Report of veterinary surgeon J. H. Steel, A. V. D., on his investigation into an obscure and fatal disease among transport mules in British Burma, which he found to be a fever of relapsing type, and probably identical with the disorder first described by Dr. Griffith Evans under the name "Surra", in a report (herewith reprinted) published by the Punjab Government, Military Department, No. 439-4467, of 3rd. December 1880-vide the Veterinary Journal (London), 1881-1882 / [By Steel, J. H.].

With Dr. Evans' compliments.

Woolwich
25 June 86

Report of Veterinary Surgeon J. H. STEEL, A. V. D., on his INVESTIGATION INTO AN OBSCURE AND FATAL DISEASE AMONG TRANSPORT MULES IN BRITISH BURMA, which he found to be a Fever of Relapsing Type, and probably identical with the disorder first described by Dr. Griffith Evans under the name "Surra", in a Report (herewith reprinted) published by the Punjab Government, Military Department, No. 439-4467, of 3rd December 1880— *vide* the Veterinary Journal (London), 1881-82.

INDIA C

vide Page 5 which had better be read for as introductory

CONTENTS OF REPORT.

Part III.—THE DISEASE (*pp.* 37—47).

PART IV.—THE PARASITE (*pp.* 47—51).

POSTSCRIPTUM (*pp.* 51—58).

ADDENDUM (*pp.* 59—89).

LIST OF PLATES, CHARTS, DIAGRAMS, &c., ILLUSTRATING THIS REPORT.

CONTENTS OF Dr. GRIFFITH EVANS' REPORT ON SURRA AT DERA ISMAEL KHAN.

REPORT ON AN INVESTIGATION INTO A DISEASE AMONG TRANSPORT MULES.

PART I.—HISTORY OF THE DISORDER.

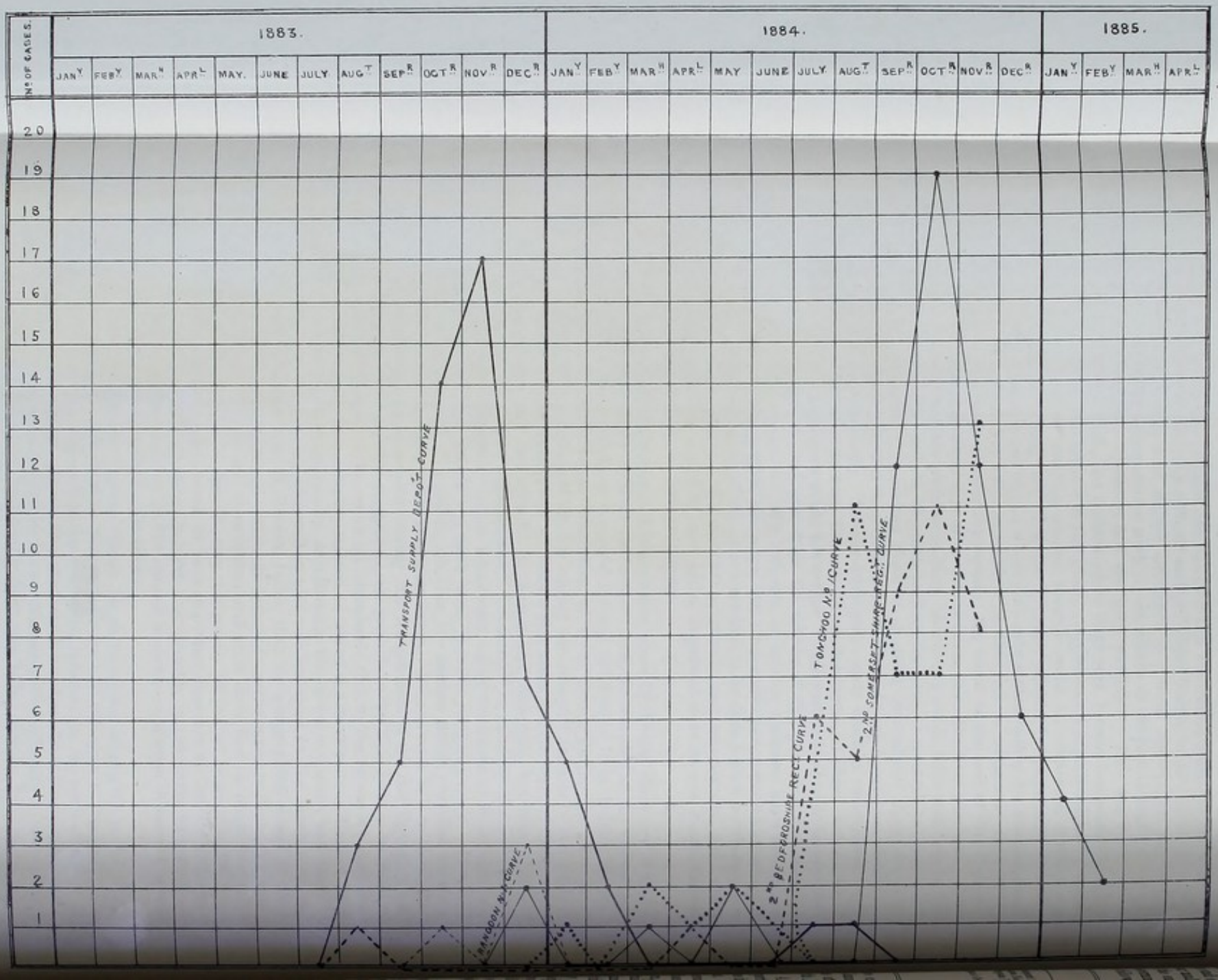
In accordance with Inspecting Veterinary Surgeon's Office No. 1208, dated Ootacamund, 24th October 1884, and under G.O.C.C., No. 547, dated 23rd idem, I proceeded to Rangoon from Bangalore as expeditiously as possible (*viâ* Calcutta). I commenced my investigations on 17th November and continued them until 20th December, when I left for Tonghoo (under D.O., No. 5, dated Rangoon, 17th December 1884) at which place I arrived on 27th idem, but left again on 29th on receipt of an urgent telegram from the Inspecting Veterinary Surgeon requesting my return to meet him in Rangoon as soon as possible. I arrived at Rangoon again on 3rd January 1885 and remained there until 17th February, when I left for Thayetmyo (under D.O., No. 3, dated Rangoon, 17th February 1885). I arrived again at Rangoon on 23rd February and occupied from 3rd March to 7th idem in visiting Moulmein on duty. Under D.O., No. 2, dated Rangoon, 10th March 1885, I left Rangoon for Madras on 11th idem. I have periodically reported to the Inspecting Veterinary Surgeon my proceedings in the investigation, with a view to keeping him *au courant* with the progress of the inquiry. I informed him that, after organizing a temporary pathological station at Rangoon with the medicines, appliances, &c., obtained from there and Madras and ascertaining the general condition of the animals belonging to Government in the station and their management, I proceeded to inquire into the fatality. I soon was enabled to arrive at the conclusion that there was *one* disease of an imperfectly-ascertained nature to which the rise of fatality above the average was to be attributed, in Rangoon at any rate, but also (as I subsequently found to be actually the case) probably in Tonghoo. I ascertained all the cases of affected animals, collected them into a sick lines (as improvised previously from one of the stables of the Somersetshire Regiment), and carefully studied the *clinical features of the disorder*. It was evidently not one of the ordinary diseases of the horse as recognized by the veterinary profession, and among the unusual diseases described by authors the only one bearing any analogy to it was Surra as described by Dr. Evans, A.V.D., from his investigation of it in the Trans-Indus territory (*vide* Veterinary Journal, 1881 and 1882), and yet there seemed such great and important differences between the disease observed by Dr. Evans and the one under inquiry by me that I hesitated much in accepting their identity. With the progress of the research, however, I found that the principal differences began to disappear, and, finally, microscopical observations enabled me to conclude that the outbreak is one of Surra, presenting some special features such as may be ascribed to range in species and to geographical, climatic, and other influences. Careful and sustained *thermometric researches* have enabled me to supplement Dr. Evans' views as to the nature of the disease by showing that it is a true relapsing fever and probably resembles closely, although it is not absolutely identical with, relapsing fever of man. *Experimental research* has enabled me to confirm most of his conclusions on the communicability of the disease by ingestion and inoculation to the dog, horse, and mule; experiments in this line on other species have been tried. *Post-mortem investigations* have thrown much light on the pathology of this disorder, especially in its relations to the frequent occurrence of ulceration of the stomach, which lesion was originally considered to be *the* disease, but has now been proved of subordinate importance. *Microscopical examination*, with the higher powers, has enabled me to study the parasites which prevail in the blood in this relapsing fever; my conclusions agree in the main with those already placed on record, but there are some minor points of difference. The causes of the disorder have been carefully investigated by me, and I have in this matter obtained information of value from the Government meteorological and statistical returns. The reports of Inspecting Veterinary Surgeon Shaw, I.V.D., and Veterinary Surgeon (First-class) Frost, A.V.D., have given me valuable information. I may remark that the complete absence of veterinary records in the transport has lessened

the amount of information available as a basis for this report and rendered the work of collection of evidence much more difficult and prolonged than it otherwise would have been. The complete absence of skilled assistance in clinical observation and record threw a large amount of manipulative work on me which might just as well have been performed by a Veterinary Assistant had one been available. This was only felt at first, for, later, Corporal Hill, 2nd Somersetshire Regiment, became competent to make intelligent observations and records of the pulse, respirations, temperature, and general symptoms of the cases, so that the clinical records could be continued during my absence at Tonghoo; I may add that the average salootri is quite unfit for any such duty. The disease is not one in which elaborate *section-cutting* for microscopical investigation is required, but such researches in this line as suggested themselves have been carried out with satisfactory results; the blood is the seat of the disorder and can best be examined while it is alive and as taken from the living animal. The parasites which occur in it are peculiar in their life-history and in their relations to the phenomena of the disease, on both of which matters the present research has increased our knowledge. A serious stumbling block to progress in study of their developmental changes was the fact that they become invisible on drying up and practically so on cessation of their movements. I made attempts to *stain* them in various ways and have obtained excellent results from the use of aniline dyes. *Culture* was a line of research for which my appliances would not suffice. This matter is of great importance as bearing on the entry into and exit from the system of the parasites, but must be worked out by future observers. My *attempts at curative treatment* have not given any important results of a positive character, but I have obtained some information as to prevention. Although much of importance remains to be done before we thoroughly understand how to grapple with this disorder practically and successfully, yet I trust the present inquiry will be found to have added important facts to our knowledge of its clinical features, causes, and true pathology.

2. History of the Disorder.—I believe that, since Dr. Evans' discovery of Surra in the Trans-Indus territory in 1880, no further scientific mention of the disease has been made until it has now been my good fortune to observe it in British Burma. Dr. Evans alludes to the probable occurrence of it in Persia during the autumn of 1876 and considers it as a cause of fatality among our transport animals in the Afghan campaigns. I have read descriptions of cases which seem to have been of this nature occurring enzootically and epizootically in Assam and Australia, but they have not been dealt with by competent veterinarians. I can recall to mind two cases of obscure disorder, considered "typhoid," which I had seen under treatment at Woolwich and Secunderabad respectively, which may have been sporadic relapsing fever. It would be worth while to ascertain, if possible, whether the *anæmia* which prevailed recently among British horses in Egypt was relapsing fever. I have no details to hand, but believe the disease which prevailed among the troop-horses in the last Egyptian war was very fatal, and that possibly relapsing fever prevails in the Nile delta as in those of the Irrawaddi and Indus. It is not at all improbable that in the future we shall find "Surra" taking a place in veterinary science as one of the most serious diseases with which we have to contend, especially as affecting the horse and mule, and there is reason to think, from its being observed in places so far apart as the north-west and south-east frontier countries of British India, that its geographical range is very extended and that it may occur on various geological formations and under very diverse climatic conditions. Dr. Evans' observations and mine show that its range in species is extensive, and it is possible that it may ultimately be proved communicable from quadrupeds to man and so be found to have important bearings on human medicine; from a study of it in animals as much light may possibly in the future be thrown on general pathology as has resulted in the case of anthrax—a disease in some respects allied to that under consideration (*vide infra*).

