

Surveying these experiments we may contemplate the alterations found (I) in the internal organs and (II) at the site of infection and in the regional lymph glands.

(I). With regard to the first we are confronted by three facts :

1. That certain lesions due probably to the plague infection were found in such organs as the liver, the spleen and the kidneys.
2. That not only in these, but in all other organs examined suspicious bacilli were found.
3. That on the other hand cultural and experimental tests yielded uniformly negative results.

On account of these negative findings (3), we must ask ourselves whether we have the right to consider the few bacteria found in the organs as plague bacilli. That we really have to do with true *B. pestis* seems proved to us for the following reasons :

1. The bacteria in question, though not always quite typical, were were nevertheless similar to *B. pestis* or its involution forms both in morphological and staining properties.
2. It is difficult to see what other bacilli could be so regularly present not only in organs within easy reach of the outside (e.g lungs), of the intestine (e.g. liver), etc., but also in others more difficult of access (e.g. spleen, lymph glands). A postmortal invasion is out of question in the killed animals, since in their case the internal organs were immediately removed and preserved in formalin. There is at present also no reason to suspect that the organs of hibernating tarabagans are invaded by unspecific bacilli. Similar looking, avirulent bacilli were found in Tb. 29, which awoke a few weeks before it was killed.

We believe therefore that the bacilli found in the organs of the tarabagans with *latent* plague ought to be considered as true plague bacilli. Their presence is in our opinion far less remarkable than the fact that they were invariably avirulent, while on the other hand virulent bacilli were present at the same time at the site of inoculation or in the regional lymph glands.

(II). Turning now to the areas in and near the site of infection it may be well to tabulate certain findings connected with them :

TABLE III.

Results in guinea-pigs infected with material from :

Tb. No.	*Inocd. Site*	*Regional lymph glands.*
17	—	—
24	—	Survived(a)
16	Died after 7 days of plague.	—
25	—	Survived(a)
26	Survived(a)	Died after 9 days of plague.
15	Died after 6 days of plague.	—
30	Died after 5 days of plague.	Survived(b)
18	—	Died after 10 days of plague.
19	—	—

Remarks : (a) Was afterwards found susceptible to plague infection.
(b) Died eventually of accidental causes, showing no evidence of chronic plague.

Before entering into a discussion of these results it is necessary to dwell shortly upon our experimental technique. The material required for the inoculations was kept until the end of the autopsy in sterile, covered Petri dishes, separate dishes being provided for internal organs, regional glands and inoculated site respectively. After the *post mortem* emulsions were made with the aid of previously sterilised normal saline and injected subcutaneously[4] into guinea-pigs by syringes which had been sterilised before the autopsy. Our invariable rule was to prepare and inject first the emulsion from the internal organs, then from the regional glands and finally from the site of infection.

As can be seen from Tables II & III we could rarely follow our general plan to infect in each case three guinea-pigs with emulsions from the internal organs, the regional glands and the inoculated site respectively. The lesions found at the site of infection and in the glands were often too insignificant or limited in size to yield sufficient material for both histological examinations and animals experiments. Hence the latter had to be sacrified for the former.

Some conclusions may be drawn from our experiments :

1. From Tables II & III it is gathered that in one case (Tb. 17) no experiments were undertaken at all[5] while in three (Tbs.

[4] In the experience of Gaiski made during a similar study upon susliks the usual percutaneous method of infection often failed. Gaiski strongly recommends peritoneal injection. This method, though undoubtedly sensitive, leads not infrequently to accidental or premature death of the test animals. We thought it best therefore to compromise by choosing subcutaneous infection.

[5] We erroneously assumed that the animal dying 12 days after infection had manifest plague and had consequently made no preparation for inoculations.

19, 24 & 25) their results were negative. In none of these last three animals was any material from the inoculated site used. On account of this and of the histological findings in all four animals, we feel convinced that they had *latent* plague in the same way as those animals yielding positive experimental results.

2. It is evident that virulent plague bacilli may be preserved not only at the site of inoculation (Tbs. 15, 16 & 30) but also in the regional lymphatic glands (Tbs. 18 & 26). As far as our limited number of experiments enable us to judge, the bacilli may be absent in the latter areas while present in the former (Tb. 30) and *vice versa* (Tb. 26).

3. The virulence of the bacteria preserved as above was undoubtedly rather impaired, the test animals succumbing as a rule considerably later than is usual with subcutaneous methods (the rule being 3 to a maximum of 5 days with our strains). This seems especially true in the cases where gland material was positive.

It will be remembered that the bacilli found in one of the two tarabagans succumbing to plague in 1926-7 after awakening at the normal end of hibernation, showed little if any loss in virulence, while in the other animal they may almost be said to have increased in virulence. If—as we have reason to think—these two tarabagans had *latent* plague while hibernating—we must assume that the plague bacilli preserved during winter at and near the site of infection increased in virulence as well as in numbers in spring and became thus able to invade the internal organs where formerly only few and avirulent bacilli seemed to have been present. Such a possibility is of great theoretical and practical interest.

4. For the reason that the infecting agent was not always strictly confined to the site of infection but may be present in the regional lymph glands we thought it advisable to apply for such cases the term *latent plague* as distinguished from a strictly local affection.

There is much resemblance between the findings made in Tb. 29 (which we believe to have suffered from residual plague) and the tarabagans displaying features of *latent* plague. In fact we consider that no sharp border line exists between the two types. In some of the animals with *latent* plague the bacilli surviving at the inoculated site or the regional glands may lose their virulence, reparatory changes may take place resulting first in *resolving* and then in *resolved* plague. We feel sure, however, that this holds true for only some of the tarabagans thus infected, while in the others the bacilli remain virulent at the site of infection or in the regional glands leading finally to generalised plague

at the end of the hibernation period. Perhaps it might have been desirable to observe such animals for longer periods than was possible in the abnormally mild winter of 1927-28. On the other hand, we may claim that the findings established in 1926-7 in the two tarabagans succumbing after awakening coincide nicely with the above scheme and thus firmly support our belief in the existence of a *latent* form of plague among tarabagans.

G. SUMMARY AND CONCLUSIONS.

Briefly we may summarise our 1927-8 researches as follows :

1. As in 1926-7 so in 1927-28 (with a specially mild winter) a considerable number of the infected hibernating tarabagans succumbed quickly to acute plague.
   Probably this was mainly due to the artificial conditions under which the animals were kept.
2. Experiences gathered from one animal dying on the third day after infection suggest that plague bacilli may invade the general system of the tarabagan quite early and in a gradual manner.
3. Some of the animals showed signs of recovery from the infection. These cases are probably more frequent in nature than under laboratory conditions.
4. A peculiar form of *latent* plague, in which virulent bacilli survive either at the site of inoculation or in the regional lymph glands or both appears to be the principal means of carrying over the disease from one season to another. This may explain the mystery of the perpetuation of plague among hibernating rodents.

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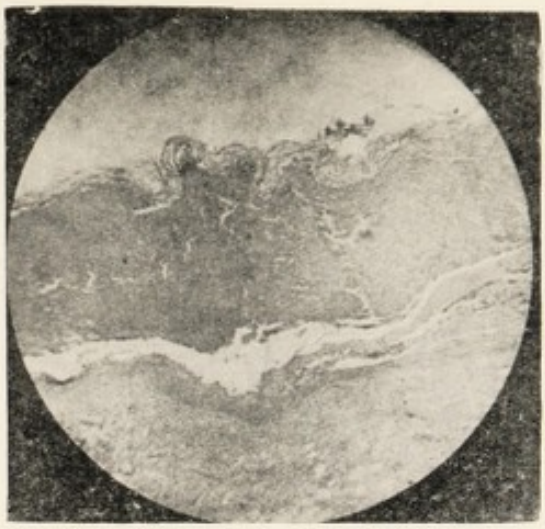

Fig. 8. Section of site of inoculation in skin. Tarab. 30. Latent plague. Showing intracutaneous abscess. Low magnif.

旱獺三十號示皮膚接種部潛伏型鼠疫示皮下膿瘍弱擴大

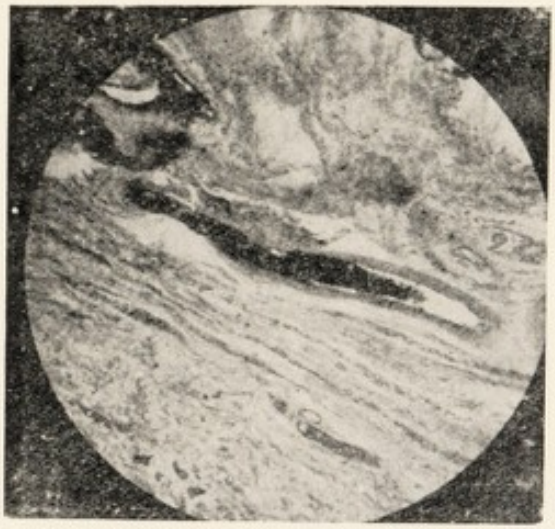

Fig. 9. Section of subcutaneous tissue near site of inoculation. Tarab. 29. Resolving plague. Showing small leucocytic agglomerations near artery at inoculated site. Low magnif.

旱獺二十九號示皮下組織附近接種部溶解性鼠疫示白血球小團聚在接種部之血管附近弱擴大

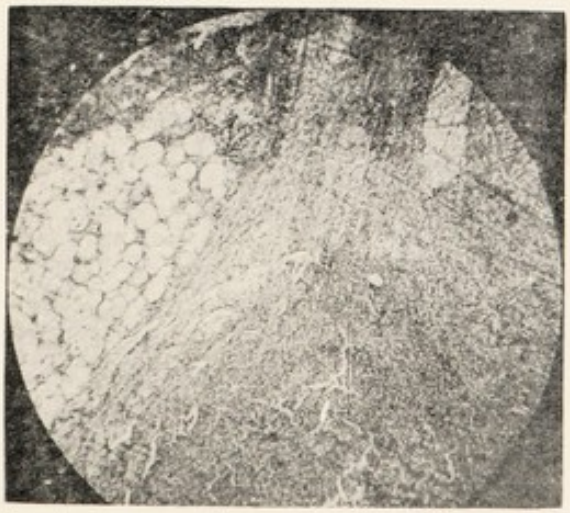

Fig. 10. Section of subcutaneous tissue near site of inocul. Tarab. 16. Latent plague. Showing local abscess surrounded by much connective tissue with infiltration and leucocytosis. Low magnif.

旱獺十六號示接種部附近之皮下組織潛伏型鼠疫示局部膿瘍有結締織及白血球浸潤圍繞之弱擴大

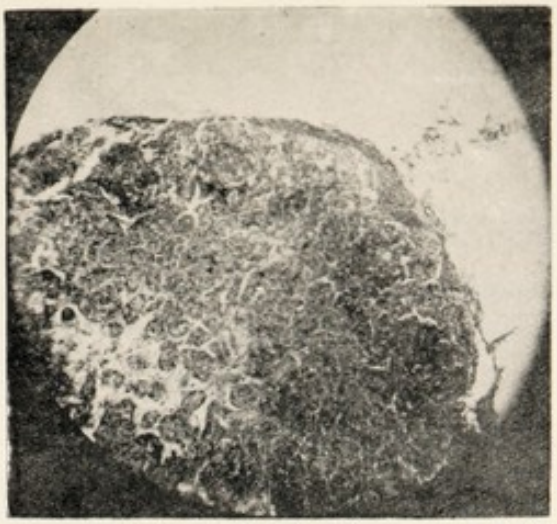

Fig. 11. Section of regional lymph gland, Tarab. 16. Latent plague. Showing no marked changes in parenchyma. Capsule thickened at places with some infiltration and cell prolifer. Low magnif.

旱獺十六號潛伏鼠疫示局部淋巴腺在腺質部著明病變缺如夾膜變厚有處見浸潤及細胞新生弱擴大

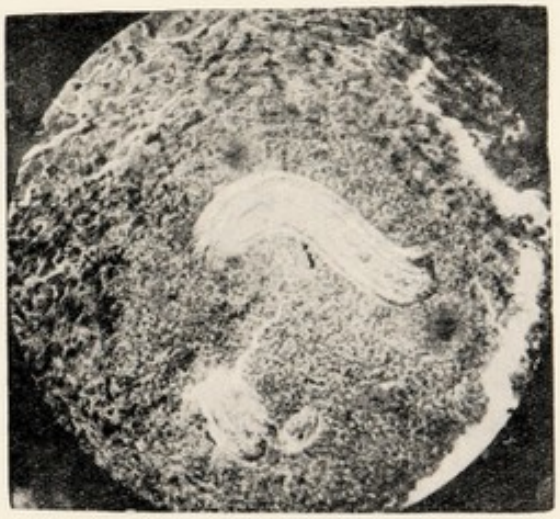

Fig. 12. Section of liver of Tarab. 17, infected with plague during winter. Died early. Showing parasitic worm cut longit. at two places. Low magnif.

旱獺十七號示肝臟此例當冬季使傳染早期致死示寄生蟲縱斷之兩處弱擴大

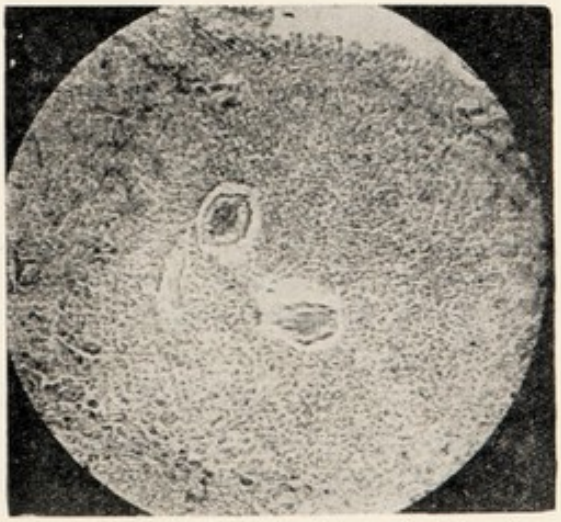

Fig. 13. Section of Liver of Tarab. 18, infected with plague in winter. Latent plague. Showing parasitic worm cut transversely with hooklets. Low magnif.

旱獺十八號示肝臟此例在冬季傳染爲潛伏型鼠疫示寄生蟲橫斷有鈎部弱擴大

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PROBLEMS OF PNEUMONIC PLAGUE.

(Read at the Far Eastern Congress of Tropical Medicine, Calcutta, 1927, and published in French in the Bulletin de l'Office International d'Hygiene Publique, 1928).

TABLE OF CONTENTS.

A. INTRODUCTION.

It is gratifying to note that the problems of pneumonic plague have received within recent years the attention they deserve. Nevertheless, considerable divergencies of opinion still exist on some important points. A discussion of these questions at such a meeting as this where plague experts from different countries are assembled is therefore welcome. The present occasion is particularly auspicious, since for the first time we are met in the country where—in addition to immortal research work done upon bubonic plague—the fundaments were early laid for our modern conception of the pneumonic form.

B. HISTORICAL SKETCH.

Before following the trend of events from this epochal period onwards it is necessary to trace the history of pneumonic plague in former times.

1. *Before the 14th century.*

Although the pneumonic form had occurred side by side with bubonic plague since time immemorial, but little evidence is now available regarding our early knowledge of the former. No doubt occasional cases of lung pest were confounded with the prevalent bubonic ones, while outbreaks with mainly pneumonic manifestations might not have been taken for plague at all. It is not surprising therefore that all we can establish is that the pneumonic type was in all probablity met with during the pandemic known as Justinian's plague (6th century), especially at Constantinople, and that an undoubted outbreak of this kind was rampant in Provence A. D. 1329.

2. *The Black Death (1346-1352).*

In spite of the scepticism still professed by some modern investigators, there is no doubt that the pneumonic type played an important role in

this worst of all known pandemics. It would lead us too far to discuss the evidence available in this respect from different countries. Suffice it to summarise that several contemporary recorders differentiated clearly between bubonic and pneumonic plague as manifestations of one and the same disease, and also noticed that those with lung affections were more infective and died quicker than the others. One prominent fact is that the pneumonic type was most prevalent at the beginning of many local outbreaks, but in only a few localities did the disease continue to rage in this form. As a rule it assumed a bubonic character after a few months. This change occurred often, though by no means always, with the approach of warm weather.

3. *From the end of the 14th to the beginning of the 19th century.*

One would expect that the clear differentiations between the various forms of plague, arrived at during the great pandemic known as the Black Death had become a matter of common knowledge. Here, however, as in other branches of medicine, we see that the truth, recognised only by a few advanced thinkers, soon became forgotten again. In this case it seems to be due mainly to the fact that—as long as plague was frequently rampant at the time soon after the Black Death—contemporary observers ceased to dwell upon an evil which had become familiar to everybody. Later on, descriptions were again voluminous, but confined almost entirely to the bubonic variety, pneumonic features, if present, having apparently been disregarded. This was the easier, because pneumonic manifestations, though undoubtedly met with in this period*, seem to have been quite rare. It may be added that the proofs we possess in this regard are almost entirely restricted to Europe. I know of only two testimonials pointing to their existence in the East :

a. Delbreil states without giving any authority, that the pneumonic type was met with in India during the sixteenth century.

b. *The Ku Chin T'u Shu* (an Encyclopedia published in Peking in 1726) says that in the year 1644 a great pestilence raged at Lu-an in the Southeast of Shansi Province. "Those attacked had hard lumps grow on the neck or arm, like clotted blood. Whole families perished. In some cases the victims vomited blood suddenly and expired."

4. *In the first half of the 19th century.*

This unfamiliarity with the pneumonic type of plague, which continued to be rare, reached such a degree that it was even doubted if the Black Death, where lung symptoms were frequent, was plague at all. It reflects great merit upon authors like Hecker, Webb and Hirsch to have proved the plague nature of this pandemic in general and of its pneumonic manifestations in particular. Webb and Hirsch at the same time pointed to the occurrence of the latter form in two modern Indian outbreaks, namely 1812-21 and 1836-38; here the pneumonic type was ex-

* For fuller information on this and other problems mentioned in this article see my "Treatise on Pneumonic Plague", Geneva, 1926.

cellently described by observers like Gilder and Whyte, Forbes, Keir, MacLean, etc. However, this form was considered peculiar to the "Indo-Chinese strain" of plague, and absent in "Western Asiatic" or "Levantine" plague.

5. *In the second half of the 19th century.*

The pneumonic type was met with occasionally, in particular the well-known outbreak at Vetlianka (South-East Russia) of 1878-79; but only a few keen and open-minded observers like Muench were convinced of the plague nature of the lung cases seen during this epidemic. It is to the everlasting credit of Childe (Bombay) that he first established definitely at the beginning of the present pandemic the pneumonic form as a special entity of plague. At this time pneumonic features were quite often met with.

6. On the whole lung pest was considered as an interesting but rare variety of the disease until the great Manchurian epidemic of 1910-11 showed that it might assume dimensions comparable to those of the Black Death. Still, the fact that this epidemic occurred during severe winter together with other data available at the time suggested to many observers that pneumonic plague was a disease peculiar to cold climates only.

7. *From 1911 to our times.*

It was only within recent years that this erroneous assumption has been revised. It is agreed now that the disease may also occur :—

(*a*) during hot seasons,

(*b*) in countries with a warm and even tropical climate.

There is no doubt that pneumonic plague is frequently encountered and an authority like Jorge (Lisbon) considers it to be more rampant now than earlier in the present pandemic. This greater frequency is perhaps more apparent than real, far more attention being now paid to this type than was the case a few decades back.

C. EPIDEMIOLOGY OF PNEUMONIC PLAGUE.

1. RISE OF EPIDEMICS.

a. *How do pneumonic epidemics start?*

Some authors have not emancipated themselves from the idea that pneumonic outbreaks arise *de novo*, i.e. that the first victim already which became infected directly from the rodents or through coming in touch with contaminated objects, may display features of primary pneumonic plague. In the course of our researches I have paid special attention to this aspect; I had the opportunity to analyse not only the etiology of the outbreaks in Transbaikalia, Mongolia and Manchuria but to consult practically all reports available from different parts of the world. The result of this study is that only in exceptional instances such a rise *de novo* is likely. Therefore the existence of outbreaks of this kind cannot be totally denied. But it must be emphatically stated that they are rare exceptions and not the rule. As proved by the ample experience gathered at the principal foci of pneumonic plague, like Egypt, Madagascar, South-

Russia, Transbaikalia and Manchuria, pneumonic plague usually originates from bubonic cases with secondary lung involvement. Some authors are inclined to ascribe a similar role to cases with skin or septicemic plague. It is evident that even when the former (skin) cases could act in such a manner, they are too rare to be of any practical importance. Those labelled as septicemic plague are more frequent but their role is also not firmly established. One point which should always be kept in mind is that one cannot be sure of the purely septicemic character of a case even where no buboes are *noted.**

b. *Why do pneumonic epidemics arise?*

While it can thus be seen that the problem as to how pneumonic outbreaks arise is satisfactorily settled, the question why such epidemics arise is still a justly much-debated one. Different theories are advocated in this respect which may be classified as follows:

(*a*) Bubonic and pneumonic plague, being due to one specific organism, are varieties of one and the same disease. The rise of epidemics of the latter type is due to chance or solely extrinsic causes.

(*b*) The rise of pneumonic epidemics is due to intrinsic causes.

(*c*) The question is still an open one, certain possible causes for the rise of pneumonic epidemics being known but not sufficiently investigated to warrant hard and fast conclusions.

Ad a. Allusion has been made already to the theory that pneumonic plague is prevalent only in cold climates, cases which may develop under warm weather remaining sporadic and not leading to epidemics. As said above, such views have not been borne out by recent observations, lung pest in epidemic form having been found under all sorts of climates and in diverse countries. In general there is much evidence against the "extrinsic" theory. It is known, for instance, that the disease is frequent in certain countries only, rare or absent in others with no milder climate or better social conditions. Another important fact is that in some areas the pneumonic type was frequent for a period immediately after introduction of infection, to become rare or absent later on. Again no change in climatic or social conditions exists which might be responsible for this peculiarity.

Ad b. One is tempted to assume that a fundamental difference exists between bubonic and pneumonic plague. Different theories have been formulated in this respect. Most of them are of historical interest only and need not detain us. One point which must be emphasized is that it has not been possible so far to find any marked difference in the bacilli causing bubonic and

* This aspect has been recently emphasized by Girard of Madagascar (Bull. Soc. Path. Exot., 1927, p. 632), and is the more interesting because formerly "septicemic" cases were thought to be a frequent cause of the pneumonic outbreaks in Madagascar.

pneumonic plague respectively; both kinds of strains are, as far as can be determined by present-day methods, identical. It is as easy to cause pneumonic plague in suitable animals by inhaling them with a strain derived from a purely bubonic human case, as to produce bubonic plague in laboratory animals by infecting them cutaneously or subcutaneously with fresh pneumonic strains or even directly with material obtained at the *postmortem* of lung victims.

There remains only one theory, once held in India and recently advocated by Norman White, which must be discussed. According to him, "the plague bacillus *alone* does not, and cannot, cause widespread epidemics of pneumonic plague..... and it seems more than probable that there is an additional organism at work—in other words, the plague bacillus in symbiosis with another organism is responsible for epidemic manifestations of pneumonic plague, which is a disease *sui generis*." Nicolle & Gobert argued that the filtrable virus of influenza might act together with the *B. pestis* in this respect and based their opinion upon epidemiological experience in Tunisia.

In my belief such theories are untenable and cannot explain the rise of pneumonic epidemics for the following reasons:

i. The pneumonic type in practically all outbreaks does not arise *de novo* but is traceable to bubonic plague, the connecting link being cases with secondary lung involvement.

ii. In most big pneumonic epidemics, a few bubonic cases are observed. Often such patients received infection more or less directly from pneumonic plague patients (or from corpses at *postmortem*) and yet developed bubonic plague, sometimes in quite mild form.

iii. Instances are known where simultaneously existing cases of bubonic and pneumonic plague could be traced to one and the same source.

iv. Influenza is not always prevalent at the time of pneumonic epidemics. In fact only very few instances are on record where simultaneous existence of both diseases was noted. In many others, where special attention was paid to a possible coexistence of influenza during pneumonic epidemics, the former disease was conspicuous by its absence.

v. No line can be drawn between sporadic and epidemic manifestations of pneumonic plague, as Norman White seems to do. Whether pneumo-pest *spreads* or not, depends upon extrinsic and not upon intrinsic factors.

vi. In general it should be kept in mind that the problem of mixed infection in plague is a very complicated one. Therefore, one cannot be too cautious in accepting any theory of this kind even when it *seems* to be supported by epidemiological or laboratory data.

Ad c. The peculiar distribution of pneumonic plague both from a geographical standpoint and in relation to the time of occurrence in certain areas, as discussed under (a), suggests that the difference between bubonic and pneumonic plague cannot be such a superficial one as advocated by the supporters of the "extrinsic" theory. To attempt a solution of this problem it may be well to analyse step by step the factors which might contribute to give an outbreak its pneumonic character. If any change in the form of the disease takes place, it might occur:

I. In the rodents, which cause the human outbreaks;

II. In the fleas which transmit the disease to man;

III. In the initial bubonic cases.

I. As to the first, the idea that the species of rodents involved in the epizootics might influence the character of subsequent epidemics is a very fascinating one. Especially, it has to be considered if a close relation does exist between epizootics in certain *wild* rodents and human outbreaks of *pneumonic* plague. In fact, a world-wide study of the disease both in rodents and in man as undertaken by our staff within recent years has yielded many data supporting this view. On the other hand, in some countries, where only ordinary rats are involved, the incidence of lung pest is conspicuous also. But before reaching any final conclusions, the following points should be considered:

i. Though ordinary rats are sometimes found to be the original source of pneumonic outbreaks, in many instances the local rodents were not involved, the disease having been imported from outside by human agency, namely by travellers incubating the disease.

ii. How long has the disease existed in the local rats? In some plague areas with rat epizootics, pneumonic plague was frequent soon after the introduction of infection but became rare afterwards. One might consequently suggest that the longer the infection continues among ordinary rats, the less chance there is for pneumonic plague to appear in man.

iii. Possibly when the epizootics for some reason receive a fresh impetus, e.g. through reimportation of infection or immigration of new animals, their character and hence that of human plague may also change.

It will thus be seen that different and often very intricate factors are at work. A final judgement may only be reached if and when the conditions present in the different plague areas are investigated according to a uniform scheme.

II. Regarding the second possibility it seems improbable that the particular species of fleas have a bearing upon the character of the epidemics. *X. cheopis* is the sole culprit in some countries where pneumonic plague is frequent. Further, it is debatable if the different fleas really play an independent part, or their apparent importance is due to the role of their hosts.

II'. When we consider that, apart from rare instances, primary pneumonic plague is not passed directly by the rodents to man but arises from human cases with secondary lung involvement, it becomes clear that factors which help to mould such secondary pneumonic features deserve our serious attention.

At the first glance it would seem likely that *frequency* of cases with secondary lung involvement ought to be of paramount importance for the rise of primary pneumonic epidemics. A careful analysis of available data suggests, however, that the decisive factor is not so much this as the *degree* of lung involvement which may develop in such cases. This degree may be marked in only a few cases, and in these cough is frequent, leading to the discharge of numerous plague bacilli in the sputum. I am sure that exact comparative investigations undertaken in this direction both in areas with and without primary pneumonic plague manifestation would yield valuable results.

If we ask ourselves what factors are primarily important for the development of marked lung involvement, mention must be made of :

i. an unusual susceptibility to respiratory diseases in general;

ii. a lessened resistance to plague infection.

That both factors are actually at work is proved by epidemiological observations. As an example we may point to the presence of severe secondary pneumonia in travellers and the frequency with which pneumonic outbreaks originate from such people.

We may thus summarise the above discussion :

i. It seems probable that not one factor but a sum of different factors, such as special character of the epizootics, susceptibility to lung diseases in general or to plague infection, etc., is responsible for the rise of pneumonic plague outbreaks. Each individual factor may not be present in every plague area where the pneumonic form exists. On the contrary it would appear that in the various localities a different chain of circumstances influences the nature of the epidemics.

ii. Although stress must be laid upon intrinsic causes, it is certain that extrinsic factors indirectly contribute to the rise of pneumonic plague. Climatic conditions may enhance the susceptibility of the lungs, while bad social conditions may impair the general resistance of the individual.

iii. In spite of the above explanations, the difficult problem as to how pneumonic epidemics arise is by no means solved. I have merely pointed out certain factors which appear to be of prime importance. Such can only be properly evaluated when their influence in the various plague areas is investigated according to a uniform scheme.

2. SPREAD OF EPIDEMICS.

While extrinsic factors alone cannot account for the rise of pneumonic epidemics they are instrumental for the spread of the disease.

The factors at work may be classified under three headings :

(*a*) Climatic influences;

(*b*) Social influences;

(*c*) Influence of measures taken.

It is obvious, however, that no sharp line can be drawn between the three groups. Thus both climatic conditions and poverty may cause overcrowding and lack of ventilation; both ignorance and extreme cold may interfere with the sanitary measures.

There is also no doubt that the influences at work are of unequal importance in the various plague areas, with their different climate, population, economic conditions and customs. It is therefore not strange that the diffusibility of pneumonic plague varies so much. The presence of many favourable factors or even a few powerful ones may foster the spread of the disease. *Vice-versa*, the absence of similar conditions tends to cut short a continuation of the outbreak.

A few points deserve special mention :

(*a*) Some authors not merely associate the influence of adverse climatic conditions with the creation of unhygienic conditions in the tightly shut and overcrowded houses, but also point to more complicated reasons. Thus, Teague and Barber assume a low water deficit of the atmosphere, as present in cold climates, to cause the droplets of sputum to float longer in the air and thus favour the spread of the disease.

This theory is in my opinion negatived by two facts :

i. Pneumonic plague occurs not only in cold climates but also in warm countries where evidently the atmospheric conditions considered as essential by Teague and Barber do not exist.

ii. Still more important is the fact that a mediate infection in pneumonic plague as presupposed by this theory, though perhaps occurring occasionally, is rare, pneumonic infection being usually contracted in the immediate vicinity of the patient. I will return to this question later on when dealing with the infectivity of the disease.

(*b*) It is a characteristic feature of many pneumonic epidemics, e.g. in Manchuria, that either no secondary infection of rats occurred at all, or that—when such resulted in rare instances—no epizootic followed. Certainly in epidemics running their course during cold winter a low flea-rate and unfavourable biological conditions for the fleas might be responsible. But such an absence of secondary rat infection was observed under other climates as well, e.g. in Upper Egypt. On the other hand, it is claimed by observers in India (Punjab) that such a secondary spread of infection to rats may occur and lead to real epizootics accompanied by human bubonic cases. The possibility of such rat infection must therefore not be underrated, and further attention should be paid to this important problem.

3. DECLINE OF EPIDEMICS.

When dealing with the spread of pneumonic plague we emphasized the value of the measures taken against the disease. The object in view is not only a limitation of the spread of infection but also a speedy termination of the epidemic. Obviously, the same factors which influence the steps taken to limit its sway are also at work in stamping out the disease. With ample means at the disposal of the medical staff and a free hand to use them, the good-will and active co-operation of the population will hasten the beneficial results. *Vice-versa*, insufficiency of means, prejudice and opposition will postpone them. Another conditional factor is the weather. Outbreaks which start under unfavourable conditions (e.g. cold, rainy seasons, etc.) will tend to diminish in severity so soon as climatic conditions show an improvement. This may be brought about directly by facilitating the anti-plague campaign or indirectly by lessening overcrowding, improving ventilation, etc. On the other hand, severe conditions of the weather setting in after the appearance of an outbreak may considerably aggravate its course.

It is thus evident that, as in the spread of pneumonic epidemics, their decline also depends upon a combination of different factors, the relative importance of which varies in different plague areas.

A much debated question is whether there may also be a tendency for the outbreaks to decline *spontaneously*. Some believers of this possibility explain it by a lessening in the virulence of the *B. pestis*. Such an assumption is, however. not borne out by facts. The bacilli, passing directly from man to man, do not become less virulent, but on the contrary there is evidence that during a pneumonic epidemic their virulence is enhanced. It might seem paradoxical to maintain a spontaneous decline of epidemics under such circumstances. But the findings made in Harbin and Vladivostok at the end of the 1920-21 epidemic tend to solve this riddle: most of the cadavers dissected at this period did not show the usual pneumonic foci, but presented—though undoubtedly infected through the respiratory tract—only hyperemic and oedematous changes in the lungs as well as marked septicemic features. We designated these cases as pulmonary plague, because, though anatomically similar to the septicemic ones encountered in bubonic plague, they were etiologically different from

them, the infection having entered through the respiratory tract. These patients with *pulmonary* plague are undoubtedly less infectious than those with the usual features of plague pneumonia.

There is no reason to assume that these *pulmonary* cases were caused by plague bacilli weakened in virulence. Most of them were not seen clinically but found dead in the streets; their illness was probably short and they were thrown out of the houses before the search parties could detect them. The only case we saw clinically died on the day after admission to hospital without showing any cough or expectoration. Thus we concluded that these cases were probably due to an enhanced virulence of the *B. pestis*, developing through passage from lung to lung. We assumed that through such repeated passage the invading organism finally became so virulent as to cause mainly *pulmonary* cases. These—though very fatally infected—are comparatively non-infectious, because the disease runs a rapid course, and as a consequence the principal medium of infection, namely the cough, is absent. In other words, our post mortem findings at the end of the 1920-21 outbreaks afford a scientific reason in favour of a spontaneous decline of pneumonic plague epidemics.

D. PATHOLOGY OF PNEUMONIC PLAGUE.

Most authors agree that primary pneumonic plague is due to an infection through the lower portion of the respiratory tract. A minority, however, take an opposite view, assuming that it enters through the tonsils, etc., and reaches the lungs secondarily through the blood or lymph stream. It appears to me that the evidence available both upon pneumonic cases and such with tonsillar lesions does not support this view. I would like to point specially to an unique case observed by Jettmar of our Service, concerning a man who committed suicide in the early stages of pneumonic infection :

Russian peasant, aged 31, suddenly felt shivers on the evening of February 18th, 1921, accompanied by headache and pains in chest. Before daybreak he left his hut and was found soon afterwards hanging from the fence of his garden. Owing to the head and neck being frozen, no complete autopsy was performed. The lungs were examined *in situ* and samples taken from them and spleen for histological examination.

Macroscopically the right lung showed hepatisation of the middle lobe, with confluent pneumonic foci in the upper lobe and early engorgement in the lower lobe. The left lung was not materially altered.

Histologically, the alveoli of lungs were filled with blood serum and exudative cells. Where the changes were early, plague bacilli were found throughout the alveoli; in the more advanced foci, organisms were concentrated near the alveolar walls adjacent to small vessels.

No plague bacilli could be seen in the capillaries; these, however, showed considerable congestion, but no thrombi. Likewise, other blood vessels were apparently free from bacilli. But the organisms could be seen forming dense clusters in the lymph spaces of the adventitia and media. At places they even reached the outer wall of the intima; here proliferation, inflation and desquamation of the endothelium were observed.

The spleen showed inflammatory hyperplasia, but *no plague bacilli could be seen anywhere.*

This case well illustrates the fact, established also by other observers, that bacilli generally appear in the blood after lung symptoms have already manifested themselves. Indeed, a few cases are on record, where death had occurred before bacteremia had developed.

Koulecha, the main advocate of the tonsillar theory, says that a final decision of this problem can only be reached by experiments and not by histological findings. This evidence may now be taken:

Strong and Teague, experimenting upon a large number of monkeys, found in animals infected by spraying that the lesions corresponded exactly to those of human pneumonic plague; the alterations in the fauces and cervical tissue were in practically all instances slight and obviously secondary in nature. On the other hand, monkeys which were infected in the fauces "all died of plague septicemia with or without bubonic infection of the cervical glands; that is, in the case in which the infection was severe and the susceptibility of the animals more marked, they succumbed to septicemia before cervical buboes developed. In none of these instances was pneumonia present."

Analogous experiments were performed in 1925 by Wu Lien-Teh and Jettmar upon tarabagans and sisels. We examined on one hand animals which succumbed to plague inhalation spontaneously, and on the other those which had been killed in early stages of the infection. In no case did we see primary foci in the fauces (tonsils), trachea or main bronchi. Occasionally they were situated in smaller bronchi. Most often, however, the portal of entry was found in the deepest parts of the respiratory tract, the *bronchioli respiratorii* and the alveoli. It may thus be concluded that our experiments, while following the line of Koulecha's wishes, have produced exactly the opposite results this author expected. In other words, we are sure that pneumonic plague invades the body through the lower portion of the respiratory tract.

E. INFECTIVITY.

This well-established fact is not only of theoretical interest but of great practical importance. For it is evident that such a mode of infection may not be easily effected by merely coming in touch with contaminated objects. Even the patients may be considered as comparatively harmless unless they cough or otherwise spray the infective material.

These theoretical expectations have been fully confirmed by practical experience. Only a few instances are on record where the evidence of a mediate infection is convincing or even suggestive. Often children and aged persons are seen to escape infection. This is certainly not due to their being immune but to the fact that they are not so apt to be in close contact with the patients as the middle-aged. Frequently simple measures of precaution, apparently useless from a theoretical point of view, have been found to save the lives of persons sharing the house or even room of the sufferers, because these helped to keep the contacts outside the range of the patients' cough. Therefore it may be safely concluded that in the same way that the transmission of bubonic plague depends

upon blood-sucking insects, particularly the rat-flea, so pneumonic plague is conveyed from man to man by the cough of patients.

Having ascertained that the real danger of pneumonic plague infection lies in the immediate range of a coughing patient, we must further ask ourselves :

(*a*) Whether a short contact of this kind is sufficient to cause infection,

(*b*) Whether the infectivity of the patient is equal under all circumstances.

Ad a. Infection through *short* contact undoubtedly takes place. As a rule, however, there is a history of *prolonged* contact with previous cases and instances are on record where persons coming quite near a patient for a short while, remained unharmed.

Ad b. Regarding the second question it has been firmly established that, owing to the absence of cough and expectoration during the first stage of the disease, the patients are then practically non-infective. Indeed it is the ideal of our preventive campaign to detect and isolate patients during this period which is usually of 24 hours' duration.

As soon as this first stage of the illness is passed, the patients become more and more dangerous on account of a continuous increase of both the cough and the number of plague bacilli emitted by it.

A controversial point raised by some observers, especially French workers in West Africa, is whether pneumonic plague is less infectious under warm than cold climates. It would seem to me that the arguments brought forward in favour of such a lessened infectivity are not convincing. Firstly it is doubtful whether *primary* pneumonic plague is so frequent in French West Africa as sometimes assumed. Apart from this, it is apparently not the infectivity of the individual patients so much as the diffusibility of the disease which is at variance. Proximity to the patients within a certain radius is apparently fraught with equal risks under all conditions. On the other hand, it is possible that the range of direct infectivity may somewhat vary under different climatic conditions. It would be well therefore if tests in this direction, as performed by Strong and his collaborators in Manchuria, could be made in as many plague areas as possible. I believe that such observations would reveal only gradual but no fundamental differences.

F. VALUE OF VACCINES AND SERUM.

1. PROPHYLAXIS WITH VACCINE AND SERUM.

It is known that a pessimistic opinion as to the value of vaccine prophylaxis in pneumonic plague is entertained by many authors. An exhaustive study of this subject shows, however, that this problem is not sufficiently elucidated to permit final conclusions. While it is possible that further tests performed upon a large scale under rigid controls may produce better results than have been obtained hitherto, I cannot help pointing to the great difficulties existing in this direction.

The inoculation of vaccine is sometimes opposed on theoretical grounds, it being maintained for instance that a negative phase follows which renders the inoculated more liable to infection. I am led to believe that this danger is more imaginary than real. In fact it appears that vaccination tends to prolong the incubation period. It is likely, however, that immunity develops not at once but gradually, and that it takes some days before full protection is afforded to the vaccinated. Under these circumstances it would seem advisable to combine vaccination with prophylactic serum administration. In fact it seems that the latter tends to confer quick though short-lasting immunity, and its combination with vaccine might be a hopeful method. In the case of the personnel, exposed to infection throughout a long epidemic, it may not be wise to administer prophylactic serum, as anaphylactic symptoms may result when therapeutic doses become necessary later on. For the ordinary contacts of the patients this objection does not hold. Here, however, the high cost of such combined vaccination and the additional work for the staff have to be considered. As already alluded to, prophylactic vaccination (and similarly serum administration) are apt to prolong the incubation period, so that the observation for contacts would have to be prolonged, involving further expense. Last but not least, it must be kept in mind, that even when no inoculations are practised, the attitude of the population towards the medical staff is often hostile. Should any of the inoculated develop plague, this might be taken by the people to have occurred not *post hoc* but *propter hoc*, and the doctors be blamed for inoculating the disease under the pretext of fighting it. I fully understand therefore why authors with great experience are sceptical in regard to prophylactic inoculations in pneumonic plague epidemics and rely upon other means of prophylaxis. It is certain that the inoculations as available to-day are no panacea, and further research is essential.

2. SEROTHERAPY.

The same may be said with respect to the therapeutic application of serum. It is known that the prognosis of pneumonic plague is well-nigh hopeless; I could collect less than 30 cases which presumably recovered from this disease among tens of thousands of records. The fact remains, however, that most of the recovering patients and also a number of others who presumably suffered from marked secondary lung involvement, had received energetic serum treatment. Certainly the best we can do at present for a pneumonic plague patient is to treat him as soon as possible with a potent immune serum, obtained—if feasible—from local strains, in combination with a careful heart therapy and adequate nursing.

G. CONCLUDING REMARKS.

In this rapid survey I have necessarily concentrated upon problems still *sub judice*, omitting those which are already agreed upon. I trust that I have not created the impression of pessimism in regard to our chances of successfully combating pneumonic plague. On the contrary, I believe that such epidemics will become less frequent and widespread for the following reasons:

1. This form of plague is dependent upon epizootics and human cases of bubonic plague. Whatever is done for the eradication of these, will effectively help to stamp out the pneumonic form.

2. More attention is now paid to the pneumonic variety than in the past. And the sooner this disease is recognised, the easier it will be to check its spread. A slow but steady change has also been noted in the attitude of the public towards the medical staff. If they co-operate with or cease opposition, much quicker results will be achieved than heretofore.

3. Finally much may be hoped from the spirit of international co-operation now prevailing, as demonstrated by this meeting of experts, initiated by the Health Section of the League of Nations. There are several problems of pneumonic plague which can only be settled by common endeavours. I trust that the day is not far distant when their solution will be firmly taken in hand.

WU LIEN TEH.

RECENT KNOWLEDGE ON PNEUMONIC PLAGUE.

(Published for the first time.)

TABLE OF CONTENTS.

A. INTRODUCTION.

Since the first edition of my Treatise on Pneumonic Plague was published in 1926, much new material bearing on the subject has been collected, particularly from Russia, India and Madagascar.

My recent visit to India early in 1928 as a member of the League of Nations Health Interchange, during which I tried to procure information from every available source, has convinced me more than ever that a considerable number of pneumonic plague outbreaks are left unobserved or unrecorded, thanks to the intransigent attitude of persons who will not move with the times, thus missing or allowing their subordinates to miss events which would otherwise have enhanced our knowledge of this most fatal of diseases.

The publication of a new volume of the Manchurian Plague Prevention Reports now enables me to make use of the information gathered during the past two years, which I trust will bring up to date what is known of the subject.

While the chapter on wild rodents formed a prominent part of the Treatise, I have thought it advisable in the present Report to treat this important problem in a separate article.

B. INCIDENCE OF PNEUMONIC PLAGUE.

I. INDIA.

In my "Treatise on Pneumonic Plague" I concluded from available records that the higher incidence of plague pneumonia as observed in the earlier years of the present pandemic, did not persist long in Southern India. I emphasised, however, that groups of these cases were not entirely absent and could in this respect point to a number of outbreaks in the Madras Presidency. During my recent tour in India (1927-8) I was able to collect additional evidence in this as well as other areas.

(a) *Madras Presidency.*

Lt.-Colonel A. J. H. Russell, Director of Public Health of the Presidency, informed me that during the ten years of his residence he has experienced three outbreaks of plague pneumonia which all occurred at Calicut, Malabar District (on the West coast of Madras Pres.):

Year.	*No. of cases.*	*Result.*	*Duration of outbreak.*
1916	? 15	All fatal	? one week.
1922	16 (2 families)	All fatal	7-10 days.
1927 (Dec.)	22 (3 families)	All fatal	10 days.

Graham (1) notes 22 fatal pneumonic cases at Maduvattam (Ponnani Taluk).

The above data, taken together with those already published in the "Treatise", show that pneumonic outbreaks are by no means rare in Madras Presidency, whose climate is distinctly hot.

(b) *Mysore State.*

Chief Medical Commissioner Dr. B. Mohamed Usmon told me that he had often noted pulmonary complications in bubonic cases characterised by fever, cough and bloody sputum. These may occasionally lead to primary pneumonic plague which latter wipes out whole families. The last instance he saw occurred in Mysore City in 1926 when a family of seven died out. Dr. Usmon said that pulse and temperature in the pneumonic cases were unusually low, the latter not being higher than

100-101°F. While some cases with secondary pneumonia recovered, the primary ones were invariably fatal.

In the Victoria General Hospital at Bangalore (the administrative center of Mysore State) an outbreak took place in the winter of 1907 during which five nurses, including the English Matron and the Chief English Nurse, died within 12 days after a patient diagnosed as "pneumonia" had been admitted into the general medical ward of the Hospital. The diagnosis of plague pneumonia was microscopically confirmed in the case of one of the nurses. A tablet dedicated to the memory of these five victims is now hung in the main hallway of the Hospital.

According to Dr. Mascarenhas (Health Officer of Bangalore) there occurred in Bangalore Cantonment 10 fatal cases of pneumonic plague among Indians between February 6 and 10, 1927.

(c) *Bombay Presidency.*

Dr. Kelkar, Secretary of the Belgaum Medical Society, told me that he personally attended 20-30 cases of primary pneumonic plague at Belgaum in the year 1907. These fatal cases occurred suddenly among a group of wrestlers living together in a gymnasium, the whole outbreak lasting less than a week.

Dr. Henkor of Belgaum was in charge of six cases of lung pest in the city in November, 1927, all belonging to one family. Three cases in a neighbouring village could be traced back to this familial outbreak.

It is difficult to believe that the above outbreaks as well as the few about which we happened to hear beforehand are the only ones occurring in the South of India. In fact some of the medical officers with whom I had the privilege of talking upon this subject said that similar outbreaks of primary pneumonic plague are by no means so rare as reported. One doctor went so far as to designate the statement regarding the non-existence of pneumonic plague in India a fable. According to him one difficulty in detecting such familial outbreaks lies in the rapidity with which they come and go. Another important fact which seemed puzzling to this colleague is that—though usually all the members of the attacked family perish—no further spread of the infection takes place. To me this seems not surprising since there is little doubt that the diffusibility of lung pest is low under the conditions prevailing in the South of India. Hence the proportion of primary pneumonic to the total plague cases appears to be low and little attention is paid to this type. However, as I have always emphasised, for a true understanding of the problems of plague pneumonia it is far less important to know how far the disease may *spread* under favourable extrinsic conditions than to investigate where and how frequently such cases may *arise*. For this reason it would be well if the lofty attitude adopted by certain workers in India in regarding the pneumonic type as a *quantité negligéable*, is given up, and a more liberal policy introduced.

It is generally admitted that pneumonic plague outbreaks are not rare in the northern parts of India. The evidence quoted in the "Treatise" may now be supplemented :

(a) *United Provinces.*

Dr. B. P. B. Naidu, Senior Research Plague Worker of the Haffkine Institute, mentioned that, when sent to Agra in February, 1927, to investigate the plague epidemic, he saw four cases of plague pneumonia occurring in a family and all dying within one week. The cases, which were all confirmed by microscopic examination, showed severe symptoms.

(b) *Punjab.*

Though additional information has been received through the kindness of Col. C. A. Gill (Director of Public Health, Punjab Presidency) and Dr. Butt (Dep. Director Public Health, Lahore), our data are still incomplete. As Gill (2) states in his "Genesis of Epidemics" (1928), "it is not possible, owing to the fact that deaths from pneumonic plague are not separately recorded, to determine with precision the distribution and intensity of pneumonic plague, but field observations show that outbreaks of this form of plague, more especially in the extreme north of the province—the districts of Attock, Rawalpindi, Jhelum, and Shahpur—occur with considerable frequency during winter and early spring."

The data in our possession may be summarised thus :

Year.	*Season.*	*Locality.*	*Pneumonic cases.*	*Remarks.*
1918	? spring	Lyallpur	4	Out of 200 total cases.
1920	January-March	Shinka & Nartoppa	1,056	Imported from Rawalpindi. Stated to have been followed by epizootic & bubon. c. (2)
1923	?	Rawalpindi	4	Out of 600 total cases. Pneum. c. in one family.
1923	?	Lahore	10	All in one family, only one child escaping.
1924	September	Upper Topa. Murree Hills	8	Imported from Rawalpindi.
1927	March	Village in district of Gurgaon.	90	Apparently preceded by mild bub. outbreak. Pneum. ep. lasted 1 week.

N.B.—In 1923 12 fatal cases out 2,000 occurring at Mooltan had lung symptoms but it was not established whether these were secondary or primary in nature.

(c) *North-West Frontier Province.*

Further data on pneumonic plague outbreaks in this area are given by Graham (1) so that the available information is now as follows :

Year.	*Season.*	*Locality.*	*Pneumonic cases.*	*Remarks.*
1920	March 10-April 2	Jalsal (Mardan tehsil)	26 (25d)	Introduced by patient with sec. pneumonia; followed by severe bubonic outbreak lasting 3½ months and causing 679 c. with 437 d.
1920	March 31 April 2	Jalbal, 5 miles from Jalsal	17 (15d)	Imported through pneumonic case from Jalsal.
?	?	Gazikhel	13 fatal c.	Lasted one week and was followed by bubonic outbreak.
1924	January	Near Taxila	49 fatal c.	Preceded by bub. outbreak in 2 adjacent villages.
?	?	Batangi	24 fatal c.	

II. JAVA.

Detailed figures for the year 1924 which have just been received (3) show that out of a total of 13,116 plague deaths 957 (or 7.3%) were due to plague pneumonia, as against 4.1% in the year 1923. An "explosion of pneumonic plague" is noted as having taken place in May and June in Kampong Ardjosari (2000 inhabitants) situated on the Tenger Hills of Eastern Java and claiming 55 victims. The type noted in two further "explosions" in November in the desa Pandansari (37 deaths) and in December in the desa Wonokerto (11 deaths) is not stated.

III. CHINA PROPER.

The occurrence of pneumonic plague at Amoy was again reported in the year 1924 together with bubonic and septicemic cases. An interesting feature of the 1923 and 1924 invasions was the prevalence of cervical buboes (4).

IV. JAPAN.

Timura in a short survey of plague epidemics in Japan (5) mentions a slight manifestation of lung pest which occurred in April, 1914, in Chiba Prefecture and was responsible for 11 deaths. He considers this as the only outbreak of this type in Japan.

V. AUSTRALIA.

That primary pneumonic plague, though rare on this continent, was not altogether absent is proved by the following data kindly furnished by Dr. Cumpston, Director General of Health, Commonwealth of Australia.

(1) Two fatal cases were seen during the plague outbreak at Brisbane in 1901.

(2) The pneumonic form played an important role in a small epidemic at Maryborough, 167 miles from Brisbane, in 1905. Here the first victim was a lad, age 17, who worked as an assistant in

a fruiterer's shop where plague infected rats had been previously caught. It is believed that he suffered from septicemic plague with secondary pneumonia and passed the infection in this form to four brothers and sisters. Burnett Ham says in this connection that "the squalid surroundings of the family, their ill-nourished bodies, and insanitary dwelling abode were doubtless predisposing causes in lowering individual vitality and the natural resistance to infection. "Plausible as this is, the *post mortem* protocols of two of the secondary cases seem to us more suggestive of primary pneumonic plague. There is no doubt that a neighbour who had voluntarily nursed the first victim, and two nurses who attended the other children in the hospital, contracted true lung plague and all succumbed. A third nurse sickened with headache, pyrexia and general malaise, but recovered, having received prophylactic serum injections.

(3) A second small pneumonic outbreak noted at Balmein (New South Wales) in 1906, will be dealt with separately because it started on board ship (6).

VI. SOUTH AMERICA (ARGENTINA).

A small epidemic of pneumonic plague was reported as occurring in July, 1927, in a village 25 miles inland from Parana, Argentina (7). It will be remembered that a similar outbreak had taken place at Tucuman, an inland center, in April, 1918 and was preceded by rat-caused bubonic cases. Probably a similar evolution has occurred in the Parana district. Anyhow there is no doubt that small endemic centers exist in the inland provinces of Argentine and that, while the bubonic type seems to prevail, the outbreaks may assume pneumonic features.

A second outbreak at Buenos Aires will be discussed later on as the disease originated on board a vessel.

According to another recent (June 1928) report (8), great alarm was caused at Buenos Aires by the death of a patient from post-operative pneumonia which was said by the attending physician to be due to pneumonic plague. Strict measures were taken but it was soon established that the diagnosis was erroneous. As far as I know this is the first instance where doubt existed as to the differentiation between plague and postoperative pneumonia.

VII. EGYPT.

The additional figures available for Egypt are as follows (9):

Year.	*Bubonic and septicem. c.*	*Pneumonic cases.*	*Remarks.*
1924	370*	7	
1925	135	3	Pneumonic cases occurring at Minia (Upper Egypt).
1926	150	4	

* Instead of 377 cases as erroneously stated in the "Treatise."

As compared with previous years the incidence of pneumonic plague was conspicuously low during the years 1924-26 together with a considerable decrease of total cases. Further events in Egypt should be watched carefully.

VIII. TUNISIA.

A further outbreak of pneumonic plague, taking place in the Achache District in February, 1927, has recently been reported (10).

IX. FRENCH WEST AFRICA.

Dealing with the events in this colony in my "Treatise" I stated that since the year 1914 no specified figures are available and that it is difficult to obtain such on account of the vast territory containing many small settlements which were visited by the epidemics. This aspect is again illustrated by Leger's study on Plague in Senegal during the years 1914-24 (11). In fact Leger, referring to the outbreak in the *cercle* of Dagana in 1917, says that here the bubonic type though predominating in the statistics was not alone met with. On the contrary it appears that numerous fatal cases of broncho-pneumonia, occurring simultaneously, have not been regarded as plague at all. Very rarely can some definite proof of the existence of pneumonic plague be gathered from Leger's material. For instance, dealing with an outbreak at Saint-Louis in 1918, which claimed 1,097 victims during the first nine months of the year, he states that pneumonic cases were much more frequent than bubonic ones. In 1919, when the town suffered again from plague from April to October, only 58 out of a total of 451 cases were pneumonic in nature.

Some problems connected with pneumonic plague in French West Africa will be dealt with in the second part of this article.

X. NIGERIA.

Though the information at hand is not complete there is little doubt that this colony has to be added to the areas visited by pneumonic plague. The figures as to the incidence of plague for the last few years are mainly culled from the League of Nations Reports:

Year.	*Cases.*	*Deaths.*	*Remarks.*
1922	0	0	—
1923	5	2	—
1924	414	344	—
1925	653	491	Disease spreading to the mainland (12).
1926	497	476	44.5% bubonic, 48.7% septicemic, 6.8% pneumonic (13).

The 1925 Report (14) refers to "a succession of hospital cases revealing the existence of a form of pneumonic plague in man where the sputum is the typical rusty sputum of ordinary pneumococcal pleuro-pneumonia, the plague bacilli being so scanty, or so transformed and whelmed among other micro-organisms as to debar diagnosis—except by inoculation of a susceptible animal." Some data as to the existence of

lung pest in 1926 are contained in the table. Connal and Paisley (15) state that in the period from October 1926—March 1927, the type incidence at Lagos was as follows:

	October.	*November.*	*December.*	*January.*	*February.*	*March.*	*Total.*
Bubonic	49	12	8	10	4	3	86
Pneumonic	31	4	6	6	1	4	47

The authors add that "there are no accurate statistics, naturally, as to the number of primary or pure pneumonic cases, but this was certainly considerable."

The rodents met with are *M. rattus* and *norvegicus*, *M. musculus*, *Cricetomys gambianus* and *Lemniscomys fasciatus*, while the ectoparasites found on them are *X. cheopis* and *brasiliensis* and *Laelaps echidinus* (14).

XI. CENTRAL AFRICA (KENYA).

The "Epidemiological Intelligence" of the League of Nations for 1925 says: "The case mortality appears to be extremely high and the proportion of septicemic and pneumonic cases is considerable." It must be remembered, however, that according to a statement of Hunter, quoted in the "Treatise" 'true inspiration pneumonia' is rare though 'pneumonic' cases are common. It would seem as if some fruitful studies upon plague pneumonia could be undertaken in this area.

XII. SOUTH AFRICA.

The information collected in the "Treatise" could be supplemented by consulting Mitchell's valuable study upon Plague in South Africa (16) as well as other new sources.

In regard to the early history of the disease, Mitchell states that the small outbreak at Queenstown in 1903 was mainly pneumonic in its early stage.

Events from the year 1914 onwards when wild rodents became responsible for the human outbreaks may now be summarised:

The first outbreak on record (Tarka and neighbouring districts, Cape Province) occurred in 1914 (July-October) and claimed 35 cases with 31 deaths. The first 26 cases were all in immediate contacts; all were of pneumonic type, and only one survived.

No detailed information is obtainable as to the character of the disease in the years following when the mortality was not so high as in 1914. Mitchell (17) gives for the years November 1914-June 1923, 212 plague cases with 130 deaths, of which roughly 75-90 per cent were bubonic. In the outbreaks recorded for the year ending June 30, 1924 (18)—total cases 372 with 325 deaths—12 per cent pneumonic cases were met with, all fatal. He adds that "in all of them there was a history of contact—often close contact including kissing—with a previous case of plague; indeed, it seems probable that kissing was the mode of infection in the majority."

During the following twelve months the type incidence was about 70 per cent bubonic, 20 per cent pneumonic, 7 per cent septicemic and 3 per cent mixed (bubonic-pneumonic) cases; the total was 112 with 68 deaths. Most of the 22 pneumonic ones occurred at De Aar (19).

Of the 71 cases (with 46 deaths) recorded in the Report for the year ended June 30, 1926, 10 per cent were pneumonic and 4 per cent mixed pneumonic and bubonic. Reference is made to instances where persons who had contracted bubonic plague through contact with rodents or through their fleas, passed the infection to others in the pneumonic form. Mitchell (16) mentions in addition that "in some instances, as in the Tarka outbreak of 1914, this type apparently developed directly as a result of infection carried by fleas from veld rodents; in four separate groups of native cases it appeared to have resulted from eating (probably after very imperfect cooking) the carcases of infected veld rodents."

XIII. MADAGASCAR.

Fairly complete figures were given in the "Treatise" up to August 31, 1925. These may now be brought up to date ((20):

a. Incidence of the disease in the Tananarive province, January 1—December, 1925. 1406 cases with 1243 deaths. All types.

b. Plague cases and deaths in Madagascar, 1926:

	Cases					*Deaths.*			
Month.	*Bubon.*	*Pneum.*	*Septic.*	*Total.*	*Month.*	*Bubon.*	*Pneum.*	*Septic.*	*Total.*
January	175	98	61	334	January	149	94	60	303
February	143	82	52	277	February	129	81	52	262
March	71	77	38	186	March	66	77	38	181
April	28	35	38	101	April	22	35	38	95
May	10	11	10	31	May	10	11	10	31
June	15	46	5	66	June	14	32	5	51
July	7	10	—	17	July	6	10	—	16
August	63	39	40	142	August	52	39	40	131
September	102	47	34	183	September	90	47	34	171
October	87	97	72	256	October	67	93	72	232
November	141	82	56	279	November	107	76	56	239
December	131	72	71	274	December	112	71	71	254
Total	973	696	477	2146	Total	824	666	476	1966

c. Plague cases and deaths in Madagascar, 1927:

	Cases				*Deaths*
Month.	*Bubon.*	*Pneum.*	*Septic.*	*Total.*	*(Total.)*
January	207	88	81	376	369
February	189	83	112	384	367
March	130	46	64	240	209
April	82	32	46	160	149
May	40	18	16	74	64
June	?	?	?	46	37
July	21	20	5	46	44
August	35	50	13	98	89
September	75	60	36	171	154
October	87	45	35	167	156
November	?	?	?	209	189
December	244	65	53	362	318
Grand Totals				2333	2185

As heretofore, the majority of general plague cases as well as pneumonic cases occurred in Tananarive province. However, other districts situated on the plateau, e.g. Ambositra and Antsirabo, suffered as well. The province of Itasy, which was infected from Tananarive in 1923, if not earlier, also shows a proportion of pneumonic cases. Tournier (21) dealing with the plague problem there, emphasizes that these rarely resulted in serious local outbreaks, ascribing such good result to prophylactic serum injections administered to the contacts. That secondary infections are, however, by no means absent in Itasy Province is best proved by an observation of Guillini (22): A patient admitted with plague pneumonia to the general ward of a hospital infected 4 others, who in turn passed the disease directly or indirectly to 21 persons.

Pneumonic plague continues to be absent or rare in the coastal districts. 5 cases of this type were met with in 1924 in the northern port of Diego Suarez besides 54 bubonic and 10 septicemic ones (23).

Girard, in an exhaustive study of the problems of lung pest in Madagascar (24) emphasizes that pneumonic cases, when arising in the coastal regions, remain sporadic, while the disease tends to spread widely on the high plateau if not nipped in the bud. He ascribes this difference partly to intrinsic causes (which will be dealt with later on) and partly to extrinsic factors, especially the climate. A table prepared by him may with advantage be reproduced:

a. COASTAL REGIONS (ABSENCE OF LUNG PEST).

	Dry and cold season.	*Warm and rainy season.*
Majunga	20-24° C.	30-36° C.
Diego-Suarez	21-29° C.	24-32° C.
Tamatave	17-28° C.	24-35° C.

b. HIGH PLATEAU (FREQUENT LUNG PEST).

Tananarive, Ambositra, Antsirabo, Miarinarivo	4-22° C.	15-30° C.

On the high plateau there is a marked difference between the day and night temperature; the humidity of the air is as high as on the coast. Pneumonic plague is comparatively frequent from April to November (cold season), the most dangerous time apparently being June and July.

Girard further states that the sparseness of the population in villages and the separation of single settlements by rice plantations are powerful factors in limiting the spread of the disease, while Tananarive Town is protected by an efficient sanitary organisation. On the other hand, the spread of the disease is fostered by overcrowding in dark and insanitary hovels and by the unhygienic habits of the people. Two specially dangerous features are:— (a) the custom of relatives and friends in collecting the expectoration of the sick in their hands (thus coming in close contact with the coughing sufferers) and (b) the anxiety of the natives to bury their dead in family vaults which leads to hiding of cases and opposition to sanitary measures. The result is a comparatively high plague mortality, the disease being undoubtedly one of the factors responsible for the deplorable excess of deaths over births in that island.

XIV. MESOPOTAMIA (IRAQ).

Further information as to the incidence of pneumonic plague is as follows :

a. Four cases occurred at Baghdad between April 20 and 23, 1928. Only the fourth, a railway coolie, was medically seen. It could be established, however, that it had been preceded by three others (one male and two females of the same family). *Post mortem* proved in all four cases the presence of plague pneumonia, while external buboes were absent. Rat plague was proved in the district. Strict measures prevented further spread of the disease (25).

b. Suspicion was aroused in the case of a female who lived in the Mehallah de Suk-el-Ghazil (east of Baghdad). She fell ill on May 18, 1928 and gave birth on the next day to a living full-term baby. On May 22 she was seen by a medical man who found a serious pneumonia. After death on May 22 the presence of *B. pestis* in the lungs was established (lung puncture only?) while buboes were absent. Striking features are that the patient did not cough blood and that the 6 contacts (husband and five children including the newborn) escaped infection. The report (26) states that this was probably a case of septicemic and not of pneumonic plague.

XV. SOUTH EAST RUSSIA.

Our previous figures are herewith supplemented :

Year.	*Foci.*	*Cases.*	*Short Description of Outbreaks.*
1924	15	26	(i) Bukeev District : 3 foci. April 26-June 11.10 c., 8 d. Bubon. (ii) Kalmuck District : (*a*) Kichkino. July 23-28. 1 fatal bubon. c. (*b*) Jasta Jandiko Mochashnogo. Nov. 23-Dec. 17. 15 c., 14 d. 14 pneum., 1 bubon. First, pneum., case infected from camel?
1925	39	255	(i) Ural Government : 2 foci. May 3-July 30. 12 c., 9 d. Bubon. (ii) Spring-summer outbreak on rt. shore of Volga : (*a*) Salski District. 6 foci. June 3-Sept. 19. 23 c., 11 d. Bubon. (*b*) Kalmuck Territory. 13 foci. May 19-Aug. 30. 85 c., 54 d. 66 bubon., 6 pneum., 12 bubon.-pneum. cases.

			(c) Stalingrad Government. 8 foci. May 25-Sept. 22. 80 c., 59 d. 68 bubon., 2 pneum., 6 bubon.-pneum., 3 sept., 1 intestinal. (iii) Dubovskii, North Caucasus Territory. Oct. 18-Nov. 9. 1 recovering bubon. c. (iv) Ural Government : (a) Bukeevski District. 3 foci. Oct. 14-Nov. 10. 9 fatal c. 1 bubon.-pneum., 8 pneumon. Mice-caused. (b) Gureevski District. 4 foci. Nov. 22-Dec. 21. 40 fatal c. Pneum. (v) Stalingrad Government : (a) Mateevski. Nov. 20-Dec. 12. 2 fatal bubon. c. Mice-caused. (b) Polovnikov. Dec. 21-Feb. 3. 3 c., 2 d. 2 bubon.. 1 pneum. Mice-caused.
1926	16	113	(i) Ural Government, Bukeevski District; (a) Aisharlik. Jan. 19-Feb. 26. 7 fatal pneum. c. Epizootic among mice. (b) Baikodam, Feb. 16-March 15. 2 foci. 28 fatal c., first bubon., others pneum. Epizootic among Mus musc. Wagneri and gerbilles. (c) Kerdere. May 9-June 8. 46 fatal c. 45 pneum., 1 bubon.-pneum. Epizootic among sand mice (gerbilles?). (d) Asbergen. June 1-19. 3 fatal bubon. c. Epizootic among mice and jerboas. (ii) North Caucasus Territory, Salski District. June-October. 7 foci. 9 recovering bubon. c. Epizootic among susliks. (iii) Stalingrad Govt. Stariza. July. 1 fatal septic. c. Suslik-caused. (iv) Astrakhan Govt. : (a) Asau. June 30-July 14. 9 fatal c. 8 pneum., 1 bubon.-pneum. Jerboa-caused. (b) Krasnojarski District. Nov. 4-Dec. 17. 5 c., 4 d. Bubon. Diseased camel and mice epizootic.

			(v) Ural Govt. Gureevski District. Nov. 23-Dec. 21. 5 fatal bubon. c. Mice-caused.
1927	5	88	(i) Ural Government, Bukeevski District: (*a*) Shutkara. Jan. 7-23. 46 fatal pneum. c. (*b*) Kos-Kulak Jan. 7-Feb. 15. 13 fatal c. 1 bubon., 12 pneum. Epizootic among mice and jerboas. (*c*) Kuanish-Kurgo. Feb. 4-17. 13 fatal pneum. c. Imported from (b). (*d*) Kok-Terek. May 5-22. 5 fatal c. 4 pneum., 1 bubon.-pneum. Epizootic among sand mice. (*e*) Chankara. April 14-June 1. 11 c., 9 d. 10 bubon., 1 intestinal. Camel-caused?

N.B.—The bubo distribution in 49 purely bubonic cases observed from October, 1925 until May, 1927, was as follows:

Undetermined	6
Femoral	6
Inguinal	11
Axillary	21
Cervical	4
Submaxillary	1
Total	49 (27).

XVI. CENTRAL ASIA (TRANSCASPIA).

A new plague focus has lately been detected in the Mangishlakski Steppes of Transcaspia (28). This vast territory is inhabited by nomadic Kirghese who lead a hard and unhygienic life and suffer from different diseases including respiratory ones.

The history of the two outbreaks observed in 1926 is as follows:

(i) About the middle of August near the well Kul-Kara on the Ust-Urt Plateau a Kirghese girl, aet. 5, fell sick and died after 8 days' illness with axillary bubo and secondary pneumonia. The infection soon spread among relatives and friends and in one month there succumbed in three settlements (consisting of 13 *kibitkas* and inhabited by 72 people) 41 persons, evidently all from primary plague pneumonia. That the plague did not spread further is due to the fact that the people near the focus, alarmed by the highly fatal character of the disease, kept away from the afflicted families many of which were wiped out.

An expedition arriving in October under Nikanoroff's leadership confirmed the diagnosis through autopsies on the first and two other victims

and also microscopical examinations. While it was too late to obtain good cultures the plague nature of the outbreak was established by observations upon an epizootic raging among sand mice, *Rhombomys opimus Licht.* (see Wild Rodents article).

The second outbreak occurred further westwards near the Buzashi peninsula in October-December, 1926. The varieties were as follows :

Bubonic (inguinal)	6 (1 with sec. pneum.)
Bubonic (axillary)	10 (5 with sec. pneum.)
Primary pneumonic	8
Total	24 (20 deaths).

Ignatiev states that of these cases 20 were traceable to the epizootic among the sand mice, one had evidently been caused by contact with plague-infected hares, while 6 apparently arose from the killing and dissecting of diseased camels. Here again the people soon realised the seriousness of the disease, and hurriedly left the infected foci.

Nikanoroff thinks that infection was imported into this area from the Ak-Kamish district, infected rodents being transported by the frequent caravans.

XVII. TRANSBAIKALIA.

In my book I have included a complete record of the plague outbreaks in this endemic area up to September, 1925. Since that time plague has caused comparatively little havoc. The following outbreaks —all of tarabagan origin—were recorded in 1926 :

Date.	*Locality.*	*No. and character of cases.*
Aug. 23	Haranor	2, fatal, bubonic (axill.)
Sept. 5	Near Manchouli	1, fatal, bubonic (?)
Sept. 9	Borzia	1, fatal, bubonic (axill.)
Sept. 18	Oloviannaya	2, fatal, septicemic.
		Total 6.

Rigid measures were taken in each instance and no further spread of infection was observed.

No case was recorded in the year 1927.

Early in August, 1928. one bubonic case was noted at Dauria. Particulars are not yet to hand.

XVIII. MONGOLIA.

1. *Outer Mongolia.*

In 1926 wide-spread alarm was caused through exaggerated news of a big pneumonic outbreak in Outer Mongolia. The real facts are as follows :

The focus was situated 35-50 miles from Chechan Han. According to Skorodumoff (29) who wrote a short paper on this outbreak, during the summer dying tarabagans had been observed in the district; this was, however, ascribed to scarcity of food on account of the prevailing dryness. The first human case occurred on October 10, resulting in the infection of 23 people (22 pneumonic and one bubonic who recovered). The epidemic involved six localities and lasted about 5 weeks.

The first victim was a girl, age 23, a shepherdess in the locality Chulutoi. The story was that she tried to catch an apparently sick tarabagan, chasing it into a hole and closing this with her gown. She then hurried home to report the matter; returning to the spot she donned her gown and was probably infected through fleas which had taken refuge in it. It is certain that soon after this encounter she fell ill, suffering from fever, headache and swellings in the arm-pit, neck and groin. She died after six days of illness. The patient's mother-in-law, father and brother were successively infected.

Then two Lamas from the adjacent monastery were summoned to treat the sick and conduct the ceremonies. One of these developed symptoms of pneumonic plague after returning to the monastery. He was visited by relatives and nursed by the pupils who also became infected. The relatives conveyed the disease to their two jurtes situated on the right bank of the Kerulen river. The inmates of these jurtes all died. The neighbours who recognised the nature of the malady and avoided the sick escaped, thus ending the epidemic.

An expedition sent by the Mongolian Government arrived afterwards and found eight corpses. Material was taken for bacteriological examination and the plague nature of the epidemic was confirmed.

It is possible that outbreaks like the above (easily detected because of occurrence near a trade route) arise from time to time in Mongolia as well as other parts of Central Asia, but remain unnoticed, being cut short by adequate though crude measures taken by the natives. On the other hand, we must consider that the Mongols are usually very careful in regard to suspicious tarabagans. As again confirmed by Skorodumoff their hunters shoot the tarabagans so that they can recognise diseased animals to which they give a wide berth. Thus the unjudicious action of the shepherdess was an exception rather than the rule.

2. *Inner Mongolia.*

Though plague outbreaks evidently occur from time to time in this territory, little information is procurable. In the past we had been able merely to establish that a suspicious "winter sickness" prevailed periodically in the Ordos country and other parts of Inner Mongolia. Plague was undoubtedly present in the Patsebolong district of South Mongolia in 1917, these parts being the main distributing centers, if not the starting point of the 1917-1918 Shansi epidemic. During the following years up to 1927 no information was forthcoming in regard to Inner Mongolia. Probably plague was in reality quiescent because there is much reason to assume that the disease displays a periodicity resulting about every tenth year in manifest outbreaks.

In autumn, 1927, an outbreak of pneumonic plague was reported to have occurred near Payintala (now called Tungliao) formerly eastern part of Inner Mongolia. Payintala is a town of about 20,000 inhabitants and the terminus of a branch railway which joins the South Manchurian Railway system at Ssu Ping Kai.

The evidence procurable at the time of the outbreak was scanty, the figures obtained from different sources having evidently been much exaggerated. We estimated the real number of deaths at the time to be about 95. This is quite well in accord with the data given in a Japanese Report by South Manchurian Railway doctors (30) which contains the following tabulation :

Date.	*Locality.*	*Deaths reported by population.*	*Verified number of deaths.*
Late Aug.	Near Tan-gol.	200	30
Sept.-Oct.	Nemugol, Olebugol, Uranga & Consobon	300	40
Late Sept.	Tsenchiawopan.	5	5
Early Oct.	Chien-Chia-Tien.	—	5
Early Oct.	Village 2 *li* east of Kai-lao.	—	8
Early Oct.	Anarkamio.	—	5
Early Nov.	Wanleoho.	—	18
			Total 111

Suspicious bacilli were seen in films of the blood, but no positive cultures or successful animal tests seem to have been obtained.

The outbreak was apparently connected with a visit of the Pan-San Lama paid in August to the Tan-gol Temple. When this priest, accompanied by a retinue of lamas and followers arrived, the people flocked together from near and far to worship, the daily gathering being estimated at 10,000 persons. The Japanese Report leaves open the question whether the infection was imported by pilgrims from their home or whether the overcrowding of the region gave an impetus to the disease quietly smouldering at the spot.

In August-September 1928 another outbreak was reported in the Tungliao rgion. The cases seen were mostly bubonic in character. Further details are furnished in the article on 'Plague Investigations at Tung Liao.' (Vol. VI).

XIX. PNEUMONIC CASES AND OUTBREAKS ON BOARD SHIP.

The number of outbreaks of this nature reported in the "Treatise" may be augmented by three instances which have recently come to my knowledge :

1. *S. S. Bega.*—This vessel was employed in the coastwise trade, returning at regular intervals to Sydney. As subsequent investigations showed she was kept as free from rats as possible by periodical fumigations. While taking aboard produce besides empty sacks, crates, etc., she was apt to harbour temporarily plague rats. It is probable that the victim infected on board had the misfortune to come into touch with such. Inquiries elicited the fact that less than a week before his attack he had picked up a dead rat with his hands and thrown it overboard. Soon afterwards he removed another rat from the engine-room, handling it this time with tongs, as he had been scolded on the first occasion for his imprudence. Though he left Sydney in good health on June 15, 1906, he fell ill the next day, stayed off and reached his home in

Balmein, a suburb of Sydney, on the 19th. The medical man visiting him then diagnosed slight catarrh; the next day the patient seemed to have recovered. Late at night on June 22 he summoned another practitioner who recognised the presence of pneumonia; the pulse and general condition seemed good. The next morning the doctor found the man collapsed and cyanotic; death occurred soon afterwards.

The patient had lived in a comfortable house which he had shared with his wife and nine children. The former, who had nursed her husband, fell ill three days after his death and died 48 hours later. Her medical attendant suspected plague-pneumonia and reported the case. Autopsy confirmed this diagnosis. Another woman who had repeatedly visited the first victim, fell sick on June 28 and died after two days of pneumonic plague confirmed by *post mortem*. Her husband and four children, who had been from time to time in her bedroom before she was hospitalised 12 hours before death, escaped infection as did the other contacts of the first victim. These as well as visitors were protected by subcutaneous serum injections. The Report (6) states also that all three patients had very little cough, hence the escape of the 29 contacts.

It can be seen that the course of the disease in the first patient was atypical and that its nature could not be definitely established. It remains an open question whether he suffered from primary or secondary pneumonia.

2. *S. S. Armedale Castle.*—Much alarm was caused in Capetown when on April 8, 1927, a case suspicious of plague was reported on board the outward bound mailboat, the *Armedale Castle,* due to sail for Southampton the same afternoon. The patient, a member of the crew, who had fallen ill on April 6, was admitted to hospital the next day under the diagnosis of *pneumonia.* He died on same day. Smears made at autopsy revealed the presence of suspicious bacilli and a presumptive diagnosis of plague was made (which was afterwards confirmed by animal experiments). Rigorous measures were at once taken and no further spread took place either in the hospital or on board the steamer, which was allowed to sail after some delay (31). If it is remembered that plague had not been known to exist in any of the coastal ports visited by the steamer one must really admire the alertness with which this case was detected and dealt with. I hope to obtain fuller details about the *post mortem* results for future publication.

3. *Steamer in South American trade.*

The correspondent of the Journal American Medical Association at Buenos Aires writes under date of May 28, 1928 (32) that the master of a boat became sick and died in Uruguay, with a diagnosis of *bronchopneumonia.* Upon arrival of the vessel at Buenos Aires three sailors became ill, and it was found that they suffered from plague pneumonia. Another sailor who left the boat, and a woman who had nursed the three aforementioned hands also died. Sanitary measures were taken and no further spread took place.

The instances described in 1 and 3 as well as others mentioned previously well illustrate the potential danger of such outbreaks, which might easily be overlooked and cause considerable havoc in a suitable climate and environment.

C. EPIDEMIOLOGY AND INFECTIVITY OF PNEUMONIC PLAGUE.

1. "ORIGINALLY PNEUMONIC" PLAGUE CASES AND OUTBREAKS.

In previous pages some instances have been quoted where pneumonic cases are believed to have been the result not of infection from a human being suffering from bubonic plague with secondary lung involvement but of more or less direct contact with infected rodents. I have to discuss now some recent publications dealing with this matter in a more general manner.

1. Connal and Paisley (15) in the course of routine examination of rats captured at Lagos (Nigeria) were struck by the temporary frequency of hemorrhage in the intestinal tract of the animals. In the bloody intestinal contents *B. pestis* was invariably found and sometimes present in great numbers. Ulcerative processes were absent, the condition apparently being due to "a massive diapedesis from the walls of the intestinal tract." The authors state that the nature of the houses in Lagos and the custom of sweeping every morning the accumulated dust into the street would facilitate an aerial infection through rat feces which might have been deposited and become dried on the mud floors. The sweeping is usually done with a short broom in a stooping attitude. Pneumonic cases were frequent at the time when rats with such intestinal changes were found. Moreover, the authors performed some experiments upon monkeys in order to show that such infection through rat feces is possible. Thus :

Mode of infection :	*No. of monkeys :*	*Results :*
a. Bloody intestinal contents mixed with sterile dust introduced intratracheally.	5	4 positive 1 negative
b. Infective material applied as dry spray over open mouth and nostrils.	3	All negative
c. Infective material applied as liquid spray.	2	1 positive 1 negative.

After reading the protocol I am not convinced that all the succumbing animals suffered from primary plague pneumonia. One had evidently septicemic symptoms while in some others gland involvement seemed to have been present in addition to plague pneumonia. This point, however, is perhaps not of paramount importance because, even if infected dust should lead to secondary lung involvement only, it might be indirectly responsible for the rise of primary pneumonic plague. However, the evidence supplied by the above experiments is not particularly strong if we disregard the highly artificial method of direct tracheal infection. The experiments with dry dust were altogether negative. This

is in accord with numerous negative epidemiological observations made by us in Manchuria and elsewhere. It might be said in general that a mediate infection, though possible under exceptional circumstances, is certainly not the rule in lung pest. Hence it would be wrong to assume that an infection from contaminated objects plays any considerable role in the genesis of pneumonic plague outbreaks.(1)

2. The idea that pneumonic plague may arise *de novo* directly from the rodents has found a zealous advocate in Gill. He states (33) that his own observations "have shown that the primary case in pneumonic plague epidemics may, and often does, occur in the absence of bubonic plague (but usually in the presence of a rat epizootic), and it is therefore inferred that a rat epizootic may be the starting point of an epidemic of pneumonic plague."

Expounding this theory in a more general manner Gill suggests (34) that "Wu Lien Teh has recorded many instances of outbreaks of pneumonic plague as result apparently of infection derived directly from plague-infected tarabagans." Because of this and other instances on record where primary lung pest has been occasioned by the accidental inhalation of plague bacilli (? laboratory cases), he concludes "that primary pneumonic plague may be due to infection derived directly from the mammalian host."

Gills thinks apparently that the opposite view ascribing an intermediate role to bubonic cases with secondary lung manifestations is mainly dependent upon observations made by Russian physicians and based merely upon the fact that pneumonic plague outbreaks are preceded by bubonic plague manifestations.

"It is now clear", he continues, "that at high latitudes where the plague season occurs in the autumn, outbreaks of bubonic plague would be expected to precede outbreaks of pneumonic plague, which latter disease, for reasons already stated, would not be expected to assume epidemic proportions until the onset of winter.

On the other hand, in the Punjab, where for climatic reasons, the bubonic plague season occurs in the spring, outbreaks of pneumonic plague, since they necessarily exhibit a winter periodicity, would be expected to precede outbreaks of bubonic plague.

In the light of these facts it is possible to reconcile the discordant results of epidemiological observations made by Russian and Indian epidemiologists in regard to the time relationship of epidemics of bubonic and pneumonic plague; and it is also held that, because outbreaks of pneumonic plague sometimes arise during the course of epidemics of bubonic plague, it does not necessarily follow that the first case of primary pneumonic plague is due to infection derived from a case of bubonic plague with pneumonic complications."

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(1) Connal and Paisley's observations are of great interest in another direction. It is possible that one of the factors which help to mould pneumonic outbreaks in areas with rat-caused plague is the peculiar nature of the epizootics prevailing. This might result from importation of a highly virulent strain or immigration of susceptible rats and the like. One wonders if such peculiar conditions did not exist at the time when intestinal hemorrhages were observed in the Lagos rats with simultaneous pneumonic plague.

In reply to these statements of Gill I must mention the following:

a. I have never said that outbreaks of pneumonic plague are due to infection derived *directly* from tarabagans. On the contrary, I was at great pains to prove that—apart from two instances one of which was certainly not well investigated—the origin of pneumonic plague outbreaks in Transbaikalia and Manchuria could always be *definitely traced* to human bubonic cases with secondary lung involvement. Early in the 1920-21 epidemic I was fortunate enough to observe this gradual transition from the bubonic to the pneumonic plague type at Hailar with my own eyes.

b. The Russian physicians likewise have not been content with the mere notion that pneumonic plague outbreaks are preceded by bubonic manifestations, but have taken the trouble to verify in an exact manner the nature of the first cases in pneumonic outbreaks by exhuming and dissecting the victims. As discussed in my "Treatise" they repeatedly satisfied themselves that these early cases, which were in causal connection with the later pneumonic ones, had bubonic plague with secondary lung involvement. Furthermore it was often established that the pneumonic outbreaks were not preceded by protracted bubonic outbreaks, but that the change of type took place very quickly so that the second or third, victim had already developed primary lung pest.

c. Similar observations upon a very short "bubonic stage" have been made as well in Egypt by Petrie, who thus adds his weighty testimony as to the causal connection between bubonic and pneumonic plague.

d. Girard, in a recent study upon lung pest in Madagascar, also concludes that bubonic cases actually intervene between rat plague on one hand and pneumonic on the other. There are outbreaks which *seem* purely pneumonic, but it must be remembered how difficult it is to trace these back to the really first case. Certain it is that bubonic cases are more frequent on the Madagascar Plateau than was believed even a short time ago.[2]

e. Thus though I am far from denying that cases of "originally pneumonic" plague may exist, they are exceedingly rare, and I can but repeat that as a rule the outbreaks do not arise *de novo*.

II. THE THEORIES OF NORMAN WHITE AND NICOLLE & GOBERT.

I have again entered into a detailed discussion of this question because it is not only of great interest *per se* but closely connected with other important problems, notably as to whether pneumonic plague is a disease *sui generis* or not. It will be remembered that Norman White believes in a fundamental difference between the bubonic and pneumonic types. As I stated when discussing his theory, perhaps the most convincing proof against it is the fact that an aetiological connection exists

(2) Girard states at the same time that the close watch now kept for the presence of buboes has shown that purely septicemic cases are rarer than it was assumed beforehand. This confirms our opinion that septicemic cases do not play such an important role in the genesis of pneumonic plague outbreaks as has formerly been believed in Madagascar (35).

between the different forms of plague. I am glad to note that Nikanoroff (36) now confirms this opinion of mine.

We must next pass to the theory of Nicolle and Gobert who argued that the filtrable virus of influenza might act together with the *B. pestis* in causing primary plague pneumonia, an aspect which has been urged again by Dopter and Jausion (37). As I have stated before epidemiological considerations speak strongly against this idea. I am able to furnish new evidence in this direction :

a. Graham (1) could not obtain any direct proof of the relationship between influenza and pneumonic plague in India.

b. Leger, in his study of plague in French West Africa (11), states that influenza did not play a role in the causation of lung pest as shown by direct observations.

c. Girard, though himself rather inclined to believe in the importance of a mixed infection in the genesis of pneumonic plague, also concludes that his findings have not enabled him "to ascribe an important and constant role to the influenza virus in the epidemiology of lung pest in Madagascar."

III. GIRARD'S VIEWS UPON PNEUMONIC PLAGUE IN MADAGASCAR.

Aside from some questions which are more or less agreed upon, Girard in his observations upon pneumonic plague in Madagascar makes the following statements :

1. The first case in a pneumonic epidemic is often found to be different from the subsequent ones inasmuch as it lasts considerably longer and has for some days an uncharacteristic expectoration in which other microorganisms, specially pneumococci, greatly outnumber the *B. pestis*. Afterwards plague bacilli come gradually to the foreground, while the sputum becomes more and more bloody and finally assumes the aspect typical of plague pneumonia. Girard concludes that these early cases must be considered as having secondary lung involvement ("plague with non-manifest or non-diagnosed buboes").

2. While persons infected from such early cases fall sick not sooner than 4-5 days after the demise of the first victims, the length of the incubation period during a pneumonic outbreak varies from 1 to 3 days. Girard concludes from this that—

   i. the incubation tends to become shorter in the course of the outbreak;

   ii. infection takes place only when plague bacilli are present in the sputum in pure culture. Hence the long interval between the first and early secondary cases.

3. An explanation of the different behaviour of the first and the later cases lies in the different bacteriological findings. As already said the pneumococcus is constantly found in the early stage of the first cases besides a few plague bacilli. In fact Girard believes himself entitled to introduce an *etape pneumococcique* (pneumococcal stage) which precedes the final *etape pesteuse* (plague stage). During the course of an outbreak only the B. *pestis* is found as a rule in the case of natives, while Eur-

opeans, even when infected at the height of an outbreak pass through a "pneumococcal stage" and show a longer course of the disease than the natives who at the same time perish quickly.

Girard concludes that a causal connection exists between the presence of pneumococci and the development of pneumonic features. These microorganisms "prepare the soil" for the plague bacillus and—while delaying its development—they give to it that exalted virulence which is necessary for the rise of a "massive" outbreak.

4. During the course of pneumonic epidemics one meets besides frankly pneumonic cases those without cough or expectoration, though after the death of the sufferers the lungs are found to be full of plague bacilli. Such patients are little or not infective at all.

5. Girard records two instances where the lungs of patients were punctured two hours after onset of the first symptoms (fever and a little dyspnoea). On both occasions plague bacilli were plentiful in the smears. (N.B. No simultaneous blood cultures were made). The author refers also to previous observations of his showing that in pneumonic plague cases the lungs alone (or merely one lung) may contain plague bacilli, while the liver is free. He concludes therefore that:

i. the lungs are attacked as soon as the first symptoms of illness appear;

ii. a bacteremia may be absent even in the terminal stages.

Nevertheless, he leaves open the question whether pneumonic plague is preceded by a septicemic phase or not.

Many of the above statements coincide with or supplement our findings. My studies have led one to the conclusion that the virulence of *B. pestis* increases during the course of a pneumonic epidemic as manifested by the occurrence of "septicemic" or better *pulmonary* cases, which though very fatally infected, are little infective owing to the absence of cough and expectoration. This statement is confirmed by Girard who furthermore asserts that the incubation period becomes shorter at the height of an epidemic—a point about which I was not quite sure. Likewise, Girard's findings regarding the early presence of the causative organisms in the lungs and the occasional absence of a bacteremia tend to support my opinion that infection in pneumonic plague occurs through the deep respiratory tract.

On the other hand, I cannot quite agree with Girard's views as to the importance of the pneumococcus in the etiology of pneumonic plague. It seems to me that the facts furnished by him in this direction could be explained in another and more satisfactory way. Our observations in Harbin have shown that the manifest stage of lung pest with severe cough and copious expectoration of plague bacilli was preceded by a latent period in which cough was practically absent, and only a few if any plague bacilli were found in the scanty and uncharacteristic sputum, while other microorganisms abounded. Gradually the plague bacilli grew in numbers while the other bacteria decreased and finally disappeared. However, this evolution seems due simply to the fact that some time is required for the specific lung process to evolve while the other bacteria play an accidental and not a causative role.

I am fully aware that our experiences made in Manchuria cannot at once be applied to conditions prevailing on the Madagascar plateau for three reasons :

i. The inhabitants of the latter area are very susceptible to pneumococcal infections while our Chinese are rather refractory to them.
ii. A mixed infection played no role in Manchuria, while in Madagascar such true cases evidently occurred as proved by one *post mortem* protocol furnished by Girard.
iii. Girard ascribes considerable importance to the pneumococcus not so much in primary pneumonic plague as in the early cases with secondary lung involvement.

I think, however, that not too much stress should be laid upon these differences. It might be argued that cases with secondary lung involvement might follow laws other than those of true plague pneumonia. But Girard is unable to draw any sharp line between the early and later cases so far as the presence of a "pneumococcal stage" is concerned. He states that such examples are rarely met with in natives during an epidemic, but are the rule among Europeans infected eventually with primary pneumonic plague. Thus, without denying that cases of true mixed infection do occur in Madagascar, we may ask whether in other instances the pneumococci are not an accidental feature. Naturally, their presence in the early cases with secondary lung involvement and slow evolution of the disease will be more conspicuous than in the later purely pneumonic ones, the majority of which run a quick course. The fact that sick Europeans (infected with primary pneumonic plague in the course of epidemics) display an evolution of the disease similar to that of the early native cases seems puzzling to Girard. I should say that this could largely be explained by the fact that the course of the disease is prolonged in their case by energetic serum administration which is sometimes started before the onset of symptoms.

For these and similar considerations I cannot believe that the pneumococcus plays any important role even in Madagascar plague. That lung pest occurs frequently on the plateau and rarely on the coast is in my opinion due not to the difference in susceptibility to *pneumococcal* infections but to the different resistance against *pulmonary infections in general*. Therefore it is easy on the plateau for the *B. pestis* to invade the lungs of the sufferers without any pace-maker.

Finally I have to deal with Girard's assumption that the patients are infectious only when their expectorations contain the *B. pestis* in pure culture, whereas earlier in the disease, when other micro-organisms are present, they fail to infect others. While admitting that in general the infectivity of the patients increases with the course of the disease, I dispute Girard's explanation of this point. That the sufferers become more dangerous is primarily due to continuous increase of both the cough and the number of plague bacilli produced by it. *Vice-versa*, early in the second stage coughing is scarce with low concentration of plague bacilli in the sputum. For this reason, and not on account of the accidental presence of other microorganisms, the patients are less infectious.

And if, on account of early detection and prompt sanitary measures or the rapid death of the patient, his contact with relatives and friends is cut short, secondary infections are less likely to occur than when prolonged contact up to the death of the patient takes place.

I have been obliged, much against my inclination, to concentrate upon our points of disagreement, which after all are far fewer than those where Girard and I feel at one. I wish therefore to conclude this review with a full appreciation of my French colleague's work, which has shed much light upon the problem I have at heart. If such profound studies could be undertaken in other plague areas, our knowledge of the infection would be vastly improved.

IV. PNEUMONIC PLAGUE IN FRENCH WEST AFRICA.

Dealing with the problem of lung pest in French West Africa I noted two pecularities :

i. A considerable number of recoveries was recorded;
ii. The sufferers were supposed to be less infectious than in other plague regions.

While some observers were more or less convinced of the presence of primary pneumonic plague, Heckenroth (38) maintained that this type was rampant only in 1914-15. Later, cases with secondary pneumonia prevailed, the infectivity and mortality of which were naturally low. These problems have received further attention. Dujardin-Beaumetz (39) believes such non-contagious and benign cases to be due to a secondary invasion of the lungs. It is possible, he adds, that the presence of other microorganisms, notably the pneumococcus, might be responsible for the non-infectivity of such patients.

Leger, in a recent study upon plague in French West Africa (11), also touches upon this problem but is, as heretofore, not very explicit. He again emphasizes the absence of hospital infections though the staff take no special precautions and patients sometimes remain for some time in the general wards. He admits, however, that the infectivity of the outbreaks was not always the same and that overcrowding and unhygienic conditions in the native dwellings appeared to play an important role in spreading the disease. Though not in favour of Heckenroth's (and Dujardin-Beaumetz') theory, Leger is unable to assert that the cases under discussion are primarily pneumonic in nature; they cannot be called 'secondary' because manifest buboes are absent; on the other hand they show neither the quick evolution nor the invariable fatality so typical of the primary form.

Though it is difficult for an outsider to reach a definite conclusion, one cannot help expressing that Leger's statements are somewhat vague. As a further proof of the opinion that some observers in French West Africa do not sharply discriminate between primary and secondary pneumonic plague, I may quote from Leger's recent paper. Speaking of an atypical case of lung pest he notes that "the clinical findings did not correspond to those ordinarily observed in plague broncho-pneumonia since no tender lymph-gland was present" (Le tableau clinique ne cadrant avec ce qui est d'ordinaire observé dans la broncho-pneumonie

pesteuse, comme il n'y a aucun ganglion sensible. . .''). The presence of tender external lymph glands certainly does not coincide with the classical features of primary pneumonic plague which Leger invokes a few pages later on!

To the unbiassed, it would seem therefore that more reliance should be placed upon Heckenroth's and Dujardin-Beaumetz' clear-cut views. It is, however, an open question if the pneumococcus is instrumental in rendering these cases with secondary pneumonia non-infective. As already discussed its presence may merely indicate a prolonged course of the disease. The real explanation for the non-infectivity of such patients is perhaps the scarcity of plague bacilli in the expectorations rather than the abundance of pneumococci.

V. SUSCEPTIBILITY TO PNEUMONIC PLAGUE INFECTION IN DIFFERENT RACES.

Our observations in Manchuria have not yielded any evidence of racial immunity against primary pneumonic plague. Russians, French, Britishers, Koreans and Japanese have succumbed as well as the Chinese, whenever exposed to infection. Similar observations have been made in other plague areas. Girard, referring to this aspect, ascribes a slight resistance against plague infection to Europeans, but adds that "if they live under the same conditions as the *Malgaches*, they pay a similar tribute to the scourge."

On the other hand, I have quoted in my "Treatise" certain observations suggesting a special susceptibility to pneumonic plague infection in certain races. Mitchell, dealing with the plague problem in South Africa in 1927 (16) again mentions this point, saying that "Asiatics appear to have the lowest resistance to the disease and to be specially liable to develop it in a virulent pneumonic or septicemic form. South African natives and mixed-coloured or Eurafricans occupy intermediate positions in this respect between Europeans and Asiatics."

While I cannot believe that a racial immunity against primary pneumonic plague exists, these observations deserve attention. A lessened resistance to plague or a tendency to pulmonary disease in general, as present in certain races, may facilitate localisation of the *B. pestis* in the lungs leading to secondary pneumonia. Now if unhygienic conditions, which favour the spread of primary pneumonic plague, are present among such people (as is often the case) the disease is likely to spread. Members of other races living in the same area may escape because of lack of the initial spark or of suitable fuel.

VI. THE MODE OF INFECTION IN PRIMARY PNEUMONIC PLAGUE.

1. When dealing with this problem I have stressed the fact that infection in primary pneumonic plague is in the overwhelming majority of cases due to a direct and close contact with sufferers. This aspect which is of paramount theoretical and practical importance, has found recent confirmation:

Girard after exhaustive studies in Madagascar comes to the conclusion that a contagion occurs only within narrow limits and is easy to prevent. Though praising the efficiency of masks and goggles he says that—especially in the early cases of an epidemic—simple precautions instinctively taken by medical men (e.g. avoiding the direct range of cough and turning the head of the patient to the side when examining the front of his chest) are sufficient to prevent infection.

2. Nikanoroff, in his 1928 study upon plague in South-Russia (27), states that several instances are on record of persons falling sick after a short contact with pneumonic plague victims. In the "Treatise" I have also quoted such instances and stressed the necessity of taking all precautions even when approaching a pneumonic plague patient for a short while only. On the other hand, I have come to the conclusion that such cases due to a passing contact with patients are the exception rather than the rule.(3)

Of great interest are Nikanoroff's remarks upon the role of inanimate objects in the spread of the disease. Formerly, when little was known of the true causes of endemicity in South Russia, undue stress was laid upon this source of transmission. Now less and less importance is ascribed to it. The plague bacilli expectorated by pneumonic plague patients may find a suitable medium for prolonged survival on the *kochma* (a thick woollen material largely used by the Kirghese). While a direct infection from such contaminated material would be difficult of explanation, Nikanoroff considers it possible that mice might contract the disease from this source and in their turn pass it to human beings.