3. In 1883 the British Burma disease began to appear among the mules of the supply depôt and of the 5th Madras Native Infantry half transport at Rangoon; one case (possibly two) also occurred in the European Infantry at the same station. It was most severe by far among the depôt mules which had been for a long time exposed in the open during the monsoon; there is a certain amount of difficulty in determining when the earliest case occurred, the earlier diagnoses being obscure, but the disorder, having caused some deaths in August and September, proved extremely fatal in October

CHART ILLUSTRATING THE DISTRIBUTION OF CASES OF RELAPSING FEVER IN TIME, PLACE, & CORPS.



and November, less so in December and January, and two cases succumbed to it at the end of February, when the outbreak ceased. Of the Native Infantry half transport, four mules (one hired out to an officer), and of the European Infantry, two mules died of the enzootic this season (1883-84). The probable losses from relapsing fever of Government transport animals at Rangoon during the year 1883-84 may be tabulated as follows:—

Corps, &c.	Mules.	Ponies.	Remarks.
Transport Supply Depôt	42	11	Number of cases among ponies perhaps exaggerated.
Madras Native Infantry	4	..	Inclusive of one mule hired out to an officer.
European Infantry	5	..	One recorded as "ulceration of stomach;" four doubtful.
Queen's Own Sappers and Miners	..	..	There have been no fatalities among these from doubtful causes.
Total ..	51	11	62 animals at Rs. 150 each; total approximate value Rs. 9,300.

Two cases seem to have occurred this season at Tonghoo; one in August and one in January in the European and Native Infantry regiments respectively. This outbreak was at an end when Inspecting Veterinary Surgeon Shaw arrived to investigate it (19th April 1884).

4. In the current season the disease re-appeared, or rather again began to assume importance, for it cannot be said to have ever quite died out. Strange to say, this time it proved most severe in those lines in which it had previously been least prevalent. This is a noteworthy fact. Is it to be attributed to last year's outbreak having thoroughly "weeded" the depôt of animals capable of taking the disease or to the efficacy of measures of prevention and improved hygiene adopted? In so far as I can learn, the only change made in the latter respect was as concerns the water-supply, certain suspected tanks being carefully avoided, and we shall see that the evidence against those tanks is not strong. But it may be that the remaining depôt mules have not this year been exposed to the climatic conditions necessary to generate this disorder; they were not, as in 1883-84, exposed during the monsoon time, which is a very likely cause of the disease. Still we must not neglect this important evidence in support of the view of Veterinary Surgeon (First-class) Frost that the disease in 1883-84 was conveyed by the water. The supply of drinking water was altered as to its source in accordance with his suggestions, and there has been no *outbreak* in the depôt this year, although *sporadic* cases have occurred there.

5. The brunt of this year's outbreak has, at Rangoon, fallen on the 2nd Somersetshire Transport Lines: a doubtful case occurred in March; two deaths are recorded for May; in September twelve were fatal; in October the maximum (nineteen) was reached; twelve died in November and six in December; four succumbed in January 1885 and two in February. I left one affected animal alive on my departure from Rangoon. In all I had about twenty fatal cases at Rangoon (experimental inclusive). I missed a few opportunities of post-mortem examinations by absence at outstations. That the disease is due to the water of special tanks only is disproved by its prevalence at Tonghoo this year. I have absolute proof, from personal investigations, that the disease which has recently caused so many deaths there is the same as that of Rangoon, but of more acute character and even more fatal. My returns show that up to their date, *i.e.*, 25th December 1884, some fifty mules of the 2nd Bedfordshire half transport had died in the current official year, the majority probably from relapsing fever; of the 3rd Palamcottah Light Infantry, about fifty-four mules died during the same period. The supposed causes of death in these cases, as given in the returns and also in the post-mortem examination committee reports, lead me to believe that I may fairly attribute at least 100 out of these 104 deaths in Tonghoo to the enzootic of which I am treating. Since the returns were sent in, more deaths have taken place [to the end of February 1885: eight among the mules of European Infantry, seven in Native Infantry, *vide* Telegram Transport 152, dated Rangoon, 17th March 1885]. No cases of this disease have been reported from Moulmein nor from Thayetmyo during these two years; on the contrary there are slight grounds for the belief that some affected animals recovered after arrival at Thayetmyo. Some fatalities occurred on line of march. We

shall not be far out in representing the money loss to Government in two years from this disease in British Burma at Rs. 30,000; and if, as is possible, the losses in elephants (so numerous lately) are also due to relapsing fever, a considerable addition must be made on that account to the above specified amount. We may here give a table showing the losses probably from relapsing fever of Government transport mules in 1884-85 :—

Station.	Corps, &c.	Mules.	Remarks.
Rangoon ..	Transport Supply Depôt	2 + 3	Inclusive of experimental mules, &c.
	2nd Battalion, Somersetshire Light Infantry ..	52 + 6	
	5th Madras Native Infantry	..	
	Queen's Own Sappers and Miners	..	
Tonghoo ..	European Infantry	50 + 8	
	Native Infantry	54 + 7	
	Royal Artillery	..	
Others ..	Various	..	
Total ..	British Burma	182	

6. The influence of this disease on the readiness of the British Burma troops for field service is self-evident. In the current year the fatalities at Tonghoo have been above 50 per cent. on the total strength of mules of the two infantry regiments, and at Rangoon the British Infantry regiment has lost one quarter of its mules. But, unsatisfactory as this present state of affairs is, it is not the only serious feature of prevalence of the disorder as concerns active operations. It is almost certain that, if the mule transport were now called on for service in the field, few animals would survive the first campaign. This view is based on the notoriously unhealthy character of the climate of British Burma, especially to animals of the horse tribe (the Burman pony being the one least liable to disorder). We have history also to support the opinion, as the following extracts from the *Report of the Commission on Indian Cattle Plagues*, 1871, show: "Mr. J. Grierson writes in the 'Transactions of the Medical and Physical Society of Calcutta' (vol. II, 1826,) that in Arracan in July 1825, during the first Anglo-Burman war, 'cattle had begun to be in the same deplorable state (as the men); the camels, with the exception of one or two miserable skeletons still retaining life, had all perished; *horses*, *elephants*, and bullocks daily diminished in number.' Mr. R. V. Burnard writes on the same subject in vol. III of the same Transactions, page 84. He describes the fever which proved so fatal among European and Native troops employed in Arracan from June to November 1825. 'The climate of Arracan appears to be equally hostile to cattle; elephants (from Bengal and Hindustan), horses, sheep, and bullocks died in great numbers * * * . Professor Coleman says that the horse is not the subject of fever, *i.e.*, of a regular succession of cold, hot, and sweating stages. In Arracan it certainly was, and it was a disease of great fatality; the mode in which it terminated was usually effusion in the chest.' Mr. W. Stevenson writes, '* * * Bruce further observes "no horse, mule, or any beast of burden will breed or even live at Sennar or many miles around it; poultry do not live there; neither dog nor cat, sheep nor bullock can be preserved a season there. *They must all go every half-year to the sand.* Though all possible care may be taken of them, they die in every place when the fat earth is about the town during the first season of the rains."' Such in some degree was the case in Arracan. Bullocks died in great numbers, and horses and elephants shared the same fate." Here we have distinct evidence of fever, emaciation, great fatality, dropsy of the chest, outbreak during the first season of the rains, among horses, mules, and other animals. It is impossible to say exactly what the "fever" was; in those days the medical uses of the microscope and thermometer were unknown; so there must always be an element of uncertainty as to whether the fever was anthrax or surra, the balance of argument being in favor of the latter. It is well known that the large number of fatal cases occurring among the artillery horses in Burma is one of the principal causes of the field batteries having been withdrawn from that province, so it would be of great interest if we could, by overhauling old veterinary records of the batteries which were stationed in Burma, learn what were the symptoms and post-mortem appearances of the prevailing disorder, which was diagnosed as Kámrí, or, in other words, was an obscure disease associated with loss of power in the loins,

just as surra in British Burma is! The balance of evidence tends to the view that relapsing fever is *not* a disease new to British Burma. It is almost impossible to exactly establish this position, because so little has been known of animal diseases in Burma until quite recently. The evidence of Veterinary Surgeon (First-class) R. Frost, however, would seem to point to its being a new disease, for, although he has been engaged professionally in Burma for some eight years, the disease is new to him; he has never seen a case before which he can recall to his mind. But his professional work has been among animals proved by experience to be least liable to it, namely, the animals housed in private stables receiving individual attention from people in charge of them. During my stay in Burma I heard of two cases at Rangoon in ponies belonging to private individuals. I did not see them, but there could be little doubt as to their nature. I have seen wretched shadows of ponies grazing in the marshes which strongly reminded me of my patients. The mortality among ghari ponies and others belonging to poor people is high, if we may judge from the not infrequent sight of a carcase by the side of the road in the early morning. These people view the loss of a pony as a "visitation from God" and enter no further into the cause of it, although there is a tradition among them, which I have heard of two or three times, that the ghari-owners could not keep their ponies alive until they hit on the expedient of giving them some gram with the paddy ration. Tonghoo seems from report to be a very unhealthy place for ponies, and, if for them, how much more for imported mules! This question of whether the disease is new to Burma is of some practical importance, for it has been suggested that the transport mules may have brought the disease with them from India, especially the Punjab, in which neighbourhood it is well known to horse-owners and cavalry officers. But, apart from the high probability that the disease is not contagious, as will be demonstrated hereafter, we have the significant facts in this relation that the mules at Tonghoo and of the European Infantry at Rangoon were not, like those of the Rangoon Supply Depôt, subjected to the outbreak the year of their arrival into the country; also that it has not yet appeared among the sappers' mules at Rangoon, the artillery at Tonghoo, or the animals at Thayetmyo and Moulmein. It rather seems that the mules came from India with good constitutions, in exact proportion to which, *plus* the care taken of their health, *plus* the healthiness of the station to which they were posted, has been their freedom from disease. According to this view it is possible that it may next season extend to the stations and corps which have not yet suffered from its ravages, and we cannot yet give an opinion as to whether removal of mules from Tonghoo and Rangoon to Moulmein and Thayetmyo would prevent recurrence of the disorder; it seems not unlikely that aggregation of a large number at Moulmein would render it the seat of an outbreak from which it is now exempted by the very small number of transport animals there. Thayetmyo has stood this test satisfactorily for two years and will probably be found an excellent centre for mule transport.

7. The serious mortality at Rangoon in 1883 was reported on (under date 9th November 1883, No. 1693,) by Veterinary Surgeon (First-class) Frost, then in veterinary charge of the British Burma Transport. He states that the disease from which so many mules have died within the last month or six weeks was ulceration of the stomach, generally confined to the left sac, but in some instances the ulceration extended to both sacs, no traces of disease being found either in the gullet or in the intestines. Some cases were acute, others chronic. He considered it due to something deleterious in grain, grass, or water (*the latter especially*). He thought that the exposure to which the depôt mules were subjected during the height of the monsoon, when quarantined at Kokine, rendered them susceptible; as a matter of fact they went out in good condition and came back in bad. In accordance with his suggestions, Major Hill, Deputy Assistant Commissary-General for Transport, removed the mules and ponies from the depôt and had them watered from a different tank. Inspecting Veterinary Surgeon Evans was referred to in the matter and was "inclined to think that the disease was parasitic" (*vide* Commissary-General Macgrath's letter to Quartermaster-General, No. 2412 T.B., dated 18th January 1884). Inspecting Veterinary Surgeon Shaw was, in the commencement of 1884, deputed to inquire into the disease. He arrived at Rangoon in April 1884, but, as the last fatal case at the depôt had terminated on 14th February 1884, the disease was not then present. He concluded from such evidence as was available that the symptoms and post-mortem

appearances clearly point to some irritant causing the disease; this irritant he thought resided in certain tanks which had been beyond doubt established as sources of infection. He suggested that the animals be kept from drinking the water of stagnant pools and tanks; that eleven "old and worn-out" mules be watered from the suspected sources until the following February, and that, should the disease reappear in the mean while, opportunity will have been afforded to carry out further inquiry. Inspecting Veterinary Surgeon Evans, writing from Ootacamund on 14th May 1884 (No. 549), concludes that there is not sufficient evidence to show what the disease was. Accordingly, the disease having reappeared, I was, in October 1884, deputed to make the suggested necessary investigation. The following is *a précis of the knowledge of the disease acquired before my arrival.*

8. Symptoms.—"The first symptom which the animals showed was loss of appetite, followed by rapid wasting and a general dejected appearance. They soon became tucked up, became unsteady on their limbs, and frequently fell down to rise again after a short time. In some cases the animals showed distinct symptoms of paralysis of the hind limbs. Although grain was refused from an early stage, the animals generally partook of grass for many days." * * * "After an interval of from five to eight or nine days the patients generally were unable to rise, and death invariably put an end to their sufferings in the course of 36 to 48 hours; in some few cases, even in a much shorter time" (Frost). Captain Johnstone of the 2nd Bedfordshire Regiment records (from Tonghoo) that he has noticed "that the majority of deaths have been preceded by a very sudden falling away and also that in many cases a running of the eyes has been a premonitory symptom. At Rangoon an animal "hanging back" on the collar chain was considered a marked symptom. The Tonghoo death reports comprise the histories of cases from the evidence of the Transport Officers. The animals were generally ill some time before death, but the fatal result occurred when not expected. Giddiness, rapid emaciation, extreme weakness, staggering, tenderness over the loins (in one case), urine thick and high-colored, poor appetite, dull heavy listless appearance, apparent recovery followed by relapse, very high temperature, sometimes apparently much pain at intervals, normal evacuations, breathing heavy and difficult shortly before death, sometimes prolonged recumbence and painless death. All these symptoms mentioned in the different returns when examined together give a very good description of those characteristic of relapsing fever. It is to the credit of the Tonghoo Transport Officers that they especially drew attention to the high fever which is present and *observed that it recurred;* they considered these recurrences as separate attacks, not relapses, but were very near a correct diagnosis in one or two cases. In Rangoon the earliest cases of the disease were entered as "fever."

9. Post-mortem Appearances and Pathology.—"Ulceration of the stomach, in the majority of cases, confined to the left sac, but in some instances extending to both sacs, sometimes very extensive, eating away large patches of the internal lining membrane; in others it was less severe" (Frost). "The only visible lesion so far discovered being a corroded or ulcerated condition of the lining membrane of the stomach, more particularly at that part where the cuticular and villose membranes meet and towards the left sac . . . The ulcerated condition appears to have been present in every case to a greater or less degree" (Inspecting Veterinary Surgeon Shaw). When Veterinary Surgeon (First-class) Frost wrote frequent and careful microscopical examinations of the affected parts had failed to throw any light on the subject." Inspecting Veterinary Surgeon Shaw states that "sometimes the patches of ulceration were confluent, and, as described by Mr. Frost, 'had the appearance of a piece of leather that had been denuded of its outer film or enamel by cockroaches.'" "The ulcerations were not deep, nor had they in any case penetrated the walls or given exit to the contents of the stomach. The course of the trachea was free from disease as were the intestines throughout their course. The other viscera were healthy, and the stomach at most presented but a faint blush of inflammatory action. In one or two cases there had been slight hæmorrhage from the ulceration. No post mortem examination appears to have been made of the brain or spinal cord." It seems that Veterinary Surgeon (First-class) Frost made "more than one examination of the blood, but detected no parasites in it;" but Inspecting Veterinary Surgeon Shaw suggests that slides of blood be sent to some spcialist for examination, and says "it is quite open to speculation that further

researches with the higher magnifying powers may reveal some pathological organism hitherto overlooked." The event has quite justified this surmise. The Chemical Examiner to Government, R. Romanis, D.SC. (Edinburgh), examined the gastric ulcers and found in them vegetable débris and low forms of life, but, not being a medical man, did not feel in a position to give any statement as to their pathological importance.

10. From Tonghoo the following list of post-mortem appearances is summarised from the evidence (in twenty-five unselected cases) given by officers of the medical department as attending the committees for post-mortem examination of mules under orders of the General Officer Commanding (there being no veterinary officer at the station):— *Stomach*, contents solid and blood-stained in places or about a quart of yellowish fluid; coats thin and flabby, or no ulceration, the mucous membrane being bile-stained, or a small patch of erosion, or villous portion congested or inflamed, cuticular ulcerated. *Liver* congested. *Kidneys* congested, soft. *Brain*, a blood-clot on the surface, congested, or soft and anæmic, or inflamed. Membranes slightly congested or inflamed. Dropsy of the lateral ventricles. In one case the brain "undoubtedly inflamed." *Lungs* congested, or the seat of abscesses, the result of acute pneumonia. *Heart* small and flabby, or right ventricle dilated and containing a clot or very dark non-coagulated blood. Dropsy of the pericardium. *Small intestines*, upper part inflamed or congested. *Whole body* much emaciated, old-looking, the seat of general atrophy and dropsy. This list, it will be observed, amply confirms my views that the disease in Tonghoo is essentially the same as that at Rangoon; at first I thought the congestion of brain, liver, and small intestine different from the lesions as seen in Rangoon, but I now conclude that they are detectable in acute cases at that station also; this is a result of my latter post-mortem examinations at Rangoon. It cannot be taken as in any way reflecting on the professional ablity of the medical officers who made the above summarised post-mortem examinations if I differ from them as to the cause of death in the majority of cases, since they were dealing with a new disease as affecting patients of a species entirely novel to them from a professional point of view. It will be remarked that I venture to differ from them, not as to the facts observed and recorded, of that disease, but, on the strength of my *special* experience of this disorder, I venture to differ from them in the conclusions to be drawn. The facts are recorded in a manner which has proved most valuable to me, and they contribute materially to our knowledge of the disease.

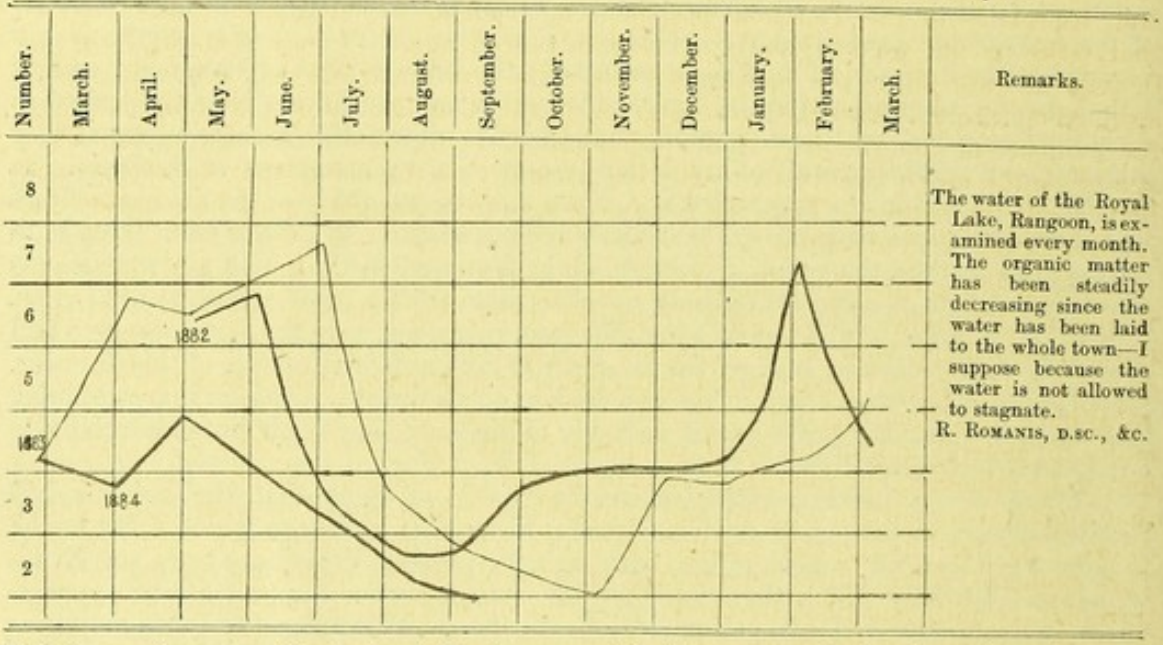
There was a lurking suspicion in the minds of some that the animals had received some irritant poison maliciously administered, a matter to which I felt bound to give very careful consideration, although Inspecting Veterinary Surgeon Evans wrote, on 14th May 1884, that "neither the symptoms nor the post-mortem appearances indicate any irritant poison * * * * . Whatever the cause may have been, it could not have been an irritant; therefore irritants should be excluded in future from our inquiry," and that possibly some mould or fungus on the grass caused the disease. It will be seen later on that I am not quite inclined to accept this view, but attribute some influence in *determining* local lesions to the ingestion of irritant food, not however maliciously administered.