These remarks are of considerable interest as this might be one of the avenues through which infection passes to the rodents during a pneumonic outbreak. However, it is unlikely that such secondary infection is common. Instructive information in this direction is available now from Madagascar. Formerly it was believed that the second (prevalently bubonic) outbreak taking place on the plateau of Madagascar in 1921-22 was caused by rats, which had become infected during the first (mainly pneumonic) epidemic occurring in June-July, 1921. The latter was said to be due to the arrival of an infected person from the coast. Now Girard believes that the infection had existed among the rats of the plateau in June already and was responsible for the first outbreak. Girard aptly remarks that a role of the rats may easily remain unperceived in a pneumonic epidemic since it is restricted to the earliest cases.

(3) Confirmation of this point is found in Bulstrode's Report on the 1910 outbreak in England which was not accessible to me when compiling the "Treatise". This careful observer says that "in both of the invaded houses there were several persons—mostly children—who, before the nature of the disease was suspected, must have had opportunities, although perhaps minor and transient, of becoming infected, but who, nevertheless, remained well. "Bulstrode concludes that "it has to be observed that more or less continuous and intimate association with the patient, and this by persons relatively ignorant of the importance of taking precautions, seems to have been necessary for infection."

3. Discussing the limited importance of a mediate infection in pneumonic plague I mentioned among other things that the personnel, employed to disinfect the premises where victims had been, usually escaped infection. A list of exceptions to this rule was at the same time recorded. The number of these exceptional cases is now augmented by Girard who records (40) one suspicious and one doubtful instance. He underlines the rarity of such cases and adds that no member of the squads charged with the burial of victims of plague pneumonia contracted the disease. It must be kept in mind that the whole personnel adopted sufficient precautions, including the wearing of masks and goggles.

D. PATHOLOGY OF PNEUMONIC PLAGUE.

Both in my "Treatise" and in the "Problems of Pneumonic Plague" embodied in this Report I have dealt with the question as to where pneumonic plague infection enters the system. I have stated that—whilst the majority of investigators including myself believe that the virus directly enters the deeper parts of the respiratory tract—some maintain that the plague bacilli invade the tonsils and upper portion of the respiratory channel, thus reaching the lungs indirectly through the blood stream. Some fresh experimental evidence bearing upon this controversy is now available:

a. Semikoz, Kotelnikov and Bessonova read at the 1927 Russian Anti-Plague Conference three papers (41) dealing with the plague infection of guinea-pigs by various routes including the conjunctiva, mouth and nares, as well as by inhalation; the latter was administered with the aid of a special apparatus described by Semikoz (42).

Being interested in other matters the authors paid little attention to the problems of pneumonic plague. Apparently they are satisfied that in this type infection enters the lungs directly. They quote, for instance, the case of a guinea-pig killed 24 hours after inhalation, where plague bacilli were found only in the lungs ("the portal of entry") but were absent from the blood.

b. At the same Conference Korobkova read a paper dealing with plague infection by mouth (43). This author infected (besides some guinea-pigs and rats) 102 susliks by dropping from a pipette thick emulsions of plague cultures (rarely plague organs) into the mouth of the animals. Many of the latter were killed with chloroform at intervals varying from 20 minutes to 48 hours after infection while the rest were left to succumb spontaneously. Short *post mortem* protocols are given of the last-mentioned animals only. Histological investigations were apparently not undertaken, reliance being chiefly placed by Korobkova upon the bacteriological findings, which are thus summarised:

PERCENTAGE OF POSITIVE RESULTS IN ORGANS OF ANIMALS KILLED AT DIFFERENT TIMES AFTER INFECTION BY MOUTH.

No. of animals	Time after infection	Cervic. glands:	Trachea:	Lungs:	Blood:	Stomach:	Intestines:	Remarks:
19	20 min–1 hour	58	68	58	5	42	21	Killed with chloroform
19	1–3 hrs.	42	58	63	21	15	5	"
13	3–6 hrs.	54,5	69	77	65	0	0	"
25	6–24 hrs	73	75	80	72	0	0	"
19	24–48 hrs.	84	84	84	84	5	15	"
24	72 hrs. or later	100	100	100	100	0	18.5	Dissected after spontan. death

The author concludes among other things that "after introduction of plague infection in the mouth simultaneous invasion of many organs occurs, especially a direct transportation of the bacilli to the upper respiratory tract followed by quickly evolving plague pneumonia and bacteremia. In this way our bacteriological findings coincide with the observations of Koulecha..." Korobkova further maintains that the pneumonic changes observed in her material were primary in nature.

The following criticisms must be made:

1. A careful perusal of the paper furnished no convincing proof for the claim that primary pneumonic plague was found in the experiments. No histological findings are quoted and among the incomplete series of *post mortem* protocols only a few instances deserve the benefit of the doubt that any pneumonic changes were present.
2. Even though the bacteriological evidence be accepted it may be pointed out that
   a. the method of killing the animals with chloroform deducts much value from Korobkova's findings in the respiratory organs. It is obvious—especially in the animals killed a short while after infection with huge doses of *B. pestis*—that aspiration would have taken place during the death struggle. In other words the abundance of plague bacilli in the trachea and lungs is perhaps more apparent than real.
   b. It is difficult to see how Korobkova's findings can support the views of Koulecha. This and other advocates of the "tonsillar theory" maintain that the plague bacilli entering the system through the upper respiratory tract are transported to the lungs by the blood stream. Korobkova, however, states repeatedly and distinctly that in her animals bacteremia was of a secondary nature, and occured after the bacilli had reached the trachea and lungs.

3. Therefore, even if any reliance be placed upon Korobkova's findings, these do not support Koulecha's views but rather discredit them.
4. Korobkova's paper contains interesting information regarding "intestinal" plague infection. It is a matter of regret therefore that she has reduced its value by introducing theories which are not strictly to the point.

E. CLINICAL FEATURES OF PNEUMONIC PLAGUE.

I. DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS.

1. Tournier (44) says that in Madagascar cases of pulmonary congestion due to a spirochaete similar to Castellani's may at first cause diagnostic difficulties, but these soon disappear because the patients always recover.

2. Bates and Avery (45) make a noteworthy contribution to the group of cases where plague-like bacilli cause lung symptoms: A coloured male suffered from diarrhoea and weakness of 3-4 days' duration and died within 20 hours after admission to hospital. At autopsy outstanding features were noted in the lungs only in the shape of scattered hemorrhagic areas. Smears showed gram-negative cocci and diplococci or diplobacilli. Two mice inoculated intraperitoneally and a guinea-pig subcutaneously succumbed. Cultural tests showed that the causative organism was identical with *Bac. mucosus capsulatus*.

An inexperienced or rash investigator might in a case like this be persuaded to make a *prima facie* diagnosis of plague pneumonia. The careful analysis of the case therefore reflects credit upon the two authors. The only criticism one would like to make is that no guinea-pig was inoculated percutaneously. This method, though less spectacular than the peritoneal and subcutaneous ones frequently yields most reliable results.

3. Petrie, in a review of Dickie's work in California (46) recommends puncture of the apices and basis of the lungs in addition to the heart in persons found dead in suspicious circumstances, and remarks that "lung puncture as a diagnostic aid during life should certainly be practised more often than hitherto."

This advice is timely. As demonstrated by Girard, plague bacilli are often present in the lungs as soon as the first symptoms develop. There is much hope therefore that a preliminary diagnosis might be speedily arrived at with the aid of lung punctures at a stage when it is difficult to secure reliable results from the sputum. Incidentally, it could be proved by simultaneous blood cultures whether or not a bacteremia is present in the first hours of the disease. I for one feel sure that such blood tests would prove negative.

4. The number of atypical plague cases is augmented by Girard:
   i. Discussing the absence of symptoms which might suggest the simultaneous presence of influenza and plague, he quotes an exceptional case of pneumonic plague of 9 days' duration where *rhinitis* (coryza) was marked. This symptom is so uncommon in pneumonic plague patients that both Delbreil and myself

ascribed a differential-diagnostic value to its presence. Naturally, however, not too much reliance should be laid upon such aids.

ii. The case of a European lady who succumbed after five days' manifest illness to plague and passed the infection in pneumonic form to fifteen other persons, deserves attention. Though in her case no systematic search for a bubo was made, this seemed absent, and she suffered apparently from primary pneumonic plague. The source of infection in this initial case is difficult of explanation. A local origin appears out of question. Girard was able to establish that for two weeks preceding the onset of manifest plague symptoms the lady was unwell. However, since she suffered from chronic malaria and had had an initial shivering fit, the vague symptoms prevailing during that period were ascribed to the old infection. It is noteworthy, however, that just before that time she had returned from Tamatave where plague showed some activity. Girard thinks it unlikely that she transported infected fleas to her home and wonders if she had not contracted plague in Tamatave already, and remained a carrier of the disease for two weeks. To support this possibility he records two other instances:

iii. The case of a child who had for a fortnight an undiagnosed cervical bubo and succumbed finally to secondary plague pneumonia, virulent *B. pestis* being cultivated towards the end from the suppurating bubo.

iv. Case of a patient who was treated with bacteriophage and who "died of primary pneumonic plague 14 days after apparent recovery from a first attack, a reinfection being out of question."

Though it is difficult to come to any final conclusion about the case of the lady, an unusually long duration of the disease is perhaps the best explanation. The last mentioned case (iv) is of unusual interest since I remember only other instance where a relapse after recovery from lung pest came into question[4]. It is to be hoped that fuller details will be forthcoming in Girard's case.

Mention was made in the "Treatise" of a few instances where plague infection was established in babies born by mothers already ill with pneumonic plague. A new case of this kind, observed by Nikanoroff in 1915, is recorded by Bikov (48):

v. A Russian woman, aet. 40, who contracted pneumonic plague near the end of pregnancy, was on the second day of illness delivered of a living child. The mother died a few hours after the birth, while the baby survived for two days. No autopsy was performed upon the child but positive cultures were obtained from its blood.

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(4) A patient was reported ill on July 23 with fever and abdominal pains. Began to cough and expectorate bloody sputum; pulse retained good quality. Cough less after 1 week. Early in the illness, sputum positive for *B. pestis* (microscopically); after a week negative. Patient again fell sick on December 4 and went through a mild attack of bubonic plague (Relapse? Reinfection?) (47).

II. TREATMENT AND PROGNOSIS.

The latest results with serum treatment may be tabulated as follows:

Author:	*General results*:	*Successfully treated cases*:
Burnett Ham (6)		Nurse who had been in contact with patient without taking due precautions, sickened with headache, fever and malaise, but recovered without showing pneumonic symptoms. Recd. prophylact. serum.
Raynal (49)	Diego-Suarez (Madagascar): No results in 5 pneum. cases which had been treated with 120—200 c.c. per day, partly intraven., partly subcut. Two had been admitted on 2nd day of illness, three on 3rd; one of the 1st group had full dose of vaccine.	
Tournier (50)	Serum usually not given to pneumonic patients.	One patient suffering from confirmed pneum. plague and treated by Rehm, received within 15 days 7,000 c.c. of serum. While recovering, still showed plague bacilli in his sputum.
Girard (51)	Tananarive: In several Europeans each receiving over 1,000 c.c., the serum merely prolonged the disease. In natives no such results, the serum seemed even to hasten death, so that patients refused the treatment.	(*a*) European monk, aet. 50, affected with primary plague pneumonia, recovered after intravenous injection of huge serum doses. (This is probably identical with above case of Tournier). (*bc*) At Majunga two patients treated by Mury with serum doses of 60 c.c. for 2-3 days, recovered. (*d*) A young European lady who had been much exposed to infection, fell ill with fever, slight cough; sputum at times bloody. During the eight days of illness no plague bacilli, only pneumococci found in expectorations. Patient who had received huge prophylactic serum doses, recovered.

N.B.—Mitchell (52) records four cases of recovery in South Africa. No details are given in regard to the first case (1914), while the other three (1924) are stated to have "received adequate treatment and care."

Dujardin-Beaumetz (39), in a discussion upon Girard's cases (a-c), doubts if the Majunga cases were primary, since—as shown by Calmette and Salimbeni at Oporto—only secondary pneumonic cases are amenable to serum treatment. It is true that some of the recorded recoveries were not primary lung plague or even plague at all. For instance, it is an open question whether Burnett Ham's case and Girard's case (d) were actually infected or merely protected by the prophylactic serum treatment, their symptoms having been due to serum disease or some intercurrent illness. On the other hand, a number of fairly well-established instances of recovery have been recorded since Calmette and Salimbeni's time, most of which are mentioned in my "Treatise". To these we may now add Tournier's isolated case. Thus one can but repeat that a timely and energetically continued serum administration offers the only fighting chance available at present for the treatment of primary pneumonic plague.

F. PROPHYLAXIS OF PNEUMONIC PLAGUE.

I. PERSONAL PROPHYLAXIS.

Certain experiences with prophylactic serum and vaccine application which have recently come to my knowledge are herewith noted:

i. *Australia.*—During the Maryborough outbreak a group of nurses and other persons who had been in more or less close contact with a pneumonic patient without taking sufficient precautions, received an injection of 20 c.c. serum; in the case of the nurses daily doses of 10 c.c. were continued for 5 days. None of the inoculated succumbed to plague. Of special interest is the case of one nurse who at first refused the injection, though continuing to attend the patient. Her treatment was started 24 hours later. She fell ill but recovered without having developed pneumonic symptoms.

None of the contacts of the Balmein cases who had been protected with subcutaneous serum injections, got ill. It must be noted, however, that the three patients had little cough.

ii. *Egypt.*—Kamel Hanna (53) states that in Egypt where all pneumonic outbreaks are traced to bubonic cases, vaccination is practised on nearly all occasions. The results are apparently quite satisfactory. Thus in the provinces of Girga and Assiut eight villages with a total population of 60,000 showed a clear record during two years following a vaccination campaign.

iii. *Gold Coast.*—Selwyn-Clarke (54) states that previous vaccination was ineffectual in warding off a fatal issue in the pneumonic and septicemic types.

iv. *Madagascar.*—The work done in regard to vaccine and serum prophylaxis of pneumonic plague may be summarised thus:

a. Couvy (55) States generally that repeated administration of vaccine does not protect against primary pneumonic plague.

b. Tournier (50) Considers a single dose of 20 c.c. serum insufficient but obtained good results with such doses administered daily for six days to persons who had been in close contact with pneumonic patients. (Children under 10 years received 10 c.c. daily).

c. Girard (22) Concludes that one prophylactic dose of even 80 c.c. is without avail. Better results may be expected from repeated injections as recommended by Tournier (b).

d. The observations of Roques (56) made in the Isolation Camp of Tananarive deserve more detailed analysis. This author states that anti-plague vaccinations in Madagascar were not attended by the desired results. The natives, not distinguishing between vaccine and serum prophylaxis, became so distrustful of injections that for a time no preventive treatment was possible. When the work was resumed a single subcutaneous dose of 20 c.c. serum (usually protecting the contacts of bubonic patients) was found to be insufficient in the case of pneumonic (and septicemic) plague. Gradually the dose and mode of administration were changed as shown by the following table :

Period :	*Total dose given :*	*Mode of administration :*
Dec. 15, 1926—Feb. 1, 1927	20 c.c.	Subcutan.
Feb. 1—April 16, 1927 ...	100 c.c.	Subcutan in doses each of 20 c c. on the 1st, 2nd, 3rd, 5th and 6th day of isolation.
April 16—23, 1927	100 c.c.	20 c.c. subcutan on 1st day, 40 c.c. intraven on 2nd same on 3rd day of isolation.
April 23—May 17, 1927...	120 c.c.	40 c.c. intraven on 1st, 2nd and 3rd day resp.
May 17	100 c.c.	Intraven on 1st day of isolation.

The general results of this prophylactic campaign may be summarised thus.

Period.	No. of pneum. pl. contacts.	No. of plague cases among them.	% of plague cases.
Jan.—Dec. 15, 1926 (No prophyl. given)	176	43	24.4
Dec. 15—May 25, 1927 (serum given)	77	11	14.4

These results are the more remarkable when we consider that at first too small doses were given and that intravenous administration was resorted to comparatively late.

Roques states that rarely slight attacks of serum disease were met with, never serious accidents[5]. In his opinion the only drawback of the injections is that those who contract the disease in spite of treatment are, while surviving longer than the uninoculated individuals, particularly infective throughout the illness. For this reason prophylaxis should be given only in hospitals, isolation camps, etc.

It would seem that conditions for the preventive treatment of lung pest are especially favourable in Tananarive Town for three reasons :

i. Contacts are speedily isolated through excellent sanitary organisation.

ii. The number of contacts is comparatively small.

iii. An abundant supply of serum is available.

One hopes therefore that these promising studies upon serum prophylaxis of pneumonic plague will be energetically pursued, not so much to confirm the value of the inoculations as to ascertain the optimal dosage and method of administration.

II. NEW METHODS OF DISINFECTION.

Two new methods of disinfection deserve notice :

1. *Chlorine gas.*—Suvorov (58) warmly recommends the use of chlorine for both disinfection and disinfestation, as this gas can be used with advantage in insufficiently sealed rooms. 4.4 to 7.3 Russian pounds (1 lb. = 420 grms.) are allowed for each 1,000 cubic feet of space. Drawbacks of the method are :

   (i) Metallic things have to be removed before disinfection.

   (ii) While the gas acts best in a moist *milieu*, it tends to spoil materials, skins, furs, etc. It is therefore necessary to remove these and treat them separately. For this the author recommends Carbon Bisulphide (exposure for 10-15 minutes, concentration 1c.c. bisulphide to 1,000-3,000 c.c. of space).

2. *Chloropicrine.*—Chloropicrine has been used for several years both as a rat—and insect-icide and as a disinfectant in Madagascar (59). This method has also attracted the attention of the authorities in South-Russia (60).

Bouffard recommends the closing of windows of the house to be disinfected and to plug other openings with mats. 15 grms. of chloropicrine per cubic meter are used for the sick room, 5 grms. for the others. Then

(5) As emphasized in my "Treatise" the danger of anaphylactic symptoms in the prevention and therapy of plague is far more imaginary than real. This point is discussed by Pal (57) who concludes that most of the failures of serum treatment in plague are due to the small dosage employed and the fear of anaphylaxis which scares many a medical man from giving it intravenously—the only reasonable mode of administration.

the house is closed for 48 hours after which it can be entered with impunity by the disinfecting squad in order to complete the work (cleaning, burning old mats, rags, etc.) The house is sealed again for a month; Bouffard thinks, however, that five days would be sufficient in cases of bubonic plague.

The Russian authors speak favourably of the action of chloropicrine upon rodents, insects, plague cultures (including stabs) and plague bacilli contained in albuminous matter (peritoneal exudate and the like). They recommend that the sealing be done as hermetically as possible. The apparatus used must produce a fine spray. Semikoz and his coworkers recommend a dose of 19 c.c. of the liquid for one cubic meter of space. This has to be augmented 2-3 times in the case of leaky huts, while in kibitkas a 10-20 fold dose is advisable, to be sprayed within 30-60 minutes. The vapours penetrate different tissues like cloth, felt, etc. and do not spoil metallic objects.

While the efficacy of chlorine gas upon plague sputum has been established (58), no such tests have been performed in the case of chloropicrine. It stands to reason, however, that this will act in a similar way as upon peritoneal exudates, etc. It may be said in general that this method possesses many obvious advantages. The drawbacks are :

i. a prolonged ventilation (48 hours as a minimum) is necessary after disinfection.

ii. On account of its volatility it can be used for the disinfection of parts of buildings, only when these are separated from the rest of the premises by substantial walls and also possess special entrances.

It is evident that these two drawbacks will be felt under urban conditions. Nevertheless this method deserves further trial since only practice can show how far it will prove equally economical, efficient and practicable under the vastly different conditions of climate, housing, etc. obtaining in the different plague areas.

G. CONCLUDING REMARKS.

I have reached the end of this survey of recent researches upon pneumonic plague. Though in certain directions satisfactory progress is noted, the question as to how pneumonic plague epidemics arise is still unsolved. Discussing this problem on previous occasions, I came to the conclusion that the salient point is why under certain conditions secondary lung affections become marked and infective, thus causing cases and outbreaks of a primary nature, while at other times they fail to do so. I have further stated that the rise of pneumonic epidemics is due perhaps not to one unvarying factor but to a sum total of different factors which may vary in the different plague areas but nevertheless lead to one and the same result. In my opinion it would not be right to assume that these factors

are all extrinsic in nature. On the contrary, there is much reason to believe that a principal role is played by intrinsic causes, while the extrinsic conditions exert a powerful though only indirect part. As I said in this connection, it is evident that the three principal intrinsic factors (i. a peculiar character of the epizootics; ii. a lessened resistance of the human victims to plague infection and iii. a lowered resistance of the victims to lung diseases in general) might be influenced by meteorological and social conditions and the like. In other words, there is ample room for that interplay between "infection" and "immunity", to which Gill in his "Genesis of Epidemics" justly ascribes a fundamental importance.

Once we believe that not one all-important cause but an interplay between different factors, both intrinsic and extrinsic in nature, moulds the character of the epidemics, it is logical to conclude that an elucidation of this complicated problem must not rely upon mere inductive methods, which try to adapt the facts to a preconceived theory. On the contrary, the deductive method has to be chosen which permits conclusions to be based upon an unbiassed study of conditions prevailing not in a few but in all plague areas. This was the main reason why I thought it desirable at the League of Nations Expert Committee Meetings at Paris (1927) and Calcutta (1928) to propose a world-wide enquiry into the problems of pneumonic plague.

For this purpose I prepared an exhaustive Questionnaire for the consideration of all interested parties. This Questionnaire is embodied in the present Report.

The reception given to my proposals at Paris (where Director Hahn of the Hygienic Institute Berlin took the chair) was most encouraging, and I received many helpful suggestions.

At Calcutta, however, certain members and co-opts of the Committee, including Cols. J. D. Graham, F. P. Mackie, C. L. Dunn and J. Taylor did not ascribe to Pneumonic Plague the importance which in my humble opinion was its due.

Consequently the proposals to send the Questionnaire through official League channels was withdrawn, and only a limited space was awarded to plague pneumonia in a general enquiry upon plague matters (see Appendix B. Memorandum upon Plague in Wild Rodents, etc.).

Some progress may still be expected if the revised Plague Research Program as adopted at Calcutta is properly carried into effect. I am confident, that the good examples set by South-East Russia, Madagascar, South Africa, etc. will stimulate the workers of other plague areas into looking at the problem from a fresh angle.

WU LIEN-TEH.

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FURTHER OBSERVATIONS UPON PLAGUE IN WILD RODENTS.

(Published for first time)

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A. INTRODUCTION.

At the Far Eastern Congress of Tropical Medicine in Singapore, 1923, a paper (1) was read by one of us summarising the knowledge then available upon the Siberian marmot (Tarabagan) as well as upon other wild rodents responsible for plague outbreaks in different parts of the world. These rodents in addition to some which had been found susceptible to artificial infection were tabulated in that paper which was reprinted in our 1923-24 Report. New data came to hand soon afterwards and were published in a pamphlet "Practical Aspects of Plague in wild Rodents" by the League of Nations (2) containing a new list

of naturally infected wild rodents. In our 1925-26 Report a revised edition of the League of Nations pamphlet was embodied. In the "Treatise on Pneumonic Plague" by Wu Lien-Teh (Geneva, 1926), the problem of plague in wild rodents was exhaustively dealt with and some fresh information was added. Since then so much matter has been received that a revised article has become necessary. The appearance of the present Report (1927-28) offers a welcome opportunity to publish this.

B. SOUTH AMERICA.

On previous occasions we have quoted a wild guinea-pig known under the local name of "Cuis" (*Cavia aperea*) as being the cause of human plague outbreaks in Argentine. Jorge in a study upon plague rodents and fleas (3) mentions another suspect rodent, namely *Ctenomys* (called "tuco-tuco" by the inhabitants).

C. CALIFORNIA.

Mention was made in the "Treatise" of a rational scheme adopted for fighting squirrels in the Los Angeles County. This aimed at wholesale destruction of the rodents within areas surrounded by "squirrel barriers" rendering a repopulation of the treated territories impossible. As far as can be judged from a report embracing the period up to June 30, 1926 (4), the results were satisfactory. Dickie states that "the total squirrel infected territory in the four plague areas amounted to 28,000 acres. In two areas eradication was complete as recent inspections have disclosed no squirrels. In the remaining areas apparent eradication has been accomplished. 90-99 per cent reduction in the squirrel population has been obtained in the treated portion of the remaining acreage, and the work is being carried on diligently as soil and feeding conditions will permit."

To date, no further enzootic among the wild rodents has been reported from the Los Angeles County. Other districts of California where such measures were not taken still show infection. A 1928 report (5) states that plague has been proved in two lots of wild rodents coming from the Alameda and Contra Costa Counties respectively. Ground squirrels sent to the laboratory from San Benito and Monterey Counties were also found to be infected; in the latter district a fatal human case occurred in July.

Energetic action was taken by the authorities in the county of Monterey as well as in the other areas "to keep zones free from rodents around the dwellings in order to prevent the spread of the disease to rats."

D. NORTH AFRICA.

Jorge, in his 1927 summary (3) states that "gerbilles are suspected to keep alive a permanent plague focus in the Cyrenaica, a district of Libya bordering on Egypt; an epidemic starting four or five years ago from this focus spread almost to Tripolis and the Tunisian frontier; one

year ago it manifested itself at Kairouan. The vectors seem to be gerbilles which emigrated to the north on account of scarcity of food and illness. The animals have not been identified; for geographical reasons one could think of *Tatera hirtipes or Gerbillus gerbillus.*"

According to Raynaud (quoted by Jorge), an outbreak in Morocco which was imported from Tafilelt (a desert region in the Sahara) was also traced to wild animals hurriedly leaving their habitat.

These notes are of considerable interest. When studying plague conditions in North Africa one notes that the outbreaks are often not of a local origin but due to importation of the disease from the interior. It seems quite possible that in a vast portion of the *hinterland* plague smoulders among wild rodents in much the same way as in the case of Central Asia.

E. FRENCH WEST AFRICA.

On previous occasions we have mentioned a large field rat involved in this plague area (6). Now Leger (7) states that this rodent (called by the natives "kantchouly") is identical with *Cricetomys gambianus.* According to him plague has also been established in French West Africa in *Mus mauretanicus* and *Mus rufinus;* the information available in regard to these two animals is not sufficient to permit of their being embodied in our list.

F. SOUTH AFRICA.

Our knowledge of the South African wild rodents has been considerably augmented by some fine studies undertaken by Pirie asd Ingram (8) which may now be summarised with some fresh evidence.

1.—MODE OF LIFE OF THE RODENTS.

In the "Treatise" some data were given of the gerbille (*Tatera lobengulae*) and the multimammate mouse (*Mastomys coucha*). Further details are supplied of the former by Powell (9):

A warren is inhabitated by a family consisting of 4-24 animals. When medium-sized the area has a diameter of 20-30 yards with 15-20 entrance holes, while the depth is 4-6 ft. There are several sleeping rooms lined with dry herbs; these often possess a sort of emergency exit.

The gerbilles are industrious, always improving their burrows, and show a tendency to shift their quarters. For instance in a territory measuring a square mile and inhabited by 30 families, there may be 130 burrows, and within a fortnight 3-4 families may have successively used one single burrow.

In winter when their food becomes scarce, the gerbilles migrate either singly or in groups to the plains where the vegetation is better, while during the rainy season they seek refuge in more elevated regions. Adult gerbilles can swim and thus save themselves when encountering rain or inundations.

Pirie (8) furnishes the following data regarding other rodents associated with the spread of plague:

Name :	*Description* :	*Mode of life* :
Ground squirrel *Geosciurus capensis*	Head & body about 10 inches, tail 8 inches. Has short, stiff, reddish-grey hair, with a white stripe on each side of body.	Live in families in large warrens, often in close association with gerbilles, suricats and mongoose. Come out in daytime, may roam widely in search of food. Have spare warrens like gerbille.
Spring hare *Pedetes caffer*	Large rodent of the kangaroo type (head & body 15-16 inches, tail slightly longer).	Live in warrens, spec. in sandy country. One pair may have several warrens between which they alternate. Travel long distances (up to 20 miles) at night to feeding grounds.
Striped mouse *Rhabdomys pumilio*	Larger than ordinary mouse, dark grey in colour with four distinct black stripes along back, separated by three lighter stripes.	Live especially in bush country, where they usually build big nests of sticks on the surface of the ground and live in families of 12-30. Diurnal in habits. Travel by little runs from one bushy patch to another. Also found in stacks of grain, etc., and in fences near farms.
Namaqua gerbille *Desmodillus auricularis*	Smaller than Lobengula gerbille; tail only about half as long, ears shorter; white spot behind each ear.	Favour lime-stone country. Food consists mainly of seeds and grains. Live in warrens in families (2-8); have spare burrows nearby. Not so migratory as Lobengula gerbille. May be found living close to farm houses or on vacant lots in settlements.
Karroo rats (general designation for diff. species)	Slightly shorter & stouter than Black rat; tail shorter.	Water-rats which have taken to a dry life and are found only in Karroo country. Dig warrens like gerbilles, but frequently also build a nest of twigs of the bushes upon which they live over part of the warren. Have runs between one warren and another. May live near homes and settlements like Namaqua gerbille.
Hare Genus *Lepus*	L. zuluensis is rather smaller than ordinary hare and has usually white spot on forehead.	Do not burrow but during the day squat under a clump of bush, etc. Nocturnal in habits they may travel 3-4 miles a night for food; in times of drought more.

Natural plague has also been found in the following species :

Name :	*Description* :	*Mode of life* :
Large-eared mouse *Malacothrix typicus*	About the size of House mouse, but with short tail covered with short, white, bristly hairs and very large ears.	Lives in light sandy grass veld. Burrows small, usually with single opening only, generally under cover of a clump of grass or bush and difficult to see. Not in the habit of visiting each other's burrows or burrows of other species, but travels about at night along roads or footpaths, often for considerable distance from its burrow.
White-tailed rat *Mystromys albicaudatus* (10)	A rat about size of Black rat.	Lives (generally on black turf or clay soil) in single holes, is slow and lazy and does not travel far.

NB.—Plague infection is said to have been found also in the *Peba gerbille* (10) Since no further details could be ascertained about this rodent it is not embodied in our list. The same holds true of a "meerkat" (order of carnivora?) which may be only a carrier of infected fleas (11).

2. EXPERIMENTAL INFECTION.

Pirie, besides giving a list of five rodents and two species of carnivora experimentally susceptible but not definitely known to suffer from natural plague, mentions the following points :

i. *Morbid anatomy*.—The animals, which were infected percutaneously, showed as a rule no marked gross changes; the buboes were usually small, the spleen slightly enlarged and free from areas of necrosis; in the lungs small patches of congestion or commencing plague pneumonia were frequent. Septicemia was unvariably present. Pirie adds that experience regarding naturally infected rodents has "failed to show features indicative of a less severe type of infection and suggests that, in nature, the plague infection is also commonly of a rapid septicemic type." No evidence of chronic plague has been observed in gerbilles and multimammate mice; however, one Namaqua gerbille and one Karroo rat found dead in the fields, yielded positive experimental results without showing any macroscopic signs. ...

ii. *Transmission of plague by feeding*.—The gerbilles display, both in captivity and under natural conditions, cannibalistic tendencies towards their sick and weak brethren. In order to learn whether they can contract infection by feeding upon plague corpses, experiments were carried out in this direction and proved positive. Practically all the animals showed either more severe congestion of the lungs than those infected cutaneously, or marked pneumonia which was apparently secondary to bubonic invasion or septicemia.

iii. *Experiments with vaccine and serum.*—Tests performed upon gerbilles and multimammate mice with intradermal vaccination (prophylactically) and with sensitized vaccines (both for prophylaxis and treatment) upon gerbilles gave negative results. A vaccine prepared from agar cultures "was found to confer a certain degree of immunity to gerbilles, not of a very high order, but perhaps as much as was to be expected in a species so susceptible to infection." Better results were obtained with living avirulent cultures. Vaccine failed in the therapeutic experiments upon gerbilles. Likewise anti-plague serum, though highly satisfactory in guinea-pigs when given early and in large doses, led in the case of the gerbilles to a prolongation of the disease only. The serum was found less efficacious for prophylaxis than vaccine in both guinea-pigs and gerbilles.

In all the above experiments the percutaneous method was used. As is to be expected, some of the animals, especially those surviving for a prolonged period, showed marked lung changes of a secondary nature.

3. SPREAD OF INFECTION.

i. *From animal to animal.*—The paramount importance of fleas has been confirmed by recent experiments of Ingram (8) who succeeded in transmitting the disease from rodent to rodent with *Dinopsyllus lypusus and Chiastopsylla rossi* and later on also with *Xenopsylla eridos*. Mitchell in his Report for 1925-26 adds that the last mentioned is "the most common flea found in wild rodents, and is probably the chief agent in spreading plague in the veld."

Although among the other rodents plague seems to spread solely by fleas, in the case of the gerbille infection by feeding is also possible. Pirie is careful to point out that this question can be definitely settled only after an extensive examination of fresh, naturally infected animals.

ii. *From animal to man.*—Here also the principal role of the fleas is not only theoretically plausible but actually suggested by the fact that the three species known to convey plague from rodent to rodent besides *X. erilli* were found capable to bite man. It is possible that direct infection may also take place in persons handling carcases of wild rodents in the fields (Mitchell). The contention that persons eating the imperfectly cooked meat of plague rodents may contract *pneumonic* infection has already been mentioned in our previous article.

Man may be attacked not only in the fields, but also in houses through multimammate mice and rarely domestic rodents. Other species of rodents, though not entering human dwellings, may live quite near and thus become dangerous.

4. EPIZOOTICS.

Not all the wild rodents found infected in South Africa are of equal epidemiological importance. In the past both Mitchell (12) and Haydon (13) considered the gerbille as the main reservoir of plague, the multimmamate mouse usually serving as the vehicle for conveying infection to man. Recent investigations confirmed the supreme importance of these two species on the High Veld, where the ground squirrel and perhaps also the spring hare play somewhat less prominent parts. In the lower and more bushy country the main role is taken apparently by the striped mouse, while in the Karroo districts (interior plateau, 3000-4000 ft. high) Namaqua gerbilles and Karroo rats seem to be the main reservoirs of plague, hares taking part in its spread (Pirie, 8).

Mitchell (14) writing in 1924, considers it probable that the infection constantly smoulders among the wild rodents and is "sometimes, usually in the warm season, when all forms of insect life are abundant, apt to flame up and spread widely and rapidly amongst the rodent population and concurrently cause cases of the disease in man." The recent investigations of Pirie have thrown much new light upon this subject. His conclusions are :

i. Field experience suggests that plague infection can be kept alive in a scanty rodent population by flea transmission, combined possibly with the occurrence of carriers amongst the veld rodents.

ii. Four field experiments carried out with a view to determining how long infection could persist in burrows were inconclusive. One of the four experiments suggested a period of at least six months, but rigorous proof was lacking.

iii. Eight further experiments planned so as to have the factors under better control showed :—

(*a*) That in the absence of fleas plague was not persistent in gerbille burrows even for one month.

(*b*) That with fleas present infection persisted abundantly for two months, to some extent for three months, and, doubtfully, perhaps even for four months.

(*c*) As a corollary, that certain fleas of veld rodents, known to be potential plague vectors and known also to be capable to bite man can survive without hosts easily for two months, sometimes, for three months, and perhaps even for four months, and are capable of re-infecting with plague after these periods.

There is no doubt that in areas ravaged by severe epizootics the rodent population may soon become numerous again. An example is quoted by Mitchell in his report for the year ended June 30, 1926; During a resurvey of certain districts where enzootic plague had raged in 1923-24, undertaken in December 1925, the rodents were found to be far more numerous than before the plague. Possibly a destruction of their natural foes had made conditions specially favourable. No evidence of infection was found.

The excentric spread of rodent plague seems to occur not only in continuous stages (15) but also *per saltum*. Several species involved are migratory or roaming in their habits. Even when infested by fleas different from those upon the animals actually involved in the epizootics, such animals may serve as vectors of the disease by temporarily carrying fleas from infected rodents. This is suspected for instance in the case of the spring hare, the ordinary hares (8) and the carnivora (16).

Areas which are unsuitable habitats for the gerbilles serve as barriers against the epizootics, though they do not absolutely check their spread. Thus it was noted in 1923-24 that, although an area of 70 miles separated enzootic districts in the Orange Free State and the Cape Colony, it was overrun by the epizootic. Likewise the same epizootic travelled round rodent-free belts, which had been created artificially. It had been hoped also that territories populated by the Namaqua gerbille and by the Karroo rats might serve as barriers, because the former animals are much less susceptible to experimental plague infection than the other species, whereas the latter seemed protected through their habits and partly through a difference of their flea parasites. However, recent experiences have shown that plague may also invade such territories.

A favourable feature is that the disease does not seem to pass readily from the wild to the domestic rodents, since infections among the latter have been exceedingly rare.

Though wild rodents were practically never found to be transported by rail, it was feared that "should infection spread to the veld rodents of areas where oats or lucerne are grown extensively, the risk of its conveyance in this way to the larger centers would be greatly increased" (Mitchell, 1926). To judge from recent information (17) the infection, spreading at an alarming rate early in 1928, has reached such a district, the Ceres Basin, from where "grain, forage and fodder are carried by rail to Capetown and other centers."

On the whole it must be said that the plague problem has become more and more serious in South Africa. The area involved at the end of June, 1924, was about 50,000 square miles (16). Pirie estimated this to have increased to 100,000 square miles. Now the territory is probably larger.

5. DESTRUCTION OF THE WILD RODENTS.

Ever since the first detection of plague among wild rodents, they have been killed wherever possible in and round the infected areas. The methods adopted are gassing with carbon bisulphide or with agricultural dynamite ("Capex Vermin and Mole Destroyer" being found specially useful) and poisoning with barium carbonate and monkey-nut pellets or with strychnin-impregnated grain. Among the gerbilles an epizootic caused by a new bacterium was detected near the Tiger river. This "Tiger-river bacillus" is highly pathogenic to gerbilles and other small rodents but harmless to rats, larger veld rodents and mammals (dogs, cats, monkeys). Much hope is entertained that this strain may be profitably used for the extermination of small wild rodents.

When clearing a belt in order to protect the Capetown district from the onrolling wave of the epizootic (17) poisoning with strychnine was resorted to in the case of the gerbilles and striped mice, gassing against the Karroo rats and near homesteads where the poison might be dangerous to domestic animals. The simple process of poisoning resulted in a kill of over 90 per cent of the animals, while the more tedious gassing killed them whole-sale.

Successful as such campaigns are, their palliative nature is fully evident. Pirie and Murray (18) considered that even the above mentioned second line of defence formed by the Roggeveld and Cedarberg Mountains and by the Doorn and Oliphants Rivers with their irrigation canals, connected up by artificial belts, would be outflanked in time so that the epizootics would spread to the coastal belt.[1] These authors conclude: "We understand that Senior Surgeon Perry, Director of Plague Prevention in California, has expressed the opinion that with an expenditure of a million dollars annually for three years, plague could be eradicated from the State. On that basis, a million sterling annually and the turning of most of our population into rodent gangs, would be required here. Which, as Euclid would say, is absurd."

The only real hope seems to rest upon the attitude of the individual farmers both towards preservation of the natural foes of wild rodents, like birds, wild cats, etc., and the extermination of the rodents themselves. Mitchell concludes in this respect: "It is not too much to say that every farmer who tries intelligently to do so can clear his farm of veld rodents without very much expense; and if every land-owner or occupier did his duty in this respect the eradication of plague would become a practicable proposition" (19).

Perhaps the more plague threatens to invade highly cultivated areas the easier will success be realised.

G. SOUTH-EAST RUSSIA.

1. NEW SPECIES INVOLVED IN THE EPIZOOTICS.

Dealing with the plague problem in South-East Russia on previous occasions we tabulated a number of species suffering from natural plague:

a. Small sisels or susliks, now designated under the common name of *Citellus pygmaeus.*

b. The large suslik, now called *Citellus fulvus.*

c. Domestic mice, including the Kirghese mouse, *Mus musculus Wagneri.*

d. Field mice, *Microtus arvalis* and *M. socialis.*

e. Jerboas, *Alactaga saliens* and *A. elater.*

f. A hamster, *Cricetus cricetus.*

g. Mention was made furthermore of a *Mus* (? *Gerbillus*) *tamaricinus.* Whilst there seemed no doubt that plague had been found in this "sand mouse", the species to which it belonged could not be definitely established. Now it is known that both the *Gerbillus tamaricinus* and the *Gerbillus meridianus* have been repeatedly found plague infected (20).

[1] This fear seems to have been realised (17).

Some data as to the mode of life of these rodents are given by Golov & Joff (20) and by Tikhomirova & Zagorskaya (21). The animals are frequent in the sandy regions of South-East Russia where they dig their holes at the base of the sand hills. Not being subjected to hibernation, they store food reserves in the burrows. One curious habit of theirs which might be of epidemiological importance is to bring pieces of material, wool and the like, from the surface into their holes (22).
(NB. The same has been observed in susliks).

h. Nikanoroff states (23) that plague has also been found in *Mus tamaricinus lasurus*. No further information about this rodent is available from Russian sources.

i. This also holds true of the *Dipodipus* (23) (Dipodipus sagitta ?). which is experimentally susceptible to the infection. We are not sure if this occurs in nature as well.

k. *The Lagurus lagurus*, a rodent belonging to the subfamily of *Microtinae* has to be added to the list of naturally infected rodents as established by Kniazevski and Grishina (24).

Tikhomirova and Zagorskaya state (21) that these rodents often settle down in the deserted corridors of suslik burrows, and that they possess cannibalistic habits, entering the suslik burrows to devour fresh corpses. No wonder that suslik fleas are found upon them!

2. MODE OF LIFE OF THE RODENTS.

(Additional notes).

a. *Susliks.*—The epidemiologically important fact that the susliks are apt to migrate has found further confirmation (25). Experiments (26) and actual observations (27) show that they swim well and are (unlike mice) able to cross streams and large rivers. Tikhomirov (25) maintains that during extensive epizootics the disease may be propagated over long distances by migration or flight of the susliks. As observed by Obolenski (28) they cover under ordinary circumstances not more than 5-6 versts (3-4 miles) in a season. It would seem, however, that this question is not definitely settled and it is proposed to study it by marking groups of the animals with metal rings (29).

b. *Mus Musc. Wagneri.*—According to Tikhomirova and Zagorskaya (21) these rodents live mainly in the open in summer but retire at the approach of cold weather to the stacks of kumarchik (*agriophyllum arenarium*) and human dwellings. Obolenski (28) while maintaining that the mice live constantly near man, admits that they visit the burrows of wild rodents and may thus become dangerous vectors of plague.

c. *Cricetus cricetus*.—Koltzov (25) remarks that the hamsters breed twice a year, in early spring and in summer, up to 20 young being born at a time. These rodents have a short hibernation period, staying out until frost appears and awakening early in spring.

Plague was only once observed in a hamster, the animals leading a solitary existence. Nevertheless Joff (28) considers them potentially dangerous because of their contact with man. He thinks that insufficient attention has been paid in the past to the hamsters. It must be remembered, however, that the fleas on these animals do not bite man.

d. *Jerboas*.—These also breed twice a year, 3 young ones being produced at a time. The beginning of their hibernation period coincides with that of the hamsters, but they awaken later than all the other rodents (Koltzov, 25).

3. EXPERIMENTAL INFECTIONS.

Experiments by Nikanoroff showing an influence of the seasons upon experimental suslik plague have been described in the "Treatise." In the article on "Perpetuation of Plague among wild Rodents" (embodied in this Report) it is stated that these results have been supplemented through experiments of Gaiski undertaken throughout a whole year. This author has now published a second paper (30) which deals separately with the findings in the hibernating animals. It seems well therefore to discuss the problem of plague in hibernating susliks once more.

Experiments upon hibernating sisels have been performed in the past. Churilina (31) infecting them by different methods observed in a few instances a prolonged course of the disease (up to 4¾ months) and came to the conclusion that the carrying over of the infection from season to season occurs in the form of "chronic" nodose plague. In her opinion such animals have not been found in nature because they probably die in the burrows—an argument which does not sound very convincing.

Further experiments undertaken by Gaiski and Nikanoroff did not lead to definite results and need not detain us.

Gaiski's recent findings upon 30 hibernating susliks may be summarised thus:

Awoke and succumbed after 2-22 days (average 8 days)	21
Were killed after 15 & 35 days after infection respect	3
Succumbed after 45-139 days	6
Total	30

Interesting details of the above experiments are:

a. In nine cases plague bacilli were found only at the inoculated site; in two their presence in the organs could be proved by animal experiments only (intraperitoneal infection); in one case organ cultures were positive but those from the blood sterile.

The percentage of atypical cases was thus 40% as compared with 12 per cent in 128 non-hibernating susliks.

b. Of the three animals killed, two (15 and 35 days after inoculation respectively) were well nourished and showed plague bacilli at the site of infection but not in the blood or organs. In the third (killed after 15 days) bacteremia was present.

c. Of the three animals dying after 139, 121 and 101 days respectively after awakening from hibernation, at least one had passed through a stage of bacteremia, one guinea-pig infected intraperitoneally with material from the organs succumbing to plague (NB. Cultures from blood and organs were negative). In the other two animals positive cultures were obtained from the local abscesses, while bacilli seemed absent from the organs.

d. Histological examination in the case of the animal succumbing on the 139th day showed necrosis at the inoculated site with reaction from the tissue ("induration") which was specially marked at the periphery. A regional lymph gland was apparently unchanged.

Gaiski raises the question why such cases are not found in nature. In his opinion this is due to the fact that a diagnosis of plague is not easy in such animals.

At the 1927 Anti-Plague Conference Gaiski reported (31) attempts to find plague in hibernating susliks under natural conditions. In two districts where intensive epizootics had raged in summer, about 800 burrows were opened in the course of the following winter. Altogether 47 dead and 16 living susliks were found. The majority of the former were quite mummified. Five corpses were much decomposed, while 4 animals had apparently succumbed lately, fresh blood being found in their fleas.

Altogether nine animals were subjected to a detailed examination (7 of the dead ones and 2 of the living animals which seemed suspicious at autopsy). Although in some cases suggestive macroscopic signs were found, microscopic examination as well as cultures and experiments yielded invariably negative results. The same holds true of a further series of animals dissected in early spring.

Gaiski concludes that :

i. Susliks which become plague-infected before hibernation continue to sicken in winter and die in the holes.

ii. The *B. pestis* in hibernating susliks assumes an invisible form.

> Doubt was expressed in regard to Gaiski's second conclusion. Nikanoroff (32) said that no convincing proof had been brought forward and that the matter needed further elucidation. Still more to the point were Barykin's remarks (33) that the fact that no bacilli were found is due more to their paucity or slight virulence than to their invisible nature.

Correlating similar experiments made by us upon tarabagans we may at first express our satisfaction that most of the facts established by Gaiski coincide with our own findings. Especially there seems little doubt that in the suslik as well as in the tarabagan the *B. pestis* introduced during hibernation may for a prolonged period rest at the site of inoculation. Our contention that such animals with local (or rather latent plague) may pass through a bacteremic stage is also well supported, one of Gaiski's animals, which survived for 101 days, showing bacilli in the organs, but apparently none in the blood.

On the other hand, we agree with Barykin's statement that the absence of visible plague bacilli was more apparent than real. In our experiments it was sometimes extremely difficult to see the *B. pestis* even in material from which positive experimental results had been obtained. However, whenever we allowed sufficient time for a thorough histological examination, we succeeded in every instance. In 1926-27, when less care was bestowed upon this matter, we had a number of cases where the findings seemed quite negative. We feel sure that such a careful observer as Gaiski will obtain similar results in future when working out his preliminary communication made at the 1927 Conference.

As already alluded to in our article "Recent Knowledge upon Pneumonic Plague" Korobkova (34) infected 102 susliks by the mouth and found no evidence of "intestinal" plague. While stating that the habit of certain rodents in devouring plague corpses might be of some epidemiological importance, she apparently does not ascribe much influence to this saying that—since plague bacilli are rare in the intestinal contents—the feces are not particularly dangerous. Semikoz, Kotelnikov and Bessonova, who worked upon guinea-pigs (35), came to the conclusion that the excreta deserve far more serious attention. The Conference decided (36) that further investigations are necessary in this direction. One thing which in our opinion is of great importance for settling this matter is that (as again confirmed at the 1927 Conference. 37) inguinal buboes preponderate in both susliks and mice dead of plague in the fields.

4. THE FLEAS.

The extensive researches of Golov and Joff (38) enable us to amplify the incomplete data recorded on previous occasions. Some of their findings are herewith tabulated:

FLEAS.

Rodents :	FLEAS.				
	Species :	Able to bite man :	No. days infection is preserved :		Transmitted experimental infection. to :
			At 14-27° C.	At 0-15° C.	
Citellus pygmaeus	Ceratophyllus tesquorum	+	90	275	C. pygmaeus, C. fulvus, M. bobac, M. arvalis, Cric. cricetus, Mesocric. ewersmanni, White rat, Polecat.
	Neopsylla setosa	+	62	180	C. pygmaeus, C. guttatus, C. fulvus, Dipod. sagitta, R. norvegic., M. muscul., Arvic. amphib., Lag. lagurus, Gerb. meridianus, G. tamaricinus, Guinea-pig.
	Ctenophthalmus breviatus	—	90	396	
	Ct. pollex	—	204	—	
	Ct. orientalis	—	220	210	
	Frontopsylla semura	+	203	—	
Citellus fulvus	Oropsylla ilovaiskii	+	14a	—	
Dipodidae	Ophthalmopsylla volgensis	+	—	—	
	O. kasakiensis	+	14a	—	
	Mesopsylla hebes	+	14a	—	
	M. lenis	+	—	—	
	M. tuschkan		—	—	
Mus musculus,	Ceratophyllus mokrzeckyi	+	30a	—	M. musculus.

M. musc. Wagneri	Cerat. consimilis	+	110	210	R. norvegicus, M. musc., M. wagneri, Microm. minutus, Micr. arvalis, Lag. lagurus, Dyromys nitedula, White rat.
	Ct. musculi	+	—	—	
	Ct. taschenbergi	+	—	—	
Microtus arvalis, Lagurus lagurus	Ct. breviatus	—	As above		
	Ct. pollex	—	As above		
	Ct. orientalis	—	As above		
	Cer. tesquorum	+	As above		
	C. consimilis	+	As above		
	Amphipsylla rossica	—	110	—	
	A. prima	—	—	—	
Cricetus cricetus	Ct. breviatus	—	As above		
	Ct. orientalis	—	As above		
	Ct. wagneri	—	—	—	
Gerbillus meridianus, G. tamaricinus	Ceratophyllus laeviceps	+	63	—	
	Xenopsylla mycerini	+	84	—	

Remarks :—a) Succumbed to accidental causes.

It will be seen that in all fleas properly investigated the *B. pestis* is preserved for considerable periods. In fact Golov and Joff state that under laboratory conditions these harbour the *B. pestis* during their life term, regardless of their being fed after infection upon healthy rodents (or even pigeons) or not. Infected fleas were able after starvation for 150 days (at a temperature of 0-15° C.) to transmit infection to rodents. Golov and Joff therefore ascribe great importance to the fleas not only as transmitters but also as preservers of plague infection.

At the 1927 Anti-Plague Conference record was made of several attempts to prove this contention by field observations :

a. Nikanoroff and Gaiski (39) obtained a large number of suslik fleas (12,770) by opening burrows in winter and early spring in two districts, where epizootics had raged during the preceding summer. Cultural and experimental tests with these fleas were totally negative.

b. Similar investigations were made by Borzenkov and his collaborators (40) with 5,845 fleas from 144 burrows. The results were equally negative.

These findings were much discussed at the 1927 Conference. Nikanoroff and Gaiski as well as Borzenkov and his collaborators maintained that the fleas did not play any important role as preservers of the infection. The answer of Golov and Joff was that for various reasons these investigations could not be considered as final. Nikanoroff retorted (41) that—even it were possible to find plague-infected fleas in winter under natural conditions, these would be exceptions rather than the rule and would not therefore be of practical importance, the reservoir of the *B. pestis* being rodents (and human carriers).

Studies upon the geographical distribution of the different flea species show the curious fact that the areas where recent epizootics occurred among the susliks coincide fairly well with those where *Ctenophthalmus pollex* is found and *Ct. breviatus* is absent (Joff, 38). Joff, though not asserting that a direct connection exists between the disease and certain flea species, nevertheless argues that "both the distribution of fleas and the localisation of the endemics depend upon some third factor which creates favourable conditions for their existence—most probably some climatic peculiarity suitable for these fleas and the plague virus." He expresses the hope that by studying the fleas valuable clues may be obtained as to conditions favouring the endemicity of plague.

5. EPIZOOTICS.

We have previously stated how susliks are responsible for plague outbreaks in the steppe regions of South-East Russia, while in the sandy southern wastes the main culprits are the mice, an occasional role being played by other species. In the foregoing pages we have demonstrated that conditions in both areas are almost ideal for a preservation of the infection. It is now established that the virus is present in the susliks throughout the year; in fact, as remarked by Gaiski (42) the seasonal changes of susceptibility protect these animals from dying out wholesale whilst at the same time guaranteeing the preservation of the infection.

It is not definitely ascertained whether the fleas take an active part in this. However, as established by Golov and Joff (43), suslik fleas remain active under laboratory conditions at winter temperatures of the burrows and are able to feed upon their hibernating hosts; similar observations have been made under natural conditions. Consequently, since bacteremia sometimes occurs in the animals in winter we may assume that the fleas are able to transmit the disease in winter as well as in summer and thus form an important link in the chain of favourable conditions the whole year round.

As the mice (principal factors in the sandy regions) do not hibernate, it is easy to understand how the disease is perpetuated there.

Hence we can see that, once plague becomes entrenched in the rodent population of a district, it shows no tendency to disappear spontaneously, though following the regular seasonal variations and sometimes reduced to a flicker after the majority of rodents have been wiped away by a preceding epizootic (Nikanoroff, 23).

Some points may now be discussed in more detail :

a. Susliks.—Gaiski (30) maintains that the epizootics suddenly become manifest in the second half of May. Possibly the soil is prepared for them by the mating period which brings the solitary susliks in close contact (Golov and Joff, 44). Ample fuel is certainly provided for the infection owing to the susceptibility of young animals to the disease, though perhaps the susliks are as a whole more liable to infection early in summer than later on (23). According to Gaiski (30) the epizootics also end abruptly. Perhaps, as in the case of the tarabagan, the seriously diseased animals soon die off while in others which start to hibernate, the illness tends to become less acute.

The percentage of susliks usually found infected in an area is not high, 3-5 per cent being the rule. Naturally such figures depend upon many circumstances and decrease as more animals are examined (Joff, 45). Small districts are found where the percentage of diseased animals is higher, reaching 50 or more (45).[2]

Interesting statistics showing the rise of an epizootic are given by Nikanoroff (23) :

Month :	*No. susliks examined :*	*No. found infected :*	*Percentage infection :*
April	133	0	0
May	1,842	11	0.6
June	2,640	156	5.8
July	968	88	9.0

NB.—In addition 483 fairly fresh corpses were found in July.

[2] Gaiski (31) in the course of his digging operations in winter found 200 burrows which had evidently been inhabited during the preceding summer. Only 8% of living susliks were found in these besides 18.5% corpses, while the others had probably died off during the epizootic.

Mention was made in the "Treatise" of the strange fact that epizootics among the small susliks do not spread to other rodents abounding in the same districts. This question is touched again by Nikanoroff (23) who says: "When the principal rodent species is the *spermophilus* (*C. musicus, mugosaricus*) this animal alone is found to be the carrier of plague. By a strange coincidence, in spite of the abundance of domestic and field mice in these districts they do not contract plague. Obviously one cannot speak of an absence of contact between these animals and the *spermophilus*. On the contrary we have frequently found the burrows to be adjacent or even common to both susliks and mice; evidence has been found as to the mice entering the suslik burrows; the passage of suslik fleas to the mice and *vice-versa* is easy.

Probably factors hitherto unknown are at work. At present we can but note that in the northern steppe regions the susliks alone are the carriers and reservoirs of the plague virus."

b. *Mice.*—Mice epizootics vary considerably in extent being sometimes widespread, sometimes limited to quite small districts. Nikanoroff (23) records an interesting instance observed by Spodarevich where an 'epizootic' was restricted to the mice inhabiting a single hut, yet was responsible for human cases.

The intensity of the real epizootics also varies from 0.5—6% to 80% (23).

A close connection exists between massive epizootics and conditions favouring the propagation of the animals. Nikanoroff records in this respect (23) that a regular correlation is observed between epizootics and good harvests of "kumarchik" (*agriophyllum arenarium*). He adds that the Kirghese have learnt to realise that an abundance of mice forebodes plague. In our opinion, this seems to support Elton's theory (46) regarding the relation between breeding and epizootics.

6. CAMPAIGNS AGAINST SUSLIKS.

As recorded in the "Treatise" a campaign against the susliks was started in 1924, consisting in the creation of rodent-free belts (up to two versts wide) round some of the villages near which epizootics were found. According to Nikanoroff (47) the immediate results were excellent.

In 1925 all 31 settlements threatened by epizootics were surrounded by belts up to four versts wide. Altogether an area of 123,864 acres was cleared and 5,187,530 inhabitated burrows were treated, mostly with chlorine (48). In spite of these extensive operations the results were not so satisfactory as in 1924, human cases occurring at different points. According to Nikanoroff the main reasons for failure were: (a) measures against the susliks were not started immediately after plague was detected and (b) the population by hunting the animals for their skin and meat was in closer contact with the rodents than before.[3]

At the 1925 Saratov Conference some speakers entertained doubts if the above mentioned method was sufficient or even reasonable. One went so far as to suggest that the non-appearance of plague was not due to the antisuslik campaign but to a mere coincidence, as in other localities where infection had been proved among the rodents, no human cases followed, though no steps had been taken. The Conference, though not agreeing with such extreme views, found it advisable to supplement and modify the program of destructive measures as follows :

(*a*) To replace as far as possible the palliative methods by wholesale extermination of the susliks in suitable localities, especially where natural boundaries prevent re-infestation by immigration;

(*b*) To create belts round the threatened localities not after but long before epizootics become manifest;

(*c*) To consider as threatened not only the settlements but other localities where contact with the susliks is likely to occur, e.g. cultivated areas and the like.

This modified program was actually tried in 1926. However, as shown by the following tabulation, it was not possible on account of insufficient funds and other difficulties to carry out all that was intended.

Great attention was paid to the question how far the methods used are likely to kill parasites in the holes besides the rodents. Traut (49) declared that—though the parasites survive in burrows containing fresh nests—they will not become dangerous for a further spread of the disease if such burrows are carefully plugged up after treatment.

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3 According to a resolution of the 1925 Anti-Plague Conference the trade with suslik skins was prohibited in the endemic regions, a policy which is still adhered to in spite of some secret hunting and open requests for abolishing the bane.

ANTI-SUSLIK CAMPAIGN IN THE YEAR 1926.

	Planned :			*Actually Done :*		
Locality :	*Kind of work :*	Acreage :	Number of holes :	*Kind of work :*	Acreage :	Number of holes :
(a) Jandiko-Mochashni District.	Wholesale destruction of rodents	143,000	3,000,000	Wholesale destruction of rodents	83,458	2,904,475
(b) Cherno-jarsk etc., Distr.	i. Creating 4-5 versts belt across distr. separating suslik-infested area from settlements & Volga River.	Ca. 71,500	2,000,000	1. Creating belt across country	59,885	1,914,570
	ii. Palliative belts to be created in spring.			ii. Narrow belts round cultivated areas.	5,374	110,848
(c) —	Palliative measures to be carried out when necessary round villages, cultivated areas, etc. Belts to be 1-3 versts wide.	22,880-28,600	1,000,000	Palliative measures carried out in all 14 areas of Stalingrad Govt. where epizootics were found. In some cases wholesale destruction was carried out in endemic areas themselves.	43,752	1,282,760

NB.—1. The work in areas (a) and (b) was carried out with carbon bisulphide while in areas (c) chlorine was used as far as the somewhat limited supply permitted. Chloropicrine, though giving satisfactory results, was not used on a practical scale.

2. Besides the above palliative work was carried out in the Salski District (North Caucasus Territory) by a special organisation.

Traut under whose leadership this work had been performed, commented at the 1927 Anti-Plague Conference upon the above figures as follows (50):

Area a.—In some districts, especially those treated by Kalmuck workmen, 1-5% of the suslik population survived. Nevertheless the area was the only one where no plague focus was detected.

Area b.—The area seemed practically free from susliks when inspected in summer 1926. Traut thinks also that the antiplague results were good.

Area c.—The results were fair.

It would seem that Traut was too optimistic as far as Area (b) is concerned. He was vigorously opposed by Ignatiev who stated that in spring, 1927, plague-infected susliks were found among healthy ones in the belt which had been cleared the previous summer. Ignatiev also upheld that the absence of susliks noted at the end of the 1926 campaign

was more apparent than real, the surviving susliks having already retired for hibernation. Traut questioned the exactness of Ignatiev's findings and declared that no harm results when a small number of rodents remain in the districts because these do not usually come in contact with one another; consequently they do not propagate and soon die off.

Be this as it may, there is no doubt that early in 1927 some plague infection was found in Area b, as confirmed by Nikanoroff. Thus the Conference was faced with the problem not so much how to carry on the work as whether to continue it at all. The necessity of doing so was unanimously agreed upon, some difference of opinion remaining only as to ways and means. Traut upheld that a 100% destruction is not absolutely necessary and that, if this be insisted upon, only 1,000 *desjatines*[4] could be cleared with the same amount of energy and money needed for making 1,300—1,500 *desjatines* practically free from rodents (1-3% remaining); in other words by the new policy two foci could be cleared up where formerly three were possible. Nevertheless, the Conference resolved that the areas under treatment must be cleared as completely as possible. Only two methods were considered as economically and prophylactically sound, viz.—

a. Wholesale destruction of the rodents;

b. Creation of protective belts early in spring and not after epizootics are detected, the latter method to be used only in cases of emergency.

Further it was stated that the method to eliminate the flea danger by plugging the burrows after the campaigns is by no means satisfactory. A second proposal to kill the fleas by pouring water in the gassed burrows was considered unreliable and expensive. Thus new methods have to be worked out in this direction.

The scope of work proposed by Traut for the year 1927 may be tabulated as follows :

PROGRAM OF ANTI-SUSLIK CAMPAIGN IN 1927.

Locality :	*Kind of work* :	*Acreage* :	*Number of holes* :
Chernojarski District	Widening the belt laid across the district in 1926 to 10 versts.	114,400	4,000,000
Chernojarski District	Going over the areas treated in 1926 where more than 2-3 susliks survived per *desjatine*.	5,720	5-6,000
Donski Distr., Stalingrad Govt.	Early creation of belts, 2-4 versts wide, round the settlements where epizootics were observed in 1926.	71,500	3,000,000
——	Usual palliative emergency work.	Up to 28,600	Up to 1,000,000
Salski Distr. North Caucasus Territory.	Usual palliative emergency work.	Up to 85,800	?

[4] A *desjatine* equals 2.86 acres.

Reliable figures of the results obtained in 1927 are not yet at hand. Passing an unbiassed opinion upon the work performed during the years 1924-1926, we must first express our admiration at the truly heroic battle waged against the susliks in the face of tremendous odds. Prominent among the difficulties are the following :

1. *Indifference of the population.*—This is the more pronounced as in most of the areas under treatment little or no agriculture is done, the susliks therefore representing no economical danger;
2. *Insufficiency of funds.*—It was rarely possible to accomplish all that was planned, since both the scope of the work and unforeseen obstacles render it difficult to form more than an approxmate forecast of expenses. Sometimes the granted sums were not forthcoming in time.

Unfortunately these two obstacles are not the only ones. The difficulty to kill off fleas simultaneously with the susliks has been mentioned already. The available funds allow for a thorough destruction of the susliks in part of the territory only, while in other places the epizootics perpetuate or even tend to spread. Finally, as alluded to by Joff (51) the campaigns have to embrace not one but all species suffering from natural plague.

Much against our inclination we are obliged here, as elsewhere regarding the destruction of wild rodents, to mention failures rather than successes. We believe, however, that in such a vital problem as war against rodents, nothing is more dangerous than excessive optimism.

H. CENTRAL ASIA.

Although there is every probability that different species are naturally infected and responsible for outbreaks, little information is available in regard to them. Mention was made already on previous occasions of the tarabagan-like animals involved in the Semiretchinsk District.[5] A subspecies, *Arctomys robustus*, is said to be the cause of human outbreaks in Thibet (55). The same animal is suspected in Kansu (55). The outbreak observed in this Chinese province in 1917 at the time of the 1917-18 Shansi epidemic is reported to have originated in a man who skinned and ate a tarabagan found dead on the hills bordering on Thibet (56).

Recently the *Rhombomys opimus* has been found responsible for plague outbreaks in Turkestan and Transcaspia. In the latter area hares (*Lepus timidus*) are also involved (57).

Nikanoroff (57), when dealing with the 1926 outbreaks in Transcaspia, gives some data regarding the *Rhombomys opimus*. He describes the animal as being the size of a rat, yellowish-grey in colour; the tail is long and ends in a black tuft. These rodents live in communicating burrows with 15-100 entrance-holes. Near the surface they have large

[5] A recent paper of Klodnitzki (52) states that large susliks (*Citellus fulvus*) are found in this territory. It is not certain whether these correspond to what the natives call tarabagans. It may be mentioned that the *C. fulvus* stands much nearer to the marmots than other susliks (53) and that according to Satunin (54) these '*Cynomys*' should be considered as a subspecies of the genus *Marmota*.

stores of hay. The sleeping rooms lie at a depth of 1-2 meters and are lined with dry herbs, pieces of camel-fur, etc.

The finding of mummified corpses, bones, pieces of fur, etc., in the holes proved that a virulent epizootic had raged among the rodents. Its plague nature was established by three methods :

i. A few animals with macroscopical changes were found giving positive tests; three of them had cervical buboes.

ii. A guinea-pig injected with an emulsion prepared from the spleen of 22 apparently healthy rodents, succumbed to plague.

iii. The same result was obtained with a guinea-pig which had been inoculated twice with an emulsion prepared with a great number of parasites (fleas and ticks) found on the rodents and specially in the burrows.

Most of the 153 fleas collected at the time from the rodents and found in burrows consisted of *Xenopsylla* (? *conformis*), *Ctenophthalmus dolichus* and *Ceratophyllus* (? *laeviceps*). There were besides a few specimens of each *Ophthalmopsylla volgensis, Rhadinopsylla cedestis, Coptopsylla* (? *lamellifer*) *and Neopsylla* (? *setosa*) (Joff, quoted by Nikanoroff and Kniazevski, 57).

At the 1927 Anti-Plague Conference (Saratov) Joff stated (38) that the following fleas are found upon the *Rhombomys* : *Xenopsylla gerbilli, X. hirtipes, X. skrjabini, Coptopsylla sp., Ceratophyllus fidus, C. laeviceps*[6]), *C. tersus, Ophthalmopsylla volgensis*[6]), *Ctenophthalmus dolichus, Rhadinopsylla cedestis.* Furthermore *Echidnophaga gallinacea* was caught in the nests.

The only hare in which plague infection was proved displayed, according to Ignatiev (57), typical features of hemorrhagic septicemia. He further stated that he saw not rarely other corpses—too decomposed for bacteriological examination. The positive animal was found not far from a *kibitka* (travelling tent) where a lad had died of plague. The relatives said that he had handled hares 3-4 days before he fell ill.

Nikanoroff suspects that infected rodents had been imported into Transcaspia from Turkestan. He says in this connection that the *Rhombomys opimus* undoubtedly lives near man : When inspecting the property of the infected families, such rodents were often detected amidst felts and bags with barley; in felts bundled together for transport sometimes whole families of rodents were found. Nikanoroff believes therefore that in this case the camel (ship of the desert) played a similar role, as the sea-going vessels do in transporting rats. This theory is interesting and perhaps of practical importance.

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[6] Known to bite man.

I. TRANSBAIKALIA.

1. NEW SPECIES FOUND PLAGUE INFECTED.

In the "Treatise" it was said that many Russian authors, while admitting the important role played by the tarabagan in the immediate causation of human plague, are reluctant to agree that the virus is permanently kept alive in this animal. Attention was specially drawn to the theory of Sukneff (58) according to which the reservoirs of plague are not the tarabagans but certain species of non-hibernating small rodents. For many reasons this theory seemed to us far-fetched. We have mentioned that—as can be judged from available evidence—the disease continues to exist in hibernating tarabagans in a modified form whereby the virus is carried over the winter. At the same time we admitted that insufficient attention had hitherto been devoted to the smaller rodents and that the presence of plague in them, if any, might have been overlooked.

New evidence is now available from two directions:

a. Our 1926-7-8 experiments have definitely established that the plague virus is preserved in hibernating tarabagans.

b. Plague infection has been recorded in two species of small rodents.

For fuller details regarding these hibernation experiments we refer our readers to the first two articles of this Report. We proceed to a discussion of the findings in the small rodents.

i. The Jerboa (*Alactaga mongolica*).—Skorodumoff reported (59) the finding of plague in two representatives of this species. The first was caught alive near the village Akurai (65 versts from Borzia) in a locality free from inhabited tarabagan burrows. The obviously sick animal died two days afterwards (September 16, 1926) showing at *post mortem* enlargement of the cervical and inguinal glands; the liver was congested and spleen mottled; petechiae were present under the intestinal serosa. The diagnosis of plague was fully established.

The second jerboa was found on September 1, 1927, at Hadabulak, a locality 100 versts distant from Akurai. It died one hour after capture and displayed at autopsy the same signs as the first (cervical and inguinal glands markedly enlarged with hemorrhages; subpleural hemorrhages; liver and spleen congested and mottled; subserous hemorrhages on stomach). Though the diagnosis of plague was confirmed from the organs of the animal, negative results were obtained with 8 fleas (*Ceratophyllus sp.*) and 21 ticks (genus *Ixodina*) found upon it. It may be added that the next inhabitated tarabagan burrows were four versts distant from the village Hadabulak.

The *Alactaga mongolica* belongs to the family of *Dipodidae* (*Jaculidae*) and is a jumping animal, its hind extremities being about 4 times longer than the front ones. The animals do not usually approach man, being when healthy of strictly nocturnal habits. They are not hunted and have a hibernation period (Radde).

ii. The Dauria sisel (*Spermophilus dauricus*).—During a mass examination of rodents in the districts of Birka, under Skorodumoff's leadership in July and August, 1927, some suspicious susliks were found (60). In the first three cases gram-negative and bipolar-stained bacilli were seen under the microscope but no further tests were made. In two other animals, captured alive, the diagnosis of plague was fully established. These in addition to the first three susliks probably had septicemic plague, no buboes having been noted at *post mortem*.

It is of interest that the two living animals were caught near the settlement in a locality where according to the inhabitants many mice (?) had died during the preceding year (1926), while in the spring of 1925 dead tarabagans were found nearby which had evidently been carried thither from the neighbouring hills and valleys by the spring flood.

The *Sp. dauricus* is the smaller of the two suslik species found in Transbaikalia, reaching a length of not more than 22 cm. This rodent is similar in appearance to the ttrabagan and squats on its hind legs uttering a characteristic whistle. Like its South-Russian relatives, the animal leaves its burrow in day-time only, and is subject to hibernation. Leading a solitary life it is very fierce and resists violently attempts to capture it. Skorodumoff (60) maintains that—though generally herbivorous—the *Sp. dauricus* hunts mice and rat-hares, and—being stronger—kills and sometimes devours them. This author also lays stress upon the fact that the holes of the Dauria sisels like those of other small rodents sometimes communicate with deserted tarabagan burrows, but says that he has never observed any actual contact between the animals (61). The flea of the *Sp. dauricus* is the *Ceratophyllus tesquorum* while its ticks belong to the genus *Ixodina* (60) and its lice to the genus *Polyplax* (62).

iii. *Conclusions*.—Valuable as these findings of Skorodumoff are their importance should not be overrated for the following reasons :

(a) Both the jerboas and the Dauria sisels hibernate in much the same way as the tarabagan. Consequently, if any doubts exist whether plague can be carried over the winter by hibernating rodents, these necessarily apply to all three species.

(b) Transmission of the infection from the tarabagan to these small rodents and *vice versa* is probably not frequent because the tarabagan fleas have a strict predilection for their own hosts, while no instance is on record where rodent fleas other than the *Oropsylla silantiewi* (*Ceratophyllus*) were found on the Siberian marmot.

(c) It is true that the jerboas as well as susliks do not have much contact with human beings. Nevertheless, should epizootics among them be frequent and widespread, then some animals at least (caught by playing children, dogs, cats, etc.) would enter courtyards or dwellings. However, we know of no instance where these species were even *suspected* in this respect.

(d) For these reasons we think that infections among the small Transbaikalia rodents are more of a sporadic nature and are perhaps the sequel rather than the cause of the constant tarabagan epizootics.

2. THE TARABAGAN FLEA (*Oropsylla silantiewi*).[7]

In the course of their investigations upon the fleas of the South-Russian rodents (38, 53) Golov and Joff found the *Oropsylla silantiewi* on the *Marmota bobac*, a close relative of the tarabagan. This rodent, though experimentally susceptible, has thus far never been found infected under natural conditions.

Golov and Joff succeeded in transmitting plague with the aid of living infected marmot fleas (*O. silantiewi*) to *Marmota bobac*, *Citellus fulvus*, *C. pygmaeus*, and *Mus musculus*. It is gratifying to note that the positive results of our own experiments upon tarabagan fleas are now confirmed.

Like other South-East Russian rodent fleas, *O. silantiewi* remains infected for prolonged periods (in fact throughout its existence) once it has sucked plague blood. The following figures were obtained:

	Not fed after infection:	*Fed after infection*:	
Temperature:	0-15°C.	14-27°C.	0-15°C.
Fleas remained infected for days:	242	194	358

As in the case of the suslik fleas, it remains to be seen whether the tarabagan flea takes an active part in the carrying over of infection. According to Golov and Joff it was very difficult to feed this flea when the temperature fell in winter, so that even those which had been fed beforehand were left starving for four months. Though it is not indicated whether these fleas were fed upon their usual hosts (*marmota bobac*) it is nevertheless an open question whether the *Oropsylla silant*, remains active during winter as some of the suslik fleas do. It may be added that Skorodumoff (64) noted the thermometer to mark 0.5°C. in a tarabagan burrow when the outer temperature was −36°C.

[7] Wagner and Joff (63) decided that the group of *oropsyllae* should be separated from the genus *Ceratophyllus*. We humbly follow these authorities, one of whom (Wagner) first described the tarabagan flea.

Our experience that the *O. silantiewi* shows a distinct predilection for its proper hosts is fully confirmed by Joff (38, 53) who says that "at present we can point to but two species of South-East Russian fleas which may be considered—as far as our material goes—as strictly specific ('monozoid'): these are the *O. silantiewi*—the flea of the marmot and *Ctenophthalmus spalacis*—the flea of *Spalax microphthalmus*." Joff is careful to point out that the absence of close relations of these rodents in the areas under investigation and their limited numbers may explain the 'specificity' of their fleas.

Golov & Joff observed, as we did, that the *O. silantiewi* have a tendency to stick to their hosts even after the death of the latter, and said that this is facilitated by the thick fur of the rodents in question. Nevertheless they believe these fleas to dwell mainly in the nests and not upon the animals. On account of their large dimensions the fleas are able to suck up considerable quantities of blood. Consequently the chances of their becoming infected when fed upon animals with moderate septicemia are greater than in the case of smaller species. They quote an example where 50% of the *Oropsylla* became infected as compared with only a few suslik fleas fed upon one and the same animal.

Our findings that the tarabagan fleas attack man is confirmed by actual observations made by Golov and Joff in the course of their field work.

K. CONCLUDING REMARKS.

When attempting to draw conclusions from the evidence assembled above, it must be emphasised that the more one learns about plague in wild rodents the more serious this problem appears. So far as we know, there is only one modern instance where a wild rodent plague focus became extinct (England). As a rule, once the infection had gained a foothold among such animals, it not only perseveres but even tends to spread whenever possible, sometimes at an alarming rate. Although the danger of a transition of the disease from the wild to the domestic rodents is not omnipresent and should therefore not be overrated, such transition has in all probability taken place in at least one area and may be imminent in others. Further, not too much stress should be laid upon the fact that the wild-rodent foci are usually situated in sparsely populated regions, and that the danger of a spread among humans is consequently not great. We can point to the example of Manchuria to show that this protection is of relative value only. And this security tends to diminish more and more since some foci are likely to extend to more populated areas, while others are approached by modern civilisation with its quick means of communication. Finally, turning to the areas where campaigns against wild rodents are practicable, we repeat that in spite of all the attempts of those in charge of the operations, the means at their disposal enable them at most to free only suitable subdivisions of the plague areas permanently from rodents, whereas in other districts mere palliative work is performed. It may be that such palliative measures, while necessary and usually accompanied by temporary success, sometimes lead in the end to an increase in the number of wild rodents by favouring the multiplication of animals, which may survive or immigrate.

One consoling feature lies in the progress evident everywhere in research work upon plague among wild rodents. It would perhaps be too much to expect that the results of these investigations will soon become common knowledge so that those in authority as well as the laymen concerned may take more interest in the fight against wild rodents. Undoubtedly, the more our knowledge increases the easier it will be to concentrate all available means and energy upon the vital points.

WU LIEN TEH, M.D.
ROBERT POLLITZER, M.D.

L. APPENDIX.

I. TABLE OF RODENTS KNOWN TO SUFFER FROM SPONTANEOUS PLAGUE OTHER THAN THE DOMESTIC RATS AND MICE (REVISED TO SEPTEMBER, 1928).

		Name :	*Locality :*	*Reference :*
Sciuridae	1.	Tarabagan *Arctomys bobac*	Transbaikalia, Mongolia, etc.	Bjeliavski & Rjeshetnikoff, Vj. Obst. Guig., 1895.
	2.	Dauria sisel *Spermophilus dauricus.*	Transbaikalia	Personal information from Prof. Skorodumoff.
	3.	Large suslik *Citellus fulvus.*	South-East Russia	Koltzov, Vratchebnaja Gaz., 1917, p. 417.
	4.	Small suslik *C. pygmaeus.*	South-East Russia	Deminski, quot. by Klodnitzki, Russki Vratch, 1913, p. 1067 and Berdnikov, Zentralbl. f. Bakt., 1913, vol 69, p 251.
	5.	Ground squirrel *Geosciurus capensis.*	South Africa	Mitchell, Publ. H. Rep. for year ended June 30, 1924.
	6.	Californian Ground squirrel *Citellus beechyi.*	California.	Wherry, Jl. of Infect. Dis., 1908, p. 485 and McCoy, U.S. Publ. Health Rep., 1908, p. 1289.
	7.	Squirrel *Fonambulus palmarum.*	Ceylon	Hirst, Colombo Rep., 1922, p. 41.
	8.	Squirrel (a) *Sciurus palmarum*	India	Simond, Ann de l'Inst. Pasteur, 1898, p. 664.
Muridae — Murinae	9.	Multimammate Mouse *Mastomys coucha*	South Africa	Mitchell, Jl. Hyg., 1921, vol. 20, p. 377 and Haydon, Lancet, 1921, II, p. 1103.
			Uganda	Uganda Protect. Ann. Med. & San. Rep. for 1921, p. 96.
			Senegal	Leger & Baury, Bull. Soc. Path. Exot. 1923, vol. 16, p. 136.
	10.	Striped mouse *Rhabdomys pumilio.*	South Africa	Mitchell, Jl. Roy. Army Med. Corps, 1906, vi, p. 130 & Report, 1924.
	11.	White-tailed Rat *Mystromys albicaudatus*	South Africa	U.S. Publ. H. Rep., Sept. 2, 1927, p. 2233 & Sept. 9, 1927, p. 2282.

Muridae	Murinae	12. Pelomys fallax *iredescens.*	East Africa	Lurz Arch. f. Schiffs-& Tropenhyg., 1913, No. 17, p. 593.
		13. Tree rat *Epimys dolichurus*	East Africa	Lurz Arch. f. Schiffs-& Tropenhyg., 1913, No. 17, p. 593.
		14. *Arvicanthis abyssinicus nubilans.*	East Africa	Buchanan, Bull. Off. Internat. d'Hyg. Publ., 1925, p. 492.
		15. Hamster-rat *Cricetomys gambianus.*	Gold Coast	Graham in Simpson, Rep. on Plague in the Gold Coast in 1908, p. 21.
			Senegal	Laveau, Bull. Soc. Path. Exot., 1919, p. 482 and Leger, Ann. de Med. et Pharm. Colon., 1926, vol. 24, p. 273.
		16. African Bush Rat *Golunda campanae*	Senegal	Leger & Baury, l.c.
		17. Field Rat *Arvicanthis niloticus*	Egypt	Egyptian Plague Rep., Cairo, 1924, p. 52.
		18. *Bandicota malabarica.*	Ceylon	Philip & Hirst, Jl. of Hyg., 1915, vol. 15, p. 543.
		19. Bandicoot *Bandicota indica* (*Nesocia bandicota*)	India	Jl. Hyg., 1907, Plague No., p. 760 & 1910, Pl. No., p. 459.
		20. Small bandicoot *Nesocia bengalensis*	India	Hossack, Jl. & Proc. Asiat. Soc. of Bengal, New Series, vol. 5, 1906.
	Gerbillinae	21. Sand mouse *Rhombomys opimus.*	Turkestan	Nikanoroff, qu. by Grekoff, Guig. & Epidemiol., 1924, vol. 3, No. 4, p. 50; Vj. Microbiol. & Epidemiol., 1924, p. 201; personal communication from Pr. Nikanoroff.
			Transcaspia	Nikanoroff, Vj. Microbiol. & Epidem. 1927, 1, p. 3.
		22. *Gerbillus tamaricinus.*	South-East Russia	Golov & Joff, Transact. of 1927 Russian Anti-Plague Conf., pp. 110, 141.
		23. *Gerbillus meridianus.*	South-East Russia	Golov & Joff, Transact. of 1927 Russian Anti-Plague Conf., pp. 110, 141.
		24. Gerbille *Tatera lobengulae*	South Africa	Mitchell and Haydon, l.c. (1921).

Muridae	Gerbillinae	25. Namaqua gerbille *Desmodillus auricularis* (b)	South Africa	Mitchell, Publ. H. Rep. for year ended June 30, 1926.
		26. Gerbille *Psammomys obesus* (c, d, e)	Tunis	Gobert, Arch. Inst. Pasteur de l'Afrique du Nord, 1921, vol. 1, p. 440.
	Microtinae	27. Field Mouse *Microtus arvalis*	South-East Russia	Damberg & Tikhomiroff, quot. by Koltzov, Vratch. Gaz., 1915, p. 335; Nikanoroff, Vj. Microbiol. & Epidem., 1922, vol. 1, p. 71 and personal information.
		28. Field mouse *M. socialis.*	South-East Russia	Koltzov, Vratchebnaja Gaz., 1917, p. 147.
		29. *Lagurus lagurus.*	South-East Russia	Kniazevski & Grishina, Transact. of 1927 Russian Anti-Plague Conf., p. 87.
	Otomyinae	30. Eastern Karroo Rat *Parotomys luteolus*	South Africa	Mitchell, l.c., 1924.
		31. Broom's Karroo Rat *Myotomys broomi*	South Africa	Pirie, The Plague Problem in South Africa, Publ. of the S. Afric. Inst. f. Med. Res., 1927.
	Cricetinae	32. Hamster *Cricetus cricetus*	South-East Russia	Koltzov, Zabolotny's Rep. on Plague in S.-E. Russia, Leningrad, 1926.
	Dendromyinae	33. Large-eared Mouse *Malacothrix typicus*	South Africa	Mitchell, l.c., 1924.
	Neotominae	34. Dusky-footed Wood rat (Brush rat) *Neotoma fuscipes.*	California	McCoy & Smith, Jl. Inf. Dis., 1910, vol. 7, p. 368.
	Sigmontodinae	35. Field rat *Hesperomys palustris.*	New Orleans	William, Amer. Jl. Publ. Health, 1920, November.

Jaculidae	36. Jerboa *Alactaga mongolica.*	Transbaikalia	Skorodumoff, Guigiena & Epidemiol., 1928, vol. 7, No. 5, p. 69.
	37. Large Jerboa *A. saliens.*	South-East Russia	Berdnikov, l.c.
	38. Small Jerboa *A. elater* (f)	South-East Russia	Koltzov, l.c., 1926.
Leporidae	39. Hare *Lepus timidus.*	Transcaspia	Ignatiev, Vj. Microbiol. & Epidem., 1927, 2, p. 160.
Leporidae	40. Karroo hare *L. saxatilis.*	South Africa	Pirie, l.c.
	41. Zulu hare *L. zuluensis.*	South Africa	Pirie, l.c.
	42. Hare. *L. europaeus.*	England	Martin & Rowland, Observ. on Rat Pl. in East Suffolk 1910 & Bulstrode, L. G. B. Rep., 1910-11, p. 36.
	43. Rabbit *Oryctolagus cuniculus* (g)	England	Martin & Rowland, l.c.
Caviidae	44. Guinea-pig *Cavia cobaya.*	Sydney	Thompson, Rep. of the Board of Health on a second Outbr. of Pl. at Sydney, 1902.
		India	Liston, Jl. Bombay Nat. Hist. Soc., 1905, vol. 16, p. 253 & Jl. Hyg., 1908, vol. 7, p. 891.
		Manila	Schoebl, Phil. Jl. of Sc., 1913, vol. 8, p. 417.
		Senegal	Noc., Rep. sur le fonct. du Lab. de l'A. O. F. en 1919, Dakar, 1920.
	45. "Cuis" *C. aperea* (h)	Argentine	Uriarte & Gonzalez, C. R. Soc. Biol., 1924, vol. 91, p. 1040.
	46. Porcupine *Hydrochoerus capybara*	Mysore (India)	Bruce Low, L. G. B. Rep., 1898-01, p. 317.
Pedetidae	47. Spring hare *Pedetes caffer.*	South Africa	Mitchell, l.c., 1924, (Report).

Unclassified	48.	Black Marmot (c)	Semiretchinsk	Russian Publ. Health Rep., 1907, p. 162.
	49.	*Mus tamaricinus lasurus* (i)	South-East Russia	Nikanoroff, Bull. Off. Internat., d'Hyg. Publ., 1928, vol. 20, p. 537.
	50.	Jerboa (k)	Baku District	Milman, Russki Vratch, 1915, p. 351.
	51.	Field rodent (Mouse)	Khorassan (Persia)	Grekoff, quot. by Clemow, Lancet, 1913, i, p. 1697 & Guigiena & Epidemiol. 1924, vol. 3, No. 4, p. 50.
	52.	Field-rat	Rhodesia	Kinghorn, 1918, quot. Trop. Dis. Bull., vol. 13, p. 324.

Remarks : (*a*) One palm-rat was found infected in Senegal (see Laveau, Bull. Soc. Path. Exot., 1919, p. 291) In addition *Mus mauretanicus* and *Mus rufinus* are suspected (see text).

(*b*) Plague infection is said to have been found also in the *Peba gerbilles*.

(*c*) The existence of plague was not definitely proved in these two.

(*d*) Besides gerbilles field-rats seem to have been involved in Tunis.

(*e*) Gerbilles apparently also play a role in other parts of North Africa (see text).

(*f*) Possibly natural plague is also found in the *Dipodipus sagitta* (see text).

(*g*) That tame rabbits are apt to contract plague during an outbreak was recently again confirmed by Girard (Bull. Soc. Path. Exot., 1928, vol. 21, p. 299).

(*h*) A *Ctenomys* is also suspected (see text).

(*i*) The data available in regard to this rodent are not complete.

(*k*) Plague infection was proved also in some mice (species ?).

II. ADDITIONAL LIST OF RODENTS IN WHICH NO NATURAL PLAGUE HAS BEEN FOUND BUT WHICH ARE SUSCEPTIBLE TO ARTIFICIAL INFECTION. (REVISED TO SEPTEMBER, 1928).

Name :	*Locality* :	*Reference* :
1. *Mus agrarius*	Formosa	Kuraoka, Rep. of Saigon Conf. Trop. Med., 1913, p. 204.
2 Field mouse (a)	Formosa	Kuraoka, Rep. of Saigon Conf. Trop. Med., 1913, p. 204.
3. Striped Hamster *Cricetulus griseus*	China (Chihli)	Hsieh, National Med. Jl., 1919, vol. 5, p. 20.
4. Spermophilus (*Citellus*) (b) *mongolicus*	South Manchuria	Wu Lien Teh & Eberson, Jl. Hyg., 1917 p. 1.
5. Hamster *Cricetulus furunculus*	Transbaikalia	Jettmar, Jl. of Transbaik. Med. Soc. 1922, No. 2, p. 95 & Sukneff, Publ. of Harbin Med. School, No. 1, 1922, p. 213.
6. Rat-Hare *Ochotona dauricus*	Transbaikalia	Jettmar, Jl. of Transbaik. Med. Soc. 1922, No. 2, p. 95 & Sukneff, Publ. of Harbin Med. School, No. 1, 1922, p. 213.
7. *Spermophilus eversmanni*	Transbaikalia	Jettmar, Ztschr. f. Hyg. & Infekt.-Kr. 1923, p. 329.
8. *Citellus guttatus*	South-East Russia	Shurupoff, Russki Vratch, 1911, p. 1301 and Zentralbl. f. Bakt., 1912, vol. 65, p. 243.
9. Marmot *Marmota bobac* (c)	South-East Russia	Golov & Joff, Transact. 1927 Anti-Pl. Conf., p. 102.
10. Jerboa *Dipodipus sagitta* (d)	South-East Russia	Golov & Joff, Transact. 1927 Anti-Pl. Conf., p. 102.
11. *Micromys minutus*	South-East Russia	Golov & Joff, Transact. 1927 Anti-Pl. Conf., p. 102.
12 *Arvivola amphibius*	South-East Russia	Golov & Joff, Transact. 1927 Anti-Pl. Conf., p. 102.
13. *Mesocricetus eversmanni*	South-East Russia	Golov & Joff, Transact. 1927 Anti-Pl. Conf., p. 102.
14. *Dyromys nitedula*	South-East Russia	Golov & Joff, Transact. 1927 Anti-Pl. Conf., p. 102.
15. Alpine marmot *Marmota marmota*	Europe	Wurtz, quot. by Dujardin-Beaumetz & Mosny; Dujardin-Beaumetz & Mosny, C. R. Acad. Sci. 1912, vol. 155, p. 339.

16.	Lerot (Door mouse) *Myoxys murinus*	Senegal	Leger & Baury, C. R. Acad. Sci., 1922, Vol. 175 p. 734.
17.	*Acomys cahirinus*	Egypt	Todd in Petrie, Progress Rep. on the work of Pl. Invest. Staff in Upper E., 1911-12, Cairo, 1912, p. 20.
18.	Jerboa	Egypt	Todd in Petrie, Progress Rep. on the work of Pl. Invest. Staff in Upper E., 1911-12, Cairo, 1912, p. 20.
19.	"Root rat" *Tachyoryctes daemon.*	East Africa	Lurz, Arch. f. Schiffs-& Tropenhyg., Sept. 1913, p. 593.
20	Dwarf mouse *Leggada*	South Africa	Pirie, Publ. of the S. Africa. Inst. f. Med. Res., 1927, No. 20, p. 122.
21.	Fat mouse *Steatomys krebsi*	South Africa	Pirie, Publ. of the S. Africa. Inst. f. Med. Res., 1927, No. 20, p. 122.
22	Grey mole-rat *Cryptomys sp.*	South Africa	Pirie, Publ. of the S. Africa. Inst. f. Med. Res., 1927, No. 20, p. 122.
23	Water rat *Otomys irroratus*	South Africa	Pirie, Publ. of the S. Africa. Inst. f. Med. Res., 1927, No. 20, p. 122.
24	Cape hare *Lepus* capensis	South Africa	Pirie, Publ. of the S. Africa. Inst. f. Med. Res., 1927, No. 20, p. 122.
25.	Field mouse *Microtus californicus*	California	McCoy, Jl. Inf. Dis., 1909, June, p. 283.
26	California pocket gopher (e) *Thomomys bottae*	California	Ibidem & Jl. Inf. Dis., Jan. 1911, p. 42.
27	Chipmunk *Callospermophilus (Citellus) chrysodeirus*	California	McCoy, Jl. Inf. Dis., 1911, Jan., p. 42.
28.	*Ammospermophilus leucurus*	California	McCoy & Chapin, U.S. Publ. H. Bull., No. 53, 1912, p. 15.
29.	Rock squirrel *Citellus gramurus*	New Mexico	McCoy & Smith, Jl. Inf. Dis., 1910 p. 374.
30	Arizona prairie dog *Cynomys ludovician. arizonensis*	New Mexico	McCoy & Smith, Jl. Inf. Dis., 1910 p. 374.
31.	Eastern desert wood rat *Neotoma albifula angusticeps*	New Mexico	McCoy & Smith, Jl. Inf. Dis., 1910 p. 374.

Remarks: (*a*) "Field and forest mice" were found susceptible by Nuttal (Centralbl. f. Bakt., XXII/4).

(*b*) Preliminary experiments were made by Strong (Mukden Conf. Rep., p. 239) and by Shibayama (ibidem, p. 31).

(*c*) Experiments with the Ural tarabagan were carried out 1903 by Shurupoff (Russki Vratch 1911, No. 33, p. 1301).
Flu (Geneesk. Tijdschr. v. Ned.-Indie, 1914, No. 5, p. 540) performed experiments with marmots (?).

(*d*) *Possibly* found naturally infected.

(*e*) "Gophers are not sufficiently susceptible to infection with *B. pestis* to be of any importance from an epidemiological point of view" (Mc Coy).

III. FLEAS OF WILD RODENTS SUFFERING FROM SPONTANEOUS PLAGUE.

Locality:	*Host*:	*Fleas*:
Transbaikalia, etc.	Arctomys bobac	Oropsylla silantiewi*
	Alactaga mongolica	Ceratophyllus sp.
	Spermophilus dauricus	Ceratophyllus tesquorum*
Transcaspia	Rhombomys opimus	Xenopsylla gerbilli X. hirtipes X. skrjabini Coptopsylla sp. Ceratophyllus fidus C. laeviceps* C. tersus Ophthalmopsylla volgensis* Ctenophthalmus dolichus Rhadinopsylla cedestis
India	Nesokia bengalensis	Xenopsylla cheopis*
	Sciurus palmarum	Xenopsylla cheopis* Fleas similar to Ceratophyllus fasciatus
South-East Russia	Citellus fulvus	Oropsylla ilovaiskii*
	Citellus pygmaeus	Ceratophyllus tesquorum* Neopsylla setosa* Ctenophthalmus breviatus Ct. pollex Ct. orientalis Frontopsylla semura*
	Dipodidae	Ophthalmopsylla volgensis* O. kasakiensis* Mesopsylla hebes* M. lenis* M. tuschkan
	Microtinae	Ceratophyllus consimilis* Amphipsylla rossica A. prima

	Gerbillinae	Xenopsylla mycerini* Ceratophyllus laeviceps*
	Cricetus cricetus	Ctenophthalmus breviatus Ct. orientalis Ct. wagneri
England	Oryctolagus cuniculus	Spinopsyllus cuniculi* Ceratophyllus fasciatus*
	Lepus europaeus	Spinopsyllus cuniculi*
South Africa	Tatera lobengulae	Dinopsyllus lypusus* Chiastopsylla rossi* Xenopsylla eridos* Listropsylla stygius Ctenophthalmus calceatus Leptopsylla musculi
	Mastomys coucha	Dinopsyllus lypusus* Xenopsylla eridos* Chiastopsylla rossi* Xenopsylla brasiliensis*? P. irritans*
	Rhabdomys pumilio	Dinopsyllus lypusus* Xenopsylla eridos* Chiastopsylla rossi* Listropsylla agrippinae L. chelurae Chiastopsylla octavii Dinopsyllus longifrons
	Malacothrix typicus	Dinopsyllus lypusus* Xenopsylla eridos* Listropsylla stygius
	Geosciurus capensis	Xenopsylla erilli* Ctenocephalus canis* Echidnophaga bradycta E. gallinaceus Xenopsylla eridos* Dinopsyllus lypusus* Pulex irritans*
	Paratomys luteolus	Chiastopsylla pitchfordi Listropsylla agrippinae Dinopsyllus longifrons Xenopsylla eridos* Chiastopsylla rossi*
	Pedetes caffer	Xenopsylla caffer
	Desmodillus auricularis	Xenopsylla eridos* ? Dinopsyllus lypusus*
	Myotomys broomi	Chiastopsylla mulleri Listropsylla agrippinae Chiastopsylla rossi* Xenopsylla eridos* Hypsophthalmus aganippea

	Lepus zuluensis L. saxatilis	Ctenocephalus canis* Xenopsylla eridos*
	Mystromys albicaudatus	Dinopsyllus lypusus* Chiastopsylla rossi* Xenopsylla cheopis*
Central Africa	Mastomys coucha ugandae	Xenopsylla brasiliensis*? X. cheopis* Dinopsyllus lypusus*
	Arvicanthis abyssinicus nubilans	Xenopsylla brasiliensis*? Dinopsyllus lypusus* Xenopsylla cheopis*
	Petomys fallax iredescens Epimys dolichurus	Xenopsylla cheopis* Leptopsylla musculi* Ceratophyllus fasciatus*
Gold Coast	Cricetomys gambianus	Xenopsylla aequisetosus X. nubicus X. cheopis*
French West Africa	Golunda campanae Mastomys coucha	Xenopsylla cheopis*
Upper Egypt	Arvicantnis niloticus	Xenopsylla cheopis*
California	Citellus beechyi	Ceratophyllus acutus* Hoplopsyllus anomalus*

Remarks: * Known to bite man.