11. ETIOLOGY.—Veterinary Surgeon (First-class) Frost showed that the grass and grain of *all* the transport mules were identical in quality and from the same sources in Rangoon during the first outbreak, of which they therefore, he argued, could not be the cause. He was so strongly convinced of the water from certain tanks being the immediate cause that Inspecting Veterinary Surgeon Shaw accepted his conclusions on the strength of the arguments brought forward by him. Dr. Romanis' water analyses led him to conclude that "beyond showing an excess of free ammonia, which is more or less present in tank waters, there is nothing in the above analysis to account for the great nervous irritation." The analysis referred to was made by himself of the waters of tanks from which the mules of Government in Rangoon were watered. It showed the following results:—

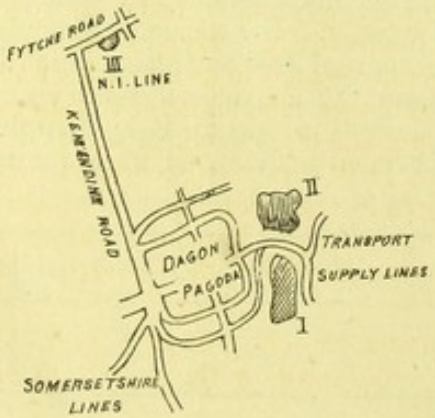
Impurities.	No. 1.	No. 2.	No. 3.
Free ammonia	0·35	0·02	0·52
Organic ammonia	0·40	0·12	0·48

In the water from tanks Nos. 1 and 3 "were over eighty specimens (species ?) of animal and vegetable parasitic (?) life, twelve of the latter (confervæ) being new to science, but whether any of them are noxious is not known. Microscopical examination of the contents of the stomach and of waters from Nos. 1 and 3 tanks was unsuccessful so far as detecting any injurious animal parasitic life goes, but there were present certain vegetable organisms of the Algæ family, some very rare varieties of decimedes, diatoms, chara, and confervæ. Among the infusoria were the harmless vorticella and rotifera" (*vide* Inspecting Veterinary Surgeon Shaw's report). The occurrence of the disease in October and the following months, *i.e.*, "after the tanks had received their supply of organic impurities," was considered a noteworthy point in relation to the cause; but, as a matter of fact, the organic impurities in relation to the bulk of water are then at a minimum, and the maximum is attained generally in May and early in June, when cases of relapsing fever actually commence. It is as well to correct the method of expressing this fact, although its bearings on the disease are the same, *i.e.*, that, after the maximum organic impurity has been attained, cases of relapsing fever begin to occur, and they increase in number with the fall in the ammonia curve as the following chart shows :—

Curves of Organic Ammonia in water from Kanaawgyi (Royal Lake), Rangoon.



Veterinary Surgeon (First-class) Frost suspected the water of the Commissariat tank because it was the ordinary watering place of the depôt mules in 1883, and the "half-transport mules, with the exception of those belonging to the 2nd Middlesex (the regiment which preceded the 2nd Somersetshire), have not partaken of water at that tank, and even the 2nd Middlesex mules have not been watered there for a considerable time since some cases of anthrax occurred at the depôt." Inspecting Veterinary Surgeon Shaw gives Mr. Frost's arguments as follows, with a diagram of the positions of the tanks which he calls Nos. 1, 2, and 3 :—



"(*a*) The Commissariat depôt mules and ponies were watered from No. 1 tank, and the disease was confined to them only (at first).

"(*b*) All the other animals were watered from tank No. 2, and they escaped the disease. Later on in the year the half transport mules of the 11th Madras Native Infantry were, for some reason unknown and without orders from the Veterinary Surgeon, watered from tank No. 3, when the disease at once showed itself. After the first death they were put on well water, but two (three ?) other cases, which were then ailing and must have been infected by drink from No. 3 tank water, died.

"(c) The remaining transport animals that continued No. 2 tank water till it became low (when it was thought prudent to remove them to well water) did not show a single case of the disease * * * *.

"(e) Total disappearance of the disease since Nos. 1 and 3 tank water ceased to be used."

We shall refer to this argument in another part of the report.

12. Veterinary Surgeon (First-class) Frost distinctly recognizes exposure as a predisposing cause, and the Transport Officers at Tonghoo attributed many cases to "exposure when on public command at Thandoun in June" 1884.

13. The possibility of locality causing the disease was recognized; accordingly the Rangoon depôt mules were in 1883 moved out of their lines on to the glacis of the Pagoda Fort, to the Commissariat Bakery, and to Kokine with a view to getting rid of it, and the Tonghoo mules were moved out several miles towards the frontier to the sanitary or cholera camp.

14. The influence of advanced age in causing fatality was thus far noted that "old age" was the verdict of many post-mortem examination boards at Tonghoo, and is entered as the cause of a number of deaths at Rangoon. It is urged that many of the mules sent from India to Burma were very old, and that thus many veterans of Afghanistan—possibly of Abyssinia even—have found graves in Burma, and, especially in the Tonghoo post-mortem reports, we constantly find it stated that "the animal was evidently of very advanced age." I may remark, however, that the disease alters the appearance of a mule very much and makes him look very aged prematurely. Also the ages as given in the Tonghoo returns are obtained from the evidence of the salootri, whose profession of being able to determine the exact age of a mule up to twenty-five years led naturally to the belief that he knows very little about the matter. Thus the returns of ages from Tonghoo must be considered as relative rather than absolute.

15. A letter from the Officer Commanding the left half of the 2nd Bedfordshire Regiment at Tonghoo (dated 29th December 1884, No. 740-84), expresses very well the non-veterinary view of the influence of size, color, age, and sex in producing the disorder. It says: "I have noticed that a larger proportion of first-class mules (*i.e.*, over 13·2) have died from this disease than of the smaller ones, and that the small thick-set mules in general appear to enjoy complete immunity. The oldest mules were the first to be attacked, but the disease was by no means confined to them. I have noticed that a greater proportion of gray and light-colored mules have died than of any other colors. By far the greater number of mules which have died have been mares." This evidence, of an officer who took great interest in his mules of regimental half transport, is quite sustained by the views of the officers and non-commissioned officers in charge of mules at Rangoon. The disproportionate loss of the larger mules was at the latter place considered mainly due to the fact that only a large mule could perform the fatiguing duty of carrying the enormous camel puckals which were then in use in the station and have only recently been replaced by those proper for mules, and it was urged that it would be difficult to prevent a puckal mule from having an occasional draught from suspected or noxious tanks from which he was bringing water for non-drinking purposes up the steep path of the pagoda hill, the journey being performed frequently during the day and the animal much exposed to damp while at the duty.

16. Treatment.—Of *preventative measures* the principal ones adopted were change of locality and of sources of drinking-water supply, especially from tanks towells. *Curative means* were tried, but in all cases ineffectually. Hyposulphite of soda, salicylic and carbolic acids were the principal agents resorted to as a means of counteracting vegetable organisms acting locally on the mucous membrane of the stomach, as was supposed to be the case. Conjee, white of egg, and other mucilaginous substances were also given. Veterinary Surgeon (First-class) Frost says: "The treatment (which I regret to add was entirely unsuccessful) consisted in the administration of astringent medicines and demulcent nourishment." On my arrival at Rangoon, I at once, with a view to studying the disease uncomplicated by the action of medicinal agents, stopped the administration of carbolic acid.

17. Such are the facts which I have been able to collect concerning the knowledge of the disease before my arrival. I found eight cases on the sick list at first (on

17th November 1884) and from time to time fresh cases occurred—nine in number; thus I have had in all 17 cases for study of the disease as occurring naturally. The needs of the inquiry have compelled me to induce the disease artificially in other patients of various species and in different ways. The disease invariably proved fatal, so that I had an opportunity of making a large number of post-mortem examinations illustrating both complicated and uncomplicated cases of the disease. I have appended an account of my methods of inquiry and record, together with a summary of diary of work done. I trust that the facts ascertained, the observations made, and the conclusions arrived at by me will be considered satisfactory; they seem to me to very materially add to our knowledge of the disorder, as was to be anticipated from the exceptional facilities which have been afforded me in the inquiry, and from Dr. Evans' "*Report on 'Surra' in the Dera Ismael Khan District*, 1880," constituting an excellent basis of work, once I had proved to my own satisfaction that I was dealing with either Surra itself or a very closely-allied disorder. I trust that this report will, in its turn, be found a stepping stone to further advances in our knowledge of relapsing fever in quadrupeds and even in man, and that such matters as I have not been able to settle will, by having attention drawn to them herein, be indirectly ascertained through its agency. The disease in question is inferior to none in pathological interest and complexity, and it promises to assume enormous importance in the future on account of its fatality, the number of species to which it is communicable, and the close relations it bears to relapsing fever of man. Under these circumstances, it is well that *ab initio* it should be the subject of careful inquiry, and I feel gratified in having been one of the workers selected to make it known to science.

Part II.—THE OUTBREAKS.

1. For some time after its first appearance the disease with which I have been dealing, and of which I have, in Part I, given the history, remained unrecognized as an epizootic or enzootic. It was supposed that the fatal result depended on a different pathological condition in almost every case. Veterinary Surgeon (First-class) Frost soon corrected this error in Rangoon; but, until quite recently, it prevailed in Tonghoo, where we find the following recorded as the *supposed causes of death*:—Congestion, softening, or impairment of functions of the brain; fever; old age; debility; imperfect action of the heart; blood-poisoning owing to imperfect action of the liver and kidneys; acute nephritis; impure condition of the blood; congestion of brain, lungs, and liver; congestion of brain and lungs; clot in right ventricle; peritonitis; stomach eroded; dilatation of the heart; inanition; senile degeneration; and so on. We have here a curious indirect record of the conditions present after death in cases of relapsing fever, which, with the symptoms and post-mortem appearances already noticed (in Part I, paragraphs 4 and 5), amply confirms my opinion of the nature of the disease causing fatality at Tonghoo. It is necessary for me to summon all my evidence on this point in order to carry as thorough conviction on it to the minds of others as there is in my own, especially since Inspecting Veterinary Surgeon Evans, in his visit to Tonghoo, arrived only at doubtful opinions on this point, and, indeed, his views tended to negative mine somewhat. But, if I understand his views rightly, he is not prepared to oppose *negative* results obtained from a necessarily hurried inquiry to my *positive* results in the same direction. I selected a number of animals considered by the Transport Officers to be affected with "the disease," and proved its existence by demonstration of the parasites in a large proportion of these cases. Dr. Evans selected thirteen animals for destruction (which were not considered by the Transport Officer as affected) on account of excessive debility; he examined their blood only on one occasion and found parasites *then* present in the blood of only one case. Although these animals were not necessarily affected with the fever, being selected merely as being the poorest-looking mules of the infantry at Tonghoo, there is no doubt in my mind that the proportion of affected animals among them was higher than the results of Dr. Evans' blood examinations would appear to indicate: firstly, they seem to have all been thought free from fever, *i.e.*, any actually affected were in that stage of the disorder when the parasites are, as

I have proved, generally non-detectable in the fresh blood; secondly, the "pocket" instrument used by Dr. Evans, although excellent for magnification and definition, is much too small in the field for exact and prompt diagnosis; thus, in Rangoon, it several times gave negative results when my larger instrument readily showed the parasites present. Dr. Evans made some post-mortem examinations at Tonghoo, which, as we shall see directly, have a most important bearing on the relations of gastric ulceration to relapsing fever.*