*? Jorge (3) states that *X. brasiliensis* "is the most common species in Uganda and Kenya and appears to be capable to transmit plague to rats and man since this was practically the only flea met with on the black rats in the huts of the infected district Kabete."

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STUDIES UPON THE MORBID HISTOLOGY OF PNEUMONIC PLAGUE.

[Comment on the material from the 1920-21 Manchurian epidemic of pneumonic plague based to a certain extent on the study of material from the 1911 epidemic and literature. With four micro-photographs.]

BY MAJOR GEO. R. CALLENDER, M.D. (*Curator, Army Medical Museum, Washington, D.C.*)

Editorial Note.—In April 1925 I spent a few very interesting days with Major Geo. R. Callender, Curator of the Army Medical Museum, Washington, D.C., who before his present appointment had done considerable work on Plague in the Philippines.

Major Callender and I freely exchanged ideas on the pathological aspect of pneumonic plague, and on my return to Harbin I sent him selections of *postmortem* material collected during the 1921 epidemic in Manchuria, so that I might obtain his expert independent opinion upon the morbid histology of pneumonic plague.

The results of Major Callender's investigations of the material thus sent him reached me in January 1927, some months after the appearance of our 1925-6 Reports.

I therefore take pleasure now, with the Major's permission, to embody his thorough and painstaking studies in the present Volume of our Manchurian Plague Reports.

It will be seen that the conclusions arrived at by Major Callender as to the mode of invasion of the virus into the system do not coincide with my own. I doubt whether this question can be settled by purely histological investigations, however careful these may be undertaken. In my opinion, for a true recognition of the portal of entry, the epidemiological and experimental evidence has to be considered as well as the strictly morphological findings.

All the same, I wish to express my deep obligation to Major Callender for the keen interest he has shown in the matter and for the very fine work he has turned out.

W. L. T.

It would appear from the material studied, that the method of infection common to this disease is by way of the respiratory tract. While in some instances organisms may make direct entrance to the pulmonary parenchyma, it appears that more frequently, if not always, the organisms enter through the mucous membrane of the upper respiratory tract, tonsils, pharyngeal wall, tracheal and upper bronchial mucosa, there, producing greater or lesser, many or few lesions, the organisms make their way into the lymphatics, thence to the hilus of the lung, where changes are produced in the local lymph-nodes. Directly and by over-flow from these structures the organisms enter the thoracic duct and the right heart, and from there are disseminated throughout the pulmonary tissue. To a certain extent, as in pneumonia, the organisms make their way out into the lung along the perivascular and peribronchial lymphatics; the normal direction of the flow toward the hilus being blocked by the lesions already produced in the nodes. Apparently in this manner, the lobular and

confluent types of pneumonic consolidation are produced, at least in a great many instances, as the oldest lesions appear to be nearest the hilus. By means of blood stream dissemination embolic processes occur giving rise to the nodular type while the isolated lobular involvements in terminal anatomical lobules may conceivably be produced in either way; those accompanied by advanced lesions in the tissues separating the lobules probably arising from lymphatic rather than vascular extension.

Involvement of the pleura apparently regularly follows whenever any type of lesion reaches the pleural surface. It probably also is caused by direct extension out from the mediastinal lymphatics at the hilus of the lung as is seen in pneumonia due to other organisms. In a similar way mediastinal lymphatics are frequently involved and thus the pericardium and thymus are undoubtedly affected although blood stream involvement cannot be entirely ruled out. It is noted however, in examining these tissues, that the organisms appear massed in lymphatics rather than plugging blood vessels.

In the extremely acute cases it would appear that organisms in a finely divided state and in enormous numbers and presumably of enhanced virulence are distributed throughout the body by means of the blood stream producing no definite focal lesions, for it is in these acute cases that evidences of invasion with this organism are seen most widely scattered throughout the body, the patient apparently dying from septicemia before localization in the sense of producing focal lesions can occur.

In the slower forms of the disease secondary lesions are produced probably as the result of an over-flow of organisms from the lung to the general circulation. As a result, the secondary lesions may differ to a considerable degree depending to a large extent upon the size of the bacterial emboli. In such cases there may be little evidence of pyemic process, the lesions in the parenchymatous organs being almost purely degenerative in type. In the kidney, the material so far examined has not shown embolic lesions while those in other organs indicate that the emboli are relatively small.

LESIONS.

Fulminant Cases.

In this form one will find necrotic lesions of small size in the tonsil, in the pharyngeal mucosa and in the bronchial mucosa. The bacilli appear somewhat disseminated in the lymphatics, rarely in large groups.

Lung: In this organ, the lesions are characterized by vascular engorgement, little escape of blood into the alveoli, edema, hyalin degeneration and leucocytic infiltration of the peribronchial tissues and alveolar walls, the most advanced lesions appearing in the upper respiratory tract where some desquamation of bronchial epithelium may occur.

Spleen: Usually shows somewhat more advanced lesions which are characterized by a leucocytic infiltration, edema and vascular engorgement most intense immediately surrounding the lymph nodules.

Liver: Aside from engorgement and degeneration of varying degrees, the capillaries are more or less filled with leucocytes with occasional small embolic lesions in the capillaries of the intermediate zones.

Kidney: Degenerative changes in all parts of the kidney structure occur, although appearing to be more marked in the convoluted tubules actually the most intense destruction is evidenced in the glomeruli by swelling, degeneration and necrosis of the capillary endothelium and the epithelial cells.

Lobular or confluent pneumonic form. Upper respiratory tract. Lesions apparently not more numerous but more advanced, necrosis more evident, larger masses of bacteria present both in the tissues and in the lymphatics; necrotic pseudomembrane may form; desquamation, necrosis and hemorrhage are more characteristic. Hilus lymph nodes show more advanced changes with necrosis and the lesions appear to extend out into the tissues surrounding these nodes. In this manner lymphogenous extensions are found in the mediastinal tissues including the thymus.

Lung: In this organ the lesions vary from the engorgement stage of pneumonic consolidation with the escape of serum and bacteria only into the alveoli to complete necrosis of the pulmonary parenchyma. All of these stages may be seen in any one case by studying sections from the center of one of the confluent pneumonic areas to the periphery. In no two cases are the lesions exact duplicates as congestion, leucocytic infiltration and necrosis all vary. In the center bacteria will be found in the tissue and air spaces in quantities varying to a considerable degree but never in small numbers. The contents of the alveoli aside from bacteria will consist of leucocytes and cellular debris in varying proportions. Sometimes leucocytes are as numerous as in typical lobar pneumonia, while at other times they form a small proportion of the exudate. Hemorrhage is pronounced only in those cases with a relatively small amount of leucocytic response. No good examples of the hemorrhagic type of lesion were seen in the 1920 cases though the 1911 epidemic showed lesions of this nature. As one approaches the periphery of confluent lesions consolidation is less complete and engorgement is more evident. The alveolar walls in the central portions appear completely necrotic no nuclei being visible, farther out connective tissue nuclei alone are seen and it is only when we reach the comparatively normal lung that the nuclei of endothelial and epithelial cells lining the alveoli can be differentiated. The toxin appears to specifically affect endothelial cells causing them to swell and undergo degeneration to necrosis.

In the lesions confined to the anatomical lobules one usually finds an evenly consolidated area sharply limited by the connective tissue of the interlobular septa. This consolidation varies as does that previously described but in any one area is uniform. The interlobular septa usually show massive leucocytic infiltration with enormous numbers of bacteria, the lymphatics are blocked, the connective tissue is swollen and nuclei have to a large extent disappeared.

Pleura: In the early stages of its involvement shows intense engorgement, hemorrhage, leucocytic infiltration and necrosis of its mesothelial covering. Fibrin which is in very small quantities in the parenchyma, sometimes being apparently absent, is more abundant in the pleural lesions and forms a plastic exudate on the surface, later becoming infiltrated with leucocytes. In addition to this, it is quite usual to find definite hemorrhages where no evidence of inflammatory reaction appears.

Spleen: The lesions in the spleen may resemble those previously described. In most of the tissues examined the lesions were variations of those seen in the fulminant type. The follicles appeared small, capillaries about them more or less dilated with blood and infiltrated with few to many leucocytes. In many instances there appeared a definite necrotic zone immediately surrounding the small lymph nodule.

In this area connective tissue fibers appeared increased but were markedly swollen and infiltrated with rather numerous bacteria. Fibers of the blood vessel wall were frequently swollen and hyalin though this was not always encountered. In two cases of the 1920 series, P. M. Nos. 7 and 23, a somewhat different lesion was seen. This consisted in a proliferation of the large cells of the follicles, the lymphoid elements being displaced to the periphery. At the periphery lesions similar to those seen in other cases were seen. It cannot be definitely stated that this proliferation of the epithelioid type of cell was caused by the plague although a considerable number of organisms are found in this tissue while the cells themselves showed definite degenerative changes. It is quite possible that some other condition was present in the individual at the time of the onset of pneumonic plague which was responsible for the epithelioid proliferation in the follicles.

Liver: In some instances the liver was found to be the same as in the fulminant cases. In other cases only degenerative changes were seen. In a few cases the nodules described by Wilm were evident and consisted of groups of epithelioid cell, leucocytes, most of which were of the lymphoid type and greater or lesser numbers of bacteria. These nodules appear to surround small capillaries containing hyalin bacteria. They were located in the intermediate zone and sometimes contained fairly numerous red cells. Nuclei of liver cells in immediate proximity failed to stain. These undoubtedly are embolic lesions and are described as much larger in cases examined by some investigators.

Kidney: The lesions vary in degree from moderate degenerative changes to cases in which a large proportion of the nuclei of the convoluted tubules and of the high type tissue in the glomeruli fail to stain.

Uterus of pregnancy: In the one case examined in which a still birth had occurred shortly before death, masses of leucocytes and bacteria were found scattered through the blood of the engorged uterine sinuses and occasionally in the lymphatics surrounding. The placental tissue in this case showed no definite lesions in the material available for examination.

Brain: In one case the brain showed embolic or pyemic lesions extending out from the blood vessels of the meninges. This condition could undoubtedly occur as easily as any of other septicemic manifestations.

SUMMARY.

The differential characteristics of the lesions of this organism are its tendency to produce rapid necrosis wherever it localizes, and this to a greater extent than any other organism with which we are familiar; the most important differential diagnostic point, however, is the relatively enormous numbers of organisms which are seen in the lesions. The lesions seen in the influenza epidemic reproduced to a considerable degree those seen in pneumonic plague although necrosis as a rule was not so evident and certainly required a greater length of time to produce. The numbers of organisms however in the lesions can never compare with the quantity seen in cases of pneumonic plague.

Accession No. 23815

"P. M. 1. Man aet? (Middle aged). Feb. 21, 1921. Patient had shown suspicious bacilli in sputum, but no blood on the day before death. Died Feb. 24.

Marked congestion upper respiratory tract.

Persistent thymus.

Trachea and bronchi contain much froth, mucosa congested.

Pleura and lungs: Old pleural adhesions of right lung and left upper lobe. Pleura of right upper lobe typically changed (thickened, yellow in color with hemorrhages beneath). Calcified tubercular nodes in left upper lobe. Extensive pneumonic area, partly in stage of grey and partly of red hepatisation in right upper lobe. Confluent lobular pneumonia in right lower lobe. Over this pleura cloudy with fine hemorrhages. Rest of lungs congested.

Bronchial glands, especially right side, show congestion besides anthracosis.

Liver: Nut-meg appearance.

Spleen: Indurated; neither enlarged nor congested. Follicles and trabeculae visible; part of the former, size of pin-head, are surrounded by hyperemic zone.

Kidneys: Swollen, hyperemic, cortex enlarged. Few hemorrhages in pelvis."

Sections received: Histological; liver and trachea.

HISTOLOGICAL EXAMINATION.

Liver: Moderate parenchymatous degeneration and leucocytic infiltration.

Trachea: Catarrhal inflammation of mucous membrane with small necrotic areas filled with bacteria immediately beneath the muscularis mucosae.

Comment: None.

Accession No. 23816.

"P. M. 2. Girl, aet. 16. March 1. Former prostitute, died on Feb. 28, having shown typical symptoms of pneumonic plague.

Well developed dead body.

Marked congestion upper respiratory tract.

Thymus large.

Trachea and bronchi much congested with some submucous hemorrhages.

Pleura and lungs: Extensive pneumonic area in right upper lobe, mainly in stage of grey hepatisation. Pleura over this area typically changed[1] (as in Case I). Lobular confluent pneumonia in other lobes of lungs; pleura over some foci shows fine fibrin nets and large hemorrhage.

Bronchial glands only slightly enlarged, anthracotic, not markedly hyperemic.

Heart: Right chamber distended.

Liver: Swollen, degenerated Hemorrhages beneath capsule.

Spleen: Moderate acute enlargement; not markedly soft or congested. Follicles visible.

Kidneys: Early fatty changes."

Sections received: Thymus.

HISTOLOGICAL EXAMINATION.

Thymus: Fairly cellular, some calcification of Hassall's corpuscles, some vascular engorgement.

Comment: None.

Accession No. 23818

"P. M. 7. Boy, aet. 8. March 18.

Well developed body. On skin over left hypogastrium a tuberculous ulcer size of a silver dollar.

Mucosa of upper respiratory tract shows marked congestion. Tonsils not enlarged. Big thymus.

Mucosa of larynx and trachea much congested, covered with frothy sputum.

Pleura and lungs: Upper and middle lobe adherent through fibrinous membranes. Over pneumonic area in upper lobe pleura typically changed. Pneumonic patch of greyish—red colour in upper lobe.

Peritracheal and bronchial glands: Those on right side enlarged, showing besides anthracosis more or less congestion.

Heart: Especially right chamber distended, musculature pale.

Hemorrhages beneath the pleural surface of *diaphragm*.

Liver: Enlarged, congested. Some "white spots" on surface.

Spleen: Enlarged, congested; follicles as in Case I.

Kidneys: Parenchymatous degeneration.

Brain: Pachymeningitis. Congested."

Sections received: Histological: kidney, liver and spleen, adrenal, small intestine.

HISTOLOGICAL EXAMINATION.

Kidney: Advanced parenchymatous degeneration and vascular engorgement, no evidence of fat change.

Adrenal: Necrosis of the central half of the glandular portion without other recognizable change. (Possibly postmortem).

Liver: Acute passive hyperaemia, moderate leucocytic infiltration, occasional small epithelioid nodules in intermediate zones, leucocytic infiltration is irregular in distribution. Tissue stains fairly well.

Spleen: Marked increase in blood with hyalinization of many capillary masses. Marked polymorphonuclear infiltration, marked epithelioid proliferation in follicles with degeneration; in the proliferated areas are many macrophage cells engulfing leucocytes. Lymphoid cells of the follicles generally very markedly decreased.

Ileum: Epithelioid proliferation in the follicles, otherwise no marked change.

Comment: Septicemic lesions accompanying pneumonic consolidation.

Accession No. 23803

"P.M. 5. Woman, age 22. March 8. Mother of above.

Big, well developed, stout woman .

Marked congestion upper respiratory tract.

Persistent thymus.

Larynx and trachea marked congestion, much froth.

Pleura and lungs: Pleura over right middle lobe typically changed. Pleura over upper part of right lower lobe opaque with hemorrhages. Fibrin nets and hemorrhage at places over the inflamed area in the other lobes. Extensive pneumonic area, mainly in stage of grey hepatisation, in right middle lobe. Extensive area, mainly in stage of red hepatisation, in right lower lobe. Lobular confluent pneumonia in the other lobes.

Peribronchial glands: Especially right side markedly swollen, anthracotic, partly congested.

Many hemorrhagic spots on the *diaphragm*.

Heart: Enlarged, distended. Lipomatosis.

Liver: Parenchymatous degeneration. Some "white patches."

Spleen: Moderately enlarged, congested. Follicles as in Case I.

Kidneys: Advanced degenerative changes.

Oesophagus: Slight congestion of lowest part.

Sexual organs congested."

Sections received: Lung, uterus, ovary, thyroid, liver, kidney.

Lung: Sections show all vessels filled with bloody fluid. Tissues contracted, firm and hemorrhagic. One section shows beginning mottling of necrosis. The other one is purely hemorrhagic while the third is nearly completely necrotic and represents the lower portion of the upper lobe. There is some fibrinous exudate on the interlobar pleura.

Bronchi—Mucosa somewhat hemorrhagic.

HISTOLOGICAL EXAMINATION.

Lung: Sections from confluent necrotic areas show alveoli filled with leucocytes, some fibrin and many bacteria, alveolar walls necrotic. Sections on the periphery of these areas show fewer leucocytes, less necrosis and more congestion until the neighboring lung is reached where but few leucocytes are present and most of these are in the alveolar walls. Marked congestion and some edema characterizes the picture. The pleura shows edema, some leucocytic infiltration in the vicinity of the necrotic areas and hemorrhagic foci throughout but more noticeable away from the more dense consolidation. Bacteria are most abundant in the more densely consolidated portions. Bronchial mucosa shows desquamation of the cells and the presence of a necrotic exudate, the basement membrane being swollen and hyalin while the surrounding vessels are engorged and several of the veins appear thrombosed. Peribronchial lymph-nodes are congested, infiltrated with leucocytes with which some of the lymph vessels are completely filled.

Uterus: Vascular engorgement, numerous hyalin vessels, mucosa somewhat atrophic, no signs of inflammation.

Ovary: Some sclerotic in places, rather vascular, a few normal appearing follicles.

Thyroid: Alveoli appear considerably dilated, filled with deep staining colloid.

Liver: Congestion and parenchymatous degeneration of moderate degree.

Kidney: Rather extreme degeneration of the tubules of the granular type.

Comment: Confluent bronchopneumonic type with final involvement of all parts of the lung in the various stages of pneumonic plague.

Accession No. 23813

"P.M. 6. Woman, aet. 17. March 14. Patient had been sent to isolation on March 6 after death of husband. On completion of isolation (5 days) she felt giddy when having bath, but walked to her home. Here she got worse, was sent to hospital in the evening. During isolation period, temperature was normal. When admitted on evening of 11th, very ill, but no sputum. On 12th morning found goaning, lying on back; cough with bloody sputum. *B. pestis* ++++. Died early on 13th.

Big woman, well nourished, much abdominal fat. Some ecchymoses on skin.

Congestion of upper respiratory tract not so marked.

Persistent thymus.

Larynx and trachea marked congestion.

Pleura and lungs: Front surface of right middle lobe covered with a layer of pus. Beneath this typically changed pleura to be seen. Extensive pneumonic area in right middle lobe, showing a larger and a smaller grey portion, while almost the whole remainder of the lobe is in the stage of red infiltration. Lobular confluent pneumonia in right lower lobe, central patch in left lobe.

Peribronchial glands: Those on left side show no marked changes apart from small calcified area in one. Those on right side are enlarged, partly congested. In some, a few hemorrhages and small greyish areas on section.

Heart: Especially right chamber distended. Musculature pale.

Liver: Marked congestion.

Spleen: Enlarged, congested. Trabeculae and follicles visible, latter as in Case I.

Kidneys: Congested, cortex enlarged.

Oesophagus: Lowest part hyperemic.

Uterus: Some congestion."

Sections received: Lung, gross and histological. Gross material presents a poorly defined confluent bronchopneumonia.

HISTOLOGICAL EXAMINATION. (6 Sections).

Lung: 1. Fairly even consolidation, numerous leucocytes, moderate compression of alveolar walls, necrosis of endothelium and epithelium, alveolar walls still show fair staining of connective tissue nuclei, congestion of considerable degree present. Pleura, beginning fibrinous exudate with some necrosis in subserosa.

2. Lung including bronchus: Moderate degeneration and some desquamation of bronchial mucosa; marked perivascular and peribronchial edema with congestion, some hemorrhage and necrotic bronchopneumonia.

3. Second section same with some areas of the lung not firmly consolidated.

4. Another section, engorgement and beginning exudation in one lobule, rather dense leucocytic consolidation in another. Irregular patches of necrosis, capillary hemorrhages beneath the pleura.

5. Another section, fibrinous exudate on secondary bronchus. interstitial pneumonia with edema, most of the leucocytes being mononuclears, some diapedesis in alveolar walls.

6. Another section of lung—In a stage of engorgement and edema with varying degrees of leucocytic infiltration.

Comment: A rapidly extending confluent hemorrhagic bronchopneumonia.

Accession No. 23811

"P.M. 9. Woman, mother of still-born, died 23rd March, 1921.

Well built woman, age 31. Cyanosed face.

Tongue slight red; *Tonsils* swollen and congested. Papillae of *pharynx* show similar changes.

Larynx and *Trachea* much congested, little mucosa.

Lungs: Anterior surface showed film of semi-purulent mucus, emphysematous. Right upper lobe pneumonic patch. Bronchopneumonia in right lower lobe. Rest congested.

Left lung markedly congested, no patches.
Peribronchial glands swollen and anthracotic.
Right upper lobe, pneumonic patch. Bronchopneumonia.

Heart: Much clear pericardial fluid. Much enlarged. Coronary vessels enlarged, aorta normal.

Diaphragm: Many hemorrhages, specially over liver areas.

Liver: Swollen; marked fatty degeneration. White patches especially in right side.

Spleen: Enlarged, much congested.

Kidneys: Right much enlarged and congested with cloudy swelling; pelvis filled with urine.

Left same, only slightly less marked.

Cortical vessels enlarged and congested.

Uterus: Much enlarged, not reduced after labour. Many big clots inside. Haemorrhages on surface of uterus and left ovary.

Ovaries, tubes and ligaments congested."

Sections received: Umbilical cord, ovary, liver, uterus, placenta.

Placenta: Shows nothing definite.

HISTOLOGICAL EXAMINATION.

Umbilical cord: No definite pathology.

Ovary: Corpus luteum of pregnancy, one blood vessel contains purulent clot, ovarian follicles and ova appear normal.

Liver: Fixation fair, leucocytic infiltration, granular degeneration and congestion of a moderate degree are present.

Uterus: Section of bladder wall and uterus including placental site—remnants of decidua in the glands, leucocytic infiltration of the mucosa, muscular tissue of the bladder wall edematous and injected, submucosal capillaries and lymphatics contain numerous bacteria.

Placenta: Considerable thickening of the vessels, no definite lesions. Two other sections of uterus, but no definite lesion seen.

Comment: The infection evidently involves the maternal terminal vessels in the uterus. No foetal tissue available for examination.

Accession No. 23814

"P.M. 10. Man, aet. 35. March 24. Patient had been admitted on 22nd. Bloody sputum on 23rd, died at 8 p.m. same day.

Congestion of upper respiratory tract not marked. Tonsils somewhat enlarged, without other macroscopic changes.

Persistent thymus.

Larynx and trachea slight congestion. Minute submucous hemorrhage.

Pleura and lungs: Some old adhesions left lower lobe. Parietal pleura intensely congested. In right upper lobe under typically changed pleura an extensive pneumonic area of greyish-red color. Confluent lobular pneumonia in left upper lobe.

Peribronchial glands: Only moderately enlarged, not markedly congested, anthracotic.

Heart: Some cloudy fluid in pericardium. Many hemorrhages beneath visceral layer of pericardium. Right side of heart distended. (Hypertrophia of both ventricles, atheroma aortae.)

Hemorrhages beneath pleural surface of *diaphragm*.

Liver: Much congested; at places hemorrhage beneath capsule.

Spleen: Swollen, very soft. Follicles only partly visible, partly surrounded by hemorrhagic zone. Trabeculae visible.

Kidneys: Some hemorrhages beneath capsule and in pelvis. Parenchyma yellowish, fatty, vessels deeply injected.

Oesophagus: Lower part somewhat hyperemic.

Parietal peritoneum shows congestion and some hemorrhage."

Sections received: Small pieces of tissue from kidney, liver, spleen, thymus.

HISTOLOGICAL EXAMINATION.

Kidney: Advanced granular degeneration with marked swelling of the cells of convoluted tubules and Henley's loop. Granular swelling and hydropic change with marked increase in blood contents in all glomeruli.

Liver: All sections show moderately advanced granular degeneration with deep eosin staining material in the sinusoids. There is a considerable degree of polymorphonuclear infiltration seen in the sinusoids, much more marked in some sections than in others. There are many small foci or collections of epithelioid cells largely in the intermediate zones in all sections. These consist of a central group of epithelioid cells with a few lymphocytes, plasma cells and polymorphonuclear leucocytes.

Spleen: Shows all blood spaces distended and the entire spleen crowded with polymorphonuclear leucocytes. Malphigian bodies seem rather small in size and each one is surrounded by a deep eosin staining homogeneous zone which under power is seen to consist of partially disintegrated red blood corpuscles forming what morphologically and pictorially. is a hyalin clot or thrombus.

Thymus: Shows some vascular engorgement, the majority of the Hassall's corpuscles are hyalin and many of them contain lime salts. Here and there throughout the section are dense masses of bacteria in the perivascular lymphatics. There is always considerable hemorrhage in the vicinity of these bacterial plugs.

Comment: A septicemic lesion accompanying extensive pneumonia.

Accession No. 23812

"P.M. 11. Adult man. March 25. Admitted for undoubted plague (bloody sputum) on March 16. Sputum contains many plague bacilli. Was fairly well up to 23rd. On 24th morning still conscious, died suddenly of heart failure at 12.30 p.m.

Well developed man. Much abdominal fat.

Upper respiratory tract shows very marked congestion.

Thymus persistent, but small.

Larynx markedly congesttd with submucous hemorrhages. Trachea not so markedly congested, contains no froth. Bronchi congested, contain froth.

Pleura and lungs: Emphysema. Central patches in right upper lobs; superficial patches under typically changed pleura in right middle lobe; lobular confluent pneumonia in right lower lobe, pleura partly changed, partly showing slight hemorrhages. In left upper lobe patches as in right middle lobe. Left lower lobe only congested.

Peribronchial glands : Enlarged, especially on right side, anthracotic, but not markedly congested.

Heart : In systole. Beginning atheroma of aorta.

Few hemorrhages in *diaphragm*.

Liver : "White patches" of irregular shape. Fatty degeneration.

Spleen : Much enlarged, congested, very soft. Follicles as in Case I. Near caudal pole an anemic infarct, size windsor bean, surrounded by a hemorrhagic zone.

Kidneys : Marked congestion.

Stomach : Submucous hemorrhages near pylorus.

Colon : Mucosa of normal appearance.

Pancreas : Few hemorrhages in substance."

Sections received : Pancreas, liver, kidney, pharynx, trachea, tonsils, lungs.

Lungs : One section shows small necrotic foci. Another section is uniformly consolidated. a reddish grey in color; other tissues, histological material only.

HISTOLOGICAL EXAMINATION.

Pancreas : Seems rather cellular

Liver : Some postmortem change, granular degeneration and moderate acute passive hyperaemia, irregularly distributed leucocytic infiltration. Fairly advanced parenchymatous degeneration and moderate to marked congestion and leucocytic infiltration. Occasional portal radicals show purulent thrombosis.

Kidney : Congestion moderate, moderate granular degeneration, congestion most marked in peripheral cortex.

Pharynx and trachea : Some desquamation of tracheal mucosa with increase of leucocytes, marked vascular congestion in upper end of oesophagus.

Tonsils : Evidence of old inflammation, recent vascular engorgement, no definite lesions.

Lungs : Marked degeneration and bacterial infiltration of walls and surrounding tissues of small blood vessels, beginning fibrinous pleuritis with marked sub-pleural hemorrhage and necrosis and vascular congestion of subserosa. Lung shows varying stages of consolidation from engorgement and leucocytic infiltration with edema to almost complete filling of alveoli with serum and leucocytes, bacteria and hemorrhage, with more or less complete necrosis of alveolar walls. Dense leucocytic infiltration of interlobular spaces with hemorrhage and massing of bacteria. In some sections there is considerable fibrino- -purulent exudate on the pleura, and in such the consolidation is advanced to a considerable degree towards complete necrosis in the lung itself.

Comment : Confluent broncho-pneumonic type shading off into the congestive stages in the more recently affected portions of the lung. General septicemic lesions in parenchymatous organs.

Accession No. 23807

"P. M. 12. Boy, aet 10. March 30. Died on 29th.

Body of fairly well developed boy.

Upper respiratory tract shows marked congestion.

Thymus large.

Trachea moderately congested, more markedly near bifurcation. Little froth.

Lungs and Pleura : Old adhesions right side. Left cavity contains considerable quantity blood tinted fluid. Walnut-sized calcified area in right upper lobe. Extended pneumonic area, mainly in the stage of red hepatisation, in left lower lobe. Pleura over it typically changed. Fibrino-purulent matter in sulcus between left lower and upper lobes.

Peribronchial glands : Enlarged, partly congested with hemorrhages. In one gland tuberculosis caseosa.

Heart : Pericard contains a small amount of fluid. Heart in systole.

Liver : Fatty degeneration. "White patches" on surface.

Spleen : Enlarged, congested. Follicles as in Case I.

Kidneys : Advanced degenerative changes.

Stomach : No marked macroscopic changes.

Brain : Both meninges and brain substance extremely congested."

Sections received : Lung, spinal cord, cerebrum, cerebellum.

Lung : Confluent bronchopneumonia. Fibrinous pleurisy.

HISTOLOGICAL EXAMINATION.

Lung: Fairly advanced degeneration of the bronchial mucosa with some hyalin change in the submucosa. Confluent bronchopneumonia characterized by almost complete necrosis of alveolar walls, granular exudate containing numerous bacteria and many leucocytes, the latter never filling more than 25% of the alveolus. Beginning involvement of the pleura in places.

2nd Section. Essentially similar picture. One portion of the section showing more advanced necrosis, particularly necrosis of the blood vessels.

Spinal cord: Some nerve cell degeneration but no characteristic changes.

Cerebrum: Vascular engorgement, purulent infiltration of meninges in one area in a sulcus

Cerebellum: Congestion of meninges.

Comment: Confluent bronchopneumonic type with fairly uniform but incomplete consolidation; septicemic secondary lesion in the meninges.

Accession No. 23801

"P. M. 13. Well developed woman, aet. 24. April 5.

Skin cyanotic.

Upper respiratory tract markedly congested.

Persistent thymus.

Trachea only moderately congested. Mucosa at bifurcation darker, much froth.

Pleura and lungs: Fine old adhesions right side. Left cavity contains considerable amount of fluid. Extensive pneumonic area in right upper lobe. Central pneumonic patch in right lower lobe, pneumonic patches in left upper lobe, lobular pneumonia in left lower lobe. Pleura over most of these areas typically changed.

Peribronchial glands: Partly markedly enlarged, anthracotic; in one a calcified tuberculous area.

Heart: In systole.

Liver: Degenerated with "white patches."

Spleen: Slightly enlarged, markedly congested and soft. Follicles not visible.

Kidneys congested. Some small hemorrhage in pelvis.

Stomach: Mucosa swollen; small submucous hemorrhage.

Ileum and colon show slight congestion. Mesenterial vessels congested, mesenterial glands slightly enlarged, congested.

Suprarenals: No marked macroscopic changes.

Sexual organs: Endometritis. Ovaries somewhat congested, tubes and ligaments more markedly so. Some small hemorrhage beneath mucosa *bladder*."

Sections received: Lung and bronchus.

Lung: Both sections show a nodular type of bronchopneumonia with confluence of some areas. The nodules show various stages of necrosis while the remaining lung tissue is hemorrhagic and partially consolidated.

Bronchi hemorrhagic with apparent partial desquamation of epithelium.

HISTOLOGICAL EXAMINATION.

Bronchus: Complete desquamation of epithelium; basement membrane covered with layer of hyalin tissue and sero-fibrinous exudate, vascular engorgement; attached lymph-node shows necrotic areas near peripheral sinuses. Edema in surrounding tissue.

Lung: The nodular consolidated areas show a purulent exudate in the alveoli with necrosis of the walls. Numerous bacteria. The nodules occur in anatomical lobules, the surrounding connective tissue trabeculae are edematous and infiltrated with leucocytes. Adjoining alveoli show congestion, serous and sero-purulent exudate which contains numerous bacteria, while the walls contain leucocytes, dilated capillaries and show necrosis of alveolar epithelium.

Comment: Confluent bronchopneumonia type, these having existed an appreciable length of time.

Accession No. 23817

"P. M. 16. Man, aet. 24. Admitted into hospital April 12. Patient who showed exquisitely staggering gait, died ½ hour after admission, was dissected 3 hours after death.

Rigor mortis. Cyanotic patches all over skin.

Moderate congestion upper respiratory tract.

Thymus persistent? (mainly fatty involution of organ).

Epiglottis and larynx show only moderate congestion. Trachea full of froth, but mucosa only pink in colour.

Pleura and lungs: Layer of fibrinous exudate over upper lobe of right lung. Locular confluent pneumonia in right upper lobe, pleura here typically changed. Pneumonic patches in right lower and left upper lobes, central patch in left lower lobe.

Peribronchial glands: Moderately enlarged, anthracotic, markedly congested.

Heart: On visceral layer of pericardium near right auricle a few hemorrhages. Corresponding to this a few hemorrhages beneath epicardium. Heart in systole.

Liver: Large, indurated. "White patches" on surface.

Spleen: Much enlarged, congested, soft. Follicles indistinct.

Kidneys: Congested. Some hemorrhages pelvis.

Brain: Oedema and congestion."

Sections received: Histological; kidney and liver.

HISTOLOGICAL EXAMINATION.

Kidney: Advanced degeneration and beginning necrosis of tubules, hydropic change in glomeruli, considerable serum in capsules, considerable vascular engorgement.

Liver: Acute passive hyperemia, fairly advanced fatty degeneration, considerable postmortem change.

Comment: None.

Accession No. 23821

"P. M. 18. Boy, aet. 14. April 19. Died rather suddenly on April 17.

Not well nourished.

Upper respiratory tract markedly swollen and congested.

Thymus rather larger.

Epiglottis, larynx and trachea markedly congested, much froth in trachea.

Pleura and lungs: Hemorrhages, especially on visceral pleura. Right lobes only congested. Left upper lobe same. Pleura over left lower lobe typically changed. Confluent lobular pneumonia in this lobe.

Peribronchial glands: Moderately enlarged, partly congested with hemorrhages.

Heart: Some clear fluid in pericardium. Hemorrhages beneath visceral layer. Right chamber of heart distended.

Hemorrhages on *diaphragm*.

Liver: Fatty changes with indistinct "white patches".

Spleen: Hardly enlarged, but congested and soft. Follicles indistinct. Four accessory spleens.

Kidneys: Appearance as in Case 10.

Oesophagus: Lower part congested.

Stomach: Small submucous hemorrhage.

Brain: Congested.

Both external and mesenterial lymph glands enlarged, but appear not acutely changed (status lymphaticus?)."

Sections received: Kidney, liver, spleen, epididymis, testes, colon.

HISTOLOGICAL EXAMINATION.

Kidney: Marked congestion, moderate granular degeneration, tubules filled with granular material in a foam-like structure, granular degeneration of the glomerular epithelium.

Liver: Moderately advanced fatty degeneration, old portal lymphocytic infiltration, moderate in degree, moderate polymorphonuclear infiltration throughout. Occasional small nodules of epithelioid cells and polymorphonuclear leucocytes in the intermediate zones.

Spleen: Marked edema, polynuclear infiltration, marked increase in blood content, groups of bacteria in small masses scattered throughout, hyperplasia of the epithelioid cells in the follicles with degeneration of follicle cells particularly on the periphery, hyalin degeneration of the walls of small blood vessels.

Epididymis: Shows some vascular engorgement, otherwise is not remarkable.

Testes: Shows considerable edema, but otherwise is a normal functioning organ.

Colon: Postmortem desquamation of superficial epithelium, slight edema and vascular engorgement of the submucosa.

Comment: A rather acute case accompanied by septicemic lesions in parenchymatous organs.

Accession No. 23802

"P. M. 19. Smallish woman, age 24. April 23, died on 22nd.

Upper respiratory tract congested.

Thymus seems involved.

Larynx and trachea not markedly congested. Some froth at bifurcation. Mucosa of bronchi markedly congested.

Pleura and lungs: Extensive pneumonia areas in right upper and lower lobes, mostly in stage of red hepatisation. Pleura over the areas mostly unchanged. At one place on upper lobe it is cloudy and injected, while at the base of the lung there is one typically changed area. Lobular confluent pneumonia in right middle lobe.

Peribronchial glands: Partly anthracotic, partly enlarged and congested.

Heart: Small quantity of fluid in pericardial sac. Heart in systole.

Liver: Parenchymatous degeneration. "White spots" and hemorrhages on surface.

Spleen: Not enlarged, soft. Follicles invisible.

Kidneys: Congested. Hemorrhages on surface.

Stomach: Numerous submucous petechiae.

Intestine: No hemorrhages. Mesenteric glands normal in size, but markedly congested.

Uterus: Congestion. Endometritis."

Sections received: Right upper and lower lobe and 2 pieces of liver.

Gross sections of lung shrunken, consolidated with a confluent broncho-pneumonia of lighter color in central portions reaching the periphery at the base of the lower lobe where fibrinous exudate is present.

HISTOLOGICAL EXAMINATION.

Liver: Moderately advanced fatty degeneration, considerable leucocytic infiltration, considerable vascular congestion in one section, occasional epithelioid nodules of small size.

Lung: Relatively even consolidation of entire section, blood vessels engorged, epithelium largely desquamated, numerous leucocytes largely polynuclear in alveolar walls, alveoli filled with serum, leucocytes and numerous bacteria. Pleura represented by a hyalin membrane, endothelium having desquamated, beneath is a dense leucocytic infiltration and some congestion with a little escape of red corpuscles. Perivascular and peribronchial lymph spaces show enormous numbers of bacteria.

Comment: Confluent type of bronchopneumonic plague.

Accession No. 23805

"P. M. 21. Man, age 61. Died on 23rd.

Well developed, not fat.

Upper respiratory tract congested.

Thymus persistent.

Epiglottis and larynx congested with small submucous hemorrhage. Trachea much congested with superficial patches of necrosis. Plenty of dark-red froth at bifurcation.

Pleura and lungs: Pleuritic adhesions on right side, especially upper lobe. Emphysema. Confluent nodular tuberculosis in upper lobe; also several calcified areas in this lobe. Right middle and lower lobe only congested. Extensive pneumonic area in left upper lobe, mainly in stage of grey hepatisation. Lower lobe only congested. Layer of fibrinous pleuritis between upper and lower left lobes.

Peribronchial glands: Enlarged, anthracotic, partly hyperemic.

Heart: 2 ounces cloudy, slightly blood tinted fluid in pericardium. Plenty of petechiae beneath both layers of pericardium. Both ventricles hypertrophic. Incipient atheroma of aorta.

Liver: Parenchymatous degeneration.
Spleen: Slightly enlarged, firm Some hemorrhages beneath capsule.
Kidneys: Congested. Some hemorrhage on surface.
Stomach: Few submucous hemorrhages.
Intestine: Slightly congested mucosa.
Brain: Congestion, slight oedema."

Sections received: Trachea, larynx, pericardium, heart (both chambers), left lung, liver.

Heart: Section of left heart wall shows epicardial hemorrhages. Section of the right ventricle shows adherent clot, probably post mortem. Hemorrhagic infiltration between the muscle bundles.

Larynx: One section for decalcification taking that point of vocal cords which shows hemorrhage.

Lung: Is contracted, deep red. Shows a confluent bronchopneumonia in the lower portion of the lobe with a fibrinous pleurisy and some hemorrhage.

HISTOLOGICAL EXAMINATION.

Trachea. Submucosa vascular engorgement with increased activity of mucous membrane and some degeneration. Submucous glands contain masses of deep eosin staining material apparently replacing the glands, or else representing necrotic structures. These are infiltrated with leucocytes and the change is not seen in all groups.

Pericardium: Not including heart. Vascular engorgement and diffuse hemorrhage, venous sinuses engorged with blood.

Heart: Including pericardium. Pericardium edematous, cardiac muscles moderately infiltrated with leucocytes, adherent postmortem clot.

Liver: Granular degeneration advanced, considerable distention of the capillaries, a few fatty cells are scattered irregularly throughout the lobules.

Larynx: Decalcified. Vascular engorgement and some edema. At the junction of the columnar and stratified squamous epithelium there is some diapedesis from dilated capillaries. Bone marrow: Portion is fairly active, blood vessels being engorged.

Lung: Pleura covered with thick layer of fibrin containing numerous leucocytes. The fibrous tissue appears necrotic in many areas beneath the exudate. Bronchi contain leucocytes, fibrin and debris. Mucosa is partially desquamated and there is some hemorrhage in the submucosa. Lung tissue shows engorgement, hemorrhage, necrosis of alveolar walls with many leucocytes in the alveolar walls and in the air spaces, the latter contain considerable serum and numerous bacilli but are not densely consolidated.

Comment: Fairly loosely consolidated confluent bronchopneumonia in the pneumonic area. The rest of the lung shows the congestive stage of the disease and the bronchi present all stages from congestion in the bronchioles to a puriform bronchitis in the larger passages. These changes are accompanied by a septicemic involvement of the heart with hemorrhages beneath the epicardium and leucocytic infiltration of the muscle.

Accession No. 23820

"P. M. 22. Man, aet. 41. April 27.
Well developed. Skin cyanotic.
Marked congestion of upper respiratory tract.
Thymus persistent.

Laryax and trachea markedly congested, much froth. Peritracheal glands congested; in some of them hemorrhages on section.

Pleura and lungs: Emphysema. Lungs hyperemic. Slight pneumonia (engorgement) in right apex.

Peribronchial glands: Moderately enlarged, anthracotic, hyperemic. On right side some calcified tubercular areas in glands.

Heart: Lipomatosis. Wall of right ventricle near apex thinned.

Liver: Parenchymatous degeneration.

Spleen: Chronic enlargement. Not soft. Some hemorrhages beneath capsule.

Kidneys: Congested.

Stomach and intestine: No abnormal findings.
Oesophagus: Mucosa of lowest part somewhat congested."
Sections received: Section of adrenal gland.

HISTOLOGICAL EXAMINATION.

Adrenal: Marked engorgement, marked increase in pigmentation, fairly marked granular degeneration of most of the cells with intercellular edema, some diapedesis, no frank hemorrhage.
Comment: None.

Accession No. 23810

"P. M. 23. Fresh body of a young man, aet.? May 1.
Undernourished with weak musculature.
Mucosa of upper respiratory tract markedly congested.
Thymus persistent.
Epiglottis congested with submucous hemorrhages. Larynx and trachea congested, contain *non-bloody* froth and greenish mucous matter.
Pleura and lungs: Right pleural cavity contains some cloudy liquid. Mucofibrinous deposits on right lung, especially between middle and lower lobes. Extensive pneumonic area, to a good part in stage of grey hepatisation, in right middle lobe. Here pleura cloudy beneath the fairly adherent fibrinous layer. Lobular confluent pneumonia right lower lobe. Superficial patches in both lobes left lung.
Peribronchial glands: Slightly enlarged, somewhat congested. Not much anthracosis.
Heart: Pericardium contains some clear liquid.
Liver: Early fatty changes.
Spleen: Not much enlarged, soft. Trabeculae just seen. Follicles as in Case 1.
Kidneys: Congested. Some hemorrhages on surface."
Sections received: Kidney, liver, spleen, pharynx, lymph-node, adrenal, tonsil, lung.
Lung: One lung shows confluent bronchopneumonia in the one portion with a strong line of demarcation outlining a necrotic area below, necrotic area being distinctly paler. Other sections show numerous nodules rather sharply outlined but tending to become confluent.

HISTOLOGICAL EXAMINATION.

Kidney: Advanced degeneration and necrosis of tubules, advanced degeneration including hydropic change of glomerular epithelium, moderate vascular engorgement.
Liver: Advanced granular and beginning fatty degeneration, sinusoids dilated and contain many leucocytes, no nodules seen.
Spleen: Follicles large, active, contain degenerated epithelioid cells, moderate polymorphonuclear infiltration, some increase in blood.
Pharynx: Section contains a necrotic abscess in tissue in which all blood vessels are engorged and capillary hemorrhage is present. Tissue has not been completely broken down but is filled with bacteria. Polymorphonuclear leucocytes moderately abundant.
Lymph-node: Acutely active and there is some fibrin in the lymph; the node shows no characteristics of plague.
Adrenal: Moderately advanced granular degeneration.
Tonsil: Marked engorgement of vessels, small areas of necrosis and follicles loaded with masses of bacteria. In some crypts epithelium is necrotic, the necrosis extending for some distance into the tissue. Small areas of hemorrhage.
Lung: Purulent bronchitis with degeneration and necrosis of epithelium and fairly complete necrosis of the wall, the tissue of the latter in places is nearly completely filled with leucocytes. Alveoli in the consolidated area show complete necrosis of the wall, the air spaces being filled with large masses of bacteria, a few leucocytes and occasional strands of fibrin. Tissue surrounding the consolidated areas shows a necrotic inflammatory reaction in the bronchial wall, the hyalin thickening of the basement membrane and a dense polymorphonuclear infiltration which

includes the submucous glands. Alveoli in these areas are largely empty, walls thickened in part from vascular engorgement but more from leucocytic infiltration in which mononuclears predominate. In one section there is a frank abscess formation apparently extending from the perivascular lymphatics of a blood vessel.

Comment: A necrotic type of bronchopneumonia accompanied by lesions in the upper respiratory tract and advanced degenerative changes in the kidney.

Accession No. 23800

"P. M. 29. Well developed middle aged man. May 14. Picked up in street.

Marked congestion in upper respiratory tract.

Thymus persistent.

Larynx and trachea congested, but contain only yellowish, non-bloody sputum.

Pleura and lungs: Old adhesions on left side. Some hemorrhage on visceral pleura. Lungs congested, but no pneumonia (*pulmonary* case). Mucosa of bronchi congested.

Peribronchial glands: Partly enlarged, anthracotic. No marked congestion.

Heart: Moderate hypertrophy of left ventricle. Lipomatosis. Right chamber distended. Incipient atheroma of aorta.

Hemorrhages on *diaphragm*.

Liver: Parenchymatous degeneration.

Spleen: Large, soft. Trabeculae and follicles visible, latter as in Case 1.

Kidneys: Congested. Some hemorrhages in pelvis.

Oesophagus: Mucosa somewhat congested.

Stomach: Many submucous hemorrhages.

Small intestin: No submucous hemorrhages.

Caecum: Many hemorrhages. Mesenterial glands somewhat enlarged and congested."

Sections received: Lung, spleen, kidney, bronchial gland, tonsil (2 pieces), thymus, pharynx.

Lung: Section of lung including bifurcation of the bronchi at hilus. Lung is firm, much shrunken, very dark color, apparently blood filled. Hemorrhagic inflammation of the bronchial mucosa

HISTOLOGICAL EXAMINATION.

Lung: Pleura shows vascular engorgement. Alveolar walls: vascular engorgement, diapedesis, leucocytic infiltration, edema and epithelial necrosis, bronchioles show no marked change. Bacteria present but not abundant in edematous fluid in tickened alveolar walls.

Spleen: Moderate polynuclear infiltration irregularly placed, considerable degeneration of cells on outside of follicles, the latter are small and most of them are dense.

Kidney: Moderate parenchymatous degeneration, irregular vascular engorgement, some diapedesis from veins.

Bronchial Node: (1) Acute inflammatory reaction, old fibrosis, marked anthracosis.

(2) Congestion marked, anthracosis moderate, peripheral edema, slight leucocytic infiltration, beginning necrosis of stroma between follicles, purulent exudate in lymphatics with a small amount of fibrin.

Adjacent Bronchus: Mucosa is covered by a necrotic fibrinous pseudomembrane in which are numerous red corpuscles and bacteria. Mucosa is in part desquamated. Basement membrane is thickened and appears hyalin, smooth muscle bundles lack striation.

Tonsil: (1) Vascular engorgement, marked proliferation of epithelioid cells in germinal centers, necrotic exudate in one crypt, no characteristic plague lesions.

(2) This tonsil shows a necrotic inflammatory reaction in most of the crypts, stratified epithelium necrotic and the necrosis extends into the lymph tissue, moderate polymorphonuclear reaction with much nuclear fragmentation in the necrotic material. Thrombosis of a deep vein. Bacteria abundant in crypt exudate, fairly numerous throughout tissue.

Thymus: Vascular engorgement.

Pharynx: No definite lesion. Vessels of the lymphatic structures markedly engorged.

Comment: A rapidly fatal case in which death occurred in the stage of pulmonary engorgement without the appearance of any exudate in the alveoli.

Accession No. 23806

"P. M. 30. Young well developed man. May 16. Picked up in street.

Only moderate congestion upper respiratory tract.

Persistent thymus. (Seems rather involved).

Larynx and trachea moderately congested; latter contains whitish mucous matter. Mucosa of bronchi more congested.

Pleura and lungs: Fairly strong (old) adhesions right side. Lungs congested. Slight oedema but no pneumonic infiltration (*pulmonary* case).

Peribronchial glands: Enlarged, especially some on right side (TB.?). In part moderate congestion.

Heart: Some clear liquid in pericardium and some hemorrhage on visceral layer. Right chamber distended.

Liver: Petechiae beneath capsule. Fatty changes with "white patches."

Spleen: Enlarged, soft. Follicles and trabeculae indistinctly visible.

Kidneys: Congested.

Oesophagus: Mucosa of middle portion slightly hyperemic.

Stomach: Numerous submucous hemorrhages.

Intestine: Mucosa of both ileum and caecum hyperemic. Mesenterial glands enlarged and congested."

Sections received: Heart, lung with bronchial glands and bronchi, liver, kidney, pharynx.

Lung: Uniform dark red in color, some suggestion of necrosis at the margin with fibrous adhesions over the surface, evidently recent. Bronchi show hemorrhagic inflammation.

Lymph-node slightly enlarged Calcification in lymph nodes at hilus.

Heart: (2) Left ventricle shows hemorrhages on the epicardium, rather deep stained muscle.

HISTOLOGICAL EXAMINATION.

Heart: 1st Section. Heart muscle hypertrophied, epicardium edematous, considerable degeneration of myocardium most marked beneath endocardium where there is considerable polymorphonuclear infiltration and some actual necrosis.

2nd Section. Purulent thrombi in cardiac sinuses, thrombi are practically one mass of leucocytes, very few red cells being present.

Liver: Moderate congestion and leucocytic infiltration, granular and slight fatty degeneration.

Pharynx: No definite lesion.

Kidney: Enormous engorgement with considerable hemorrhage in the cortex, advanced parenchymatous degeneration, hydropic change in the glomeruli.

Lung: Bronchi show desquamation and purulent infiltration with considerable numbers of bacteria relatively superficially located. Alveolar walls are engorged. There is considerable diapedesis of red corpuscles and a moderate degree of leucocytic infiltration. Practically no exudate in the alveoli.

Comment: Acute fulminant type of reaction, death occurring in the congestive phase; most advanced lesions in the bronchi.

Accession No. 23809

"P. M. 31. Man, apparently 40 years of age. May 16.

Well developed. Morphine spots all over.

Only moderate congestion of upper respiratory tract.

Thymus persistent (mostly fatty tissue).

One cervical gland enlarged, but shows no marked acute changes or periadenitis.

Epiglottis and larynx not congested. Only slight congestion of trachea which contains no blood, only greenish-white mucous matter.

Pleura and lungs: Adhesions (old) right upper lobe. Emphysema. Early engorgement (Group B) of both upper and right lower lobes.

Peribronchial glands: Those on right side enlarged, anthracotic, but not markedly congested. In a few caseous or calcified tubercular areas.

Heart: Not distended Lipomatosis.

Liver: Fatty changes with "white patches."

Spleen: Large, very soft. Follicles invisible, trabeculae just distinguishable.

Kidneys: Fatty changes and injection as in Case 10.

Stomach: Mucosa somewhat swollen. Plenty of submucous hemorrhage.

Intestine: No marked changes.

Mesenteric glands: No marked changes."

Sections received: Hilus node, bronchus, lung.

HISTOLOGICAL EXAMINATION.

Hilus Node. Old fibrosis and recent caseous active tuberculosis.

Bronchus: No definite pathology

Lung: Emphysema, old fibrosis and some odema and atelectasis. Mild grade of interstitial pneumonia near pleura.

Comment: These tissues not sufficiently well preserved to justify any diagnosis; they would not be considered plague in the ordinary run of examinations.

Accession No. 23819

"P.M. 36. Fairly developed man, aet. about 25. May 19. Picked up in street.

Marked congestion of upper respiratory tract. Cervical glands somewhat enlarged, one on left side size of a hazelnut, congested. No periadenitis. Tonsils not enlarged, their mucosa congested. In substance of right tonsil a small abscess (size of half pea) containing thick pus. Apparently older change.

Mucosa of larynx congested, trachea only slightly congested, contains no froth.

Pleura and lungs: Some hemorrhagic dots on surface left lung. Lungs congested, no marked pneumonia, (*pulmonary* case). Bronchus mucosa congested.

Peribronchial glands: Slightly congested, anthracotic; one on right side slightly enlarged.

Heart: Some hemorrhage epicardium. Left hypertrophia. Beginning atheroma of aorta.

Liver: Petechiae beneath capsule. Some "white patches" on surface. On section nut-meg appearance and indistinct "white patches" surrounded by congested areas.

Spleen: Enlarged, soft. Follicles not clearly visible, trabeculae visible.

Kidneys: Congested.

Oesophagus: Upmost part slightly congested.

Stomach: Petechiae beneath mucosa of pyloric part.

Small intestine: No marked changes.

Large intestine: Submucous petechiae in caecum. Mesenterial glands not swollen, but congested."

Sections received: Lung, bronchial lymph-node, kidney, ileum, stomach, spleen.

HISTOLOGICAL EXAMINATION.

Lung: Marked congestion, considerable edema, moderate polymorphonuclear infiltration of alveolar walls and perivascular tissue, serous bronchitis, no consolidation. Bronchus shows hyalinization of the basement membrane.

Bronchial lymph-node: Marked vascular engorgement, hyalin thrombi in nearly 50% of capillary vessels; lymph spaces filled with serum, macrophages and leucocytes. Macrophages have engulfed both leucocytes and numerous corpuscles. Some capillary diapedesis in most follicles, occasional ones contain necrotic epithelioid cells and moderately numerous polymorphonuclears.

Kidneys: Extreme engorgement of vessels so that vessels surrounding tubules have practically one-half the diameter of the tubules. Advanced granular degeneration of the tubules, granular and hydropic degeneration of the glomeruli. Some intertubular hemorrhage.

Fig. 14. Section of Human Lung, P.M. 11, 1920 epidemic, showing Confluent Pneumonia. Low magnif.

偏桃腺組織標本解剖十一例一九二零年流行的示慢性壞死及在濾胞部見新鮮多液管之侵襲

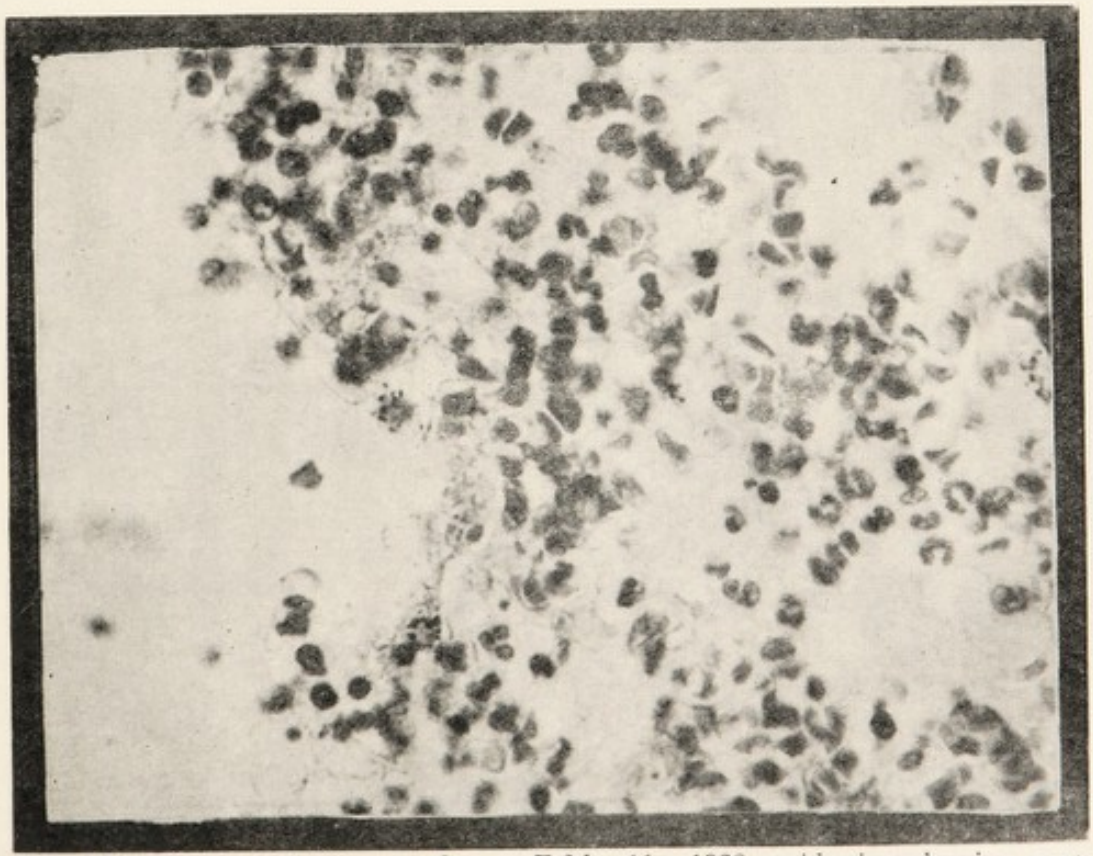

Fig. 15. Section of Human Lung, P.M. 11. 1920 epidemic, showing acute leucocytic reaction in the alveolar walls. Plague bacilli are clearly seen lining the walls. Hign magnif.

毛肝示的疫行流年零二九一號十剖解本標織組肝細管內有鼠疫菌栓塞

ROYAL COLLEGE OF SURGEONS OF ENGLAND
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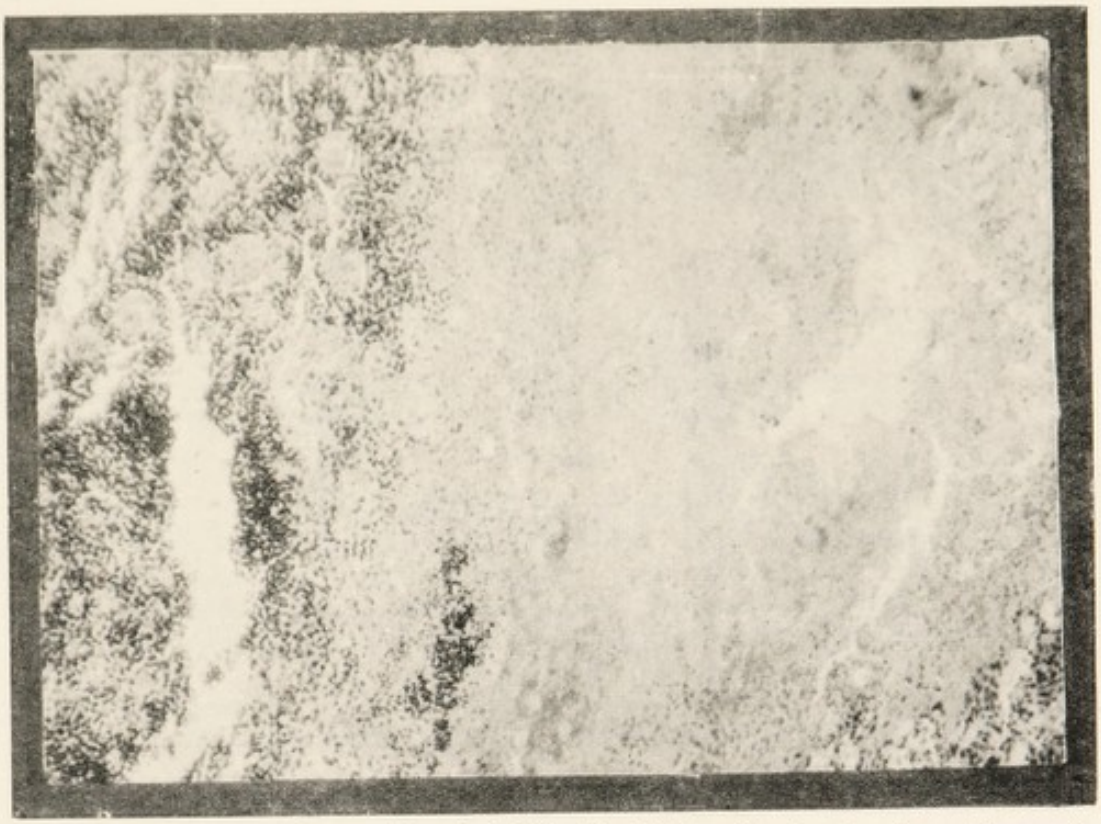

Fig. 16. Section of Human Tonsil, P.M. 11, 1920 epidemic, showing old necrosis as well as fresh vascular invasion in crypts. Mod. Magnif.

肺組織標本解剖十一號一九二零年流行的示注
流性肺炎用弱擴大

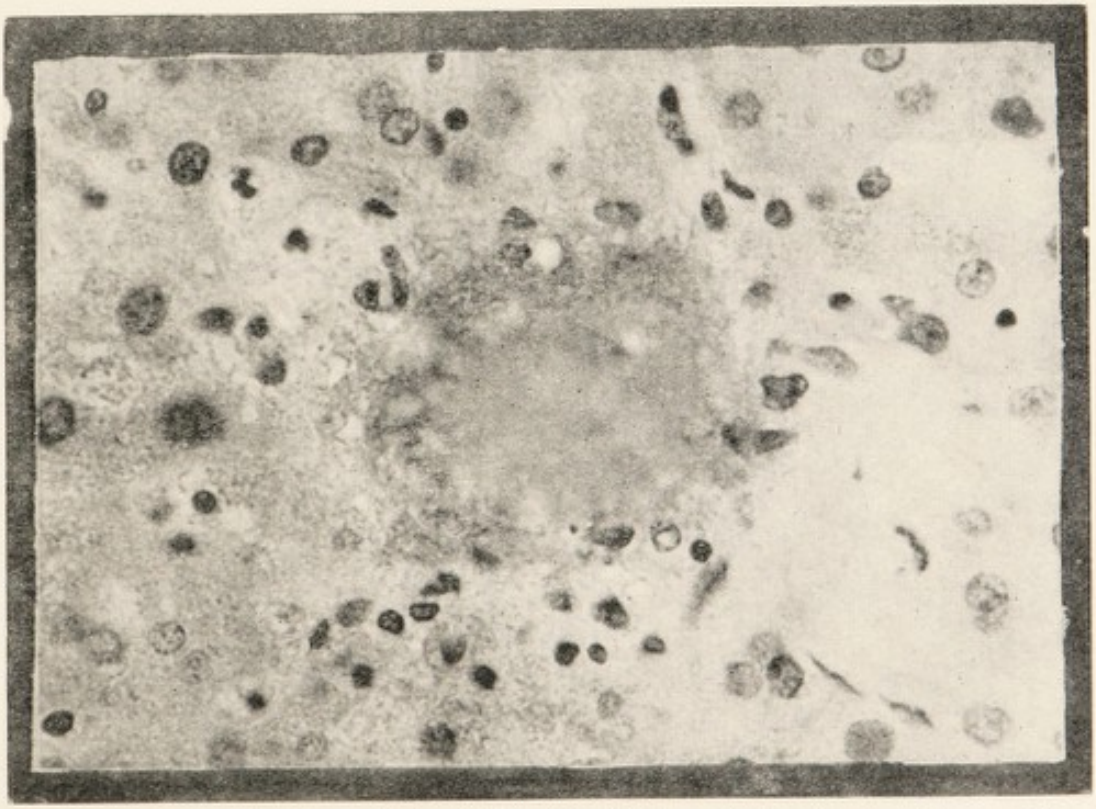

Fig. 17. Section of Human Liver, P.M. 10, 1920 epidemic, showing liver capillary plugged with B. pestis. High magnif.

肺組織標本解剖十一號一九二零年流行的示肺胞壁之急性白血
球反應疫桿菌在肺壁交界部能明視用强擴大

Spleen: Marked increase in blood, which in many places, forms hyalin homogeneous masses in which the corpuscles can be made out with difficulty; enormous polymorphonuclear infiltration, considerable diapedesis, considerable degeneration and bacterial infiltration in and about the follicles which appear somewhat decreased in size.

Stomach: Shows congestion and considerable edema, with desquamation of the superficial epithelial cells probably due to postmortem change.

Ileum: Postmortem desquamation, edema, congestion and considerable activity in the lymph follicles in a few of which bacterial masses are seen. These lymph nodules contain quite a few macrophages but relatively few polymorphonuclears.

Comment: Acute fulminant case dying in the state of congestion.

Accession No. 23808

"P. M. 39. Strong, well developed young man, aet. 25. May 21. Was admitted the day before for fever and suspected plague. Had neither cough nor sputum.

Only slight congestion upper respiratory tract.

Larynx and epiglottis fairly congested, trachea only pink, no contents.

Pleura and Lungs: Lungs only congested and oedematous, but no pneumonia (*pulmonary* case).

Heart: Distended.

Liver: Hemorrhages beneath capsule. Degenerated with slight "white patches."

Spleen: Large, soft.

Kidneys: Very congested.

Stomach: Submucous hemorrhages all over.

Caecum: Some hemorrhages."

Sections received: Lung, bronchus.

Lung: Uniformly hemorrhagic, firm and fibrous. Hemorrhages on pleura.

HISTOLOGICAL EXAMINATION.

Primary bronchus: Section does not show the mucosa, no definite lesions except in lymph-node where there is an occasional necrotic area.

Lung: Marked postmortem change, evidently interstitial pneumonia present. Bronchus shows vascular engorgement and some desquamation, bronchial lymph-node engorged and has a few small necrotic areas containing numerous bacilli. Alveoli show leucocytic infiltration of wall with engorgement and some diapedesis. Bacteria are present but not in large numbers.

Comment: An acute fulminant case dying in the stage of congestion and beginning interstitial inflammatory reaction.

MEMORANDUM SUBMITTED TO THE LEAGUE OF NATIONS, HEALTH SECTION, ON THE PROPOSED SURVEY OF PLAGUE IN WILD RODENTS BY AN EXPERT COMMISSION OF THE HEALTH ORGANISATION OF THE LEAGUE OF NATIONS.*

By Wu-Lien-Teh, M.A., M.D. (Cantab.), LL.D., etc.

1. The original suggestion for a Survey of Epizootics among Tarabagans by an International Commission to be set up by the Health Organisation of the League of Nations as submitted by me on June 15, 1926, recommended :

I. An intensive study of tarabagan plague in the endemic centers of Transbaikalia (and Manchuria).

II An investigation of the endemic regions in Central Asia in general.

These proposals are here discussed in detail, and a questionnaire for securing information is appended.

I. Intensive study of tarabagan plague.

In some respects we already possess satisfactory information regarding the problems of tarabagan plague.

It has been finally established that these rodents suffer from natural plague. The morbid appearance in both naturally and experimentally infected animals have been thoroughly studied. It has been proved moreover that both tarabagan fleas and lice often harbour plague bacilli and are able to transmit the disease from animal to animal. Both parasites have been proved to bite men, and there is no doubt that the fleas especially play an important part in the spread of the disease from rodents to man. Our experiments have shown that the susceptibility of tarabagans to infection and the nature of their illness vary markedly at different season of the year, the disease assuming a protracted course during hibernation whenever infection takes place.

In other respects little or no definite information is as yet available regarding tarabagan plague. Among these are :—

a. A few workers doubt if the tarabagans are *the reservoir* of plague in the Transbaikalian, etc., regions. Some believe that certain species of *small* rodents are the real sources of infection, the tarabagans only becoming periodically infected through them.

b. Consequently it is not certain how the epizootics among tarabagans *may arise*.

* Some of the problems mentioned in this Memorandum have been partially solved as will be seen in the article appearing in this Volume of Reports on "Further observations upon Plague in Wild Rodents".

c. Nothing definite is known as to *the areas* covered by tarabagan epizootics. It is generally agreed that they are usually limited in extent, though this may not always be the case, and exact data are still lacking. It is not certain if all burrows within the epizootic areas may become infected or not. Further it is not definitely established if these areas grow only by continuous spread of infection, or if a spread *per saltum* takes place. In the latter case the tarabagans may migrate under exceptional circumstances, or infection may be propagated by other means (small rodents, beasts and birds of prey).

d. Little is known as to *the time* covered by tarabagan epizootics. It seems likely from epidemiological observations that epizootics may continue in certain localities through two or more seasons. No *definite* information exists on this point and there is at least one observation on record which suggests that epizootics may decline quickly.

e. No exact data are available about the decline of epizootics. We think it unlikely that all the animals within an infected area die out. It is not established, however, what percentage of animals escape or recover. Likewise it is unknown if animals surviving in a region previously visited by an epizootic become immune or less susceptible to infection.

f. Of much practical importance is another problem suggested by epidemiological observations. Human infections from the tarabagan occur mainly in late summer and autumn. Spring outbreaks, though possible, are rare. That the incidence of human infections is highest in late summer and autumn is undoubtedly due in part to the fact that—as opposed to spring time—in addition to the hunters a majority of the population live out on the steppes for the hay harvest. In our opinion, however, two other factors deserve serious consideration :

i. Seasonal changes in the character of tarabagan plague. Possibly the disease is at times (e.g. spring) of a more localised character. At others (e.g. late summer and autumn) it may be more generalised and accompanied by severe bacteremia. In the latter case obviously a spread both by direct contact (skinning and cutting of the hunted marmots) and through their fleas becomes easier.

ii. Flea rate of the tarabagan. If the fleas grow more numerous during the warm season, infections would *ipso facto* be more numerous than in spring.

In order to investigate the above and related problems the following program is suggested for the work of the Commission :—

a. Workers should be ready on the spot when the tarabagans leave their burrows after hibernation, and stay there until late autumn (period of hibernation).

b. Investigations should be conducted, if possible, in districts :—

i. Where an acute epizootic prevails,

ii. Where such is known to have existed in the past (comprising areas invaded during the previous season and also two or more years before),

iii. Healthy areas.

It would not be easy to find situations where acute epizootics are present. This point should be emphasised when the Commission decides upon the district for investigation.

c. In areas where an acute epizootic exists, the following points are specially important :—

i. Determination of the extent of the epizootic. Does it coincide with the natural borders within which the tarabagans live? If not, is there any evidence in favour of a continuous spread of the epizootic? Are there any signs of migration, etc. suggesting a spread of infection *per saltum*?

ii. Are all burrows within such an area affected or not? This problem may be solved by constantly observing the number and condition of the animals of the different burrows, by exposing guinea-pigs or healthy tarabagans in cages at the entrances. Where practicable, one may resort to capturing (or shooting) of the animals belonging to the different burrows.*

iii. Investigations of burrows found infected. Again the number and behaviour of the inhabitants of such burrows should be watched. Capturing or shooting of animals may be resorted to; in other cases infected burrows will be opened and their contents, including parasites, carefully examined. Test animals may be exposed in burrows where the tarabagans have died out.*

iv. All animals captured or found dead should be systematically examined. This includes :—

1. Counting and examining of parasites; some of the living parasites may be exposed on test animals.
2. All animals captured alive will be observed for some time and—if possible—accommodated in individual cages. Serological tests will be made with apparently healthy animals. In suitable cases their susceptibility to plague infection may be studied.
3. Both the animals found dead and those captured (which will die or be killed eventually) should be dissected and serological and bacteriological tests carried out to establish the existence of plague.

v. Attention should be paid to small rodents living in the same area for mortality or illness among them. Corpses will be examined for plague and sick animals isolated. In addition such rodents may at a suitable time be captured

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* See also g.

en masse and examined for the presence of infection. Finally special attention should be paid to the parasites of such rodents so as to find tarabagan parasites, if any, upon them; likewise a constant look-out should be kept for the existence of non-specific parasites upon the tarabagans.

d. In areas where epizootics formerly existed, the following investigations might be performed:

i. Determination of number of inhabited burrows and estimation of number of animals in the single burrows. The data found should be compared with those observed formerly by local plague workers and hunters. Enquiries should be made to find out whether or not any immigration has taken place.

ii. As many tarabagans as possible should be captured and investigated. It is important to know whether plague still exists among them; if so, in what form. Does their serum show any reaction pointing to past infection? To what degree are the animals susceptible to experimental infection? All dead animals should be carefully examined for signs of residual plague.

iii. Deserted burrows should be opened and examined. Test animals may be exposed in them.

e. Comparative investigations should be carried out in areas free from epizootics, specially as regards serological reactions and susceptibility to plague of the animals. In order to save time and expense, advantage may be taken of animals captured by professional hunters, to whom the furs of the animals might be returned.

f. In all the areas studied certain investigations upon the parasites should be carried out:—

i. Collection of all data regarding the flea rate and its changes in the course of time.

ii. Observations upon plague infected fleas to determine how long infected fleas may survive and remain infectious, study the infectivity of their faeces, collect material for histological studies of infected fleas.

iii. Investigations as to whether the fleas of tarabagans and other rodents emancipate themselves in autumn from their hosts and lead an independent life on the ground as asserted in some quarters. The fleas found "free living" should be collected separately in order to determine afterwards to which species they belong.

iv. If time and circumstances permit an attempt should be made to transmit plague from one species of rodents to another with the aid of parasites of the first kind and *vice versa*. The result of such researches would throw

much light on the problem of transmission of plague from one species to another. At the same time this would permit the collection of further data regarding the infestation of small rodents.

v. The ticks infecting tarabagans should be collected and identified. Are there more than one species? Experiments should be continued to establish their role, if any, in the transmission of plague from one animal to another.

vi. Material should be collected for a further study of the entero—and blood parasites of the tarabagan.

g. Different methods for destruction of wild rodents should be tried in order to find a suitable means of destroying the tarabagans. If would be advantageous to perform such tests in infected areas where it is possible at the same time to procure suitable material for examination of infected animals, their burrows, etc.

If possible a strain of the "Tiger River" bacilli (South Africa) should be procured and the susceptibility of the tarabagan and other species to such be tested.

h. An investigation should be made to determine the best methods for disinfecting tarabagan skins. This may be done in late autumn after the tarabagan has begun to hibernate.

i. As far as possible investigations should be made and information collected as to the districts inhabited by the tarabagan, special attention being paid to their northern and southern boundaries.

II. INVESTIGATION OF THE ENDEMIC REGIONS IN CENTRAL ASIA.

The following table gives summaries of the information upon the Central Asia endemic areas.

TABULATION OF PLAGUE AREAS IN CENTRAL ASIA.

No. Locality:	*Year of last outbreak on record:*	*Rodents implicated or suspected:*	*Remarks:*
1. Manchuria	1925	Tarabagan (*Arctomys bobac*)	
2. Transbaikalia	1926	"	
3. Outer Mongolia	1908?	"	Information about human outbreaks incomplete.
4. Inner Mongolia a. Patsebolong District	1917	?	"
b. Ordos Country	?	?	A suspicious "winter sickness" is said to exist here.
5. Shansi	1924	?	Probably connected with area 4.

No. Locality:	*Year of last outbreak on record:*	*Rodents implicated or suspected:*	*Remarks:*
6. Kansu	1917	Tarabagan (Arctomys robustus)	Human infection apparently imported from area 7.
7. Thibet	?	Same?	Information unsatisfactory.
8. Chinese Turkestan	1902?	?	Connected with area 7?
9. Semiretchinsk (Russian Turkestan)	1913	Implicated: "Black marmots" or "tarabagans"	Not established whether these rodents are a variety of the *Arct. bob.* or identical with the *Cynomys* fulvus as suggested by Klodnitzki.
10. Western Turkestan (on the Amu Darya)	1924	*Rhombomys opimus* Licht.	See text.
11. Khorassan (Persia)	1921	Field mice implicated (in 1912). There exists a "tarabagan" (*Citellus fulvus Parthianus*)	The 1921 epidemic spread to the adjacent Russian territory.
12. South-East Russia a. Ural Territory	1926	Small sisel (*Sperm. mugozaricus*) Large sisel (*Cynomys fulvus*) Field mice: *Arvicola arvalis* *Microtus socialis** House mouse (*M. musc.*) *Jerboas* (*Alactaga elater*)* Hamster (Cricetus cricet.)*	
* Only few specimens found infected.			
b. Kirghese Steepes	1926	Small sisel (*Sperm. musicus*) Large sisel (*Sperm. rufescens*) Field mouse (*Arvicola arvalis*) Kirghese mouse (*M. muse. Wagneri*) House mouse (*M. musc.*) Jerboa (*Alactaga saliens*) Sand mouse *Mus* (*Gerbillus?*) *tamaricinus*	
c. Don Territory	1925	Small susliks Field mice	

A study of the above table shows that the available information is unsatisfactory in many respects. In fact it may be said that exact data exist only in regard to Transbaikalia and Manchuria on the one hand, and South-East Russia on the other, while little is known of outbreaks in Central Asia proper. In my opinion it would be erroneous to assume *a priori* that such outbreaks are rare. It stands to reason that many plague manifestations in Central Asia are unnoticed because they do not spread widely either on account of the sparseness of population and slowness of communication or, as is claimed for Mongolia and Thibet, on account of crude preventive measures by the people. On the other hand it must be admitted that the disease is not always active in every area known to have been affected at one time or another.* This flaring up and abating of the infection in individual foci speaks against a purely local origin of the disease and suggests the presence of some common primary cause. As I concluded in the article on the "Original Home of Plague" it seems that "in Central Asia the virus is constantly kept alive among the various species of susceptible rodents, which suffer from periodical epizootics."

These were the main reasons which actuated me to propose in addition to the intensive study of the tarabagan foci in Transbaikalia and Manchuria, an extensive investigation of the Central Asiatic areas and southern region of Himalayas. A discussion of the problems awaiting solution follows :—

a. *Outer Mongolia.* It is well known that here both tarabagan and human plague has existed on and off in various districts. Also the distribution of the tarabagan throughout these areas is known on general lines. I hope to obtain further information on these points before the expedition starts; thus Dr. Dudakalov (Urga) who is in charge of Public Health work in Outer Mongolia, promised to collect information for me. It will therefore not be necessary for the expedition to devote too much time to Outer Mongolia.

b. *Inner Mongolia.* These areas are of great epidemiological importance as they apparently form the starting point of the Shansi Plague outbreaks, the aetiology of which has never been properly investigated. It appears that no tarabagans live within the knee of the Huang-ho river in the Ordos country; according to Prshevalski hares and different species of small rodents live there. Yet vague information exists about a "winter sickness" prevailing there which excites suspicion. Tarabagans are probably absent also in the districts immediately north of the Huang-ho; here besides hares (*Lepus Tolai*) a *Spermophilus sp.* prevails (Prshevalski). The Patsebolong district, situated in this area, was the apparent starting point of the big 1917-18 Shansi epidemic. It would be very important therefore to investigate the rodents and search

* Thus it may be mentioned that the Amu Darya region where an epidemic was found in 1924, was apparently never before visited in our times. Likewise foci which had been rather active in former times, seem dormant now; this was recently stated by Neligan (Lancet 1926, I. p. 690) for Khorassan in Central and Kurdistan in Western Asia.

for epizootics and human outbreaks in these parts. Then only will it be possible to make definite recommendations for the establishment of a plague prevention bureau at a point of vantage. A new focus—the Payintala district reached by the Ssupingkai-Taonan Railway (South Manchurian trunk) which gave rise to 60 pneumonic cases in 1927—will have to be studied.

c. *Kansu and adjacent parts of Thibet.* There is no doubt that plague had occurred in Kansu; one limited pneumonic outbreak was noted in 1917 at the time when the Shansi epidemic started and was traced to Thibet, where the first victim became infected while handling a marmot found dead in the hills. This marmot is probably identical with the *arctomys robustus* abounding in the Kansu mountains and Northern Thibet. This relative of the tarabagan of Transbaikalia, etc., has never been properly examined from a medical standpoint and it would be desirable to test its susceptibility to plague infection, investigate its parasites and search for evidence of epizootics. At the same time the other rodents abounding in these parts could be examined and investigations undertaken to find out the incidence of human plague. This is indeed a *terra incognita* for modern medicine.

d. *Weichang District.* If time and circumstances permit, similar investigations may be carried out here also. This area deserves less attention than the former because, though plague was very active here towards the beginning of the present century, it has apparently died out during the last 25 years.

e. *Semiretchinsk. District.* Here several plague outbreaks are on record (the last in 1913) evidently brought about by "black marmots" or "tarabagans". Only circumstantial evidence is available in regard to the epidemiological importance of these rodents and—as far as our information goes—it is not definitely established which species was (or were) involved. It must be kept in mind that the population gives the name tarabagan not only to the different varieties of *arctomys* but also to other fair-size rodents. It would be desirable therefore to establish whether subspecies of the *arctomys* or other species are responsible for the plague outbreaks in these parts and to conduct researches upon their parasites, epizootics, human manifestations, etc. Here also new ground may be covered.

f. *East Turkestan, Khorassan.* If possible similar investigations should be undertaken in East Turkestan on one hand, and Khorassan (Persia) on the other. No information about any rôle of wild rodents is available in the former area, very indefinite in the latter. It would seem that both foci are quiescent now.

In my preliminary programme two expeditions were recommended, one starting (a) *southwards*, the other (b) *westwards*, for the purpose of:

1. Studying the rodent fauna and their parasites in the different areas,
2. Finding evidence of epizootics including the appearances of plague in the animals, when infected,

3. Testing the suspectibility to plague infection of species or subspecies not hitherto tested,
4. Performing biting and transmitting experiments with parasites not hitherto tested,
5. Obtaining evidence of human outbreaks, if possible by actual observation, otherwise by enquiries among the people,
6. Studying the contact between the population and the rodents.

(a) The expedition going out *southwards* could study Outer Mongolia *en route* and then concentrate upon Inner Mongolia including the Ordos Country on one hand, upon Kansu Province and adjacent parts of Thibet on the other. If enough personnel is available it would be well to have one party work in Inner Mongolia (then pass via Weichang to Peking), the other in Kansu, entering Thibet, if possible.

(b) The expedition going *westwards* could study Outer Mongolia *en route* and work then in Central Asia, especially the Semiretchinsk area.

Satisfactory results might possibly be obtained from the work in the tarabagan regions, where the previous experience of our Anti-plague Service and our Russian colleagues is available. Likewise no unsurmountable difficulties need be expected in Mongolia if timely arrangements are made. The same holds true for China (Kansu, Weichang). It would also be possible to arrange matters in Thibet. The most difficult trip will be that proceeding westwards. Here a great distance has to be covered in a territory about which we and our Transbaikalian colleagues have no experience; apart from this the necessity of not delaying too long in one place will interfere with the program. Certainly this part of the work of the Commission needs serious preparation.

I propose that, in addition to our Manchurian Plague Prevention Service and the Russian Anti-plague Organisation of Chita and Irtusk, representatives of organisations in other areas where plague exists among wild rodents, should collaborate. It would be well if the workers from the following areas could be represented :

i. South East Russia.
ii. South Africa.
iii. U.S.A. e.g. California (Louisiana).

Advantage could be taken of such an assembly of representatives from areas interested in wild rodents in several ways. I would propose that the members of the Commission meet in late winter in Russia, and that an International Conference on Plague in Wild Rodents be held, preferably at Saratov, where advantage could be taken of the interesting institute and museum of Prof. Nikanoroff. Representatives from the following organisations might be invited to this Conference :

1. Health Section, League of Nations;
2. Commissariat for Public Health, Moscow.
3. N. Manch. Plague Prevention Service, representating at the same time the Chinese Government,
4. State Institute for Microbiology and Epidemiology, Saratov,
5. Bacteriological Institute of the Osbek Republic, Tashkent,

6. Plague Prevention Centre, Irkutsk.
7. Antiplague Service, Chita,
8. Other Russian Institutes proposed by the Commissariat for Public Health,
9. Plague Prevention Service, South Africa,
10. Plague Prevention Service, U.S.A.
11. Mongolian Public Health Service,
12. Persian Public Health authorities or Conseil Sanitaire at Teheran,
13. Indian Public Health Service,
14. Any other.

LEAGUE OF NATIONS, HEALTH ORGANISATION, NOTE ON THE STUDY OF PROBLEMS RELATING TO PLAGUE.

During its First Session held at Singapore January 4th to 6th, 1926, the Advisory Council of the Eastern Bureau, which acts as a Commission of the Health Committee, adopted a Resolution of which the following is an extract :

"The Advisory Council;

"Considering that the International Health Organisation should include in its programme of activities the promotion of studies and investigations into the great problems of public health in the East ...

"Considering that investigation into the rat-flea fauna of different countries with respect to plague is a matter of great importance as a basis for the formulation of methods of control of non-pneumonic plague, and particularly for its control in ports :

"Considering that the study of conditions concerning the epidemiology of pneumonic plague with regard to its endemic centres is a matter of much importance .....

"Recommends that a special Expert Committee or Committees be set up composed of directors of research and other experts of the several countries, in order to draw up a programme of study and to agree on the procedure to be followed for the inter-communication of results obtained and the mutual co-operation in the carrying out of the enquiry;

"Proposes that the technical secretariat of the Expert Committee or Committees be provided by the Health Organisation of the League".

During its Sixth Session held at Geneva from April 26th to May 1st, 1926, the Health Committee noted with interest the recommendation contained in the Resolution of the Advisory Council concerning the creation of Expert Committees to co-ordinate research in the Far East in public health questions of great international importance and invited the Medical Director to obtain from the members of the Advisory Council of the Eastern Bureau further information regarding the subjects for co-ordinated research for which the creation of Expert Committees was recommended.

At the Second Session of the Advisory Council of the Eastern Bureau held at Singapore January 6th to 10th, 1927, a Resolution was adopted recommending that an Expert Committee to deal with plague research be set up, suggesting that the Committee should meet for the first time in December, 1927, at the time of the Congress of the Far Eastern Association of Tropical Medicine in Calcutta. The Advisory Council also made some recommendations in regard to the membership of the Committee, and referred to it a Note by Dr. Wu Lien Teh containing "Suggestions for a Survey by a Commission of the League of Nations to study Epizootics among Tarabagans".

Acting on the Resolutions and Minutes of the Second Session of the Advisory Council, the Health Committee during its Ninth Session held at Geneva from February 14th to 18th, 1927, adopted a Resolution of which Part III follows:

> "The Health Committee accepts the suggestion of the Advisory Council that the co-ordination of research mentioned above will derive considerable advantage from the setting up of expert committees, and it approves the proposal for the constitution of a new Committee of Experts which will undertake the study of research on plague. The Committee accepts the proposal with regard to the nomination of experts put forward by the Advisory Council, proposed to complete the composition of the Committee by the addition of other experts, and decides to convene the members at a convenient date to be arranged with the President of the Advisory Council, the constitution of this Committee of Experts being determined by Article 10 of the Rules of Procedure of the Health Committee (Annex 1)".

In order to take advantage of the presence in Paris of a number of experts on questions relating to plague, it has been decided to invite them to give their views on the proposals of Dr. Wu Lien Teh with regard to a survey of wild rodents by an international Expert Commission in various areas where plague is endemic, and to send a Questionnaire to the directors of the different Plague Prevention Institutes.

The experts will meet in Paris on April 26th at the Pasteur Institute at an hour to be announced later.

A summary of the opinions of the experts present will be prepared for the use of the Health Committee and of its Commission of Experts on Plague.

Meeting in Paris, April 1927.

Accordingly, advantage was taken of the presence of experts attending the International Rabies Conference at Paris to convene a meeting of plague experts at the Institute Pasteur at 5.30 p.m. on April 26, 1927 to discuss Dr. Wu's proposals. There were present on the occasion:

Prof. Hahn (Director of the Hygienic Institute, Berlin) Chairman,
Dr. Wu Lien Teh (China),
Prof. S. Zlatogoroff (Kharkov),
Capt. S. R. Douglas (National Inst. of Medical Research, London),
Prof. C. Praussnitz (Director, Hygienic Institute, Breslau),
Prof. T. Madsen (Director, Serum Institute, Copenhagen),

Lt.-Col. J. Taylor (British India),
Drs. Norman White, Boudreau and Olsen (League of Nations).

The greatest interest was shown in the proposed Questionnaire submitted by Dr. Wu Lien Teh, and much valuable material was added to the original draft. The revised Questionnaire published in this Report is therefore the combined work of the plague experts assembled at Paris in 1927.

Meeting at Calcutta, December, 1927.

As the Far Eastern Association of Tropical Medicine was holding its seventh Congress at Calcutta in December 1927, it was thought desirable for the Sub-committee on Plague Research to meet about the same time. Accordingly, the following gentlemen attended by invitation of the Health Section of the League of Nations :

Dr. T. Madsen (President of Health Section), Chairman,
Col. Graham (Sanitary Commissioner, India),
Lt.-Col. Mackie (Director, Haffkine Institute, Bombay),
Dr. F. Hirst (Micro-biologist, Colombo),
Dr. Wu Lien Teh (China),
Dr. Jourdran (Health Commissioner, Hanoi),
Col. Forster (Director, Public Health, Punjab),
Dr. S. Hata (Kitasato Institute, Tokio),
Dr. S. Shiga (Government Research Institute, Keijo),
Dr. S. M. Nikanoroff (Saratov),
Dr. E. P. Hicks (Municipal Health Dept., Shanghai),
Lt- Cols. J. Taylor (Rangoon), Gloster and Dunn (Br. India),
Dr. d'Herelle (Alexandria),
Dr. B. P. B. Naidu (Bombay) and
Dr. A. N. Goyle (Lucknow).
} co-opted

This Expert Plague Committee first considered the advisability of sending to Health Administrations the questionnaire (Appendix A) prepared by Dr. Wu Lien Teh on pneumonic plague. The majority were against sending of this questionnaire through official League channels. The Committee then drew up a list of subjects requiring, in their opinion, investigations. This list is attached (Appendix B) and gives in priority of importance, eight subjects for enquiry concerning bubonic plague alone and two regarding pneumonic plague.

The Committee was of opinion that the role of the Eastern Bureau in this matter was to send this list of proposed investigations to the Health Administrations of the Eastern countries. The Health Administrations which have any plague investigations under consideration should be asked to undertake them if possible along the lines suggested in the list and to notify to the Bureau any which they may resolve to undertake.

In the matter of plague as in the matter of vaccination the role of the Bureau was, in the Sub-Committee's opinion, more in the nature of stimulating and arousing interest on certain specific points and of guiding investigation on the lines drawn by experts than of actually inducing

unwilling Administrations to undertake investigations. The Sub-Committee appreciated the fact that research on plague would be carried out in India along certain of the lines suggested by the Expert Plague Committee and believed that similar selections could be made by Health Administrations and individual workers in other countries, as results obtained from these investigations along the predetermined lines would be comparable and probably of benefit to all. The meeting then dispersed.

APPENDIX A.

INQUIRY ON PNEUMONIC PLAGUE.

QUESTIONNAIRE.

Passed by Expert Plague Committee in Paris, April 1927, but withdrawn by another Committee sitting in Calcutta, December 1927.

Area..... Institute.....

I. General Plague Situation in Area. (Include photographs if possible).

1. Short description of area, inhabitants, (including statistics of age, sex, occupation, as well as information re: feeding, housing, domestic animals, etc.) and climate.
2. Short statement as to when and how plague was introduced into area.
3. Species of rodents and/or other animals involved in epizootics. Their Biology, (years of abnormal fertility, etc.). Appearances and type of plague observed in animals. Is plague endemic among these or does reimportation of infection take place?
4. Species of fleas and/or other parasites known or suspected to transmit plague from:
   (a) Animal to animal.
   (b) Animal to man.
   Their biology, seasonal frequency, etc.
5. History of human plague in area, comprising a short individual description of all outbreaks or at least the primary pneumonic ones.
6. Summary of (5) with general statement as to frequency of different types of plague in man, accompanied, if possible, by statistical data.
7. Notes on Bubonic plague in man, especially its
   Fatality,
   Distribution of buboes (if possible with statistics).
8. Notes on primary septicemic plague in man, prognosis, etc.

II. *Secondary Plague Pneumonia in Area.*

1. Frequency,
2. Clinical features mentioning specially
   (a) frequency of cough,
   (b) character and bacterial contents of sputum; ratio of *B. pestis* to other organisms present,
   (c) duration and prognosis.

3. Is there any connection between localisation of buboes in certain gland areas (e.g. cervical, axillary) and frequency of secondary pneumonia?

4. Are there other factors influencing frequency of secondary lung involvement? Note here :—

(a) Susceptibility of inhabitants to respiratory diseases in general, including statistics and notes on seasonal prevalence, frequency and fatality of

pulmonary tuberculosis,
pneumonia,
influenza, etc.

N.B.—Such susceptibility may exist permanently or may be brought about by special conditions. In the former case it may be common to all inhabitants of area or restricted to certain races or classes of population. Special conditions may be created by climatic influences, epidemics, famine, war, etc.

(b) Susceptibility of inhabitants to plague infection in general. Is there any evidence that an unusual susceptibility to plague infection in general accounts for heavy incidence of secondary lung involvement?

N.B.—Again such an unusual susceptibility may be permanent or temporary, affecting all inhabitants or restricted to certain races or classes of population.

(c) Other factors which may seem important.

III. Rise of Primary Pneumonic Plague Epidemics.

1. How do the outbreaks arise in area?

(a) Are they always traceable to bubonic cases with secondary lung involvement?

(b) Do primary septicemic cases play any causative role?

(c) Are there instances of "original" pneumonic plague outbreaks, where the initial case displays features of primary pneumonic plague?

N.B.—If such instances are on record, provide as complete details as possible. Stress etiology (e.g., furs, skins, eating of rodent meat, etc.).

2. Why do pneumonic outbreaks arise?

To what causes do you ascribe the rise of such epidemics?

(a) Extrinsic causes alone?

Discuss relation between climatic influences and pneumonic outbreaks.

(b) Intrinsic causes (like mixed infection, simultaneous prevalence of other diseases, e.g., influenza, etc.)?

(c) Discuss connection between pneumonic plague outbreaks and

i. Species of rodents and/or other animals involved. Mention for each outbreak how far local rodents are responsible, or if infection is imported by human agency (travellers developing secondary pneumonia). If the local rodents are responsible mention for each outbreak whether preceding epizootic was of long standing or short duration.

If of long standing, whether it might have received new impetus by reimportation of infection or immigration of new rodents.

ii. Species of fleas involved.

iii. Frequency and degree of cases with secondary lung involvement leading to pneumonic outbreaks.

IV. *Spread and Diffusion of Primary Pneumonic Plague.*

1. Characterise spreading power of primary pneumonic plague in area.

(a) Is its diffusibility great, leading to a series of primary pneumonic cases, or

(b) small, so that cases remain sporadic?

2. Discuss causes for diffusibility existing in area, particularly

A. *Climatic influences.*

i. Is spread favoured by absolutely or comparatively inclement weather (cold, rainy reasons, prevailing winds, etc.)?

a. Is this brought about merely by creation of unhygienic conditions (overcrowding, tightly shutting of the houses, etc.) or

b. are intrinsic factors, like atmospheric humidity, nature of soil, water supply, etc., important?

ii. Is diffusibility lessened by a warm climate prevailing? If so, what are the causes?

N.B.—To permit a further study of these problems, the following data would be valuable:

a. Exact data as to presence or absence of spread of the disease, both in houses of patients (among relatives, friends, etc.) and in hospitals and camps, etc. (among sanitary personnel, relatives accompanying the patients to hospital non-plague patients and other contacts).

b. Comprehensive description of housing conditions especially presence or absence of overcrowding, ventilation, exposure to sunlight, etc.

c. Comprehensive description of conditions prevailing in hospitals, especially their arrangement, ventilation, exposure to sunlight, arrangements for isolation of pneumonic plague patients, manner and length of their contact with personnel and others, precautions prescribed, thoroughness with which personnel and others carry them out, etc.

d. Ease or difficulty with which prophylactic measures are carried out under the climatic conditions prevailing.

e. Clinical and anatomical signs as different from those usually found.
Specially important are:—Frequency of cough, quantity of sputum expectorated, number of bacilli therein, ratio of *B. pestis* to others, extension and character of lung involvement.

f. Any other relevant data.

g. Whenever possible, the range of infectivity of pneumonic plague patients should be directly tested by the method of Strong and Teague (exposure of agar dishes in front of patients at varying distances).

B. *Social influences.*

Discussion of other factors, habits of life of population which are conducive to spread of plague, e.g., hospitality, customs when attending the sick and funerals, using clothes of dead, attitude towards measures taken by the medical authorities, willingness to co-operate, etc. Does the disease spread among all or only certain strata of the population?

C. *Other influences.*

Discussion of other factors, like racial problems, etc.

B. *Decline of Epidemics.*

Discussion of causes contributing to the decline of epidemics. Special stress should be laid upon—

1. Evidence of a tendency of the outbreaks to decline spontaneously?

N.B.—Data should be obtained regarding *pulmonary* cases (i.e. cases which succumb quickly to respiratory infection without developing marked lung symptoms), especially their frequency towards the end of the outbreaks.

2. Climatic influences.
3. Importance of measures taken by :—
   a. The authorities.
   b. The people themselves.

VI. *Incidence of Bubonic Cases in Primary Pneumonic Plague Epidemics.*

N.B.—A distinction should be made between two types of pneumonic plague outbreaks :

a. Those where the pneumonia cases form but an episode in a bubonic outbreak, i.e. "mixed" outbreaks and
b. so-called "pure" pneumonic plague epidemics, where the disease spreads mainly in pneumonic form.

Even in the latter category a small number of bubonic cases may be met with, either during the course or at the end of the outbreaks. It is very desirable to obtain fuller information upon such cases.

Discuss :

1. Frequency of bubonic cases in "pure" pneumonic outbreaks, stating whether they are observed during the course or at the end of the epidemics or on both occasions.
2. Distribution of such buboes.
3. Length of such illness and case fatality.
4. Etiology.

N.B.—For example, such cases may be caused by :—

a. Direct contact with pneuonmic plague sufferers, sputum (or other infectious material) entering the conjunctiva, nose, mouth, skin wounds, etc. of the contact.
b. Bite of human or other parasites (see VIII).
c. Coming in touch with inanimate objects soiled by the sputum (or other excreta) of the patients,
d. Handling pneumonic plague corpses.