2. To Veterinary Surgeon (First-class) Frost belongs the credit of drawing attention to the state of the stomach, ulcers in which organ are very marked in a large proportion of the cases. This is a lesion generally associated with the action of an irritant poison, and, apart from such influence, is rarely seen in veterinary practice as may be inferred from the record of a case in vol. XIII, Veterinary Journal, page 201, where it is noticed editorially as quite an exceptional condition. Under these circumstances it was natural that the cause of irritation be sought for in the food and surroundings of the animal. Firstly, the question of *malicious poisoning* has to be dealt with. It seems, from the evidence bearing on this matter, that there has been at times some dissatisfaction among the transport drivers, and it was thought that this could be directly associated with the prevalence of the disease in different lines at Rangoon at different times; the influence of attendants on the development of the disease was thus far perceptible that some drivers have lost fewer of their mules than others, or even none of those under their charge. In one or two cases the driver, after losing all his three mules, deserted. Chemical analysis of the viscera was resorted to, but no poison detected; the gastric contents administered to a mule from two fatal cases failed to communicate the disease, but the cases might have been chronic in which all poison had passed off from the system in the earlier stages of attack, so it was necessary to get other evidence. The disease practically occurred only among Government animals, and it was difficult to see how poisoning the animals could benefit any one—contractor, drivers, chumars (the carcases being burnt generally, and, even if they were not, the skins of little value intrinsically and generally much bruised and spoiled in this disease), or others. Again, the drivers in general seem fond of their animals, and it is not infrequent for favorite mules to die, such as one ridden by the Native Infantry Jemadar at Tonghoo. Further, we are acquainted with no poisons such as would produce the lesions of the disease. Still it seemed possible, but not likely, that some poisonous herb known only to natives had been used maliciously. However, the clinical features of the disease were not those of poisoning, especially was this evident when the fever was proved to be a relapsing one; also I asked myself, why, if due to poison, should it occur mainly at certain seasons of the year? So I found that the balance of evidence absolutely excluded the possibility of malicious poisoning. We have seen how an irritant was sought for in the water used for drinking purposes and supposed to be traced to a particular tank, which was forbidden for use. On the same supposition the Somersetshire mules were moved for watering from the Cemetery tank in about March 1884 to wells, in about two months after that to the Royal Lakes, and in October 1884 to the wells again, and yet, while obtaining well water, tank water of excellent quality from the Royal Lakes, of inferior quality from the cemetery tank, these mules have succumbed to the disease; moreover, of two troops watered from the lower well (C and D), one has lost a large number of mules and the other very few, and those fairly attributable to other causes. Thus, since of two bodies of mules watered from the same source constantly, a large proportion of the one died and the other practically escaped, it is evident here that the cause lies somewhere else than in water from a special source. The experiment suggested by Inspecting Veterinary Surgeon Shaw unfortunately was not thoroughly carried out; the eleven experimental mules were watered from the suspected tank for eight months—April to November 1884—but of them, some were used for other purposes; thus four of them were exposed on anthrax graves, being allowed to shift for themselves in the matter of food and water; of them, two completely resisted disease and two died, their stomachs being found ulcerated on post-mortem examination; but whether or not they suffered

* It is right to state that Dr. Evans assures me he took sufficient time and care to examine the blood of the mules at Tonghoo, and he is quite certain that they were all free from the parasite *then*, excepting one, though more or less ulceration of the stomach was found in eleven out of thirteen.

from relapsing fever was not ascertained, and we have no evidence to show that the ulceration depended on any cause other than their vagabond mode of existence. One of the experimental mules died of "ulceration of the stomach," *i.e.*, that lesion was found after death; another died on 27th November 1884, and was carefully examined by me. He showed no signs of relapsing fever and his stomach was not ulcerated. I have carefully examined five of these mules for some time and found them anæmic, but not suffering from relapsing fever, as I am in a position to state positively. The remainder of these mules got fat and have been sent to the regiments for duty. Thus three cases of "ulceration of the stomach" have followed in eleven mules experimented on; of these, two are excluded as having been not treated in accordance with the conditions of the experiment, and the single remaining case may have been due to other causes operative in the station at the time. Six animals certainly, and two more probably, did not suffer from drinking the water of tank No. 1. Such results as we can obtain from this imperfect experiment are, therefore, opposed to the view that some irritant in tank No. 1 water caused the disease. The water of the tank in question was in November 1884 bright and sparkling, but I find that in January 1885 it has become almost green from plant growth and contains much organic suspended matter, both animal and vegetable, and there is a scum on its surface in places, especially at the corner near the road from the Somersetshire lines, where the tank is freely accessible for drinking purposes. The corner near the Depôt Lines where the animals used to be watered is comparatively pure, and there is no possibility of sewage contamination of this tank. It contains fish and tortoises. The arguments against this special tank are defective. My researches have brought to light error in some matters which are advanced as facts; thus one case of the disease occurred in the European Infantry mules in December 1883; again these mules seem to have been watered at the cemetery tank, not No. 2, but, even if they were watered at the latter, it is not likely that on all occasions they would have passed the accessible eastern corner of tank No. 1 to go a quarter of a mile farther on to tank No. 2. The supply of water to the depôt mules while in quarantine at Kokine is not mentioned, and it is not considered that a transport mule waters almost anywhere, if used for puckal work at the tank to which he goes, if on orderly duty at every place where opportunity offers, and the same with other duties. It is practically impossible, from the very nature of the service, to prevent mules obtaining water from various sources, and the probable fact that private animals—certainly the hired mules and ponies—have not been involved in the outbreak seems to indicate that the drinking water may be very bad and constantly varied without causing the disease. It is, on the other hand, difficult to prove that an animal has not had water more or less directly associated with a suspected source; thus the latter may have been brought up in mussack or puckal, used afterwards for the drinking water. I may sum up my views on the question of water contaminated from certain sources by saying that, although not absolutely disproved, it is not probable; the balance of evidence is against any single collection of water being the focus of disease and as such acting as an irritant in the generation of ulceration of the stomach. The holders of this view of the nature of the British Burma disorder argue, feasibly enough, that the occurrence of the disease at a special season of the year is *probably* due to that being the season when low organisms in water throw off their active reproductive elements which may be noxious to mules, but this involves a supposition which requires to be substantiated. They further argue that non-specific inflammations sometimes, especially in the tropics, manifest a certain degree of periodicity. The former argument may possibly bear on the established pathology of relapsing fever; the latter may be dealt with by saying that we can explain the periodicity, which is more regular and systematic than in other diseases, much more satisfactorily, for we find it *in direct association* with an organism in the blood. Having thus completed my argument against the disease being due to the action of an irritant locally on the stomach, *i.e.*, ulceration of the stomach pure and simple, I must now examine the question of the *relations which ulceration of the stomach bears to relapsing fever in this outbreak.*

3. The disorder was at one time mentioned in official returns as "ulceration of the stomach," and measures of treatment were adopted accordingly. Having been specially detailed for the investigation of this disease, I was enabled to devote to it

that care and attention to exact and continued observations which my predecessors had not been able to find time for in consequence of other urgent duties. I had been nearly a month in Rangoon observing the clinical features of the disease before (on 11th December 1884) I recognized in it the parasite of Surra. This observation, soon amply and thoroughly substantiated by frequent repetitions, at once afforded me a solid basis for future work. It was evident to me, from the very diffused nature of the lesions as observed post-mortem, and on other grounds, that the blood was the seat of disorder. But the question arose whether the ulceration of the stomach would not fully account for this; no doubt such extensive ulceration as was generally found would admit of absorption of unaltered gastric juice and of imperfectly prepared products of digestion. The post-mortem evidences are not contradictory to this possibility, and the most evident symptoms of the disease might be accounted for on this theory. The persistently acid state of the urine, too, indicated excessive acidity of the blood, which might be due to prematurely absorbed gastric contents, and the occasional occurrence of albumen in the urine might be accounted for in this manner. And yet there were difficulties in the way of this view, especially it would not account for the periodicity of the disease which was not in association with the phases of digestion nor with any other detectible gastric influence. Further, it soon became evident that in a sufficiently large proportion of cases post-mortem examination showed ulceration of the stomach only to an insignificant extent or else entirely absent. This led me to conclude that *gastric ulceration is not the disease, although evidently in very close association with it either as a cause or an effect.* A mule having been destroyed for broken leg was found to have ulcers of its stomach; this fact still further confirmed my view that the gastric ulceration was but secondary, although (considering the fever cases are not diagnosed at their commencement) it is possible that the animal suffered from the disorder without it having been recognized and should at the time (November 1884) have been on the sick list for it. Gastric ulceration having been proved but secondary to the principal disorder, the important question next suggested itself whether the relations between the two conditions are accidental contemporaneous existence, causal, or the ulcers but a local manifestation of specific disorder. (*a*) The two states might be due to a common cause acting as a poison on the system and exerting a local influence on the stomach; (*b*) or the ulcers in the stomach might be the portal of entry of the specific element into the blood; (*c*) or they might be the vents through which the blood expelled it from the system; or (*d*) the two states on account of the great frequency of both among a limited number of mules might occur simultaneously in many cases. In endeavouring to solve this difficult question, I found it necessary to examine the pathology of the stomach ulcers with very great care. I obtained the following results from post-mortem examinations:—

Observer.	Number of animals examined post mortem.			Cases of ulceration found.	Remarks.
	Relapsing Fever.	Doubtful.	Fever not considered present.		
Self	9	..	1	7	Cuticular diseased in 6, villous in 4, both 1.
Tonghoo Post-mortem Boards.	..	31	..	18	
Dr. Evans	1	..	12	11	
My cases during absence ..	2	..	..	2	Cuticular diseased in 1, villous in 1, both 0.
Total ..	12	31^{+3}	13^{+6}	38	Per cent. ulceration = $67\frac{9}{7}$.

Out of 12 *cases certainly affected with Relapsing Fever* 9 *had ulcers in the stomach;* thus *ulceration of the stomach has been found more or less marked in two-thirds of the cases examined and in* 75 *per cent. of those known to be affected with Relapsing Fever*, which seems to indicate something more than accidental coincidence*. We must examine the condition of the stomach to enable us to determine whether it at all assists us in the inquiry.

4. We may supplement Veterinary Surgeon (First-class) Frost's account of the ulcers by the following précis of observations made by the Tonghoo Boards, Dr. Evans,

* Out of 38 cases of ulceration of the stomach as found post-mortem only 12 (about one-third) were known to have suffered from relapsing fever, but the diagnosis was often most unsatisfactory.