VII. *Infectivity of Pneumonic Plague.*

1. Discuss in general—
   a. Importance of inanimate objects in spread of disease.
   b. Infectivity of fomites after removal of the patients or corpses;
   c. Infective role of pneumonic plague corpses;
   d. Food and other sources of infection.

N.B.—Of special interest are those rare cases which, though not directly infected from the patients, develop *primary pneumonic* plague.

2. If such instances are observed, record them in detail.

VIII. *Spread of Pneumonic Plague Infection to Animals.*

Does such secondary infection occur? If so, give details.

IX. *Clinical Features of Pneumonic Plague.*

Characteristic features observed in area as different from those usually described (see also IVb). Supply data on:

1. Preinfectious period.
2. Length of incubation period.
3. General symptoms, like frequency of cough, copiousness of sputum, admixture of other micro-organisms, mental, nervous and cardiac conditions (including blood pressure).
4. Atypical cases.
5. Pulmonary cases (see V. 3).
6. Slight cases,
7. Diagnosis and differential diagnosis.
8. Recoveries. Influence of therapy with
   i. Serum,
   ii. Vaccine,
   iii. Drugs.
9. Other noteworthy features.

X. *Question of immunes and carriers.*

XI. *Pathology of Pneumonic Plague.*

Description of features peculiar to area (see IVe). Discuss:

1. Lung changes,
2. Changes in upper respiratory tract,
3. Other characteristic anatomical findings,
4. Noteworthy bacteriological and serological findings, Evidence regarding mixed infection,
5. Post mortem diagnosis, especially value of lung and spleen punctures,
6. Views as to mode of infection,
7. Views as to pathology proper.

XII. *Personal prophylaxis.*

Describe measures taken in area, dwelling especially upon use and efficiency of

1. Masks,
2. Prophylactic vaccine inoculation (source?),
3. Prophylactic serum administration (source?),
4. Housing and other noteworthy measures (including measures in regard to domestic animals).

XIII. *General prophylaxis.*

Discussion of measures taken and their efficiency. Dwell upon :

1. Rodent destruction,
2. Disinfection and disinfestation,
3. Regulation of traffic,
4. Quarantine,
5. Other noteworthy measures.

XIV. *If possible forward copies of unpublished and/or not easily accessible reports, papers, etc.*

.....

Director of Institute.

City.....

Date.....

Please return the answer to this questionnaire to :

....

.....

if possible before .....

APPENDIX B.

LEAGUE OF NATIONS : HEALTH SECTION : EASTERN BUREAU.

PLAGUE RESEARCH PROGRAMME

Adopted by the Plague Expert Committee at its First Session at Calcutta, 5th—9th December, 1927.

The following investigations were considered of particular interest and were approved by the Plague Expert Committee in the following order of importance :—

A. BUBONIC PLAGUE.

1. Further investigations into the methods of destruction of rats and fleas.
2. Investigation on the comparative epidemiological rôle of the various species of fleas in plague transmission in selected areas of India, as being the most heavily infected country, the species of fleas concerned and their viability under natural conditions.
3. Survey of Plague in wild rodents of Northern Asia (Transbaikalia, Manchuria and other Chinese provinces) by an international mission, provided such mission receives substantial support from the countries concerned.
4. Investigation on the part played by grain and cotton in the dissemination of Plague and measures to prevent this spread (disinfestation).

5. Investigation of the conditions under which Plague is carried over from one season of incidence to another (problem of its recrudescence).
6. Investigations on the relative importance of rodents other than rats in the transmission of plague in various countries.
7. Investigation of rat and flea conditions in ports (shore, lighters, ships), the ship fauna being investigated both in ports and during the voyages, in eastern and western areas. This information should be collected by the Singapore Bureau for providing information applicable to quarantine measures.
8. Prophylaxis and therapeutics :
   (a) speedy preparation of anti-plague vaccine,
   (b) possibility of reducing local reaction to anti-plague vaccine,
   (c) possibility of producing a plague anti-toxic serum,
   (d) further studies on anti-plague bacteriophage and its practical applications,
   (e) chemiotherapy of plague.

B. PNEUMONIC PLAGUE.

1. Investigation of the incidence of bubonic plague cases in outbreaks of pneumonic plague; relative incidences of cases of bubonic plague, secondary pulmonary plague and primary pneumonic plague in the various outbreaks.
2. Study of the possibility of existence of a special ultra-virus or filter-passing form of B. Pestis as the causative agent of pneumonic plague.

INTERNATIONAL ASPECTS OF PNEUMONIC PLAGUE.

Pneumonic plague was until within recent years considered as an exotic disease either limited to a few areas or occurring under special climatic conditions, e.g. cold. It is realised now that this form occurs not only in isolated but in general plague areas, at both cold and warm latitudes. In fact, there are quite a number of places where the appearance of the pneumonic type is to be anticipated whenever plague breaks out; instances of such countries are Upper Egypt, French West Africa, the Gold Coast, Madagascar, the Azores, Khorassan (Persia), South-East Russia, certain parts of Central Asia and Transbaikalia, Mongolia and the adjoining territories. Frequent pneumonic outbreaks have been reported recently from Tunisia and it remains to be seen what will be their further development in this country. In a number of other plague foci, like the Punjab and other districts of Northern India, Java, California, South Africa, Morocco, Portugal, the pneumonic type—though not of such common occurrence as in the above-mentioned countries—is frequent enough to influence local plague conditions. In other places, such as, China Proper, South America, Algeria, Central Africa, Eastern Europe and the Near East, plague pneumonia—though not overshadowing the character of the prevailing bubonic epidemics—is also observed now and then. Finally, it must be kept in mind that in a further group of plague areas, where lung pest is now rare, it has existed soon after the original introduction. It is possible that in future, when new territories are invaded by the disease, a similar development may occasionally take place.

This rapid review shows that, so far as geographical distribution is concerned, pneumonic plague may claim some international attention, and it may be wise to discuss certain features in this connection. One may first review shortly the relation of this type of the disease to other forms. It is known that in some quarters pneumonic plague is considered as a disease *sui generis*, due not to an infection with the *B. pestis* alone but to a mixed infection with some other causative agent. Many observers, including myself, think that this hypothesis cannot be upheld. In almost all outbreaks the pneumonic form did not arise *de novo* but could be traced back to bubonic plague, the connecting link being cases showing secondary lung involvement. Sometimes simultaneously existing cases of bubonic and pneumonic plague could be traced back to one and the same source. In practically all big pneumonic outbreaks some bubonic cases are observed, many of which received infection more or less directly from the pneumonic patients. Further it is claimed, specially by Indian observers, that during pneumonic plague epidemics rats may become secondarily infected and in their turn are responsible for bubonic cases among man. All this epidemiological as well as the experimental evidence speaks against any fundamental difference between the bubonic and the pneumonic type, and it may be said that the appearance of the latter is nearly always dependent upon preceding epizootics as well as bubonic

plague. On the other hand I am far from denying that certain influences which are not yet fully elucidated may favour the supervention of secondary lung involvement and thus of primary plague pneumonia. Thus it may be mentioned that travellers suffering from bubonic plague are liable to develop marked secondary lung involvement, and numerous instances are on record where such individuals were the cause of pneumonic outbreaks in localities hitherto freed from plague of any kind.

However, while admitting that certain factors, like the species of rodents involved in the epizootics, the length of time the latter existed, susceptibility of the early victims to respiratory diseases or plague infection, etc., may help to shape the character of the subsequent epidemics, I must emphasize that their influence is restricted to the genesis of the outbreaks and that, once primary pneumonic plague is established, its further spread depends not upon intrinsic causes but upon extrinsic conditions.

Turning to the question as to how primary pneumonic plague infection is propagated particular emphasis should be laid upon its direct spread from man to man. Reliable instances, where not the patients directly but their houses or infected inanimate objects were responsible for further cases, are rare and it cannot be assumed that such indirect factors contribute materially to the spread of the disease. A secondary infection of rats, as mentioned above, has thus far been recorded from a few plague areas only, whereas during many extensive pneumonic outbreaks either the rodents remained free or only sporadic cases, not a widespread epizootic, was noted among them. It might be argued that under these circumstances pneumonic plague epidemics are problems of local interest only; in other words,—being spread solely from man to man—they are easily suppressible by hospitalisation of the patients, isolation of the contacts and similar means. There is no doubt that such argument is theoretically sound, but experience has shown that theory and practice coincide only when such conditions as the following exist :

(*a*) When the existence of pneumonic plague is quickly diagnosed in a country with a good sanitary organisation and a population living under hygienic conditions, and willing to co-operate with the medical authorities or at least not opposing them;

(*b*) When the people themselves are well acquainted with the disease, know how to take care of themselves and have—owing to their nomadic habits—the chance to practise crude isolation of the patients, evacuation of the healthy, etc.

If such favourable circumstances are absent, there will be more or less spread of infection. Often the disease will not only gain a firm hold at the place of its origin but also spread *per saltum*, being introduced by travellers incubating or actually suffering from lung pest to other localities. Such spread will be slow in sparsely populated countries or these with slow communication. *Vice versa*, an epidemic will cover long distances with almost lightning rapidity, as soon as modern means of communication are available.

It may be well to discuss now in some detail these various modes of spread. The Shansi epidemic of 1917-18 spread at first slowly, infection being effected by people travelling in carts and making 20-30 miles' progress a day. When some railheads were reached, quicker and wider spread took place. The Manchurian epidemics are examples of such rapid spread through railways. Recently the motor-car has added to our difficulties. Traffic between Manchuria and Mongolia is now done by motor-car, and during the 1926 (Nov.-Dec.) outbreak in Mongolia, it was feared that persons leaving Mongolia soon after infection might reach Manchuria in the incubating stage. Fortunately, thanks to early preventive measures, our fears were not realised.

A special role is played by steamers as these may not only serve as vehicles for persons incubating or suffering from lung pest but also for plague rats which in their turn may infect human beings and thus give rise first to bubonic and then to pneumonic cases. We have records of instances of both kinds.

A. *Examples of pneumonic infection introduced on board ship* during pneumonic outbreaks were seen during the 1921 Vladivostock epidemic :

1. S.S. Kisheneff : Left Vladivostock on April 28, 1921, for Chefoo. April 29 one fatal case of pneumonic plague detected in a Chinese passenger. All possible measures were taken on board. On May 3 another Chinese, who had slept side by side with deceased, showed symptoms of plague. Steamer arrived same day at Chefoo and was quarantined at Kentucky Island. Unfortunately no strict watch could be kept so that many of the quarantined escaped on May 5. On the island further cases developed, giving a total of 12.

   April 29 ..... 1 death,
   May 4 ..... 6 deaths,
   May 5 ..... 5 deaths.

   Strict measures were taken at Chefoo port and no cases occurred. However, 5 deaths occurred in a neighbouring village, 10 miles east of Chefoo. These were traced to one of the escaping contacts.

2. On June 8 the British steamer *Ralph Moeller* arrived at Chefoo from Vladivostock and reported three deaths from plague during the voyage. Quarantined at Kentucky Island, which had been fitted out in the meantime. No further mishap.

3. The British steamer *Kiangsi* arrived from Vladivostock at Tsingtao on June 4 with one case of pneumonic plague on board. On the 7th a second case (mother of victim) occurred. No further cases.

B. *Examples where pneumonic cases developed on board ship* :

1. *S.S. Friary* : This ship, with a crew of 21 persons, left the port of Alexandria on December 22, 1900 after a stay of 12 days; the whole crew had been on shore during this time. The

vessel, carrying cotton-seed from Alexandria to Hull was at Algiers between December 30-31 and arrived at Hull on January 10, 1901. Some dead rats had been found in her after leaving the Egyptian port. Twelve hours before arriving at Hull, one sailor died of 'Influenza'. On January 12 two more sailors fell sick with lung symptoms, diagnosed afterwards as pneumonic plague. Up to January 21, 8-9 persons, including a private practitioner, were attacked. All these died except the doctor, who recovered having shown but few plague bacilli in his sputum. The first patient had not been seen medically, and it is therefore difficult to say definitely what type of plague he died from.

2. *S.S. Cheongshing*: Arrived from Hongkong at Tientsin on May 31, 1912. The wife of the 1st purser, who had lived in a house at Hongkong where two deaths from plague had occurred just before her coming on board, died on board on May 29. On June 2, her husband fell ill with pneumonia and died on June 3 in a hospital. On June 4, the second purser fell ill, who had been in close contact with foregoing and died next day. Pneumonic plague was established in his case. In the meantime steamer had left for Chefoo, but the authorities there having been informed by wire did not permit landing. The ship proceeded to Shanghai where it was quarantined. Two more cases occurred during the journey to Shanghai. Thus at least five cases occurred on board.

3. *S.S. Nagoya*. Arrived in London on October 25, 1919, from the Far East with 16 officers, 150 native crew, 29 European and 200 deck passengers. On arrival she reported one death from "influenza" during voyage and six cases of same disease among native crew. The plague nature of these and subsequent cases was subsequently established, and after some search rat plague was also detected on board. Altogether there were 8 human cases, all fatal. The early history of this outbreak is interesting:

   The ship surgeon considered the first case occurring during the journey (October 21-23) as malaria and the later ones with lung symptoms as influenza. When the ship arrived at Plymouth on October 24, (before reaching London) no plague was suspected there, though besides 3 "influenza" cases one was seen who had an enlarged and indurated cervical gland which had existed for 14 days; this was taken to be ordinary adenitis. Consequently permission was given for some 50 passengers to land and for the vessel to proceed to London. Luckily the passengers had evidently had no contact with the patients and no further mishap occurred.

4. *S.S. Bega*: This vessel was employed in the coastal trade of Australia, returning to Sydney at regular intervals for fumigations. Besides produce, she carried empty sacks, crates, etc. which might harbour rats. On June 16, 1906 a seaman reported ill on board, stayed off and reached his home at Balmein (suburb

of Sydney) on June 19. He was quite well on the 15th, though he confessed afterwards that a week before he had picked up a dead rat with his fingers. Later on, after being scolded he picked up another rat with iron tongs in the engine room.

The medical man attending the patient diagnosed slight catarrh, and next day improvement was apparent. On June 22, another doctor was called in, who found pneumonia. Next morning patient was collapsed and cyanotic, and he died soon afterwards.

The patient lived in a comfortable house with wife and nine children. The wife who nursed him fell ill on June 26 (three days after his death) and died 48 hours later. Her medical attendant suspected plague pneumonia and reported the case. Autopsy confirmed this diagnosis. Another woman who repeatedly attended the first victim fell ill on June 28 and died after two days of pneumonic plague, confirmed at postmortem. Her husband and four children, who had been in and out of her room until 12 hours before she was sent to hospital, escaped infection, as did other contacts of the first victim. All these visitors received subcutaneous injections of serum. The Report stated that all three patients had little cough, hence the escape of the 29 contacts.

5. *S.S. Calypso*: This steamer plied between the two healthy ports of Venice and Trieste. Towards the end of Oct. 1906, she took into Trieste a cargo of cotton and jute, which had been brought into Venice from India by another steamer. The quartermaster who had handled the transhipping fell sick and died at Trieste on Nov. 8. Postmortem examination roused a suspicion of pneumonic plague, which bacteriological diagnosis confirmed. Two rats and 18 mice, found on board after fumigation, were found healthy. This case may be termed a true 'original' case of pneumonic plague.

Besides steamers, danger from smaller craft has to be considered. Though these may proceed more slowly and make shorter journeys, they are in a way more dangerous than steamers as they may leave infected ports stealthily and land passengers outside the regular ports. In fact during the 1910-11 Manchurian epidemic this occurred repeatedly, and coolies—in order to evade quarantine at Dairen—hired junks and crossed over to the Shantung coast; one instance is on record where two men reached the shore dying from pneumonic plague.

So far we have considered only instances where *pneumonic* plague cases were imported by ship. Another important feature must not be forgotten: Plague infection imported by ships through infected rats may give rise to human outbreaks on shore, in which case pneumonic cases preponderate. One notable example of this is the 1914-15 epidemic in French West Africa. Infection was introduced into the capital Dakar in April 1914, most probably by the steamer *Mingrelie* from Casablanca (Morocco). It led to almost 1500 cases in this City—at first mainly

septicaemic and pneumonic in nature; later on the epizootic spread and the bubonic type became preponderant in man. The epidemic extended also to other places in the colony and the cape Verde Islands, and claimed altogether 9,000 victims.

The above evidence shows that the danger of spread of pneumonic plague infection (directly or indirectly) through ships should not be underrated.

If we ask what measures should be taken against such a spread of pneumonic infection per saltum, it is certainly true from a theoretical viewpoint that the measures taken within the infected area should be sufficient to prevent further spread. As practice has shown, however, it is absolutely necessary to supplement them by a preventive campaign outside the foci in which not only the authorities but also private undertakings, like railways, steamship companies, etc., must take part.

It is particularly difficult to carry out this complicated system of measures if not one but several countries are involved or threatened by pneumonic plague epidemics. A prominent instance is Manchuria, which country is practically free from indigenous plague and yet is threatened yearly by an invasion from Transbaikalia or Mongolia. The epidemics of 1910-11 and 1920-21 can all be traced to the endemic region of Transbaikalia

It is hoped closer international co-operation may be obtained against pneumonic plague, so that through early notification of cases and concerted action the outbreaks will be early stamped out.

Up to date very little notice has been taken of pneumonic plague in laws relating to maritime quarantine. Perhaps these humble remarks of mine may help to focus attention upon the need of revising present laws with a view to ensuring greater safety among trading nations.

Lastly international enquiries in the problems of plague pneumonia will undoubtedly contribute towards a fuller knowledge of this problem and thus strengthen our defenses against this scourge.

LIN CHIA SWEE, M.D.,
Senior Resident Med. Officer, Harbin.

RESEARCHES UPON PLAGUE IN CHINA.

(Being an address delivered at the opening of New Science Buildings, Lingnan University, Canton, Oct. 19, 1928).

The inauguration of the new Science buildings at Lingnan University is another reminder of the splendid progress which your Institution has made in the development of higher education, and I may be permitted to add my humble congratulations to you upon this achievement.

The history of independent Chinese researches upon plague, which I wish to speak to you about to-day, dates back to the year 1910-11. As you are probably aware, a great epidemic of pneumonic plague invaded Manchuria and North China at that time claiming 60,000 lives and monetary losses amounting to at least 100 million dollars. The only consoling thought is that by cutting short the new Black Death, we prevented it from invading other parts of the world. In China herself medical progress and a better understanding of public health among the people has made considerable headway after that event. Many of those in authority who had formerly pledged their faith to the old-fashioned medicine now saw that its methods were powerless against such severe epidemics. For the first time in history, an International Medical Conference was called together by the Chinese Government, the deliberations of which shed much light upon the almost unknown problem of pneumonic plague. The most gratifying result, however, was the establishment in 1912 of the North Manchurian Plague Prevention Service with headquarters at Harbin. This is one of the few institutions in the world whose primary function is to fight plague by all possible means, and its influence upon medical and health progress as well as Chinese outlook upon scientific research has been far-reaching.

The main principle of modern medicine is to replace the cruder methods of former days (when medical practitioners were satisfied with the *curing* of patients) by rational *preventive* measures based upon a thorough understanding of the causes of sickness. In the case of pneumonic plague, which only occasionally invades Manchuria, it was by no means easy to realise this object at once. It is true that ancient folk lore passed among the natives of Transbaikalia and Mongolia from one generation to another had pointed to the Siberian marmot or (in Mongol) *tarabagan* as the cause of plague. They believe that a mysterious disease has been rampant since time immemorial among these animals, and that it may pass over to those human beings who disturb their peace. But whatever evidence available was circumstantial only, no one having properly investigated the diseased animals. Immediately after the 1910-11 epidemic, a few sick tarabagans were found by a Russian doctor but his information was not definite enough to vouchsafe final conclusions. For no widespread disease seemed present then among the marmots of that region, and the infection of the few animals might easily have been the result rather than the cause of the human outbreak. Our Service members were obliged to

wait for over ten years before another opportunity came to visit Siberia and trace the origin of plague among these tarabagans. In the meantime, we sought to obtain further knowledge by investigating step by step their natural habits, their morphology, their parasites, etc., and by studying their susceptibility to plague in the laboratory. We began to acquire a thorough knowledge of the healthy tarabagan and its mode of life. We ascertained that its specific fleas could bite human beings as well as guinea-pigs and other rodents. We found finally that the tarabagan was susceptible to plague infection not only through the ordinary channels common to rats, e.g. in the skin (percutaneous method), under the skin (subcutaneous method, etc.), but also by inhalation, in which case an illness developed comparable in most aspects to pneumonic plague in man with fever, coughing, bloody sputum and sudden death. By taking advantage of this fact, we have been able to contribute to the elucidation of many problems connected with pneumonic plague and confirm that in this disease the lungs become directly infected through the lower respiratory tract whereas it was formerly believed by many that the bacilli first entered the throat and only reached the lungs secondarily through the blood stream.

Our studies upon tarabagans had prepared us for the time when the opportunity for investigating naturally-infected rodents offered itself in 1923. As soon as this news did reach us, our Service sent an expedition to the affected parts of Transbaikalia and co-operated with the Russian doctors working there. We were soon able to establish irrefutable proof that outbreaks of plague do occur in certain areas among these Siberian marmots. Besides obtaining cultures from the several organs of animals found sick or dead in the fields we have made comprehensive histological examinations which fully confirmed our belief as to the existence of natural plague among tarabagans.

Satisfactory as these results were, it was clear that many problems still awaited solution. Foremost among them was the question as to the role of the tarabagan fleas in the spread of the disease. Should these parasites be found capable of carrying infection, then they might be equally dangerous to man and rodents. After several attempts our experiments finally proved that various susceptible rodents may be infected by fleas collected from diseased marmots.

The next problem to occupy our attention was whether the tarabagans, which had now been proved to be the immediate cause of the human outbreaks, occurring almost yearly in the fields of Transbaikalia and Mongolia, were at the same time the original and permanent source of the plague. It is known that in certain regions where plague is entrenched, as in India, the bacilli are constantly harboured in ordinary rats, in which evidence of infection may be found the whole year round. The Siberian marmot seemed to lie in a class by itself on account of its prolonged hibernation period. It was once maintained by some scientists as well as laymen that animals which become infected with plague in the course of the warm season do not retire to the burrows with approach of the winter but stay in the open to die. In this way only healthy animals would survive the winter and if—as is now established—plague is present among the marmots every summer, they must contract infection in spring

from some source which is not influenced by the winter, for instance, from some non-hibernating rodent species. We have always entertained grave doubts about the accuracy of this theory because early experiments had convinced us that hibernating tarabagans are liable to contract plague, their illness often lasting considerably longer than in summer. A systematic study of the problem was hence desirable, and we have during the last three years devoted much energy to its solution.

These researches have now confirmed our previous findings that hibernating tarabagans do contract plague in much the same way as non-sleeping animals. Further the disease in many animals follows in winter a much prolonged and unusual course. Some of those infected in winter apparently recover from the disease, while in the case of others it lies dormant and only becomes acute and fatal in spring.

Though there still remain a few unsolved problems it may be claimed that the fundamental facts about tarabagan plague have been established, showing that these Siberian marmots are the main reservoirs of plague in the north eastern steppes of Asia. And this fact is by no means of local importance only. To explain this, we must briefly survey the history of plague in general. As most probably known to you, plague which had become almost forgotten in the course of the nineteenth century again attracted attention after 1894. In that year a widespread epidemic of bubonic plague started in Hongkong. It was in that island that the bacillus causing the disease was claimed to have been simultaneously discovered by both Kitasato (a Japanese) and Yersin (a Frenchman). Without prejudice we may say that we owe to the former the earliest account of the organism, and to the latter its first detailed and accurate description.

Sir W. J. Simpson, who has published a treatise on plague, contended that the pest invading Hongkong in 1894 had come from Yunnan province and concluded—rather hastily—that this area was a permanent home of the disease. This assumption is disproved by our enquiries which show that for at least 30 years Yunnan has been and is freed from infection. Furthermore our historical studies establish that that western province of China was itself invaded from the west. In my opinion, the present pandemic of plague had certainly started from Central Asia long before it became manifest in Hongkong in 1894. It is also highly probable that this vast Central Asian plateau is the original home of the disease which in previous centuries had been known to spread from there westwards as well as eastwards. Moreover, we know that Central Asia is populated by big rodents more or less related to the Siberian marmot. In fact the latter, which inhabit Transbaikalia, Mongolia and Northwest Manchuria up to the western slopes of the Hingan mountains, may be called the eastern representatives of a vast group of rodents similar to them in constitution and habits. There is no doubt that in parts at least of the Central Asian plateau these rodents periodically develope plague and transmit the disease to man. And we may assume that the infection entrenched in those almost inaccessible regions follows the same laws as those applicable for the tarabagan.

I have always considered it desirable to support this by actual studies of conditions in those remote areas. I am glad to say that there is now every hope that the first steps is this direction will be taken next summer. In this connection, may I express the hope that simultaneously with our work in the north, similar researches could also be undertaken in other regions of China which are threatened by plague such as Kansu, Shansi, Kwangtung and Fukien provinces? Regarding Kwangtung, in whose capital the famous Lingnan is situated, the disease in its bubonic form has implanted itself for nearly half a century now in a serious, now in a mild form. May I express the hope that the spirit of research which has filled so many of you engaged in various scientific and industrial activities, may also move some keen-minded young physician to devote his time to this vastly interesting problem, which affects the physical and material welfare of so many people in these parts? Whatever discoveries be made in this direction will surely redound to the credit of Kwangtung as well as his alma mater.

WU LIEN TEH, M.D., LL.D.

ENDOPARASITES DU TARABAGAN.

INTRODUCTION.

On sait maintenant que le Tarabagan est la source de la peste pulmonaire dans les régions du Nord de Manchourie et de la Sibérie.

Chaque année le N.M.P.P. service est approvisionné d'un grand nombre de ces animaux capturés vivants, en Mongolie, pour les recherches, afin d'éclaircir la question d'épidémiologie de cette maladie. Comme nous faisons partie de ce service, nous avons eu l'occasion d'examiner de nombreux Tarabagans et nous avons trouvé chez eux des parasites intestinaux qui nous ont semblé intéressants.

Cet article est le compte-rendu succint des résultats que nous avons obtenus pendant ces derniéres années.

DESCRIPTION DES ESPECES TROUVEES.

1. *Ascaris sp.* : (Voir : "Report of the N.M.P.P.S. ' de 1927)

Cet Ascaris a été vu aussi per Monsieur Sukneff de Tchita en 1923. Cette année, nous en avons rencontré deux, chez un Tarabagan et dix, chez un autre.

2. *Entamœba sp.* :

Cette amibe ressemble beaucoup morphologiquement à l' Entamœba coli. Son diamètre est de 15-30 u. Celui du noyau qui prend fortement les colourants est de 5 à 7 environ. On distingue difficilement l'endoplasme de l' ectoplasme. Ses mouvements sont très lents. Nous avons souvent observé des bacteries et des sphaerites ingérées dans cette amibe

3. *Enteromonas sp.* :

Nous avons vu dans les selles de la plupart des Tarabagans présentant un syndrôme dysentérique, un flagellé qui ressemble à l'Enteromonas hominis da Fonseca. Cet Enteromonas est de forme sphérique ou ovalaire et animé de mouvements extrêmement rapides, connus sous l'expression "rapid dancing mouvements" de Wenyon et O'Connor. Il change de forme d'une façon brusque et frêquente. Il n'y a ni cytostome ni membrane ondulante Les jeunes flagellés présentent à leur partie posterieure un prolongement à extrémité pointue, long de 6 à 8 u. La forme prékystique mesure 16 à 18 u. de longueur et renferme dans son protoplasme de nombreuses granulations trés chromophiles. Ce dernier caractère différencie ce flagellé des autres Enteromonas.

Le noyau est situé à la partie antèrieure du corps. La partie entre le Karyosome et la membrane nucléaire ne prend aucun colorant. Du blépharoplaste situé en avant du noyau, partent quatre flagelles dont trois antèrieures sont libres et une postèrieure passe le long du corps avec lequel il est intimement en contact. Les flagelles se colorent difficilement, Parfois on ne voit qu'une ou deux flagelles antèrieures au lieu de trois et la flagelle postèrieure ne se colore que dans sa partie intro-protoplasmique. La forme prékistique renferme des masses de glycogène une grande ou bien trois ou quatre petites, décelables par le Lugol double ou par la méthode de Best.

CULTURE.

a.—Ces Enteromonas contenus dans les selles de Tarabagans peuvent être conservés pendant plus d'un mois dans un sérum artificiel normal et à la température de 20°C. Nous n'avons pu observer les formes de division.

b.—*Milieu de Musgrave Clegg.*

Quand on les ensemence dans ce milieu à la température de 37° C. dés le lendemain, on peut rencontrer de nombreuses formes de division de ces flagellés.

La dégénération sera retardée si on les cultive à la température de 25°C environ—Dans cette condition les flagellés commencent à se diviser au bout de 3 ou 4 jours.

c.—*Milieu ovomucoïde de Hogue.*

d.—Sérum de Tarabagan :

Sérum	1
Solution salée à 1.5%	10

Dans ces deux derniers milieux (c et d) ils se multiplient très rapidement, mais par contre la dégénération est aussi précoce; on ne peut donc les conserver dans ces milieux aussi longtemps que dans le milieu de Musgrave-Clegg.

ROLE PATHOGENE.

On trouve constamment ces flagellés en nombre plus ou moins grand dans les selles de tout Tarabagan atteint de syndrôme dysentérique; mais on ne peut dire à l'heure actuelle, si ces flagellés sont la cause même de ce syndrôme chez les Tarabagans ou bien si ce ne sont que des "germes de sortie". Cette question pour être élucidée demande des recherches ultèrieures.

CONCLUSION.

En résumé, on peut rencontrer dans les selles de Tarabagans :

1.—Ascaris sp.
2.—Entamœba sp.
3.—Enteromonas sp.

En outre, on note parfois, l'existence de kystes ressemblant à ceux de Blastocystes hominis.

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ENTAMOEBA BOBACI N. SP. DES TARABAGANS (MARMOTA BOBAC).

(Reprinted from Annales de Parasitologie, 1928).

Nous avons trouvé dans les matieres fecales, chez 7 des 43 tarabagans capturés en Mongolie, en dehors des Trichomonas, des Tricomastix, des Hexamitus et des Chilomastix, une espèce spéciale d'amibes. Cette amibe se trouve généralement dans le cæcum et dans le colon des tarabagans et ressemble beaucoup a l'Entamœba coli.

1. Examen a l'état frais.—A l'ètat frais, ses mouvements sont relativement plus lents que ceux de l'Entamœba dysenteriæ et ses pseudopodes sont plus hyalins et plus gros que ceux de l'Entamœba coli; sous la forme de repos, on ne distingue plus l'ectoplasme de l'endoplasme. Le noyau est presque toujours visible pendant la locomotion.

2. Examen après coloration.—La fixation a été faite dans le liquide de Bouin et la coloration a l'hématoxyline ferrique. Les dimensions de cette amibe sont ordinairement de 20 à 35 M; on trouve également des formes de 15 à 40 M, ce pui est à peu près la taille de l'Entamœba coli (20-30 M, Brumpt, Dobell). Nous avons mesuré 100 amibes et nous avons fait une moyenne qui est de 24 M.

Le protoplasme granuleux est relativement plus pâle et moins dense que celui de l'Entamœba coli et de l'Entamœba dysenteriæ. On y trouve un grand nombre de vacuoles contenant diverses substances ingérées par l'amibe. Parfois, les pseudopodes sont très grands (fig. I et III), comme ceux de l'Entamœba muris. Cette amibe est très vorace : on trouve dans ses vacuoles toutes sortes d'objets : bactéries, levures, Hexamitus, Eutrichomastix, etc., ces derniers morts ou dégénérés; en somme, tout le contenu normal de l'intestin du tarabagan, mais on n'a jamais observé l'existence de globules rouges. A peu près 60 p. 100 de ces amibes sont parasitées par une espèce de Sphærita (fig. I et IV). Le parasitisme des Sphaerita a été signalé pour la première fois par Dangeard et constaté par differents auteurs (Léger et Duboscq, Chatton et. Brodsky, Dobell, Wenyon, Nöller, Brug, Mattes et Brumpt, etc.) chez diverses amibes. Mais on a rarement observé un pourcentage aussi élevé d'amibes parasitées. Ces parasites, de forme sphérique, sont de volume très variable, depuis la limite de la visibilité jusqu'a 2 M 5 : leur nombre varie de l'unité jusqu'à quarantaine. La destruction des amibes par les Sphaerita a déjà été démontrée par divers auteurs. Nous avons observé qu'à un certain degré de développement, elles causent une hypertrophie considérable du protoplasme de l'amibe; celle-cit finit par éclater. Une autre espèce de Sphaerita, à spores en forme d'anneau (fig. II et VI), mesurant 4 M sur 2 M 5, signalée par Nöller et Brug chez l'Entamœba coli, parasite parfois l'amibe en question. Cette Sphaerita est de couleur plus foncée à son extremité anterieure; le reste est brun clair. Ces deux Sphaerita peuvent coexister dans la meme amibe (fig. VI).

Le noyau, qui mesure de 3 M 5 à 7 M est excentrique ou subcentrique et caractéristique du genre Entamœba; il est sphérique, vésiculeux, avec une couche de chromatine périphérique constituée par des granules sphériques rapprochés les uns des autres: on voit dans les préparations bien différenciées "des nodules appliqués contre la membrane, bien individualisée faisant saillie dans la cavité du noyau." (Chatton).

Le karyosome volumineux est en général excentrique ou subcentrique, très rarement central. Il existe des granules chromatiques très fins dans la zone intermédiaire entre la membrane et le karyosome.

Dans un seul cas, nous avons rencontré une amibe (fig. VII) du diamètre de 15 M avec un noyau ressemblant tout a fait à un noyau de Karyamœbina (Kofoid). Nous ne pouvons pas dire actuellement si cette amibe est une forme spéciale de l'Entamœba bobaci ou bien s'il s'agit d'une autre espéce.

3 Enkystement.—Chez ces 7 tarabagans nous avons examine les matières fecales et nous avons fait aussi l'autopsie après leur mort, mais nous n'avons trouvé de kystes ni dans le caecum, ni dans le colon.

Grâce a l'obligeance du prof. Brumpt, nous avons pu récemment observer plusieurs fois, dans son laboratoire, la presence en grand nombre de kystes d'Entamœba muris, principalement dans le caecum des souris grises. Dans son article de 1923, Kessel dit que l'enkystement de Councilmania (Entamœba) muris se fait la plupart du temps dans le caecum. Etant donnée la constation de Kessel, nous sommes étonné de ne trouver de kystes de l'Entamœba bobaci ni dans le caecum, ni même dans le colon des tarabagans, bien que nous ayons trouvé de nombreuses amibes sous la forme végétative et prékystique (fig. VIII). Peut-être l'enkystement de cette amibe est très rare, comme dans le cas de l'Entamœba cobayae, signalé par Holmes. Sul ce sujet, nous allons faire d'autres recherches.

4. Rôle pathogène.—Cette amibe n'est pas hématophage et par conséquent n'est probablement pas pathogène pour son hôte. Nous avons, d'ailleurs, examiné microscopiquement le caecum et le colon de quelques tarabagans et nous n'avons trouvé aucune lésion histologique spéciale, ni constaté la pénétration des amibes dans les tissus.

5. Classification.—Quoique cette amibe ressemble a première vue à l'Entamœba coli, elle se distingue, cependant, de cette dernière par ses pseudopodes plus hyalins et plus gros et son protoplasme moins dense. Elle doit donc former une espèce tout a fait à part. Nous l'avons nommée, d'après son hôte, Entamœba bobaci.

Je désire exprimer ma reconnaissance au Prof. Wu Lien Teh, Directeur du Bureau antipesteux a Kharbine, qui a bien voulu me permettre de terminer ce travail, ainsi qu'au Prof. Brumpt et au Dr. Langeron, qui m'ont aimablement aidé de leur conseils au cours de mes recherches.

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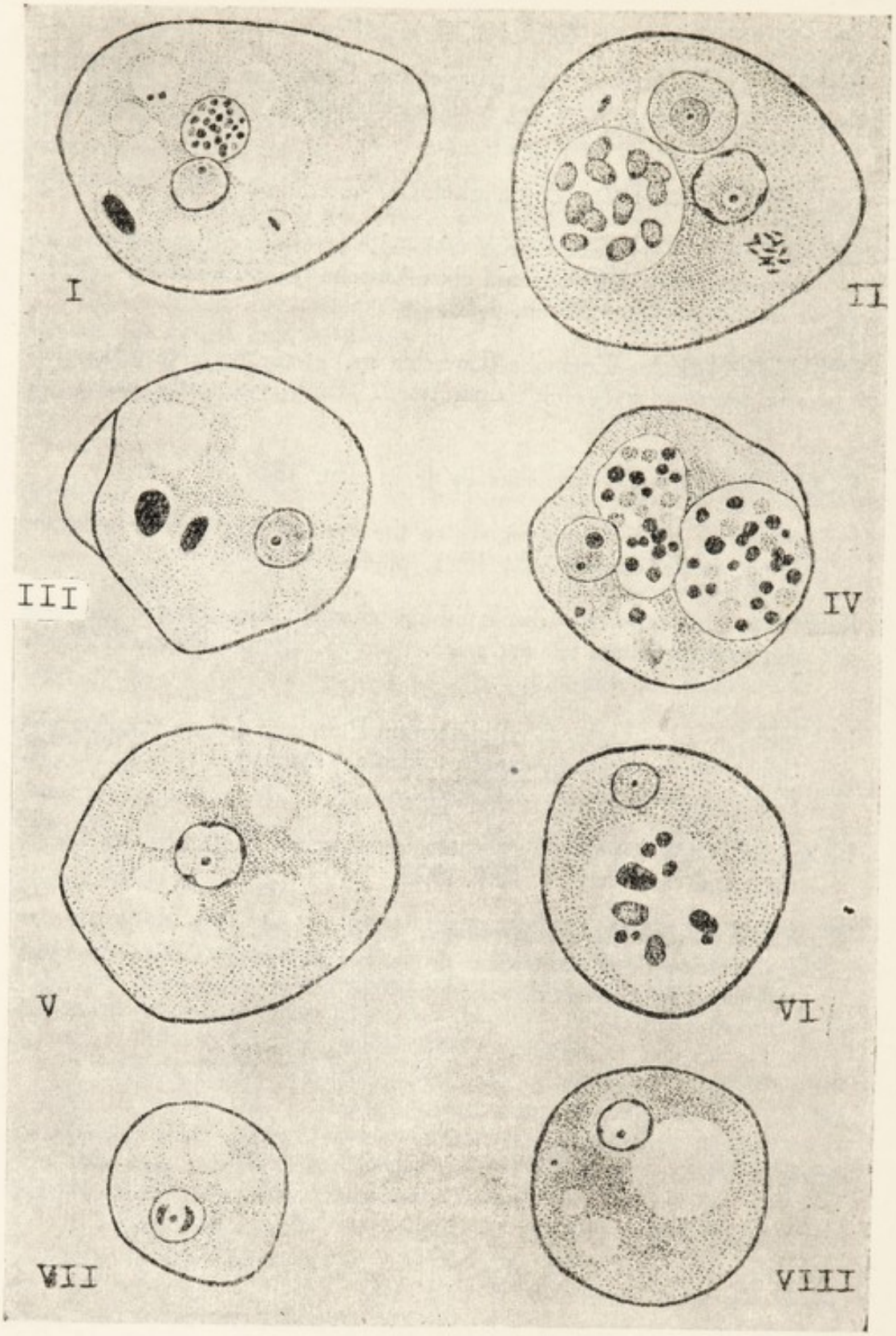

Fig. 18.—Entamoeba bobaci n. sp. I et II, formes végétatives, parasitées par deux Sphaerita différents; III, forme végétative présentant, ainsi que I et II, un pseudopode hyalin, très développé; IV, forme végétative, parasitée par deux individus d'un Sphaerita; V, forme végétative; VI, forme végétative parasitée à la fois par les deux Sphaerita; VII, forme végétative du type Karyamoebina; VIII, forme prékystique.

BEITRAEGE ZUM STUDIUM DER PEST UNTER DEN INSEKTEN.

II. Mitteilung.

(*Reprinted from Zeitschrift für Hyg. u. Infkh. Bd.* 107, p. 498.)

ALSO ENGLISH SUMMARY.

Während über die Verbreitung der Pest durch Blutparasiten aus dem Reiche der Insekten und über das Schicksal der letzteren nach erfolgter Pestinfektion sehr umfangreiche Studien angestellt wurden, ist die Literatur über pestinfizierte Insekten, welche keine Blutparasiten darstellen, verhältnismässig spärlich und nicht frei von widersprechenden Angaben.

Man interessierte sich gewöhnlich nur für die Lebensfähigkeit der Pestbacillen beim Passieren durch den Darm der jeweiligen Insekten und machte namentlich den von Insekten abgeschiedenen Kot zum Ausgangspunkt von Untersuchungen.

So fand z. B. *Küster*[9, 10]), dass die Pestbacillen bisweilen imstande sind, den Darm von Küchenschaben (Blatta orientalis) nach Fütterung der Tiere mit infektiösem Material (Agarkulturen) noch in virulenten Zustande zu passieren: *Knauff*, einem Mitarbeiter *Küsters*, gelang es, nach einer Reihe von negativen Versuchen, mit ganz frisch abgesetzten infizierten Faeces von schwarzen Küchenschaben eine Ratte durch peritoneale Injektion mit Pest zu infizieren. Es wurde hierbei betont, dass nur *ganz frische* Faeces die Infektion herbeiführen konnten. Nach *Guiseppe Cao*[3]) passiert der Pestbacillus Käfer und Schaben lebend und virulent [zit. nach *Dieudonné* und *Otto*[4])].

Ueber die Möglichkeit einer Uebertragung von Pest durch Fliegenkot und besudelte Fliegenbeine berichten *Albrecht* und *Ghon*[1]), *Nuttal*[12]) und *Hunter*[7]).

Auch Ameisen werden von *Hankin*[6]) beschuldigt, Pestübertragung teils durch Benagen von Pestleichen, teils durch Verschleppung von infektiösem Material bewirkt zu haben.

Eine besonders grosse Rolle spielen in der Geschichte der Pest die Heuschrecken. Sie wurden seit dem Altertume als Verkünder und Verbreiter von Pestepidemien[13, 17]) angesehen.

Ueber das *Schicksal der Insekten selbst* nach der Pestinfektion existieren nur wenige und oft widersprechende Angaben.

Hankin[6]) berichtet, dass Ameisen, welche virulente Pestkeime verschleppen, dabei selbst der Seuche zum Opfer fallen können.

Yersin[14—16]) vermutet, dass die Fliegen durch Aufnahme pestinfizierter Nahrung zugrund gehen. *Nuttal*[12]), welcher in einer grösseren Versuchsreihe Fliegen mit Pest infizierte und weiter beobachtete, kam zum Schluss, dass diese Tiere nach Fressen von pesthaltigem Material zugrunde gehen, und zwar um so schneller, je höher die Temperatur ist, bei welcher sie gehalten wurden. *Sticker*[13]) beobachtete unter den Aas-

fliegen zu Bombay, welche an pesthaltigem Sektionsmaterial gefressen hatten, ein Massensterben. *Albrecht* und *Ghon*[1]) sowie *Hunter*[7]) hingegen konnten unter den Fliegen ihres Sektionsraumes kein auffallendes Sterben wahrnehmen.

Gosio[5]) berichtet, dass mit Pestfleisch gefütterte Larven nekrophiler Insekten (von Musca domestica. Calliphora vomitoria und Lucillia macellaria) am Leben bleiben und sich ungestört zum vollentwickelten Insekt ausbilden können; letzteres geht jedoch dann sehr rasch an der Pestinfektion zugrunde (nach 15—24 Stunden). - Auch ausgewachsene Fliegen sterben nach diesem Autor kurz nach Einführung der Pestkeime.

Ueber *Schaben* berichtet *Küster*, dass die (einmalige) Nahrungsaufnahme von Pestbacillen scheinbar auf sie keine Einfluss gehabt hatte. Barber[2]), welcher 61 Schaben verschiedener Arten (Periplaneta americana und Ryparobia maderae) mit einer Glascapillare eine Pestbouillonemulsion in die Coxae injizierte, erzeugte unter denselben durch diese Operation eine relativ grosse Sterblichkeit, welche jedoch meist auf Mischinfektion durch ein bewegliches kleines schabenpathogenes Stäbchen mit den biologischen Eigenschaften der Bac.-enteritidis-Gruppe zurückzuführen war. Nur in einem Falle gelang es ihm, nach dem Tode der Schaben durch den Tierversuch Pest nachzuweisen.

Dieser Autor kommt in seiner Zusammenfassung zum Schlusse, dass die Schaben sich durch Injektion grosser Dosen hochvirulenter Pestbacillen in die Körperkavität infizieren lassen, dass jedoch auf Grund der zahlreichen negativen Impfversuche anzunehmen ist, dass diese Insekten, speziell Ryparobia maderae gegenüber einer Pestinokulation in die Körperhöhle wenig empfänglich sind. Meine Versuche, die sich auf Küchenschaben, Fliegen und Heuschrecken erstrecken, hatten hauptsächlich die Aufgabe, nachzuweisen, ob die Pestinfektion die Tiere schädigt, und ob die Sterblichkeit unter diesen Insekten durch Aufnahme von Pestmaterial in den Organismus erhöht wird.

Diese Frage musste vor allem durch möglichst gleiche Versuchsanordnung und Versuchsbedingungen zwischen den Tieren des Experimentes und den Kontrolltieren, welche mit pestigem Material nicht in Berührung kamen, gelöst werden.

I. VERSUCHE MIT KUECHENSCHABEN (BLATTA GERMANICA).

Die Tiere wurden teils in Fernbach-Kolben, zu je 10 Stück, teils in sterilisierten weithalsigen Flaschen einzeln gehalten. Der Boden der Gefasse wurde mit Papier ausgelegt und in der Mitte, direkt unter der Oeffnung, ein grösseres Watte resp. Gazestück gelegt, auf welches nach Einbringung der Schaben das pesthaltige Material gelegt bzw. getropft wurde. Das Auslegen des Glasbodens mit Papier und Watte oder Gaze ist unerlässlich, einerseits, um die Feuchtigkeit aufzusaugen, andererseits um den Tieren emen Unterschlupf zu geben. Die Gefässe mit den Versuchstieren wurden ausnahmslos im Thermostaten bei 24—29° gehalten; das Optimum der Lebensäusserungen der Kuchenschaben und der Entwicklung des Pestbacillus fällt bei dieser Temperatur zusammen.

1. Versuch: Einmalige Tränkung mit virulenter Pestbouillon.

Es wurden in je 2 Flaschen (für Spezialnährboden nach *Omeljansky*) je 10 Schaben gesetzt. Die ein Serie wurde mit in Wasser getränkten, die andere mit in Pestbouillon getränkten Zuckerstückchen gefüttert.

Pest				Kontrolle				
1. Flasche		2. Flasche		1. Flasche		2. Flasche		
tot	lebend	tot	lebend	tot	lebend	tot	lebend	
0	10	0	10	0	10	0	10	23. VI.
1	9	2	8	0	10	1	9	25. VI., nach 2 Tagen
2	8	2	8	2	8	3	7	30. VI., nach 7 Tagen
2	8	2	8	2	8	3	7	1. VIII., nach 8 Tagen

Ergebnis: Kein Unterschied in der Sterblichkeit zwischen Kontroll- und Pesttieren.

2. Versuch: Füttern von Küchenschaben mit frischem, hochvirulenten Leichenmaterial.

Frisch gefangene Schaben werden zu je 10 Stück in zwei Fernbach-Kolben gesetzt. In einen derselben werden frisch aus einer Pestmeerschweinchenleiche entnommene ausserordentlich bakterienreiche Organe (Herz mit Blutgerinnsel, ein Stück Milz, ein Bubo) geworfen. Alle Schaben sogen allsogleich am Pestblut. Di Kontrolltiere wurden mit den gleichartigen, einem frisch getöteten, wutkranken Kaninchen entnommenen Organen gefüttert und unter völlig gleichen Bedingungen gehalten.

VERSUCHSVERLAUF.

Datum	Pestkolben		Kontrollkolben		Bemerkungen
	tot	lebend	tot	lebend	
10. X. ...	0	10	0	10	Ein Tier im Pestkolben und ein Kontrolltier scheint im Organsaft ertrunken zu sein.
12. X. ...	2	8	0	10	
13. X. ...	3	7	1	9	
15. X. ...	4	6	1	9	Tränkung der Tiere durch Befeuchtung der Organe mit H^2O.
17. X. ...	4	6	1	9	Neuerliche Fütterung mit frischen Pestorganen.
20. X. ...	4	6	2	8	
24. X. ...	4	6	4	6	Ein Kontrollier möglicherweise im Organsaft ertrunken.
30. X. ...	4	6	6	4	

In dem Kolben mit den Pestorganen laufen zahlreiche junge, aus den Eiern geschlüpfte Tiere umher. Liquidierung des Versuches infolge zunehmender Fäulnis des Materials.

Derartige Versuche wurden mehrmals wiederholt, wobei die Schaben des öfteren mit Organstücken gefallener Pestmeerschweinchen gefüttert wurden. Einer dieser Versuche dehnte sich über 5 Wochen aus. Die 10 Tiere des Pestkolbens erhielten während dieser Zeit ausschliesslich Pestorgane zur Nahrung, welche bisweilen mit destilliertem Wasser befeuchtet wurden. Die Tiere vermehrten sich während des Versuches in den Kolben; so konnten nach Abschluss des Versuches, nach 5 Wochen, im Pestkolben über 30 Tiere gezählt werden.

Diese Versuche beweisen, dass bei hoher Temperatur gehaltene und daher stets einem lebhaften Stoffwechsel unterworfene Küchenschaben sich wochenlang ausschliesslich von pesthaltigen Leichenteilen ernähren konnten, wobei die Sterblichkeit nicht oder nur unbedeutend höher war (in einigen Versuchen sogar niedriger) als bei den unter gleichen Bedingungen gehaltenen Kontrolltieren.

3. Versuch: Wiederholte Tränkungen von Küchenschaben mit Pest-, Cholera- und Typhusbouillonkulturen.

Datum	Pest		Typhus		Cholera	
	lebend	tot	lebend	tot	lebend	tot
12. XII.	10	0	10	0	10	0
13. XII.	10	0	8	2	9	1
14. XII.	10	0	6	4	8	2
15. XII.	9	1	5	5	6	4
20. XII.	8	2	5	5	6	4
24. XII.	7	3	5	5	6	4
31. XII.	7	3	4	6	6	4
2. I.	7	3	4	6	5	5
8. I.	6	4	4	6	4	6
15. I.	5	5	4	6	3	7

Die Tiere wurden einzeln in sterilen Flaschen gehalten und 2 Tage hungern gelassen; hierauf erhielten sie ein *steriles*, vollkommen trockenes Brotstück, welches *täglich* mit 24—72 stündiger Bouillonkultur von den entsprechenden Bakterien betropft wurde. Die Mehrzahl der Tiere trank meist bald nach dem Zugiessen der Bouillon vor meinen Augen. Die Tiere wurden von Zeit zu Zeit in trockene, mit sterilem Brotstück versehene Flaschen umgesetzt.

Während des Versuchsverlaufes legte ein mit Pestbouillon dauernd gefüttertes Weibchen ein Ei ab, aus welchem Junge ausschlüpften, die während des weiteren Versuches zum grössten Teil am Leben blieben (ein Teil ertrank in der Bouillon); dasselbe Tier brachte nachher noch ein Ei zur Reife. Zwei reife Nymphen häuteten sich während des Versuches zum vollentwickelten Insekt.

Ergebnisse: 1. Die Mortalität bei den mit Pestbouillon gefütterten Tieren war in dieser Versuchsreihe geringer als die Mortalität der Typhus-resp. Choleratiere. 2. Die namentlich in den ersten Tagen beträchtliche Mortalität unter den mit Typhusbouillon gefütterten Tieren konnte in Zusammenhang gebracht werden mit der Angabe von *Barber*, dass Bacillen des Gärtner-Typus für Küchenschaben hochpathogen werden können. 3. Es konnten durch tägliche Tränkungen ausschliesslich mit hochvirulenter pesthaltiger Bouillon 50% der Schaben über einen Monat am Leben erhalten werden, wobei sich die Tiere sogar vermehrten bzw. häuteten.

4. *Versuch*. Infektion von Meerschweinchen mit dem Darminhalt von Küchenschaben, welche mit pesthaltigem Material gefüttert wurden.

Es gelang nur einmal, ein Meerschweinchen mit dem Darminhalt pestinfizierter Küchenschaben zu infizieren. Auffallend war hierbei die lange Inkubations-resp. Krankheitsdauer und die Erscheinungen einer subchronischen Pest.

7. XII. Es wurden je 10 Schaben, welche 1—3 Tage gehungert hatten, in zwei Fernbach-Kolben verteilt und ihnen zur Nahrung Pestorgane eines eben gefallenen Meerschweinchens vorgeworfen. Die Schaben machten sich sogleich alle an das Futter. Die beiden Fernbach-Kolben wurden im Thermostaten bei 28° gehalten.

10. XII. Alle 20 mit Pestmaterial gefütterten Schaben am Leben. Die 10 Tiere des einen Kolbens werden mit Chloroformdämpfen getötet. Zwei dieser Tiere werden sorgfältig gereinigt, in 5% Carbolsäure rasch abgespült und hierauf in mehreren Schalen mit steriler physiologischer Kochsalzlösung gründlich gewaschen. Nach dem Trocknen der Tiere wurde der Darm sorgfältig herauspräpariert, wobei eine Berührung desselben mit dem Chitinpanzer vermieden wurde, und in eine sterile Schale gelegt. Hierauf wurde daraus eine Emulsion hergestellt, mit welcher ein Meerschweinchen subcutan geimpft wurde. Gleichzeitig wurde ein Meerschweinchen mit der Ausgangspestkultur subcutan geimpft.

Das mit der Pestkultur geimpfte Kontrollmeerschweinchen ging nach 4 Tagen an typischer akuter Bubonenpest zugrunde.

Das mit der Emulsion von Schabendarm infizierte Meerschweinchen zeigte erst nach 6 Tagen Krankheitserscheinungen und wurde in der Frühe des 12. Tages tot aufgefunden.

Sektionsbefund: Hämorrhagien an der Injektionsstelle; grosse *im Zentrum vereiterte* Leistenbubonen. In den Lungen ausgedehnte Verdichtungsherde und Hämorrhagien. Nekrotische Herde in der Leber; Milz auf das Dreifache vergrössert mit zahlreichen Nekroseherden von der Grösse eines Zündhölzchenkopfes. Nephritis haemorrhagica.

Die Ausstrichpräparate aus dem Bubo ergaben die Anwesenheit einer mässigen Menge von Pestbacillen, worunter sich zahlreiche Involutionsformen vorfanden. Pestbacillen in Herz und Milz in Reinkultur. Pest bakteriologisch und biologisch nachgewiesen.

Diesem Versuche gingen voraus und folgten zahlreiche ähnliche Experimente mit *negativem* Ergebnis.

Infektion von Meerschweinchen mit dem Darminhalt von Küchenschaben führt oft zu unerwünschten Tierverlusten, da diese Insekten in ihrem Darminhalt meist höchst maligne anaerobe Keime enthalten, denen die überwiegende Mehrzahl der Meerschweinchen bei subcutaner Infektion unter zunehmender Kachexie auch zum Opfer fällt. Die so gefallenen Tiere erwiesen sich bei der Sektion als frei von pestigen Veränderungen.

Es ist daher anzunehmen, dass die Pestbacillen im Darm der Küchenschabe rasch vernichtet werden oder ihre Virulenz einbüssen.

Es wurde zuerst versucht, auf histologischem Wege durch Serienschnitte, die nach der Methode von Kossel gefärbt wurden, das Schicksal der Pestbacillen im Schabendarm zu verfolgen. Dies gelang jedoch nicht, da es nicht möglich ist, die Pestbacillen von den zahlreichen autochthonen Darmbewohnern der Küchenschaben mit Sicherheit auseinanderzuhalten.

Da anzunehmen war, dass möglicherweise im Darm der Küchenschabe Stoffe vorhanden sind, welche gegenüber den Pestbacillen starke baktericide Wirkung entfalten, wurden mit sterilen Darmfiltraten einige Versuche angestellt:

5. *Versuch*: Untersuchung des filtrierten Darminhaltes von mit Pest lange vorbehandelten Schaben auf Vorhandensein eines Bakteriophagen.

Eine Serie von Küchenschaben wurde im Verlaufe eines Monats 12mal mit frischen Organen von an Pest gefallenen Meerschweinchen gefüttert. Von 10 Tieren starben während der ganzen Versuchszeit nur zwei.

Nach 18 Tagen wurden 3 Tiere getötet, die Därme vorsichtig herauspräpariert, in 3 ccm Bouillon verrieben und durch einen Seitz-Filter filtriert. Hierauf wurde je ein Tropfen des Filtrats auf 6 frisch mit Pestkultur bestrichene Agarplatten auftropfen gelassen. Kontrollplatten mit Typhus-und Cholerakulturen. Ausserdem wurden mehrere Bouillonröhrchen mit 0,1, 0,5 und 1,0 ccm des Filtrats beschickt (9 ccm neutrale Bouillon). Bebrütung im Thermostaten bei 37°.

Ergebnis: Ueberall gleichmässiges Wachstum des Pestbacillus.

Nach 4 Wochen wurden weitere 3 dieser Schaben getötet, *zerrieben*, in neutraler Bouillon aufgeschwemmt und durch Seitz-Filter filtriert. Es wurde etwa 6 ccm Filtrat erhalten.

Es wurden zwei Röhrchen: a) 9 ccm neutraler Bouillon allein und b) 8 ccm neutraler Bouillon + 1 ccm Filtrat mit je einer Oese einer hochvirulenten Pestkultur geimpft und im Thermostaten bebrütet.

Ergebnis: Völlig gleichmässiges Wachstum in beiden Röhrchen.

Nach 48stündigem Verweilen der beiden Röhrchen im Thermostaten (35—36°) wurden 2 Meerschweinchen subcutan infiziert, und zwar erhielt Meerschweinchen Nr. 1 400 g. 0,1 ccm des Bouillonröhrchens + Filtrat; Meerschweinchen, Nr. 2 370 g 0,1 ccm des Bouillonröhrchens "Pest allein".

Meerschweinchen Nr. 2 ("Pest allein") starb am 6. Tage an typischer Bubonenpest, Meerschweinchen Nr. 1 (Pest + Filtrat) erst am 10. Tage mit Vereiterung der Einstichstelle, wobei hier die Pestbacillen Degenerationserscheinungen und Polymorphismus zeigten. Grosse Nekroseherde in der Milz, sonst alle Anzeichen typischer Bubonenpest.

Es hatte demnach den Anschein, als ob die Pestkultur durch ihr Beisammensein mit dem verdünnten Darmfiltrate einigermassen abgeschwächt wurde.

Eine Wiederholung des Versuches nach 12 Tagen an 4 Meerschweinchen, wobei zwei mit der filtratfreien, 2 mit der filtrathaltigen Pestbouillon geimpft wurden, ergab jedoch eindeutige negative Resultate: Alle 4 Tiere gingen ziemlich gleichzeitig nach 5—6 Tagen an typischer akuter Bubonenpest zugrunde. *Ergebnis*: Es gelang durch obige Versuche nicht, einen wirksamen Bakteriophagen aus den Schabendarmfiltraten zu gewinnen.

6. *Versuch*: Infektion von Meerschweinchen mit frisch abgelegten Exkrementen pestinfizierter Schaben.

Es wurden 10 Küchenschaben 2 Tage lang hungern gelassen. Hierauf wurden sie einzeln in sterile Eprouvetten gesetzt und mit je einem kleinen Stück einer frischen Pestmilz vom Meerschweinchen (ungeheure Bakterienmassen) gefüttert. Nach 12 Stunden wurden die Tiere in frische Eprouvetten umgesetzt, nach weiteren 12 Stunden*) wurden die Exkremente gesammelt (10 Stück), mit steriler Kochsalzlösung verrieben und diese Emulsion 2 Meerschweinchen in die rasierte Bauchhaut eingerieben

Die Küchenschaben erhielten gezuckerte feuchte Brotschnitten zum Futter, wurden nach weiteren 12 Stunden abermals umgesetzt und die 36—47 Stunden nach Fütterung mit pesthaltigem Material abgelegten Exkremente (12 an der Zahl) in einer Emulsion zwei weiteren Meerschweinchen in die rasierte Bauchhaut eingerieben.

Wiederholung des Experimentes mit den 67—71 Stunden nach der Fütterung mit pesthaltigem Material abgelegten Exkrementen.

Ergebnis: Alle 6 Meerschweinchen blieben völlig gesund. Keines der Tiere zeigte Drüsenschwellungen. Nach 14 Tagen wurden die Tiere getötet: Normale Milzen, nirgends Veränderungen, welche auf eine Pestinfektion hindeuten könnten, nachgewiesen.

Dieser Versuch steht im Einklang mit den schon von *Küster* (*Knauff*) betonten Angaben, dass es nur äusserst selten gelingt, und dann nur bei peritonealer Impfung und bei Anwendung eben abgelegter Exkremente, Meerschweinchen erfolgreich mit Pest zu infizieren.

7. Zahlreiche *Versuche mit "chirurgischen" Infektionsmethoden* (Amputation des Femur III im oberen Drittel und Plombieren der Wunde mit der Pestkulturmasse eines Agarröhrchens) ergaben stets negative Resultate: Niemals konnte in den nach der Operation gelegentlich gefallenen Insekten durch den Tierversuch die Anwesenheit virulenter Pestkeime nachgewiesen werden. Die Sterblichkeit unter diesen Tieren war eine höhere als unter den gefütterten Tieren, doch ist dies nicht auf die Pestinfektion zurückzuführen, da auch unter den entsprechenden Kontrolltieren, denen ohne nachfolgende Pestinfektion der Femur auf die gleiche Weise amputiert wurde, die Mortalität ungefähr gleich hoch war.

II. VERSUCHE MIT FLIEGEN.

Am 13. IX. wurden je 10 Fleischfliegen (Sarkophaginen) in zwei Fernbach-Kolben gesetzt. In den ersten Kolben wurde die bacillenreiche Leber eines an demselben Tage an der Pest verendeten Meerschweinchens, in den zweiten Kolben normale parenchymatöse Organstücke eines Kaninchens geworfen. Die Kolben werden bei Zimmertemperatur gehalten (zu Anfang des Versuches 13—15°). Die Organstücke wurden von Zeit zu Zeit mit einigen Tropfen Wasser befeuchtet bzw. durch frische ersetzt. Alle Tiere wurden beim Saugen des Pestsaftes beobachtet.

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* Auf Grund der Untersuchungen von Küster[9] vollzieht sich die Fortbewegung des Darminhaltes bei den Küchenschaben sehr langsam. Es war daher ein Auftreten von Pestbacillen in den Entleerungen vor Ablauf der ersten 24 Stunden kaum anzunehmen.

VERSUCHSVERLAUF.

Datum	Kolben mit der Pestleber		Kontrollkolben		Bemerkungen
	lebend	tot	lebend	tot	
13. IX. ...	10	0	10	0	
14. IX. ...	10	0	10	0	
22. IX. ...	10	0	10	0	
24. IX. ...	8	2	9	1	Kaltes, frostiges Wetter; Sinken der Zimmertemperatur auf 10°.
27. IX. ...	5	5	7	3	Nachtfröste; starkes Sinken der Temperatur des Versuchsraumes.
2. X. ...	1	9	3	7	Liquidierung des Versuches.

Derartige Versuche wurden mehrmals sowohl mit Sarkophaginen als auch mit Stubenfliegen (Musca domestica) wiederholt und ergaben mit obigen Versuchen übereinstimmende Resultate.

Ergebnis: Die mit Pestmaterial genährten und bei Zimmertemperatur gehaltenen Fliegen konnten alle über eine Woche lang am Leben erhalten werden. In der Folge war die Sterblichkeit derselben gegenüber den Kontrolltieren nicht auffallend erhöht.

III. VERSUCHE MIT HEUSCHRECKEN.

Es gelangten über 100 grosse, auf den Grassteppen in der Umgebung der Tschitaer Bakteriologischen Station gesammelte Heuschrecken zur Untersuchung. Sie wurden in grossen, einen Liter fassenden Glaszylindern, wie sie zur Blutentnahme bei Pferden üblich sind, gehalten. Als Futter erhielten sie hauptsächlich Disteln, an denen sie im Freien häufig angetroffen worden waren, und deren Blütenblätter sie mit Vorliebe frassen. Die Tiere wurden ausnahmslos bei Zimmertemperatur gehalten.

1. Es wurden je fünf eben gefangene grosse Heuschrecken in Glaszylinder gesetzt.

Die *I. Gruppe* wurde folgendermassen mit Pest infiziert: Amputation eines Femurs des dritten Beinpaares im obern Drittel, Plombieren einer Oese einer hochvirulenten Pestagarkultur in die Höhle zwischen Chitinpanzer und retrahierter Muskelmasse.

Der *II. Gruppe* wurden die Blumen mit Pestbouillon übergossen.

Die *III. Gruppe* wurde mit Typhus abdominalis analog der ersten Gruppe infiziert.

Die IV. Gruppe wurde unbehandelt gelassen und diente als Kontrolle.

VERSUCHSVERLAUF.

Datum	I. Gruppe Chirurg. Pest		II. Gruppe Fütterungspest		III. Gruppe Chir. Typh. Abd.		IV. Gruppe Kontrolle	
	lebend	tot	lebend	tot	lebend	tot	lebend	tot
27. VIII.	5	0	5	0	5	0	5	0
29. VIII.	5	0	5	0	5	0	5	0
30. VIII.	5	0	4	1	5	0	5	0
31. VIII.	2	3	1	4	5	0	5	0
3. IX.	1	4	1	4	4	1	5	0
2. Wiederholung des Versuches unter genau denselben Versuchsbedingungen :								
3. IX.	5	0	5	0	5	0	5	0
4. IX.	5	0	5	0	5	0	5	0
5. IX.	5	0	5	0	5	0	5	0
6. IX.	5	0	5	0	5	5	5	0

3. Es wurde eine 3. Serie mit am Tage vorher gefangenen Heuschrecken angesetzt : Die *erste* Gruppe wurde infiziert durch intensives Einschmieren einer Pestkultur (Agarrasen) in die Mundwerkzeuge; die *zweite* Gruppe auf gleiche Weise durch Einschmieren einer Typhus-abdominalis-Kultur. Die *dritte* Gruppe durch Plombieren des Agarpestrasens in die Wunde der Coxa nach totaler Amputation eines Femurs des hinteren Beinpaares. Die *vierte* Gruppe wurde in gleicher Weise behandelt wie Gruppe 3, nur folgte nach der Amputation keine Infektion. Sie diente als Kontrolle.

VERSUCHSVERLAUF.

Datum	I. Gruppe Einschmieren von *Pest* in den Mund		II. Gruppe Einschmieren von Ty. abd. in den Mund		III. Gruppe Plombieren von Pest mat. in d. Coxastumpf		IV. Gruppe Amputation ohne Infektion	
	lebend	tot	lebend	tot	lebend	tot	lebend	tot
7. IX.	5	0	5	0	5	0	5	0
11. IX.	5	0	4	1	4	1	5	0
13. IX.	4	1	4	1	3	2	4	1
17. IX.	4	1	2	3	1	4	0	5

Die Leichen der auf chirurgischem Wege mit Pest infizierten Heuschrecken wurden einige Sekunden mit warmer 5 proz. Carbolsäurelösung abgespült, hierauf wiederholte Male längere Zeit mit steriler physiologischer Kochsalzlösung gewaschen und gespült. Dann wurde in der sterilen Lösung das Abdomen vom Thorax abgetrennt und in physiologischer Kochsalzlösung verrieben. Mit der so gewonnenen Emulsion wurden zwei Meerschweinchen, eins subcutan, das andere cutan, geimpft : Die Meerschweinchen blieben gesund; Beobachtungsdauer über einen Monat.

Diese Versuche mit Heuschrecken ergaben bisweilen eine grössere Sterblichkeit unter den pestinfizierten Tieren (Versuch 1). Es gelang jedoch, weder bakteriologisch noch durch den Tierversuch bei den gefallenen Tieren Pest nachzuweisen. Es erscheint daher unwahrscheinlich, dass die Tiere der Pest zum Opfer fielen. Die Heuschrecken vertrugen die Gefangenschaft überhaupt schlecht und begannen nach einer

Woche unterschiedslos einzugehen, was zum Teil auch auf die vorgeschrittene Jahreszeit und die hereinbrechende Kälte zurückgeführt werden kann.

ZUSAMMENFASSUNG.

1. Küchenschaben konnten bei ausschliesslicher Fütterung mit pesthaltigem Material über einen Monat lang am Leben erhalten bleiben. Aus den während des Versuchsverlaufes zur Reife gelangten Eiern entwickelten sich lebenskräftige Küchenschaben.

2. Durch Tierversuche konnte festgestellt werden, dass die Pestbacillen offenbar im Darmtrakt der Küchenschabe meist rasch zugrunde gehen bzw. völlig ihre Virulenz verlieren. Versuche, einen wirksamen Bakteriophagen reinzuzüchten, schlugen fehl.

3. Es gelang niemals, mit frisch abgelegten Exkrementen von mit Pestmaterial gefütterten Küchenschaben Meerschweinchen durch *cutane* Impfung mit Pest zu infizieren.

4. Durch Verimpfung von Pestmaterial in den Coxastumpf eines amputierten Hinterbeines konnte bei Küchenschaben und Heuschrecken keine tödliche Pesterkrankung hervorgerufen werden.

5. Es gelang nicht, Heuschrecken per os mit Pest zu infizieren.

6. Die von mehreren Autoren vertretene Ansicht, dass Fliegen infolge pestinfizierter Nahrung rasch zugrunde gehen, konnte bei Versuchen mit Stuben-bzw. Fleischfliegen nicht bestätigt werden.

LITERATURVERZEICHNIS.

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PLAGUE EXPERIMENTS UPON INSECTS.

(SECOND COMMUNICATION).

SUMMARY IN ENGLISH.

Note :—The first communication has already appeared in Vol. IV of the Manchurian Plague Reports, pp. 231-4.

1. Cockroaches (Blatta germanica) have been observed to live for more than one month after feeding upon material containing *B. pestis*. Their eggs produce healthy offspring.
2. Experiments upon guinea-pigs show that plague bacilli perish or soon lose their virulence in the alimentary tract of cockroaches.

   Attempts to produce an active bacteriophage from the alimentary tract of cockroaches fail to produce positive results.
3. It has not been possible to infect guinea-pigs with fresh excrements from cockroaches fed on plague material.
4. Inoculations of *B. pestis* into the coxa-stumps of an amputated leg did not produce plague in either cockroaches or locusts.
5. Locusts could not be infected by feeding.
6. The opinion of some authors that flies succumb after feeding upon plague infected food has not been confirmed by my experiments.

H. M. JETTMAR, M.D.,

Serologist, P. P. S.

DIE BAUE EINIGER TRANSBAIKALISCHER SAEUGETIERE IN SCHEMATISCHER DARSTELLUNG.

(Erweiterter Abdruck aus der Zeitschrift fuer Saeugetierkunde I. Band, I. Heft.)

MIT 12 ABBILDUNGEN.

In der mir zur Verfuegung stehenden Literatur ist wenig ueber transbaikalische Nagetierbauten zu finden. So fehlt z.B. in der neuesten Auflage des "Brehm," in welcher den transbaikalischen Nagetieren besondere Aufmerksamkeit zugewendet wird, eine eingehende Baubeschreibung. Daher entwarf ich, als ich im Jahre 1920 zwecks Peststudien die Baue einiger transbaikalischer Nager ausgrub, gleich an Ort und Stelle die schematischen Zeichnungen, nach denen die hier beigefuegten Figuren angefertigt wurden. Im Fruehling und Sommer 1923 hatte ich abermals Gelegenheit, 2 Tarbaganhoehlen, ungefaehr dreissig Bauten vom Eversmannschen Ziesel und eine betraechtliche Anzahl von Bauen kleiner Steppennager auszugraben. Die Studien wurden im Jahre 1927 in der Mongolei in der Umgebung von Urga fortgesetzt, wo zahlreiche Nagetierbaue, unter anderem zwei Tarbaganhoehlen und mehrere Baue vom Erdspringhasen ausgegraben wurden. Die Bewohner aller Hoehlen, mit Ausnahme je eines Tarbagan-und eines Iltisbaues, wurden gefongen und durch Vergleich mit dem Material des Tschitaer Museums oder durch Vermittlung des Herrn Dr. Jordan (London) identifiziert.

Die Nager der transbaikalischen Steppen gelten, wie bekannt, als Pestüberträger und sind daher nicht bloss für den Zoologen, sondern auch für den Mediziner von Interess.e Alle Nagetierbauten waren von einer grossen Menge verschiedener Käfer, Wanzen, Fliegen, Tausendfüssler, Spinnen, Insektenlarven bewohnt, welche sich z. B. von den Höhlengängen aus eigene Blindgänge graben. Diese Insekten können in der Höhle eines pestkranken Tieres als Verbreiter der Pest in Betracht kommen. Fand ich doch in der nächsten Nähe der Schlafkammer in einem Tarbaganbau einen Käfer, an dessen Brustschild drei Zeckennymphen hafteten. Auch die Ektoparasiten des Nagers, wie Flöhe und Zecken, sind recht häufig in seinem Bau anzutreffen, besonders die Schlafkammer wimmelt oft von ihnen. Flöhe und Zeckennymphen kann man auch sehr zahlreich häufig im Ausgang der Höhle antreffen, wenn man ein paar Hände voll Erde aus diesen herausholt und absucht.

Zu den Abbildungen sei im allgemeinen bemerkt, dass die Massangaben in cm gemacht sind. Eine Zahl neben einem kleinen Kreise gibt die Tiefe der betreffenden Stelle unter der Erdoberfläche an. Das Zeichen → gibt die Richtung an, in der der betreffende Gang sich senkt. Das Zeichen →← gibt ebenen Verlauf des Ganges an. Buchstabenerklärung siehe im Text.

I. TARBAGAN (*Arctomys bobac* PALL).

Der Tarbaganbau hat zahlreiche Darsteller gefunden, ich erinnere bloss an die Arbeiten von RADDE, TSCHAUSSOW, ULRICH, DUDTSCHENKO und WU LIEN-TEH. In letzter Zeit hat auch SUKNEFF den Tarbaganbau eingehend studiert und von ihm mehrere Schemata veroeffentlicht. Ich habe mich daher hier auf einige ergaenzende Details beschraenkt, wobei ich besonderen Wert auf genaue Ausmessung der Baue legte.

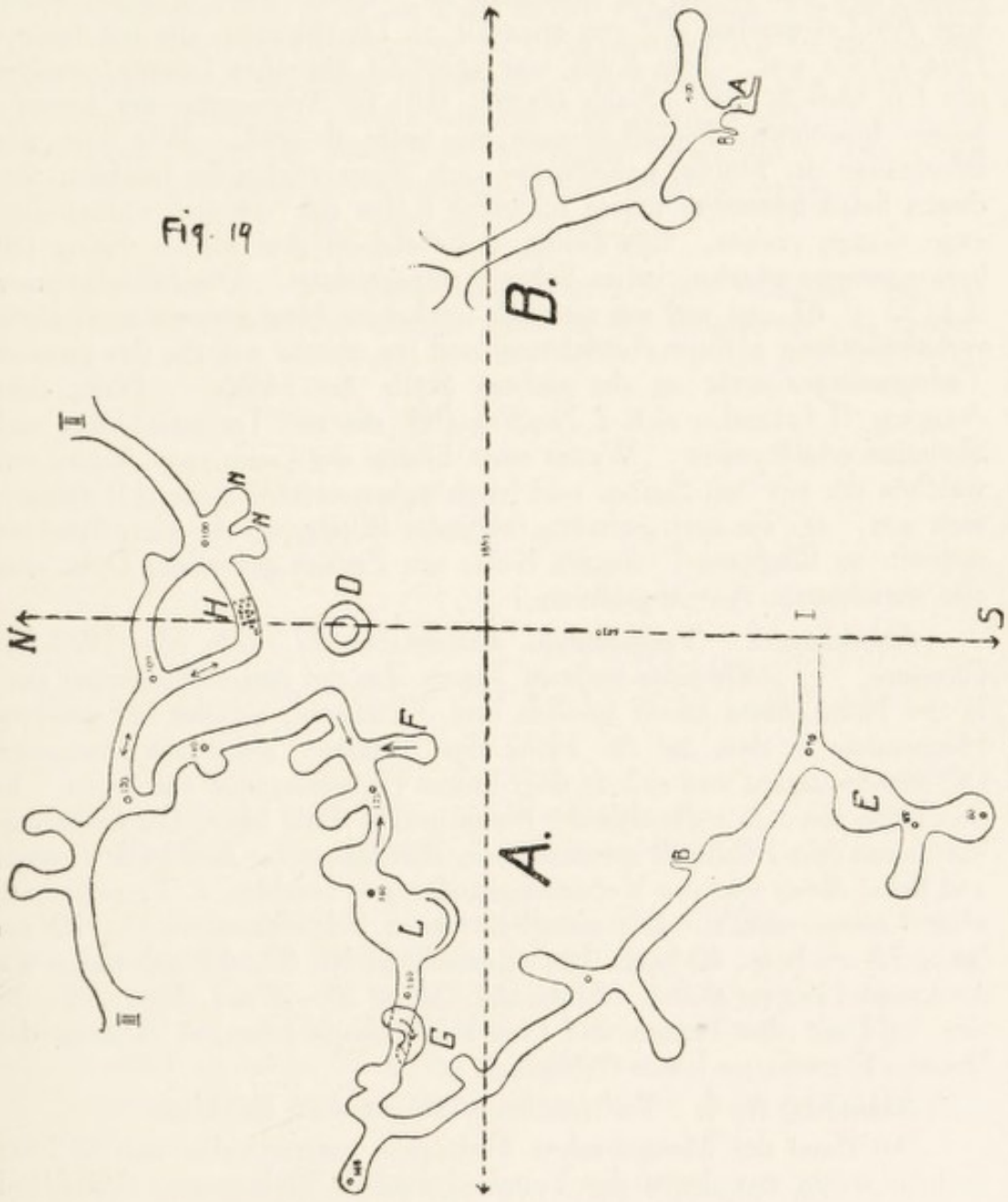


Abbildung 1. Bau von *Arctomys bobac* PALL.

Abbildung 1. Bau des Tarbagans, ausgegraben bei Charanor 27. 5. 1923. Die Höhle war von einem Tiere bewohnt, das während des Ausgrabens durch einen der mit Steinen verbarrikadierten Ausgänge entkam.

Von aussen stellte der Bau einen flachen Hügel (sg. Butan) von 18,9 m Längen-, 14,7 m Breitendurchmesser dar. Seine Höhe war ungefähr 1 m. Der Hauptbau (A) hatte 3 Ausgangsöflnungen. In der Nähe des Hauptbaues, noch in demselben Butangebiete befand sich ein grösserer, allem Anscheine nach verlassener Blindgang (B), der nicht besonders tief nach abwärts führte (1 m). In die Erweiterung nare an seinem Ende mündete der in den Abhang des Hügels gegrabene Bau (A) eines Erdhasen (die kleinen transbaikalischen Steppennager graben sehr gerne ihre Gänge in den Tarbaganbau ein). Vom Ausgange I des Hauptbaues zweigt in einer Tiefe von 70 cm ein Blindgang ab. Dieser hatte in seiner Mitte eine Art Erweiterung (E) von etwa 70 cm Durchmesser, die mit faulem Heu gefüllt war. Sein Ende war angefüllt mit alter Losung, welche mit Eis bedeckt war. Nach RADDE fällt lie Temperatur der benachbarten bewohnten Schlafkammern nie unter 0 grad. Wie fast alle Blindgänge der Höhle, beherbergte auch dieser zahlreiche Insekten; von diesen fielen besonders grosse schwarze Käfer auf, die sich selbständige enge Gänge graben. Ein Gang, aus welchem drei solcher Käfer (B) herausgezogen wurden, ist im Schema eingezeichnet. Die Schlafkammer (L) (52 × 87 cm) war mit ziemlich trockenem Heu ausgepolstert, hatte verhältnismässig geringe Ausdehnung und lag ebenso wie die des zweiten Tarbaganbaues nicht an der tiefsten Stelle der Höhle. Nahe dem Ausgang II befanden sich 2 Nischen (N), die mit Tarbaganlosung und Skeletten erfüllt waren. Weiter innen bildete der Gang zwei Arme, von welchen der eine mit Steinen und frisch aufgeworfener Erde (H) verrammelt war. (G ein nach aufwärts führender Blindgang, der viele Insekten enthielt; im Bindgang F wurden Käfer mit Zecken gefunden; D ist eine alte verschüttete Ausgangsöflnung.)

Abbildung 2. Tarbaganbau, ausgegraben 27.—28. Mai 1923 bei Charanor. Er stellt einen anderen Typus, den mit einem Ausgange, dar. In der Nähe dieses Baues spielten drei Tarbagane, welche bei unserem Herannahen in dem auf der Höhe des "Butans" gelegenen Ausgange (X) verschwanden und sich in dem linken Haupteingange verbargen. In der Tiefe von 1 m teilte sich der Hauptgang. Bald hinter der Gabelung führte von seiner linken Fortsetzung ein Blindgang (G) steil nach abwärts und links; dieser war von Verwesungsstoffen und feuchter, z. T. gefrorener alter Losung erfüllt. Die ziemlich weiten Schlafkammern (LI 70 cm breit, 78 cm lang, 40 hoch; LII entsprechend 70, 82, 40 cm) waren mit trockenem Heu gepolstert. Höhe der Gänge 20—25 cm, Breite 25—30 cm.) (N mit alter Losung und Heu angefüllte Nischen, A Abhang des Butans, F trockener leerer Sackgang).

Abbildung 3, 4. Tarbaganbaue, ausgegraben bei Urga.

Die Baue der Mongolischen Tarbagane unterscheiden sich in ihrer Anlage wenig von denen der Transbaikalischen Tarbagane. Auffallend ist etwa nur, dass der ueber dem Bau aufgeworfene Erdhuegel-der Butan-meist nicht so deutlich ausgesprochen ist, wie beim Bau der Transbaikalischen Tarbagane. Die Baue,—die meisten derselben haben nur einen Ausgang,—sind in ihrer Anlage etwas groesser und mahr verzweigt, als die entsprechenden Daurischen Tarbaganbautypen mit einem Ausgange.

Der Tarbaganbau Abbildung 3 wurde von drei Tieren bewohnt, von welchen jedes am Ende eines der Blindgaenge erbeutet wurde. Die Tiere hatten, obgleich ihnen hierzu ueber 20 Stunden zur Verfuegung standen, keinerlei Versuche gemacht, weiter zu graben, oder die Gaenge zu verschliessen. Die beiden links gelegenen Blindgaenge endigten in einem Erdreich, das mit fest aneinander gepressten grossen spitzen Steinen erfuellt war.

Tarbaganbau No. 4 ist zwar etwas kuerzer und weniger verzweigt als Tarbaganbau No. 3, er imponiert jedoch durch seine ungewoehnliche Tiefe. So liegt die Schlafkammer 2m 90cm unterhalb der Erdoberflaeche. Zum Ausgraben dieses Baues benoetigten sechs Maenner ueber zwei Tage. Die beiden Tarbagane, welche den Bau bewohnten, wurden endlich aus dem linken, langen 2m 90cm tiefen Blindgang hervorgeholt. Auch hier hatten die Tiere keinerlei Versuche gemacht, weiter zu graben.

II. ZIESEL (*Citellus*).

Abbildung 5. Bau des daurischen Ziesels *Citellus dauricus* BRANDT, ausgegraben im Sommer 1920 in der Umgebung von Soktui.

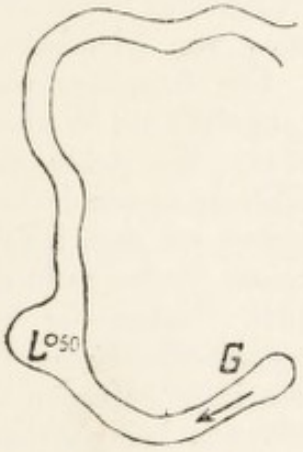


Abbildung 5. Bau von *Citellus dauricus* BRANDT. Massstab 1 :25.

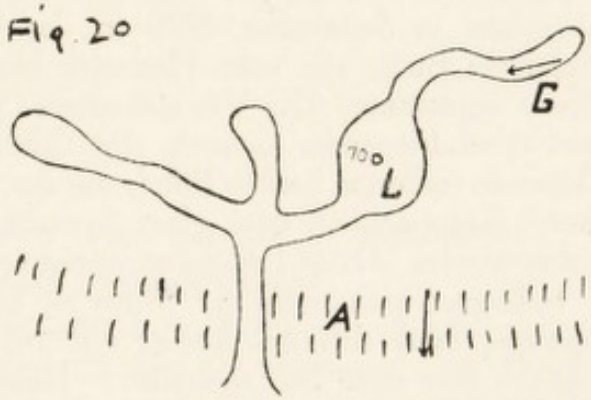


Abbildung 6. Bau von *Citellus eversmanni* BRANDT. Massstab 1 :25.

Er war von einem einzelnen Tier bewohnt. Fast alle Bauten des daurischen Ziesels sind nach dem gleichen Schema angelegt : nur ein Ausgang, ein verzweigter Gang, der in der Nähe seiner tiefsten Stelle seine Ausbuchtung (L) hat. Diese ist mit Heu, Fetzen und Haaren ausgepolstert. Von ihr führt ein kurzer Blindgang (G) nach aufwärts. Beim Ausgraben des Ganges verstopft das Tier häufig denselben mit einem festen Pfropfen aus Erde.

Abbildung 6. Bau des Eversmannschen Ziesels, *Citellus eversmanni* BRANDT, ausgegraben bei Rasmachnino im Nertschinsker Kreis, August 1923. Er war von einem einzelnen Tiere bewohnt. Der Bau ist geräumiger and tiefer als der des daurischen Ziesels, verzweigt sich auch häufiger, aber im wesentlichen ist er doch nach dem gleichen Plane angelegt (A Abhang, L Lager, G aufwärtsführender Blindgang).

III. BURUNDUK (*Eutamias asiaticus* GMEL).

Abbildung 7. Bau von
Eutamias asiaticus GMEL.
Massstab 1 :25.

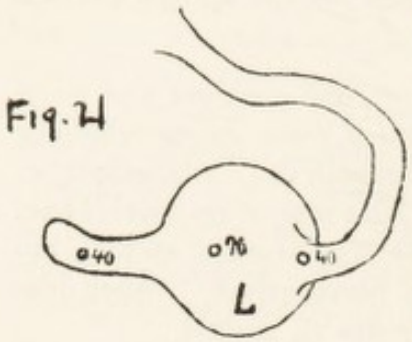


Abbildung 7. Bau des sibirischen gestreiften Erdhörnchens, *Eutamias asiaticus* GMEL., ausgegraben im Birkenwalde nahe der Station Burjatskaja 23. VII. 20. Die Höhle war von einem einzelnen Tier bewohnt. Die Gänge waren sorgsam mit Birkenblättern ausgelegt. Der Schlaf- und Vorratsraum L stellt eine geräumige, runde Höhle dar, welche zur Zeit des Ausgrabens zur Hälfte mit Beeren und Samen von Waldpflanzen angefüllt war. Auf der dem Eingang entgegengesetzten Seite fand sich ein kurzer Blindgang, der nach aufwärts führte.

IV. ZWERGHAMSTER (*Cricetulus furunculosus* PALL.)

Abbildung 8. Bau des daurischen Hamsters (*Cricetulus furunculosus*) ausgegraben in September 1920 in der Umgebung von Mandschuria. Mittelgrosse Burg, von zehn Hamstern bewohnt. Die Ausgänge waren im Kreis angeordnet. Die Vorratskammer (V) war angefüllt mit Wermuth, dessen durchdringender Geruch alle Gänge erfüllte. Sie stellte einen vollkommen isolierten breiten Blindgang dar, dessen Decke in dem lockeren Erdreich hauptsächlich dank eines Systems von Säulen vor dem Einsturz bewahrt wurde. Diese Erdsäulen waren an den beiden Enden recht dick und verjüngten sich nach der Mitte zu beträchtlich. Neben der Vorratskammer fand sich ein leerer, erst frisch ausgegrabener Raum (E), vermutlich eine neue Schlafkammer. Rings um die Burg befanden sich einige Blindgänge (G), welche offenbar zum Verbergen bei plötzlicher Gefahr bestimmt waren. L = Lager.

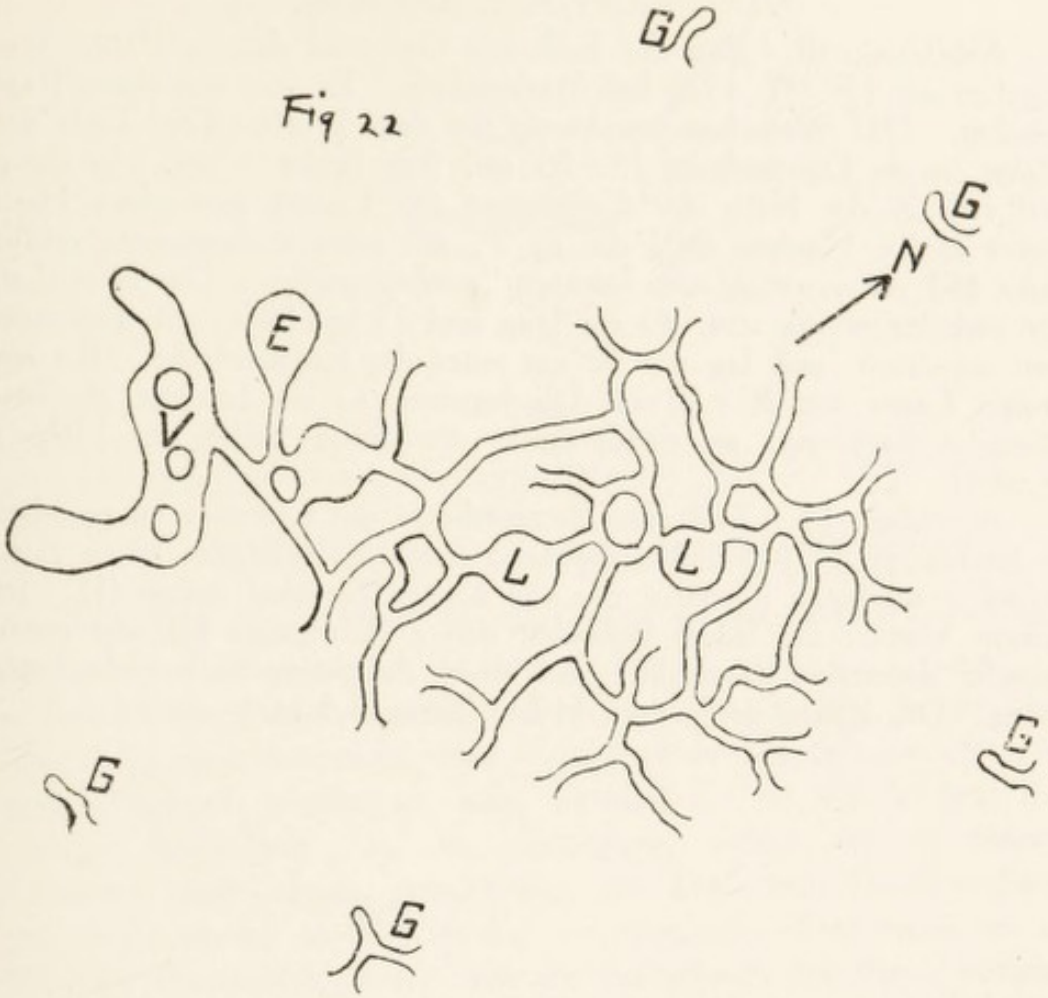


Abbildung 8 Bau von *Cricetulus furunculosus* PALL. Masstab 1 :25.

V. FELDMAEUSE (*Microtus*).

Abbildung 9. Bau von *Microtus brandti* RADDE, ausgegraben in der Nähe des Sees Tschinda-Nor, 25 km von Charanor, am 31. V. 1923. Er war von einem Muttertier und 6 Jungen bewohnt. Das trockene Nest (L) lag 20 cm unter der Erodberfläche, ganz im Innern des Baues. Am Ende desselben ein weiter leerer Raum (E) (zukünftige Vorratskammer ?)

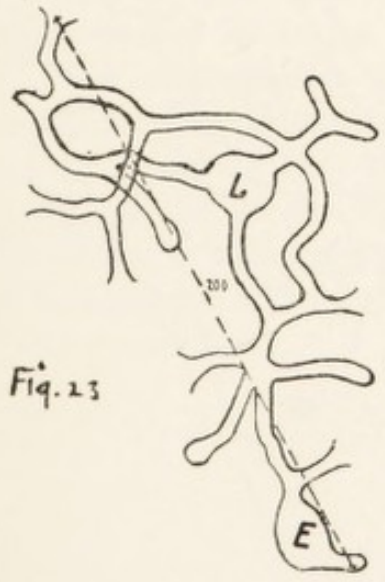


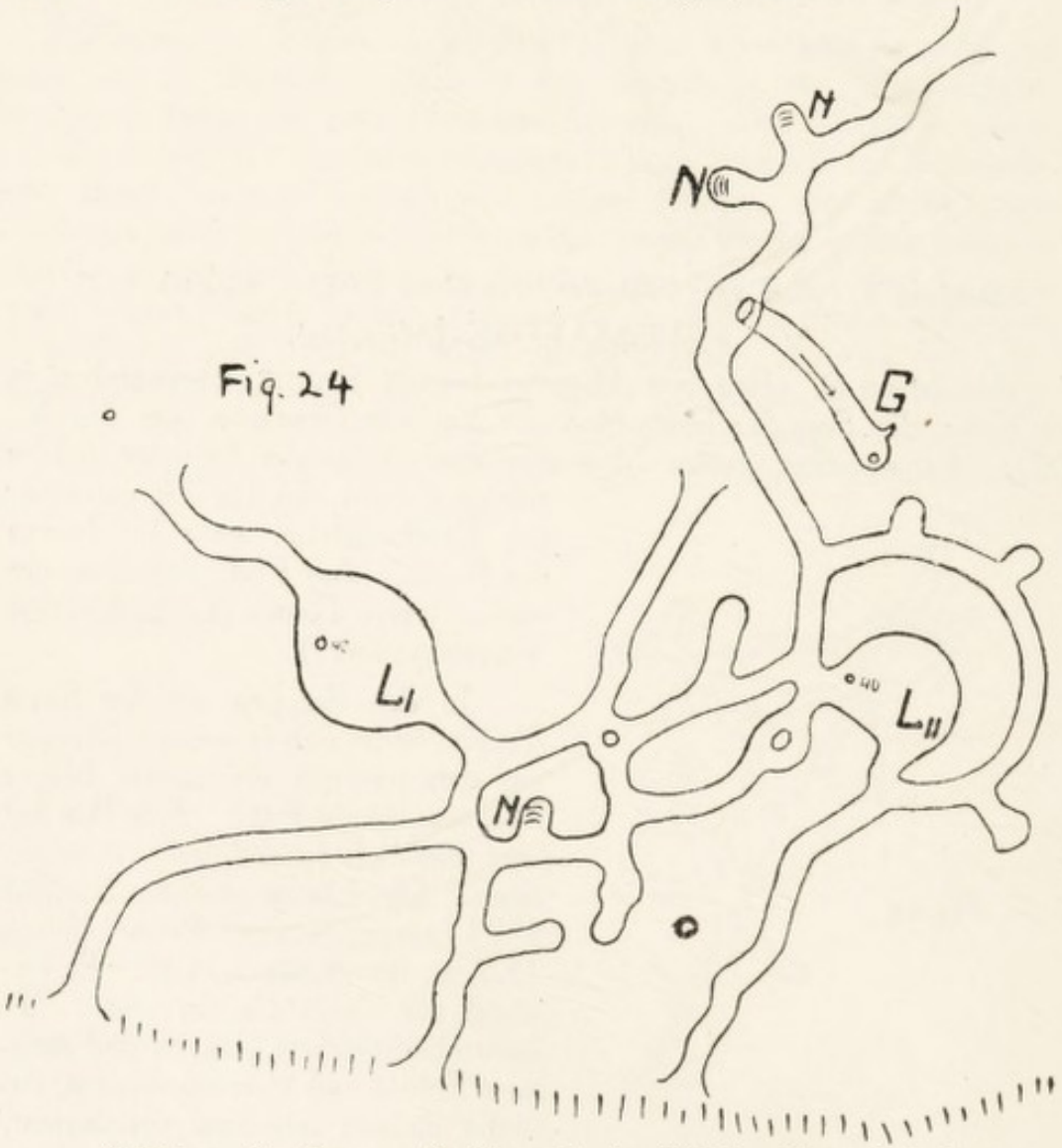
Abbildung 9. Bau von *Microtus brandti* RADDE. Massstab 1 :25.

In den Steppen um die Stadt Tschita findet sich in grossen Mengen ein dem vorigen verwandter Nager *Microtus arvalis* PALL. Sein Bau hat eine ziemlich beträchtliche Ausdehnung. Die Länge eines im Sommer 1923 ausgegrabenen Baues betrug 13,7 m, die Breite 3,75 m. Er bestand aus einem Gewirr von z. T. halb oberirdischen Gängen und mehreren Schlaf-und Vorratskammern, darunter einigen scheinbar verlassenen.