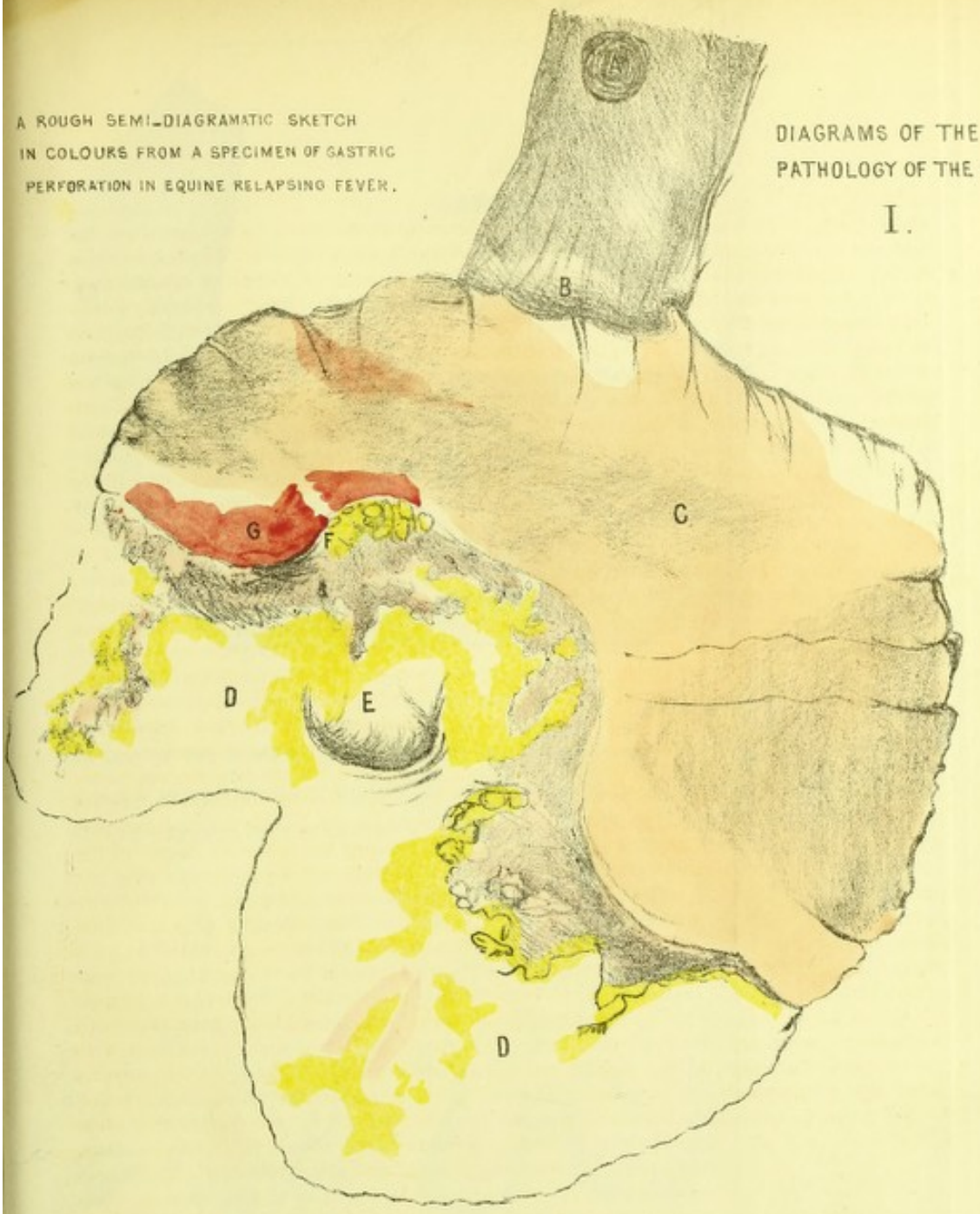
and myself :—In one of Dr. Evans' cases the stomach was much ulcerated in two patches —one small, the other very large ; the small one presented a " bloody slough " and the stomach contained black fluid of a very fœtid odour. There was yellow discoloration of the cuticular portion in this case, and it seems from the record of symptoms to have been a true case of relapsing fever. In another, which, when examined, gave no evidence of being affected with the fever, he found a patch (1 inch by $\frac{3}{4}$ inch), irregular in shape, on the cuticular portion, of a brownish leathery appearance, not detached nor presenting any tendency to separate, thinner than the normal membrane. There was neither congestion nor inflammation of the parts around this patch of what Dr. Evans considers " degenerated membrane which probably would become a slough." He found in almost all cases yellow pigmentation of the lining membrane and " dry sloughing " without inflammation along the line of meeting of the cuticular and villous parts. In the Tonghoo death returns the yellow coloration is mentioned as bile-staining, and the pathological states of the stomach are noted as extensive ulceration, erosion in patches, inflammation, and congestion ; sometimes the ulcers are noted as superficial and shallow, at others they are " evidently of old standing " or " large and irregular." One case is specially worthy of record, for in it there was a " large ragged ulcer on the cardiac extremity " and the organ " contained a large hard mass of undigested paddy and grass, one side of which, *i.e.*, that in contact with the ulcer, was covered with blood." In Dr. Evans' case of gastric hæmorrhage he also noted the presence of rough, indigestible matter in the form of a bunch of unmasticated straw. In my post-mortem researches I have found ample evidence that, as Veterinary Surgeon (First-class) Frost observed and recorded, both the cuticular and villous portions of the gastric mucous membrane may be involved in the disease, but the former actually is involved more frequently than the latter. I find that sometimes the cuticular may be the sole seat of disease, sometimes the villous, and sometimes both are simultaneously diseased or both healthy. I also observe that there are, so to speak, favorite positions for the occurrence of the lesions (as Inspecting Veterinary Surgeon Shaw indicated) at the junction of the two portions and also at the lesser curvature rather than at the greater, and at either side of the middle line. Further, I find that the lesions of the villous coat differ from those of the cuticular only in minor respects, such as may fairly be attributed to their anatomical differences. As the lesions of the cuticular are more frequent than those of the villous portion, I will deal with them first. The bright yellow color presented when the stomach is opened at once attracts attention, for it contrasts remarkably with the normal whiteness of the epithelium. In the Tonghoo reports the stomach is mentioned as extensively bile-stained, but we have no evidence that the coloring is due to bile ; on microscopical examination we find that it results from degeneration of epithelium and other tissues. Careful examination very soon makes it clear that *there are several different ways in which the degeneration commences*—(*a*) An irregular thickening of the epithelium takes place in lines with generally a tendency to radiate from the œsophageal opening, or to extend along the line of junction of the cuticular with the villous portion. This is due to a proliferation of the epithelium and at first results in increased opacity, but later the epithelial cells begin to break up into yellow débris from those parts which have attained a certain thickness ; thus yellow superficial ulcers are formed which run together along the disease ridges and extend laterally, so that a tendency to linear arrangement of the ulcers is generally marked in this diseased state. (*b*) At definite spots the epithelium becomes depressed and semi-transparent from the loss of its deeper cells which have commenced to degenerate. Next the superficial cells give way and small ulcers—circular, shallow, and yellowish—result. These in time run together ; they do not show any tendency to linear arrangement, but by their extension and confluence frequently isolate irregularly-shaped portions of the healthy epithelium from their surroundings, and these healthy parts separate from their margins and gradually curl up until they are shed. (*c*) Sometimes a small, generally circular, loss of the glistening dense outer layer of epithelium is the first noticeable change. The effect of either of these processes of degeneration of the epithelial layer is to expose the corium, which in its turn undergoes degenerative changes which have probably already advanced considerably by the time the epithelium is thoroughly removed. Sooner or later the corium disappears and the white muscular fibre is exposed, only in its turn to undergo the yellow degenerative change becoming thinner and thinner

Vide diagram.

A ROUGH SEMI-DIAGRAMATIC SKETCH
IN COLOURS FROM A SPECIMEN OF GASTRIC
PERFORATION IN EQUINE RELAPSING FEVER.

DIAGRAMS OF THE PATHOLOGY OF THE STOMACH.

I.



CASE G: DIED 20TH NOVEMBER 1884, PERFORATION OF THE STOMACH BEING THE IMMEDIATE CAUSE OF DEATH.

A. OPENING OF BILE DUCT.
B. PYLORIC OPENING.
C. VILLOUS PORTION OF STOMACH.
D. CUTICULAR SHOWING YELLOW CHANGES & ULCERATION.
E. TERMINAL EXTREMITY OF ŒSOPHAGUS.
F. PERFORATION
G REDDENED CONDITION OF THE VILLOUS PORTION AGAINST THE PERFORATION.

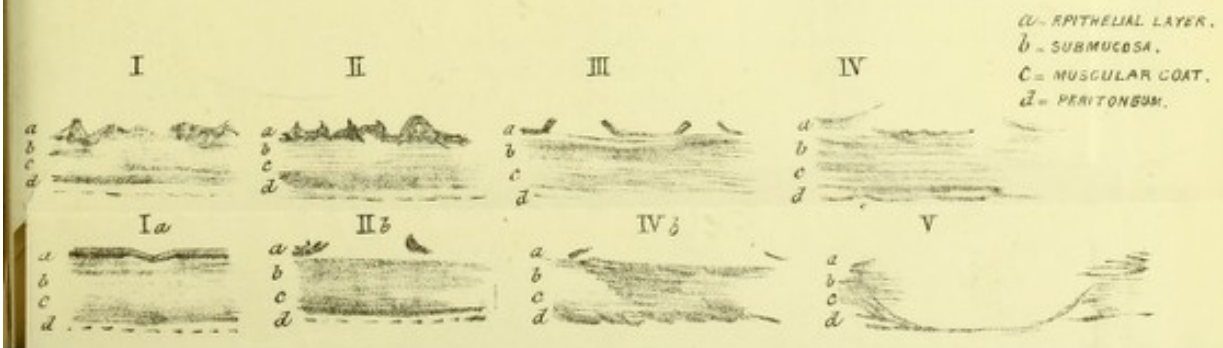
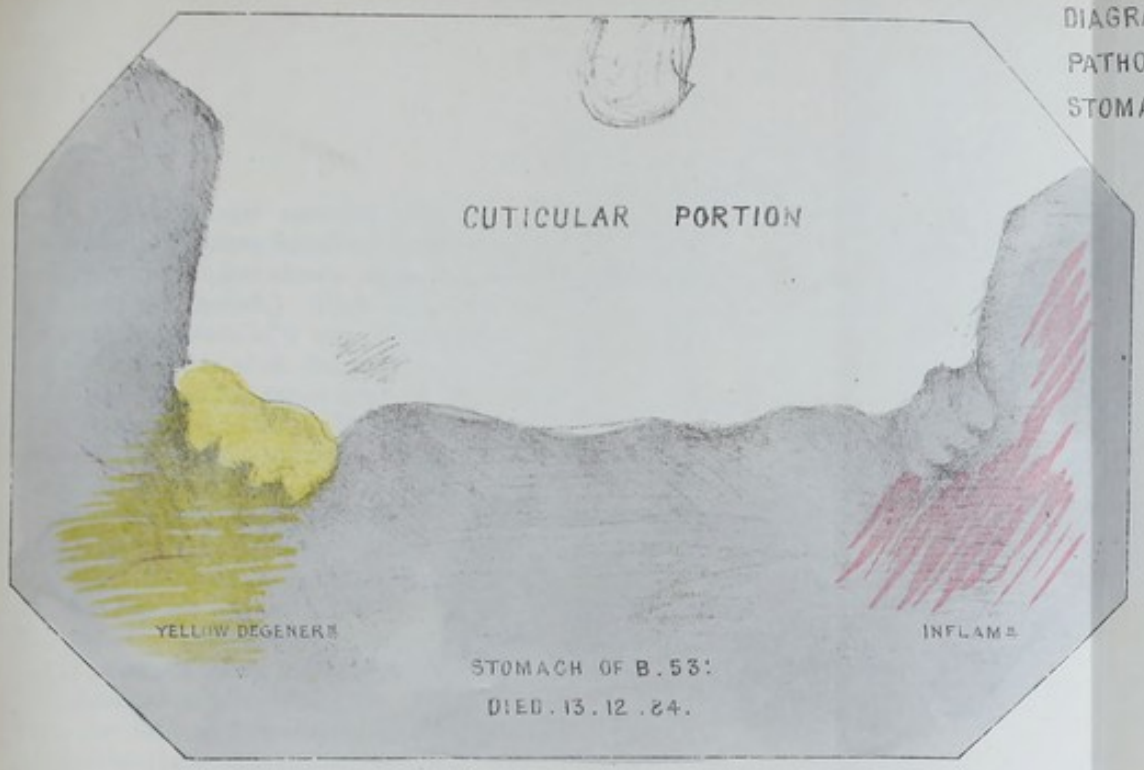


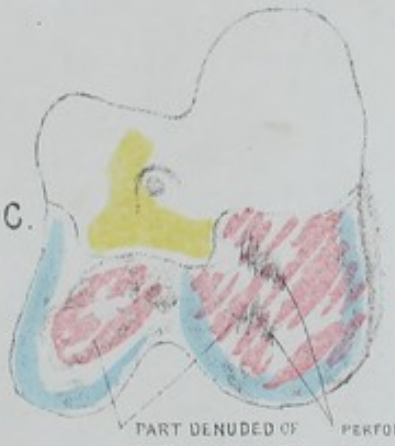
DIAGRAM TO ILLUSTRATE THE STAGES BY WHICH GASTRIC PERFORATION IS BROUGHT ABOUT IN EQUINE RELAPSING FEVER, AS DESCRIBED IN THE TEXT.

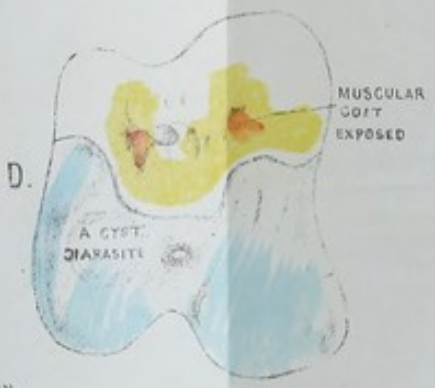
A

DIAGRAMS OF THE PATHOLOGY OF THE STOMACH II.



DIAGRAMS OF THE MORBID CONDITIONS OF THE STOMACH FOUND IN THE BRITISH BURMA DISEASE.
GHT YELLOW DEGENERATION OF EPITHELIUM ASSOCIATED WITH AN, APPARENTLY NATURAL, EXTENSION OF THE
US AT THE EXPENSE OF THE CUTICULAR PORTION





SE IV. A. 39: ULCERATION OF VILLOUS
YELLOW DEGENERATION OF EPITHELIUM
NG FROM CUTICULAR.

C. CASE XI. B. 55. DIED 31ST DECR 84.
PERFORATION OF VILLOUS COAT.
CUTICULAR PORTION UNALTERED.

D. B. 56: THE CUTICULAR
UNDERGOING YELLOW DEGENERATION.
MUSCULAR COAT EXPOSED.

III.

AS CLOSE A COPY AS POSSIBLE OF A BLOOD CLOT FROM A CASE OF RELAPSING FEVER IN A MULE: INTENDED TO ILLUSTRATE THE EXTRAORDINARY VARIETY OF COLOURS SEEN IN THESE CLOTS.
(VIDE TEXT.)

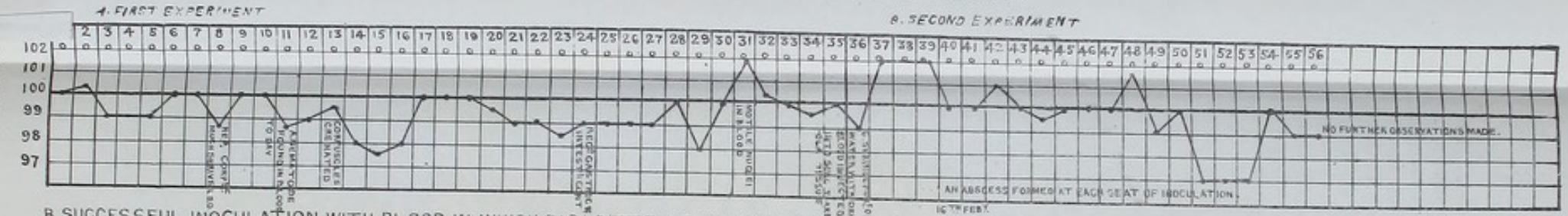
until, if the animal remains alive long enough, perforation occurs. Probably the peritoneum becomes involved in the general atrophy, but I have not seen a single case in which it has shown signs of inflammation opposite the ulcers. (In Tonghoo one case was noted.) Here and there a stellate congestion of the corium may be detected by means of a magnifying glass. Condensation of the areolar tissue connecting the muscular with the mucous coat and sometimes thickening of the former takes place, and later the three coats of the stomach may become united by consolidation of their connective tissue. Perforation may result from extension of the ulceration and is generally a minute opening through the peritoneum, its length corresponding to the direction of the outer layer of muscular fibres. It is sometimes seen in the cuticular portion, sometimes in the villous. Where the muscular layer is exposed, its blood-vessels are often distinguishable as black lines between the fibres; they are filled with coagula. This leads us to the important *question of whether the blood organisms are the producers of the ulceration,* and gives a clue to the manner in which they might produce that effect. The villous portion is less adapted to show the lesions above described, but it presents them, one and all, in different cases. We frequently find that part of its substance is dense and thicker than the rest, but not uniformly so, for here and there are thin spots resembling ulcers, or we may find lines of ulceration looking very much like rents converging towards the pylorus, or the membrane may be merely congested and present blood-spots. In some cases there are changes going on at the line of junction of the two portions which look like (and probably are) a normal change of nature of the membrane, an increase in size of the villous at the expense of the cuticular, especially wherever the latter normally projects into the former. I have also seen cases in which the ulcers were apparently undergoing gradual, but not very advanced, repair. Various forms of parasites are to be found, but this is nothing unusual. The abodes of sclerostomes and the gelatinous mucus in which those on the mucous membrane are embedded are constantly alluded to in the post-mortem records, but apparently have no direct relation to the disease. Bots leave behind them marks resembling some of the smaller ulcers on the cuticular part, but these will not be confounded with the extensive removals of tissue found in true cases of gastric ulcer. Dr. Romanis has found, on microscopical examinations of the ulcers, low organisms and tissue débris—in fact such matters as we find in the gastric contents. My results are the same with well-marked and advanced ulcers, but in the earliest stage of epithelial change in the cuticular portion I have noted no organisms, simple breaking up of the cells, which seems generally to commence about the orifices of the follicles which open into the stomach. Thus from post-mortem inquiry we obtain some important items of information of which we may note especially (1) that the ulcers are specially frequent in certain definite situations; (2) that they may originate in the deep-seated epithelial cells as well as the superficial; and (3) that sometimes they are in direct association with plugged vessels. To these we may add that (4) similar ulcers are observed in the nose and mouth during the progress of a few cases. To the first matter we must not attach special significance because it is also noted in simple ulceration of stomach in man and probably depends on the manner in which the blood-vessels are distributed to the organ. Items (2) and (4) tend to prove that the ulceration is a consequence of disease of the body in general, arising from within and not from the direct action of irritants taken into the stomach, and item (3) gives us a feasible explanation of how this might take place in the course of an attack of relapsing fever, the parasites plugging the small vessels of the gastric coats and thus causing circumscribed ulcerations of them. We must remember, on the other hand, that ulceration of the stomach was not found by Dr. Evans in his surra cases and is not noted in relapsing fever of man (it seemed also to be absent from the stomach of my dog,* which succumbed to the disease as artificially induced), and that we have ample evidence of the use of paddy, uncrushed gram, and rice straw as food having been long-continued—irritant substances which might possibly give rise to rents and ulcers of the gastric mucous membrane, as also might the sharp-edged cutting grass, which is often given as fodder. We have a certain amount of evidence that the use of uncrushed paddy is liable to cause stomach lesions. The native horse-owners at

* And was not found in my experimental monkey.

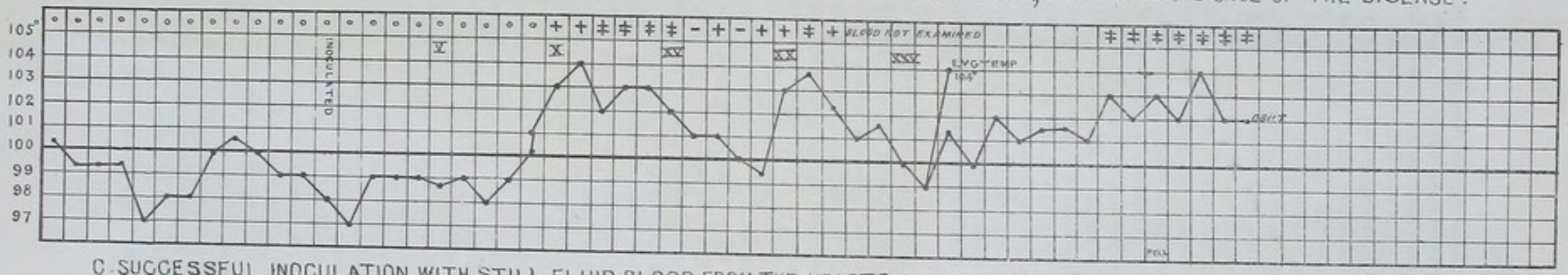
Rangoon bruise the paddy before use with a view to breaking off the irritant sharp points, which they think injurious to the stomach. The Tonghoo Committees noted the occurrence in the stomach of unbroken paddy in several cases, and one Board suggested "that paddy be no longer given and some light easily digestible substitute be made." In one of my *experimental* cases which died on the fortieth day of the disease, I find well marked ulceration of both cuticular and villous coats bearing a fairly direct relation to the known duration of the case. He has been fed with soft readily digestible food since admission. In my experimental cases which succumbed subsequently to this I always found gastric lesions, more or less intense. Altogether the evidence very decidedly tends to the conclusion that *gastric ulceration must be directly associated with this outbreak of relapsing fever as a special feature of it and one materially aggravating the symptoms.* The latter point must not, however, be exaggerated; thus the persistent acidity and occurrence of albumen in the urine must not be attributed entirely to this lesion, for I have found them in a case where the lining membrane of the stomach was proved to be normal after death, and the acidity of the stomach contents is constantly diminished in fever, so would be less likely to affect the blood in relapsing fever than in health. Again marked pain in the region of the stomach, indicated by the animal turning his head to the left side and biting the hypochondrium, occurs sometimes, and in one instance of it I found no ulceration of the stomach. Having thus decided that we must, at any rate provisionally, accept ulceration of the stomach as a lesion of the disease, and not simply in the light of a cause or accidental accompaniment, the next matter to be dealt with was whether the disease is an eruptive disorder having the local manifestations of the constitutional derangement in the stomach.

5. I was at first inclined to accept this view. I find from my records that before I detected the parasite in the blood, I thought the disease was malarious and decided to, *pro tem.*, term it *gastric typhoid;* there certainly was well-marked cachexia, fever was a prominent symptom, one part of the alimentary canal (the stomach) was the seat of ulceration presenting special features, and sometimes there were circumscribed congestion of the mucous membrane of the small bowel, often somewhat most marked on Peyer's patches. In some cases just before death the fæces became soft, reddish, and most offensive to the smell. I think the following extract from my note-book of an entry made at this stage of the inquiry will be found sufficiently interesting to warrant its insertion here:—"The *theory* of malaria in the production of this affection must be accepted as by no means established because it is incapable of exact demonstration, and the majority of the conditions seen in this disease are referrible to local states. But analyse the symptoms, post-mortem appearances, and (other) causes as we may, there is still a something deficient, which 'something' may be considered as specially acting on the blood, probably superadded to it. Its effects are remarkably like those exerted by miasma on man, notably the periodic exacerbations of fever and occurrence of crops of petechiæ, and the characteristic cachexia and embolic complications. We cannot attribute them to ordinary poisons; they are not such as would result from premature absorption of semi-digested albuminoids or acid secretions through the gastric ulcers; they are something more than simple anæmia. To the influence of miasma only can I attribute them with the remark that miasma is, in all probability, parasitic in its nature, essentially consisting of minute organisms generated, or finding their most suitable nidus of growth in decaying organic matter, but having a parasitic stage, during which they inhabit the bodies of mules, horses, possibly also men. Whether these organisms be animal or vegetable cannot here be discussed. Such argument would be premature!" Here I was manifestly verging on determination of organisms in the blood. I had decided that the disease usually found in the kidneys and brain is evidently, from its nature, secondary; its absence in some cases also proves this, and although the disease in some respects resembles uræmia, yet it presents very markedly different phenomena in others. The generally diffused character of the lesions, the height and special relapsing nature of the fever, the absence of any definite and well-defined local lesion invariably present in association with the characteristic symptoms of the disease, the fact of different cases assuming different forms and of petechiæ appearing on the mucous membranes clearly pointed to its being a disease of the blood. This important fact having been

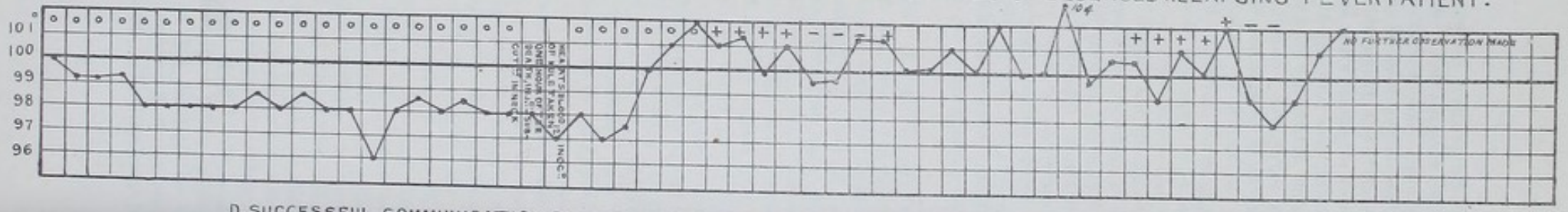
A. UNSUCCESSFUL ADMINISTRATION OF GASTRIC & INTESTINAL CONTENTS INTO THE STOMACH; (b) AND OF THOROUGHLY DRIED BLOOD INTO SUBCUTANEOUS AREOLAR TISSUE.