VI. PFEIFHASE (*Ochotona*).

Abbildung 10. Bau des Erdhasen *Ochotona* daurica PALL. ausgegraben am 15. VI. 1920 bei Maziewskaja. Er war von einem Paar bewohnt. Das Weibchen war knapp vor dem Wurfe. Die Tiefe der Gänge ist im Durchschnitt 15 – 20 cm, ihre Breite 5 cm. In ihnen fand sich in der Nähe der Oeffnungen mit Losung gemischtes Heu. Ferner kleine Nischen (N), die z. T. mit alten Exkrementen erfüllt waren und von verschiedenen Insekten bewohnt wurden. Das Nest (LII) war verhältnismässig rein, 30 cm lang und 25 cm breit, mit trockenem Heu gepolstert, und lag nur 40 cm unter der Erdoberfläche. (LI ein zweites Lager von 30 × 40 cm Durchmesser, G ein feuchter abwärtsführender Sackgang; an einem Ende die tiefste Stelle der Höhle: 65 cm.)

Abbildung 11. Teil eines Sommerbaues des Erdhasen ausgegraben im September 1920 bei Mandschuria. In der Nähe des einen Ausganges 2 rundliche Nischen, die von Kröten bewohnt waren (1). Im weitern Verlauf des Baues befanden sich 2 Blindgänge (2), die genau einander gegenüber angeordnet und allem Anscheine nach nicht fertig waren. Die Länge der ganzen Höhle betrug 3,5 m.



Abbidung 10. Bau von *Ochotona daurica* PALL. Massstab 1 :25.

Abbildung 11.

Teil eines Sommerbaues von *Ochotona daurica* PALL. Massstab 1 :25.

VII. SPRINGHASE (*Alactaga mongolica RADDE*).

Die Baue dieser Tiere, welche sowohl in den Daurischen als auch Mongolischen Steppen sich in grosser Menge vorfinden, wurden ausschliesslich des Nachts ausgegraben. Die Tiere, welche in der freien Steppe mit dem Automobil unter Zuhilfenahme des Scheinwerfers aufgesucht wurden, wurden so lange verfolgt, bis sie sich in eine ihrer Hoehlen fluechteten. Bei der Verfolgung, welche sich oft ueber 5 Kilometer lange hinzog, entwickelten die Tiere eine Geschwindigkeit von 15-20 Meilen in der Stunde, wie durch das Tachymeter an der Maschine festgestellt wurde. Da die Springhasen bei der Verfolgung sich stets ebene Steppengebiete aussuchen, und Abhaenge und Schluchten vermeiden, ist die Methode mit dem Automobil fuer den Fang dieser Tiere sehr geeignet. Obwohl zahlreiche Baue von Tarbaganen, Zieseln und Pfeifhasen in der Steppe lagen, sprangen die Alacdagen stets nur in Hoehlen von einem bestimmten Typ, welche ihnen offenbar zugehoerten. Beim Ausgraben eines dieser Baue wurden ausser dem Verfolgten noch ein zweiter-kranker-Springhase hervorgeholt, dem der Bau sicher angehoerte. Die ausgegrabenen Baue sind alle von einem Typus: Ein ziemlich kurzer Gang fuehrt steil nach abwaerts in eine Tiefe von etwa 70 cm, wo er sich zu einer kleinen Schlafkammer erweitert. Von der Kammer fuehrt in gerader Richtung weiter ein kurzer Sackgang, der zum Schluss leicht nach aufwaerts fuehrt. Verzweigungen in Form von kurzen Sackgaengen sind selten. Die totale Laenge des Baues betraegt kaum ueber zwei Meter.

VIII. ILTIS (*Putorius*).

Abbildung 12. Bau des Iltis (*Putorius spec.*) ausgegraben an einem steilen Abbang (A) in der Umgebung des Bades Schiwanda, August 1923. Um den Ausgang lagen im Sande Ueberreste von 5 aufgefressenen daurischen Zieseln. Felle und Skelette dieser Zieselart fanden sich

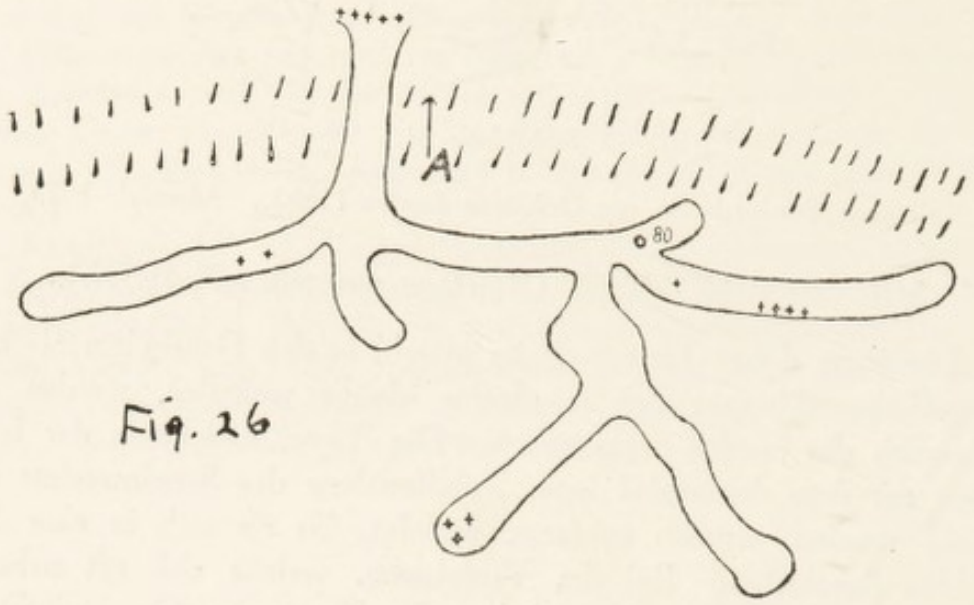


Abbildung 12. Von einem Iltis (*Putorius*) bewohnter Bau. Massstab 1 :25.

auch fast überall in den Gängen des Baues. Die Anzahl und Fundstellen dieser Zieselreste sind in der Figur durch Kreuze bezeichnet. Das Tier hatte es in einem Sommer fertiggebracht, die zahlreichen Ziesel einer grossen Halde vollkommen auszurotten. Zur Zeit des Ausgrabens war es nicht in der Höhle.

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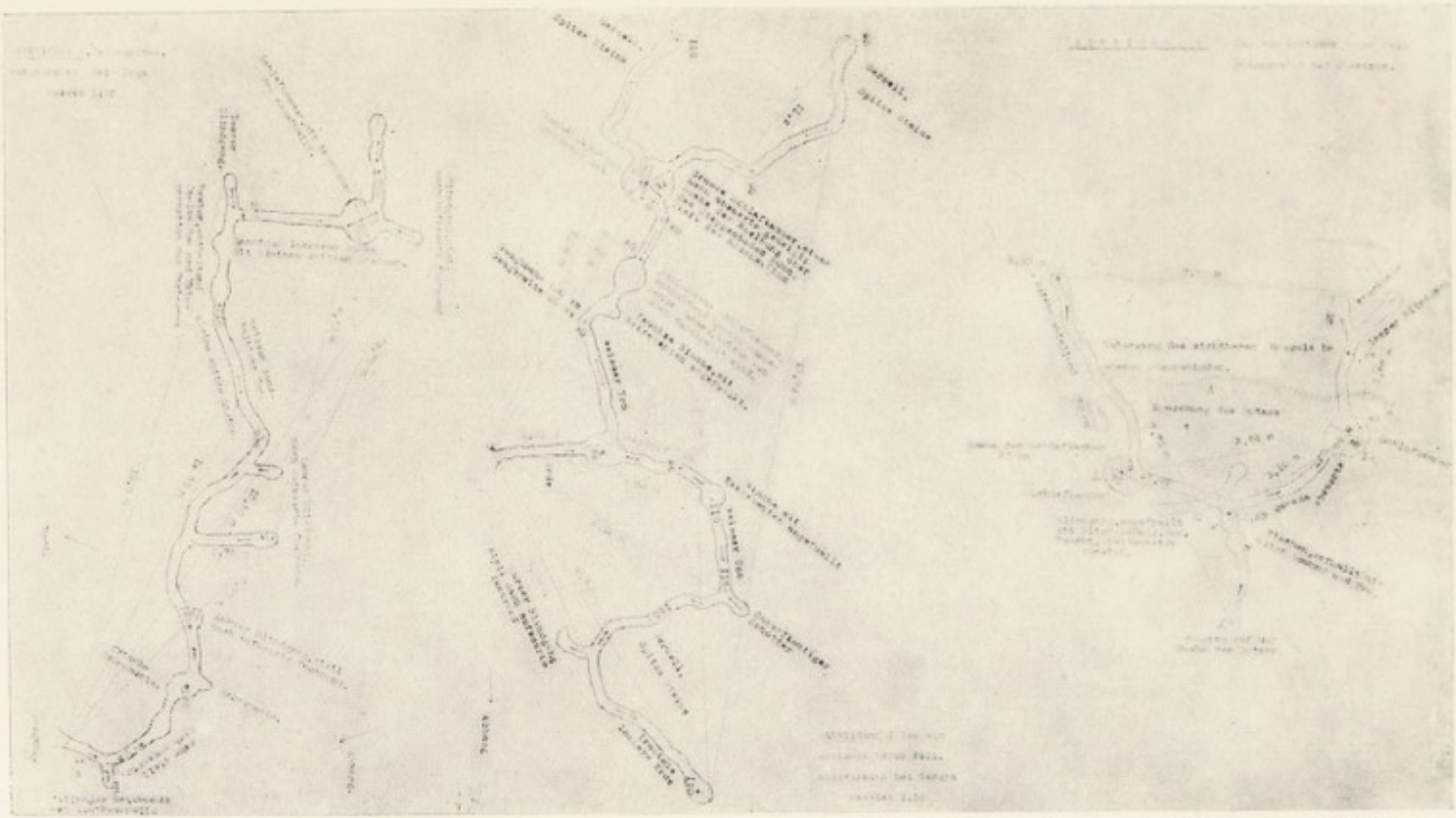

Fig. 27. Three plans of underground borrows of tarabagans, first two in Mongolia and third in Transbaikalia.

示旱獺所造之地洞道 第一二兩圖爲蒙古的 第三爲東西比利亞的

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H. M. Jettmar, M.D. (Wien),

Serologist, P.P.S.

PLAGUE INVESTIGATIONS AT TUNG LIAO 1928.

WITH AN ADDITIONAL NOTE BY DR. J. W. H. CHUN.

For several days towards the end of July and beginning of August, Chinese and Japanese newspapers had published alarming accounts of Pneumonic Plague having occurred in the Tung Liao (Payintala in Mongolian) district in Fengtien Province, where one village was supposed to have lost 20 persons and another (north of the River Liao) practically all 60 families except three children. On August 15 the Health Section of the League of Nations wired me for confirmation, and on August 18, another telegram arrived from the Eastern Bureau at Singapore asking for details. I decided to investigate the matter myself and therefore left Harbin by the night train on August 18, changed into South Manchurian express at eight the next morning and arrived at Ssupingkai two hours later. Here I stayed for the day making enquiries from the medical and lay staff of the Ssupingkai-Tungliao Railway, whose headquarters are situated in this city. At 7 a.m. on the 20th I started again by train and reached Tungliao station at 2 o'clock the same afternoon. The country everywhere is flat and suitable for cultivation. The district engineer Mr. Siao kindly put me up at his house since no hotel accommodation was available.

Tungliao is a newly opened-up flat area of Fengtien Province with Mr. Chi as Magistrate under the *Taoyin* (Superintendent of Circuit) who resides at Chengchiatun. The town itself is quite unimposing, a straight loose earthen road lined with grey brick houses proceeding direct from the up-to-date station. At right angles to this main thoroughfare are intersecting roads, all non-metalled because of the absence of granite in the vicinity. The total population is under 20,000, consisting principally of immigrants from Shantung, Chihli and the Liao district of Fengtien. Their occupation is mostly agricultural, some business being done in horses, cattle and clothing with Mongol tribes across the River Liao in the north. The crops grown consist mainly of beans, kaoliang, wheat, millet, and hemp. The soil is fertile and water sufficient for most purposes.

Interviewed the magistrate, head of police and local railway staff, who acknowledged having heard of suspicious deaths in one or two villages 10-15 miles away, but the symptoms were vague and might have been due to mushroom poisoning, influenza, meningitis or enteritis as well as plague. There was a young Korean doctor Li, who was positive about having seen a plague case with cough last year. Dr. Cheng, an observant and experienced practitioner, who had worked in Harbin in the 1921 epidemic informed me that early in August four patients coming successively from one compound in a neighboring village consulted him at his office. They said that three others living with them had died of high fever and unconsciousness; one complained of a bubo in the right groin. The four patients seen by Dr. Cheng were as follows:

1. F. 46. with fever, cough, diarrhoea and vomiting. Died 2 days after.

2. M. 40. with fever, headache and delirium, no cough. Died next day.
3. F. 24. with fever and vomiting. Died 2 days after.
4. M. 52. with T. 102, rigor, backache, died suddenly.

He made no blood examination during life or after death, although the rapid deaths were significant.

The police could not give any definite information, and although I stayed in the city for four days I did not see or hear of any suspicious case.

In the meantime, I tried to collect some rodents in houses and the open fields. No rats were caught. Of the wild rodents, apparently the country from Tungliao eastwards as far as Tahan (60 miles away) is inhabited by the *spermophilus*, jumping hare *Zapus* and small mice, whose different burrows can be clearly seen from the railway train. I caught samples of the two former species (seven in number), but none showed any sign of disease. The *Arctomys* (tarabagan or large Siberian marmot) certainly does not exist for hundreds of miles around. Since the question of the endemicity or not of the Tungliao region is most important in the etiology of plague, I have prepared a map of the tarabagan areas in North-east Asia (Fig. To be fixed in final proof with illustrations).

Roughly the boundaries are :

North. Great Siberian forest known as Taiga, through which pass at one stage or another the River Shilka, Argun, Unda and Onon.

South. Gobi Desert, where the deep sand dunes are unfavorable for tarabagan. Some parts have not been traced.

East. Khingan Range of mountains. The Chinese Eastern Railway traverses this region.

West. Rather indefinite, but probably extends to over 200 miles west of Urga. In the neighborhood of Urga the animals are very numerous.

Outer Mongolia with Urga as a center has for centuries been an endemic focus. Caravans carrying produce in the form of grain, fur, hides, clothing, fodder, etc. ply regularly between Mongolia on one hand and Kalgan (through Suiyuan), Tungliao and Taonan on the other. The increase of traffic brought about by newly opened railways, such as, Chengchiatun-Taonan-Tsitsikar, Tungliao-Chengchiatun-Ssupingkai, Tungliao-Tahusan-Mukden, Tungliao-Yinkow, besides the regular land route from Urga to Kalgan via Suiyuan and Poutouchen, has all added to those potential dangers.

Our Dr. Jettmar after one year's work in the midst of Urga (1926-7) calls the Mongols taciturn and self-contained, where plague outbreaks are concerned and is convinced that there is a yearly recurrence of both epizootics and epidemics on a big or small scale. During recent years we have already recorded the following human outbreaks as having arisen from Mongolia :

1917-18 Shansi epidemic with 16,000 deaths. This began in Outer Mongolia, passed through Poutouchen and Suiyuan, invaded Fengchen, Tatung, Peking and even reached Nanking. Cases were mostly pneumonic.

1919 Localised outbreak at Linhsien (Shansi) with 350 deaths, principally bubonic.

1924 Localised outbreak at Hsing-hsien (Shansi) in Oct.-Nov. killing nearly 800 persons, mainly bubonic.

1926 Limited epidemic in Tsechan-Han in Outer Mongolia, definitely traced to tarabagans, involving 6 localities, lasting 5 weeks. 24 cases were recorded.

1927 Scattered outbreak in Tungliao region, probably in the main bubonic and killing 95 persons. Lasted Aug.-Oct. From one corpse were obtained films showing *B. pestis*.

This year another explosion has taken place at Tungliao and Chienchiatien, involving up to September 15th about sixty persons, with buboes and septicamia but it is hoped that by early preventive measures and systematic vaccination among the inhabitants the epidemic will be limited to the sparsely populated regions. Unless it takes on a pneumonic form, there is every hope that its spread will be stayed.

Railway connections.

Since the rapid means of communication may enhance the spread of plague in these regions, the various operating railways may be mentioned in detail :

1. *Peking-Mukden* (*523 miles*). At Tahusan (between Koupangtzu and Fengtien) a branch line proceeds northwards to Tungliao, distance 256 miles. There is daily through service between Fengtien (Mukden) and Tungliao (350 miles) and also between Newchwang (Yingkow) and Tungliao (387 miles).
2. *South Manchurian*, Dairen to Changchun (436 miles). At Ssupingkai (62 miles south of Changchun) the Ssu-Tao Railway operated by Chinese runs on one hand northwards to Taonan to join the Chinese Eastern at Anganchi, and on the other westwards to Tungliao. Both branches pass the old city of Chengchiatun, where the Taoyin or superintendent of circuit resides.

It is thus seen that the Tungliao region is brought into close touch with big cities like Mukden (Fengtien), Harbin, Tsitsikar (capital of Heilungkiang), Newchwang (seaport), Dairen (main seaport of Manchuria), Tientsin and Peking. It is also in communication with the main trunks—South Manchurian, Peking-Mukden, and Chinese Eastern. We cannot be too careful in watching the progress of the infection.

TABLE OF RAILWAYS IN MANCHURIA.

Peking-Mukden	523	miles.
Dairen-Changchun	436	,,
Newchwang-Tungliao	242	,,
Tahusan-Tungliao	157	,,
Chengchiatun-Tungliao	71	,,
Ssupingkai-Tungliao	126	,,
Ssupingkai-Taonan	195	,,
Ssupingkai-Chengchiatun	55	,,
Changchun-Harbin	150	,,
Manchouli-Vladivostock	1070	,,

WU LIEN TEH.

ADDITIONAL NOTE BY DR. J. W. H. CHUN (SENIOR M. O.)

Dresser Wang and I left Harbin on the 5th September and arrived at Ssupingkai on the morning of the 6th. At 3 p.m., Drs. Li, Chien and later Dr. Wang (all of the Ssu-Tao Railway) went with us to the junction, Cheng Chia Tun, whence we went aboard a special car which was pulled to the station Chien Chia Tien early on the morning of the 7th.

Dr. Li, (Chief of the Railway Hospital) reports that for the last four years, he has noticed deaths from some infectious disease in the region of Tungliao. On the 30th August, he received news from the station master that for the previous two weeks 2-3 deaths have occurred daily in the eastern section of the village of Chien Chia Tien. Altogether there might have been some 20-50 cases of these deaths from some acute illness lasting only a few days. They seemed to be scattered over that part of the village, mostly isolated ones in a house, but there were sometimes 2-3 in the same household.

He went down to investigate forthwith, but just missed a corpse with cervical bubo, because it had been nailed up in a coffin. The *symptoms* were said to be fever, red eyes, unconsciousness (? delirium), tightness in the chest; some cases had buboes, rashes, and diarrhoea near death. The disease seemed to last usually 3 days. No cough or spitting of blood has been noticed.

Dr. Wang's report. For the last week Dr. Wang has been going to the village from Tungliao (23 miles) daily and inspecting the neighboring region with two policemen. He said he saw no buboes (?). The patients had fever. From two patients he took blood from the basilic veins and made smears but could see no bacteria (the microscope was not powerful enough), but there seemed to be leucocytosis. None had lung symptoms.

Two Japanese S. M. R. doctors went down on the 2nd September and saw a young male cake-seller. He had fever, and right femoral bubo from which blood was withdrawn and proved by Dr. Kanai in Dairen to be full of *B. pestis*. Later on, all tests, including animal and agglutination tests confirmed Bubonic Plague. The patient in question was seen on the second day of illness and died on the third day. He also showed no lung symptoms.

Description of the village. The surrounding country is sandy and sparsely cultivated. Kaoliang is the principal crop. No tarabagans abound. The villagers are mostly farmers. There are 100-200 mud houses with flat-thatched roofs, and mud floors. The village is situated about ½ mile from the station on north side. The police are very apathetic. The infected east side was supposed to be isolated from the west by stretching 3 ropes across the main street. The whole village was quarantined by the cancelling of passenger traffic, and by the stopping of foot passengers by police (this was not enforced strictly owing to the open country).

On arrival, we were told by one of the station staff that a Mahomedan native doctor (Li) had died that morning. We hurried to the police station, and obtained two men to go with us to visit the house. On going along the main street, we saw a sick young man in a barber shop. He was said to have been ill for some 14 days. He had fever and furred tongue. No diarrhoea. No buboes. No enlarged spleen. No physical signs anywhere. Fever was about 101°F. The mother of the patient was reduced to hysterics by our presence, and "make-up" (white apron, mask, hood and gloves). The second case was a man of about 40, living in a small isolated hut with his wife and child. He was seen by Dr. Wang the day before, and some blood was taken for examination. At that time the result was negative. He also had fever. No buboes. No lung signs. No enlarged spleen. He was in his third day of illness.

We then directed our attention to the Mahomedan's house. There were four members of the family left. Wife, sons and brother were all well. No history of death of rats about the premises. The corpse was laid out in the inner room, covered with a blue cloth. The wife (an old lady) refused to allow us to see it until we threatened her with eviction. On removing the cloth, the body was that of a strong man of about 59 years of age. Bluish in colour. Rigor mortis. A patch of subcutaneous haemorrhage on the left hip. No cervical or axillary buboes. But there was a large left femoral bubo. On incision, blood was withdrawn on knife and smears and cultures were made. Dr. Li withdrew some blood with a syringe, made smears for himself and injected two white mice which died 15 hours afterwards. The smears were examined afterwards and found to be full of typical *B. pestis*.

The deceased had died on the morning of third day of illness. The appearance of the corpse gave an impression of severe septicemia. Owing to loud protests from the wife, nothing further could be done in the way of investigation.

Measures already taken. Beyond stopping the sale of tickets to and from Chien Chia Tien, and asking the police to keep the affected village isolated, the railway has done nothing more than keeping a sharp look-out on the number of deaths reported daily both in the village and Tungliao (reports from this place seemed to indicate the presence of two suspicious deaths on the 7th).

Measures recommended.

1. The stoppage of all passenger traffic from the junction Cheng Chia Tun to Tungliao, and from Tungliao to Tahusan connecting with the P.M.R. and thus Fengtien and Peking.

Alternately 2. The stoppage of tickets at Chien Chia Tien, Ta Lin, Ta Han, and enforcement of quarantine measures at Tungliao or medical inspection. The same for Cheng Chia Tun and Ssupingkai.

3. In connection with 1 or 2, the local authorities must be pressed to keep a sharper look-out on the isolation work by strictly surrounding the village, and stopping all foot passengers.

4. The establishment of an antiplague bureau which will have to carry out the work of hospitalising the sick, quarantining of the contacts, disinfection of the houses, disposal of corpses, etc.

REMARKS.

1. Dr. Li thinks the infection must have been smouldering at a little distance from the village, and that it has now finally infected it.

2. The patients are among family people, not travellers or inn-dwellers. It was very difficult to get hold of any material for examination, from patients or corpses because some relative is sure to object. It was lucky that we came across a Mahomedan, because of the custom of not having any clothes on the corpses, so that no undressing need be done.

   The people tried to bury the dead as soon as possible. They offered much passive resistance, and no doubt tried to hide their dead. Some are said to have run away to Tungliao after deaths have occurred in the family.

3. The Magistrate in Tungliao does not seem to take any notice and the police are apathetic. They need to be stirred by pressure from Fengtien authorities.

4. The Ssupingkai Taonan Railway heads are afraid to acknowledge the presence of plague officially, owing to the attitude of the Fengtien authorities last year, and also the lack of funds for antiplague measures. The railway heads did not wish to hold a meeting to discuss the plague situation.

5. They look to the Plague Prevention Service to furnish everything, including funds. The most urgent seems to be vaccine and serum.

   The Railway doctors are keen men, willing to work, but with little knowledge or experience.

J. W. H. CHUN.

Harbin, Sept. 9, 1928.

NOTES ON PLAGUE IN INNER MONGOLIA.

(Reprinted from League of Nations Health Rep. Oct. 25 and Nov. 8, 1928).

I. The following information is extracted from a report received by the Health Section from the *North Manchurian Plague Prevention Service* :

Rumours have been heard during the last five years of mysterious outbreaks among the Mongol population north of the river Liao, which runs practically parallel to the railway from Chengchiatun to Tungliao (Payintala). No bacteriological work was done until 1927, when the Director of the South Manchurian Railway Medical Service, accompanied by Chinese doctors, visited the region. They made a post-mortem on a corpse found on the wayside. It was believed that *B. pestis* was demonstrated in films of liver, but the cultures were contaminated and animal experiments gave negative results.

In August 1928, there were again rumours of epidemics and Tungliao was visited, but no plague case found. Early in September, deaths occurred at Chienchiatien and plague bacilli were demonstrated by our Dr. Chun in the bubo (groin) of a Mohammedan native physician who became ill two days after visiting his last patient and died the next day. The culture brought back proved positive and 30,000 doses of anti-plague vaccine were forthwith manufactured by the agar method.

The clinical and post-mortem investigations performed at Chienchiatien proved that this is practically a pure bubonic epidemic. Only in few corpses were no evident buboes found. Over 50 per cent. of the buboes are in the region of the lower limbs, but a large percentage are found in the neck and armpit. The disease is unusually rapid. A large number of cases end fatally within twenty-four hours after the first symptoms. The majority of deaths occurred within three days, but some cases live for seven days. The incubation period is usually three days. Septicæmic cases are met with and one skin case has been seen. On September 25th, a post-mortem revealed small broncho-pneumonic nodules in lungs containing plague bacilli. Again, on October 4th, a post-mortem revealed almost primary morbid changes of pneumonic plague in the lungs; no buboes were present and the patient coughed up bloody sputum. There have been a number of recoveries in inguinal, cervical, femoral and even one in a popliteal case. About 92 per cent of the cases seen have so far ended fatally.

Rats were numerous at the beginning of the epidemic, but they seem to have died out when it was at its height. Attempts to recover sick rats have usually been negative, but on October 3rd a dead rat with plague-infected blood was found. Three human fleas from a recently dead patient were ground up in emulsion which was inoculated in a guinea-pig, with the result that it died from plague within three days. Numerous plague bacilli were recovered. The same results were obtained with bed-bugs.

In 1927, probably about 200 deaths occurred in Outer Mongolia and less than 50 among the Chinese in the vicinity of Tungliao. In 1928, the outbreak probably began in July in Outer Mongolia. The epidemic was apparently general, for appeals for vaccines, sera and medical supplies were received from Urga through the Russian Medical Service. The disease appeared probably in Tungliao in the first week of August, and four cases coming from a village 20 miles north of Tungliao were attended by one of our doctors at his office in Tungliao. These patients died within two or three days. The first case at Chienchiatien was reported in a Mongol who had travelled from a neighbouring camp 7 miles to the north. The first Chinese case occurred on August 21st and the first bacteriologically verified case on September 7th. Since the beginning of the epidemic there have been about 300 deaths at Chienchiatien in a population of 1,800. Some 500 of the inhabitants have run away to various places and the isolated cases occurring at Chengchiatun, Talin, Pamiencheng, Sanlin and Tungliao have been traced to these refugees. Only at Tungliao was there any considerable number of cases *i.e.*, about 18. The total number of deaths to date (October 6th) is less than 400. The epidemic has been well in hand since September 26th, and only two or three new cases are now occurring daily, all confined to Chienchiatien.

Another interesting focus is Chan Yu, which is not on the railway but connected with it by motor-buses, which do the 40 odd miles in two hours. This Chinese town contains about 20,000 inhabitants and is in frequent communication with the neighbouring Mongol camps. One of our doctors stationed there reports that 11 plague cases occurred in August and 21 in September, since when the epidemic has abated. Evidently the outbreak has arisen independently of that at Chienchiatien. Taonan, which is in close touch with other Mongol camps, has remained free from plague.

Funds for combating the epidemic have promptly been granted by the Chinese authorities. A quarantine station has been established at the railway junction of Chengchiatun by stationing 50 empty freight-cars and retaining passengers from Tungliao for the Taonan line for five days. Medical officers travel on all passenger routes.

The North Manchurian Plague Prevention Service is collaborating with the Sanitary Service of the South Manchurian Railway Company.

II. Additional information dated Szepingkai, October 20th, has been received from the *North Manchurian Plague Prevention Service*:

The plague epidemic has practically come to an end and only occasional cases occur at Chienchiatien. These cases come from outlying districts, while it appears that the village itself is now free from plague. The little village of Chienchiatien, with its 1,800 inhabitants, has borne the brunt of the epidemic and lost about one-fifth of its inhabitants. The second centre was Chan-Yu, while the majority of the cases occurring in

other towns were among refugees from Chienchiatien. Nine-tenths of the cases occurred in the eastern part of the village, which consists of mud and straw houses. The western quarter, which consists of houses with brick walls, was less affected.

The number of victims of the epidemic has been roughly as follows :

Chienchiatien	340
Tungliao and neighbourhood	40
Talin	12
Sanlin	22
Chan-Yu (Mongol settlement not on the railway)	50
Chengchiatun	2
Pamiencheng	1
Other localities	20
Total	487

Only 2 cases, out of 15 post-mortems, showed secondary lesions in the lungs; one of these had had cough and bloody sputum. Treatment with locally prepared vaccine appears to have been successful in several cases. The second experiment with human *Pulex irritans* proved successful; the guinea-pig died on the ninth day after inoculation, showing all signs of subacute plague.

Fig. 28. Flat country on the way from Chengchiatun to Tungliao with small bootans (mounds) inhabited by small wild rodents.

示由通遼至鄭家屯之大平原有一樹堆附近處有小齧齒類居留

Fig. 29. Railway station Tungliao completed in 1926.

示通遼車站係一九二六年落成者

Fig. 30. Main street of Tungliao with roads made with earth dug from trenches on either side.

示通遼街市大道係由兩邊陽溝中所掘之土築成者

Fig. 31. Horse market in summer at Tungliao. Note the simple and effective straw hats.

示通遼夏季之馬市注意最單簡而實用之草帽

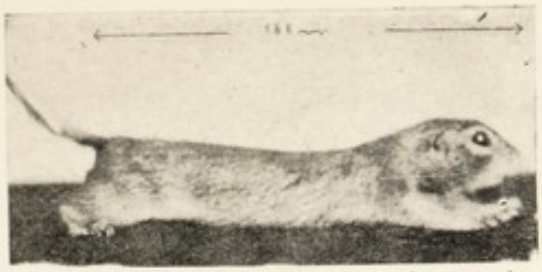

Fig. 32. Sisel (Spermophilus spec.), Chinese taoshu caught at Tungliao fields August 23, 1928.

Desc.: adult male. Back dark brown with interspersed black spots, sides of head, neck, and flanks hazel-brown to whitish yellow, belly sandy yellow, almost cream colour. Inner side of legs almost white; eyelids whitish-yellow, whiskers black. Ears very short, finely haired. Sides of the nasal and the whole maxillar region light yellow. Tail short, with long hairs partly black, partly yellow.

Measurements:		
	total length:	220 mm.
	tail:	28 ,,
	Ear distance:	31 ,,
	Pupillar distance:	29 ,,

斯士兒鼠中國名爲豆鼠於一九二八年八月二十三日由通遼田間所捕者

解剖）雄鼠背部褐灰色分散各部中有黑點頭之兩旁頸及脇腹部帶棕色或白色黃色腹部爲砂黃或爲乳白色腿之內側爲白色眼面爲黃白色鬚爲黑色其兩耳甚短毛極細緻鼻側及全腋部爲淡黃色尾短有長毛一部份爲黑色一部份爲黃色

體之量度		
	身體長	二二〇密米
	尾　長	二八密米
	耳距離	三八密米
	趾距離	二九密米

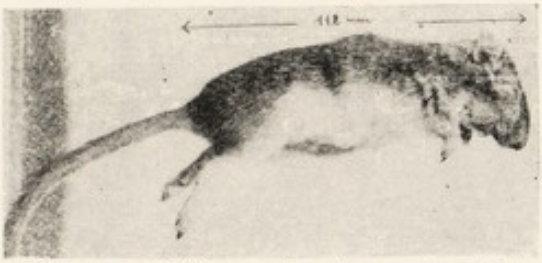

Fig. 33. Field spring mouse; Chinese: tiao-shu, rodent, belonging to the family Jaculidae, subfamily Zapodinae, genus Zapus, spec.? caught at Tungliao fields, August 23, 1928.

Desc: adult female body, bottle shaped, neck short, thick head elongated, narrow, mouth small, Ears round oval shaped. Eyes rather small, whiskers somewhat longer than the length of the head, anterior ones all white, posterior ones all black on the basis. Fore-legs short, with 4 toes, hind legs long with 5 toes. Tail round, *as long as the body*, with long yellow hairs, on the dorsal side interspersed with black hairs.

Back dark yellow with black hairs; belly as well as inner sides of legs white.

Measurements:		
	total length:	112 mm.
	tail with hairs:	118 ,,
	Ear distance:	30 ,,
	Pupillar distance:	18 ,,
	Nostrils-edge of upper incisivi:	9 ,,

田跳鼠中國俗稱跳鼠此齧齒動物爲紮古力打屬又爲紮坡顚尼副屬金勞斯屬等由通遼之田間一九二八年八月二十三日所捕者

(解剖) 該雌鼠體如瓶形頸短頭長尖口小耳卵圓眼小鬚比頭長前面部白色後脊背爲黑色前腿短有四趾後腿長有五趾尾圓與體長相等尾又長帶黃毛其背黑色散佈於內甚多間雜有褐色腹部及腿內爲白色

量　度		
	全體長	二二密米
	尾帶毛部	二八密米
	耳距離	三〇密米
	趾距離	一八密米
	上門齒至鼻樑	九密米

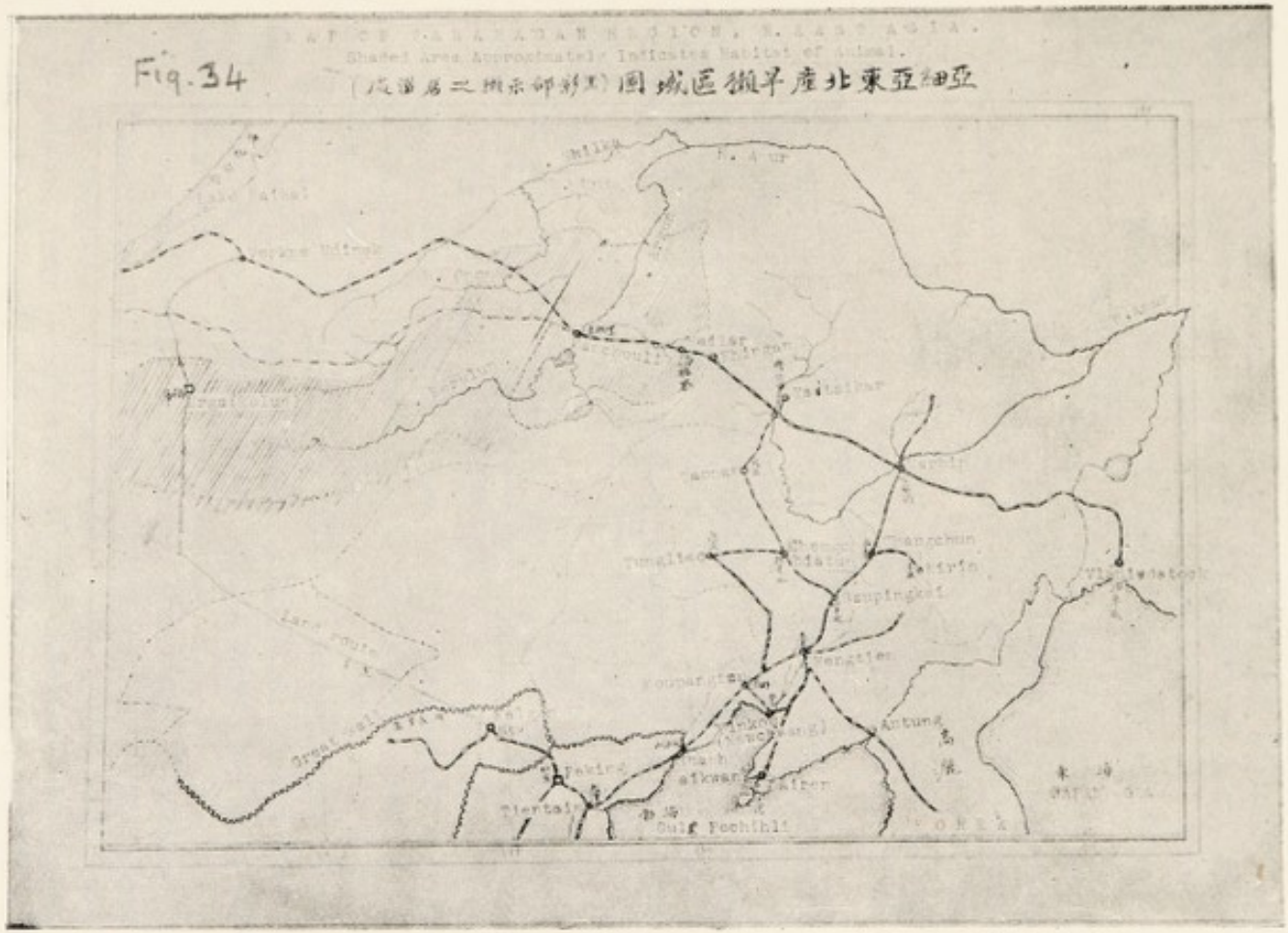

Fig. 34. Plan of Manchuria, Siberia and Mongolia showing Habitat of the Tarabagan.

東省西比利亞及蒙古全圖示旱獺之居留地

ONE YEAR IN A MONGOLIAN LABORATORY, 1927.

(English summary from an Article in German: Bericht ueber meine Kommandierung in die aeussere Mongolei und ueber meine Taetigkeit im Urgaer Veterinaerinstitute).

In autumn 1926 I was invited by Dr. Dudukaloff, Head of the Veterinary Service in Urga, to work under his direction in the Sangin-Rinderpest station near Urga. In November of the same year, when reports came from Mongolia of an outbreak of Pneumonic Plague, I received an urgent telegram from Urga to procure anti-plague equipment, and proceed at once to Urga.

Having received a year's leave from the Director of the Manchurian Plague Prevention Service, and provided myself with laboratory and anti-plague equipment, I left Harbin for Manchouli on December 11, 1926.

The further journey of 800 kilometer was somewhat tiring, as our motor-car got broken not far from Gurbu-Bain on the Manchuria-Mongolian border (220 kilometers), and I had to continue my travel on camels to Urga. The whole trip took over one month.

As I had left Harbin without any Mongolian documents, I found some difficulty at Sanbese in proceeding further to Urga. After some days' delay, thanks to the efforts of the local Veterinary Department my "dsara" (Mongolian passport for travellers) was ready, and I was given permission to travel by the military route, and to take advantage of night shelters at the stations (*ourtons*), placed along the route at distances of 25-40 versts.

The journey to Urga was undertaken on sleighs pulled mainly by camels, while the weather was cold, snowy, and stormy most of the time. I suffered much from the cold and snowy storms of the desert (*shourgans*), which were the cause of our losing the way sometimes in spite of instructions obtained from ourton to ourton and the presence of local Mongolian guides.

Finally, on the January 13, 1927 I reached Urga, where I learned that the small Pneumonic Plague outbreak at Tsetsen Han was over, and Mongolia was entirely freed from plague (as far as human cases were concerned).

I was employed by the chief of the Urga Veterinary Department at the Rinderpest-station of Sangin (18 versts from Urga), where I worked until my departure in autumn 1927. During this time I was engaged upon administrative work, and had not sufficient time to perform systematic research work, although the material was abundant, and the conditions in the Sangin-station with its well equipped laboratory were favourable for different scientific investigations.

I was able, however, to make various observations upon medical and veterinarian questions in Mongolia, and had occasion to study the biology of local rodents. These observations, which are presented in a more detailed form in German, may be dealt with here.

The Veterinarian organisation in Outer Mongolia was founded and perfected by Dr. A. A. Dudukaloff (originally of Kharkov), the present head of the Chief Veterinarian Department in Urga. This organisation is very important for a country like Mongolia with its stock-farming population. It also does considerable work in the suppression of Rinderpest, the most fatal disease among cattle.

The epizootic has spread widely over Mongolia and neighboring regions during the last decades, so that districts, formerly entirely free from plague, are often in danger of being invaded.

Expeditions were once sent, mainly from Russia, into Mongolia in order to fight the cattle-plague by means of extensive vaccinations. But these expeditions usually arrived too late, and did not possess sufficient staff or material, to deal with such epizootics on a big scale.

Only after the establishment of cattle-plague-stations in the enzootic areas was it possible to fight the disease successfully.

At the present time there are in the Outer Mongolia four Rinderpest-stations :

1. The Sangin Station near Urga with 400 immune cattle,
2. The Sain-Shabé-Station with 400 immune cattle,
3. The Uljassutai-Station with 100, and
4. The Orchon-Station with 400 immune cattle.

Thanks to these Stations which altogether produce over 70,000 liters of antiplague serum per annum, and a well organized vaccination campaign spread widely over the whole country, the early dangers have been considerably reduced, and the disease is now almost stamped out. According to the latest reports, Mongolia is now altogether free from Rinderpest after liquidation of the last small outbreaks by means of universal vaccination of cattle around the infected areas.

The immune serum as prepared is mixed in the proportion 1 : 10 with 5% carbolic acid, and issued in bottles of 500g. As it is rather inconvenient to transport big quantities of serum on the Mongolian roads, especially in winter time, the Sangin Laboratory is now engaged in working out a rational method of preparing dry antiplague serum.

Formerly there were used as immune cattle mainly the Mongolian variety, but at present there are used almost entirely cross breeds between the Mongolian cattle and the Yack, the so called *Hainyks*.

These *Hainyks*, are highly susceptible against Rinderpest. Their susceptibility is even higher than that of the most cultivated European races. The mortality usually is about 100%.

These cross breeds (hainyks) are usually bigger and sturdier than their parents, and prosper only in cold high mountainous regions.

On account of their high susceptibility against cattle plague it is necessary to use bigger doses of serum. The hyper-immunised animal produces a large quantity of serum with a high titer.

At present immunisation is performed by intravenous injection of plague blood. This method has been introduced since 1921 by Dudukaloff, and saves a lot of trouble and expense. Should an animal under

observation (either yack or Mongol cattle) show plague infection, it is killed before death, and the defibrinated blood used for intravenous injection into the hainyks for immunisation purposes. Nowadays there is need to use fewer such donors for plague blood than formerly when the subcutaneous method of immunisation was used.

Of other infectious diseases among cattle Peripneumonia is common, especially in the northern regions of Mongolia. Anthrax also occurs, although it is not so often observed as in neighbouring Siberia.

The small horned herd of Mongolia (e.g. sheep, goats,) also suffer from various diseases, which have been insufficiently studied until now.

For instance, in the South Western districts of Outer Mongolia, there is often observed a rapid spread of a devastating epidemic among flocks of sheep, the so called "godoron"—a lung disease with a high mortality.

Also diseases of the alimentary tract of sheep are common. Thus in the Tsetsen-Han district there occurred in summer 1927 a devastating infectious disease, where inflammation of the bowels constituted the most important findings at *post mortem*.

Trypanosomiasis among the yacks and *piroplasmosis* among horses were also observed.

Among camels (*Camelus bactrianus*) I was not able to observe many serious infectious diseases producing high mortality, although these animals may occasionally suffer from rinderpest in sporadic form.

Among human infections in Mongolia, *Syphilis* takes the first position. Especially in the last few decades after the wide-spread immigration of Europeans this disease became prevalent on a very large scale. There are now few among young Mongolia people who do not show some sign of syphilis. Gonorrhoea is also exceedingly common. When I had to investigate students of 20 years in a Municipal school in Urga, I found one half of them suffering from *both* syphilis and gonorrhoea, while almost 100% were infected with syphilis.

Only in recent years has Salvarsan been used for treatment on a large scale, and the Mongols are now convinced of the evident therapeutical value of this remedy.

The universal propagation of syphilis among the population represents in the present time one of the most important dangers confronting the Mongolian population.

Regarding the causes I think the lamas play an important role in the distribution of syphilis. I am told that these people often take advantage of their position, and the Mongolian women in the jurtes do not dare to object. Polyandry is also an important cause. The typical Mongolian carelessness also favours the wide prevalence of the infection. The Mongol does not bother about a small ulcer on the top of his penis. He takes lama advice only when the symptoms are fully developed. About prostitution in Mongolia I have no authentic information. I know only that in one school most of the 18 years old boys were infected already with syphilis.

Tuberculosis is very rare among the country people. Nevertheless, Mongols do not show any natural resistance against this disease. When they have occasion to become infected with Tuberculosis in an European town, they often succumb to it.

Typhoid fever also occurs in small sporadic outbreaks in the outskirts and town of Urga. *Typhus Exanthematicus* and *Relapsing fever* have not been observed by me in Mongolia.

Smallpox is common, although vaccination is performed in schools, institutions and among the soldiers of the Mongolian army.

Infectious diseases of children seem to be comparatively rare, but may give rise to a high mortality among babies.

Malaria was not observed by me, although anopheline mosquitoes are common in the valley of the river Tola near Urga. Anthrax among men also occurs. Rabies in men was observed several times in the hospital. It is very common among the domestic animals. Not only dogs and wolves suffer from Rabies, but also cattle and horses are often bitten and succumb to the disease.

Dysentery is very common during the summer and autumn. The large number of flies during the hot season seems to be the main cause of these epidemics.

Among diseases of the skin *scabies* is very common. Sometimes it appears in a severe form, causing exhaustion, and even death of the patient. For instance, in summer 1927 a severe outbreak of scabies occurred in the Tsetsen Han district with a comparatively high mortality.

According to the reports of the local doctors *trachoma* is common. I myself observed only a few cases among the Mongols.

Common cold, pneumonia and rheumatism are rare. Their frequent exposure to the roughness of the climate, starting from the earliest stage of life, seems to protect the natives of Mongolia from these affections.

Bubonic Plague in man has been observed in Mongolia several times. Thanks to the simple though practical methods of quarantine used by the natives themselves plague outbreaks scarcely ever develope into real epidemics, particularly in regions where the density of the population is low.

I am convinced that outbreaks of plague occur in Mongolia almost every year, but often remain unknown because of carelessness and taciturnity, which are prominent characteristics of the people. I give the following instance: In autumn of 1927 two deaths were reported from the plague focus of Tzetzen Han. I was recommended by Dr. Dudukaloff as leader of an antiplague expedition to the district. The field laboratory was packed in a hurry during the night, and next morning we were ready to start. But the order for the motor-cars was not ready, and we waited for some days. I personally addressed the Department of Health, where I learnt that some days previously an express rider had been sent to the spot to find out 'what the matter was'. This man returned with the news that three men had died suddenly, but after that all had remained quiet. The Urga authorities on the strength of this cancelled the expedition. The

Fig. 35. Motor car caravan in Mongolia in winter.

在冬季蒙古運貨氣車

Fig. 36. Mongolian summer cart led by a camel and donkey ridden by woman.

蒙古人在夏季用駝車及乘騾之婦人

Fig. 37. Sacred dogs of the Mongols kept and fed carefully. These animals rapidly eat up dead people thrown to them.

蒙古之神犬愼于豢養此犬與以屍體速盡食之

Fig. 38. Veterinary Institute of Urga with immunised cattle in the foreground.

庫倫之獸醫研究所示前院內之已接種免疫牛

Fig. 39. Hyper-immunised cattle being bled for preparing immune serum against rinderpest

已施足量免疫牛取其血配製免疫血清作防牛瘟之用

Fig. 40. Sample of a hainyk (cross-breed between Mongol bull and yack) which has been immunised.

已種免疫之牽力克獸（係蒙古牡牛與西藏馬雜種所產之獸）

cause of death of the three persons remains unknown unto this day! The last outbreak was reported from the Tsetsen-Han district where tarabagans are abundant (Nov. 1926)

This animal is hunted at the present time very frequently. In 1927 there were exported from Mongolia to Kalgan about three millions of skins. The skins of the tarabagan from the eastern Mongolian districts are very valuable and worth about three dollars (*tuchriks*) per piece. There is another kind of tarabagan in the Kobdo regions, which is considerably larger than the eastern species but its black fur is not so valuable.

It seems to me that the Manchurian and the Mongolian Tarbagan near Urga are of the same species. When two tarabagans brought from Manchouli and two tarabagans from the steppes near Urga once were mixed together in one cage, they did not fight each other, and it was impossible to distinguish one from the other.

The structure of their holes is of about the same scheme. There seems to be only a slight difference: The hills erected over their burrows, the so called "butans" are bigger and more characteristic among Dahurian tarabagans than among those near Urga.

The Mongolian tarabagan inhabits almost the whole eastern part of Outer Mongolia with the exception of the areas northwards from Urga and Tzetzen-Han where there is woodland, the valley of the River Kerulen, and the deserts of the Diriganga-districts. It is found frequently in the steppes near San-Bese, in the mountains surrounding the Kerulen valley on the both sides, in the highland southwards from Tzetzen-Han and San-Bese, and also near Urga.

Quite often it is found on the slopes of the sacred mountains of Bogdo-Ul (opposite Urga), where all kinds of hunting are strictly prohibited.

The Mongolian tarabagans harbour on their skin a considerable number of fleas, and chauffeurs, engaged in the transport of fresh tarabagan-skins along the routes Urga—Sanbese and Urga Kalgan complain of the large number of fleas disturbing them during the journey.

In the Mongolian steppes there is a large number of different rodents. Small hamsters inhabit the districts of Sanbese, Tzetzen-Han, and Urga, in such numbers that they finally clear that part of any scanty vegetation left. Their extirpation by means of bacteriological methods is very difficult, because these animals do not touch the poisoned baits. Near Urga many experiments have been made in this direction but without any striking results.

The most active enemies of these small rodents are birds of prey and snakes, which live in abundant numbers in the steppes and mountains near Urga.

H. M. JETTMAR.

BIOLOGICAL INVESTIGATIONS AMONG ABORIGINAL TRIBES IN NORTH MANCHURIA.

(With 22 illustrations).

In May 1928 I received permission to make a tour of investigation in North Manchuria beginning with Sansing then proceeding to Lahasusu, onwards to Taheiho, through Mergen, southwards to Tsitsikar, and then back to Harbin.

The objects of my trip were :

1.—Investigation of the Biological Index (blood grouping) among the Fish Skin Tartars and other native tribes.

2.—Study of indigenous diseases among the aborigines, with some statistical data if possible.

3.—Study and collection of wild rodents.

4.—Study of the prevalence of malaria, and collection of Anopheles and Culex.

5.—Miscellaneous knowledge regarding Natural History.

I started for Sansing, on May, 18, where I was mainly engaged upon the study of wild rodents and their parasites. The whole June and the beginning of July I spent at Lahasusu where I investigated the Nabojé-tribe of Fish-skin Tartars and collected mosquitoes and assorted zoological and botanical material. In July I started for Taheiho, thence to Nunkiang (Mergen), where I investigated the population of the Solon tartar village Charitun and then the Dahurian tribe, whose villages are distributed around Nunkiang. But the continuous heavy rains and disastrous inundation of the rivers prevented me from proceeding with my studies, and I had to return on a sailing boat down the Nonni river to Tsitsikar, where I arrived early in August.

The whole material collected during the excursion has not been fully classified and worked up until now, and this report may be considered therefore as only a preliminary one.

1. Investigation of the Biological Index (blood grouping) among the Fish Skin Tartars and other native tribes.

Only the work upon the Fish-skin Tartars (Nabojé) near Lahasusu gave enough material for publication, while that upon the Solon tribe and other natives has not yielded sufficient material for drawing any conclusion. These studies will be continued at a convenient opportunity.

THE RACE INDEX AMONG THE GOLDI (NABOJE TRIBE) NEAR LAHASUSU.

The Nabojé-tribe,—Sungari-Ussuri group of the goldi (Chinese : Yu-p'i-ta-tzu or Lao-ta-tzu) are living in small villages around Lahasusu, down the river Amur to Habarovsk and the mouth of the river Ussuri.

Along the Amur river between Habarovsk and Novo Nikolejewsk there lives another closely related tribe of the goldi, the Chodin Bojé (lower people), the real "Fish-skin Tartars."

While this tribe is still numerous and numbers some thousands, the Lao-tatzu or Naboje (the Sungari-Ussuri group of the goldi) seems to be

definitely dying out and counts at the present moment no more than one thousand souls. The accompanying sketch shows the distribution of the tribe at present.

SKETCH SHOWING THE PRESENT DISTRIBUTION OF THE GOLDI (FISH SKIN TARTARS).

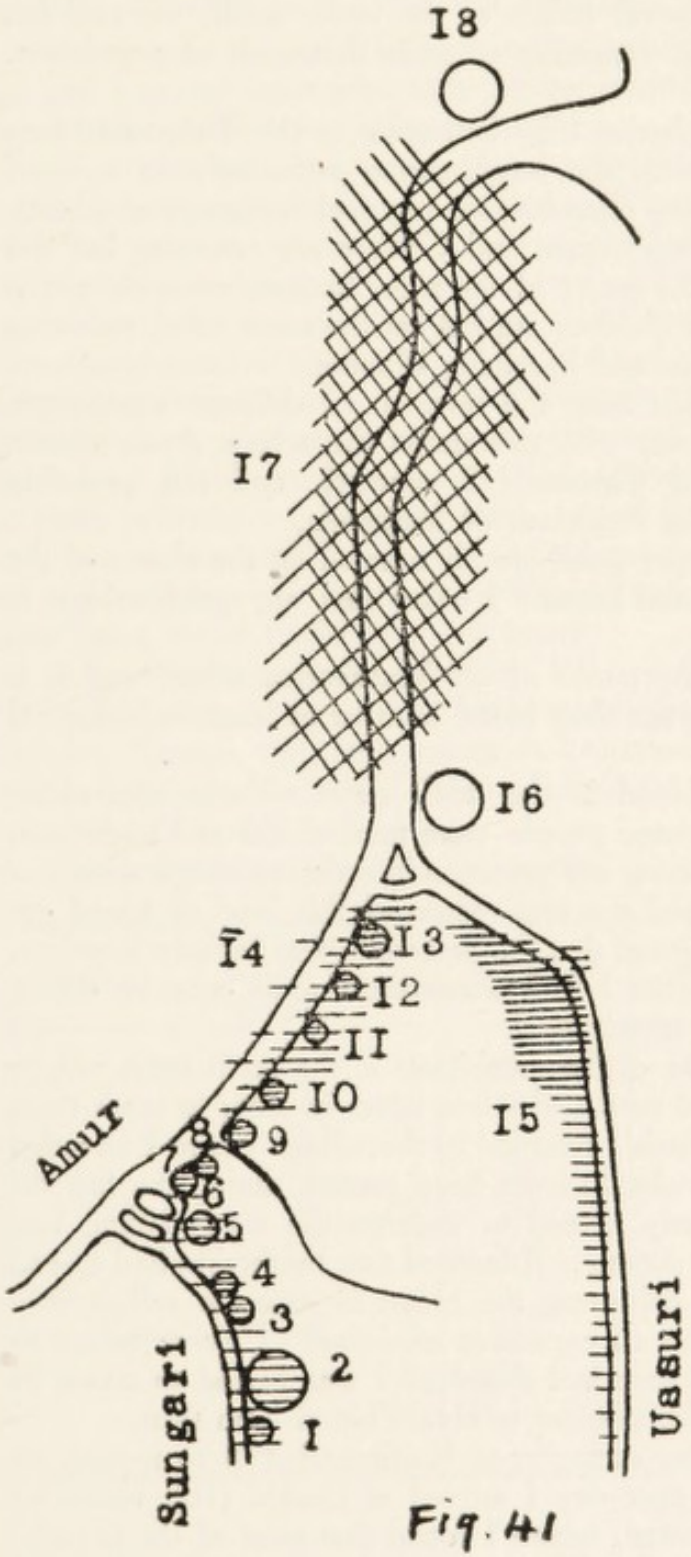

Fig. 41

In straight shaded line: Distribution of the upper goldi (Sungari-Ussuri group or the Nabojé).

In cross shaded line: Distribution of the lower goldi: The real fish skin tartars or the Chodin bojé.

1. Dathun, southermost colony of the Nabojé tribe (about 80 people).

2. Big Chinese town Fuchin containing 50-100 of the Nabojé tribe.

3. Tusheho (100 people).

4. Nuergu (30 people).

(pure tartar villages)

5. Chinese town Tungkiang (Lahasusu) near the mouth of the Sungari containing a small colony of goldi (about 40).

6. Chichikha at the mouth of the Sungari and

7. Molochunkho (almost pure tartar villages; altogether 80 persons).

8. Chai, a small tartar village (20 people).

9. Kaitikhautzu, a tartar village, mixed with Chinese colonists (about 50 tartars).

10. Derkhi (Telochi) almost pure tartar village (about 80 people).

11. 12. Etu, Kaitali and other tartar village

on the right side of the Amur river near the mouth of the Ussuri river (about 200 tartars).

13. Chinese town Sui-Yuan.

14. Small isolated colonies on the Russian border of the Amur.

15 Nabojé villages on the left side of the Ussuri river (denser population; altogether about 500-600).

16. Habarovsk.

17. Yu-p'i-ta-tzu colonies on both sides of the Amur river between Habarovsk and Novonikolayevsk (trible of the lower goldi; the real fish skin tartars or Chodin bojé), consisting of some thousands of population.

18. Nikolayevsk.

The goldi are a Manchurian tribe belonging to the Tungu-sian race group. They are mixed with the neighbouring races only in a small degree, most of them showing characteristic physical features (see photo). Intermarriages between Nabojéwomen and Chinese are common, but this fact has little influence on the purity of the race, because once the girl is married with a Chinese, her children are lost for the tartar tribe, receiving exclusively Chinese education and becoming Chinese.

The tribe suffered much from the invasion of different conquetors, Mongols under Chingis Khan, who devastated the whole Amur district (13 century), Russians and Chinese (17 century), and this probably accounts for the people being suspicious of foreigners.

The villages of the upper goldi are all situated on the shores of the big rivers Sungari, Amur, and Ussuri; I never saw any goldi-colony in inland places.

This tribe is one of the purest of all Manchurian tribes, and it is therefore of great interest to test their blood in order to learn its biological index.

The investigation of blood in the small tartar villages was rather difficult because the uncultivated people were most afraid and suspicious, and many left the village during my presence in order to escape examination. Only a few understood the real reason of this kind of blood examination. and became convinced that the procedure was entirely harmless.

To illustrate the difficulties I had to surmount, some episodes during these investigations may be mentioned.

After blood investigation of the inhabitants in the small tartar village Chichikha (6) I collected the used slides into boxes in order to wash them at home. Sometime afterwards I returned to the village where I intended to investigate more persons who had not been present during the first investigation. I failed: nobody agreed to undergo the investigation, and I was met in a rather hostile manner. I learned that busibodies had spread the information that I was collecting the blood in order to sell it very dear at Harbin, for producing an expensive medicine. Chinese refuse to give their blood for this purpose and therefore I was forced to travel to the poor and defenseless goldi in order to obtain blood from them.

Another time I made an excursion to Kaitikaotzu (9), to investigate some tartar villages. The next day I arrived at Derkhi (10), about 15 km downwards from Kaitikaotzu, where I found that most of the population of the village had escaped. We were met by the Gashendá (head

of the colony), who explained that the evening before there came a Chinese into the village who said that the goldi are defenseless dogs, and anyone can do as he likes with them. To prove his friendly declarations he foretold that the next morning a foreign doctor would enter the village with two Chinese soldiers to collect the tartar blood in bottles. If anyone dares oppose, he will be beaten to death.

It was very difficult for the Mr. Gashendá to persuade his people to return and undergo the harmless investigation.

Fortunately the goldi believe in the usefulness of small pox vaccination and I gained some popularity by performing *gratis* vaccination among the children of the tartar colonies.

As to the technique of the blood examination I used exclusively the so-called open slide method of Moss-Lee-Vincent.

This gave as satisfactory results as the test tube method, as exclusively hemotests of high titre (higher than 1 :20) were used. The hemotests were taken from the same individuals, who have repeatedly shown high isohaemagglutinative titre. This fact allowed exact comparison with the biological race index of the Chinese living in the same province (Kirin), because half a year ago there were performed investigations on the distribution of blood groups amongst Kirin-Chinese with the hemotests of the same individuals (vide Nat. Med. Jrl. of China 1928). All precautions were taken to prevent the appearance of unspecific autoagglutination and care was also taken in performing the reaction, the issue of the serum being dated back from 2-4 months.

Altogether there were examined 196 individuals, that is, about one fifth of the whole tribe. There were investigated the villages : Dathun, Tusheho, Nuergu, the tartar colony at Lahasusu, Chichikha, Molochunkho, Chai, Kaitihaotzu, and Derkhi (Telochi). There were examined 75 male and 121 female individuals.

The following table gives the results.

Locality.	*How many investigated.*	*m.*	*f.*	*O.*	*A.*	*B.*	*AB.*	*Biolog. Index.*
Dathun	35	6	29	13	10	9	3	1,08
Tusheho	34	11	23	5	5	20	4	0,33
Nuergu	15	7	8	12	1	2	0	0,5
Lahasusu	13	4	9	1	4	8	0	0,5
Chichikha	20	10	10	11	2	7	0	0,29
Molochunkhó ..	30	13	17	8	4	15	3	0,39
Chai	8	5	3	4	3	0	1	(4,0)
Kaitikhaotzu ..	11	4	7	6	2	3	0	0,67
Derkhi (Telochi)	30	16	14	17	6	7	0	0,86
Total	196	76	120	77	37	71	11	0,59
%	100	38,8	61,2	39,3	18,9	36,2	5,6	0,59

Although the number of persons investigated is small, it is possible to draw the conclusion that considerable differences exist between the indices in the tartar villages. Dathun, situated to the West of all other villages, has the highest index; in the villages on the mouth of the Sungari this is much lower and rises again in the villages lying Eastwards on the Amur (Kaitikhaotzu and Derkhi). Altogether the index is remarkably low, and is similar to that of the Annamites to whom the Manchus are said by Hirszfeld to be related. Also the high percentage of the O group is considerable.

The index of the Nabojé tribe of the Goldi is considerably lower than the lowest index of the Chinese in the Kirin province. It is also lower than that of the Koreans and the Manchus from Mukden. The following table shows the comparison between the indices of the Northern Chinese, the Eastern Mongols, the Mukden Manchus, and the Sungari Goldes :

Author :	*Race (Locality of investigation)*	*Number Examd.*	*O%*	GROUP			*Index.*
				A%	*B%*	*AB%*	
Jettmar & Lin	North Chinese Kirin	99	33'3	26'3	33'3	7'1	*0'8*
,,	North Fengtien	103	32'0	27'2	33'1	9'7	*0'9*
Jettmar	Mongols (Eastern Mongolia)	114	28'6	23'2	31'3	16'9	*0'83*
Fukamachi	Manchus (Mukden)	199	26'6	26'6	38'2	8'5	*0'75*
Kirihara & Haku	Manchus (Mukden)	236	30'9	25'9	33'9	9'3	*0'81*
Jettmar	Sungari Goldi	196	39'3	18'9	36'2	5'6	*0'59*

This table shows that the index of the Goldi is even lower than the comparatively low index of the Mukden Manchus (0'75, resp. 0'81). This fact may be explained by the Goldi being the purest representatives of the Manchu race investigated until now.

2. STUDY OF INDIGENOUS DISEASES AMONG THE GOLDI AND OTHER NATIVE TRIBES.

In this respect the Goldi have been most fully investigated. In the villages where I tested the blood groups I tried also to have a thorough medical investigation. My observations upon over 200 Goldi may be dealt with briefly here :

Information given by the natives and a study of monographs upon Goldi (for literature see end of the article), I believe the tribe is dying out.

There are many reasons for this extinction of the Sungari Goldi.

1. The male sex numbers more than the female.
2. Killing or giving away children born out of wedlock.
3. High mortality among children.

4. Callousness toward new born children, who are only regarded as valid human beings when the soul enters into the body (that is, when the child begins to speak.)
5. The women of the tribe prefer to marry rich Chinese. Their children then receive Chinese education.
6. Diseases, especially tuberculosis and small pox.
7. Deprivation of their natural conditions of life owing to the expansive force of Chinese colonists.
8. A certain laziness amongst men, who are easily defeated in the struggle for life against the laborious Chinese colonists. The men are also subject to alcoholism and often spend their whole money immediately after earning it.
9. Permanent humiliation by the colonists; prohibition of foundation of schools, prevention of celebration of religious festivals.

The depopulation is counterbalanced to a certain degree by the following facts: 1. Every girl marries soon after puberty. 2. The birth rate is high, and preventive measures of conception are unknown.

From the medical standpoint the Sungari goldi show the following characteristics:

1. *Growth of the hair.* Bald-headed people are rare (practically absent). I saw a 90 year old woman still with dense hair on the head. The hair of the beard appears late only (20th year). The hair of the head commonly turns grey somewhat early.

2. *Eyes.* Eye diseases, especially trachoma, are common. Blind people have been seen by me repeatedly. Amblyopia has been observed several times. Blepharitis with its complications is common. The strength of vision amongst healthy people is considerable. They are trained and experienced hunters and fishermen.

3. *Ears.* Otitis media with complications was observed several times. Deafness is not rare.

4. *Teeth.* In most of the colonies *caries dentium* is practically absent, almost all Goldi have wonderful sets of teeth. Only in one colony Dathun (1) I found among the women a considerable amount of caries. For instance, one woman 25 years of age had already lost one third of her teeth. In Chichikha (6) out of 20 grown-up people (between 20-40 years) only one individual had suffered from caries. One tooth was extracted. He traced this defect to the partaking of sugar when young. Old people have as a rule very strong and healthy teeth. For instance, at Derkhi (10), I investigated a 62 years old man whose complete set of teeth was still in a fine condition.

5. *Maxillar—and Jugular glands* are found in some colonies to be enlarged, even amongst small children. They are probably tuberculous.

6. *Lungs.* Tuberculosis is common. Especially in the small colony Dathun near Fuchin almost all the children investigated suffered from cough, with cervical glands and rhonchi. Many children died from "blood cough". Many of the grown-up people, especially women with clinical features of progressive tuberculosis, suffered from night sweats. In Chichikha and Molochun, also in Tusheho and Nuergu the population was practically free from Tuberculosis, while at Lahasusu, Kaitikhaotzu

and Telochi a comparatively high percentage of Tb. infected individuals was found.

7. *Heart.* Sometimes old people complain of heart troubles which may be traced back to a progressive arteriosclerosis. Angina is common, but rheumatic polyarthritis and defects of the heart are rare.

8. *Alimentary tract.* Many of the small children die of dysentery. Adults often suffer from enteritis during the warm season. Their monotonous food consists mainly of dried and rough fish and is favourable for the development of different kinds of gastrointestinal disorders.

9. *Venereal diseases.* The third stage of syphilis with destroying gummata of the soft palate has been observed several times. But the disease is not so common or severe as among other aborigines e.g. inhabitants of Outer Mongolia. About *gonorhoea* and *Ulcus molle* I was not able to collect statistical data.

10. *Skin disease,* are common. Scabies I have never seen among the Goldi. Although the people rarely wash their skin is as a rule free from acne or other inflammatory process.

11. *Gynecology and Obstetrics.* Amenorrhoea sometimes occurs, and the childless women try all kinds of remedies to beget children.

If in the course of labour some complications occur, an old woman with special knowledge and the shaman are invited to help. The shaman dances around the woman and does not touch her at all, while the old woman assists and pricks the patient with a pin in the abdominal skin at a spot, where the obstacle is supposed to be situated. The best remedy [1] for securing a normal labour is the embryo of a female deer. The doe's embryos may also be used but these are not considered so active, being cheaper than the embryos of the former. Other embryos are considered quite useless.

12. *Infectious diseases.* Small pox is very common. People with pockmarks are often seen. The mortality is high and a considerable percentage of children succumb to this disease. The Sungari Goldi admit the importance of vaccination against small pox. They consider vaccination as active only when there is marked heavy skin reaction. Thus when I arrived at Derkhi a Chinese dresser had performed vaccination amongst the children of the colony, and almost all of them showed severe inflammation on the arm leading in some cases even to cellulitis. The parents were pleased with the results and regarded the vaccine as a very strong and active one.

Typhoid fever is well known, and I was told sometimes that children died of "typhoid fever", but I have not been able to prove the presence of this disease amongst the goldi.

Malaria was not observed by me. Enlarged spleens are rare and typical malaria spleens with other typical clinical features I never saw.

Lepra. According to the report of Lopatin leprosy is common among the lower Amur-goldi. I myself never saw a single case.

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1 The manner of preparing this remedy is as follows: The embryo together with all the membranes is cooked in a pan for some hours. After cooling, the gelatinous mass is cut in parallel strips and sold very dear (one solotnik, i.e. 4¼ grams costs five local dollars). The woman in the late state of pregnancy throws a piece of this jelly-like mass into a glass of boiling water and drinks the emulsion.

13. *Nervous and mental diseases.* Women with hysterical fits occur. Epileptic strokes are not rare. For instance, the head (gashenda) of one of the investigated villages, a very intelligent and polite gentleman who knew perfectly well 3 languages (Goldi, Russian and Chinese), suffered severely from epilepsy.

14. *Infant mortality* is considerably high. The following tables demonstrate this, and show that unless precautions are quickly taken. the natives will die out very soon.

Very striking in this respect is the investigation of the married female population at Dathun where I had the occasion to examine almost all women of the colony:

TABLE A: INFANT MORTALITY IN THE GOLDI COLONY AT DATHUN

Married Fem. No.	Age	*State of health.*	*CHILDREN* birth	living	died	*Cause of death.*
1	25	Anaemia, Furunculosis	2	—	2	?
2	18	Cerv. glands, cough	—	—	—	—
3	21	Without findings	—	—	—	—
4	44	Bad teeth	4	—	4	3 dysentery, 1 small pox
5	33	a. Amblyopia, b. Amaurosis, Staphyloma anticum, cough, Furunculosis.	2	1	1	?
6	23	Caries dentium; one third of the teeth extr.	1	—	1	?
7	31	Without findings.	2	abortions		—
8	35	Luet. ulcer in pal. molle	1	—	1	?
9	39	Pockmarks, blind on one eye	5	1	4	"typhus"
10	31	Without findings	14	2	12	?
11	23	Without findings	3	—	3	one child of small pox
12	37	Cervical glands	4	2	2	one child of small pox
13	26	Cervical glands	3	—	3	?
14	18	Glossitis	—	—	—	—
15	51	Slight arteriosclerosis	6	3	3	?
16	30	Bad teeth, gingivitis Cervical glands	2	—	2	one small pox, one eclampsia
17	33	?	5	1	4	Idied 14 years old of "bloody cough".
18	32	Struma	6	3	3	all 3 died of "bloody cough."
19	26	Without findings	3	1	2	unknown
20	43	Amblyopia, Symblepharon	6	3	3	unknown
21	22	Cervical glands	2	1	1	eclampsia
22	31	Without findings	—	—	—	—
23	28	Struma, bad teeth	3	3	—	—
Total	23		72	21	51	

In other colonies investigated the infant mortality was rather high as shown in table B (Kaitikhaotzu). Table C (Chai), and Table D (Derkhi).

TABLE B: INFANT MORTALITY IN THE GOLDI COLONY AT KAITIKHAOTZU.

Married Fem. No.	Age	State of health.	CHILDREN birth	living	died	Cause of death.
1	53	a. Amaurosis, b. Amblyopia	13	6	7	Some children suffered from cough and died in the age of 10-15; others died from diarrhoea.
2	20	Irregular menstruation	—	—	—	—
3	35	Varicous veins	6	5	1	diarrhoea 5 years old.
4	24	Anaemia, rhonchi, stutterer	1	—	1	bloody cough
5	19	Heart troubles	1	—	1	eclampsia.
Total:	5		21	11	10	

TABLE C: INFANT MORTALITY IN THE GOLDI COLONY CHAI.

Married Fem. No.	Age	State of health.	CHILDREN birth	living	died	Cause of death.
1	51	Amblyopia, Trachoma, cough heart troubles, gastritis	8	3	5	All under 3 years of diarrhoea.
2	46	no findings	5	4	1	unknown
3	44	Cervical glands	11	6	5	died at early age.
Total:	3		24	13	11	

TABLE D: INFANT MORTALITY IN THE GOLDI COLONY DERKHI.

Married Fem. No.	Age	State of health.	CHILDREN birth	living	died	Cause of death.
1	37	Anaemia Ulcus ventr.	8	5	3	All died of eclampsia
2	38	Heavy headaches, Gumma destr. palat. Cough with blood	2	1	—	1 abortion.
3	36	Pains over the chest Susp. quoad. Tbc.	6	2	4	All died of "bloody cough" among them a 13 years old girl!
4	30	Luet. anamnesis	—	—	—	—
5	35	Without findings	6	3	3	One died 4 years old of "troubles from the throat".
6	46	Without findings	5	4	1	Diarrhoea.
7	25	Anaemia; no findings	4	4	—	—
Total:	7		31	19	11	

I also began the examination of the Solons—a small Manchurian tribe of the Tungusian group, living in the mountain regions of the Khingan. I found it only possible to examine the village Charithun situated on the River Munli 90 km. northwards from Nunkiang. Their language is so like that of the Sungari goldis that the latter could fairly understand them. The tribe numbers about 1000. They seem to be healthier, stronger and more active than those of the Nabojé tribe. Out of 20 people investigated only one suffered from Tuberculosis. The infant mortality also was high, and it was surprising that in the village of about 60-70 people there were only 4-5 children.

TABLE E: INFANT MORTALITY IN THE SOLON VILLAGE CHARITHUN NEAR NUNKIANG.

Married Fem. No.	*Age*	*State of health.*	*CHILDREN*			*Cause of death.*
			birth	*living*	*died*	
1	59	Healthy	10	2	8	?
2	39	Pregnant, healthy	3	1	2	One child of fracture of the mandibula.
3	22	Healthy	1	—	1	Smallpox.
4	23	Without pathol. findings	1	—	1	Died one month old.
5	21	Without pathol. findings	1	—	1	Died 2 weeks old.
6	30	Irregular menstruations	—	—	—	—
Total :	6		16	3	13	

3. STUDY AND INVESTIGATION OF WILD RODENTS AND THEIR BLOOD PARASITES.

Systematic investigations of rodents were performed only at Sansing while at other places these were collected and preserved for future identification.

In the fields at Sansing I was able to obtain four kinds of rodents :

1. One small striped yellow rodent belonging apparently to the subgenus Microtus (a vole).
2. One big black vole, looking like a combination between a hamster and a house rat (see photo).
3. One brown field mouse with one narrow black sagittal stripe (Micromys agrarius Pall?) and
4. One big mole rat of ash grey colour.[2]

The small hamster-like voles (Rodent 1 and 2) live mostly in the trenches situated between the cultivated fields where they have almost invisible holes under the high grass. The mole rat (*Spalax*) and the field mouse (*Micromys*) captured and investigated in large numbers did not show in their blood any parasites. The voles on the other hand suffered much from Bartonella-and Trypanosoma infection.

[2] Dr. Koller of the Vienna Museum of Natural History has kindly determined species 1, 2 and 3 as follows :

1. *Cricetulus furunculus* Pall.
2. *Cricetulus arenarius* Pall.
3. *Siphneus aspalax* Pall.

The burrows of these voles are not complicated: There is usually only one exit and one passage leading to the dormitory which is situated 50-70 cm under-ground, and contains dry grass and seeds from the fields. From here a blind passage leads upwards, the latter showing sometimes some ramifications.

A short description and measurement of the two infected voles may be mentioned:

Rodent No. 1. A small hamster-like vole. Back and sides brown to reddish yellow. Belly white ash-grey. On the back, beginning behind the head a dark, not very sharply limited and not entirely straight line of black hairs leading to the tail. The breadth of this black stripe is 2 mm. The whole tail is covered with dense downy hairs. The rings on the tail are not visible. The upper aspect of the tail is black, the lower white. The fur although dense and long haired, is exceedingly tender and soft. The frontal-nasal line is regularly curved. Ears oval, rather big, covered with black hairs with white marginal border.

Measurements: (adult female) Total length:	12'3 cm.
Tail:	2'0 cm.
Pupillar distance:	1'4 cm.
Distance between tops of ears:	2'4 cm.

Upper incisivi light yellow, lower incisivi white, thin and long.

Rodent No. 2. Back: greyish-black, belly: grey-silver white. Thickset, rather short body. Head very thick, legs short and clumsy. Tail rather short, getting thinner towards the tip. Around the base of the tail a dense, downy ring of hairs, from which the short, bald tail arises. The shed-rings of the tail indistinct, very narrow, without any structure.

Measurements of adult male: total length:	22 cm.
Top of nose to base of tail:	16½ cm.
Tail:	5½ cm.
Circumference of chest:	10 cm.
Distance between the tops of the ears:	4 cm.
Pupillar distance:	1'8 cm.
Distance between top of nose and upper incisivi: ...	0'6 cm.

The anterior surface of upper and lower incisivi is dark yellow. The well-developed cheek pouches make the head very thick, clumsy and hamster-like.

A. TRYPANOSOMIASIS AMONG SANSING VOLES.

Both the small yellow and the larger black Sansing vole were found suffering from trypanosomiasis.

Altogether there were investigated 22 recently caught animals: 17 small yellow, and 7 bigger black ones.

The following table shows the result of microscopical investigation of the blood taken from the tip of the tail:

a. Small yellow rodent No. 1.	Trypanosoma.	Bartonella.
specimen 1	+	+
2	+	−
3	+	+
4	−	−
5	−	−
6	−	−
7	−	−
8	−	−
9	−	−
10	−	−
11	+	+
12	+	−
13	−	−
14	−	−
15	−	−
16	−	+
17	−	+
b. Big black vole Rodent No. 2.	Trypanosoma.	Bartonella.
Specimen 1	+	+
2	−	−
3	−	+
4	−	+
5	−	−
6	−	−
7	−	−

The percentage of infected individuals is rather high: Among 17 specimens of the smaller yellow type of rodents 5 were infected with trypanosoma, i.e. 29'4%, out of 7 specimens of the big black vole one proved to be infected (14'3%).

The number of trypanosomes in the blood is sometimes considerable. Especially when the blood is taken from the heart at some spot of the smears two or more trypanosomes can be seen in almost every field of vision.

The trypanosoma has the following characteristic features[3]: The body is comparatively slender, breadth almost never exceeds 1¾ mikrons. It is therefore considerably thinner than other kinds of trypanosomes, being closely related to the trypanosome of the Sansin voles. (See following table).

Regarding length it is difficult to draw any conclusion, because many specimens are much curved. The length measurements were taken by me in this way:

The single trypanosoma to be measured was placed under the micrometer-ocular, and by means of rotation the maximal distance was deter-

[3] For comparison of the measurements I used mainly Boshenko's table from the article "The trypanosomes of the susliks" in the Vjestnik Mikrobiologyi, Saratov 1927, VI p. 166. This author performed the conversion of ratios of different rodents' trypanosoma, : thus the Trypanosoma spermophili, trypanosoma lewisi and the Tr. criceti.

mined, covered on the scale by the trypanosoma. When the single distances were determined: 1. posterior end-blepharoplast, 2. blepharoplast—posterior border of the trophonucleus, 3. length of the trophonucleus, 4. anterior border of the nucleus—Anterior extremity (end of the flagellum) and these measurements were summed up, higher proportions were always obtained.

The posterior (aflagellar) end is sometimes markedly pointed and somewhat long. The distance between the aflagellar end and the blepharoplast is longer than that of the other investigated rodent trypanosomes. Out of 10 specimens measured carefully only one showed a length less than 4 microns. 3 measured more than 5 microns, and one up to 7 microns for this distance. The distance: "Blepharoplast—posterior wall of the nucleus" on the other hand is rather short, as a rule not exceeding 6 microns, while in the trypanosoma spermophili this distance amounts more than 9 microns.

The nucleus is small; on an average 2¼ microns long. Chromatoid granules are not seen distinctly (All smears were stained after Giemsa's method). The flagellum gradually becomes free, and it is often impossible to determine exactly the spot where the free flagellum begins. The posterior part of the trypanosoma (from the posterior border of the nucleus to the flagellar extremity) is on an average longer than that of the trypanosoma lewisi, and shorter than of trypanosoma spermophili and Tr. criceti.

The following table gives the detailed measures of the Sansing trypanosoma compared with those of Trypanosoma spermophili (from Citellus musicus, Cit. mugosaricus, and Cit. fulvus from South Russia), Trypanosoma lewisi (from Rattus norvegicus), and Trypanosoma criceti from Cricetus cricetus of Dyskovka (South Russia).

According to this measurement it seems that we have to deal with a new species, to which temporarily the nme Trypanosoma Sansingense may be given.

Table: Comparision of the Tr. spermophili, lewisi, and criceti with the Sansing Trypanosoma. All measurements in microns.

Distance:	*Sansing Trypan. Average.*	*Trypan. Spermophili Average.*	*Trypan. Lewisi Average.*	*Trypan. Criceti Average.*
1. Posterior end-blepharoplast.	4'02-7'0 *4'31*	2'3-5'85 *3'12*	3'1-4'65 *3'82*	3'1-6'2 *4'8*
2. Blepharoplast posterior border the trophonucleus.	5'25-7'0 *6'03*	7'75-10'85 *9'33*	7'3-9'5 *7'98*	6'2-7'75 *6'52*
3. Length of the trophonucleus.	1'75-2'62 *2'25*	1'7-3'1 *2'8*	7'3-9'4 *7'98*	6'2-7'75 *6'52*
4. Anterior border of the nucleus-end of the flagellum.	10'5-16'2 *13'2*	12'4-22'2 *17'12*	10'05-16¾ *12'84*	9'3-20'15 *14'17*
5. Breadth.	1'92-2'62 *2'15*	1'55-3'1 *2'53*	2'3-3'85 *2'85*	2'32-3'1 *2'97*
6. Total length.	22'7-34'5 *25'6*	30'2-35'8 *33'4*	23'3-31'1 *27¾*	27'1-32'5 *29¾*

The trypanosomes of rats (Tr. lewisi) are generally considered to be non-pathogenic. Anyhow, according to the experience of Boshenko on suslik trypanosomes, the animals infected with them succumb earlier in captivity than the healthy ones. I also made a similar observation : at the end of May after concluding the investigations at Sansing in order to start for Lahasusu, I sent 22 living specimens of the two kinds of rodents susceptible for trypanosoma to Harbin, as I intended to continue the experiments after my return from the excursion. Four out of the 22 animals were infected with trypanosomes. After my return in August there survived only one animal; the other three had died. Of the healthy animals there died only a small percentage. But the high mortality among the trypanosomes infected voles may be traced also to the mixed infection with Bartonella, because only the animal infected with trypanosoma, survived, while the other three voles, suffering from both Bartinella and Trypanosomiasis succumbed.

Experiments with transmission of the trypanosomiasis on other voles and rodents are now in operation and will be dealt with in future.

B. THE BARTONELLA INFECTION AMONG SANSING VOLES.

It is well known that Bartonella bodies are present in the blood of normal rats and certain small rodents such as, moles (*Spalax*) and gerbilles (*gerbillus*). These bodies as a rule occur in the blood stream in such small numbers that they evade observation. But after splenectomy of the rodent these bodies rapidly multiply by producing anemia and death.

Dudshenko who studied the blood of Transbaikalian rodents, describes these blood parasites in the erythrocytes of small hamsters living near Chita. (*cricetulus*). This author says that 25% of this rodent are infected with Bartonella.

M. Zuelzer, who investigated the field mice (*arvicola arvalis*) near Potsdam in Germany found that in some districts 80-90% were infected with Bartonella.

In Summer 1927 while investigating Mongolian rodents near Urga (around the Rinderpest station at Sangin), I found that the spring hares (*Alactaga mongolica*) often harbour Bartonella in their erythrocytes in more or less numbers. Spring hares infected on a larger scale show also in their blood well developed poikilochromatosis and die soon after having been captured.

The *Alactaga mongolica* sometimes harbour in addition in their erythrocytes a larger number of *hepatozoon spec.* which accumulate and block the small capillaries of the lung. These infected erythrocytes become pale and oval-shaped when harbouring the parasite, which lies along the greatest axis; when deeply affected, the cells become mere shadows, so that the elongated parasites seem to lie freely in the blood stream.

In autumn of 1927 I examined the blood of large numbers of small hamsters (*cricetulus spec.*) living around the Rinderpest station, and found a high percentage with the bacilliform bodies of the Bartonella in

their erythrocytes. Out of 29 small hamsters investigated 17 were positive, that is about 60%, while in 12 these blood parasites could not be demonstrated in the smears after one microscopical examination. The percentage of the really infected animals is probably larger than 60%, as the bartonella, according to my experience, sometimes disappear from the blood stream and reappear after some time. Thus while investigating the blood from the tail of a *cricetulus spec.* I once found a considerable number of bartonella-containing erythrocytes (2-3 almost in every field of vision). In order to study this infection histologically I killed the animal 5 hours afterwards but this time I found the organism only sporadically in the blood (one infected erythrocytes in every 200-300th field of vision).

A close connection seems to exist between the development of the poikilochromatosis and the bartonella : My few specimens of the cricetuli, free from poikilochromatosis, never showed in their blood any bartonella, while all severely affected with poikilochromatosis showed the parasites.

Other Mongolian rodents, the rat hare (*Ochotona*), the sisel (*spermophilus*) and the tarabagan (Arctomys), although being sometimes affected with poikilochromatosis, never showed these blood parasites in red cells.

I also detected at Sansing the rod-shaped bacilliform bodies in May 1928 in the erythrocytes of 2 species of voles living on the fields in the outskirts of the town (rodent No. 1, 2).

The percentage of the infected individuals among the voles is high : Among 17 specimens of the smaller yellow vole (rodent No. 1) five, i.e. 43%, proved infected. The number of bodies included in the infected red blood corpuscle is as a rule considerably high. The erythrocyte is often so full of them that it is very difficult to count them. The rod-shaped forms of the blood parasite are straight or slightly curved. When numerous, they often lie parallel to one another : sometimes they also show radiary arrangement, being concentrated near the border of the erythrocyte. When there are only few in the erythrocyte, they remind one in their morphology and arrangement of Bac. diphtheriae, showing also more intensively coloured ends (metachromatous granules or polar stainings), while the central part of the microorganism is considerably paler. When the rods are longer than usual, there may appear in the centre some granules, thus giving the appearance of a chain. The affected red blood corpuscle is as a rule not enlarged but often shows poikilocytosis while the bodies project out from the border. Finally, the erythrocyte is entirely destroyed, and in its place only a dense round-shaped mass is seen. Then these rods swim out, and single individuals enter a healthy erythrocyte.

While comparing the bartonella from different rodents, I found some morphological differences.

The bartonella of the Mongolian spring hare (*Alactaga mongolica*) usually occurs in large numbers in the red blood corpuscle; on an average one crythrocyte harbours 30-40 and more rods. The single individuals

are short and thick. On the other hand, the bartonella of the small hamster is somewhat long and slender. The erythrocytes as a rule harbour a small number, those with 40 and more rods being comparatively rare. Here the morphology of the single individual can be studied best. The bartonella of the Sansing-voles is found in large numbers in the blood corpuscles (on an average 40 and more). They are shorter, and resemble more those in spring hare.

Although hamsters affected with bartonella (from Mongolia) lived more than a year in captivity before finally getting rid of their parasites, it seems to me that this disease is rather serious and sometimes fatal for the rodent, producing a severe blood disease, like pernicious anemia.

Especially serious is a mixed infection with the hepatozoon as found in the Mongolian spring hare, or with trypanosoma as in the Sansing voles: Three voles, infected with both bartonella and trypanosoma, succumbed soon after capture by us.

4. STUDY OF THE PREVALENCE OF MALARIA IN THE TERRITORY VISITED.

For this kind of investigation the season was not favourable. At Sansing, at the end of May, I collected only some specimens of culex although I was told that on the mountains surrounding the town there is a considerable number of mosquitoes appearing in June and multiplying in July to such a high degree that it is not advisable to camp out.

At Lahasusu in June there was already a considerable number of mosquitoes in the marshes bothering one in the evening and night time. But they were all different kinds of culex or other closely related genera, and I was at first unable to obtain any anopheles, although I examined carefully the houses and stables in Lahasusu and the villages surrounding the town. This was surprising for me as at the same season (June) at Harbin I repeatedly found in the houses of New Town specimens of Anopheles sinensis.

At the end of June there came into our Lahasusu hospital a young Chinese colonist from the neighboring village of Lasheho, who recently contracted *Malaria tertiana*. His blood contained a large number of plasmodium vivax. According to the anamnesis he was never ill before, and never left the village or its environs the whole time before he fell sick.

I made an excursion the next day to the village Lasheho in order to investigate the houses, stables for anophelines. I failed, although I tried to examine every place. The inhabitants of the village also refused to undergo the blood investigation.

Some days afterwards I went to Kaidza village. Returning from this trip, at evening time,—between Kaitikaotzu and the village Althun (not far from Lasheho)—we were attacked by clouds of mosquitoes, all culex. I detected among them one anopheles, and afterwards five more. The place where I found the anophelines was in the marshes of the small Kaidza river, and far away from houses. (The nearest hut was about 5 km. distance!). Three specimens were caught, all belonging to one

species, probably anopheles sinensis.[4] Immediately afterwards I obtained hundreds of mosquitoes and investigated them at home, but no anopheles was present.

The region eastwards from Lahasusu (between the mouth of the Sungari and the Kaitza river) has therefore to be considered as a malaria focus.

Another time, during the journey from Lahasusu to Taheiho (early July) I founl on the steamer one specimen of a female anopheles, probably of the same kind. Where this mosquito embarked, remains unknown, probably already at Harbin.

This finding deserves attention because it shows, thanks to the present steamship traffic, how quickly different kinds of insects, and with them diseases, may spread throughout the Province.

During my journey via Nunkiang to Tsitsikar I only found various kinds of culex but no anopheles.

5. MISCELLANEOUS KNOWLEDGE REGARDING NATURAL HISTORY.

I endeavoured to collect zoological and botanical material wherever there was any possibility.

At Sansing a large number of rodents (4 kinds) were preserved and sent to Harbin Museum. The ectoparasites on them also have been collected and sent to an expert for determination.

At Lahasusu there was collected a large number of different mosquitoes and blood sucking flies. A herbarium containing the phanerogamina blooming in June in the marshes near the mouth of the Sungari river has been collected consisting of 105 species with 234 specimens. Besides, about 100 kinds of beetles (200 specimens) were caught and preserved. Finally mosquito larvae and other insects (butterflies, flies of prey, dragon flies and so on) have been preserved in the Harbin Museum.

On the trip from Taheiho to Nunkiang and in the surrounding country of Nunkiang I collected rodents, bats and their ectoparasites, a herbarium, containing the phanerogamina blooming in July in the mountains of the small Hingan and near Nunkiang, consisting of 69 species with more than 200 specimens. There have been collected butterflies and beetles, also a large number of tabanidae (7 different species and 50 specimens).

Some of the material have been sent already to proper experts for determination, others are kept in the Harbin Museum for further identification.

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[4] The mosquitoes have been sent to Europe for determination.

第四十一圖 民國十七年防疫醫官在錢家店臨時防疫醫院攝影（前係客店）用泥草蓋造

Fig. 41. showing antiplague staff outside temporary plague hospital (formerly village inn), Chien Chia-tien, Oct. 1928. Note the mud and straw construction.

Fig. 42. Rodent I from Sansing (Cricetulus furunculus).

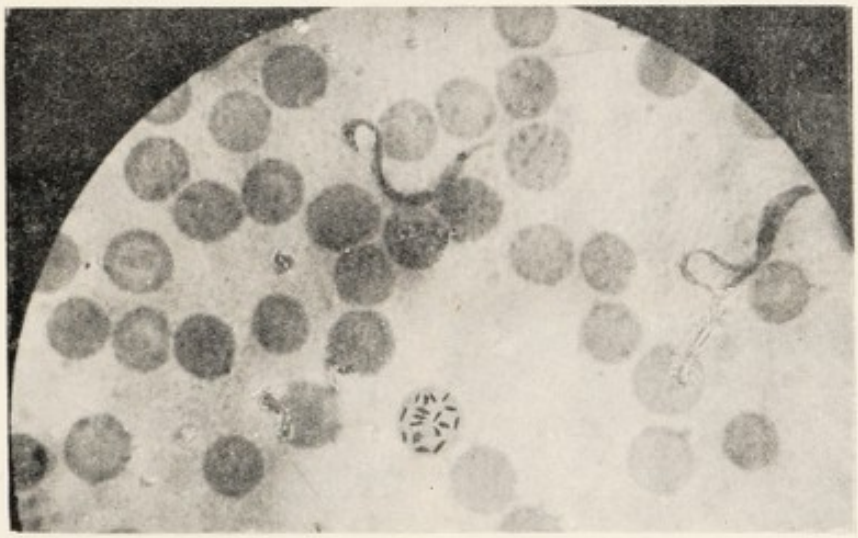


Fig. 43. Microphoto of blood of Rodent I showing both Trypanosomes (2) and Bartonella (1) High magnif.

Fig. 44. Rodent 2 from Sansing (Cricetulus arenarius).

齧齒動物甲產于三姓（米古勞篤斯類）圖四十三號卽齧齒動物甲血液顯微照像示篤利巴雖疏米及巴篤尼來原虫（强擴大）

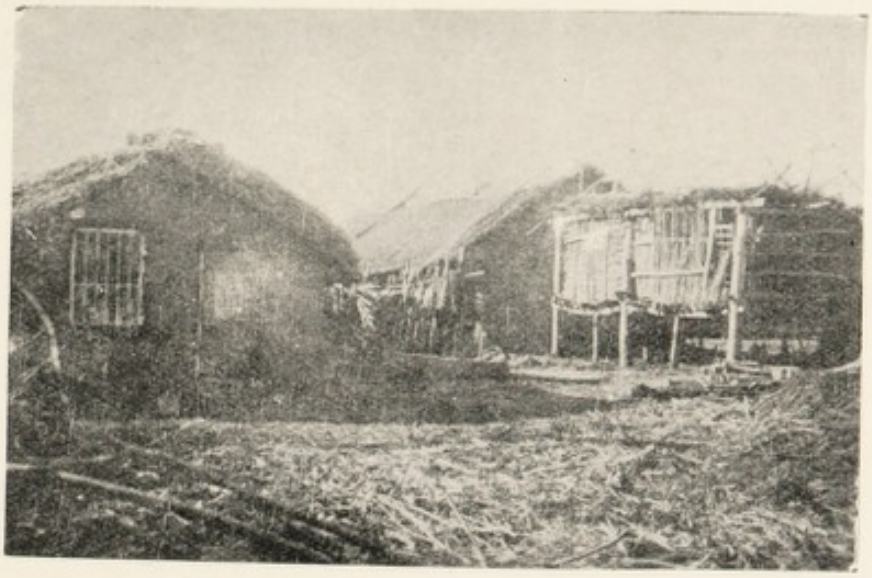
圖四十四齧齒動物產于三姓（米古篤斯類）

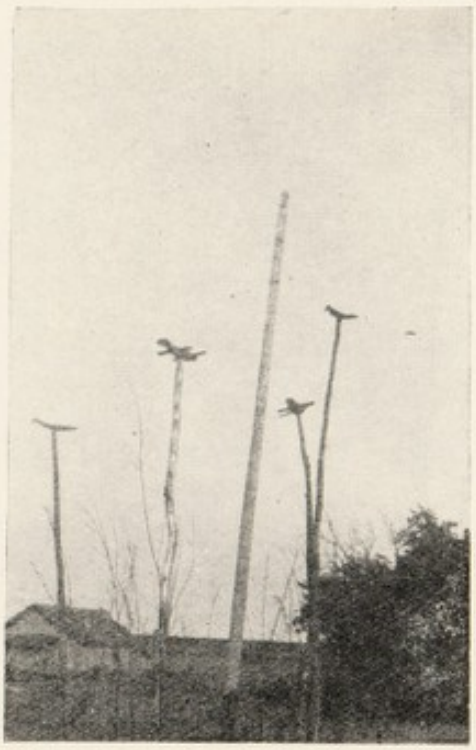
Figs. 45-51. Types of Goldi (Nabojé) tribe Yu Pi T'a Tzu.

示哥兒的族（拿坡治）卽中國俗稱魚皮韃子是也

Figs. 52-55. Above three indicate Goldi types, Bottom : the Solon village Charithun.

上三圖示哥兒的民族下一圖示蘇倫村境況





Figs. 56-59. Top and bottom show houses of Goldi. Center left: Solon woman Center right: wooden idols on top of poles (Nabojé village Tusheho).

上下兩圖示哥兒的住戶中左圖示索倫女子
中右圖每桿之頂有木偶（都土河之拿坡治村）

Figs. 60-62. Above, a dancing shaman (priest), Chichikha; center, wooden idols under cave of hut (Tusheho); buttom, a Goldi rowing boat on Amur river (near Chai).

上圖示沙爾（道士）跳舞芝芝哈 中圖在茅洞內之木偶（都士河）
下圖爲哥兒的在阿櫻爾河駛行之小艇

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PROBLEMS OF CHOLERA IN CHINA AND JAPAN.

(*With an Appendix*).

The importance of the cholera problem in the Orient cannot be over-emphasised. The vast Empire of India is notorious as the original home of this fatal infection, and scarcely one year passes, but thousands of cases are recorded in some part or another of this vast dependency of Great Britain. As we travel eastwards, we find constant outbreaks in Burmah, Siam, French Indo-China, South and Central China, in most of which endemic centers probably exist.

If we take the year 1926 (a rather bad year for cholera) we find:

a. India recording at least 72,859 deaths (actual cases probably over 150,000).

b. Siam recording 10,655 cases.

c. French Indo-China with 19,029 cases (13,925 fatal).

d. Philippines with 690 cases (in 1925 there were 1059 cases).

Japan with her fine quarantine system was successful in keeping the disease at bay, and reported only 26 cases in that year.

Korea, in spite of a vigorous campaign, had 252 cases with 159 deaths.

South and Central China were badly affected in 1926, serious outbreaks having been reported in almost every city in the Yangtze Valley, such as, Nanking, Soochow, Wusieh, Huchow, Anking, Hankow, Wuchang, etc., besides cities further south, such as, Foochow, Amoy, Swatow, Canton, Hainan, etc. In Shanghai district there were at least 20,000 cases, the two Chinese Summer Disease Hospitals admitting in the week August 1-8 2481 cases.

In Manchuria, which is in close relation with both China Proper and Japan, thanks to close co-operation between the Chinese and Japanese Health Services, only 1500 cases occurred in 1926 as compared with over 10,000 in the previous cholera year of 1919, when no such co-operation existed. It can be definitely said that both in Manchuria and Japan, there are no endemic centers, all outbreaks occurring being traceable to importations from the south.

The task of combating cholera in the Far East is obviously of a two-fold nature.

(1) Offensive measures to be taken in the endemic centers.

(2) Defensive measures in areas threatened by importation of the infection.

Solid progress can only be hoped for if the actual fight against the disease in endemic centers is everywhere taken up with equal energy and efficiency. Defensive measures, on the other hand, adopted in cholera-free areas, must be reduced to the absolutely necessary minimum in order not to interfere unduly with commerce and traffic.

Let me concentrate at present upon the defensive measures. The weapons recommended by the International Sanitary Convention of 1926

may first be scrutinised. From the articles dealing with cholera (see Appendix) it may be gathered that the Convention gives vast powers to the sanitary port authorities in the case of *cholera infected* ships, but limits them in the case of *cholera suspected* ships and still more in the case of *healthy* ships. In fact according to Article 33 free pratique should immediately be given to such vessels. Article 34 finally emphasizes anti-cholera vaccination as a specially reliable preventive measure.

The Japanese Delegates at the 1926 Paris Conference, while signing the Convention, reserved the right for their Government "to take all measures considered as necessary by the sanitary authorities against carriers of the *V. cholerae*." Therefore it is clear that the intention of the Japanese authorities is to continue enforcing rigorous measures for the detection of carriers among ship passengers. The procedure adopted by them may be shortly described as follows:

1. Enforced defecation by all members of crew and passengers on the morning of arrival, and collection of small samples in little Petri dishes.
2. Placing of small particle of specimen with sterile tooth-pick in test tube containing peptone water.
3. Handing over of all the peptone cultures (4-6 hours old) in racks of thirty tubes each to the quarantine authorities.
4. Making of films (five on one slide), drying over a spirit lamp, staining with dilute carbol fuchsin for ½ min., drying between filter paper without washing in water, then examining under the microscope.[1]

Apparently quite apart from their reservation, the Japanese authorities are technically entitled to enforce these rigorous methods (see end

[1] In this way, Dr. Yano of the Naval Quarantine Service could with the aid of another trained doctor assistant inspect six slides, that is 30 specimens, in one minute. While I was in the Laboratory the examination of 280 specimens took just 40 minutes from the time of arrival of the peptone cultures to the final report by telephone to the shipping authorities. In addition to Dr. Yano, there were two medical assistants and six technicians in August 1926 undertaking these fecal examinations for steamers arriving from Shanghai.

of Art. 29 and also Art. 15 of the Convention.) [2] The fact that stool examinations are not mentioned in the Convention (as contrasted with the recommendation of vaccination) and that the Japanese Delegates reserved the right of their Government to take steps in this direction, however, shows that in other quarters the necessity or perhaps even the value of these stool examinations is doubted. At the FEATM Conference in Calcutta (December, 1927), Colonel Graham, Sanitary Commissioner for India, strongly spoke against the stool examinations as enforced by the Japanese Health authorities. Thus it may be said that there are two schools of thought, one relying mainly upon vaccination, the other regarding stool examinations as indispensable.

Let us try to consider these two views impartially. We all agree that anti-cholera vaccination "is a method of proved efficacy in staying cholera epidemics and consequently in lessening the likelihood of the spread of the disease" (1926 Convention, Art. 34). Another question is whether vaccination, even when carried out on a large scale, will be instrumental in doing away with carriers of the cholera germ. Opinions on this point are rather divided. Authors like Frank & Wengraf, Erdheim and Schopper, etc. believe that the number of carriers in an epidemic is diminished by vaccination; Rogers records an instance where "in a camp of some 3,500 Servian prisoners, inoculation during an epidemic produced no bad effect, but on the contrary this measure brought the disease to an end from 8 to 14 days, while the number of carriers rapidly fell to zero." On the other hand, it is on record that not rarely vaccinated persons become carriers (Kaup, Nomura, Sotoma and Harata). To hope, therefore, that vaccination, even when universal, will abolish the carrier problem, would be vain. Carriers will probably be diminished in number, but at least some will remain.

Turning now to the stool examinations, we must confess that apart from its inconvenience to individuals and the delay to traffic it causes, this method is by no means free from sources of error. I am ready to admit that some of these errors may be obviated by great care and efficiency of the sanitary staff. Such are :

(1) *Prevention of frauds.* Certainly it would be ideal to obtain rectal swabs from each individual, but this would entail so much inconvenience and delay as to be extremely difficult on a large scale. If defecation is left to the individual, great care is necessary to prevent fraud.

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[2] Article 15—Any ship, whatever its port of departure, may be subjected by the sanitary authority to a medical inspection, and if circumstances require it, to a thorough examination.

The sanitary measures and procedure to which a ship may be subjected on arrival shall be determined by the actual condition found to exist on board and the medical history of the voyage.

It rests with each Government, taking into account the information furnished under the provisions of Section I of Chapter I and of Article 14 of this Convention, as well as the obligations it has undertaken under Section II of Chapter I, to determine what procedure is applicable in its own ports to arrivals from any foreign port, and in particular to decide whether, from the point of view of the procedure to be applied, a particular foreign port should be considered as infected.

(2) *Evaluation of results.* Where a skilful staff is present, there should be little fear of vibrios (if present) being overlooked in the stress of microscopic examination. In this connection, it is more likely that cholera-like vibrios may lead to the detention of harmless passengers.

There is, however, one unavoidable obstacle. As established by many authorities, e.g. by Heiser, Greig, Yagasaki, Noda, Nomura and Harata, carriers may not void cholera vibrios with each stool and not even on each day. In other words an examination of a single specimen does not entitle us to consider an individual as a non-carrier. Interesting data in this regard were supplied by Amata, Nagawo, Nakamura and Hasegawa among 391 persons quarantined as contacts during the 1922 epidemic at Tokyo, where 33 carriers and 4 cases were found. Of these 37 instances there were detected ·

Through one examination	17
Through two examinations	5
Through three examinations	12
Through four examinations	2
Through five examinations	1
	[3]
	37

Consequently, in order to be sure that no carrier is missed, it would be necessary to examine travellers not once, but at least two or three times. To detain passengers of a healthy ship for such a prolonged period would be absolutely against the spirit of the 1926 Convention.

In conclusion we may say therefore that neither vaccination nor stool examination can be considered as absolutely efficient. Both methods have their weak points as well as their technical difficulties. It would be vain to hope that either will absolutely prevent importation of the disease. Under these circumstances it is clear that defensive measures alone are insufficient to protect a country against the importation of cholera, and one must necessarily pay more serious attention to an active campaign against the disease in neighbouring endemic centers. In the case of China and Japan such measures would comprise :

(1) An intensive study of the endemic areas in the Yangtze Valley.
(2) Removal of causes wherever possible, e.g. control of water works, sterilisation of wells by chemicals, etc. (A promising beginning in this direction has been made by the Shanghai Chinese Public Health authorities).
(3) Mass vaccination in the endemic areas; vaccination of each passenger leaving for Japan or Manchuria.
(4) Fullest co-operation among the two health authorities in the spirit of mutual confidence and helpfulness.

[3] The Japanese references are mainly gathered from "Studies of Cholera in Japan" by Rokuro Takano, Itsuya Ohtsubo and Zenjuro Inouye, Geneva, 1926.

APPENDIX.

INTERNATIONAL SANITARY CONVENTION, 1926.
CHOLERA.

Article 29.—Infected Ship.—A ship shall be regarded as infected if there is a case of cholera on board, or if there has been a case of cholera during the five days previous to the arrival of the ship in port.

Suspected Ship.—A ship shall be regarded as suspected if there has been a case of cholera at the time of departure or during the voyage, but no fresh case in the five days previous to arrival. The ship shall continue to be regarded as suspect until it has subjected to the measures prescribed by this Convention.

Healthy Ship.—A ship shall be considered healthy, although arriving from an infected port or having on board persons proceeding from an infected area, if there has been no case of cholera either at the time of departure, during the voyage, or on arrival.

Cases presenting the clinical symptoms of cholera, in which no cholera vibrios have been found or in which vibrios not strictly conforming to the character of cholera vibrios have been found, shall be subject to all measures required in the case of cholera.

Germ carriers discovered on the arrival of the ship shall be submitted after disembarkation to all the obligations which may be imposed in such a case by the laws of the country of arrival on its own nationals.

Article 30.—Cholera Infected Ships.—In the case of cholera, infected ships shall undergo the following measures :—

(1) Medical inspection;

(2) The sick shall be immediately disembarked and isolated;

(3) The crew and passengers may also be disembarked and either be kept under observation or subjected to surveillance during a period not exceeding five days reckoned from the date of arrival.

However, persons who can show that they have been protected against cholera by vaccination effected within the period of the previous six months, excluding the last six days thereof, may be subjected to surveillance, but not to observation.

(4) Bedding which has been used, soiled linen, wearing apparel and other articles, including foodstuffs, which in the opinion of the sanitary authority of the port, have been recently contaminated, shall be disinfected;

(5) The parts of the ship that have been occupied by persons infected with cholera or that the sanitary authority regard as infected, shall be disinfected;

(6) Unloading shall be carried out under the supervision of the sanitary authority, which shall take all measures necessary to prevent the infection of the staff engaged in unloading. The staff shall be subjected to observation or to surveillance which may not exceed five days from the time when they ceased unloading,

(7) When the drinking water stored on board is suspected it shall be emptied out after disinfection and replaced, after disinfection of the reservoirs, by a supply of wholesome drinking water.

(8) The sanitary authority may prohibit the emptying of water ballast in port without previous disinfection if it has been taken in at an infected port.

(9) The emptying or discharge of human dejecta, as well as the waste waters of the ship, into the waters of the port may be forbidden, unless they have been previously disinfected.

Article 31.—Cholera Suspected Ships.—In the case of cholera, suspected ships shall undergo the measures prescribed in (1), (4), (5), (7), (8) and (9) of Article 30.

The crew and passengers may be subjected to surveillance during a period which shall not exceed five days, reckoned from the arrival of the ship. It is recommended that the crew be prevented during the same period from leaving the ship except on duty notified to the sanitary authority.

Article 32.—Clinical Cholera.—If the ship has been declared infected or suspected on account only of a case on board presenting the clinical features of cholera, and two bacteriological examinations, made with an interval of not less than 24 hours, between them, have not revealed the presence of cholera or other suspected vibrios, the ship shall be considered healthy.

Article 33.—Healthy Ships.—In the case of cholera, healthy ships shall be given free pratique immediately.

The sanitary authority of the port of arrival may prescribe as regards these ships the measures specified in (1), (7), (8) and (9) of Article 30.

The crew and passengers may be subjected to surveillance during a period which shall not exceed five days reckoned from the date of arrival of the ship. The crew may be prevented during the same period from leaving the ship except on duty notified to the sanitary authority.

Article 34.—Since anti-cholera vaccination is a method of proved efficacy in staying cholera epidemics, and consequently in lessening the likelihood of the spread of the disease, sanitary administrations are recommended to employ, in the largest measure possible and as often as practicable, specific vaccination in cholera foyers and to grant certain advantages as regards restrictive measures to persons who elect to be vaccinated.

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THE SIGNIFICANCE OF SCARLET FEVER IN CHINA.

During recent years the problem of scarlet fever, especially in North China and Manchuria, has become acute, and it is felt that popular attention drawn to the menace of this disease, both among Chinese and foreigners residing in this country, might help to limit its spread among the community.

It is generally understood that scarlet fever is a disease of temperate climates, being almost unknown in the tropics and seldom seen in sub-tropical countries. As China (including Manchuria) lies between 20° latitude in the south (Hainan) and 54° latitude in the north (Aigun), variations may be expected in the incidence of this communicable disease. A few years ago I sent out a questionnaire to physicians scattered in various parts of China, asking for detailed information as to its prevalence. From information thus gathered, I came to the following conclusions :

1.—It is practically absent or very mild in South China, fairly severe in Shanghai and the Central Provinces, and very severe in the North. Regarding Hongkong during the years 1911-22, out of 14,493 cases of infectious diseases, only 41 were classed as scarlet fever: 10 of these were among white people. During the same period 7,090 cases of bubonic plague, and 3,039 of smallpox were encountered.

2.—The disease generally seems to be more frequent among Westerners residing in the country. In the north, although several deaths are yearly recorded among them, the infection assumes greater severity among similar classes of Chinese.

3.—The special virulence of the disease as manifested when Chinese are attacked in an epidemic form, may be explained partly by its recent introduction in the country, before any natural immunity has yet evolved among the population. The tendency in Europe and America during the last decade is for the infection to appear in a mild form.

A few words regarding its history in the Far East may not be amiss. Although in Europe it is of great antiquity, having been described by Galen (131-201 A.D.), and a careful account given by the celebrated English physician Thomas Sydenham as far back as the 17th century, in China little or nothing was known of scarlet fever until 1873, when the first authentic case was recorded in Shanghai. Dr. Arthur Stanley (former Health Officer of the International Settlement there) in an able review in 1917 said :

> "The first recorded death from Scarlet Fever in the Foreign settlement of Shanghai was in 1873. It seems probable that the infection was imported. At about this time, cases are believed to have been reported at Chefoo. The occurrence of Scarlet Fever in Japan appears to have been officially notified in 1897, but it is

probable that a few cases occurred prior to this. Indeed, Scarlet Fever appears to have reached Japan and China at about the same time and to have been previously unknown. By the year 1902 there had been introduced into Shanghai a quantity of infection sufficient to gather epidemic momentum, and the Chinese death record from Scarlet Fever in that year, 1500, does not appear to have been exaggerated."

Stanley adds that four scarlet fever cases were imported from England into Hongkong in 1898, previous to which time the disease had been "practically unknown" there.

The admittances into the Shanghai Isolation Hospital for various years are :

	1902-1921		1922-1925	
	Foreigners	Chinese	Foreigners	Chinese
Cases	761	1,071	203	261
Deaths	118	270	13	57
Case Fatality	15.5%	25.2%	6.40%	21.85%

The lessened case fatality in the later period, especially among foreigners, is worth noticing.

Unfortunately, in Tientsin, Peking, Mukden, Harbin, in fact all cities in Chihli, Shantung and Manchuria, where careful records have been kept, scarlet fever seems particularly virulent among Chinese and produces a high fatality. In the Government Isolation Hospital of Peking, between 1915 and 1923 there were admitted 638 patients (352 male and 286 female) with a death rate of 20.8 per cent. In Tientsin for nearly fourteen years we have observed very severe cases among both wealthy and poor people. Owing to the widespread ignorance and superstition of the women folk, even after having undergone a general education, infection generally spreads from child to child in the same family until almost every one is attacked. The constant communication between the children and servants of the locality also helps to spread the disease broadcast. Even adults are often attacked as the following cases show :—

(*a*) An experienced Chinese old-style physician aged 62 attended in February 1923 the family of the late General Chang Hsun for scarlet fever. He himself developed high fever, sore throat, headache, swollen glands and, later, nephritis, etc. Within 36 hours he was speechless and delirious, but with stimulants and serum he gradually recovered after 4 months. A younger son, seeing the serious condition of the father, remained in the same room for most of the time and even slept in the same bed. Three days after, he showed all signs of a severe infection with vomiting, pneumonia, etc., and died the same evening.

A married daughter (*aet.* 28) who had been nursing the father, also became ill and returned to her house with shivering, headache, sore throat, dry cough and severe vomiting. Her temperature rose to 106 with pulse of 126, when the rash appeared. Serum was given and after three days the disease abated and she slowly convalesced. Her three children,

though isolated early, caught the infection, resulting in the death of the youngest.

(b) Among the Chang Hsun family in 1923, one adult woman and two children died, though several persons were sick.

A similar virulence was noted throughout the Tientsin district during January-February 1924, and should give a serious warning to those in charge of public health work regarding the need of establishing more hospitals for the proper reception and care of such cases. In the winter of 1926 the disease again destroyed large numbers of children.

In Harbin scarlet fever has caused much havoc during the last five years and is an important cause of death every autumn and winter. Sometimes families, rich and poor, lose all their children within a fortnight from this cause. Adults and children are often attacked together, but a considerable percentage of the former survive while the tender aged perish. Owing to dread of hospitalisation and insufficient knowledge of the value of isolation, it has been difficult for us to save lives, even where such were possible, or to keep accurate records of the incidence of the disease among the people. Of 16 families studied in 1926 when the disease prevailed, we found a very high death rate among the children. Thus :

	Adults		Children	
	Male	Female	Male	Female
Number	19	27	18	28
Died	0	1	9	20
% death	0.0	3.7	50.0	71.4

Dr. Iimura reported in 1925 to the League of Nations on the incidence of scarlet fever in Japan as follows :

"It was only in 1887 that the occurrence of this disease was recorded with any degree of certainty. After the war of 1894 its presence in Japan was confirmed.

"By the alteration of the Law for the Prevention of Infectious Diseases in 1897, scarlet fever was included among the notifiable diseases, and this made it possible to know the exact number of cases occurring each year. In that year the total was 37. The number increased to 125 in 1902, to 500 in 1907, and from 1909 to the present time the annual case incidence has never fallen below 1,000.

"Scarlet fever in Japan is purely sporadic. It varies little from year to year or from place to place. Its seasonal activities are most marked in November and December and to a lesser degree in the six months beginning with January and ending with June, August and September being the months of minimum incidence.

"The case mortality rates show a marked undulation of a very interesting nature. The following shows the death-rates from 1897 onwards.

	Percentage	Average
1897-1905 (9 years)	8.11- 9.46	9.22*
1906-1912 (7 ,,)	12.10-21.93	17.05
1913-1923 (11 ,,)	4.17- 9.23	6.24

* The year 1900 alone showed the extraordinarily high rate of 15.58.

"In Japan, children from four to seven years of age are most liable to this disease; youths up to twenty years are occasional sufferers. Very few cases occur among people over thirty years of age.

"Scarlet fever in Japan is of a very mild description. It has often occurred that the patient has only been made aware of his real ailment when seeking medical advice after the onset of nephritic symptoms."

Japanese records from South Manchuria show that in the years 1911-1919, 1249 cases with 153 deaths occurred (mortality rate 12.2 per cent.).

Since a knowledge of the signs and symptoms of scarlet fever is important for differentiating it from other infections, a short description of these may now be given.

The incubation period is short, varying usually from two to six days. The disease begins suddenly with fever, general weakness, pains in the throat, and vomiting. In typical cases all symptoms point to the coming of a serious illness. Examination of the throat shows the tonsils to be reddened and swollen and the tongue coated. During the first day or two the characteristic skin rash (colour like that of a boiled lobster) appears. The face becomes red all over, only the parts round the lips being free. On the body the rash usually appears first on neck and chest, and later spreads to the extremities. The tonsils become coated while the tongue assumes a raspberry-like aspect. The temperature remains high after the initial vomiting, for 3-5 days; then it slowly goes down and reaches normal after 9-12 days. The rash disappears at the end of the first or early in the second week; the skin then peels off in the form of thick scales, often of considerable size. These scales were formerly dreaded as the principal means of infection, but modern knowledge points to their comparative harmlessness, and emphasis is laid upon the early infectivity of both slight and severe cases.

The above is the course of the disease seen in typical uncomplicated cases. Atypical cases are, however, frequent. On one hand slight cases are encountered with short duration and ill defined symptoms. Such patients sometimes do not lie down and being unrecognized are particularly dangerous for the spread of infection. On the other hand, severe and complicated cases are met with. In the first, the disease may take a fulminant course ending in death within a few days. The most dreaded complications are severe angina, leading to necrosis, suppuration of the lymphatic glands on the neck, purulent inflammation of the middle ear and inflammation of the kidney. These complications, some of which may invalid the sufferer for a lifetime, make scarlet fever the dreaded disease it is, and cause anxiety to the doctor even when its course seems mild at the beginning. The gravity of the cases and the frequency of complications vary in different epidemics.

What can be done to defend ourselves, especially the younger generation against scarlet fever? Fortunately, thanks to incessant research throughout the world, certain important data have been established. These are:

1.—Scarlet fever is caused mainly, if not entirely, by the *Streptococcus hæmolyticus* (round bacteria occurring in chains), found in practically all cases of the infection.

2.—The disease has been produced experimentally in human volunteers and white goats, with formation of typical skin rashes.

3.—Satisfactory results have been obtained from the use of a serum similar to the antitoxin of diphtheria, produced from horses, but this serum is potent only when given early. In Harbin we have not obtained as encouraging results as in America, largely because the cases have been more severe and the majority have been admitted into hospital when too late.

4.—An interesting test, introduced by Drs. Dick and known as the Dick test, can differentiate persons liable to attack (positive Dick) from those not liable to attack (negative Dick).

5.—During an epidemic of scarlet fever, the positive persons should be immunized with a diluted toxin prepared from the *streptococcus*. Although such immunization lasts as a rule less than one year, sometimes longer, it wards off immediate danger and should be generally applied.

6.—It is possible that, as in the case of diphtheria, a more efficient method of immunization for scarlet fever will be found in the combined use of toxin and serum in graduated doses.

The splendid achievement of the Drs. Dick (husband and wife) at Chicago has stimulated workers in Europe and Asia to go thoroughly into the question. Our laboratory staff at Harbin were the pioneers in the Orient to undertake this new line of research since they feel that much can be done by up-to-date physicians to relieve the distress caused by this newly imported and exceedingly virulent disease throughout the country.

Among the results observed by us in Harbin may be mentioned the following :

1.—Out of 1275 Dick tests made on the Chinese population in Harbin ranging in ages from 1 to over 30, positives were found in 47.7 per cent.

2.—Out of 542 school children from six different schools examined the most positives were shown in the kindergarten class (86 per cent.).

3.—Investigations made of persons from different provinces of China are as yet too few to be of value but should be prosecuted on the spot with a view to ascertaining their comparative susceptibility, especially as among northerners and southerners.

4.—More extensive investigations were started during the autumn of 1926 upon 10,000 to 15,000 school children resident in different cities along the Chinese Eastern Railway in order to protect the susceptible children, whether Chinese or Russian. The results will be published later.

5.—Our Anti-Plague Laboratory in Harbin has prepared all the necessary material for the Dick test, immunization and treatment.

Finally, it should not be forgotten that, however valuable a remedy this new toxin and antitoxin may be, the general public should remember the prior importance of (1) strict isolation of scarlet fever patients, especially in the early infectious stage, and (2) careful nursing so that serious complications such as kidney inflammation, otitis, severe tonsillar and glandular trouble, etc., may not occur. Under all circumstances proper hospitalisation is preferable to home nursing and when combined with modern methods of immunization this would produce the best results for all concerned.

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STUDIEN UEBER DIE VITALITAET DER SCHARLACHSTREPTOKOKKEN.

(*Reprinted from Zeits. fur Hygiene und Infekt. 1927.*)

MIT 2 TEXTABBILDUNGEN. ALSO ENGLISH SUMMARY.

Bereits um die Mitte der 80 er Jahre des vorigen Jahrhunderts wurde von *Loeffler*[33]) und *Klein*[28]) das regelmässige Vorkommen von Streptokokken im Rachen Scharlachkranker mitgeteilt.

Diese Tatsache wurde von sehr zahlreichen Forschern bestätigt. Es stellte sich wohl in der Folge heraus, dass bei allen normalen erwachsenen Personen hämolytische Streptokokken nach mehrmaligen und eingehenden Prüfungen, und zwar zumeist in den Krypten der Tonsillen nachweisbar sind[12]), doch gibt es keine Infektionskrankheit im engeren Sinne, bei welcher dieselben in so grosser Menge und Regelmässigkeit auftreten, wie bei Scharlach.

Immer wieder fanden sich Autoren, welche sich für die Streptokokkenätiologie des Scharlachs aussprechen; namentlich in den letzten Jahren, seitdem von seiten zahlreicher Amerikaner das Problem der Streptokokkenätiologie neuerdings aufgerollt wurde, hat sich die Ansicht vielfach verbreitet, dass für eine Erkrankung an Scharlach einzig und allein eine serologisch [2, 21, 22, 25, 41—43, 49]) und durch ihr spezifisches Toxin [14—18, 53]) ziemlich gut, wenn auch nicht einwandfrei [38, 46]) differenzierbare Art eines hämolytischen Streptokokkus verantwortlich zu machen sei.

Auch die *Schultz-Charltonschen* Substanzen verdanken ihre Entstehung der Einwirkung der spezifischen Antigene des hämolytischen Scharlachstreptokokkus auf den menschlichen und tierischen Organismus.

Die so nun wieder aktuell gewordene Theorie der Streptokokkenätiologie des Scharlachs wurde in der letzten Zeit durch die interessanten Forschungsergebnisse russischer Autoren [23, 54]) wieder einigermassen erschüttert.

Es hat nach *Zlatogoroff* eher den Anschein, als ob die Resultate der amerikanischen Autoren in Paraimmunitätserscheinungen ihre Erklärung fänden.

Im schroffen Gegensatz zur amerikanischen Schule stehen die Ergebnisse italienischer Autoren [7, 8]), welche in einem durch Bakterienfilter hindurchgehenden grampositiven Diplokokken den wahren Erreger des Scharlachs sehen.

Trotz dieser Widersprüche sind alle Autoren darin einig, dass hämolytische Streptokokken bereits in den ersten Krankheitstagen im Rachen Scharlachkranker in grossen Mengen regelmässig anzutreffen und für die zahlreichen Komplikationen der zweiten Krankheitsperiode zum grossen Teil verantwortlich sind.

Möglicherweise werden diese Mikroorganismen, wenn sie nicht selbst den Scharlacherreger darstellen, durch den noch unbekannten filtrierbaren Scharlacherreger "aktiviert" und nach *Zlatogoroff* mit "besonderen antigenen Eigenschaften" ausgerüstet[54]).

Da die beim Scharlach auftretenden hämolytischen Streptokokken den Verlauf der Krankheit in so hohem Grade beeinflussen, und vermutlich in engster Symbiose mit dem wirklichen Scharlacherreger stehen—wenn sie nicht mit ihm identisch sind—, erscheint es von Wichtigkeit, ihre Vitalität auch ausserhalb des Organismus einem eingehenden Studium zu unterziehen.

In der Nordmanschurei (Harbin) ist der Scharlach eine ziemlich häufige und oft in schweren Epidemien auftretende Krankheit[9, 31, 52]).

Die Krankheit konnte nach unseren Erfahrungen leider weder durch das *Dick*sche noch durch das *Dochez*sche Scharlachserum—trotz früher und reichlicher Anwendung desselben—in überzeugender Weise günstig beeinflusst werden[11]).

Systematische Untersuchungen, welche von mir seit 1½ Jahren an über 120 Scharlachfällen ausgeführt wurden, ergaben die Anwesenheit von hämolytischen Streptokokken in *genau* 100% der Fälle*) in der ersten Krankheitsperiode. (Das Material wurde meist zwischen dem 3. und 5. Krankheitstage entnommen.)

Technik: Der Tonsillenabstrich wurde nach Herabdrücken der Zunge mit sterilem Spatel durch leichtes Betupfen der hinteren Rachenwand mit sterilem Tampon gewonnen. Der in steriler Eprouvette aufbewahrte Tampon wurde bald nach der Entnahme (spätestens nach 3—5 Stunden) auf eine frisch gegossene Hammelblutagarplatte 4—6 mal aufgetupft. Hierauf wurde mit einem Drigalskispatel die 1. Platte und davon noch weitere 5 Platten der Reihe nach bestrichen.

Von 50 der untersuchten Scharlachfälle, welche zum Ausgangspunkt für weitere Studien dienten, wurde ferner das Verhältnis zwischen Strept. haem.-Kolonien und den übrigen aufgegangenen Kolonien meist durch Zählen genau bestimmt. Die erhaltenen Resultate sind in Tab. I wiedergegeben.

Die in den Rubriken der Platten I—VI befindlichen, oberen, gesperrt gedruckten Zahlen geben die absolute Anzahl der aufgegangenen Strept. häm.-Kolonien wieder. Die unterhalb derselben befindlichen drei Zahlen geben der Reihe nach in Prozenten das Zahlenverhältnis

1. der hämolytischen Streptokokken,
2. der grün wachsenden Streptokokken, und
3. anderweitiger Kolonien zueinander auf der Platte an.

Wo dies Verhältnis infolge allzu dichter Bewachsung der Platten nicht festgestellt werden konnte, wurde ein Fragezeichen (?) gesetzt.

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*) Das Vorkommen der hämolytischen Streptokokken in glatt 100% der untersuchten Scharlachfälle wurde bereits von *Dochez* und *Sherman* (Journ. of the Americ. med. assoc. **82**, 542) *Dick* und *Dick*[17]; *Bliss*[3]), *Bristol*[4]), *Chen*[10]), *Deicher*[13]), *Moody* und *Irons*[34]) u. a. festgestellt.

Diese Tabelle zeigt vor allem das fast ausschliessliche Vorkommen von Streptokokken auf der hinteren Rachenwand bei akutem Scharlach. Andere Mikroorganismen fanden sich nur selten und in geringem Prozentsatz. Es waren dies zum grossten Teil Staphylokokken, welche namentlich in Fällen von Angina necroticans die ersten Platten des Satzes verunreinigten. Aus Stäbchen (Diphtherie u. a.) bestehende Kolonien wurden auf den Platten selten beobachtet.

Es verdient hier hervorgehoben zu werden, dass in den Sommermonaten desselben Jahres, in welchen die Scharlachmortalität eine beträchtlich höhere war, die Platten der einzelnen Fälle viel häufiger absolute Reinkulturen des Streptococcus hämolyticus ergaben, als im Herbst, während welcher Zeit der Genius epidemicus milder wurde, was vielleicht auch mit dem reichlicheren und oft überwiegenden Auftreten der grünen Streptokokken im Zusammenhang zu bringen ist.

Ganz auffallend war, dass bei den schweren Fällen die grünen Streptokokken meist vollkommen zurücktraten und in den ersten Platten vorkommende Verunreinigungen hauptsächlich von Staphylokokkenkolonien herrührten.

Auf die prozentuellen Beziehungen der grünen zu den hämolytischen Streptokokken wurde deshalb grosser Wert gelegt, da alle in der Tabelle angeführten Tampone nach der Beimpfung aufbewahrt wurden, um die Vitalität der auf ihnen befindlichen Bakterienflora weiter zu untersuchen, wovon später noch die Rede sein wird.

Kulturell konnte bereits in den ersten Krankheitsstunden das Ueberwiegen des Str. haem. auf der hinteren Rachenwand nachgewiesen werden (Fall 1.) Hämolytische Streptokokken waren in etwa 20% der Fälle am Tage der Entlassung des Patienten, nach völliger Abschuppung desselben, auf den Tonsillen noch nachweisbar. (Vgl. die ziemlich analogen Ergebnisse von *Stevens* und *Dochez*[44]) an Scharlachrekonvaleszenten !)

Die wichtige Rolle, welche den hämolytischen Streptokokken bei Scharlach zukommt, veranlasste mich, die Vitalität dieser Streptokokken

Tabelle 1. *50 Tampone von 50 Scharlachfällen, gewonnen in der Zeit vom 7. IX. 1923 bis Dezember 1925.*

Fall Nr., Krankheits-verlauf	Krank-heitstag, Datum	Platte Nr. I	II	III	IV	V	VI
1. Kon.	7.	*>500*	*<500*	*<50*	*<50*	*2*	*0*
leicht. Fall	7. IX.	20 :70 :0	20 :70 :0	10 :90 :0	5 :95 :0	0 :100 :0	—
2. Sem.	8.	*>1000*	*110*	*26*	*1*	*3*	*1*
letal	22. IX.	90 :0 :10	90 :0 :10	95 :0 :5	100 :0 :0	100 :0 :0	33 :0 :66
3. Stok.	4.	*>2000*	*1500*	*500*	*400*	*200*	*150*
leicht	22. IX.	?	99 :0 :1	100 :0 :0	100 :0 :0	99 :0 :1	100 :0 :0
4. Wor.	6.	*>1000*	*>500*	*200*	*200*	*100*	*50*
leicht	25. IX.	50 :50 :0	50 :50 :0	20 :80 :0	20 :70 :10	30 :70 :0	30 :60 :10
5. Phil.	5.	*>2000*	*1000*	*500*	*500*	*100*	*50*
leicht	25. IX.	?	50 :50 :0	50 :50 :0	50 :50 :0	30 :70 :0	30 :70 :0
6. Ign.	3.	*>1000*	*<1000*	*500*	*100*	*50*	*30*
leicht	25. IX.	40 :40 :20	50 :40 :10	70 :20 :10	80 :15 :5	80 :20 :0	80 :20 :0
7. Fjod.	3.	*>1000*	*500*	*200*	*200*	*50*	*50*
mittelschw.	25. IX.	?	20 :70 :10	20 :70 :10	30 :70 :0	30 :70 :0	30 :70 :0
8. Rub.	3.	*>2000*	*1000*	*1000*	*500*	*200*	*50*
schwer	28. IX.	?	95 :3 :2	99 :0 :1	100 :0 :0	100 :0 :0	98 :0 :2
9. Kras.	3.	*>2000*	*1000*	*500*	*100*	*100*	*50*
leicht	1. X.	?	95 :1 :4	95 :2 :3	95 :2 :3	95 :0 :5	95 :5 :0
10. Germ.	3.	*>1000*	*200*	*50*			
schwer	1. X.	98 :0 :2	100 :0 :0	100 :0 :0	steril	steril	steril
11. Sim.	4.	*500*	*200*	*100*	*100*	*50*	*20*
leicht	1. X.	50 :25 :25	50 :30 :20	0 :20 :10	80 :15 :5	95 :5 :0	95 :0 :5
12. Bsar.	4.	Kolonien des Streptococcus haemolyticus in allen					
leicht	5. X.	Platten *ueberwiegend*.					
13. Zar.	4.	Streptococcus haemolyticus, vermischt mit grünen					
leicht	6. X.	Streptokokken, letztere in der Ueberzahl.					
14. Kuch.	5.	Streptococcus haemolyticus-Kolonien, gemischt mit					
leicht	6. X.	grünen Streptokokken; erstere überwiegend.					
15. Osp.	4.	Streptococcus haemolyticus-Kolonien, gemischt mit					
leicht	6. X.	grünen Streptokokken. aa.					
16. Wol., A.	4.	Streptococcus haemolyticus-Kolonien, gemischt mit					
schwer	5. X.	grünen Streptokokken. aa.					
17. Arj.	3.	Hämolytische Streptokokken in der Ueberzahl.					
leicht	6. X.						
18. Ivan.	4.	Hämolytische Streptokokken in der Ueberzahl.					
leicht	6. X.						
19. Wol., B.	5.	Hämolytische Streptokokken in der Ueberzahl.					
schwer	6. X.						
20. Alek.	4.	<2000	*500*	*100*	9	*2*	*1*
schwer	8. X.	?	25 :75 :0	20 :80 :0	10 :90 :0	5 :95 :0	10 :90 :0
21. Pan.	3.	*>1000*	*100*	*4*	*0*	*0*	*0*
leicht	8. X.	?	50 :50 :0	20 :80 :0	0 :75 :25	0 :100 :0	—
22. Mig.	2.	*1000*	*200*	*50*	*10*	*4*	*1*
leicht	8. X.	?	30 :70 :0	40 :60 :0	20 :80 :0	20 :80 :0	sonst ster.
23. Bond.	2.	*500*	*100*	*5*	*1*	*1*	*0*
leicht	8. X.	?	30 :70 :0	5 :95 :0	1 :99 :0	1 :99 :0	0 :100 :0

Tabelle 1. (*Fortsetzung.*)

Fall Nr., Krankheits-verlauf	Krank-heitstag, Datum	Platte Nr. I	II	III	IV	V	VI
24. Klo.	6.	*2000*	*1000*	*500*	*100*	*50*	*50*
letal	8. X.	?	95 :0 :5	100 :0 :0	100 :0 :0	100 :0 :0	100 :0 :0
25. Kus.	7.	*70*	*7*	*0*	*0*		
leicht	8. X.	10 :90 :0	20 :80 :0	0 :100 :0	50 :50 :0	steril	steril
26. Gol.	3.	*500*	*100*	*30*	*2*	*6*	*1*
schwer	8. X.	50 :50 :0	30 :70 :0	20 :70 :10	20 :80 :0	40 :60 :0	25 :75 :0
27. Gla.	4.	*>2000*	*600*	*200*	*150*	*100*	*30*
letal	13. X	90 :10 ?	95 :0 :5	95 :0 :5	100 :0 :0	95 :0 :5	100 :0 :0
28. Ne., L.	3.	*500*	*100*	*50*	*8*		*1*
leicht	13. X	?	80 :20 :0	60 :40 :0	60 :40 :0	1 gr. Str.	sonst ster.
29. Tche.	4.	*200*	*20*	*2*	*3*		
mittelschwer	13. X	viel grüne	50 :50 :0	100 :0 :0	100 :0 :0	steril	steril
30. Leg.	5.	*500*	*200*	*50*	*50*	*9*	*4*
leicht	13. X	viel grüne	80 :20 :0	60 :40 :0	80 :20 :0	70 :30 :0	80 :20 :0
31. Ne., K.	6.	*500*	*200*	*505*	*5*	*8*	*2*
leicht	13. X	viel grüne	40 :40 :20	40 :40 :20	einzelne	grüne Str.	einz. Sta.
32. Los.	2.	*500*	*100*	*150*	*200*	*150*	*50*
leicht	13. X	viel Sta.	80 :10 :10	95 :5 :0	95 :5 :0	95 :5 :0	fast Reink.
33. Wed.	5.	*250*	*50*	*0*	*0*		
leicht	20. X.	33 :66 :0	20 :80 :0	0 :100 :0	einige vir.	steril	1 Verunr.
34. Mak.	4.	*>2000*	*1000*	*500*	*60*	*40*	*50*
leicht	28. X.	?	70 :25 :5	66 :33 :0	95 :5 :0	80 :20 :0	90 :10 :0
35. Sij.	3.	*>2000*	*200*	*20*	*4*	*7*	*14*
leicht	28. X.	?	20 :70 :10	5 :90 :5	2 :98 :0	5 :95 :0	10 :90 :0
36. Büt.	4.	*>1000*	*500*	*200*	*20*	*8*	*8*
leicht	7. XI.	100 :0 :0	100 :0 :0	100 :0 :0	100 :0 :0	100 :0 :0	1 Verunr.
37. Fel.	4.	*2000*	*500*	*600*	*20*	*25*	*60*
leicht	7. XI.	100 :0 :0	100 :0 :0	100 :0 :0	100 :0 :0	100 :0 :0	100 :0 :0
38. Str.	3.	*>1000*	*>500*	*>100*	*20*	*8*	*8*
leicht	7. XI.	90 :5 :5	95 :3 :2	100 :0 :0	95 :0 :5	100 :0 :0	88 :0 :12
39. Mend.	11.	*>2000*	*500*	*50*	*80*	*40*	*17*
schwer	10. XI.	95 : ? :5	100 :0 :0	100 :0 :0	100 :0 :0	100 :0 :0	100 :0 :0
40. Tscher.	7.	*50*					
leicht	16. XI.	95 :0 :5	steril	steril	steril	steril	steril
41. Bal.	4.	*>600*	*30*	*3*	*1*	*0*	*0*
leicht	16. XI.	?	10 :90 :0	10 :90 :0	5 :95 :0	0 :100 :0	4 Verunr.
42. Wor.	5.	*>500*	*25*	*2*	*0*	*0*	*0*
leicht	16. XI.	?	100 :0 :0	100 :0 :0	einz. Sta.	einz. Sta.	einz. Sta.
43. Tsche.	7.	*>2000*	*>1000*	*500*	*100*	*50*	*80*
letal	17. XI.	?	?	60 :30 :10	49 :49 :2	60 :40 :0	70 :30 :0
44. Tün.	6.	*1500*	*1000*	*250*	*150*		
schwer	17. XI.	?	?	90 :10 :0	80 :20 :0	—	—
45. Wat.	4.	*>2000*	*1500*	*1000*	*200*	*90*	*100*
leicht	17. XI.	?	?	85 :10 :5	75 :24 :1	70 :30 :0	80 :20 :0
46. Lop.	4.	*2000*	*1000*	*100*	*30*	*10*	*1*
schwer	17 XI.	?	?	70 :30 :0	50 :50 :0	50 :50 :0	sonst ster.

Tabelle 1. (*Fortsetzung.*)

Fall Nr., Krankheits-verlauf	Krank-heitstag, Datum	Platte Nr. I	II	III	IV	V	VI
47. Sim.	7.	*500*	*100*	*50*	*80*	*10*	5
letal	17. XI.	40 : ? :60	30 :0 :70	25 :0 :75	20 :0 :80	20 :0 :80	10 :0 :90
48. Kus.	2.	*>2000*	*1000*	*500*	*20*	*8*	25
schwer	23. XI.	?	?	90 :10 :0	100 :0 :0	100 :0 :0	95 :0 :5
49. Kri.	2.	*>2000*	*1000*	*200*	*50*	*50*	*6*
leicht	23 XI.	?	40 :40 :20	40 :60 :0	60 :40 :0	60 :40 :0	45 :55 :0
50. Dja.	5.	*>1000*	*>500*	*200*	*100*	*150*	*50*
schwer	23. XI.	?	?	50 :50 :0	50 :50 :0	80 :20 :0	95 :5 :0

auch ausserhalb des menschlichen Organismus in Kulturen, hauptsächlich aber in getrocknetem Zustande systematisch zu untersuchen.

Ueber die Lebensfähigkeit der Streptokokken *in Kulturen* liegen bereits zahlreiche Angaben vor. In den Handbüchern findet sich meist die Angabe, dass die grünen Streptokokkenformen alle 14 Tage, die hämolytischen etwa alle 6 bis 8 Wochen überimpft werden müssen, um die Stämme sicher am Leben zu erhalten. Allerdings gibt es unter den grünen Streptokokken Formen, welche bereits nach 14 Tagen in den Bouillonkulturen abgestorben sind[40]). Diese Angaben gelten für die gewöhnlichen Nährböden, einschliesslich des Blutagars. Andererseits berichtet *Brown*[5]) über eine bedeutend längere Lebensdauer seiner Streptokokkenstämme in Kulturen. Dieser Autor züchtete seine Stämme auf defibriniertem Pferdeblut, welches auf Schrägagar aufgestrichen war. Die Kulturen werden nur eine Nacht bebrütet, worauf die mit Paraffin verschlossenen Röhrchen im Eisschrank bei 40—45° F aufbewahrt blieben. Alle seine Stämme, mit Ausnahme des Str. mucosus und der Pneumokokken, wurden innerhalb 4—5 Monaten nur einmal überimpft; von 50 Stämmen verschiedener Typen waren nach 7 monatlicher Aufbewahrung alle mit Ausnahme von zweien noch am Leben.

Nach dem Ungermannschen[50]) Verfahren konservierte hämolytische Streptokokken halten sich nach *Pulvermacher*[39]) bisweilen über 1 Jahr lebensfähig, namentlich dann, wenn die Stämme durch lange Fortzüchtung bereits künstlichen Medieh sich angepasst haben, während frisch gezüchtete hämolytische Stämme nur ungefähr 6—8 Monate im Ungermannschen Serumröhrchen lebensfähig bleiben[39]). Grüne Stämme beginnen auch in diesem Nährmedium bereits nach 2 Monaten, einige sogar noch früher abzusterben. *Ayers and Johnson*[1]) empfehlen eine Casein und Phosphat enthaltende Nährbouillon, der Gelatine, Glucose und nach Filtration verflüssigter Agar und Natrium citricum zugefügt wird. In diesem, nach ziemlich komplizierter Vorschrift hergestelltem Nährboden brauchen nach Angabe der Autoren Streptokokkenkulturen nur alle 4 Monate überimpft zu werden.

Speziell über die hämolytischen Scharlachstreptokokken liegen folgende Angaben vor: Im Hämoglobinafar nach Levinthal in Tiefenstich konservierte Scharlachstreptokokken halten sich monatelang am Leben. Nach *Deicher*[13]) waren selbst nach 10 Monaten einzelne auf diese Weise

im Eisschrank konservierte Kulturen noch lebend, vollvirulent und bildeten Toxin. Nach *Trotzky* und *Karassewa*[47]), welche ihre Streptokokken in Glycerinagarstichkulturen unter Paraffinöl aufbewahrten, gelingt es, Scharlachstreptokokken, aus dem Herzblut von Scharlachleichen gewonnen, über 3 Monate lebensfähig zu erhalten, während von 4 Streptokokkenstämmen derselben Herkunft, welche 8 Monate so konserviert worden waren, nur 1 Stamm lebensfähig blieb. Diese Autoren konnten hämolytische Streptokokken über 1 Jahr lang lebensfähig erhalten, wenn sie das gewonnene Blut in zugelöteten Ampullen aufbewahrten.

Die Methode *Neufelds*[36]): Eintrocknen der Organe infizierter Tiere (Pneumokokken) ergibt auch für Streptokokken gute Resultate.

Alle diese Methoden kamen nicht in Anwendung, da ich durch verschiedene Nährböden- und Tierpassagen die Mikroorganismen in ihren Eigenschaften nicht beeinflussen wollte. Abgesehen davon sind Tierpassagen für diese Streptokokkenart schon deshalb bedenklich, da dieselben im Organismus der Versuchstiere häufig unter Vergrünung eingehen.

In meinen systematischen Versuchen prüfte ich die Lebensdauer 40 *frisch gewonnener**) Scharlachstreptokokkenstämme auf Hammelblutagarröhrchen (9 ccm alkal. Agar, 1 ccm defibriniertes Blut) in der Schrägkultur. Die 40 von den Scharlachfällen reingezüchteten hämolytischen Streptokokkenstämme wurden auf Blutagarröhrchen überimpft, welche nach 24stündiger Bebrütung im Kühlschrank bei 10—15° C dauernd aufbewahrt wurden. Nach bestimmten Zeitabschnitten wurde je eine ganze Platinöse, gewonnen durch Strich vom Grunde des Röhrchens nach aufwärts, auf je eine Hammelblutagarplatte ausgestrichen und das Resultat nach 24 resp. 48 Stunden notiert.

Bereits nach 4monatlichem Verweilen der Röhrchen im Kühlschrank bemerkt man auffallende Wachstumsverzögerung bei zahlreichen Stämmen. Einige der Platten zeigten nach 24 Stunden noch kein Wachstum und erst nach 48stündigem Verweilen im Thermostaten gingen üppige und typische Streptokokkenkolonien mit normal grossen (1,0 bis 2,4 mm) Resorptionshöfen auf. Nur sehr wenige 4 Monate alte Stämme zeigten nach 24stündigem Wachstum bereits normal grosse Resorptionshöfe, die Hämolyse war nach dieser Zeit erst im Beginne. (Bei frisch aus dem Rachen gezüchteten Scharlachstreptokokken sind nach 24 Stunden alle Resorptionshöfe gleich gross (1,6—2,2 mm) und gleichmässig ausgebildet).

Nach 5monatlichem Verweilen der Röhrchen in Kühlschrank war dieselbe Wachstumsverzögerung bemerkbar, doch waren alle Stämme noch am Leben.

Nach 6½ Monaten zeigten nach 24 stündiger Bebrütung nur noch ein einziger Stamm von den 40 üppiges Wachstum und zwei Stämme spärliche Kolonien, während alle anderen selbst nach 48 Stunden kein Wachstum erkennen liessen.

Ueber die Haltbarkeit von Streptokokken *im angetrockneten Zustande* liegen bis jetzt relativ wenige präzise Angaben vor.

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*) Nach *Pulvermacher* gelingt es bei *frisch von Menschen gezüchteten* hämolytischen Streptokokkenstämmen nicht immer, Ueberimpfbarkeit und Virulenz so lange zu erhalten, wie bei den an lange Fortzüchtung schon gewöhnten Laboratoriumsstämmen.

Nach *Haegler*[24]) halten sich, an Mull angetrocknet, Erysipelstreptokokken 14—36 Tage lebensfähig; an Seidenfäden angetrocknet, bewahrt der Erysipelstreptokkous noch nach 52 Tagen seine Lebensfähigkeit[32]). Die Hartnäckigkeit, mit welcher in manchen Krankenzimmern die Erysipelerreger haften, spricht jedoch für ihre bedeutend längere Lebensdauer in der Natur ausserhalb des menschlichen Organismus. Nach *Kurth*[29]) sind rasch lufttrocken gemachte Streptokokken 5—6 Wochen haltbar. Nach *Lingelsheim*[32]) lassen sich im Blut und Eiter angetrocknete Streptokokken fast doppelt so lange konservieren, als angetrocknete Bouillon- oder Agarkulturen. In feinsten Tröpfchen verspritzte Streptokokken hatten nach den *Kirsteinschen* Versuchen[27]) eine Lebensdauer von 10 Tagen. Nach *Germano*[19]) erweist sich angetrocknetes Streptokokkenmaterial ziemlich widerstandsfähig; nach seinen Ergebnissen hielten sich die Streptokokken in Trockenstaubgemengen wenigstens 7 Wochen, in angetrockneter diphtherischer Membran über 3 Monate lang lecensfähig. In getrocknetem pneumonischen Sputum hielten sich nach diesem Autor die Streptokokken bis über 150 Tage am Leben. (Auf Grund der grossen Resistenz und langen Lebensdauer der angetrockneten Streptokokken kommt *Germano* zum Schluss, dass der Luft zweifellos ein wesentlicher Anteil an der Uebertragung der Streptokokkeninfektion zukommt.)

Untersuchungen über das Vorkommen der *Scharlachstreptokokken* in der Aussenwelt wurden bereits von *Tunicliff*[49]) und in jüngster Zeit von *Vas*[51]) ausgeführt. *Tunicliff* bewies, dass der Strept. haem. von den Wänden und Böden von Scharlach-und Diphtheriekrankenzimmern sowie von Fingernägeln, Gesicht und Schuhen der bediensteten Wärterinnen, endlich auch von den Essgeräten gezüchtet werden kann. *Vas* konnte in der Luft von Krankenzimmern sowohl vor als auch nach der Lüftung und Fegung derselben regelmässig das Vorkommen der Scharlachstreptokokken nachweisen. Auf Grund dieser Befunde spricht sich *Vas* für die wichtige Rolle der Luftinfektion beim Scharlach aus.

Mir erschien es von besonderem Interesse, die Vitalität der Scharlachstreptokokken in dem Medium, in welchem sie beim Kranken vorkommen, d. i. *im Rachenschleim*, eingehender zu prüfen. Es wurden zu diesen Untersuchungen Tampone benutzt, welche mit dem Rachenschleim der Kranken kontaminiert waren.

1. Versuch: 39 Tampone, gesammelt in der Zeit vom 7. IX. bis 23. XI. 1925 (vgl. Tab. 1).

Alle diese Tampons wurden nach Bestreichen der Agarplatte in ihrer Eprouvette dauernd bei 10—18° C, vor Licht geschützt, aufbewahrt. Das Röhrchen war mit einem Wattepfropfen, durch welchen das Holzstäbchen des Tampons durchgestossen war, lose verschlossen, so dass eine ziemlich rasche Austrocknung ermöglicht war. In bestimmten Zeitabständen wurden die Tampone auf Blutagarplatten mehrmals aufgeklopft, nachdem sie vorher mit steriler Pinzette leicht gelockert worden waren. Später (7. Prüfung) ging ich zu folgender Methode über, welche das Material auf den Tamponen mehr schonte und bessere Resultate ergab*):

*) Diese Methode wurde von mir in der Folge bei der Beimpfung von Blutagarplatten mit von Plattenmaterial bestrichenen Tamponen stets angewendet (siehe später !).

Der vollkommen ausgetrocknete Tampon wurde über der Blutagarplatte mit steriler feiner Pinzette leicht gezupft. Der auf die Blutagarfläche fallende feine Staub wurde mit Drigalskispatel verrieben. Durch diese Prozedur wurde eine Berührung des Tampons mit Nährbodenoberfläche vollkommen vermieden.

Nach der 8. Prüfung, welche bei den zwei noch aufbewahrten Tamponen (alle anderen waren nach der 7. Prüfung liquidiert worden) ausschliesslich negative Resultate ergab, wurden die Tampone bei der 9. Prüfung durch Eingiessen einiger Kubikzentimeter alkalischer Bouillon in die Röhrchen befeuchtet und in den Thermostaten gestellt. Nach 4stündigem Verweilen der Tampone im Brutschrank wurden sie auf Blutagarplatten mehrmals aufgeklopft, nachdem vorher die überschüssige Flüssigkeit an der Wand ihrer Eprouvetten leicht abgepresst worden war; dann wurden sie wieder in ihren Röhrchen im Thermostaten weiter bebrütet.

Nach 24 stündiger Bebrütung wurden die Tampone mit einer Platinöse allenthalben abgewischt und mit dem Material je zwei Blutagarplatten beimpft (10. Prüfung). Die Reinzüchtung der Streptokokken erfolgte in letzterem Falle ausschliesslich von der zweiten Blutagarplatte.

Tab. 2 gibt die Resultate der Versuchsreihe mit den aufbewahrten Originaltamponen wieder.

Die in den Rubriken stehenden ersten Zahlen geben die Anzahl der aufgegangenen Str. haem.-Kolonien an, die zweiten Zahlen die Menge der anderweitigen Kolonien. Diese Verunreinigungen bestehen hauptsächlich aus kleinen, milchweissen Staphylokokkenkolonien und vereinzelten Kolonien des Heubacillus, während grüne Streptokokken von der zweiten Prüfung an nur selten und vereinzelt anzutreffen waren. Da diese Experimente in einem staubigen Stadtteil ausgeführt wurden, und die Manupulationen immerhin ein längeres Offenhalten der Blutagarplatten nötig machten, ist anzunehmen, dass ein Teil dieser "Verunreinigungen" sekundären Ursprunges ist. Die rechts befindliche, gesperrt gedruckte Zahl gibt die Tage an, welche seit der Bestreichung der Tampone verflossen sind. War das Experiment für Str. haem. positiv, so wurde diese Zahl, um die Tabelle übersichtlicher zu gestalten, durch Druck hervorgehoben.

Auffallend ist vor allem das rasche Absterben aller grünen Streptokokken, was beim Vergleich der beiden Tabellen sehr in die Augen fällt.

Bei den über 100 Tage alten Tamponen ist bereits fast regelmässig eine deutliche Wachstumsverzögerung der hämolytischen Streptokokken wahrnehmbar. So waren z. B. bei der 7. Prüfung (150—200 Tage alte Tampone) nach 24 Stunden nur 8, nach 48 Stunden jedoch 11 Platten positiv. Die nach 24 Stunden bereits aufgegangenen Streptokokken zeigten meist noch kleine, erst im Wachstum begriffene Resorptionshöfe,

Tabelle 2.

Nr.	Fall	Datum	Prüfung I sofort	Prüfung II 10. XI. 1925	Prüfung III 9. XII. 1925	Prüfung IV 7. I. 1926	Prüfung V 9. II. 1926
1	Kon.	7. IX 1925		180 :150 *64*	0 :3 93	1 :4 *122*	**4** :5 *155*
2	Sem.	22. IX. 1925		60 :6 *49*	200 :0 *78*	200 :1 *140*	**1** :4 *169*
3	Stok.	22. IX. 1925		H (Reink.)	H :0 *78*	1000 :0 *107*	**600** :2 *140*
4	Wor.	25. IX. 1925		2 :10 *46*	0 :4 *75*	0 :5 104	**0** :1 137
5	Phil	25. IX. 1925		25 :2 *46*	1 :2 *75*	0 :2 104	**11** :1 *137*
6	Ign.	25. IX. 1925		2 :10 *46*	8 :1 *75*	1 :4 *104*	**100** :10 *137*
7	Fjod.	25. IX. 1925		100 :2 *46*	1 :0 *75*	3 :1 *104*	0 :1 *137*
8	Rub.	28. IX. 1925		H (Reink.) *43*	H :0 *72*	1000 :0 *101*	**700** :0 *134*
9	Kras.	1. X. 1925		150 :0 *40*	40 :0 *69*	100 :0 *98*	**50** :0 *131*
10	Ger	1. X. 1925		H :20 *40*	10 :0 *69*	8 :2 *98*	**14** :3 *131*
11	Sin.	1. X. 1925		H (Reink.) *40*	0 :0 69	30 :3 *98*	**1500** :3 *131*
12	Bsar.	5. X. 1925		100 :2 *36*	0 :1 65	1 :4 *94*	**5** :10 *127*
13	Zar	5. X. 1925		H (Reink.) *36*	1 :2 *65*	5 :5 *94*	**4** :3 *127*
14	Kuch	5. X. 1925		200 :4 *36*	0 :0 65	7 :3 *94*	**0** :0 127
15	Osp.	5. X. 1925		6 :3 *36*	1 :40 *65*	0 :3 94	**0** :1 127
16	Wol. A	5. X. 1925		200 :10 *36*	10 :0 *65*	200 :15 94	**2000** :2 *127*
17	Arj.	6. X. 1925		1000 :10 *35*	1 :0 *64*	1 :5 *93*	**0** :0 126
18	Ivan.	6. X. 1925		10 :10 *35*	1 :1 *64*	0 :3 93	**3** :0 *126*
19	Wol. B	6. X. 1925		H (Reink.) *35*	70 :0 *64*	100 :0 *93*	**60** :5 *126*
20	Alek.	8. X. 1925		150 :4 *33*	4 :0 *62*	35 :5 *91*	**4** :3 *124*
21	Pan	8. X. 1925		1000 :20 *33*	15 :100 *62*	40 :1 *91*	**2** :3 *124*
22	Mig.	8. X. 1925		200 :25 *33*	7 :2 *62*	22 :5 *91*	**25** :4 *124*
23	Bond.	8. X. 1925		600 :0 *33*	1 :0 *62*	2 :2 *91*	**80** :1 *124*
24	Klo.	8. X. 1925		H :10 *33*	12 :1 *62*	5 :10 *91*	**1** :0 *124*
25	Kus.	8. X. 1925		2 :0 *33*	0 :0 62	0 :1 91	**0** :4 124
26	Gol.	8. X. 1925		100 :50 *33*	0 :0 62	0 :3 91	**0** :5 124
27	Gla.	13. X. 1925		H :8 *28*	H :0 *57*	H :1 *86*	**H** :1 *119*
28	Ne. L.	13. X. 1925		H :1 *28*	40 :0 *57*	100 :1 *86*	**16** :2 *119*
29	Teghe	13. X. 1925		200 :6 *28*	0 :0 57	0 :1 86	**0** :2 119
30	Leg.	13. X. 1925		0 :1 28	0 :2 57	0 :9 86	**0** :1 119
31	Ne. K	13. X. 1925		12 :15 *28*	0 :10 57	0 :4 86	**0** :0 119
32	Los.	13. X. 1925		1000 :50 *28*	600 :50 *57*	26 :20 *86*	**200** :0 *119*
33	Wed.	20. X. 1925		20 :100 *21*	3 :0 *50*	0 :1 79	**0** :2 112
34	Mak.	28. X. 1925		H :0 *13*	2 :0 *42*	2 :3 *71*	**0** :3 104
35	Sij.	28. X. 1925		H :50 *13*	0 :1 42	0 :1 *71*	**0** :6 104
36	Büt.	5. XI. 1925		H :2 *5*	35 :0 *34*	100 :0 *63*	**12** :1 *96*
37	Fel.	5. XI. 1925		H :1 *5*	H :0 *34*	200 :5 *63*	**120** :1 *96*
38	Str.	5. XI. 1925		H :2 *5*	H :0 *34*	150 :0 *63*	**200** :1 *96*
39	Mend.	9. XI. 1925		H :0 *1*	H :0 *30*	1000 :20 *59*	H :150 *92*
		% positiv :	100	97,43%	71,79%	71,79%	66,67%

Vergleiche

wodurch bedeutende Unterschiede in der Grösse der einzelnen Kolonien resultierten. Diese Unterschiede waren jedoch nach 48 Stunden meist völlig ausgeglichen. Nach einer weiteren Subkultur dieser träge wachsenden, gingen stets völlig normal rasch wachsende Kolonien mit

Tabelle 2.

Prüfung VI 10. III. 1926	Prüfung VII 7. IV. 1926	Prüfung VIII 17. V. 1926	Prüfung IX 2. VI. 1926	Prüfung X 3. VI. 1926
1 :1 *184*	0 :2 212	liquidiert		
1 :4 *169*	0 :20 197	liquidiert		
150 :1 *169*	80 :0 *197*	0 :3 237	1 :3 *253*	positiv *253/54*
0 :1 166	liquidiert			
0 :2 166	liquidiert			
20 :5 *166*	0 :20 197	liquidiert		
0 :0 166	0 :0 197	liquidiert		
600 :0 *163*	1500 :1 *191*	0 :2 231	500 :15 *247*	positiv *247/48*
40 :0 *160*	30 :0 *188*	0 :1 228	verunr. 244	verunr. 244/45
1 :4 *160*	verunr. 188	liquidiert		
1000 :2 *160*	500 :0 *188*	0 :5 228		
1 :10 *156*	0 :1000 184	liquidiert		
3 :0 *156*	0 :4 184	liquidiert		
0 :50 156	liquidiert			
0 :6 156	liquidiert			
1500 :0 *156*	600 :1 *184*	0 :4 224	verunr. 244	verunr. 244/45
4 :1 *155*	verunr. 183	liquidiert		
0 :0 155	0 :0 183	liquidiert		
30 :50 *155*	0 :0 183	liquidiert		
1 :1 *153*	0 :400 181	liquidiert		
0 :1 153	0 :1 181	liquidiert		
2 :0 *153*	0 :0 181	liquidiert		
50 :0 *153*	6 :1 *181*	0 :3 221	verunr. 240	verunr. 240/41
0 :1 153	0 :3 181	liquidiert		
0 :1 153	liquidiert			
0 :3 153	liquidiert			
1000 :1 *148*	900 :1 *176*	0 :1 216	verunr. 237	verunr. 237/38
3 :1 *148*	0 :3 176	liquidiert		
0 :1 148	liquidiert			
0 :100 148	liquidiert			
0 :0 148	liquidiert			
4 :50 *148*	0 :0 176	liquidiert	4 :3 *232*	positiv *232/33*
0 :0 141	liquidiert			
0 :0 133	liquidiert			
0 :0 133	liquidiert			
10 :0 *125*	15 :0 *153*	0 :2 193	verunr. 219	verunr. 219/20
200 :2 *125*	7 :200 *153*	0 :2 193	verunr. 219	verunr. 219/20
15 :50 *125*	6 :2 *153*	0 :5 193	verunr. 219	verunr. 219/20
H :30 *121*	500 :0 *149*	0 :1 189	verunr. 215	positiv *215/16*
58,97%	39,28%	0%	27,27%	36,36%

gleich stark und rasch sich entwickelnden hämolysierenden Fähigkeiten auf.

Ganz anders verhielten sich die Streptokokkenkolonien bei der 10. Prüfung. Hier waren sie bereits durch das 24stündige Verweilen in der Bouillon aufgefrischt resp. aktiviert worden und wuchsen rasch auf den Blutplatten. Hier hatten hereits nach 22 Stunden alle Kolonien des haem. Strept. normale Grösse und gleichmässig grosse, im Durchschnitt 1,8—2,2 mm messende Resorptionshöfe.

Es wurden nun in einem weiteren Versuche 11 Tampone, welche von anderen Scharlachpatienten (Fälle 40—50, vgl. Tab. 1) am 16., 17. und 23. XI. 1925 entnommen worden waren, bis zum 1. VI. 1926 bei einer Temperatur von 10—18° C, vor Licht geschützt, in ihrer mit Watte lose verschlossenen Eprouvette *unberührt* aufbewahrt.

Am 1. VI., also nach 197 resp. 196 and 190 Tagen, wurde von allen diesen Tamponen mit steriler Scheere ein kleines Stück abgeschnitten und hierauf mit sterilen Pinzetten über der Oberfläche der Hammelblutagarplatten zerzupft.

Tab. 3 zeigt das Ergebnis dieses Versuches:

Tabelle 3. *Anzahl der aufgegangenen Kolonien.*

Nr.	Fall	Datum	Am Tage der Entnahme*)	Am 1. VI.		Nach Tagen
				a) nach 24 Std.	b) nach 48 Std.	
1	40 Tscher.	16. XI.	50 :3	0 :2 (Staph.)	0 :4 (Staph. subt.)	197
2	41 Bal.	16. XI.	600	0 :5 (Staph.)	0 :5 (Staph.)	197
3	42 Wor.	16. XI.	500	0 :0	0 :0	197
4	43 Tsche.	17. XI.	H	9 :1	26 :2 (Staph.)	196
5	44 Tun	17. XI.	H	0 :0	0 :0	196
6	45 Wat.	17. XI.	H	3 :0	7 :0	196
7	46 Lop	17. XI	H	0 :1	0 :1 (Staph.)	196
8	47 Sim.	17. XI.	>700	3 :0	5 :0	196
9	48 Kus.	23. XI	H	0 :1	0 :2 (Staph.)	190
10	49 Kri.	23. XI.	H	1 :5	8 :5 (Staph.)	190
11	50 Dja.	23. XI.	H	100 :0	150 :0	190

Diese Versuche ergeben folgende wesentliche Resultate:

1. Die im frischen Tampon in recht beträchtlichen Mengen vorhandenen grünen Streptokokken waren vollkommen verschwunden (vgl. Tab. 1).

2. Auch die anderen verunreinigenden Begleitbakterien, fast ausschliesslich kleine weisse Staphylokokkenkolonien, waren, wenn überhaupt, nur sehr vereinzelt vorhanden.

3. Von den 11 Tamponen erhielten 5 nach 190 resp. 196 Tagen noch lebende hämolytische Streptokokken. Dies ist 45½%. Da etwa nur ein Zehntel des ganzen kontaminierten Tamponmaterials zur Untersuchung gelangte, ist anzunehmen, dass die tatsächliche Prozentzahl der positiven Resultate noch höher angeschlagen werden kann.

4. Die überlebenden hämolytischen Streptokokken zeigten meist eine deutliche Wachstumsverzögerung und erst nach 48stündiger Bebrütung waren alle Kolonien gleichmässig ausgebildet und wiesen normal grosse (2,2—2,5 mm) Resorptionshöfe auf. Diese Wachstumsverzögerung war auch hier nur vorübergehend, denn schon in der ersten Subkultur hatten bereits nach 22 Stunden die Kolonien überall normale Grösse erreicht.

5. In den von schweren und letalen Fällen gewonnenen Tamponen konnten viel länger lebende Streptokokken nachgewiesen werden, was zum Teil wohl auch auf ihre grössere Menge zurückgeführt werden kann.

*) Vgl. Tab. 1.

Um zu untersuchen, ob die hämolytischen Streptokokken auch im Tamponmaterial, das durch Abstreichen von *Streptokokkenreinkulturen* gewonnen wurde, ebenso lange am Leben erhalten bleiben, wie in ihrem natürlichen Medium in Rachenabstrichen, wurde folgender Versuch angestellt:

Die am 10. XI. durch Bestreichen mit den alten Originaltamponen (vgl. Tab. 2, 2. Prüfung) kontaminierten Blutagarplatten wurden nach der Bebrütung mit sterilen Tamponen abgestrichen, wobei die einzelnen Verunreinigungen auf den Platten nach Möglichkeit sorgfältig umfahren wurden. Die so mit Streptokokkenkolonien kontaminierten Tampone wurden in Eprouvetten unter losem Watteverschluss im Dunkeln bei 10—18° C. dauernd aufbewahrt.

Am 12. III. (1. Prüfung), nach 125 Tagen, wurden sie einige Male auf Blutagarplatten abgetupft.

Am 12. IV. (2. Prüfung), nach 151 Tagen, wurden die Tampone leicht gezupft und der dadurch erzeugte Staub auf die Blutagarplatten auffallen gelassen und verstrichen.

Am 20. V. (3. Prüfung), nach 189 Tagen, wurden die Tampone ein zweites Mal gründlich über der Blutplatte gezupft und die Platten wie in der 2. Prüfung behandelt.

Tab. 4 gibt das Ergebnis dieses Versuches.

Aus dieser Tabelle ist ersichtlich, dass von den 37 zur Untersuchung gelangten Tamponen 35, das ist über 94½%, nach über 6 Monaten noch lebensfähige Streptokokken, und zwar meist in sehr beträchtlichen Mengen, enthielten.

In einem weiteren Experiment wurde die Resistenz der Scharlachstreptokokken gegen *direktes Sonnenlicht und Austrocknung durch Wind* geprüft (1. bis 3. VI. 1926).

Es wurden hierzu fünf verschiedene Stämme, und zwar drei lange, an Tamponen getrocknete, und zwei ganz frische Scharlachstämme in Anwendung gebracht, um gleichzeitig eventuelle Unterschiede in der Resistenz dieser verschiedenen Stämme zu beobachten. Die Scharlachstreptokokkenstämme wurden ausschliesslich direkt von den Tamponen auf Blutplatten überimpft, von welchen dann die ersten Subkulturen nach 24stündiger Bebrütung mit sterilem Tampon abgestrichen wurden. Es sind dies die Stämme:

1. Wat. (Nr. 45, vgl. Tab. 1 und 3) ...196 Tage am Tampon getrocknet.
2. Sim. (Nr. 47, vgl. Tab. 1 und 3) ...196 Tage am Tampon getrocknet.
3. Raiss .....frischer Stamm, vor 2 Tagen vom Rachen des Scharlachpatienten gewonnen (letaler Fall).
4. Rum. .....frischer Stamm, vor 8 Tagen vom Rachen des Scharlachpatienten gewonnen (schwerer Fall).
5. Dja. (Nr. 50, vgl. Tab. 1 und 3) ...190 Tage am Tampon getrocknet.

Tabelle 4. *Zahl der aufgegangenen Streptococcus haemolyticus-Kolonien.*

Nr.		I. nach 125 Tg. (7. III.)	II. nach 151 Tg. (12. VI.)	III. nach 189 Tg. (20.V.)
1	Kon.	20	steril	steril
2	Sem.	50	40	9
3	Stok.	H	H	H
4	Wor.	H	H	H
5	Phil.	H	100	25
6	Ign.	10	50	50
7	Fjod.	450	200	300
8	Rub.	H	600	80
9	Kras.	600	1000	500
10	Germ.	H	H	1200
11	Sim.	H	H	1000
12	Bsar.	500	100	200
13	Zar.	2000	H	300
14	Kuch.	500	600	1000
15	Osp.	40	50	120
16	Wol. A.	100	100	200
17	Arj.	30	H	250
18	Ivan.	500	20	26
19	Wol. B.	100	100	200
20	Alek.	200	200	22
21	Pan.	verunreingt	200	500
22	Mig.	200	500	150
23	Bond.	H	H	2000
24	Klo.	H	H	H
25	Kus.	300	100	45
26	Gol.	verunreingt	2	8
27	Gla.	1500	H	H
28	Ne. L.	H	500	1500
29	Tsche.	100	30	60
31	Ne. K.	40	4	1
32	Los.	200	600	25
33	Wed.	30	steril	steril
34	Mak.	150	1000	H
35	Sij.	200	60	80
36	Büt.	H	H	H
38	Str.	H	H	1500
39	Mend.	600	2000	400

Von Nr. 30 und 37 wurden keine Platten angelegt.

Die mit den Streptokokkenrasen von den Blutplatten bestrichenen Tampone (3 cm lange und 8 mm breite, auf dünne Stäbchen aufmontierte Wattebäuschchen) wurden auf dem Geländer der Dachterrasse des vier Stock hohen Laboratoriumsturmes befestigt. Die Tampone waren von allen Seiten unbeschattet der Wirkung der Sonne und des Windes vollkommen ausgesetzt. Zu Beginn des Experimentes herrschte sehr windiges und warmes Wetter mit geringer Bewölkung.

Am nächsten Tage war warmes, ungewöhnlich trockenes, fast wolkenloses, ziemlich windiges Wetter. Um 2 Uhr nachmittags, genau nach 24 Stunden, wurden die Tampone untersucht. Sie wurden a) gründlich auf die Agaroberfläche aufgeklopft und b) kleine Stückchen mit steriler Scheere abgeschnitten und hierauf über der Blutagaroberfläche zerzupft.

Am dritten Tage [heisses, windiges Wetter*)], genau nach 48 Stunden, wurde neuerdings von jedem Tampon ein etwas grösseres Stück abgeschnieen und über Blutagarplatten zerzupft.

Am achten Tage wurden die Tampone nochmals geprüft, doch konnten bei dieser letzten Untersuchung die Resultate infolge zu starker Verunreinigung der Tamponoberfläche durch Heubacillen nicht mehr verwertet werden.

Tab. 5 gibt die Ergebnisse dieses Experimentes wieder:

Aus dieser Tabelle ist ersichtlich, dass nach 48stündigem Verweilen der Tampone im Freien bei windigem, heissem, sehr trockenem und sonnigem Wetter noch 80% der untersuchten Streptokokkenstämme lebensfähig war und normal rasch und üppig wuchs.

Datum	Temperatur °C	Barometer mm	Absol. Feuchtigkeit mm	Relat. Feuchtigkeit %	Windgeschwindigkeit Sek./m	Sonnenschein
1. VI. 7 Uhr vorm.	12,9	743,7	7,9	72	6	ununterbrochen von 6 Uhr vorm. bis 7 Uhr nachm.
1 Uhr nachm.	26,8	742,6	6,6	25	5	
9 Uhr nachm.	20,9	741,8	6,5	36	4	
2. VI. 7 Uhr vorm.	17,3	742,8	6,9	47	8	
1 Uhr nachm.	30,4	741,8	7,0	22	7	ununterbrochen von 5 Uhr vorm. bis 6 Uhr nachm.
9 Uhr nachm.	24,0	740,5	5,8	25	8	
3. VI. 7 Uhr vorm.	22,5	741,2	4,4	22	12	
1 Uhr nachm.	33,0	739,6	4,8	13	12	ununterbrochen von 8 Uhr vorm. bis 4 Uhr nachm.
9 Uhr nachm.	27,8	739,7	4,7	18	12	

*) Die meteorologischen Daten für die 3 Versuchstage (1.—3. VI. 1926), welche mir Herr *Pavloff*, Leiter der Harbiner meteorologischen Station freundlichst zur Verfügung stellte, seien hier wiedergegeben:

Tabelle 5. *Resistenz der Scharlachstreptokokken gegenüber Sonnenbestrahlung und Austrocknung durch Wind. Anzahl der aufgegangenen Str. haem.-Kolonien nach 24-bzw. 48 stündiger Exposition der Tampone.*

Tampon Nr.	1. Nach 24 Std.		2. Nach 48 Std.
	a) Oberfläche	b) Zerzupftes Stückchen	Zerzupftes Stückchen
1. Wat.	4 (stark verunreinigt durch Subtilis)	1500 (4 weisse Staph.-Kol., sonst Reinkultur)	0 (6 Heubac.-Kol., 1 kleine Staph.-Kol.)
2. Sim.	0 (4 kl. gr. Strept.-Kol., starke Verunreinigung durch Subtilis)	120 Reinkultur	60 (einzelne Subt.-Kol.)
3. Raiss.	4*) (Verunreinigung durch Subtilis)	12*) (Starke Verunreinigung durch Subtilis)	H (tot, H. durch Strept.-haem.-Kol., einzelne Subt.-Kol.)
4. Rum.	0 (viele Verunreinigungen)	30*) (5 Subt.-Kol.)	H (tot, H. durch Strept.-haem.-Kol., einzelne Subt.-Kol.)
5. Dja.	5*) (viele Verunreinigungen)	180 (1 gr. Verunreinigung durch Bac. subt.)	H (Strept. haem. fast in Reinkultur)

Da die untersuchten Stückchen stets nahe der Spitze der Tampone abgekappt wurden, war ihre Oberfläche während der ganzen Tageszeit der direkten Sonnenbestrahlung ausgesetzt gewesen.

In einem weiteren Versuche wurde *die Vitalität vergrünter Scharlachstreptokokken* im Vergleich zu ihrem Ausgangsstamme geprüft.

Durch stundenlange Einwirkung filtrierten Mundschleims eines gesunden Menschen auf eine frische Strept. haem.-Kultur eines Scharlachkranken (im Beginn des Rekonvaleszenzstadiums) gelang es, bei geringer Einsaat, grüne Streptokokkenkolonien abzuspalten. Dieselben wurden zugleich mit der hämolytischen Ausgangskultur ex tempore auf neue Blutagarplatten ausgesät und je ein Tampon mit dem Rasen gleichmässig kontaminiert. Beide Tampone wurden bei 15—17° C. aufbewahrt.

Nach 10 Tagen wurden beide Tampone über Blutagarplatten gründlich gezupft. *Resultat*: Ueppigstes Wachstum des Strept. haem. der Ausgangskultur, während die mit der vergrünten Modifikation bestrichenen Platten steril blieben.

Es erschien nun von Interesse, nachzuforschen, ob die Streptokokkenstämme durch ihr langes latentes Leben auf den trockenen Tamponen nicht in ihren Eigenschaften dauernd beeinflusst wurden.

Vor allem war es von Interesse, zu erfahren, ob sie in ihrer *Virulenz* abgeschwächt wurden.

Um dies zu ergründen, wurde zunächst folgender biologischer Versuch angestellt:

*) Da die Verunreinigungen einen mehr oder weniger grossen Teil der Platten überdecken, entzog sich auch ein grosser Prozentsatz der überwucherten Streptokokkenkolonien der Zählung.

Tierversuch mit weissen Mäusen: Auf Grund einiger Vorversuche wurde festgestellt, dass normal virulente, frische Scharlachstreptokokken nach 24stündigem Wachstum in alkalischer Bouillon in der Menge von 0.5 ccm der aufgeschüttelten Bouillonflüssigkeit Mäusen intraperitoneal injiziert, dieselben meist innerhalb 24 Stunden, spätestens nach 48 Stunden töten.

Zu diesem Versuche wurden folgende Scharlachstreptokokkenstämme auf alkalische Bouillon überimpft:

1. Eine unmittelbar vom Originaltampon Rub. (Nr. 8, vgl. Tab. 2) gewonnene Streptococcus haemolyticus-Kolonie: nach 247 tägiger Trocknung am Tampon.
2. Eine unmittelbar vom Originaltampon Gla. (Nr. 27, vgl. Tab. 2) gewonnene Streptococcus haemolyticus-Kolonie: nach 232tägiger Trocknung.
3. Eine vollkommen frische, direkt vom Tampon gewonnene Streptococcus haemolyticus-Kolonie, von einem leichten Scharlachfall, ungetrocknet (dient als Vergleich).
4. Eine unmittelbar vom Blutagartampon Germ. (Nr. 10, vgl. Tab. 4) gewonnene Streptococcus haemolyticus-Kolonie: nach 180tägiger Trocknung.
5. Eine unmittelbar vom Blutagartampon Sem. (Nr. 2, vgl. Tab. 4) gewonnene Streptococcus haemolyticus-Kolonie: nach 189tägiger Trocknung.
6. Eine unmittelbar vom Originaltampon vor 3 Tagen gewonnene Streptococcus haemolyticus-Kolonie von Fall Tsche. (Nr. 45 bzw. 4, vgl. Tab. 1 und 3): nach 196tägiger Trocknung.
7. Idem, von Fall Wat. (Nr. 45 bzw. 6, vgl. Tab. 1 und 3): nach 196tägiger Trocknung.
8. Idem, von Sim. (Fall 47 bzw. 8): nach 196tägiger Trocknung, und
9. Idem, von Dja. (Fall 50 bzw. 11): nach 196tägiger Trocknung.

Nach 24stündigem Wachstum der Kulturen bei 37° wurden die Bouillonröhrchen gut durchgeschüttelt und mit je 0.5 ccm der Emulsion eine weisse Maus intraperitoneal geimpft.

Bei der Sektion zeigten alle Tiere die Zeichen einer akuten Streptokokkensepticämie mit ungeheuren Bakterienmassen in Peritoneum, Milz und Herzblut, meist in kurzen Ketten oder in Diploform. Die sowohl vom Peritoneum als auch Herzblut angelegten Blutagarplatten ergaben Wachstum von hämolytischen Streptokokkenkolonien mit verhältnismässig kleinen, nach 24 Stunden selten 1,5 mm überschreitenden Resorptionshöfen. Vergrünung der Kolonien wurde auf den Platten nicht beobachtet.

Auffallend ist bei diesen Versuchen, dass der frische Stamm sich am wenigsten virulent erwies: die mit ihm infizierte 17 g schwere Maus blieb am längsten am Leben. Dieser Stamm wurde allerdings von einem *leichten* Scharlachfall reingezüchtet, während alle anderen zur Prüfung gelangten alten Stämme von schweren bzw. letalen Fällen herrührten.

Durch diese Versuchsreihe wurde festgestellt, dass die angetrockneten Streptokokkenstämme vollvirulent geblieben waren, ein Befund, der

Tabelle 6. *Mäuse, infiziert mit 9 verschiedenen Streptokokkenstämmen.*

Maus Nr.	Gewicht in g	Geschlecht	Infiziert mit Stamm	Getrocknet Tage	Gestorben nach Std.
1	20	♂	Rub. Originaltampon	247	18
2	17	♂	Gla. Originaltampon	232	18
3	17	♂	Sarm. Originaltampon	frisch	36
4	19	♂	Ger. Blutagartampon	189	18
5	18	♂	Sem. Blutagartampon	189	18
6	15	♂	Tsche. Originaltampon	196	30
7	21	♀	Wat. Originaltampon	196	18
8	13	♂	Sim. Originaltampon	196	30
9	15	♀	Dja. Originaltampon	190	18

im Einklang steht mit den unlängst veröffentlichten Ergebnissen von *B. Lange*[30]), welcher bei angetrockneten Tuberkelbacillen ebenfalls keinerlei Virulenzabschwächung feststellen konnte.

Ferner wurden zwei verschiedene Scharlachstämme nach der von *Bumm* modifizierten[6]) *Ruge-Phillipp*schen Versuchsanordnung auf ihre Virulenz geprüft.

Es wurde frisch gewonnenes defibriniertes Blut in Eprouvetten zu je 10 ccm verteilt, hierauf mit bestimmten Mengen einer frisch bereiteten Streptokokkenemulsion beschickt und nach gutem Durchschütteln sofort mit je 1 ccm Blut und 9 ccm 42 proz. Agar die 1. Plattenserie gegossen. Die Röhrchen mit der restlichen infizierten Blutmenge kamen hierauf in den Thermostaten, wo sie während der ganzen übrigen Versuchszeit gehalten wurden; sie wurden von Zeit zu Zeit gründlich durchgeschüttelt. Nach bestimmten Zeitabschnitten wurden in gleicher Weise neue Plattenserien gegossen. Die so gewonnenen Platten wurden im Thermostaten bei 37° bebrütet, nach 24 Stunden untersucht und die Kolonien gezählt. Die Zählung erfolgte stets mindestens 2 mal und wurde getrennt von mir und meinem Laboratoriumsgehilfen ausgeführt. Bei bisweilen sich ergebenden kleinen Differnzen wurde das Mittel der Zählungsresultate angenommen.

Besonders grosse Schwierigkeiten bereitete mir die Wahl einer entsprechenden Anzahl von Streptokokken für die Einsaat in die Blutröhrchen. Schliesslich wählte ich als Standardmass 20stündige Einzelkolonien auf Meerschweinchenblutagar, die, von mittlerer Grösse mit einem 1,6—1,8 mm breiten hämolytischen Ring, an den Randpartien der Platte, von anderen Kolonien möglichst weit isoliert, aufgegangen waren. Derartige Kolonien wurden mit der Platinschaufel vorsichtig umstochen und das Agarplättchen mit der unversehrten Kolonie im Zentrum in eine Eprouvette geworfen, welche 9 ccm steriler Bouillon enthielt. Nach sehr

gründlichem Durchschütteln wurde nun 0.1 ccm dieser Emulsion in ein neues Röhrchen mit 9 ccm steriler Bouillon übertragen und abermals gut durchgeschüttelt. 0.001 ccm dieser 2. Verdünnung in 10 ccm Blut eingeimpft, brachte so im Durchschnitt 10—100 Kolonien pro 1 ccm Blut hervor, je nach der in Anwendung gebrachten Blutart, wie durch vorhergegangene Erfahrungen festgestellt wurde.

Oft waren die Keime durch das kurze unvermeidliche Beisammensein mit dem Blute *vor* dem Giessen der 1. Plattenserie (5 bis höchstens 10 Min.) möglicherweise durch Agglutination schenbar oder in der Tat stark reduziert, was namentlich bei der Prüfung mit Rinderblut stark in die Augen fiel. Es dürfte sich hier möglicherweise um Kong'utinationsvorgänge [*Streng*[45]] handeln. Es muss demnach die Einsaat bei Blutarten, welche stärkere und rasch wirkende Bakterienagglutinine aufweisen, dementsprechend erhöht werden. Da die Bakterienemulsion möglichst rasch dem Blute einverleibt wurde, war auch der bactericide Einfluss der physiologischen Kochsalzlösung nur von sehr kurzer Dauer.

Vergleichende Untersuchungen frisch gewonnener und alter, lang getrockneter Scharlachstreptokokkenstämme wurden von mir derzeit nur mit Menschenblut als Indicator ausgeführt.

Im folgenden sind die erhaltenen Resultate tabellarisch sowie graphisch wiedergegeben.

Es wurden verwendet:

1. Scharlachstreptokokkenstamm "Ry" (sehr toxischer letaler Fall), frisch reingezüchtet. Indicator: Blut eines normalen, 36 Jahre alten Mannes. (Ueberstandene Krankheiten: Masern, Scharlach, Gelenkrheumatismus, Parotitis epidemica, Typhus abd. und exanthematicus.)

Einsaat ccm	Anzahl der Kolonien nach Stunden							
	sofort	1	2	3	4	6	8	20
0,001	97	90	88	199	257	490	632	
0,005	227	175	175	395	511	ca. 1000	V :I	fast tot, H*)
0,01	375	325	375	ca.700	1000	III :I	tot H	tot H
0,02	450	>400	>400	ca.800	1500	V :I	tot H	tot H
0,05	500	600	> 800	1500	III :I	tot H	tot H	tot H

2. Scharlachstreptokokkenstamm "Dja" (Nr. 50, ziemlich schwerer, doch zu völliger Genesung führender Fall), 190 Tage am Tampon getrocknet.

Einsaat ccm	Anzahl der Kolonien nach Stunden							
	sofort	1	2	3	4	6	8	20
0,001	70	60	11	9	18	14	15	tot H
0,002	92	75	10	6	17	40	tot H	tot H
0,005	198	112	14	13	21	15	tot H	tot H

Beim Vergleich dieser beiden Kurven fällt die stärkere und länger andauernde bactericide Wirkung des frischen Menschenblutes gegenüber dem alten, getrockneten Scharlachstamm auf. Dies könnte im Sinne einer Abschwächung dieses Stammes gedeutet werden. Es verdient jedoch,

*) Diese Zahlen geben das Verhältnis der hämolysierten Agarfläche zur nicht hämolysierten wieder.

betont zu werden, dass der frische Stamm von einem ausserordentlich toxischen, letalen Falle gewonnen wurde, während der lange getrocknete Stamm von einem zwar ziemlich schweren, aber schliesslich zu völliger Gesundung führenden Falle reingezüchtet wurde.

In keinem Falle konnte, selbst bei geringer Einsaat, völlige Sterilität des Blutes erzielt werden. Es kam schliesslich überall zu schrankenlosem Wachstum der beiden Streptokkenstämme.

Zum Schlusse wurde untersucht, ob durch das lange Verweilen der Scharlachstreptokokken an den trockenen Tamponen die Toxinbildung beeinflusst wird.

Zu diesem Zwecke wurden Hautproben nach Dick ausgeführt.

Durch mehrere Vorversuche wurde festgestellt, dass von beliebigen frischen lokalen Scharlachstämmen hergestelltes Toxin in der Verdünnung 1 : 1000 meist etwas stärkere, im wesentlichen aber ganz analoge Hautreaktionen ergibt wie das uns von *Dick & Dick* freundlichst überlassene Scharlachstreptokokken-Standardtoxin.

Es wurden von 10 mit verschiedenen Scharlachstreptokokken bestrichenen Tamponen, welche seit dem 14.III. in Eprouvetten bei hoher Zimmertemperatur über den ganzen Sommer aufbewahrt worden waren, am 25.X., das ist nach 224 Tagen, Aussaaten gemacht. Zwei davon zeigten nach 48 Stunden das Wachstum vereinzelter hämolytischer Streptokokkenkolonien, welche nach einer weiteren Ueberimpfung Reinkulturen mit allen morphologischen und kulturellen Eigenschaften der typischen Scharlachstreptokokken ergaben.

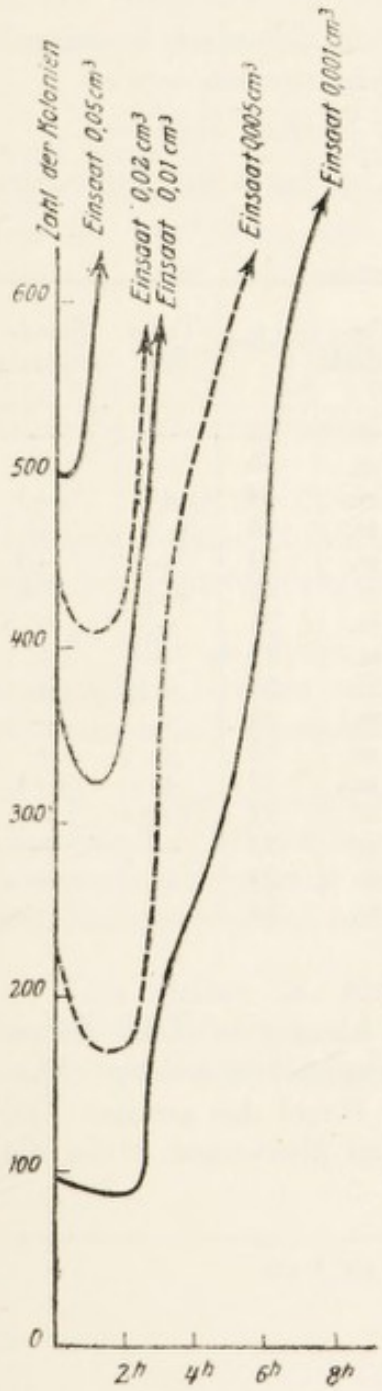

Abb. 1.

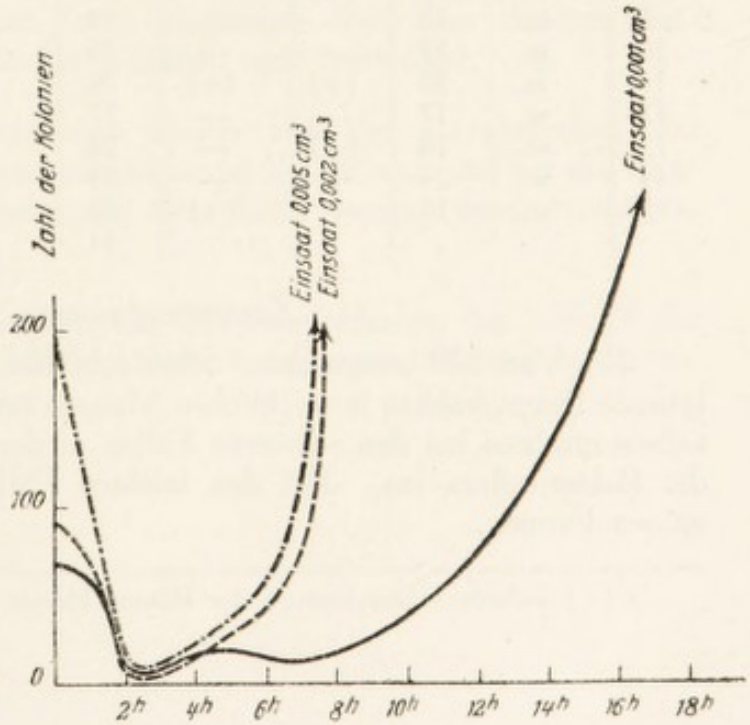

Abb. 2.

Eine dieser Kulturen "Büt" (Nr. 36 der Tab. 1 und 2; Erste Kultur vom Rachenabstrich des Kranken nach 125 Tagen Trocknen direkt vom Tampon reingezüchtet. Die erste Subkultur davon vom Blutagar mit einem Tampon abgewischt und 224 Tage in einer Eprouvette bei hoher Zimmertemperatur im Laboratorium aufbewahrt) wurde nun in mit Menschenblut beschickten Bouillonflaschen 5 Tage lang bebrütet und die Bouillonflüssigkeit durch Filterpapier und Berkefeld "N" und "W" Filter keimfrei filtriert.

Das Filtrat wurde auf 1 : 1000 verdünnt und gleichzeitig mit der ex tempore hergestellten Dickschen Standardtoxinverdünnung an 31 erwachsenen Chinesen, deren Empfindlichkeit zum Teil schon früher festgestellt worden war, geprüft. Die Impflinge erhielten an der Innenseite des linken Unterarmes das Toxin "Büt" und an der analogen Stelle des rechten Unterarmes das Standardtoxin intracutan eingespritzt.

Eine verlässliche Anamnese über stattgehabten Scharlach konnte mit Sicherheit nur bei einem Patienten (Nr. 16) aufgenommen werden.

Die Reaktion wurde 20 Stunden nach der Impfung abgelesen.

Wie aus nachstehender Tabelle ersichtlich ist, ergab sich *keine* Abschwächung der Toxinbildung.

Impfling Nr.	Geschlecht	Alter	Toxin "Büt"	Standard-toxin	Impfling Nr.	Geschlecht	Alter	Toxin "Büt"	Standard-toxin
1	m.	19	+	+	16	m.	16	—	—
2	m.	17	++	++	17	m.	16	—	—
3	m.	18	—	—	18	m.	34	+	(+)
4	m.	26	++	+	19	m.	46	++	+
5	m.	17	—	—	20	m.	29	+	(+)
6	m.	17	(+)*)	(+)	21	m.	46	++	++
7	m.	19	—	—	22	m.	26	+	+
8	m.	21	(+)	—	23	m.	27	+	+
9	m.	18	—	—	24	m.	29	+	+
10	m.	17	—	—	25	m.	26	+	—
11	m.	20	(+)	(+)	26	m.	32	+	+
12	w.	17	—	—	27	m.	32	+	(+)
13	w.	18	(+)	—	28	m.	33	(+)	—
14	w.	24	+	—	29	m.	32	—	—
15	w.	16	+	(+)	30	m.	29	+	+
					31	m.	35	++	+

Zusammenfassung.

1. Von 120 untersuchten Scharlachfällen konnten in 100% hämolytische Streptokokken in reichlichen Mengen reingezüchtet werden. Dieselben machten bei den schweren Fällen in der Regel den grössten Teil der Bakterienflora aus. Bei den leichten Fällen überwogen häufig die grünen Formen.

*) (+) bedeutet: Durchmesser der Rötung kleiner als 1 cm

2. Die auf Tamponen angetrockneten hämolytischen Scharlachstreptokokken (gleichgültig, ob die Tampone mit Rachenschleim oder Reinkulturen beschickt wurden) blieben bei 8—18°, im Dunkeln aufbewahrt, bis über ½ Jahr lang am Leben.

3. Der von solchen Tamponen abgekratzte, flugfähige Staub enthält lebensfähige, vollvirulente Streptokokken, wie durch den Tierversuch nachgewiesen werden konnte.

4. Von der auf Tamponen angetrockneten Streptokokkenflora (Rachenabstriche von Scharlachkranken) halten sich die hämolytischen Streptokokken am längsten, während die grün wachsenden Arten bereits nach einigen Wochen Trocknen völlig abgestorben sind. Es begünstigt demnach der Trocknungsvorgang geradezu die Reinzüchtung der hämolytischen Streptokokken, was zahlenmässig an einer grossen Anzahl von Tamponen festgestellt werden konnte.

5. Die lange getrockneten hämolytischen Streptokokken zeigten kulturell meist eine anfängliche Wachstumsverzögerung, die nach 48 stündiger Bebrütung aber bereits wieder ausgeglichen war. Im Tierversuch und bei der Prüfung nach der Ruge-Philippschen Methode erwiesen sich über ½ Jahr lang getrocknete Scharlachstreptokokken als vollvirulent.

6. Die Toxinbildung der Scharlachstreptokokken wurde durch langes Trocknen (125 + 224 Tage) am Tampon nicht beeinflusst, wie durch Dicksche Hautreaktionen festgestellt werden konnte.

7. Durch den Speichel eines Gesunden konnten hämolytische Scharlachstreptokokken künstlich vergrünt werden. Derartige Formen waren, wenn auf Tamponen angetrocknet, bereits nach einigen Tagen abgestorben.

8. Die in dünner Schicht auf Watte angetrockneten Scharlachstreptokokken werden selbst durch tagelanges Einwirken direkter Sonnenstrahlen und atmosphärischer Einflüsse nicht vernichtet.

9. Diese Versuchsergebnisse weisen auf die Langlebigkeit der hämolytischen Scharlachstreptokokken in der Aussenwelt und auf die möglicherweise überragende Bedeutung ihrer Uebertragung durch trockenes, staubhaltiges Material hin.

Den Herren Dr. *Korjelkin* und *Meibom*, *Harbin*, bin ich für die freundliche Erlaubnis, das Material ihrer Abteilungen benutzen zu dürfen, zu grossem Danke verpflichtet.

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VITALITY OF SCARLET FEVER STREPTOCOCCI

(English Summary).

1. Hemolytic streptococci were obtained from every one of the investigated cases of Scarlet Fever. In severe cases the streptococcus hemolyticus predominated while in mild cases the green forms of the organism often prevailed.

2. The hemolytic scarlet fever streptococci dried on gauze and kept in a dark room at 8 to 18 C. remained alive for more than half a year.

3. The dust scratched off from such contaminated swabs contained viable and fully virulent streptococci, as confirmed by experiments upon white mice.

4. Out of different strains dried on swabs, the hemolytic streptococci survived longest, while the green ones died out after some weeks of drying. The drying process is, therefore, favorable for the pure cultivation of hemolytic streptococci in scarlet fever.

5. Hemolytic streptococci dried on swabs for more than half a year were still virulent, as proved after Ruge-Philipp's methods and also by biological experiments.

6. The production of toxin was not influenced by the permanent drying process (125 + 224 days) as proved by numerous Dick tests.

7. Hemolytic streptococci, when mixed with the filtered saliva of a healthy individual, could be transformed into the green variety. Such transmutated forms died easily within a few days after drying.

8. The scarlet fever streptococci (hemolytic), when dried in thin layers on cotton wool and exposed to direct sunlight and atmospheric influences, remained alive after two days.

9. The results of these experiments prove the longevity of the hemolytic streptococci outside the human body, and the possible significance of their transmission through dry dusty material.

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(Serologist, P.P.S.)

ON THE DISTRIBUTION OF BLOOD GROUPS AMONGST CHINESE IN FUCHIATIEN (HARBIN).

(*Reprinted from National Med. Jl. of China*, 1928).

It has been well established by modern investigators that the isohemagglutinating qualities of the human blood form an important anthropological and ethnographic feature. Since L. and H. Hirszfeld published their fundamental paper on the subject, numerous authors have made additional investigations on most nations throughout the world.

In China also several investigations were made to determine the blood index of the Chinese. The first paper dealing with this question was that of Kilgore & Liu. Examining 100 Chinese of Shanghai they found a biological index of *1'3*. J. Heng Liu and H. S. Wang made an examination of 1000 Chinese in Peking, and found a blood index of *0'8* for the Northern Chinese. This means that the Chinese of the South have a higher index. These results correspond to the data reported by B. Liang when investigating 1000 Chinese in Shanghai. The total index, found by this author was *1'13*. His Chinese were inhabitants of the middle and the southern provinces of Shantung, Anhwei, Kiangsu, Szechwan, Chekiang and Kwangtung. When he calculated the index separately for each of these six provinces, he found that the most southern (Kwangtung) had the highest (*1'26*), and the most northern province examined (Shantung), the lowest (*0'89*) index.

This author explains the lower index of the Northern Chinese by the fact that the inhabitants of these provinces have mixed long ago with the Mongols and Manchus. The latter nations are known to have a lower blood index than the Chinese themselves. On the other hand, the inhabitants of Kwangtung and the neighbouring provinces are relatively "pure" Chinese, and therefore have a higher index.

Examinations of the blood of 1500 Chinese from Honan and some other Southern Provinces performed by Li Chi Pan confirmed these statements. The total index found by this author was about *1'5*. Speaking generally, the blood index of the "pure" Chinese is considerably higher than *1'0*.

Investigations of some other authors (Bais and Verhoff, Coca and Deibert, Fukamachi) on a smaller number of persons led to the same results: The index of the Chinese as a whole is about *1'0*, the index of the "pure" Chinese of the South is higher than *1'0*.

Thus, as may be seen from the preceeding statements, the biological index of the southern and middle provinces of China has been thoroughly investigated, while examinations of the inhabitants of North Manchuria are still lacking.

It would be of interest, therefore, to study the blood index in the latter, because there the influence of other races, such as Mongols and Manchus, must be especially marked. Examinations of Koreans, Man-

chus and Mongols showed that the biological index of these races is considerably lower than that of the "pure" Chinese.

Thus the index of the Koreans is *1'1*, according to Fukamachi and *1'01* according to Kirihara, while according to W. Liang it is even less than *1'0*. Fukamachi stated that the biological index of the (Southern) Manchus is *0'75*, and Jettmar found an index of *0'8* among the Mongols of Outer Mongolia.

Our own investigations were made in Harbin, and include up to date only 668 cases, mainly from Kirin, Fengtien, Chihli and Shantung.

We examined the blood of:

1. 55 students of the Harbin Medical School,
2. 22 men from the staff of our hospital,
3. 12 patients of the hospital, and 47 outdoor patients,
4. 127 children of the orphanage Tun San Juan,
5. 28 men from the staff of the firm Ta Lo Sin, and
6. 377 shopmen and workmen of the firm Tung Kee & Co.

Total ..... 668 cases.

As to the technic of our examinations, we used the so-called open slide method by Moss-Lee-Vincent.

Although some authors do not support this method, (Lattes, Schiff) it gave us as satisfactory results as the test tube method, as hemotests of high titre were used (higher than 20), and great care was taken in performing the reaction. The hemo-tests were at first compared with the hemotest of the Serotherapeutical Institute, Vienna; then different dilutions of these were titrated. Only those which agglutinated the blood corpuscles of the other group in a dilution of at least 1:20 were chosen.

To prevent as far as possible autoagglutination (according to Lattes Panagglutination). the following precautions were taken:

1. The sera to be used for hemotest were kept in the refrigerator so as to be separated from the clot.
2. Before titration the sera were kept for 5-7 days at room temperature.
3. The reaction was performed at warm room temperature (according to Lattes the optimum is 25°C).
4. Owing to the high specificity and activity of our hemotests the reaction could be observed already within one minute, and was controlled and registered after 3 minutes. In this way the result was always obtained before a non-specific reaction could have taken place. In the few doubtful cases iso-agglutination was distinguished from autoagglutination by adding a drop of isotonic sodium chloride solution to the mixture of blood corpuscles and serum. We used only hemotests within two months of issue. In every case the blood was taken from the finger.

Out of the 668 cases

188 = 28'1% belong to the group O,
197 = 29'5% ,, ,, ,, ,, A,
222 = 33'2% ,, ,, ,, ,, B, and
61 = 9'1% ,, ,, ,, ,, AB.

Special attention was paid as to where the patients originated from. In every case it was registered, where their parents came from, and if possible even where their forefathers lived.

If the cases are classified after the four provinces, from which most of them originated, the following results are obtained:

Province:	*Group 0%.*	*A%.*	*...B%.*	*AB%.*	*Total cases.*	*Biochemical index.*
KIRIN	33'3	26'3	33'3	7'1	99	0·8
FENGTIEN ...	32·0	27'2	31'1	9'7	103	0'9
CHIHLI	27'8	28'4	34·7	9'1	334	0'85
SHANTUNG ..	22'9	31'6	36'9	8'7	114	0'9

Though the number of persons is so far very small, the above mentioned table shows clearly that at any rate the biological index is remarkably lower than *1·0*.

It may be concluded from the fact that other nations have considerably influenced the blood index of the Chinese population of North Manchuria.

We intend to continue our investigations in order to contribute in this way to the ethnographical analysis of the Chinese nation.

Finally, we give a table of the results obtained by different authors who have investigated the blood index of the Chinese and the neighbouring races.

Author.	*Race (locality of investigation)*	*Number of investigated persons:*	*GROUP:* 0%	A%	B%	AB%	*Index.*
BAIS & VERHOFF	East Indies (Sumatra)	546	43'7	23'0	29'0	44'3	*0'82*
BAIS & VERHOFF	East Indies (Java)	1346	39'9	25'7	29'0	5'4	*0'9*
HIRSZFELD	Indo-Chinese	500	42'0	22'4	28'4	7'2	*-0'8*
BAIS & VERHOFF	Chinese (East Indies)	592	40'2	25'0	27'6	7'2	*0'92*
KILGORE & LIU	Chinese (Shanghai)	100	28'0	36'0	25'0	10'0	*-1'3*
LIU & WANG	Chinese (Peking)	1000	30'0	25'0	34'0	10'0	*0'79*
COCA-DEIBERT	Chinese (America)	111	29'0	32'0	29'0	10'0	*1'08*
W. LIANG	Chinese (Shanghai)	1000	38'8	30'3	25'7	6'0	*1'13*
W. LIANG	Chinese (Shantung)		21'1	31'6	36'8	10'5	*0'89*
W. LIANG	Chinese (Anhwei)		47'9	21'7	21'7	8'7	*1'00*
W. LIANG	Chinese (Kiangsu)		39'0	29'7	26'4	4'9	*1'11*
W. LIANG	Chinese (Szechwan)		44'8	28'9	23'7	2'6	*1'19*
W. LIANG	Chinese (Chekiang)		37'0	29'8	22'5	10'7	*1'22*
W. LIANG	Chinese (Kwangtung)		40'0	31'4	23'8	4'8	*1·26*
LI CHI PAN	Chinese (South)	1500	31'3	38'1	20'7	9'9	*1'5*
LI CHI PAN	Chinese from Honan only	1926	31'9	38'9	19'5	9'8	*1'6*
JETTMAR & LIN	Chinese (Harbin)	668	28'1	29'5	33'2	9'1	*0'9*
JETTMAR & LIN	Chinese prov. KIRIN	99	33'3	26'3	33'3	7'1	*0'8*
JETTMAR & LIN	Chinese prov. FENGTIEN	103	32'0	27'2	31'1	9'7	*0'9*
JETTMAR & LIN	Chinese prov. CHIHLI	331	27'8	28'4	34'7	9'1	*0'85*
JETTMAR & LIN	Chinese prov. SHANTUNG	114	22'9	31'7	36'9	8'7	*0'9*
FUKAMACHI	Chinese	80	37'7	33'7	25'0	7'6	*1'2*
FUKAMACHI	COREANS	363	28'2	32'8	26'4	12'6	*1'1*
KIRIHARA	COREANS	948	26'3	32'7	32'2	8'8	*1'01*
FUKAMACHI	MANCHUS	199	26'6	26'6	38'2	8'6	*0'75*
JETTMAR	Inhabitants of Outer Mong.	166	26'5	27'7	30'7	15'1	*0'93*
JETTMAR	MONGOLS only	112	28'6	23'2	31'2	16'9	*0'83*

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BIOCHEMICAL RACE INDEX OF MONGOLS.

(Reprinted from National Med. Jl. 1928).

While numerous investigations have been made to determine the blood index of the Chinese in different parts of the Republic, as far as I know, none have been made on Mongols. The average Mongol is very suspicious of foreign methods, and it is not so easy to get his support for blood investigations.

This is one of the reasons why my material is not so big as it should have been for a thorough analysis of blood groups among this people. The persons examined by me were from Urga and its surroundings; 114 of them were Mongols, 50 Buriats, one a Tungut, and one a Bargine. Two-thirds of them were inhabitants of jurtes around the Veterinary Station which is situated 20 km westwards of Urga. Thus almost the entire population of a valley was examined, and not "selected" cases, as it often occurs when samples are taken from hospitals, barracks, and so on. Only one third of my material consisted of the pupils of the school for veterinary dressers and of the staff of the Municipal slaughter-house.

Marriages between Mongols and Buriats are rarely observed, and all the examined Mongols were "pure" representatives of their race.

The investigations were made after the method of Moss-Lee-Vincent with high titred (higher than 1 :20) hemotest. In many cases a control was made with the hemotest of the Serotherapeutic Institute, Vienna. The results were the same.

The following table shows the results obtained :

Group :	*0*	*A*	*B*	*AB*	*Index*	*No of cases*
TOTAL	26'6	27'7	30'7	15'1	*0'93.*	166
MONGOLS only	28'6	23'2	31'3	16'9	*0'83.*	114

(The relatively high percentage of the AB-group originated from a large family whose members mostly belong to this group.)

As may be seen by this table, the biological blood index of the Mongols is considerably lower than *1'0.* It is similar to the Chinese of the Northern Provinces who in their turn have a remarkably lower index than the Southern Chinese. This fact may seem somewhat strange at first because Mongols and Chinese are quite different as to their language and habits. The explanation for this fact is that the Chinese of the Northern provinces have mixed with other races of lower index (Manchus, Mongols and others) on a large scale.

Although the number of investigated Buriats is very small, the result shows anyhow that the titre of this race is much higher. The Buriats in Mongolia are mostly immigrants from Soviet Russia, and have mixed with the Russian people, whose index is higher than *1'5.*

I am much obliged to Mr. Dsham Dsaran Dsurun, the toushemyl (comptroller) of the Veterinary Station who made it possible for me to start with these investigations.

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ON THE COMPARATIVE FREQUENCY OF NON-PULMONARY TUBERCULOSIS IN NORTH CHINA.

(Reprinted from the National Medical Jl. 1928).

That tuberculosis is exceedingly common among the Chinese is well-known. We see so much of it in our daily practice. Young adults from every social stratum are struck down in the prime of their lives and succumb to this "white plague". In Hongkong where mortality statistics are well kept, the rate is 311 per 100,000 living in 1922. In Shanghai, the figures from 1923, 1924, 1925, 1926 were 102, 108, 109 and 140 respectively per 100,000 living. Whereas in London there were only 95 per 100,000 living in 1925, and in the United States, registration area, 114 per 100,000 living. Also it has been shown that the mortality from tuberculosis among the Chinese living in the United States is relatively high. The United States Census for 1900 gives 658.5 per 100,000 living, nearly four times that of the general white population. Nor is this all. For no one can ascertain accurately the morbidity rate and the melancholy picture to be conjured up in this connection is appalling.

Having touched upon this devastating disease in a general way, it may be worth while to draw attention to the distribution of the different forms. An attempt will be made to show that the non-pulmonary forms are exceedingly rare in tropical areas such as the Federated Malay States. By non-pulmonary forms we mean tubercular involvement of the lymphatic glands, bones, joints, skin, peritoneum, intestines, etc. As we look away from the equator, more non-pulmonary tuberculosis is encountered until it reaches a big proportion in North China. As we shall see, the pulmonary form comprises of but a small percentage of all forms of tuberculosis seen at the Harbin Hospital out-patients department.

Dr. J. Tertius Clarke (1) stated that for seven years (up to 1903) he had not seen a case of non-pulmonary tuberculosis in Malaya. Nathan Raw also remarked on the rarity of these forms of tuberculosis, and explained it by the opinion that the human and bovine bacilli cause separate and distinct lesions in the human body. Namely that the bovine bacillus was probably the cause of tabes mesenterica, tuberculosis of lymph glands, tubercular joints and lupus, whereas phthisis pulmonalis, laryngeal phthisis and primary intestinal lesions were caused by the human bacillus. As there is no bovine tuberculosis in Malaya, it seems as if Nathan Raw's theory is correct. We know now, however, that there is no such distinct etiological demarkation between pulmonary and non-pulmonary tuberculosis, because bovine bacilli have been recovered from pulmonary tuberculosis and human bacilli from the non-pulmonary forms. Whatever is the explanation, the fact remains that according to the Government Medical Report of the F. M. S., in Parak for instance, out of 96,000 deaths recorded not one was ascribed to non-pulmonary tuberculosis, and in the annual report of the Medical Department of the F. M. S. for 1925, under the heading "Tuberculosis" there were 2571 cases treated in the Government hospitals, but under "local

diseases" such as those of joints, bone, skin, glands, there is no mention of tubercle. In that part of the report relating to the surgical work of the chief surgeon of Perak and Lelangor out of 1860 operations there was only one case of tubercular gland of the neck.

Coming up further north, in Hongkong, we find that out of 5381 cases treated in the Civil Hospital in 1926, there were 121 cases of pulmonary tuberculosis, 30 cases of tubercular lymphatic glands, and 32 of joints.

TABLE I.

	P.U.M.C. (1916-1917) Out-pat. D.		*Shanghai Red Cross (1917-1918) Out-pat. D.*		*Tsinan (1921-1922)*		*Shantung Rd. Hospital (1917, 1919, 1922, 1924 & 1925 in-pat.)*	
		%		%		%		
Lung	117	41.2	80	63.5	469	66.5	189	42.6
Bone	50	17.6	6	4.7	85	12.0	74	16.7
Glands	84	29.3	20	16.0	105	14.8	64	14.4
Abdomen	9	3.2	4	3.1	44	6.2		
Joints	24	8.4	16	12.7	3	.4	116	25.9
	284	99.7	26	100.0	706	99.9	443	99.6

TABLE II.

HARBIN HOSPITAL

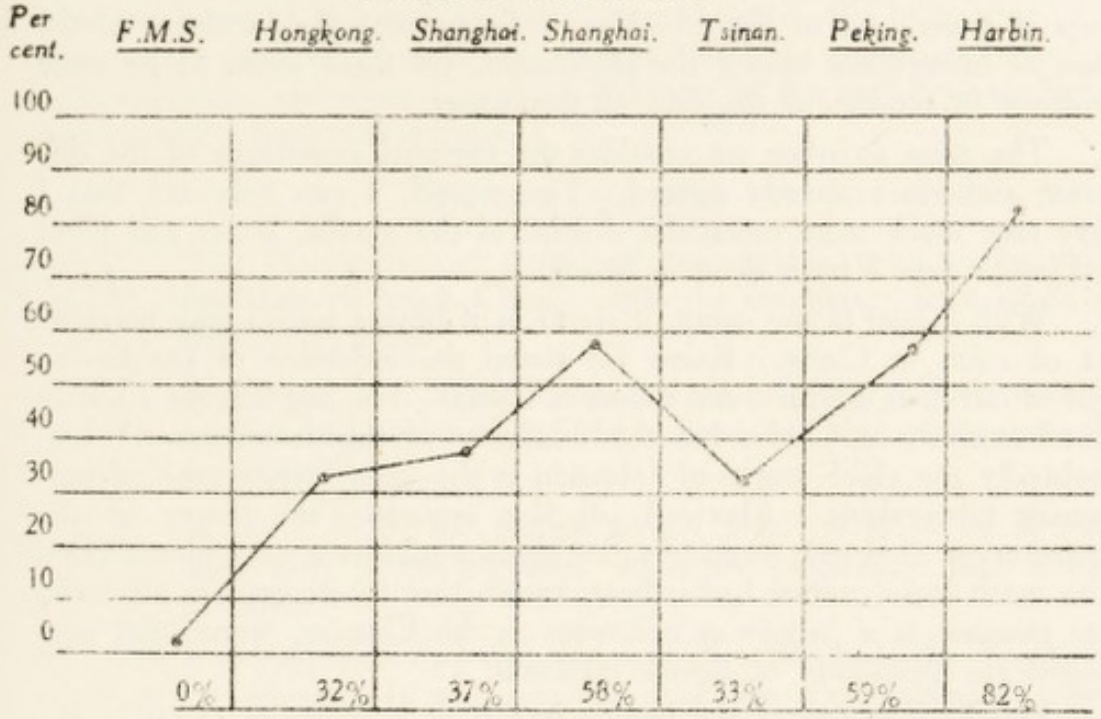
Out-patients

April 1924—Sept. 1927.

	Number.	*Percentage.*
Lung	538	18.1
Bone	620	20.9
Skin	327	11.1
Abdomen	28	0.95
Glands	1065	35.9
Testis	41	1.4
Joints	296	9.9
Elsewhere	47	1.6
		99.85

TABLE III.

PERCENTAGE OF NON-PULMONARY TUBERCULOSIS TRACED FROM F. M. S. TO HARBIN.



From Table I we gather that in Shanghai, the Shantung Road Hospital in-patient figures for five years show 42.6 per cent. of pulmonary tuberculosis, 16.7% of bone, 14.4% of glands and 25.9% of joints (of all forms of tuberculosis). The Shanghai Red Cross Hospital out-patient figures show 63.5% of the pulmonary form. The Tsinan University Hospital in-patient figures show 66.5%. As we reach Chihli province, the P.U.M.C. out-patient records show 41.2% of the pulmonary form while the glandular form has risen to 29.3%. The Report of the Roberts Memorial Hospital, Tsangchow, Chihli (2) mentions "these cases of tubercular glands of the neck, together with tubercular bone and joint cases provide us with the majority of the operations in the women's hospital. ..... Some of the bone cases live with us for months." The Medical Officer in charge expounds on the predisposing causes of tuberculous cervical glands in this way :— The stalks of the giant millet are still universally employed as fuel in the country districts. The young women burn them under the cooking pots, and the room is quickly filled with acid choking fumes. They are quite accustomed to this and do not mind it, but the delicate lining of the nose objects to such treatment, and quickly becomes inflamed. Dust storms blow every few days in the spring, and aggravate matters, and soon ulcers begin to form. These give little trouble, however, until lumps begin to form in the neck. Consumptives there are in almost every family, and hygiene is unknown. Soon the inflamed glands get infected with tubercle, and the subsequent history of the patient is a long misery of unsightly sores and undermined health.

When we come to Harbin, of all forms of tuberculosis only 18.1% are comprised of the pulmonary form, 35.9% are of the glandular form.

Though statistical evidence is not absolute, though we quote in-patient records where we fail to obtain out-patient figures and though hospital records are of limited value of determining the incidence of the form of tuberculosis among the population, yet there seems to be some evidence in support of the title of this paper.

The more so when we consider the personal experience of the different authorities already quoted. For myself, I can only say that I have seen much more tubercular disease of the glands, bones and joints in Harbin than I have done in Shanghai.

With regard to the cause, it seems as if bovine bacilli may be ruled out of court in China. Korns (3) stated the incidence of the bovine type of infection in man is not known in China. The hog may be a source of tuberculosis in China, but the incidence of this is unknown. Undoubtedly the chief source of infection is the open human type of pulmonary tuberculosis. Maxwell (4) also remarks "the theory of the bovine origin of certain forms of tuberculosis would have been promulgated with much more caution had it been known that tuberculosis in all forms was rampant in a people as numerous as the Chinese, whose diet precluded any possibility of bovine infection."

Having disposed of the bovine bacilli as a causative factor in the prevalence of the non-pulmonary form in North China, we have to consider the climatic and housing conditions. It is possible that the cold and dry climate does not predispose to pulmonary tuberculosis whereas the hot and moist climate in the tropical and subtropical areas has an opposite effect. Moreover, the cold climate forces people to live in confined spaces, thus causing overcrowding. The lack of hygiene among the common people in the presence of open pulmonary cases still favours the spread of tuberculosis in all forms.

This brings us to the big problem of tuberculosis. Whether the introduction of sanitation has by itself caused the diminution of the disease in European countries or not need not be discussed. All, however, are agreed that phthisis is an infectious disease, that the risk of infection depends in part on the quantity of bacilli to which the individual is exposed, and that the bacilli have the best chance to infect people in dark, damp, ill-ventilated and over-crowded houses.

Sir Arthur Newsholme showed that the most important factors in the decrease of the disease in England were :—

1. Better housing,
2. Improvements in the habits of the people,
3. Better segregation of the sick.

He said "improved housing doubtless increases the resistance to tuberculosis, still more it implies diminished opportunities for infection."

Dr. Clarke (5) submitted that the most important needs are the removal of the advanced, open, and therefore the most infectious cases from the houses.

In China, until the sanitary conscience of the nation is awakened, the two above desiderata will be difficult to obtain. This much we can do, and that is to educate the people both in personal hygiene and in simple ideas of Public Health, to the end that in the fullness of time, we may see the awakening of the national conscience in matters connected with sanitation, and show a national reduction in the appalling morbidity and mortality from tuberculosis.

CONCLUSIONS.

1. The Non-pulmonary forms of tuberculosis seem to be more prevalent in North China, while the pulmonary form is not so frequently seen.
2. In Central and South China the non-pulmonary forms seem to be less in proportion and the pulmonary form correspondingly more prevalent.
3. In the F.M.S the non-pulmonary forms seem to be exceedingly rare, unless imported, while the pulmonary form is very prevalent.
4. It is suggested that the cold and dry climate in the North has an influence on the prevalence of the non-pulmonary forms, either per se, or together with the ill-ventilated and crowded conditions of housing obtained in those regions, and that the hot and damp climate in the tropics favours the pulmonary form of tuberculosis.

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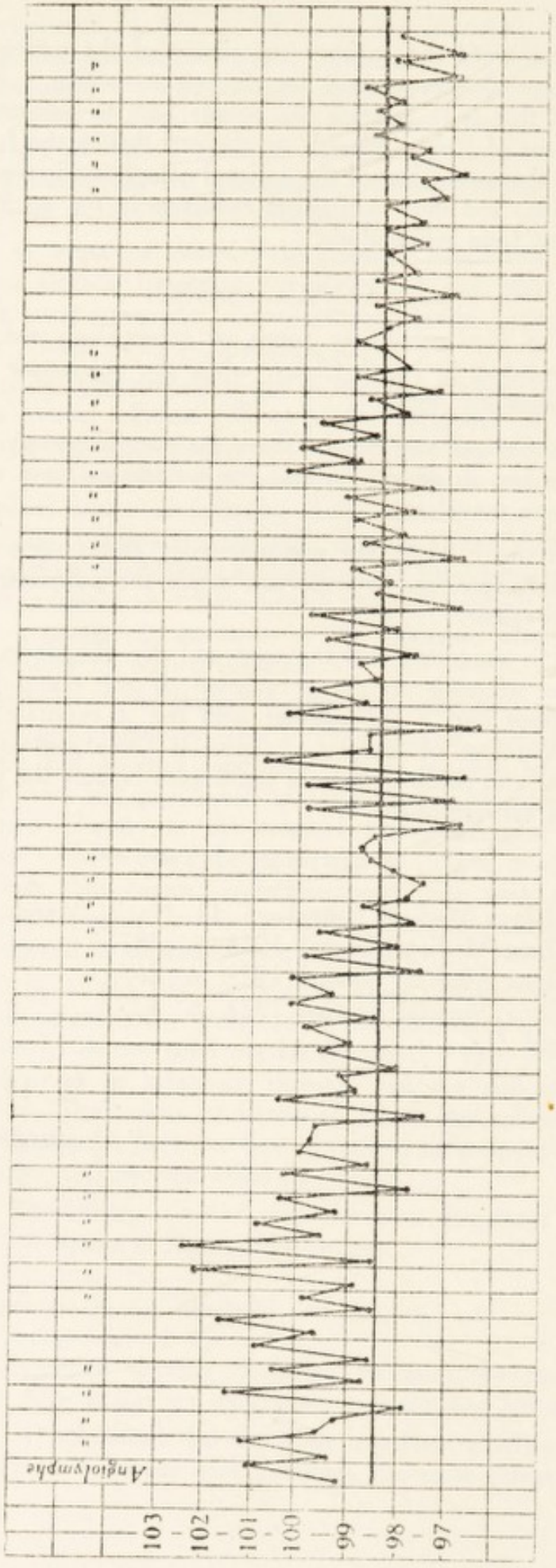

Temperature Chart
A case of tuberculous adenitis.

A CASE OF TUBERCULOUS ADENITIS WITH CONSTITUTIONAL SYMPTOMS TREATED WITH ANGIOLYMPHE.

(Reprinted from the National Med. Jl. 1927).

On the 5th May 1927, Mrs. Han, nullipara, aged 16, came to us for treatment for tuberculous adenitis of the right neck. The history was that three or four months ago she noticed there was a swelling of the neck which was gradually increasing in size, but with no accompanying pain. Past history was negative, except there was cough in the winter. No serious illness of any kind was recorded.

On examination, she was fairly well nourished, pale complexioned, with a hectic flush on each cheek. She did not appear to be ill. The temperature was 102° F., and pulse 97 per minute. There were two enlarged glands under the right jaw, and in front of the sterno-mastoid muscle. Each of the glands was about one inch in diameter, non-tender and not matted together. The teeth and tonsils were healthy. No other glands were palpable in the outer parts of the body.

On examination of the lungs, the back of the left lung showed dullness on percussion. Tubular breathing was heard at the upper part and harsh breathing was heard at the lower part. No crepitations were heard anywhere. No abnormal signs were found elsewhere. The sputum was repeatedly examined for B. tuberculosis, but none was seen.

Other organs in the body were all found to be healthy.

Von Pirquet reaction was done, and found to be strongly positive. For four weeks after admission, and inspite of the rest in bed, a remittent fever was kept up daily as shown by the temperature chart. During this time, expectorants, quinine, syrup ferri iodide and creosote were given in rotation, but no effect on the temperature was apparent.

Commencing on the 2nd June, one ampoule of Angiolymphe was injected subcutaneously every day for six days. At that time, the temperature was oscillating between 99 and 101° F.

On the 9th June, two ampoules were injected daily for six days.

On the 21st June, one ampoule was given daily for six days. At this time, the temperature was oscillating between 98.5 and 100 F.

On the 8th July, a course of twelve injections was started. Now the temperature came down to normal for several days, but the pulse was still high.

A final course of six injections was given from the 25th July, and the patient was discharged on the 1st August. By this time, the temperature and pulse were normal; she was fatter, and felt well. She had a good appetite; the hectic flush was gone; there was no cough or sputum. The glands were not affected in any way, however, for they have not diminished in size.

Von Pirquet reaction was done again, and this time it seemed to be more strongly positive. ...

As the temperature came down, she was allowed to get up and do a little needle work. She was encouraged to take gentle walks in the garden and to sit in the sun as much as possible.

After the first four weeks, and since the angiolymphe injections were started, no medicines of any kind were given.

Altogether then, fourty-two ampoules were given, spreading over a period of two months. The result may be summed up as satisfactory, so far as the constitutional symptoms are concerned.

Now as to a word about Angiolymphe. To quote the pamphlet which accompanies the ampoules, "Angiolymphe is a perfectly harmless plant extract which may be given with success in all forms of tuberculosis. Angiolymphe has a curative effect on tuberculosis of the lungs, larynx, skin and bones, in all cases of surgical tuberculosis, in urino-genital cases (tuberculosis of the testicle and ovary), in miliary tuberculosis, in short, in every case in which the morbid condition can be traced to infection with tubercle bacilli. Apical catarrh, which has just begun or has lasted for some time, is completely cured, often in a surprisingly short time, by Angiolymphe. Even in very severe cases of tuberculosis (if they are not quite hopeless) Angiolymphe effects at least an unmistakable and permanent improvement in the general condition and, as a rule, a complete recovery.

Angiolymphe differs from tuberculin and similar remedies in not being a vaccine or serum, it is a chemo-therapeutic preparation. It is a plant extract prepared from various Irideae, and it contains as its active principle glucosides of these plants, but no alkaloids. This plant extract is prepared in a special manner and is sterilised so as to be suitable for injection."

Dr. Rous of Paris was the originator of this preparation. The formula is given as follows :—

Orchis maculata	15 g.
Ixia rosea	5 ,,
Morea sinensis	4 ,,
Aqua distillata	1000 ,,

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SOME CLINICAL NOTES (HARBIN HOSPITAL).

(Reprinted from the National Med. Jl. 1927).

Novasurol as a Diuretic.

Li, aged 49, was admitted for double mitral regurgitation. He was cyanotic, and short of breath. His feet and legs were swollen, showing the usual picture of heart decompensation. He complained also of cough and sleeplessness. He was fairly well nourished and well built. There was no history of rheumatism or syphilis. He stated that three months ago he found his feet began to swell, more so during the past month.

On examination, his heart was enlarged leftwards and downwards, the apex beat being in the 6th space and ½ inch outside the nippleline. Systolic and diastolic murmur was heard at the apex and conducted towards the axilla. No murmurs heard at the aortic area. The right heart seemed to be slightly enlarged.

The chief complaint was scanty urine, which was dark in colour, but containing no albumen or casts. Digitalis, strophanthus and diuretin were given with no benefit. After six days' trial, novasurol was injected approximately thrice weekly. The result was very encouraging. Diuresis was improved and the feet were no longer swollen, the patient began to walk about three weeks after the commencement of the injections. The most striking effect was when the novasurol immediately started urinary secretion after suppression for 4 days.

At the end of the 7th week, however the patient seemed to have got worse. Digalen was given three times daily but did not help him. He gradually lost ground. His cough was blood-stained towards the end and he died soon afterwards.

The charts may show more clearly the effect of the novasurol on the urine secretion.

Novasurol in a Case of Chronic Nephritis.

Mr. Chu, a young man of 23, an office worker, complained of swollen legs and face for one year.

Family History. Nil.

Past History. No fevers of any kind, no syphilis.

Clinical History. One year ago he noticed his face began to be puffy. Then gradually his legs were involved. He noticed he was short of breath. He coughed up quite a lot of yellow sputum. His urine became scanty and thick. He was treated by many doctors and with many kinds of medicine.

On examination he was puffy all over, and very anæmic. His heart was somewhat enlarged, the second sound being accentuated.

Blood pressure :— Systolic 130, diastolic 90.

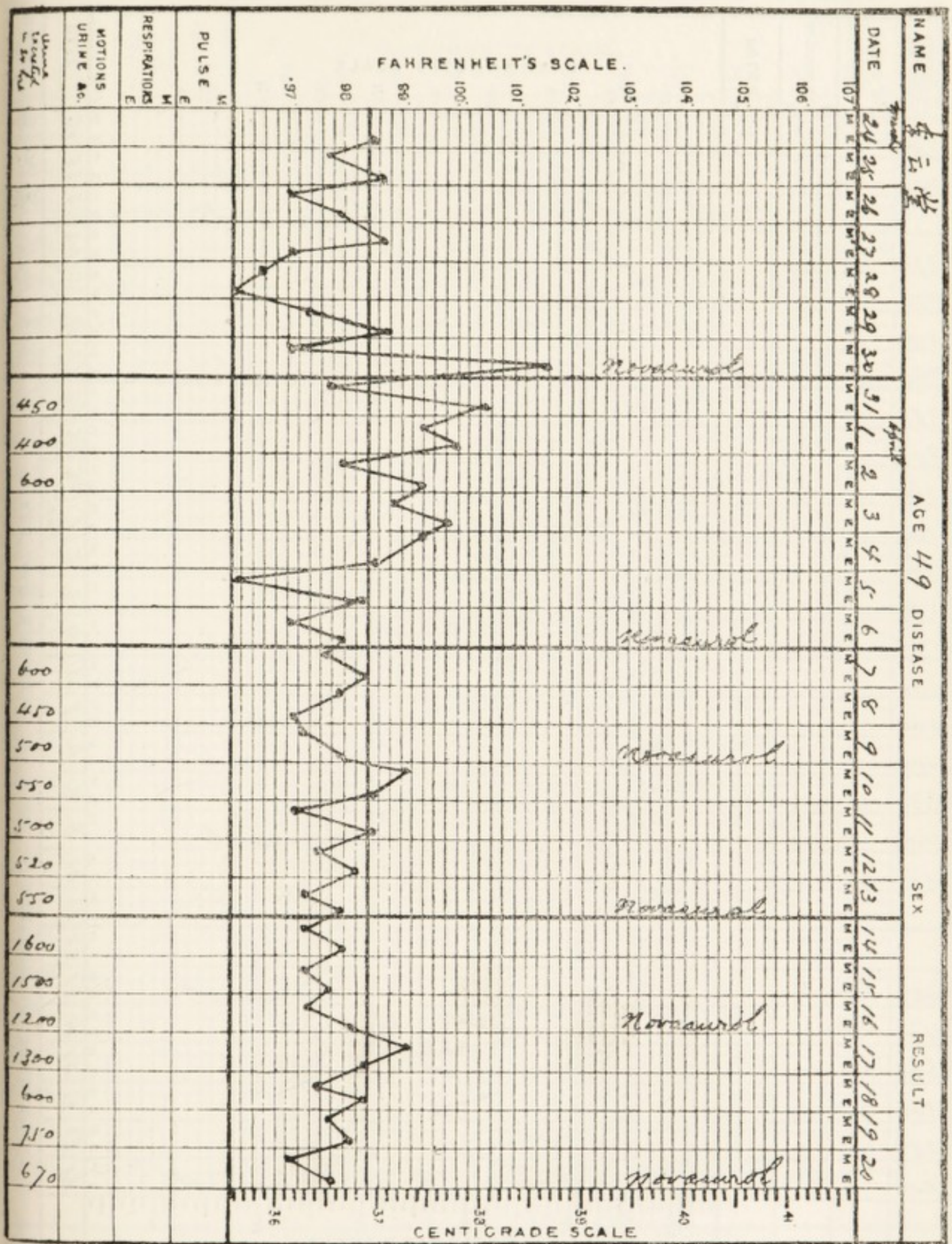
Urine : Sp. gravity 1025; Acid; Albumen, a thick cloud; No sugar; casts, granular and hyaline.

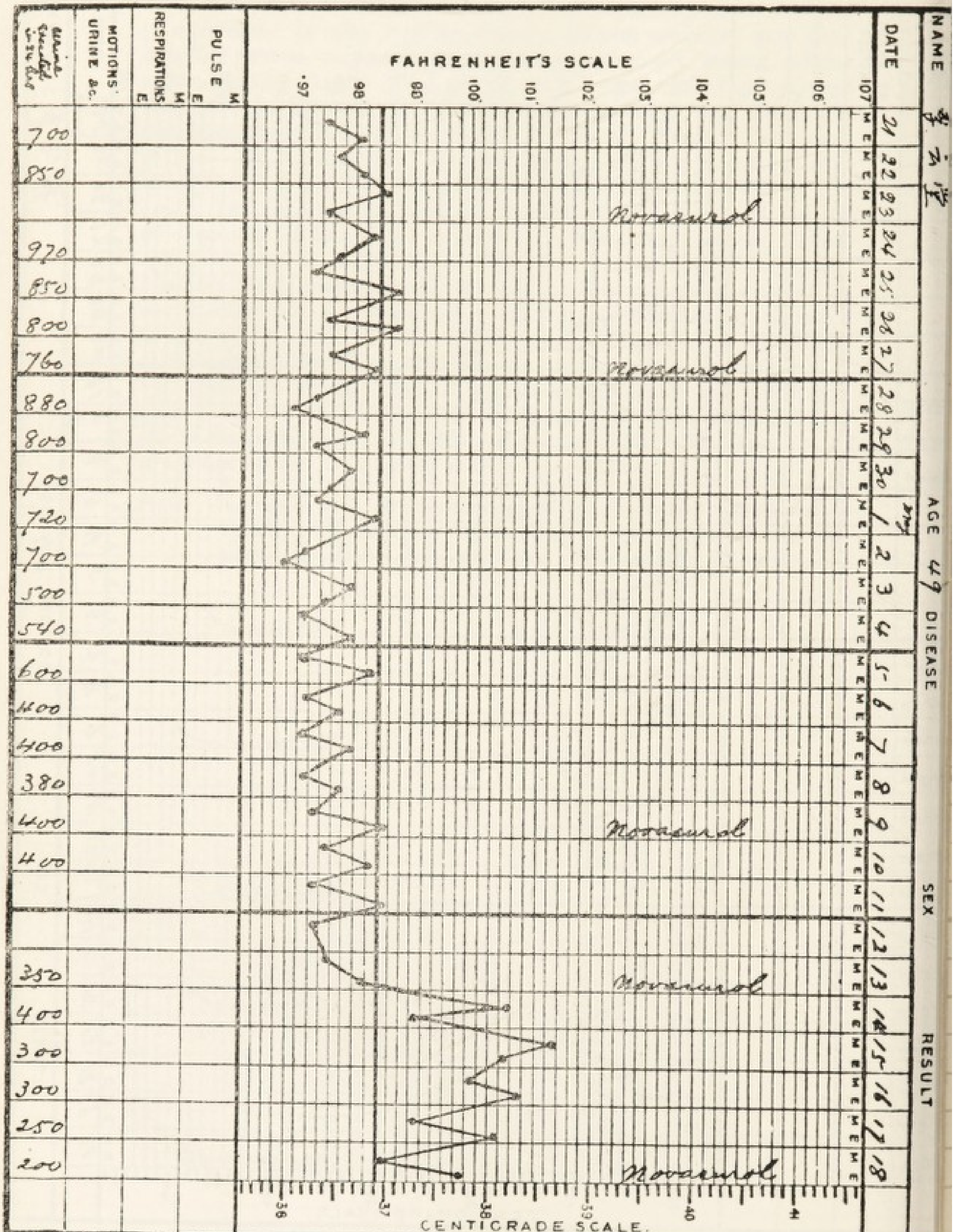
Blood test :— by Sachs-Georgi method, positive.

The condition to be dealt with was chronic parenchymatous nephritis (? syphilitic). In order to induce diuresis, different medicines were given including diuretin, mag. sulph. digitalis, strophanthus, pot. acetate, caffeine citrate, theocin, thyroid gland tablets, etc., but no marked improvement was noticed. Because of the positive Sachs-Georgi test, Bismuthine was given three times and neosalvarsan 0,3 twice. Novasurol was given in 1 c.c. doses, altogether six times intravenously and intramuscularly. It may be seen from the charts that the urine which was as low as 153 c.c. in 24 hours increased after the third injection of Novasurol and the third injection of Bismuthine to 2600 c.c. From thence onward the excretion of urine was never below 1000 c.c. The patient became better. His face which was tremendously swollen before was now reduced by one third in size. The amount of albumen as estimated by Esbach's method was not reduced, varying between 4 to 9%, though the volume of the urine had much increased and the patient was greatly improved.

After two months' stay, he left the hospital, quite slim and happy again. At that time he was still somewhat pale. His urine excretion was in the neighbourhood of 1500 c.c., albumen 9%. No casts.
Blood test by the Wassermann method was negative.

Note. Novasurol is a (combination of sedium-mercuri-o-chlorphenoxyl-acetate with diethylmalonylurea. It is put up in 1,2 c.c. ampoules by Bayer for intravenous and intramuscular injections.


NAME
AGE 49 DISEASE
SEX
RESULT
DATE
FAHRENHEIT'S SCALE.
97 98 99 100 101 102 103 104 105 106 107
CENTIGRADE SCALE
36 37 38 39 40 41
PULSE
RESPIRATIONS
MOTIONS
URINE &c.
Normandi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SEX
RESULT
FAHRENHEIT'S SCALE
PULSE
RESPIRATIONS
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Novasurol
Novasurol
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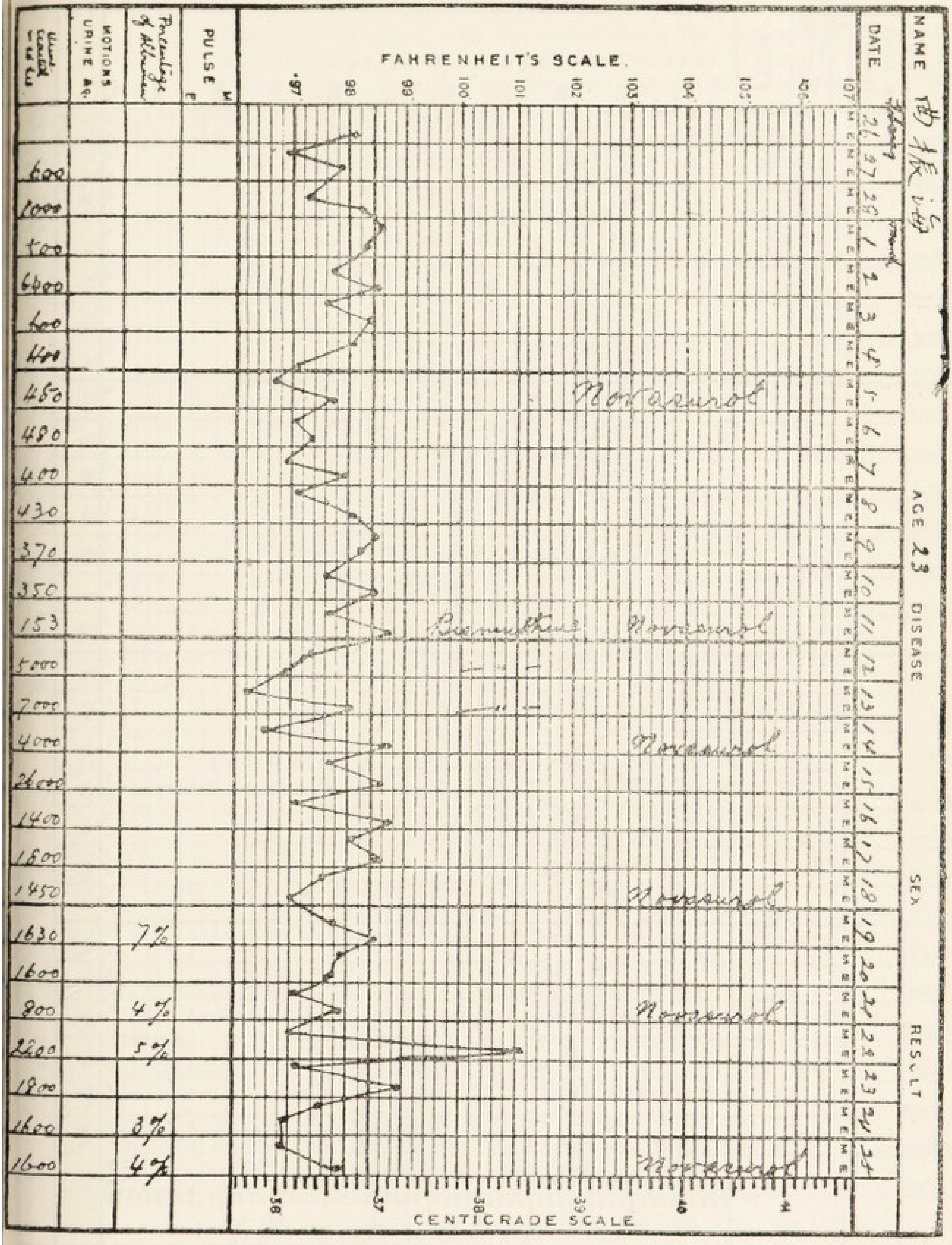
Novasurol
Novasurol
CENTIGRADE SCALE.


NAME
DATE
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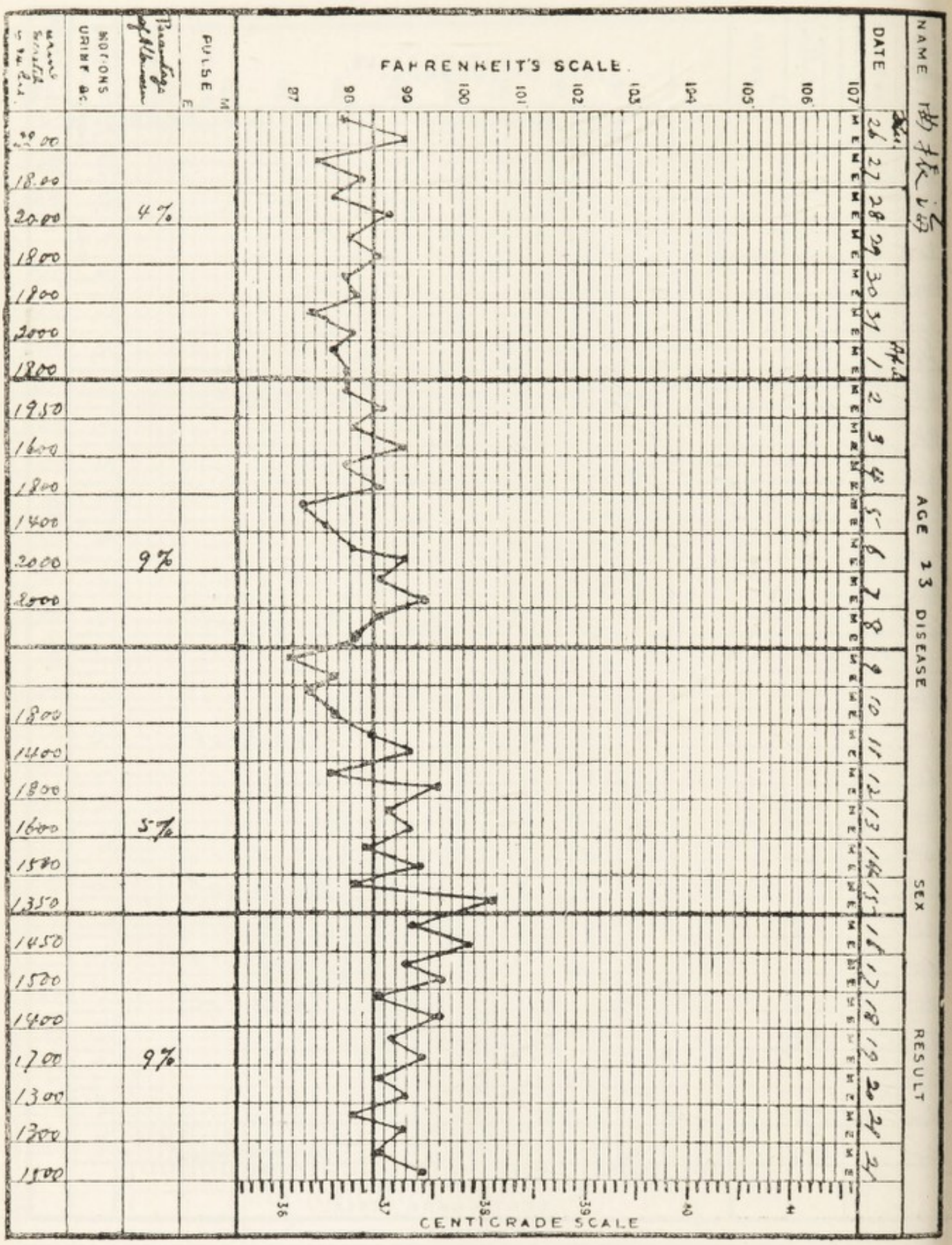
DISEASE
SEX
RESULT
FAHRENHEIT'S SCALE
CENTIGRADE SCALE
PULSE
MOTIONS URINE &c.
Novasurol


NAME
AGE 23 DISEASE
SEX
RESULT
DATE
FAHRENKEIT'S SCALE.
CENTIGRADE SCALE
PULSE
MOTIONS URINE &c.
Breakfast
4%
9%
5·7%
9%

The mercurial element therein is utilized to treat syphilis. The drug may be mixed with the arsenobenzols for simultaneous administration. It has also a hydragogue effect in cases of cardiac dropsy. Its action may be said to be due to the irritant effect on the renal epithelium. In our case, there was a definite element of risk in its exhibition. But it was employed because of the desperate condition of the patient and as a last resource, I am inclined to believe the improvement was due to novasurol, because three injections of Bismuthine could not have such a good effect. Also improvement had already set in before the two neosalvarsan injections were given. I must admit however the benefit to the patient was perhaps as much due to its anti-syphilic action as its diuretic action.

A CASE OF MENINGITIS CEREBRO-SPINALIS.

Lu, age 16, male, apprentice to a hat factory, was admitted on the 25th March 1927 with a history of 3 days' sudden illness with complaint of severe headache, high fever and delirium. Past and family histories were unimportant. No other companions were ill simultaneously or just previously. There was no diarrhœa.

On admission, a young, fairly well nourished boy, with a flush face, delirious, temperature 39.5°C. Pulse 98. No rash was seen. Tongue not coated. Abdomen not distended or tender. Slight rigidity of the neck with Kernig's sign present. Tache cerebral marked. No other abnormal physical signs. 20 c.c. of 1% mercurochrome was injected intravenously at once. His condition was improved and when seen on 27th he was conscious and took some nourishment, temperature being 37.3°C.

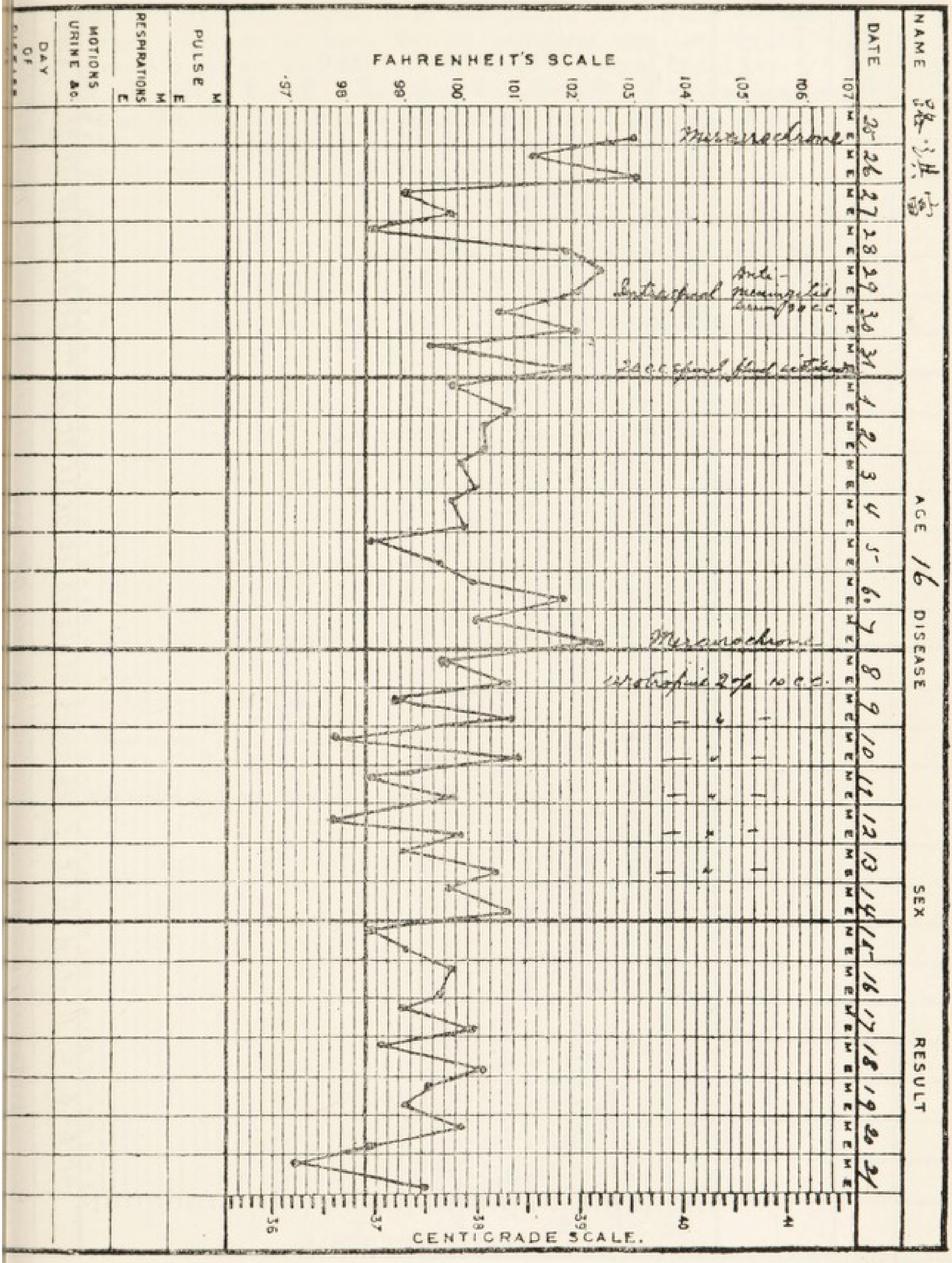
On the 28th his temperature went up again to 38.9°C. but he did not become delirious.

Spinal fluid was drawn under pressure, turbid; leucocytes and meningococcus were seen by Dr. Chow. On the 29th, Dr. Chow administered 30 c.c. of Anti-meningococci serum (Mulford) intraspinally, after withdrawing an equal amount of fluid. The patient continued to improve. Headache was less, though the neck and knees were still stiff. On the 31st Dr. Chow again tapped and withdraw 20 c.c. spinal fluid. The patient is now making good progress. Temperature falling by lysis, until it came to 36.9°C. up to 5th. April. On the 6th, however, his temperature went up again to 39.3°C. There was no delirium. Mercurochrome was again given intravenously. During the next six days, 10 c.c. of 20% urotropine was given intravenously daily and the following week showed much improvement. He was able to sleep and eat and feeling pretty well. There was still some stiffness of the neck and pain in the muscles of the thighs. After the 21st his temperature was normal and he stayed with us five weeks more in convalescence. When he left the hospital, he was quite well in every way. He was thinner than before, but his mental faculties were in no way impaired.

Notes : The effect of the first injection of mercurochrome seemed striking, for the temperature was brought down the next day, and the delirium disappeared.

A CASE OF TETANUS TREATED SUCCESSFULLY WITH SERUM.

Miss Chang, 20, was admitted for tuberculous knee of the left side. The history was that four years ago, the left knee became swollen and painful. She was unable to walk and had to keep the knee flexed. Soon afterwards three sinuses were formed, two on the inner side and one on the outer side. The sinuses have been discharging since. On examination, she was a pale woman, of frail physique. The left knee was undoubtedly tuberculous. The disease was chronic and extensive, pus tracking upwards between the muscle planes to the middle of the thigh. Amputation was advised and performed on the 17th May, the limb being removed at the junction of upper and middle thirds. Chloroform was the anæsthetic used. During operation, there was considerable bleeding and oozing on the dressings afterwards. Drainage tube was inserted for one day. The next day, the patient removed all the dressings herself and we had to dress the wound again. After five days, the wound did not heal well and on the seventh day the wound gaped open on the removal of the stitches. There were pale granulations, but not much pus. On the 10th day after operation she complained of stiffness of the jaw and inability to open her mouth. There was also some fever. Tetanus was diagnosed and 25 c.c. anti-tetanus (4000 units) were injected subcutaneously. On the next day convulsions commenced, 3-4 times per hour. 20 c.c. serum was again injected. On the third day convulsions occurred every five or ten minutes. 10 c.c. serum were injected subcutaneously and 10 c.c. intra-spinally. On the fourth day, 10 c.c. (5000 units) were injected subcutaneously and 10 c.c. intra-spinally. On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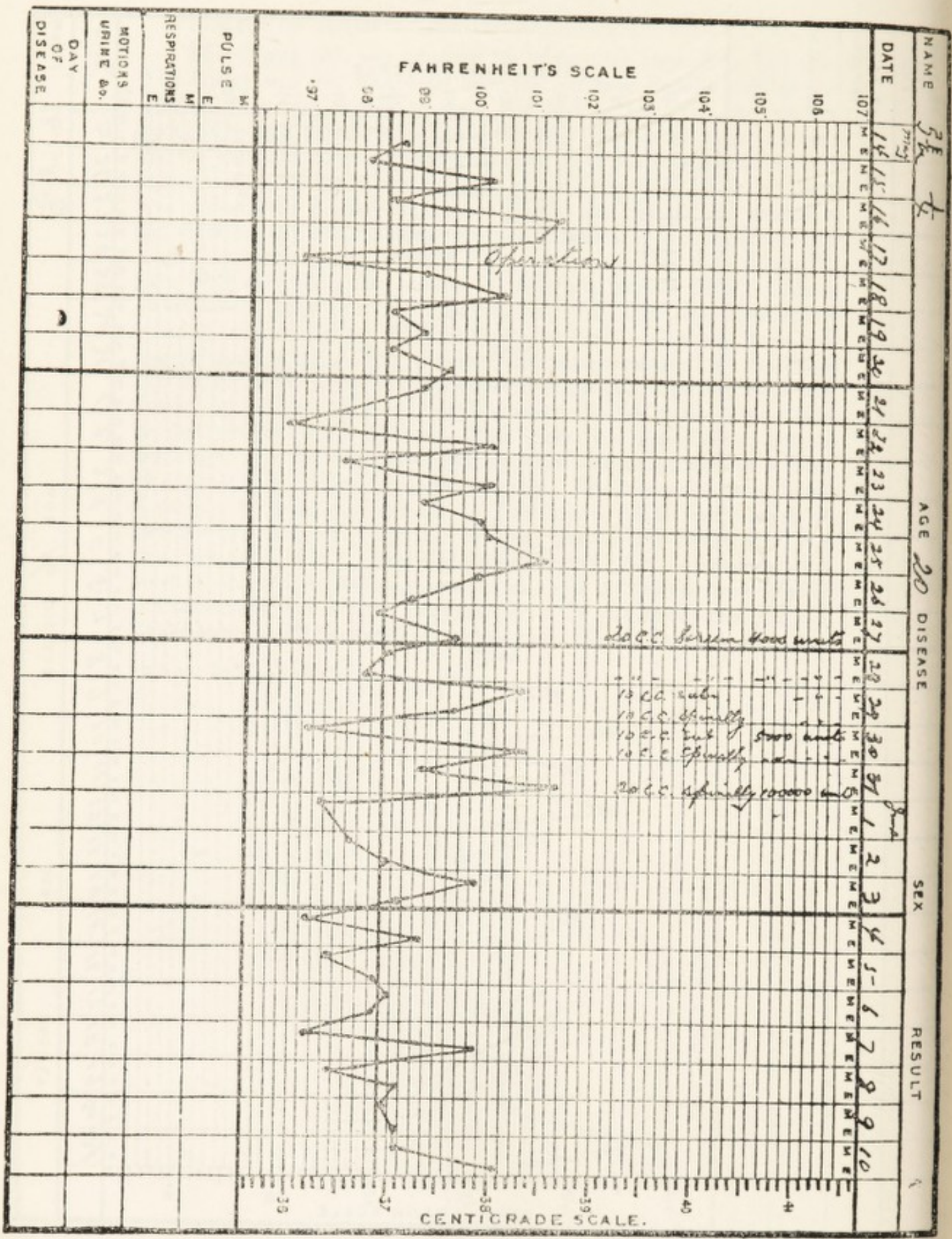
CENTIGRADE SCALE
PULSE
RESPIRATIONS
MOTIONS
URINE &c.
DAY OF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SEX
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PULSE
RESPIRATIONS
MOTIONS URINE &c.
DAY OF DISEASE
Operation
20 C.C. Serum 4000 units
20 C.C. sub
20 C.C. Specially
20 C.C. sub
20 C.C. Specially
20 C.C. Specially 100000

the fifth day 20 c.c. (10000 units) intra-spinally. On the sixth day the patient began to feel better and the convulsions were less frequent. Gradually she got well and now the wound has almost healed after a month's stay in hospital. Convalescence was marked only by a moderate serum rash two weeks after the first injection. As to medicines, we gave Pot. Brom. and Chloretone in an attempt to relieve the convulsions. No special treatment to the wound was undertaken.

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URINE ANALYSIS IN PRIVATE PRACTICE.

(Reprinted from the National Medical Journal 1927).

Dr. Willheim, Assistant of the University Laboratory for Medical Chemistry in Vienna, gives an excellent survey of this problem[1] which may prove valuable not only to practitioners but also small hospitals in China.

In the introduction to his article the author reminds us that the most important urine tests are at the same time the most simple ones, easily to be carried out by the practitioner. It is necessary, however, that the latter be informed not only in regard to their technique but also to their diagnostic importance. It is always necessary to weigh the evidence obtained by them together with the other clinical findings.

Reaction of urine. It is erroneous to conclude at once from an alkaline reaction upon decomposition of the urine. Apart from this, such a reaction may be present after alkali has been ingested, when the diet consists mainly of vegetables or in patients suffering from hyperacidosis immediately after ingestion of food. The alkaline reaction in decomposed urine is caused by ammoniacal fermentation; this may be recognised by the fact that litmus paper moistened with such urine is apt to loose its bluish colour again when dried in the air. Moistened litmus paper becomes blue when simply held over heated urine of this kind.

Specific gravity: Far too little attention is paid to a low specific gravity which is of great importance for the diagnosis not only of contracted kidney but also of acute toxic nephritis and pyelitis.[2]

Tests for albumin. Willheim strongly recommends the use of sulphosalicylic acid.[3] When carrying out the heat test addition of too much acetic acid may lead to errors; in the acetic acid—potassium ferrocyanide test too little of the former reagent or a surplus of the latter may prove misleading. The nitric acid test has the drawback that a white ring may be obtained in spite of absence of albuminuria when balsamica have been taken by the patient; uric acid may also give such a ring which, however, is situated above and not at the line of contact. In the house of the patient often the heat test will have to be resorted to: a little urine may be placed on a spoon, some sodium chloride and vinegar are added and the whole is heated over a candle.[4]

The author justly emphasises the importance of testing for the presence of albuminous substances precipitable by acetic acid alone. These are often designated as Nucleo-albumin; undoubtedly, however, other substances give the reaction as well. The test for these bodies is very simple: a sample of urine is three times diluted with distilled water and distributed in two test tubes, to one of which 10-20 drops of acetic acid are added. When the reaction is positive, a distinct opalescence appears after acidification. This test is mainly of prognostic value: if in renal disease albuminuria is present but acetic-acid-precipitable bodies absent, there is little or no hope for recovery, whereas as long as the bodies are present, a *restitutio ad integrum* may be hoped for. Thus in

chronic nephritis "Nucleo-albumin" is absent, while it is plentiful in orthostatic albuminuria. It must be kept in mind that this "Nucleo-albumin" gives positive reactions also with reagents for albumin, e. g. with sulpho-salicylic acid. However, these bodies never appear in great quantities so that this possible source of error can be neglected for practical purposes.

For a quantitative determination of the albumin contents the old Esbach test,[5] though not quite correct, is the best for the practitioner. Perhaps, however, use may be made also of the method of Brandberg-Stolnikov basing upon the fact that, when concentrated nitric acid is stratified under an albumin solution of 0.0033% content a just distinguishable ring is formed at the line of contact; the urine to be tested has to be diluted until this result is obtained. An orientation can be obtained by the heat-test insofar as an albumin solution of 0.1% or more yields diffuse, non-transparent opaqueness.

In certain cases Freund's alizarine reaction may be useful: a drop of urine is placed on a slide and mixed with the aid of a cover-glass with one drop of aqueous 1% alizarine solution, then inspected with naked eye or under low power. Normal urine shows a bright red, plate-shaped precipitate: when a diffuse process is present in the upper urinary tract (renal parenchyma, pelvis) this is yellowish-red, consisting of fine floccules or may even be altogether absent.

Tests for blood: A little guiac resin is taken with the tip of a knife and dissolved in a few c. c. of alcohol, the same quantity of urine and a drop of perhydrol[6] added. If blood (or pus) is present, a blue colour appears; if the urine is boiled before the test is made, the colour reaction shows the presence of blood only.

Sugar tests. Wilhelm recommends Fehling's test. That of Trommer requires far more experience, whereas Nylander's test is unpleasant to carry out as it presupposes longer boiling of a strongly alkaline liquid; moreover it is essential with this to free the urine from any albumin present, whereas with Fehling's test only larger amounts of albumin have to be previously removed by boiling and filtering. In concentrated urines a certain degree of reduction may be obtained with the Fehling test in spite of the absence of sugar; though no typical precipitate is formed. In such doubtful cases Nylander's reaction has to be resorted too.

For a quantitative determination of the sugar contents mention may be made of the fermentation-test, using a tube like Einhorn's or Lohnstein's at room temperature; then readings must be made after 12 hours as against 3 hours when an incubator is available. One source of error is the development of carbon dioxide on account of decomposition of the urine. It is well therefore to boil the urine for a short time before carrying out the test and to acidify it with tartaric acid if necessary. After the fermentation the reaction has again to be tested; if this is not acid, the test must be repeated.[7]

More exact are tests based upon reduction of the sugar. The method of Benedict is recommendable as it calls for one reagent only which keeps well: 200 grams of Sodium citrate, the same amount of crystallized

Sodium carbonate and 125 grams of Potassium sulphocyanide are dissolved in 800 c. c. of distilled water under gentle heating; the solution is filtered. 18 grams of Copper sulphate are solved in 100 c.c. of water and added to the first solution under constant stirring; some more water is poured into the vessel where the copper sulphate was dissolved in order to take up the remants of this and added to the mixture. Finally 5 c.c. of a 5% solution of Potassium ferrocyanide are added and the total brought up to a volume of exactly 1000 c.c. (All contents except the Copper sulphate can be weighed in the ordinary way, but every precaution must be taken to add the exact amount of the latter). In carrying out the test 25 c.c. of the solution are given in a porcelain dish, 20 grams crystallized Sodium carbonate (or 10 grams anhydride) and a little pumice are added and the whole is heated over a flame to boiling. Now add the urine from a burette or simply from a graduated 10 c.c. pipette, at first quickly, then, when the blue colour begins to disappear, more slowly, finally drop by drop. The final disappearance of the blue colour is easily noticed as it is replaced by a white precipitate. The amount of urine used corresponds, when 25 c.c. of the reagent were used, to 0.05 grams of dextrose.[8]

Tests for acetone. Legal's test with Sodium nitroprusside is preferable to the iodoform test, especially in the modification of Rothera: To a few c.c. of urine a knife-tip-full of Ammonium sulphate and a crystal of Sodium nitroprusside are added; shake, alkalinise with ammonia. When acetone is present, a red-violet colour develops slowly.

Tests for Aceto-acetic acid. Willheim thinks that Gerhardt's test (adding of 1-2 drops of a ferric chlorid solution to the urine resulting in positive cases in a Bordeaux-red colour) is out of place in the hand of the practitioner as many other substances give a similar reaction.[9]

Test for Urobilinogen. This is important for the diagnosis of liver diseases, internal haemorrhages, etc. and is performed with Ehrlich's Aldehyde reagent.[10] Heating is not to be resorted too, as then a red colour is obtained with normal urine as well.

Schlesinger's test for Urobilin gives excellent results, but this is too tedious for the practitioner.[11]

Tests for biliary pigments. Willheim justly points out that the practitioner can have but little hope from these tests, as some, like Gmelin's with nitric acid are clearly positive only when icterus can be diagnosed clinically, whereas others are for various reasons too difficult for the practitioner. Best is to stratify Tct. of Iodine, 10 times diluted with alcohol, upon the urine, a green ring appearing on the line of contact in positive cases. The fact that icteric urine forms a yellowish foam when shaken, is also helpful.

Test for indican. Indican is one of the substances neglected in routine urine analysis though its presence often forms a valuable hint for the presence of pathological conditions in the intestine and the test for it is simple: A test-tube is almost half filled with urine, the same amount of Obermayer's reagent[12] and a few c.c. of chloroform are added. Avoid shaking but extract the indican by pouring the whole in a second

test-tube, repeating this pouring to and fro until the chloroform is stratified. The presence of indican in manifested by dark colouration of the urine and blue colouration of the chloroform when this has absorbed the colour.

Test for urochromogen. This is easily carried out by three times diluting the urine and adding a few drops of a one per mille aqueous Potassium permanganate solution. Positive result is marked by the appearance of a yellow colour after thorough shaking. The test is positive in comsumptive disease, especially in lung tuberculosis (and also in typhoid fever), while but rarely in carcinoma cases. Weisz, who described this test, recommends it specially as an aid for making a prognosis in cases of tuberculosis. A positive test would make the prognosis more serious and contra-indicate the use of tuberculin.

REFERENCES.

1. Mitteilungen des Volksgesundheitsamtes, Wien, 1926, No. 10, pp. 262-264.
2. In the reviewers' experience especially pyelitis remains often undiagnosed because no attention is paid to the presence of low specific gravity together with traces of albumin and pus cells in the urine. Thus a wrong diagnosis of appendicitis, gastritis, etc. may be arrived at.
3. "Sulpho-salicylic acid may be used either in the form of a 20 per cent solution or in the solid state. If the solution be used it is added to the acidified urine in such a way that a distinct line of contact is formed. Albumin will be shown by a distinct white ring at the point of contact. It is preferable, however, to add to the urine a small fragment of this substance, when in the presence of albumin a turbidity or a white flocculent precipitate will be observed, depending upon the amount of albumin. The albumoses are also precipitated by this reagent, but dissolve on heating." (Webster, Diagnostic Methods, 1920, p. 285).
4. However, as Webster points out, sulpho-salicylic acid in substance may be readily carried in the medicine bag for an emergency.
5. Esbach's reagent is prepared by adding 10 grams of picric acid and 20 grams of citric acid to 1000 c.c. of distilled water.
6. Instead of perhydrol ozonized oil of turpentine (i.e. such which has been kept for long time in an unstoppered bottle) may be used. The test is best made by stratifying a mixture of the freshly prepared guiac tincture (see text) and an equal amount of the turpentine oil upon the urine, a ring forming at the line contact and gradually becoming blue in positive cases. Acid reaction of the urine is essential. When an ether extract of the urine and not the urine itself is used the test is positive with blood alone, not with pus. Especially when the reagents have not been used for a long time, it is well to make a control with a diluted blood solution.
7. Other possibilities of error are present as well when using simple tubes. An authority like Sahli considers "Einhorn's attempt to simplify the method for the practising physician.....as a failure" (Diagnostic Methods, 2nd English Edition, p. 624).

8. Benedict's test may be used with great advantage also for qualitative work. A good and fairly stable solution for this is prepared as follows:

"With the aid of heat dissolve 173 grams of Sodium (or Potassium) citrate and 100 grams of anhydrous (or 200 grams of crystallised) sodium carbonate in about 700 c.c. of distilled water. Dissolve 17.3 grammes of pure crystallised copper sulphate in about 100 c.c. of water. Cool the solutions to the room temperature, pour the second into the first, slowly and with constant stirring; make up to 1000 c.c. with distilled water." (Cammidge, Lancet, 1917, ii, p. 24): 5 c.c. of the reagent are mixed with 8-10 drops of urine in a test-tube; the mixture is vigorously boiled for 1-2 minutes. When sugar is present a red, yellow or more greenish precipitate forms while boiling or (if little sugar is present) on cooling. Sugar-free urines yield a clear solution or only faint turbidity, blue in colour.

This test is not only very convenient but free of sources of error peculiar to the other reduction tests. It must be kept in mind, however, that some substances, notably preservatives like chloroform, formaldehyde, etc., cause reduction (Webster, l. c., p. 305-06).

9. Good advantage can be taken, however, of two controls recommended by Sahli (l.c., p. 602):
   1. When boiled urine is used, the red colour is much fainter.
   2. The urine is acidified with sulphuric acid and shaken with ether. Aceto-acetic acid goes over in this and if the ether extract is shaken with a diluted solution of ferric chloride the aqueous layer turns red, *this colouring disappearing spontaneously in 24-48 hours.*
10. According to Hildebrandt (Z. f. Klin. Med., 1906, quot. by Sahli, l. c., p. 585) the reagent is prepared as follows: 20 grams of paradimethyl-amidobenzaldehyde are rubbed up in a mortar with 100 c.c. of concentrated hydrochloric acid, more acid added, until the volume is 500 c.c., and then the whole made up with water to 1000 c.c. Two drops of the reagent are added to the urine, a dark-red colour appears and with the spectroscope a band is seen in the orange yellow between D and E.
11. A simple test for urobilin is to alkalinise the urine strongly with ammonia, filter and add some drops of a 10% solution (aqueous or alcoholic) of zinc chloride. Green fluorescence shows the presence of urobilin.
12. This reagent is prepared by adding 4 grams of ferric chloride to 1000 c.c. of fuming concentrated hydrochloric acid.

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REPORT ON A VISIT TO HEALTH CENTERS IN EUROPE, 1927.

(Submitted to the Director, Health Section, League of Nations, Geneva).

I beg to submit a Report on my trip to Europe as a Travelling Fellow of the Health Section of the League of Nations. Owing to preparations connected with an outbreak of Cholera in China soon after my return to Harbin, this Report has been somewhat delayed.

2. I left Harbin on March 1st and returned on June 27th, the intervening days being spent as follows:

March 1-10	Travelling, Manchuria and U.S.S.R.
March 10-15	Warsaw.
March 16-20	Vienna.
March 22-23	Berne.
March 24-Ap. 2	Geneva.
April 4	Lubliana.
April 5-7	Zagreb and Mraclin.
April 8-11	Belgrad.
April 14-May 2	Paris, Havre, Tergnier.
May 2-31	London, Cambridge, etc.
June 1-3	Hamburg.
June 3-12	Berlin.
June 12-27	Travelling via Siberian Railway.

3. Since my last visit to Europe was in 1913 (year before the World War), I naturally observed immense changes in practically every aspect of human activity. The cost of living, of building and scientific equipment has increased at least twice, in some cases more. Nevertheless, the restlessness of the several nations and of individuals to 'get on' is seen everywhere, and this is reflected in the wonderful progress made in public health. Every government seems to appreciate the invaluable aid which medicine from a new angle could contribute to the economical as well as physical rehabilitation of the nation after years of strife. In this great work of construction, and amelioration, the Health Section of the League of Nations has played and is playing a leading part.

4. I will now proceed to a short description of my experiences at different centers visited by me.

At Warsaw, I was treated as a guest of Dr. Wroczynski (Director-General of the Health Service) at the new School of Hygiene, adjoining which is the well-equipped State Health Institute. I was fortunate in meeting also Dr. Chodzko (former Minister of Health and now Director of the School of Hygiene), Dr. Hirszfeld (Acting Director of the Institute), Dr. Kacprzak (Head of Dept. of Vital Statistics) and Dr. Anigstein (who has obtained noteworthy results in the difficult work on Typhus). I was very favourably impressed by the keenness with which every member of the staff showed in establishing an independent reputation for Polish science.

Thanks to the courtesy of Dr. Chodzko, I was invited to deliver an address on Pneumonic Plague to the staff.

5. From Warsaw I proceeded to Vienna, where I called upon Dr. P. Scherrer, Head of the Hygienic Department of the Ministry of Social Welfare, who explained to me the new health activities of the Republic though his work was much handicapped by insufficient funds. I was fortunate in seeing Prof. von Pirquet, whose Children's Hospital is still a model for those seeking knowledge and inspiration in Pedriatics. I also visited the famous Institute of Pathology (now presided by Prof. R. Maresch), where cadavers are as numerous and foreign students as enthusiastic as ever in acquiring the latest technic. A new field of medicine has been created by the remarkable investigations of Prof. S. Jellinek on Electro-Pathology, which in view of the widespread use of electric power is sure to play an important part in the domain of modern medicine. The Museum fitted up by Dr. Jellinek in a disused church is well worth a visit.

6. I stayed in Geneva from April 24 to May 2. Though the weather was bad most of the time, the working atmosphere was all that could be desired. Here one met persons of both sexes, perhaps with the most diverse views, who were willing to listen to each other and to seek compromise and co-operation for the most difficult problems affecting mankind. The friendly spirit that pervaded the various departments of the Health Section of the League of Nations was a wonderful stimulus to me, and I felt that China should at the first opportunity send one of her promising young doctors there to learn the technic of international health work, as well as supply all available knowledge regarding medical conditions in this country.

Having for nearly twenty years been interested in anti-opium work I spent some time collecting information at the Secretariat regarding this subject.

While at Geneva I received the utmost courtesy from every member of the Section from Dr. Rajchman downwards.

7. From Geneva I travelled through Trieste and Venice to Jugo-Slavia and stayed for a day at Lubliana, where Dr. Pirc, the Health Officer, showed me the first evidence of that most remarkable of modern health work undertaken by the Government under Dr. Ignatius Stampar. It would take too long to describe my impressions of this unique medical achievement in a corner of the Balkan States, where formerly sanitary conditions were not further advanced than what we witness in most parts of China to-day. In many respects, especially in the eastern regions, the habits of the people may be compared to our own, but each day since Dr. Stampar started his work in1920 there has been considerable improvement, so that if one were asked to-day which country in the world has the best record for health progress, one would certainly say Jugo-Slavia.

For in a population of 12,000,000 and a total budget of 12,000,-000,000 dinar (or G.$60,000,000), G.$6,000,000 is earmarked for Social Service. Of this sum G.$2,200,000 is for actual health work, that is, nearly 0.20 gold cents per inhabitant are spent upon health.

Both government (of whatever party) and peasants have the utmost confidence in Dr. Stampar, who has since the starting of the work in 1920 built, equipped and staffed 212 up-to-date medical and health institutions. In 1926 alone 126 buildings were constructed at a cost of 17,000,000 dinar. There are at present employed 230 full-time doctors and specialists as well as 1500 half-time men and women. The salaries of the medical staff are not high, the chiefs of institutions receiving 5,500 dinar (G.$100.) per mensem. But under the forceful direction of the Chief, the most diversified health work is undertaken in urban and rural areas, from immunisation of chicken, pigs, cattle, infants, etc. against their several infections to the most delicate problem in biochemistry.

I also visited Zagreb, Mraclin, Novisad and Belgrad, reluctantly leaving the country after a profitable stay of eight days.

8. A journey of two days took me to Paris, where I saw the same old institutions that I used to know in 1902-3. I called at the Office International d'Hygiène Publique, the Institut Pasteur and some hospitals where I once worked. Dr. Rajchman kindly lent me the services of Dr. La Touche of the Paris Office of the Health Section. I visited on April 21 the Tuberculosis Clinic at 17 Rue des Serpentes Pontaine (outside Paris), where two nurses and five part-time physicians cared for 300,000 inhabitants, on the 22nd. I called at the headquarters of the Service Nationale contre Tuberculose and also at the Office Publique d'Hygiene Sociale (61 Rue de Vasco di Gama XV). The work at both places is much influenced by American methods.

I also visited Havre to see the Quarantine institutions and happily met Dr. Loir, the widely-read chief of the local Office Hygiene, who took me the Musee Leseuer founded by a contemporary of Cuvier. Dr. La Touche accompanied me to the small town of Tergnier, where a garden city has been constructed side by side with the railway works since the Great War. Here are model dispensaries, schools and infant homes to supply the needs of 1,100 families. The cost of building this city is 66,000,000 francs. The International Conference on Rabies was held in Paris from April 25 to 30, at which I attended as Official delegate from China. A resume of the proceedings has already been published. Under the presidency of Dr. Hahn (Germany) an informal meeting of plague experts was held on April 26 at the Institut Pasteur. It was decided to circulate to various institutes a memorandum on Pneumonic Plague prepared by Dr. Wu Lien Teh.

9. From France I crossed to London on May 2 and stayed in England until May 31. Among institutions that I visited are the laboratory of the Medical Research Council, University College, St. Mary's Hospital, the new Laboratories at Cambridge as well as various centers where school hygiene has been so well developed. When one studies the steady and elaborate way in which practical health problems are tackled in England, one is not surprised at the splendid decrease in the mortality statistics both in cities and country. The establishment of the Ministry of Health since the War, thus co-ordinating all government health work, has contributed much to the efficiency and increased knowledge of the people.

10. I stayed at Hamburg for three days and spent most of the time at the famous Tropen Institut founded by Dr. Nocht and completed just before the Great War broke out in 1914. Here I was kindly shown round by Profs. Giemsa and Meyer. Classes on Malaria under the auspices of the Health Section, League of Nations, were being held at the time. In spite of the loss of colonies by Germany, there were as many workers as ever at the Institute and as many patients in the Tropical Wards as before. I was told that rare specimens continue to arrive from the South American Republics to maintain the efficiency of the School.

While in Berlin, I visited the Koch Institute, the Charité, and other hospitals, where I used to spend fruitful days. I found the staff and workers as keenly bent as ever upon new problems of research, and left Germany fully convinced that in spite of financial losses the high reputation of German medicine will continue and thus attract numberless students to her universities and laboratories.

11. I cannot conclude this Report without emphasing the significant part which China may play toward health betterment in the world. Until now, the ancient idea of treatment of diseases still grips her masses, and even most of her modern practitioners. Health experts are still few and far between, but these can be increased and their words more appreciated if the League of Nations will use its diplomatic as well as technical machinery in furthering this work. A fine start has been made by Dr. Norman White in his Quarantine Survey, but this needs to be completed, so that the annual outbreaks of Cholera largely spread by foreign steamers from the International Settlement of Shanghai may be more easily controlled.

Owing to military strife in the country, it has been most difficult for responsible health officers to ascertain where their duties begin and end, but this difficulty can be minimised by more attention being given to the actual workers in various localities of the vast country. During these exceptional times there should not be too rigid an adherence to surface routine if the health of the world could be protected by appropriate means. In this respect, the example of Great Britain in dealing with authorities on the spot during 1926-7 may be followed.

12. Finally, I wish to express my deep obligation to the Director for his kindness in enabling me to visit Europe and personally witnessing the striking advances made in international hygiene since the Great War.

WU LIEN TEH.

Harbin, September 20th, 1927.

WU LIEN-TEH—A SHORT AUTOBIOGRAPHY.

At forty-seven years of age one looks back upon the past with a rather detached mind. One's early ambitions are somewhat toned down, criticisms of one's fellow creatures become fewer and less severe, and while surveying present-day conditions one tends to view things in a more optimistic spirit than in the earlier years.

Hence, in writing this short autobiography I would ask my readers to excuse me if I miss out the unessential parts and devote the following pages to persons, institutions and surroundings I have been associated with in the manner of a moving picture screen, leaving to my readers any deductions to be gathered in whatever way they consider fit.

I was born in 1879 in the Island of Penang, Straits Settlements, that stretch of British territory formerly belonging to the Malays and now intensely prosperous as a result of the tin and rubber industries. My father was of the usual Cantonese emigrant type who left his village home in Sinning (now Taishan) to make his fortune across the "Seven Seas." My mother belonged to the second generation of Chinese in Malaya, her father being an emigrant but her mother was descended from resident Hakka parents, those sturdy fearless peasants of China who though possessing no province of their own are nevertheless able to succeed in business where others fail. Until within recent years the education given by the local government was purely in the English language. The fees were low, varying from six to twelve dollars a year, so that even the poorest children could take advantage of it. I stayed in the Penang Free School for nearly ten years (1886-1896) and learnt not only how to write and read English but also a lot about English History and Literature, English Geography (including details of the smallest hamlet), Latin, Chemistry and Geometry. I won many prizes at school, but the most difficult to obtain, namely, the Queen's Scholarship, for which candidates had to proceed to Singapore to be examined, required three atttempts before I was successful. On the first two occasions I was under-age, but in 1896 I reached the goal of my boy's ambition and was awarded the much coveted prize of that year, thus enabling me to proceed to England and enter my name as an undergraduate of science and medicine at Emmanuel College, Cambridge. The value of this scholarship was £200 per annum, out of which income tax had to be deducted by the London Government. Those who are conversant with university life in England will understand that this sum, even in those days, was barely sufficient to carry a student through the year, especially as travelling expenses, clothing, college fees, and holidays were included therein. However, by exercising the strictest economy and avoiding luxuries of every sort I managed to keep my head above water. The first year was the hardest, for the fees were then the highest and mistakes were frequent. Fortunately I had a sympathetic tutor at College in the person of Dr. William Napier Shaw (now Sir W. N. Shaw, Head of the Meteorological Office, London) who did all he could to encourage me in my studies and aspirations. At

the end of my second year (June 1898) I won a College Exhibition of £40, which enhanced my slender income and enabled me to partially return the hospitality which I had earlier received from my friends. The next year (1899) brought still better luck, for I obtained First Class Honours in the Natural Sciences Tripos for the B.A. and was rewarded with a Foundation Scholarship of £50. In addition I passed all the necessary examinations for the second M.B. Examination in the shortest possible time.

In September, 1899, I removed to London, competed for and won the University Scholarship at St. Mary's Hospital open to graduates of British Universities. The money from this new prize just covered all tuition fees required during the next three years at the Hospital. I found student life in London quite tame and unexciting after the "jolly time" at the 'Varsity. Gone were the four o'clock teas, evening squashes, May Weeks, Bumping Boat races, noisy rags and wooden spoons. In their place I had to be satisfied with attendance at Casualty, Clinical clerkships, surgical dressing, midwifery cases among the London poor, witnessing post mortems and all sorts of surgical operations. But the change was good for it sobered one to the serious problems of life and opened one's eyes to the vastness and complexities of human toil. From that time medicine appealed to me more than ever, and its numerous possibilities in China were rendered more and more evident.

After two and a half years in the British metropolis, I qualified as a Bachelor of Medicine and Bachelor of Surgery (M.B., B.C.) of Cambridge in April, 1902, being the only medical student of that University out of a class of 135 who entered in 1896 to have accomplished that task in the short period of 5¼ years. I also won the Cheadle Gold Medal in Clinical Medicine and the Kerslake Scholarship in Pathology and Bacteriology, besides various prizes in surgery and medicine. The next problem was to increase one's experience, for it has been truly said that 'a man only begins to learn after obtaining his degree.' My old College ((Emmanuel) offered me a Research studentship of £150 a year for 1902-3 to carry on scientific investigations in various laboratories. This opportunity I readily seized and spent the ensuing year working first under Dr. Ronald Ross (now Sir Ronald Ross) at the School of Tropical Medicine, Liverpool, then under Professor Karl Fraenkel at Halle a/S in Germany, and lastly under Professor Elie Metchnikoff at the Pasteur Institute, Paris. I devoted my time principally to research upon malaria and tetanus, and thanks to association with prominent thinkers in the world of Science I managed to imbibe its true spirit and to hope for the time when I might pass on the same to others who came after me. Living among continental people and learning their languages enabled one to become simpler in habits, more tolerant and broadminded. I acquired a fair knowledge of German and French without a teacher by mixing freely among the people and sharing their tastes and enjoyments, however humble these might be.

On my return to England in the spring of 1903 I was advised by the then Regius Professor of Physic of Cambridge (Sir Thomas Clifford

Allbutt)—most kind-hearted and brilliant man—to utilize the results of my investigation on tetanus for my M. D. thesis. This examination I successfully passed that year, though I had to wait another two before the degree was actually conferred upon me. I also accepted the post of House Physician at Brompton Hospital for Consumption and Diseases of the Chest, London, for six months, where I gained an insight into the wide prevalence of Tuberculosis and its effective control.

I returned to the Straits at the end of 1903 with another year of the College Fellowship and joined the Institute for Medical Research, Kuala Lumpur, for the study of Beri-Beri, which disease killed thousands of Chinese and Indians annually in those regions. The next three years (1904-07) were spent upon private practice in Penang, but although my bank balances increased day by day, my heart always yearned for a chance to use my unusual experience for higher purposes than the mere treatment of common ailments so often associated with general practice. The declaration in the British Parliament of Mr. John Morley (then Secretary of State for India) in 1906 that the Indo-Chinese opium trade was an immoral and indefensible one had given new life to anti-opium workers, and I entered the fray with the greatest enthusiasm, helping my friends to subscribe tens of thousands of dollars for the cause, establishing homes for addicts and giving free medical services to them. Unfortunately this work conflicted with the aims of several rich families whose source of income was mainly derived from the opium monopoly and resulted in much opposition and abuse from both officials and would-be-patients. However, the campaign against the opium habit rapidly spread and encouraged fellow-workers in China and elsewhere to persist in their efforts. The year 1907-8 could certainly be designated a red letter day in anti-opium history. In August 1905 I married Miss Huang Shu Chiung, second daughter of Mr. Huang Nai Siong of Foochow. We first met one another at the home of Dr. Lin Wen Ching, a leading practitioner of Singapore, whose wife was Mrs. Wu's sister. Our married life has been an unusually happy one, and our only son Chang Keng is now studying medicine at John Hopkins University, U.S.A.

In July 1907 I paid my first visit to China and visited Tientsin, where I was urged by Viceroy Yuan-Shih-Kai (as he then was) to join the Chinese Army Medical Service. At the critical moment I was attacked by acute dysentery, which necessitated my leaving the north for a time, though it also made possible my visit to military institutions in London and Berlin for further experience. Next year 1908 when I arrived at Peking, both the Empress Dowager and Emperor Kwang-Hsu had died, Yuan-Shi-Kai had been banished to Honan, and his principal enemy, the Manchu Tieh-Liang, was in charge of the Board of War. However, through the influence of my old friend Admiral Tan Hsueh Heng, I was appointed to the post of Vice-Director of the Army Medical College, Tientsin, where young men were being prepared as army medical officers for various units of the modern army. The teaching was done in the Japanese language by Japanese and Japanese-trained Chinese, and insufficient emphasis was laid upon practical work and the prevention of diseases.

In December 1910 came news of the outbreak of pneumonic plague in Manchuria, where the local officials and old-style physicians were helpless in the presence of the rapidly growing number of victims. Political complications were also feared because Russia and Japan had both threatened to send their own medical staff and military into the infected areas unless more radical measures were adopted by the Chinese. Mr. Alfred Sze (then Councillor of the Foreign Office) sought me out and asked me to proceed immediately to Harbin with another Chinese doctor connected with the Board of Navy. I readily agreed and within two days after receiving instructions was off. My colleague did not come and so missed a fine opportunity.

The campaign against the Manuhurian Plague Epidemic of 1910-11 is pretty well known to most readers of modern history and need not be mentioned in detail here. From a scientific stand-point this dreadful disease (which killed almost 100% of those attacked) offered unrivalled opportunities for research, for up to then very little had been known of the infection and much initiative as well as courage was required for its prevention and treatment. I acted virtually as Commander-in-Chief of the huge organisation and gave orders to doctors, police, military and civil officials alike. The most dramatic phase of that historical fight was when I boldly asked for Imperial Sanction to cremate three thousand odd plague corpses which had for weeks been lying unburied on the ground because of the lack of coolies and frozen state of the ground. Nearly one-third of the whole population had either died or run away. I waited anxiously for 48 hours for news from Peking, and when permission was finally granted it took only two days to dispose of the dead. After that the medical staff and assistants breathed easier and worked with a more willing heart. The last case in Harbin was recorded on March 1st, that is four weeks after Imperial Sanction was received for cremation. The plague of 1910-11 spread to Chihli and Shantung (including Tientsin, Peking, Tsinan, Chefoo, etc.) and claimed altogether 60,000 deaths.

The successful control of this epidemic considerably affected my fortunes, for together with H.E. Alfred Sze I was commanded by the Imperial Government to organise the Mukden Plague Conference (April, 1911), at which experts from 11 leading nations participated. I sat throughout as Chairman of the Conference and with the other Delegates recommended the establishment of the present North Manchurian Plague Prevention Service to study the disease and cope with future outbreaks. The high rank of Major of the Imperial Army with Blue Button was conferred upon me overnight so as to enable me to receive Imperial audience without unnecessary formalities. It seemed as if modern medical science, which in China used to be compared to a barber's profession and had stood under a cloud for centuries, had at last come into its own. Its future prospects were indeed bright. I was also awarded the degree of Chin-Shih or Litt. D. of the Imperial University without the usual examination. The Czar of Russia conferred upon me the Order of Stanislaus Second Order, while the French Government gave me the coveted distinction of Legion d'honneur.

The Manchurian Plague Prevention Service was started in 1912 with initial funds totalling $180,000 for buildings and equipment provided by Viceroy Chao Erh Sun of Manchuria, while the annual appropriation of $120,000 for maintenance is defrayed by the Maritime Customs. Hospitals are established at Harbin (Headquarters with Central Library and Research Institute), Manchouli, Sansing, Lahasusu, Taheiho and Newchwang. There are affiliated hospitals at Antung, Hailar and Tsitsikar. Besides accommodation for plague patients the Manchurian Plague Service treats other communicable diseases like cholera, smallpox, scarlet fever, typhus, etc., carries on routine hospital work of the city by treating medical and surgical cases, manufactures sera and vaccines for plague, cholera, rabies, scarlet fever, etc., performs chemical and bacteriological analyses on food stuffs, water, patent medicines, etc., and advises the local municipal administrations on health matters.

The effects of this Service have been far reaching, for since 1912, post mortems were officially permitted in 1913, the National Medical Association of China with a membership of 500-600 was established in 1914, western medicine was officially recognised by the Central Government in 1915, and the Central Epidemic Bureau (Peking) was established in 1919.

Returning to the personal factor, I was appointed one of the three Imperial Delegates to attend the Hague Opium Conference in 1911 and signed with the representatives from other countries the historical document known as the Hague Opium Convention of 1912. Next year I visited Europe again and joined His Excellency W. W. Yen (later Prime Minister) as delegate of the second Hague Opium Conference. When the Revolution came in 1911 and China became a Republic I received the appointment of Physician Extraordinary to President Yuan Shih-Kai. This appointment has been continued by successive presidents. In 1915 I proceeded to Hongkong to accept the honorary degree of Doctor of Laws (LL.D.) of the University. On that occasion I took the opportunity of pleading for a more liberal attitude in the appointment of promising Chinese doctors to various teaching posts in the University, and emphasised that besides teaching and the granting of degrees, a University had other functions, such as, research and training of teachers for the hinterland, both of which could best be done by natives of the soil. I am glad to notice that the Professorship of Pathology has since that date been occupied by a Chinese, and that more Chinese have been appointed to responsible posts. Incidentally the second Conference of National Medical Association with me as President was held at Canton in 1916 side by side with the China Missionary Medical Association. At that meeting I urged my colleagues, especially those in charge of medical colleges, to emphasize the needs of public health in their curriculum, for the results so far as actual saving of lives was concerned would far exceed the benefits derived from mere treatment.

In 1916 Mr. Chou Hsueh Hsi (son of the famous Viceroy Chou Fu of Liangkwang) was Minister of Finance under President Yuan-Shih-Kai. He asked me to visit him at the Western Hills, Peking, and help him to build a Sanatorium for tuberculous patients at that lovely spot

surrounded by 200-400 year-old cypresses and silver-pines, many of them planted under the supervision of Emperor Chien-Lung who used the place as a hunting park. Having considered the matter I made bold to suggest the erection of a model general hospital in the heart of the capital rather than a restricted sanatorium in the country, however well equipped. He concurred with my views and called a meeting of the leading officials and philanthropists of Peking to discuss the matter, promising to place the $120.000 dollars which he had saved from the salt revenue at the disposal of the Committee. Thus was born the nucleus of the Peking Central Hospital, of which I was soon elected Organiser and Medical Director. The task imposed upon me was unusually difficult, for I had to interview influential persons for subscriptions as well as consult architects regarding plans, supervise the complicated ferro-concrete building as it grew up, order the thousand and one articles of equipment, and see to their transportation at the least possible expense, with duty free, half-rate on railways, special discount from firms, etc. In this work of preparation I had the close cooperation of Mr. Sze Sao-Tseng (brother of Minister Alfred Sze of Washington). The Central Hospital costing just over $300,000 was finally opened in January 1918 amid universal approval, for it was then the finest and most up-to-date institution of its kind in China. Even the Director of the Dairen Hospital (South Manchurian Railway) asked me for a copy of our plans in order that he might use them for modelling the new hospital at Dairen. This latter institution was completed in 1926 at a cost of over seven million yen.

Towards the end of 1917 Pneumonic Plague again invaded China, this time from Inner Mongolia by way of Patsebolong, Suiyuan and Tatung (Shansi). Along with two American volunteer missionary doctors we made our headquarters at Fengchen (Shansi) but the local officials and inhabitants were obstinate, and the special car in which we lived was one day surrounded by a mob and nearly burnt. Other doctors later arrived and the epidemic dragged on, though the region through which it traversed was a sparsely populated one, and some cases reached Peking and Nanking. The total death roll was 16,000, the last case being reported at the end of May (seven months after the commencement of the outbreak).

We went through a serious cholera epidemic in Manchuria in 1919, followed the next winter by the second Manchurian Plague outbreak. Fortunately, on this occasion we were well organised for fighting the pest, which claimed only 8,000 victims instead of 60,000 as in 1910-11, although the population of Manchuria had doubled during the intervening ten years. Our staff was also able to undertake valuable research work, and our reports contributed considerably to the advancement of knowledge on this important fatal disease. The world now accepts our contention that the Mongolian marmot (tarabagan), whose size is that of a cat and whose thick fur is much valued, is the main reservoir of the germ causing pneumonic plague and that simple handling of the sick animal or freshly dissected skin by the hunter is liable to pass on the infection to him. The insanitary underground inns where these hunters live in winter to escape

the severe cold contribute largely to the spread of an epidemic. Instead of entire prohibition of hunting, we have introduced laws for its regulation and have taught the hunters to detect and avoid sick animals, to be vaccinated against plague, and to have the animal skins stored and disinfected at our stations at Manchouli and Hailar. We are confident that although there will always be sick tarabagans, human infections will become rarer and rarer, and even when these do occur widespread epidemics like those of 1910-11 and 1920-21 will be things of the past.

In 1922, I received the honorary degree of Doctor of Science (Sc. D.) from St. John's University, Shanghai. In 1923 I was asked by Marshal Chang Tso Lin of Manchuria to build the North-Eastern Hospital at Mukden capable of accommodating 500 patients. This new structure was completed in June 1924, and consists of a Central Administration Block with covered pathways leading to eighteen separate verandah units, each with its own Arcola heating system and lavatory arrangements. A railway platform reaching the gate enables wounded patients to be conveyed to the wards without undue exertion. The total cost of this new Hospital is $700,000.

In August 1924 I obtained a Fellowship of the International Health Board of the Rockefeller Foundation to study modern health work in America and spent the greater part of a year at the Johns Hopkins School of Hygiene where the C. P. H. was granted me. I also visited other health and research centers, and brought back new methods for the protection of children against scarlet fever, which malady was particularly virulent in the north of China. For nearly two years now we have conducted this new line of investigation and have obtained very encouraging results.

My 'Treatise on Pneumonic Plague', upon which I had been engaged ever since 1911, was at last finished in 1926 and was submitted as a Thesis to the Imperial University of Tokio. For this work the rare honour of *igaku-hakushi* (Tokio) was conferred upon me, such a degree not having previously been given to any non-Japanese, Chinese or European. The League of Nations, Geneva, published this book in November, 1926.

Early in 1927 I was invited by the Health Section of the League of Nations to visit certain health centers and research institutions in Europe. I accordingly left Harbin on March 1, travelled *via* the Siberian Railway, visited in succession Warsaw, Vienna, Berne, Geneva, Lubliana, Zagreb, Belgrade, Paris, Havre, London, Cambridge, Hamburg and Berlin, before returning by the same route. The whole trip covered just four months, but as I had been to most of these places in previous years and could speak French and German besides English I managed to see more than the average traveller in such a short time. I was indeed impressed by the extraordinary changes that had taken place since the Great War. Instead of despondency in the defeated countries there prevailed the greatest determination among all classes to make the best of the situation and to adapt themselves to the fresh needs of the times. The victor countries, on the other hand, received additional stimulus and utilised every effort to come up to the standard of America in regard

to efficiency and mass production. Education has become more practical and business methods have been made less wasteful. Even medical training is becoming more *preventive*, so that the fewer births now taking place may live healthier and longer and cost the country less through sickness. A new world is being born!

I proceeded to Calcutta (India) in December 1927 to attend with two other Chinese colleagues the seventh Congress of the Far Eastern Association of Tropical Medicine and for the first time had the pleasure of meeting the leaders of medicine in India. In spite of the forty-century-old civilisation of that vast Empire and the consequent conservatism and religious bigotry of the masses, large numbers of Indian doctors have distinguished themselves in the fields of Ophthalmology, Surgery, Inner Medicine, Pharmacology, Bacteriology, etc., in addition to Biology, Physics and Mathematics. The names of Bose, Raman, Row and Chopra are as familiar in the west as in the east. But the application of modern health knowledge to the daily life of the average peasant and toiler is still much needed, and strenuous efforts are required in every direction to guide the ignorant populace in the right direction. The same difficulties which we have encountered in China are being experienced in India, perhaps to a greater degree.

My three months tour in India, during which I visited north and south as well as east and west and travelled over 12,000 miles, will long live in my memory.

Now I have reached the end of my autobiography. I have left out the part I took in founding and editing the National Medical Journal, my literary contributions to various medical and scientific journals in China, America, Europe and Japan, my lecture tours in Japan, Europe and Malaya, India and Burmah, my attendances at medical congresses in London, America, China, India and Japan, and my co-operation with a long list of friends (Chinese and foreign) in the work of the Y.M.C.A., Y.W.C.A., Council of Health Education, National and International Anti-opium Associations, Medical Schools scattered in different parts of China, etc.

For although much has been accomplished, still more has to be done, and I am always on the lookout for colleagues, who do not mind hard tasks, long hours, a simple life and severe training. For I verily believe that China, with her many-thousand-year-old civilisation, industrious peasants and brilliant thinkers and artistic scholars, as well as inherent democratic spirit ,can easily get into line with modern nations. But in order to achieve the quickest and most permanent results in the most economical way, her leaders should absorb the best that the West can offer, such as, seriousness of purpose, service to others as well as self, a scientific temperament, rigid scrupulousness in the management of business undertakings, attention to details and a willingness to learn from outsiders even at the height of success. On the other hand, they should eschew the weaker points of western civilisation, such as, undue worship of material success at the expense of the soul, over-indulgence in the ordinary comforts of life, numberless luxuries and lack of discipline in the family.

WU LIEN-TEH.

Harbin, September, 1928.

SUMMARY OF FIFTEENTH ANNUAL GENERAL REPORT, 1927.

Harbin, Oct. 12th, 1927.

To His Excellency,
The Minister for Foreign Affairs,
Peking.

Sir,—I have the honour to submit a summary of the Fifteenth Annual General Report of the North Manchurian Plague Prevention Service for the year ending September 1927.

2. *Plague*. The plague situation has been fairly good on the whole, India particularly showing a marked diminution of cases as compared with previous years. Hongkong has for the fourth consecutive year been freed from any infection. In freshly imported areas, however, such as Union of South Africa, California, etc. the bubonic plague has shown a tendency to extend among the wild rodents, resulting in occasional human cases. This problem of the pest among wild rodents deserves a more intensive study and I am glad to state that the Health Committee of the League of Nations has circulated a memorandum prepared by me among various governmental authorities of the world for an expert commission to proceed to the enzootic areas. Both in Manchuria and the Transbaikal regions we have encountered no cases whatever of human or rodent plague.

In Outer Mongolia, however, a localised outbreak of Pneumonic Plague occurred in the neighborhood of Chechan Chan, about 200 English miles south-east of Urga. The first victim was a Mongolian shepherdess, aged 23 years, who attempted to catch a tarabagan and afterwards fell ill with fever. headache and swellings in the armpit, neck and groin. She died on October 10th, and passed the disease to her mother-in-law, father, brother and other contacts who attended the sick. Altogether 24 cases were recorded (22 pneumonic and two bubonic). involving six localities. The epidemic lasted until December 13. Senior Medical Officer Chun of our Service with a contingent at once proceeded to Manchouli, Hailar and Dalainor and instituted preventive measures.

At least eight of the cases were confirmed bacteriologically by two experts sent by the Mongolian government.

As I write (Oct. 12th), some cases of Pneumonic Plague have been reported near Tung Liao (Paiyintala) in Inner Mongolia, within 20 miles of the Tungliao Branch of the Szepingkai-Taonan Railway, which runs into the South Manchurian trunk. Chinese and Japanese medical officers have proceeded to the spot and confirmed the disease bacteriologically. Our Manchurian Service has in addition dispatched the necessary anti-plague vaccines and adopted all preventive measures. Apparently, the outbreak has been smouldering since early September, first among the Mongols, then among the emigrant Chinese. It is a significant fact that

the last epidemic arising from Inner Mongolia was in 1918 (exactly ten years ago), proceeded along the Fengchen-Kalgan-Peking railway route and killed over 16,000 persons in the course of five months.

3. *Cholera.* Cholera continues to be a menace in the East from India to Japan. The former has been its original home for centuries and in spite of all efforts epidemics break out almost yearly in large and small cities. Siam reports thousands of cases annually, while in China the Yangtze Valley, with Shanghai as a centre because of its immense local and foreign shipping, has become quite a danger spot of the world.

A considerable outbreak occurring in 1926 and affecting northern as well as southern ports has already been mentioned in last year's Report. This summer again cholera cases commenced to appear in Shanghai in June, and though not so fatal as in 1926 caused widespread anxiety in Manchuria and Japan, where the authorities had to spend much money in preventing the spread of the infection.

Fortunately only a few sporadic cases occurred in Tsingtao, Tientsin, Dairen, Newchwang, Kobe, etc., having mostly been imported by ships from Shanghai. Shanghai itself claimed about 300 cases.

It is a pity that in spite of the new knowledge available respecting its prevention and treatment, authorities and commercial bodies with so much at stake have not yet united to make a systematic effort in ridding this easily controlled disease from their midst.

4. *Other communicable diseases.* Influenza appeared in a mild epidemic form during the spring, causing few fatalities. The incidence of Typhoid, Typhus, and Small pox is about the same as last year. Scarlet fever has been unusually mild and scarce. There has been a distinct increase of Acute Bacillary Dysentery during recent years, due partly to the increased consumption of bananas imported from Formosa where the fruit is forcibly ripened in the holds of steamers with rags soaked in polluted water. Several samples of the fruit examined bacteriologically have shown live dysentery bacilli on their skins.

5. Thanks to a Travelling Fellowship offered by the Health Section of the League of Nations, I left Harbin on March 1st on a visit to Europe for a study of the Health Organisations of twelve countries. Since my last visit there in 1913, considerable improvement has taken place everywhere, particularly in new countries like Poland, Jugo-Slavia, where a substantial part of their budget is devoted to health matters. For instance, Jugo-Slavia with a population of 12 millions and a budget of G$60,000,000 appropriates one-tenth for the health service. In that country they possess a remarkable chief medical officer and organiser in the person of Dr. Stampar (formerly country practitioner educated in Vienna) who has been able to build, equip and staff over 200 modern hospitals, laboratories and health centres during the course of 5 years. The main secret of his success lies in winning the hearts of the peasants by first saving their poultry, cattle and horses with suitable inoculations of vaccine and serums and then talking to them about the preservation of their own families.

In China, this laudable means may be adopted because the majority of farmers and agriculturists seem to lay more stress upon the welfare of their animals.

Although the birth-rate of such leading countries as Great Britain, France and Germany has decreased since the War, greater care is now being exercised upon saving the lives of children through welfare agencies, health insurance, school clinics, tuberculosis dispensaries and sanatoria. As a result a healthier and more intelligent generation is undoubtedly springing up to replace the old one.

Alcoholism and syphilis are far less frequent than formerly, and typhoid fever has practically disappeared where water supplies are properly guarded as in the cities of England. The future medical practitioner will be a consultant of health rather than a prescriber of drugs.

6. Among cities I visited were Warsaw, Vienna, Berne, Geneva, Zagreb, Belgrad, Paris, Havre, London, Cambridge, Hamburg, Berlin and Moscow. In Paris I sat as China's delegate at the International Conference on Rabies. I also delivered lectures in Warsaw, Geneva, Paris and London, and I returned to Harbin on June 28, having thus been away from China for nearly four months.

7. Modern health work has also made some strides in China, though not as fast as I could wish. Among large cities with properly trained Chinese doctors in charge may be mentioned Peking, Canton, Shanghai, Hankow, Nanking, Harbin, Tsingtao, Newchwang. In some of these proper vital statistics are being kept, but there is still a lack of co-ordination and insufficient support from the lay authorities, especially the police. A creditable exception lies in Mukden, capital city of Manchuria, where the Chief of Police has invited our Plague Prevention Service to help him organise an efficient health unit to serve as an example for the rest of that large province.

It is hoped that the medical schools in China, whether foreign of Chinese, will soon revise their curriculum, so as to turn out a larger number of graduates equipped with the latest ideas on the health movement. Otherwise, the task of supplying trained men and women for the 400,000,000 inhabitants of this country would be too immense within the time at our disposal.

8. I regret to learn of the death in Peking of his Excellency Chao Erh-sun, who passed away on September 3rd, at the ripe old age of 84. His late Excellency held the highest posts in the Ching Dynasty, and during his Viceroyalty in Szechuen (1907-09) was the first to set an example to other officials in ridding that province (which used to produce half the opium of China) entirely of the fatal poppy during the short space of two years. This great statesman succeeded Hsi Liang as Viceroy of Manchuria in 1911, soon after the devastating plague epidemic, and it was to him that our Manchuria Plague Prevention Service owes its actual establishment, for he sanctioned the sum of Taels 120,000 from the Fengtien Treasury for constructing our permanent hospitals at Harbin, Manchouli and Taheiho. The memory of Viceroy Chao Erh-sun will ever be green among Chinese anti-plague workers.

9. The past summer has been one of the hottest on record in most parts of China. The continued troubles in the South have intensified emigration into Manchuria from the provinces of Shantung and Chihli. During the past five years, at least 2,000,000 people have made their home in these fertile regions, and as a result more and more land is being opened up and cultivated. Mukden, Dairen, Harbin, Changchun and other cities on the Sungari River have all increased in population. That of Harbin is nearing the half-million limit. Thanks to the good government of the Chinese Eastern Railway Area under General Chang Huan-hsiang, complete security and steady progress are now assured, and trade has prospered exceedingly. In Harbin, electric trams are running after nearly ten years' preparation, and now waterworks for the city are being planned. The whole city certainly looks smarter and brisker than during the Russian regime.

All along the Railway, such towns as Anta, Mankou, Suichingshan are showing ever increasing signs of prosperity because of the unlimited output of beans and wheat. Manchuria is rapidly earning for itself a name as one of the leading granaries of the world.

10. The following figures show the number of out-patients treated at various station hospitals of our Service :

	1926-27	1925-26	1924-25	1923-24	1922-23
Harbin	12077	16943	22874	15661	15343
Taheiho	5443	3246	4669	7327	8037
Sansing	4105	3816	6603	4379	5235
Lahasusu	1356	2388	1629	1569	1470
Newchwang	6588	6263	4675	4100	3693
Manchouli	2038	1674	3234	2214	2347
Hailar	5420	2567	—	—	—
	39027	36891	43684	35250	36125

The in-patients at Harbin numbered 570 (213 medical and 357 surgical). There is still much advanced tuberculosis of the bones and joints, for which only amputation can save the lives of the patients. Many of our people are still not alive to the benefits of modern medicine.

11. The personnel of the Service for 1926-27 are as follows :—

Dr. Wu Lien Teh, Director and Chief Medical Officer.
Dr. J. W. H. Chun, Senior Medical Officer, Harbin.
Mr. P. Barentzen (Commissioner of Customs) Lay Director and Treasurer, replacing Mr. U. Marconi.
Dr. Lin Chia Swee, Senior Resident Medical Officer, Harbin.
Dr. R. Politzer, Bacteriologist, Harbin.
Dr. N. B. Young, Senior Resident Medical Officer, Newchwang.
Dr. C. L. Shih, Resident Medical Officer, Harbin.
Dr. Y. M. Kwan, Resident Medical Officer, Taheiho.
Dr. Li Yuan Po, On leave, study in Paris from September.
Dr. W. H. Shih, Resident Medical Officer, Manchouli.
Dr. Li En Chang, Resident Medical Officer, Sansing.

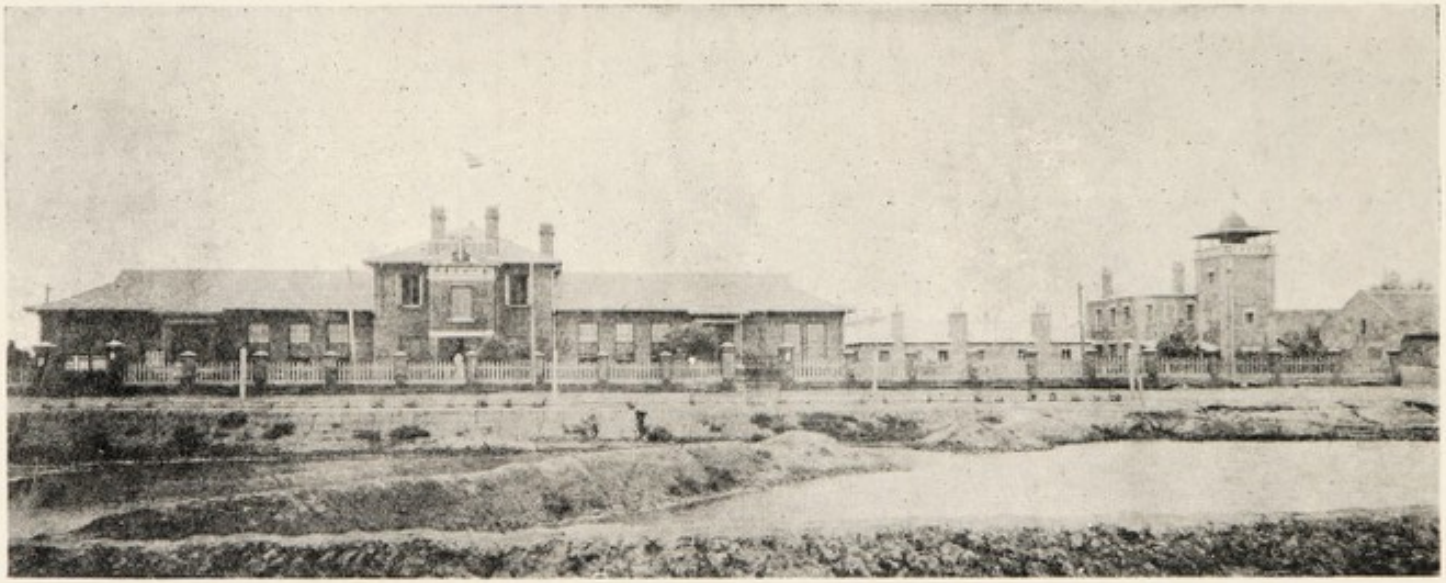

Fig. 63. General view of Newchwang Quarantine Hospital, from River (1928)—
Hospital Block on left; Quarantine Block on Right.

牛莊海口檢疫醫院由河邊看之全圖 （民國十七年落成） 左邊卽爲醫院右邊爲隔離所

Fig. 64. Exterior of New pneumonic plague block completed 1927, having separate entrances for medical staff and patients and exit for cadavers.

民國十六年落成之新築肺疫病室外部示醫員與患者有不同入口部及屍體出口部

Fig. 65. Interior of ward showing medical staff in observation niche injecting serum into patient's arm through opening in glass partition without exposure to infection

同上內部醫員診療肺疫症者及在預防窗口外施行血清注射療法疫患者之手由玻璃大障下之窗口伸出以防醫員診治時之意外傳染

Fig. 66. Interior of same ward, showing patient extending arm outwards into the niche to have blood tested, etc.

同上病室於大障內部示疫者將手由病預防窗口伸出以備醫員取血檢查

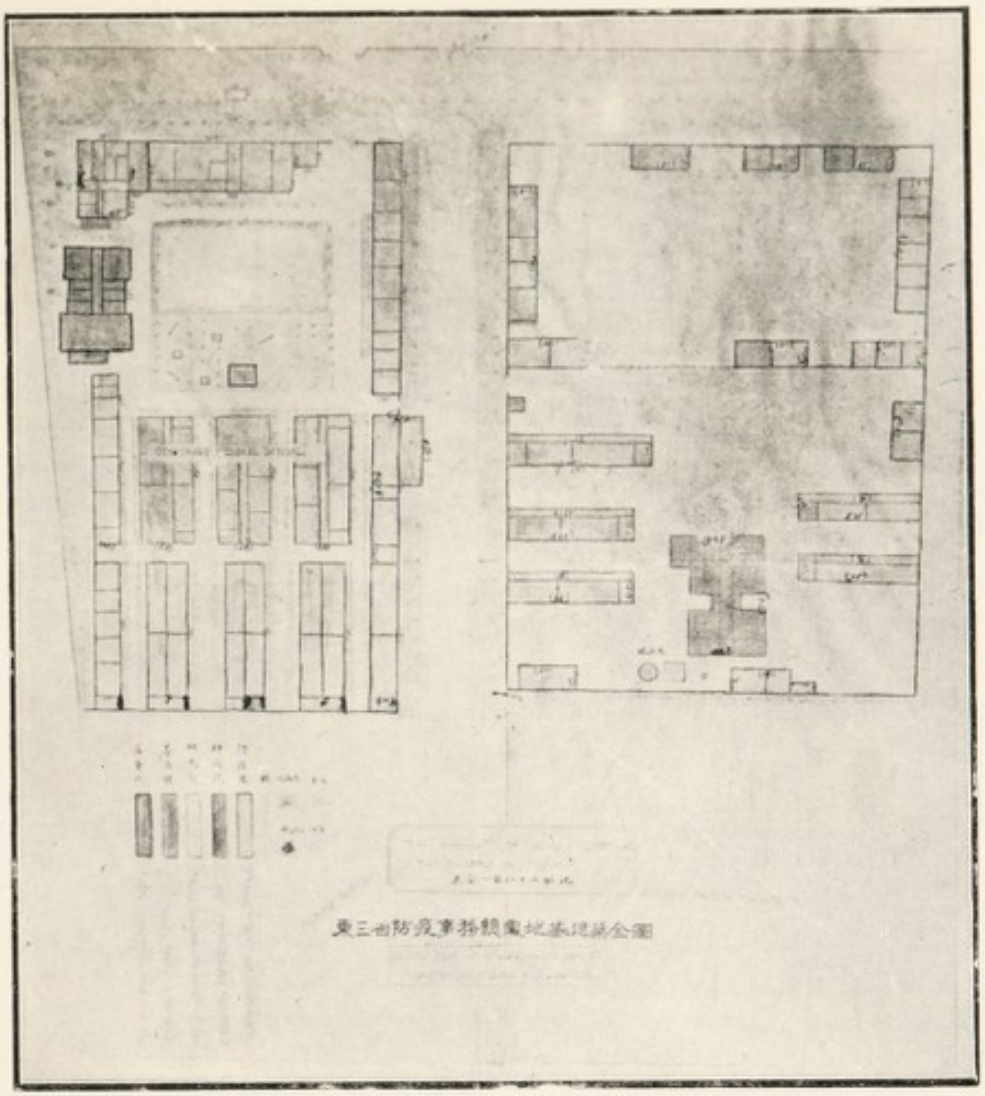

Fig. 67. Plan of Harbin Hospital Grounds (187 mou or 61 acres).

濱江醫院平面圖計一八七畝合六十一英畝

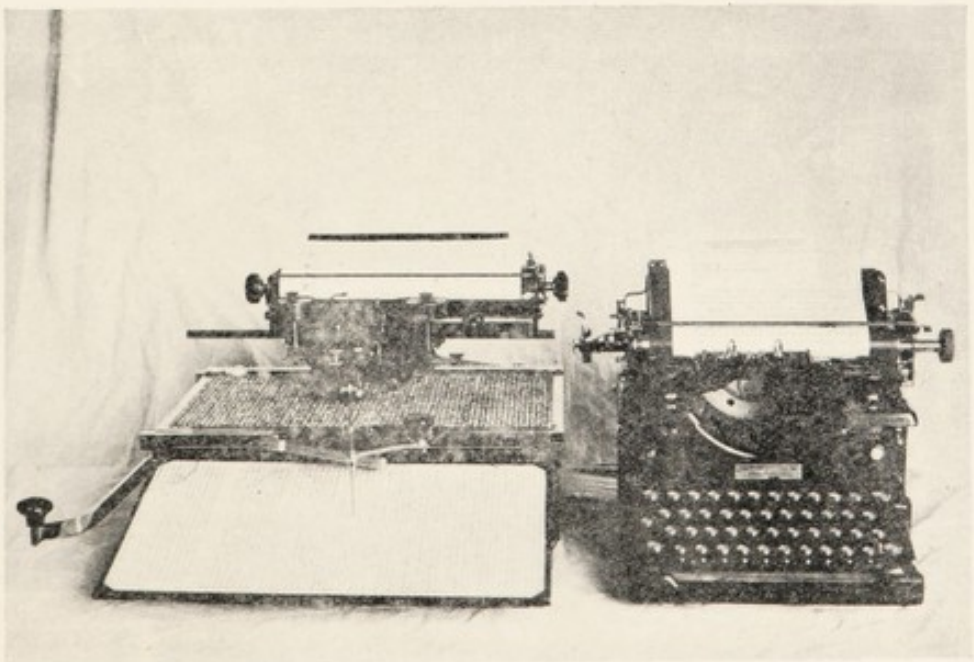

Fig. 68. Comparative sizes of Chinese and American typewriters as used in our Office.

中西打字機兩圖之比較卽防疫處辦公用者

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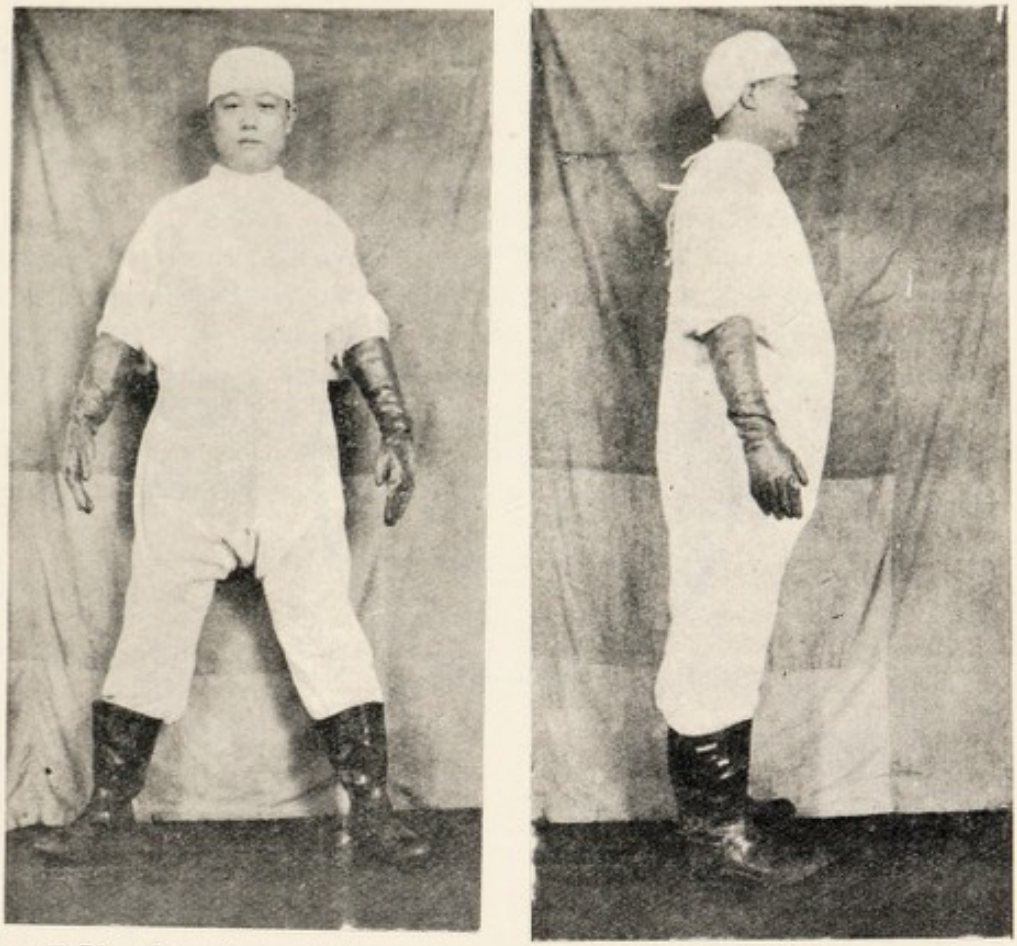

Figs. 69-70. One-piece anti-flea costume as used during bubonic plague outbreak in Tungliao 1928.

示防跳蚤之裝束于通遼一九二八年腺型鼠疫流行時所用之全連防疫衣

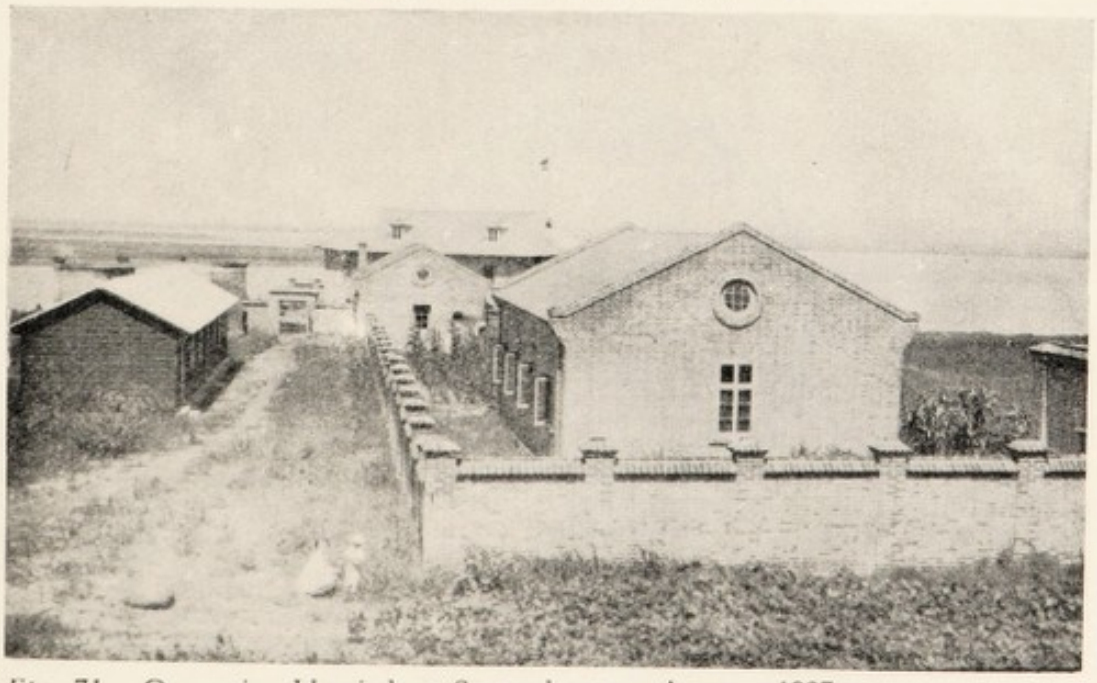

Fig. 71. Quarantine Hospital at Santau-langtau, Antung, 1927.

民國十六年落成之安東山頭浪頭之隔離檢疫所

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Mr. Tung Lan Chi, Senior Dresser in charge, Lahasusu.
Miss Liu Chi Chieh, Deputy Female Medical Officer, Taheiho.
Miss N. C. Chung, Senior Nurse, Harbin.
Miss Liu Tzu Ming, Senior Nurse, Newchwang.

12. In conclusion, I wish to take this opportunity of expressing my thanks to various departments of the Chinese Government, to the Chinese Eastern Railway and the South Manchurian Railway for facilities granted to us in the prosecution of our work. As in the former years, the Soviet Government has offered frequent facilities for our investigations. We are also indebted to the Customs Officers throughout Manchuria for assistance rendered to our Service. Finally, I beg to record the faithful and unfailing devotion to duty shown by members of our staff.

I have the honour to be,

Sir,

Your obedient Servant,

WU LIEN-TEH,

Director & Chief Medical Officer.

SUMMARY OR SIXTEEN ANNUAL GENERAL REPORT, 1928.

Harbin, September 30th, 1928.

To

His Excellency,

The Minister for Foreign Affairs,

Nanking.

Sir,

I have the honour to submit a summary of the Sixteenth Annual General Report of the North Manchurian Plague Prevention Service for the year ending September, 1928.

2. *Plague.* This infection continues to be widely distributed, fresh areas being added to the already long list of infected localities. The British colony of Aden (off Arabian Coast) which had for years been free from the disease reported 19 cases in December 1927, 178 in January 1928, 343 in February, since when there has been a regular flow of cases, until now a total of 2000 has been recorded. India reports an average of 20,000 cases monthly. French Indo-China is widely invaded. Hongkong with a clean bill of health since 1923 has since this spring recorded half a dozen human cases. Siam and Ceylon are also attacked though not seriously.

In China and Manchuria all seemed well until last August when suspicious deaths were reported from the Tung Liao district, which had been recently opened up for cultivation mainly by the building of the Ssupingkai-Chengchiatun-Taonan (Ssu-Tao) and Ssupingkai-Chengchiatun-Tungliao Railways, which branch off at Szu Ping Kai from the South Manchurian Trunk line. Also the completion of the 250 miles track between Tungliao in the north and Tahusan in the south (on the Peking-Mukden Line) has brought this vastly fertile new region within easy reach of Mukden and Peking. Unfortunately, such easy access by rail has also introduced attendant dangers of Plague from the endemic centers of Outer Mongolia.

Telegraphic enquiries were sent me both from the League of Nations in Geneva and the Eastern Health Bureau at Singapore for information regarding 20 suspicious deaths at Tungliao early in August. I immediately took train and visited the spot (410 English miles from Harbin).

It appeared that local doctors had seen some patients complaining of fever, headache, and unconsciousness, sometimes diarrhoea, and dying within two to three days after first appearance of symptoms.

In one of the six persons living within the same compound a bubo in the groin was suspected. Blood-spitting was never seen. There was no unusual mortality among domestic rats. I caught six wild rodents belonging mostly to the *Spermophilus* group but none of these showed any disease.

In the first week of September, a request came from the authorities of the Ssu-Tao Railway asking for medical help as some suspicious deaths had occurred at Chienchiatien (錢家店) a village of 1600 persons lying 23 miles east of the railway terminus at Tungliao. Senior Medical Officer Chun Winghan at once proceeded there on September 5. On the 7th he wired back that he had seen a case with femoral bubo, the contents of which showed plague bacilli under the microscope. Cultures and other bacteriological tests in our Harbin Laboratory have since confirmed the diagnosis of true Plague. It is probable that, like last year, some isolated cases have been smouldering on the Mongolian frontier since early summer and have now taken an active form.

I have duly informed our Central and Fengtien authorities of the findings and urged them to take all possible precautions in the way of isolating the sick, quarantining the village, rat destruction, rigid control of passenger traffic from the affected districts, and systematic vaccination of the inhabitants with antiplague preparations made at our Institute.

Being at present of the bubonic form, the dangers of a rapid spread among the masses are not so great as in the case of pneumonic plague, but this epidemic is fresh and evidently very virulent, and no pains should be spared in stamping it out from the beginning.

3. From Urga we have also received telegraphic information about a human plague outbreak, and the Mongolian and Russian Medical Departments have requested us to send 3000 doses of vaccine and 10 liters of serum to them. This request we have carried out, the boxes containing the medicine being sent *via* the Siberian Railway as far as Verkne-Udinsk, whence they were transported to Urga by aeroplane. So, even in far off Manchuria and Mongolia we are making full use of the latest means of communication for aiding distressed humanity.

4. *Cholera and other Infectious Diseases.*

Thanks largely to early precautions undertaken by the Health Departments in Shanghai, there have only been sporadic cases of *Cholera* in that area during 1928. Apart from a cleaner water supply (the Chinese have erected a splendid new plant with rapid sand filtration at Woosung). Dr. Houki Hu, Health Commissioner of Greater Shanghai, has vaccinated 250,000 employees of local mills and factories against Cholera during this summer. Because no epidemic has occurred at Shanghai, the whole of Manchuria is spared.

Influenza seems to have taken firm hold of this Province, for quite a number of outbreaks have been reported from various localities. In some pneumonia set in resulting in death, but as a rule the symptoms were mild.

Small-pox continues to be endemic but is rarely fatal among Chinese.

Scarlet Fever has not taken a severe form for some years, though thanks to the exodus of immigrants from Shantung there was an unusually large number of *Typhus* cases among them. At Dairen over 100 cases were received in hospital, while along the route to Harbin we had quite a stream of such cases during the spring.

Typhoid Fever and *Dysentery* are present, particularly the latter, which seems to be particularly virulent among Japanese residents.

5. *Visit to India.* Under orders from the Central Government, I attended the Seventh Conference of the Far Eastern Association of Tropical Medicine held at Calcutta from December 5 to 22, 1927, with Drs. Chin Tzu Chih and Hu Cheng Hsiang as colleagues. The opening speech of welcome was made by H. E. the Governor of Bengal (Sir Stanley Jackson), while the President of the Conference was Surgeon-General T. H. Symons (Director-General of the Indian Medical Service). About 1,000 names of doctors were registered, of whom nearly 200 came from abroad.

I read two papers based upon our researches conducted in Harbin during the last two years, namely:

1. The Perpetuation of Plague among Wild Rodents.
2. Problems of Pneumonic Plague.

The actual sessions of the Congress terminated on December 10 with a grand banquet at Belvedere, former home of the Viceroy. Covers were here laid for 1,100 persons (including families of the delegates), and I was one of the six speakers chosen for the evening.

At the final session, it was provisionally decided to hold the next Conference at Peking in 1930, if political conditions at that time allowed. There was unnecessary nervousness among some of the delegates as to the safety of Peking as a *venue* for the Conference, and a Committee of five representatives (from China, Japan, Siam, Philippines and Netherland Indies) was appointed to finally settle the matter.

The foreign delegates were on December 11 taken on a round tour, some over Northern India, others over Southern India, which lasted until December 22, when they all met at Bombay and then dispersed. During these three weeks, the official delegates were treated as guests of the Indian Government, all hotel and travelling expenses being defrayed by the Government.

6. After the above Congress, I represented our Government on the Advisory Council of the League of Nations Eastern Health Bureau, which for convenience sake held its annual meeting from December 26 to 29 at Delhi (new capital of India) instead of Singapore. The results of our deliberations have already been submitted in a special dispatch to the Wai Chiao Pu last April.

7.—My third mission in India was to represent the Chinese Government together with Dr. Chin Tzu Chih on the Health Interchange organised by the League of Nations. This work started on January 1, 1928 from Delhi, and took us over all parts of India, north as far as Rawalpindi and Darjeeling (gateway to Thibet), south as far as Mandapan (quarantine station opposite Ceylon), east as far as Mariani (tea gardens of Assam, where fatal epidemics of Kala-azar had occurred for years) and west as far as Bombay (where much plague existed). Our party consisted of 18 doctors representing China, Japan, Siam, Chosen, Philippines, United States, Netherland East Indies, Australia, New Zealand, Egypt, Singapore, Malay States, Ceylon, French Indo-China, League of Nations Health Section and Indian Medical Service.

The tour terminated on February 17 at Bombay, after which the delegates separated. Among institutions visited were:

Hospitals	28
Waterworks	11
Medical Colleges	11
Sewage farms	6
Vaccine Institutes	7
Jails	5
Health centers	7
Pasteur Institutes (Rabies)	5
Leper settlements	5
Mental hospitals	3

besides hospitals for infectious diseases, emigrant stations, coal mines, municipal markets, melas (religious fairs), etc. Altogether we covered a distance of 12,000 miles by rail (sometimes automobile) travelling mostly by night and inspecting places by day. To those who had not been to that vast ancient empire before, this tour with its extremes of wealth and poverty, splendid art and crude industries well-endowed educational institutions and mass illiteracy, lofty culture and primitive practices, was indeed a never-to-be-forgotten experience.

I reached Harbin on April 7, having investigated medical conditions at Rangoon in Burmah and Bangkok (capital of Siam) on the way back.

8. The occupation of Peking in June by the Nationalist forces and the transfer of the capital to Nanking mean that henceforth all our communications will be addressed to the latter city. In July I made a trip to Peking and thence Nanking *via* Chengchow, Kaifeng, Hsuchow and Pukow to pay my respects to the new Minister of Foreign Affairs (Dr. Wang Chen Ting) and Minister of Interior (Mr. Huseh Tu Pi), incidentally discussing with them the prospects of new medical, health and quarantine work under the more active regime. I trust that greater and more systematic efforts will now be made by our Government to promote health and medical work in this country, for the strength, happiness and economic welfare of the nation depend largely upon it. This can best be effected by proper concentration of all health activities in a separate Ministry of Health such as has been established in Great Britain, France and other forward countries.

7. The assassination of Marshal Chang Tso Lin and General Wu Chun Sheng by some unknown agency with some land mine at daybreak on June 4 while their train was passing under the South Manchurian Railway viaduct near Mukden was a great shock to the inhabitants of this Province. In the new Marshal Chang Hsueh Liang, however, we have a young, active, conscientious and modern-trained chief who will surely lead the people to further prosperity and content.

8. The summer began with some very hot days, but throughout July, August and September there has been almost unceasing rain throughout the greater part of North Manchuria, so that this year may be called one of the wettest on record. The Rivers Sungari and Amur have both surpassed their usual level, so that in the Amur district there have been disastrous floods entailing considerable suffering and monetary losses

among the population. Because millions of pieces of firewood have been washed away, there may be a timber famine this winter.

On the whole, however, the country has been blessed with bumper crops of beans, wheat, kaoliang, millet and hemp. Thanks to the extensive migration of people from the famine and war-stricken areas of Shantung, the virgin soils of these parts, the Taonan and Tungliao districts are now cultivated with profitable crops, which will increase in quantity as time goes on. In fact, with rapid means of transit and an abundant and industrious population developing the fertile enormous territory, it is quite easy to predict that before long Manchuria will become one of the biggest granaries of the world.

The city of Harbin has made remarkable strides and can now boast a population of over 400,000. Many new schools have been built including a medical one attached to our Harbin Hospital. Electric trams are running and new waterworks will soon be built. The streets are cleaner and busier than ever, and for five local cents one can take a bus ride from the Chinese city to the Special Area two miles away. Land, house-rent and the cost of living have all gone up, and plenty of ready cash seems to be in evidence. Both the Chinese Eastern Railway and the South Manchurian Railway each declared a net profit of 40,000,000 Yen last year. The acme of prosperity has not yet been reached!

9. The next Volume (VI) of our Plague Prevention Service Reports for the years 1927-8 is now in the press and covers most of the research and routine work accomplished by our staff during the past two years. There will be both a Chinese and an English edition of over 300 pages each.

10. The following figures show the number of out-patients treated at various station hospitals of our Service:

	1927-28	1926-27	1925-26	1924-25	1923-24
Harbin	8883	12077	16943	22874	15661
Taheiho	4434	5443	3246	4669	7327
Sansing	3643	4105	3816	6603	4379
Lahasusu	1284	1356	2388	1629	1569
Newchwang	6637	6588	6263	4675	4100
Manchouli	1977	2038	1674	3234	2214
Hailar	5905	5420	2567	—	—
	32763	39027	36891	43684	35250

11. The personnel of the Service for 1927-28 are as follows:

Dr. Wu Lien Teh, Director and Chief Medical Officer.
Dr. Chun Wing Han, Senior Medical Officer, Harbin.
Mr. Paul Barentzen (Commissioner of Customs), Lay Director and Treasurer.
Dr. Lin Chia Swee, Senior Resident Medical Officer and Dean of the Pingchiang Medical School, Harbin.
Dr. R. Pollitzer, Bacteriologist of the Service.
Dr. H. Jettmar, Serologist of the Service.

Dr. Yang Ting Kuang, Senior Medical Officer, Newchwang.
Dr. Shih Chi Liang, Resident Medical Officer, Harbin.
Dr. Kwan Jen Min, Resident Medical Officer, Taheiho.
Dr. Li Yuan Po, Parasitologist and Resident Medical Officer, Manchouli.
Dr. Shih Wei Hua, Resident Medical Officer, Manchouli (resigned on 23rd July, 1928).
Dr. Li En Chang, Resident Medical Officer, Sansing.
Mr. Tung Lan Chi, Assistant in charge, Lahasusu.
Miss Liu Chi Chieh, Deputy Female Medical Officer, Taheiho.
Miss N. C. Chung, Senior Nurse, Harbin.
Miss Liu Tzu Ming, Senior Nurse, Newchwang.

12. With this Report ends the sixteenth year of our Service. I am indeed obliged to my superiors for the continuous encouragement and support they have given me in maintaining what is now one of the best known health organisations of the world. Although I could well have done with a bigger budget for the better prosecution of this much-needed work, still, considering the immense troubles which this country has gone through during the past years, I am thankful that so much has been accomplished with the limited means at my disposal, not only in keeping all our institution in an efficient state but also in erecting and equipping new hospitals and laboratories to replace old ones without any special appropriation. For this credit is due as much to my faithful and hard-working staff as to my humble self.

In conclusion, I wish to take this opportunity of expressing my thanks to various departments of the Chinese Government and to the Chinese Eastern and South Manchurian Railways for facilities to me and my staff in the prosecution of our work. The Soviet authorities have on frequent occasions rendered much assistance in our investigations. I am also indebted to the Customs officers throughout Manchuria for helping our Medical Officers whenever needed.

I have the honour to be,

Sir,

Your obedient Servant,

WU LIEN-TEH,

Director and Chief Medical Officer.

SUMMARY OF THE HARBIN HOSPITAL REPORTS FROM OCTOBER 1926 TO AUGUST 1928.

1. Pneumonic Plague Epidemic in Outer Mongolia in October 1926.

The focus was situated 35-50 miles from Chechan Han. The first case occurred on October 10, resulting in the infection of 24 people (23 pneumonic and 1 bubonic who recovered). The epidemic involved six localities and lasted about five weeks.

The first victim was a girl, Purba, age 23, a shepherdess in the locality called Chulutoi. The story was that she tried to catch a tarabagan and chased it into a hole. Soon after this encounter, she fell ill, suffering from fever, headache and swellings in the arm-pit, the neck and groins. She died after six days of illness. The patient's mother-in-law, her father and brother were successively infected.

Then two lamas from the adjacent monastery were summoned to treat the sick and observe the ceremonies. One of these lamas showed symptoms of pneumonic plague after returning to the monastery. He was visited by relatives and nursed by his pupils with the result that they became infected. These infected relatives took the disease to their *jurtes* which were two in number, and situated on the right bank of the Kerulen river. The inmates of these *jurtes* were all attacked and died. Then the neighbours recognized the nature of the disease, and avoided contact with them. Thus the epidemic was stopped.

An expedition sent by the Mongolian Government arrived afterwards and found 8 corpses. Materials were removed for bacteriological examination and found to confirm the plague nature of the epidemic.

The infected territory being 200 miles east of Urga and on the direct route to Manchouli and Hailar, it was apparent that the danger of plague invasion of Manchuria became very real. Moreover, the advent of motor traffic in this region made this news doubly serious because the infected center was well within two days' journey from the Chinese border, while in former days it would have taken seven days' continuous travelling. In order to avert this invasion, our Service sent a medical contingent to Manchouli and Hailar to arrange preventive measures, such as restriction and inspection of travellers, and facilities for quarantine. These were accordingly put into effect for a month, with the happy result that no plague invasion of Manchuria occurred.

2. At the beginning of 1927, an epidemic of influenza swept over Harbin. Though the disease was exceedingly prevalent, it was not a very fatal type. Usually after 3-7 days the patients recovered quickly, though some pneumonic complications did supervene. At that time, influenza was simultaneously creating havoc in England and Japan, and Manchuria and North China were not spared.

During March, nine cases of cerebro-spinal meningitis were reported. The type was very severe, for the mortality was 66%. These cases occurred mostly among children, both Chinese and Russian.

With the advent of summer in 1927, preparations were made against any possible invasion of cholera from the south. On the appearance of some true cases in Shanghai, anti-cholera inoculations were done at the communicating ports of Newchwang and Dairen. Further and more stringent steps were ready to be applied if the disease were to spread. But fortunately we were spared of its visitation. In Harbin, however, we had the usual number of summer intestinal diseases, such as dysentery and enteritis. Three cases of choleraic diarrhoea were admitted, and successfully treated by hypertonic saline injections. The clinical signs closely resembled true cholera, and only bacteriological investigations were able to distinguish the difference.

3. A plague outbreak was reported to have occurred during September and October 1927 in villages within 20 miles north of Tungliao (Payintala), which is the railway-head of the Tungliao branch of the Szepingai-Taonan Railway. The nature of the disease was ascertained microscopically by Japanese doctors, the type being stated to be both bubonic and pneumonic. Our Service rendered assistance both in personnel and equipment to the Szepingai Railway in their campaign against the spread of the infection which fortunately died out after a month or so. The number of victims both Mongols and Chinese were about 95, and the disease was said to have been brought from Inner Mongolia to the villages by some lamas. On this occasion, our laboratory experts were able to show their usefulness by the manufacture of vaccine and settling the question of one 'suspect' case on the Harbin-Changchun train. This case of sudden death was alarming at the time, but we were able to prove that plague was not the cause of death.

4. During the winter of 1927, many communicable diseases occurred in epidemic form. Scarlet fever has been quite preponderant, our hospital recording 48, 22, 26, and 14 cases respectively in October, November, December and January. 32 cases of small pox were recorded in January in Harbin. Measles, influenza, relapsing fever, and typhus also prevailed.

In connection with small pox, typhus and relapsing fever, mention must be made of their frequent presence among the exceedingly numerous immigrants from Shantung. In former years, immigration only took place in the spring, but owing to civil war and famine, hundreds of thousands have continued to come to Manchuria throughout the winter, and many cases of infectious diseases were brought along with them. Whole families travelled together, men, women and children, taxing the accommodation of the Chinese town to its limit.

5. The summer of 1928 brought its usual train of bowel diseases such as enteritis and dysentery (dysentery 29 in July). Two cases of choleraic diarrhoea were treated in the hospital. No true cholera cases were seen, neither were we apprehensive, because the situation in Shanghai seemed reassuring.

6. *Public Health Measures.* Our Service has taken steps to induce the authorities in several important towns in Manchuria to take interest in

Public Health and to adopt certain preliminary measures such as the recording of vital statistics and notification of infectious diseases. Encouraging responses have been forthcoming, and it is not too much to hope for the actual attainment of certain basic steps.

As an important Public Health measure, we give free vaccination against small pox every year in the spring. Large number of people take advantage of this, and bring their children to the hospital for vaccination, or "plant the flower", as the Chinese call it. They also believe that vaccination in the spring, a time when all vegetation starts to grow, is the best time for ensuring a successful inoculation.

Our laboratory prepared large quantities of anti-scarlatinal toxin for systematic immunisation of the school children along the Chinese Eastern Railway and within the Municipal area of Harbin. The usefulness of this method of preventing a highly infectious and dangerous disease is proved beyond question and if put into practice persistently and widely, only good results can occur in the long run.

The examination and analysis of many patent medicines takes quite a deal of our time. Like the people of other nations, the Chinese are very fond of trying this form of self-cure. In many instances, no serious harm can be incurred, but often the reverse is the case. We have on two occasions found the presence of cocaine in anti-opium pills or drops. It would indeed be jumping from the frying pan into the fire, if one were to contract the cocaine habit, though cured of opium craving.

7. During the period under review, 648 in-patients were admitted, and 210 operations were performed. Our operation technique is now so perfected that healing by first intention almost invariably follows a clean operative manouvre. In our clinical work, much assistance has been rendered by laboratory facilities which are always willingly given by Drs. Pollitzer and Jettmar who take great pains in eliciting the true nature of morbid specimens and discharges. Special mention may be made of the ready availability of the Kahn test for syphilis, a test which is so handy and reliable for this disease only too common among our patients.

We have been able to make good use of some of the newer and patent drugs which are put in the market in such large numbers and in such quick succession. We have found Novasurol, for kidney stimulation, Mercuro-chrome for typhoid and meningitis, Plasmoquine for malaria, Angiolymphe for tuberculosis, and Monsol as an antiseptic particularly effective, and they seemed to possess the distinctive properties that are claimed for them.

It may be worth while to cite a few cases to show the type of disease which passed through our hands.

a. *Liver abscess bursting into pleura.* Wang, aged 24, was admitted for shortness of breath for two months. Past history was negative for dysentery On examination, he was a pale man of medium physique. He was obviously distressed, and somewhat emaciated. The temperature was in the neighbourhood of 101° F. and pulse about 100. The right chest and right side

of the abdomen were distended, and there was evidence of fluid in the pleura. The liver was enlarged. On tapping the pleura, 1000 c.c. anchovy-like pus was evacuated. Meanwhile emetine injections were started. In was decided to resect a rib, and accordingly the 9th rib in the post-axillary line was resected under novocaine anaesthesia. Five litres of typical amoebic pus was let out. After operation, the patient seemed to go down-hill, and lost strength; gradually he became weaker and died on the 6th day. The pus was examined for entamoeba histolytica, but none was seen. In spite of emetine injections, and washing of the cavity with quinine lotion, he did not respond to treatment.

b. *Traumatic aneurysm of the femoral artery.* Wang, aged 55, was shot accidently in the right thigh three weeks before admission. It was a revolver bullet and it passed antero-posteriorly at the junction of the middle and lower thirds. On examination, the wounds of entrance and exit were healed. They were both relatively small and about the same size. No bones were fractured. The patient was fairly well nourished, and in good physical condition. There was a pulsating tumour, round in shape, having the circumference of about 3 inches in the lower third of the thigh. This tumour was expansile and with a thrill. Loud systolic bruit was also heard over the mass. On compressing the femoral artery, the tumour became smaller. Operation wos decided upon under local anaesthesia, and the femoral artery was tied at the apex of Scarpa's triangle. The wound healed well, and there was no gangrene of the toes afterwards. The aneurysm also stopped pulsating and began to diminish in size. Two weeks after the operation, however, the patient began to have fever and swelling of the aneurysm. There was evidently suppuration of the sac. The skin over it was red and swollen, and oedematous. It was decided to evacuate the pus under novocaine, and about 3 oz of dark saneous pus was let out. The cavity was washed with acriflavine daily and it began to shrink, healing being completed by granulation.

c. *A case of ascites.* The patient was a young man of 21, barber by profession, complaining of enlarged abdomen for the past year. On examination, he was a thin, brown-skinned and poorly developed man. There was much free fluid in the abdomen. The superficial veins were dilated. The liver and spleen could be easily felt, down to half way between the sterum and umbilicus. On tapping, half a bucket of straw coloured fluid was drawn off, but the accumulation returned. It was determined to operate on him, and Talma-Morison's operation was successfully performed. Healing was by first intention, but after ten days or so, the fluid again accumulated. At operation, the liver and spleen were examined and found to

be large and congested. The surfaces were smooth, and adherent to the diaphragm above. Before operation, the patient was carefully examined for blood and intestinal diseases, but nothing abnormal was found. This is another of those numerous ascites cases with obscure etiology. The question of alcohol as a cause of the disease did not come in. Kala-azar was also ruled out.

d. *Sarcoma of the optic nerve.* A boy, 7 years of age, was admitted for growth of the right eye. The history was that 4 months ago, his eye began to bulge, and within one month the cornea was destroyed. There was considerable pain in the head and eye. On admission, the right eye was pushed out by something in the orbit, or the eye itself might have been involved by a tumour. No glands were felt. The sclerotic was much congested, and the cornea quite white. On operation, the eye was removed, but after the optic nerve was cut, another tumour was seen to be at the bottom of the orbit. This seemed to be incorporated with the nerve, and about the size of an eye-ball. There was some adhesion to the walls of the orbit. The tumour was removed, and the soft tissues of the orbit eviscerated. The patient did well, and the wound healed up gradually. The pathologist reported that the tumour was composed chiefly of loose fibrous tissue somewhat resembling glioma. But at the periphery of the growth, there were sarcomatous cells of the mixed type.

e. *Traumatic aneurysm of the left brachial artery.* Patient was a young married woman of 18. While her husband was examining a revolver in the next room, it went off, the bullet passing through the wooden partition and shot her in the left fore-arm. It entered the limb on the inner aspect just below the elbow, and passed upwards and outwards, wounding the brachial artery in its lower third. The larger wound of exit was near the insertion of the deltoid. The bone was not touched. There was considerable bleeding. The patient lived a long distance from the hospital in another town, so she did not come till two weeks after the accident. On examination, she was very pale and there was constant oozing from the wound of exit. At the front of the lower part of the arm, there was a pulsating tumour of the size of a dollar. The whole area was more or less swollen. There was no severe infection and the patient was not feverish. Operation was advised, but we had to wait for the consent of the husband. One day, after a week in the hospital, there was severe bleeding from the wound of exit, and the tourniquet had to be applied. Operation was performed forthwith under local anaesthesia. An incision was made on the inner aspect of the arm, between the biceps and triceps muscles in order to tie the brachial artery in the middle of the arm. The whole part was swollen and it was rather difficult

to find the artery. But after careful searching and releasing the tourniquet, it was found and tied with cat-gut. The wound was closed without drainage. The wound of exit was also enlarged, cleaned, and clots removed. The patient rallied well with sub-cutaneous saline injections. An attempt was made to give blood transfusion, the donor being her mother of 60, but some difficulty was met with in obtaining the free flow of blood. Still, though the patient was very anaemic, she recovered slowly but surely. The aneurysm diminished. The wound became a little septic, but healed well. The radial artery of the left arm was found to be obliterated on admission, and remained so after operation. There was no gangrene of the fingers, and eventually collateral circulation was well established.

f. *A case of splenomegaly.* Liu Chia Tai, aged 17, son of a beggar, stated that he was brought up in Manchuria from the third year of life. The complaint was that the abdomen gradually enlarged within the last year. On examination, he was an underdeveloped boy of about five feet in height. He was fairly well nourished. a little pale, and bleeding easily from the oral mucous membrane. The abdomen was enlarged. The spleen was two inches below the umbilicus. The liver was not palpable. There was slight amount of free fluid in the peritoneal cavity. A few superficial veins were seen. Sexual development was very poort Other organs were healthy. Faecal examination revealed only oxyuris ova. The temperature was normal. The blood showed a secondary anaemia picture. Spleen puncture did not reveal kala-azar, or malaria. Splenectomy was decided upon. On opening the abdomen, slight manipulation caused much of bleeding in the peritoneum. Unfortunately, the spleen was found to be firmly adherent to the diaphragm and to the ribs. So the operation was abandoned, and the abdomen was closed. Two days afterwards, the patient developed fever of 104° F. and died on the third day.

g. *A case of strangulated hernia, for ten days.* Lu, a young man of 20, suffered from right inguinal hernia for 5 years. Ten days before admission, he found the hernia would not go back. There was a great deal of pain. The bowels were confined, and for the last two or three days there was vomiting of bile and faecal smelling fluid. On examination, he did not seem very ill. The temperature was subnormal, and the pulse was 90. There were no signs of peritonitis. Operation was performed at once. On opening the sac, omentum was found to be adherent to a knockle of intestine and to the wall of the sac. At the bottom of the sac there was some serous fluid, and it was here that a piece of the omentum was found to be necrosed. The intestine was more or less normal in appearance, but was friable. There was no gangrene or excessive congestion. The

omentum was carefully freed from the intestine, but in separating the two, two small holes were made in the latter. These were sewn up. The necrosed part of the omentum was resected and the intestine was returned to the abdomen. Radical cure by Bassini's method was carried out. On coming out of the anaesthesia, the patient seemed to be doing well. Glucose saline was given per rectum. Next morning the abdomen was somewhat distended. No gas or faeces were passed. Turpentine enema and pituitrin injections did not help, and the patient died on the third day. It seemed remarkable that an irreducible hernia should cause such slight signs on the intestine. The only explanation is that the blockage could not have been complete. If the patient had been brought to the hospital earlier, he might have been saved.

h. *Ununited fracture of the left humerus.* Suen, aged 24, fell on his left arm four months ago, and the fragments never united, because the lower portion was pulled forwards probably due to the weight of the fore-arm, and improper treatment. On operation, an incision was made on the outer aspect of the arm, and after the bones were cleared and freshened, they were joined together with a silver wire. Healing was by first intention, and the fracture became united. The arm could not be fully flexed owing to the atrophy of the biceps muscle. There was evidence of injury to the musculo-cutaneous nerve at the time of injury. With massage and exercise he gained back quite a useful arm.

i. *A case of tetanus after operation.* Mrs. Chang, aged 20, was admitted for T. B. knee. There were three sinuses and pus tracked up to the middle of the thigh. She was pale but not too thin. Amputation at the junction of the upper and middle thirds was done under chloroform and ether. At the time, there was much bleeding and cozing of tre wound afterwards. The next day, the patient removed all the dressings, and the wound had to be dressed again. After five days, the wound did not heal well, and gaped open, showing pale granulations. On the ninth day after operation, the patient complained of stiffness of the jaw. There was also a little fever. 20 c.c. anti-tetanus serum was injected subcutaneously. The next day convulsions commenced. Again 20 c.c. serum was given. Convulsions every five minutes. On the fourth day, the same treatment. On the fifth day, 20 c.c. intraspinally. On the sixth day, patient began to feel better, and the convulsions were much less frequent. Gradually she got well, and the wound also started to heal. In addition, hydrogen peroxide was applied to the wound several times daily and chloretone in 10 grain doses given three times daily.

j. *A case of meningitis serosa.* A young married woman of 23, Mrs. Li, was admitted in an unconscious condition. The history was that she had been suffering from headache off and on

for the last two months. Then on the journey from Pinchow to Harbin, she became unconscious and was brought to the hospital. On examination, there was no fever, no rigidity of the neck, but Kernig's sign was present. Reflexes were present and equal. Sphincters were not in control. Urine examination proved negative. Wassermann reaction was negative. Lumbar puncture was performed. The cerebro-spinal fluid was under pressure, clear, no leucocytes or bacteria. As a therapeutic measure, lumbar puncture was performed twice in four days. On the fifth day, she recovered consciousness.

k. *Vesical calculus.* A man, Kuo, aged 51, came for treatment for difficult micturition. The urine contained pus. On examination, a stone was found to be in the bladder. Supra-pubic operation was performed under spinal anaesthesia and two large phosphatic stones were extracted from the bladder. A rubber catheter was tied in, and the supra-pubic wound was closed without drainage. The patient recovered very well. But the next day, he showed paralytic abdominal distention. Pituitrin injections and turpentine enemas were given, but with no result. Intravenous injection of hypertonic saline given with a view of relieving the adynamic ileus, but no effect was observed, and the patient died on the fifth day after operation.

J. W. H. CHUN,
(*Senior Med. Off., Harbin*).

SHIH CHI LIANG,
(*Resident Med. Off., Harbin*).

EIGHTH ANNUAL REPORT, NEWCHWANG QUARANTINE HOSPITAL, 1927.

I beg to submit herewith the Eighth Annual Report of the Newchwang Quarantine Hospital from June 19th, 1926; to June 18th, 1927.

2. Medical Inspections were started on July 9, 1926 though actual quarantine against Shanghai for "Cholera Infected" was not declared until July 19, 1928.

Canton was declared "Cholera Infected" on July 16, 1926. No cholera cases nor passengers were detained from Canton.

Although no actual quarantine was declared against Tientsin and Lungkow yet we started Medical Inspections on steamers from those ports from September 1st, lasting to September 30, 1926. One cholera case was detained from Tientsin and recovered.

The port of Tsingtao was declared "Cholera Infected" from September 13, to November 10, 1926.

3. Quarantine against all steamers was discontinued as from November 10, 1926. The following are the list of passengers and crews we inspected as from July 9, 1926, to November 10, 1926.

Chinese Passengers	20,488	male.
Chinese Passengers	1,058	female.
Foreign Passengers	60	male.
Foreign Passengers	14	female.
Foreign & Chinese crews	8,251	
Total	29,871	

4. The following are the list of steamers we inspected during this period.

Nationality steamer	Number.
Chinese	63
British	46
Japanese	25
Norway	4
Germany	2
Dutch	1
Total	141

The following are the number of junks and passengers we inspected during this period.

No. of junks	208
Passengers and Crews	413

Three junks were detained one week each because there was a death from cholera on each junk.

5. Cholera in the city of Newchwang—Owing to the prevalence of cholera epidemic in the city of Newchwang and the inability of the local Isolation Hospital to care for them, we had opened our isolation wards for the local authority. During the period of August 19, to

October 10, 1926 we have cared for the following number of patients *gratis*.

No. of Suspects 30
No. of Cholera 36
No. of Deaths ..... 5 from cholera

Total No. received into our hospital is 66, including 5 deaths.

There are 22 deaths from cholera in the local Isolation Hospital.

6. During the year 1926-1927, we had treated 6,034 out-patients including 1,097 female cases. The number of Inpatients was surgical 35, medical 30, cholera suspects 30, true cholera 36.

The following statistics are since the opening of the hospital on July 10th, 1920.

(a) *Dates.*	*Outpatients treated*	*Inpatients treated*				*Total*
1920-1	3225	Med.	21	Surg.	22	43
1921-2	3367	,,	10	,,	—	10
1922-3	2919	,,	44	,,	21	65
1923-4	4412	,,	10	,,	18	28
1924-5	4343	,,	10	,,	42	52
1925-6	5812	,,	18	,,	44	62
1926-7	6034	,,	30	,,	35	65
1926-7	Cholera suspects 30, Cholera cases 36					66

(b) Infectious Diseases treated in same period :—

Names	1920-1	1921-2	1922-3	1923-4	1924-5	1925-6	1926-7
Cholera	—	—	—	—	—	—	36
Erysipelas	—	—	—	—	—	—	3
Diphtheria	1	—	—	—	—	—	—
Scarlet fever ...	2	—	—	—	—	—	1
Measles	—	—	—	—	—	—	1
Smallpox	1	—	—	—	—	—	—
Typhoid	2	—	—	1	—	—	—
Parotitis	—	—	—	—	—	—	29

(c) Percentage of three important diseases among outpatients were :

Names	1920-1	1921-2	1922-3	1923-4	1924-5	1925-6	1926-7
	Percentage %						
Gonorrhea	12	15	20	18	14	9	.06
Syphilis	12	21	20	10	8	6	4
Tuberculosis ...	16	12	10	9	8	6	3

7. The following figures regarding population are supplied by the police :

Chinese	103,603	
British	72	
Americans	2	
French	2	
Germany	14	
Holland	4	
Japanese	317	in Chinese city.
Japanese	2613	in South Manchurian Area.
Korean	230	in South Manchurian Area.
Korean	116	in Chinese city.

Russian	27		
Sweden	4		
Number births	693	Number of deaths.....	701

The number of Prostitutes are as follows :—

Class 1.	49
2.	105
3.	342
4.	157
5.	17
Japanese SMR area	61
Japanese in Chinese city	35
Total	766

8. Movements of Personnel are as follows :

Mr. F. W. Lyons replaced Mr. Y. Kurematsu as Commissioner of Customs as from September 8, 1926.

Mr. P. P. P. M. Kremer relieved Mr. F. W. Lyons as Commissioner of Customs as from June 1, 1927.

Mr. J. M. Nisbet took office as Harbourmaster as from 13th, December, 1926.

Mr. Liu Tung Pan who was acting Mayor since March 16, 1926, was promoted to the Mayorship as from February 2, 1927.

9. Up to date we have spent the following sums upon the hospital :

Original hospital blocks .....Mex.	$30,000
Mortuary	1,200
Store Room	276
New Quarantine (Detention) blocks 1924	40,900
Medical Examination block 1927	8,600
Total Mex.	$80,976

This year we have spent about Mex. $1,000 upon drugs and surgical dressings.

10. The first snow fall of the year was on October 28, 1926. The Liao river was closed to traffic from January 1, 1927, to March 19, 1927. The first steamer come in was the S.S. Yinkow Maru on March 24th, 1927.

In conclusion may I express my appreciation of the valuable services rendered by the local officials, especially during the anti-cholera campaign of the city of Newchwang from August 19, to October 10, 1926. Commissioner of Customs F. W. Lyons also took great interest in the building of our new Medical Examination block, by sending the Maritime Customs' supervising engineer Mr. F. Nightingale to inspect our workmanship on several occasions during the progress of the building, which will be finished about the first week in July, 1927. Lastly, I desire to express my appreciation to our staff for their faithful performance of duty.

YANG TING KWANG, M.D.,
Senior Medical Officer.

Newchwang, June 25, 1927.

NINTH ANNUAL REPORT, NEWCHWANG QUARANTINE HOSPITAL, 1928.

I beg to submit herewith the ninth Annual Report of the Newchwang Quarantine Hospital from June 20, 1927, to June 30, 1928.

During this period we have treated the following :—

7960 Outpatients including 1630 female cases.
38 Medical Inpatients.
32 Surgical Inpatients.

8030 Total from June 20, 1927, to June 30, 1928.

Our New Medical Examination Block: Construction of this new block began on April 16, 1927 and was completed on July 23, 1927, same costing under Mexican Dollars Eight thousand.

Medical Inspections were started on August 13, 1927, though actual quarantine against Shanghai for "Cholera Infected" was not declared until August 31, 1927. Quarantine against Shanghai was withdrawn on October 21, 1927.

A quartermaster from the S.S. Livingston was treated for "Colitis" and recovered. A dead corpse was found on board the S.S. Ichang, death being due to heart failure.

Medical Inspections on Tientsin steamers began on September 17, 1927, and withdrawn on October 2, 1927. No steamers or passengers were detained from Tientsin.

The following is a list of passengers and crews we inspected as from August 18, 1927, to October 21, 1927.

13,326	Chinese passengers.
45	Foreign passengers.
1,067	Foreign crews.
3.089	Chinese crews.
Total 17,527	

The following is a list of steamers we inspected during this period.

Nationality steamer	Number.
Chinese	37
Japanese	13
British	10
Norway	7
Holland	2
French	1
German	1
Total	71

Cholera in the City of Newchwang: We have taken care of about 55 male and 59 female cases with symptoms resembling "Cholera" from the city sent in by the local police authority. Out of these numbers 22 male and 9 female cases died from various stages of "Colitis". No Cholera vibrios were found in any of them. One true cholera in a woman of 50 years old recovered.

Black Smallpox in the City of Newchwang: A case of Black smallpox occurred in a boy of 16 years old at the "Yu Chang Chan". The said boy died on the afternoon of June 22, 1928. The two contacts were quarantined in our Isolation wards from June 22 to 24, 1928. The said room where the death of the boy occurred was fumigated and disinfected by our staff.

The following are statistics since the opening of the hospital on July 10, 1920.

(a) *Dates.*	*Outpatients treated.*	*Inpatients treated.*				*Total*
1920-1	3225	Med.	21	Surg.	22	43
1921-2	3367	,,	10	,,	—	10
1922-3	2919	,,	44	,,	21	65
1923-4	4412	,,	10	,,	18	28
1924-5	4343	,,	10	,,	42	52
1925-6	5812	,,	18	,,	44	62
1926-7	6034	,,	60	,,	35	95
1927-8	7960	,,	38	,,	32	70

(b) Infectious Diseases treated during same period:—

Names	1920-1	1921-2	1922-3	1923-4	1924-5	1925-6	1926-7	1927-8
Cholera ...	—	—	36	—	—	—	35	01
Erysipelas	—	—	—	—	—	—	03	08
Diphtheria	01	—	—	—	—	—	—	09
Scarlet Fever	02	—	—	—	—	—	01	—
Measles ...	—	—	—	—	—	—	01	01
Smallpox ...	01	—	—	—	—	—	—	01
Typhoid ..	02	—	—	01	—	—	—	—
Parotitis ...	—	—	—	—	—	—	29	05

(c) Percentage of three important diseases among outpatients:—

Names	1920-1	1921-2	1922-3	1923-4	1924-5	1925-6	1926-7	1927-8
	Percentage %							
Gonorrhea	12	15	20	18	14	9	.06	6
Syphilis	12	21	20	10	8	6	4	3
Tuberculosis	16	12	10	9	8	6	3	3

The following figures represent the population of different nationalities in the city of Newchwang.

Chinese	103,027	including	31,857	females.
British	54	,,	24	,,
American ...	06	,,	03	,,
French	02	,,	01	,,

German	10	,,	04	,,
Japanese	214	,,	99	,,
Korean	123	,,	65	,,
Russian	27	,,	16	,,
Japanese ...	2905	,,	1443	,, IN S.M.R. area.
Korean	403 in S. M. Railway area.			

The number of prostitutes are as follows :—

Chinese class 1.	83	
2.	105	
3.	386	
4.	201	
5.	023	
Japanese	37	in S. M. R. area.
Japanese	14	in Chinese city.
	849	total in Newchwang.

The number of Births and Deaths (not complete) are as follows :—
Births 579 males, 519 females.
Deaths 566 males, 436 females.

Movements of personel are as follows :—

Mr. R. L. Warren was in temporary charge as Commissioner of Customs from October 14, to November 10, 1927, when Mr. P. P. P. M. Kremer was transferred from here to Chinkiang.

Mr. C. N. Holwill relieved Mr. Warren as Commissioner of Customs as from November 10, 1927.

Mr. W. E. Clark relieved Mr. J. M. Nisbet as Harbourmaster as from May 9, 1928.

Mr. Yang Wen Hua relieved Mr. Liu Tung Pan as Mayor of Newchwang as from June 11, 1928.

I went to Tungliao on October 15, to investigate the plague condition there and returned on October 19, 1927. Found no other deaths or new outbreaks than the case reported by the Japanese authority. This case was being disposed of before my arrival.

This year we have spent about Mex. $1000 upon drugs and chemicals and surgical supplies. Besides Hongping Taels 3900 for a new hot water heating system which was installed on December 22. 1927. Same kept the hospital quite warm during last winter.

We have bought two bicycles for the dressers in their Public Health work, same costing Mex. $88.00 in all.

Am enclosing the reports of the Public Health campaign for Newchwang as from January to June 1928. Same are made out in Chinese.

On November 15, 1927, railway traffic from Tungliao to Tahushan connecting with the Peking Moukden line was opened. Direct traffic from Tungliao to Newchwang via the Peking Moukden line was opened as from Mrch 15, 1928, thus making the port of Newchwang qurte an important exporting centre.

The first snowfall of the year was on October 28, 1927. The Liao River was closed to traffic as from January to March 19, 1928. The first steamer come in was the S.S. Sagami Maru on March 22, 1928.

It is reported that over 600 children under 3 years died from Influenza in Tokio in the beginning of the year. Also that 1000 adult deaths from Influenza occurred during January and February, 1928. Cases of Scarlet Fever with 20% deaths occurred during January, 1928 in Dairen, and 8 cases of Small-pox with 4 deaths among Shantung emigrants during February in Dairen.

We have examined about 668 prostitutes on June 5, 1928, and 130 of them have not come forward for examination on account of having infections. Hereafter they will be examined monthly. Out of the number we found 17 with marks of morphine injections in their arms and bodies.

YANG TING KWANG, M.D.,

Newchwang, July 2, 1928. *Senior Med. Officer.*

ANNUAL REPORT OF ANTUNG QUARANTINE HOSPITAL, 1928.

I beg to forward you a general report of our Hospital for 1927-8.

Our work of last year was not very much, because our quarantine began as late as August 26. Last year our quarantine was instituted when the Customs Commissioner was notified by the American Consul to the effect that Shanghai was officially declared to be Cholera Infected. I do not know whether quarantine against other infected ports is similarly instituted in Newchwang or not. Only 12 steamers arriving from Shanghai and Tientsin, including 1003 passengers, were examined: no infectious diseases were found on board them. There was no case of Cholera reported in Antung last year.

In February of this year I had one month's leave to study the subject of Cholera and some laboratory work in the Peking Union Medical College. On my way back from Peking I visited some Japanese Hospitals in Dairen, including the S. M. R. Hospital, where I met Dr. Totani, who inquired if you were back yet. Upon my return from Peking, I felt I had a lot of work to do for the Hospital. I succeeded in asking the Board of Directors to convene together so as to discuss some questions regarding the developing of the Hospital, with the result that a sum of $1600 has been recently granted to set up (improve) our Laboratory, including the purchase of some ordinary drugs for our Dispensary. Our Laborator Apparatus, Chemicals and Drugs are being ordered, through the Customs Commissioner, from Peking, Shanghai and Japan.

Premises of the Hospital. The Hospital Buildings, as you know, occupy about 8 *mou* of land, and consist of 6 separate blocks, including the Ward and the Detention Camp. All these blocks have plenty sunlight, and with exception of the Detention Camp, are of very good brick construction. Although our Ward can hold only 12 beds, its capacity seems to be large enough to meet our present need. The reason is because our Hospital is so far away from the city that patients admitted to our Ward are generally patients from vessels. On the other hand, our Detention Camp is, to my opinion, altogether too small, and is too hot to accommodate any contacts in summer time. We have, however, decided to build an extra mat-shed above its tin roof this summer so as to make it useful. There is still sufficient empty space in the Hospital Compound for further expansion of both the Ward and the Detention Camp in case of need.

Our well, which was a laborious work and which cost several thousand dollars to build, is no more good for use. Repairs will be done to it presently. Unfortunately, the people did not take the trouble to think over how to get water before they started to build the Hospital on the top of a rocky hill.

The same mistake is found with the building of the Main Block, where all the 7 rooms on the ground floor (basement) are too damp for any use. For that reason, I was obliged to propose to the Board for the

building of one more block to contain at least three rooms for the use of Laboratory and Dispensary. The new block will be built next year, as has been approved.

The Staff Personnel. The present Medical Staff consists of one doctor, the Port Health Officer, who is living in the Hospital, one Dispenser and one Dresser. The Low Staff has 8 employes: namely, 2 guards, 2 servants, 1 boatman, 1 coolie, 1 letter carrier, and 1 cook.

In time of quarantine, during former years, it was the custom to borrow male nurses and sometimes one or more doctors from the Danish Hospital in Antung. We also borrowed police to guard the contacts from running away. In my opinion, it is more practical and advantageous to engage, if necessary, a trained laboratory technician to assist our work during quarantine time than to engage a special (extra) doctor or extra nurses. Because we have usually more cases to be diagnosed by laboratory methods than cases to be treated in our Ward. Furthermore, most of the local qualified doctors lack laboratory training; besides it is more difficult to get any doctor for temporary term at this end,—not to mention the fact that the pay of a doctor is higher than that of a technician.

Our Work. I have during the last several months grasped every opportunity to explain to the Customs Commissioner the importance and necessity of equipping our Hospital so as to fulfil our object. Dr. Yeo, as well as the Tao-Yin, is anxious to see our Hospital efficiently managed so far as we have a good budget and a handsome balance left. Now, as we are expecting a good bunch of apparatus for our Laboratory, we can do better work this year than the last.

But here we are at least facing a difficult problem and that is the distance of the Station from the city. In the first place, we cannot have electric fittings for our Laboratory out here, thus our work suffers a great handicap. Secondly, owing to the distance and inconvenience of traffic, patients in Antung cannot come to the Hospital for treatment and, therefore, our work is only limited to inspection of vessels in quarantine time; while in ordinary times, such as in spring and winter seasons, we have practically nothing to do. I believe that our Hospital can be best functioned by doing its share of work for the public in winter months as well. In order to attain this aim, it would be best to move the Medical Staff up to Antung as soon as navigation ceases so that not only the health of the city can be looked into, but the doctor and the assistants can also keep up their training and knowledge. I have suggested this to Mr. Fukumoto, the Co-Director and Treasurer, but I don't know whether it can be put into practice or not.

TANG TSUNG MIEN, M.D.

Resident M. O.

SUMMARY OF IN-PATIENT DISEASES (HARBIN HOSPITAL).

From October 1926 to August 1928.

	Harbin Hospital	*Newchwang Hospital*
Fracture and Dislocations. 骨骨折折及脫臼		
Fracture Thigh 大腿	1	0
,, Tibia 脛折骨	16	1
,, Spine 脊柱骨折	3	0
,, Clavical 鎖骨骨折	1	0
,, Humerus 上膊骨折	3	1
Dislocation Big toe 大趾脫臼	1	0
,, Hip 大腿關節脫臼	0	1
,, Shoulder 肩關節脫臼	1	0
Injuries.		
Sprain 扭傷	1	3
Contusions 挫傷	4	0
Frost bite 凍傷	4	0
Gun Shot 槍彈傷	43	6
Stabs and Wounds 刺及創傷	34	8
Scalds 燙傷	4	0
Burn 火傷	8	1
Diseases of Genito-Urinary System. 生殖及泌尿器病		
Nephritis 腎炎	3	0
Phimosis 包莖	0	1
Stricture urethra 尿管狹窄	2	0
Bubo 橫痃	17	0
Gonorrhoea 淋病	6	6
Orchitis 睾丸炎	5	0
Chancre 下疳	1	0
Obliteration vagina 膣口閉塞	1	0
Vesical Calculus 膀胱結石	2	0
Cystitis 膀炎	3	1
Prostatitis 攝腺炎	1	0
Perineal fistula 會陰瘻管	1	0
Incontinence urine 遺尿	1	0
Rupture urethra 尿道破裂	1	0
Diseases of Alimentary Canal. 消化管病		
Dysentery 赤痢	15	1
Typhoid 傷寒症	17	0
T.B. Peritonitis 腹膜結核	4	0
Hernia 小腸氣	3	0
Piles 痔核	12	2
Fistula in ano 痔漏	37	5
Constipation 便秘	3	4
Stomatitis 口腔炎	2	0

Gastritis 胃炎	1	0
Dyspepsia 消化不良	15	4
Appendicitis 盲腸炎	1	0
Gastroenteritis 胃腹炎	9	3
Diarrhoea 瀉洩	3	4
Fissure anus 痔瘺	1	0
Condyloma 梅毒疣肉	1	0
Prolapse rectum 直腸脫	1	0
Ascites 腹水	3	3
Colitis 腸炎	0	1
Jaundice 黃胆症	3	0
	Harbin Hospital	*Newchwang Hospital*
Diseases of Skin. 皮膚病		
Lupus 皮膚結核	1	1
Psoriasis 白癬	1	0
Eczema 濕疹	6	4
Scabies 疥癬	1	0
Ulcer 潰瘍	1	0
Diseases of Women. 婦科病		
Dysmenorrhoea 月經痛	1	0
Endometritis 子宮內膜炎	1	2
Eclampsia 產褥症	1	0
Abortion 小產	2	0
Puerperal fever 產褥熱	0	1
Circulatory and Respiratory System. 循環及呼吸系病		
Pneumonia 肺炎	5	8
Mitral 僧帽瓣症	12	5
Phthisis 肺結核	25	7
Pleurisy 肺膜炎	8	0
Bronchitis 氣管支炎	9	1
Phlebitis 靜脉炎	1	0
T.B. Adenitis 結核腺腫	11	3
Apoplexy 腦率中	2	0
Asthma 喘息	3	0
Endocarditis 心囊炎	1	0
Empyema 肺氣腫	2	0
Tumours. 瘤		
Granuloma 芽瘤	1	0
Fibroma 纖維瘤	1	0
Sarcoma 肉腫	7	0
Carcinoma 癌腫	7	0
Papilloma 刺瘤	3	0
Cyst 囊腫	3	3
Keloid 瘢瘤	2	0
Sebaceous cyst 皮脂腺腫	1	0

Gumma 護膜腫	1	0
Epithelioma 上皮腫	1	0
Venereal Wart 花柳腫	1	1
Lipoma 脂肪瘤	4	0
Diseases of Nervous System. 神經系病		
Paraplegia 腦梅毒	5	3
Meningitis 顱腦衣炎	4	0
Tabes Dorsalis 脊髓癆	2	0
Neurasthenia 神經衰弱	1	1
Convulsion 抽搐	3	0
Sciatica 坐骨神經痛	1	0
Facial paralysis 顏面神經痲痺	2	0
General Paralysis Insane 全痲痺狂	1	0
Epilepsy 癲癎	3	1
Dementia 癡症	2	0

	Harbin Hospital	*Newchwang Hospital*
Septic Cases. 化膿症		
Abscess 膿瘍	30	8
Ulcer 潰瘍	9	0
Gangrene 壞疽	5	0
Boil 癤	1	0
Mastitis 乳腺部炎	1	0
Carbuncle 癰	4	0
Erysipelas 丹毒	1	0
Cellulitis 蜂窩炎	8	0
Diseases of Eye. 眼病		
Pterygium 胬翳	1	0
Entropion 瞼捲內	1	0
Trachoma 胬粒炎	3	4
Pannus 角膜派勞斯	1	1
Conjunctivitis 胬炎	2	0
Traumatic Cataract 白內障	1	0
Leucoma 白翳	1	0
Diseases of Bones, Muscle and Joints 骨節及關節病		
Rheumatism 寒濕節痛	13	0
T. B. Bone 骨結核	41	1
T. B. Joint 關節結核	13	3
Necrosis 骨死	2	0
Periostitis 骨膜炎	1	0
Gon. Arthritis 淋症節炎	3	1
T. B. Spine 脊髓結核	0	1
Arthritis 關節炎	0	2
Fevers. 熱病		
Influenza 流行性感冒	4	0
Scarlet fever 猩紅熱	2	2
Acute Rheumatism 急性僂痲質斯	2	0

Measles 痲疹	1	0
Typhus 發疹傷寒	2	0
Catarrhal fever 加答兒熱	1	0
Diphtheria 白喉	0	1
Small pox 天花	1	1
Various. 雜症		
Opium Habit 中鴉片毒	3	0
Undiagnosed 未診斷的	4	6
Maternity 生產	13	10
Syphilis 梅毒	7	3
Morphine Habit 中嗎啡毒	2	4
Polydactyly 孖指	1	0
Poison 中毒	2	0
Ankylostomiasis 腹虫症	1	0
Beri-beri 脚氣	1	4
Tetanus 破傷風	1	0
Hare-lip 兎唇	2	0
Splenomegaly 脾臟腫大	2	0
Nasal catarrh 鼻炎	1	0
Septicaemia 血中毒	2	0
Thread worms 線虫	2	0
Heat-stroke 感暑症	0	1
Cholera 霍亂	0	12
Cholera contact 霍亂接觸者	0	1
	648	158

1926 TO AUGUST 1928, HARBIN HOSPITAL.

LIST OF OPERATIONS BETWEEN SEPTEMBER,

Amputations. 肢截斷術

Toe 脚趾	4
Fingers 手指	2
Leg 小腿	11
Thigh 大腿	11
Foot 足	18
Arm 臂	1
Breast 乳腺	5
Fore-arm 前膊	2

Bones and Joints. 骨及關節

Resection Metatarsus 蹠骨切除術	3
Osteo-myelitis tibia 大腿骨膜炎	2
Plating tibia 大腿骨釘術	3
Reset old fracture 整舊骨折	1
Remove plate 除釘術	1
Excision of head of humerus 上膊骨切斷術	1
Plating humerus 上膊骨釘術	1
Draining sinus 廔管排膿術	1
Comp fracture Tib and Fib 脛腓骨複雜骨折	3
Scraping Necrosed bone 括腐骨	8
Resection rib 脇骨切除	1

Genito-urinary. 生殖器及尿道

Stricture urethra 尿道狹窄	1
Castration 割去睾丸	1
Vesical Calculus 膀胱石	1
Phimosis 包莖術	1
Ruptured urethra 尿道破裂	2

Alimentary Canal and Abdomen. 消化道及腹

Piles 痔核	10
Fistula in ano 痔瘻	31
Exploratory laporatomy 開腹術	4
Strangulated hernia 拑頓小腸氣	3
Hernia 小腸氣	1
Talma-Morison operation 摩爾遜氏手術	1

Eye. 眼

Scraping Trachoma 括胖粒眼	4
Iridectomy 虹彩切除	2
Pterygium 胖翳	1
Symblepharon 瞼球相粘	1
Entropion 眼內捲	1

Plastic. 畸形術

Obliterated vagina 膣閉塞	1
Hare-lip 兎唇	2
Skin-grafting 植皮術	2
Supernumerary digit 巨指	1
On cheek 頰部	1

Various. 雜類

Remove bullet 除彈術	1
Needle in knee 針在膝內	1
Adenoids 腺腫	2
Transverse Presentation 橫產	1
Ligature Brachial artery 上臂動脉結紮	1
Ligature femoral artery 大腿動脉結紮	1
Extract bullet 取彈	2
	210

Skin, Fascia, Tendon. 皮膚筋鞘靱帶	
T. B. Gland 腺結核	4
Bubo 橫痃	9
Cellulitis 蜂窩織炎	4
Abscess 膿瘍	11
Scraping ulcer 括潰瘍	1
Carbuncle 癰	1
Tumours. 瘤	
Cyst 嚢腫	2
Sebaceous cyst 皮脂腫	1
Sarcoma 肉腫	3
Ganglion 坼死	1
Keloid 瘢瘤	3
Lipoma 脂肪瘤	3
Polypus 贅疣肉	1
Fibroma 纖維腫	2
Carcinoma 小腸氣	1
Papilloma 癌腫	1
Chondroma 綠腫	1
Venereal wart 梅毒性疣肉	1

SUMMARY OF OUT-PATIENTS TREATED AT MANCHURIAN PLAGUE PREVENTION SERVICE HOSPITALS FROM OCTOBER 1926 TO AUGUST 1928.

	Harbin Hospital.				Taheiho Hospital.				Sansing Hospital				Manchouli Hospital				Lahasusu Hospital				Newchwang Hospital				Hailar Hospital			
	1926.	1927.	1928.	Total.	1926.	1927.	1928.	Total.	1926.	1927.	1928.	Total.	1926.	1927.	1928.	Total.	1926.	1927.	1928.	Total.	1926.	1927.	1928.	Total.	1926.	1927.	1928.	Total.
I. SPECIFIC INFECTIOUS DISEASES 特別傳染病																												
(a.) BACTERIAL DISEASES. 細菌病																												
1. Typhoid fever 傷寒熱症	24	23	9	56	0	7	11	18	0	0	0	0	0	1	2	3	0	0	0	0	0	0	0	0	13	22	0	35
2. Erysipelas 丹毒	3	30	11	44	0	2	2	4	0	0	0	0	0	2	0	2	0	0	0	0	2	6	4	12	12	40	13	65
3. Diphtheria 白喉症	4	0	0	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	6	2	8	1	0	0	1
4. Pneumonia 肺炎	18	20	6	44	2	20	13	35	0	0	0	0	1	16	0	17	0	0	0	0	0	0	0	0	9	35	12	56
5. Influenza 流行性感冒	72	213	50	335	0	6	33	39	0	0	0	0	1	12	77	90	4	17	22	43	0	0	0	0	38	178	137	354
6. Whooping cough 白日咳	5	16	15	36	0	2	1	3	6	36	34	76	0	11	0	11	1	2	0	3	2	2	0	4	0	69	1	70
7. Gonococcus infections 淋毒傳染症	135	127	63	325	19	68	22	109	0	21	53	74	8	99	46	153	40	40	42	122	11	75	79	165	155	328	168	651
8. Dysentery 赤痢症	37	129	89	255	5	32	36	73	0	18	10	28	1	18	22	41	6	8	8	22	1	5	5	11	5	81	48	134
9. Cholera 霍亂症	0	2	0	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	4
10. Plague 鼠疫症	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
11. Tetanus 痙傷風	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	3	0	3	0	0	0	0	0	1	1	2
12 Leprosy 癩瘋	0	2	4	6	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
13. Tuberculosis 肺結核症	171	113	72	356	85	226	123	434	18	138	100	256	26	64	25	115	18	100	64	182	47	225	116	388	52	235	101	388
(b.) NON-BACTERIAL FUNGUS INFECTIONS. 非細菌傳染病													2	0	0	2												
(c.) PROTOZOAN INFECTIONS. 原生動物傳染症																												
1. Malaria 瘧症	7	8	6	21	5	55	60	120	5	0	12	17	0	2	11	13	1	24	19	44	1	10	2	13	11	87	28	126
2. Relapsing fever 回歸熱症	27	0	0	27	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1
3. Syphilis 梅毒	260	280	101	641	47	304	127	478	10	71	102	183	24	192	121	337	22	68	39	129	52	285	86	423	157	492	213	862
4. Yellow fever 黃熱症	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
(d.) METAZOAN DISEASES. 原蟲症																												
1. Intestinal Cestodes, Tapeworms 觸蟲類 帶蟲類	13	18	12	43	0	16	6	22	0	4	0	4	2	6	0	8	0	0	0	0	0	0	0	0	3	27	6	36
2. Diseases caused by Nematodes 線蟲類	3	30	20	53	0	2	5	7	4	0	0	4	0	0	0	0	0	0	0	0	2	16	5	23	6	50	18	74
3. Parasitic Insects 寄生蟲	30	19	11	60	0	0	5	5	0	6	0	6	0	1	42	43	0	4	0	4	23	151	97	271	42	270	141	453
(e.) INFECTIOUS DISEASES OF UNKOWN ETIOLOGY																												
1. Small pox 天花 未知病原之傳染病	3	0	6	9	0	9	0	9	0	0	6	6	0	0	0	0	0	0	0	0	0	1	0	1	0	4	0	4
2. Chicken-pox 水痘	23	6	9	38	0	0	3	3	0	0	0	0	0	0	0	0	0	1	0	1	0	0	0	0	1	17	0	18
3. Measles 麻疹	12	39	19	70	0	2	17	19	0	13	49	62	0	2	0	2	0	0	0	0	0	1	0	1	0	4	4	8
4. Scarlet fever 猩紅熱	6	4	4	14	0	0	1	1	0	0	0	0	0	1	0	1	0	0	3	3	0	2	1	3	1	43	30	74
5. Epidemic Parotitis (Mumps) 流行性耳下腺炎症	3	16	19	38	1	4	3	8	0	0	0	0	0	1	0	1	0	0	1	1	0	26	8	34	10	72	9	91
6. Typhus 發疹傷寒	0	1	1	2	0	1	1	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	22	11	34
7. Rabies 狂犬症	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
8. Rhrumatic fever 傻麻質斯熱症	24	0	0	24	1	5	6	12	0	0	0	0	6	30	0	36	0	0	0	0	0	1	0	1	41	221	104	366
9. Acute Tonsillitis 急性扁桃腺炎	71	115	54	240	3	19	21	43	0	0	0	0	0	6	2	8	0	0	0	0	9	28	18	55	21	105	46	172
10. Acute Catarrhal fever 急性加答兒熱症	17	0	0	17	0	0	1	1	0	0	0	0	0	8	0	8	0	0	0	0	0	0	1	1	1	5	2	8
II. INTOXICATIONS. 中毒																												
a. Alcoholism 酒精中毒	16	0	0	16	0	1	0	1	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0	2	0	2
b. Morphia Habit 嗎啡中毒	46	15	2	63	1	4	8	13	0	0	0	0	0	0	0	0	0	0	0	0	0	13	11	24	62	30	2	94
c. Lead poisoning 鴉片中毒	0	5	12	17	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	0	4	0	1	27	28
d. Arsenical poisoning 砒霜中毒	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	6	7	13	0	0	0	0
e. Food poisoning 食物中毒	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	2	0	0	0	0
f. Beri-beri 脚氣	0	0	0	0	0	0	0	0	0	75	24	99	0	12	0	12	0	0	0	0	0	0	5	5	0	1	0	1
III. DISEASES OF METABOLISM. 新陳代謝病																												
a. Gout 痛風症	5	2	1	8	0	10	0	10	0	7	0	7	0	0	0	0	0	0	0	0	0	0	0	0	17	139	90	246
b. Diabetes 糖尿症	7	0	1	8	0	0	0	0	0	6	0	6	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	2
c. Rickets and Scurvy 軟骨及血枯症	0	0	1	1	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	7	0	7
d. Rheumatism 傻麻質斯症	84	425	280	789	16	87	62	165	81	334	231	646	18	54	8	80	4	53	51	108	0	12	10	22	29	195	63	287
IV. DISEASES OF THE DIGESTIVE SYSTEM 消化系病	150	801	609	1560	171	519	360	1050	166	369	280	755	43	201	102	346	73	107	92	272	97	505	266	868	104	458	195	757
V. " " " RESPIRATORY SYSTEM 呼吸系病	256	862	495	1613	82	281	135	498	7	36	30	73	44	180	177	401	22	88	55	165	49	208	161	418	114	430	197	741
VI. " " " GENITO-URINARY SYSTEM 泌尿生殖器病	132	86	38	256	10	63	17	90	0	0	0	0	0	60	0	60	4	22	6	32	13	121	24	158	20	158	88	266
VII. " " " THE BLOOD 血液病	31	34	14	79	4	6	7	17	5	11	0	16	0	6	0	6	0	0	0	0	0	0	0	0	25	143	73	241
VIII. " " " CIRCULATORY SYSTEM 循環系病	24	57	27	108	3	64	39	106	0	4	0	4	0	24	4	28	3	19	2	24	0	4	0	4	22	142	73	237
IX. " " " DUCTLESS GLANDS 無管腺病	0	30	14	44	13	44	8	65	0	0	0	0	0	6	0	6	30	37	10	77	0	0	0	0	35	181	119	335
X. " " " NERVOUS SYSTEM 神經系病	79	44	14	137	22	105	57	184	19	48	33	100	0	82	2	84	0	3	1	4	11	53	22	86	33	218	95	346
XI. " " " LOCOMOTOR SYSTEM 運動系病	0	21	20	41	0	8	4	12	0	9	0	9	0	11	0	11	0	91	60	151	0	0	0	0	18	78	56	152
XII. " " " EYE 眼病	235	697	574	1506	70	430	364	864	54	333	246	633	19	123	48	190	79	249	166	494	77	870	405	1352	72	358	191	621
XIII. " " " SKIN 皮膚病	226	856	676	1758	120	624	543	1287	113	435	261	809	26	114	34	174	21	64	49	134	54	353	59	466	136	545	206	887
XIV. " " " NOSE, THROAT AND EAR 耳鼻咽喉 病	151	197	99	447	37	167	136	340	13	128	140	281	20	105	5	130	3	50	21	74	125	610	533	1268	90	478	276	844
XV. " " " WOMEN 婦人病	88	161	75	324	25	32	45	102	35	105	116	256	4	24	6	34	74	334	228	636	6	77	27	110	46	298	106	450
XVI. SURGICAL CASES 外科	750	4818	2976	8544	272	1546	953	2771	473	1746	1018	3237	133	744	710	1587	0	30	24	54	598	3884	2258	6740	54	388	180	622
XVII. VACCINATION 種痘	33	249	335	617	0	273	217	490	0	26	29	55	0	6	0	6	0	0	0	0	0	87	51	138	0	319	337	656
XVIII. MIDWIFERY 產科	6	2	0	8	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	8	9	0	6	5	11
Total.....	3287	10575	6844	20706	1014	5045	3453	9512	949	3979	2774	7702	379	2224	1444	4047	405	1414	963	2782	1180	7650	4271	13101	1458	6986	3476	11920