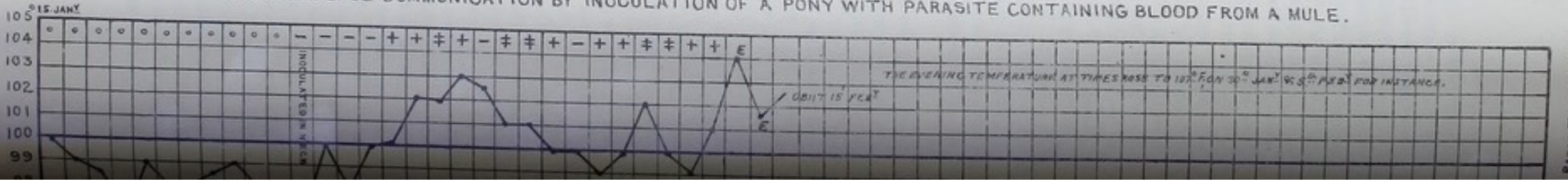
B. SUCCESSFUL INOCULATION WITH BLOOD IN WHICH PARASITES WERE NOT AT THE TIME DETECTIBLE; FROM A NATURAL CASE OF THE DISEASE.



C. SUCCESSFUL INOCULATION WITH STILL FLUID BLOOD FROM THE HEARTS CAVITIES OF DECEASED RELAPSING FEVER PATIENT.



D. SUCCESSFUL COMMUNICATION BY INOCULATION OF A PONY WITH PARASITE CONTAINING BLOOD FROM A MULE.



TEMPERATURE CHARTS OF EXPERIMENTAL ANIMALS—No. I.

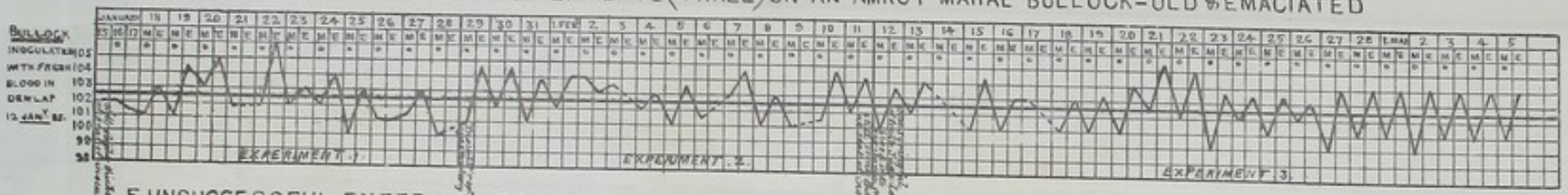
ROMAN NUMBERS (THUS V) = DAY AFTER INOCULATION.
SMALL NOS. (THUS 36) = DAY OF OBSERVATION OF ANIMAL.

PARASITES PRESENT.
NO PARASITES DETECTED
PARA NUMEROUS & ACTIVE.
NO APPRECIABLE ABNORMALITY OF BLOOD.

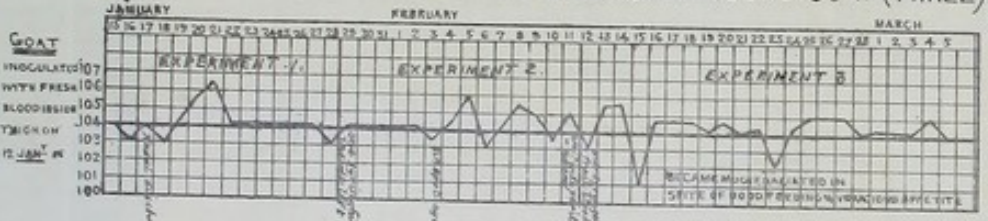
TEMPERATURE CHARTS OF EXPERIMENTAL ANIMALS Nº 2.

TO FOLLOW Nº 1 AND BOTH TO FACE P. 17.

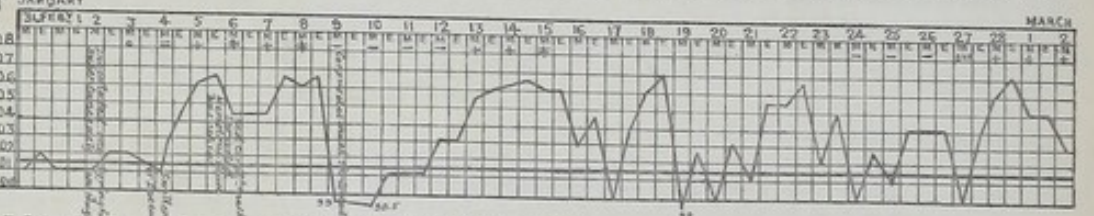
E. UNSUCCESSFUL EXPERIMENTS (THREE) ON AN AMRUT MAHAL BULLOCK – OLD & EMACIATED



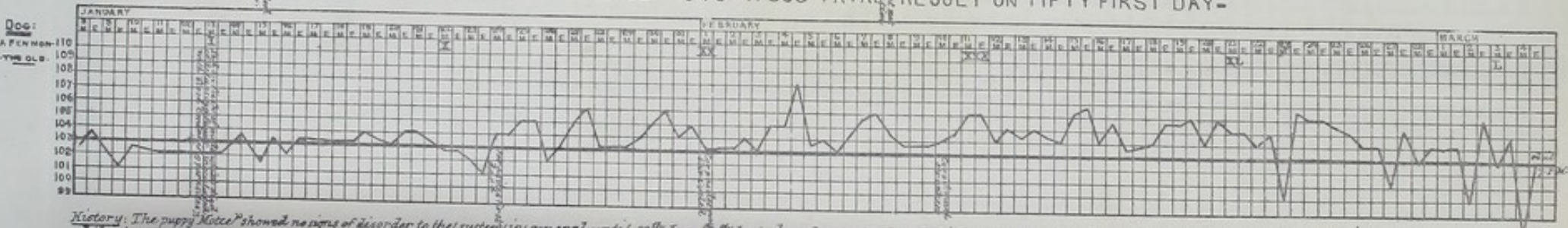
F. UNSUCCESSFUL EXPERIMENTS ON A YOUNG GOAT – (THREE)



G. SUCCESSFUL INOCULATION: EXPERIMENTAL MONKEY – STILL ALIVE BUT VERY THIN & WEAK.



H. SUCCESSFUL INOCULATION OF A DOG – FATAL RESULT ON FIFTY FIRST DAY –



History: The puppy "Kitty" showed no signs of disorder to the system in general until 28th Jan. (12th day) when she was dull, had slight cough, and exhibited pain when pressed on the loins – She was very feverish while the blood contained parasites and the conjunctiva became pale in the extreme. She was allowed to eat portions of all the mules which died but her appetite became very capricious. On my return from Thayetmyo 25th Feb. 1885 I found the puppy very dull, feverish & thin – with a swelling on each side of the head, much altering her appearance – also the left inguinal gland swollen & the body in general so tender that the animal shrieked when lifted from the ground – a large number of disintegrating corpuscles but no parasites detected in blood – The swelling of the head increased & on 1st March there appeared edema of the perineum & the throat & muzzle were of great size – on 2nd March she seemed a little better but this improvement was only temporary – on 4th March she lay on a side, struggling in delirium just as the mules generally do, and became very cold, with the hind quarters much swollen – she died at 7 P.M. on that date.

Autopsy: (as I was away could not be made until some four days after death) – in so far as I could ascertain the lining membrane of the stomach was not ulcerated.

settled, it was next necessary to decide in what respects that fluid is modified. The earlier lesions noted were those of simple anæmia, a paucity of red corpuscles, a shrivelled or imperfect condition of the blood elements, an apparent preponderance of white corpuscles, which were soon found to be not *white* but decolorised *red* ones, a tendency to dropsy, petechiæ of membranes, cardiac murmurs immediately preceding death, extreme pallor of membranes, the dry harsh state of the skin and acidity of urine (as though the animal, for want of sufficient vegetable diet, had to become practically carnivorous in living on its own tissues); these were conditions enough to warrant me in seriously considering whether I had not been sent simply to investigate a disease which I should have to report as under-feeding pure and simple! In starvation there may be exaggeration of normal daily variations in temperature, and a certain periodicity dependent on the phases of digestion might be present in association with gastric ulceration, but, as the temperature tracings gradually developed themselves, I became assured that simple anæmia would not suffice for the explanation of a *Relapsing* Fever, nor explain why the disease occurred at certain seasons especially. For some short time after I subjected the blood to elaborate scrutiny, only small, spherical, mobile, highly refractive bodies attracted my attention as unusual. I thought they might be the micrococci of malaria as described by Tommasi Crudeli and others. Soon, however, the characteristic organisms became evident and at once dispelled, in a most satisfactory manner, all ideas of typhoid, ulceration of stomach from irritant ingesta, malicious poisoning, and simple anæmia, but not absolutely the theory of malarial influence. It may at first seem strange that with active living beings in the blood Veterinary Surgeon (First-class) Frost and myself (for a month) had not observed them, but in this relation it must be remembered that it is only at certain phases of the fever (which happen to be at the time when the animal is least likely to be brought to the notice of the veterinary officer) that they are detectible in the blood; secondly, the parasites are, under ordinary powers, detectible only by most intent observation *when we know what to look for*. Dr. Evans' Surra observations "gave us a good lead," but we were "thrown off the scent" by the gastric ulceration, which induced us to believe that we had to deal with a disease other than Surra. What led me back to the right track was especially the markedly relapsing character of the fever as shown by the beautiful thermographic tracings which the cases afforded in the first month; and, as Relapsing Fever in man is associated with spirilla in the blood, I was led to seek and find spiral organisms in mules affected with the British Burma disease. Then, with increased familiarity with the disease, I found many of the differences between it and Surra (as observed by Dr. Evans) melt away, and now but few remain. The symptoms in a pony which obtained the disease artificially almost exactly answered to Dr. Evans' description of his cases in Punjabi horses.

6. To illustrate the results of clinical observations made by me and to give an idea of the symptoms and post-mortem appearances found in the mules, I will insert in the Appendix my note-book records of clinical work for two days consisting of pulse, temperature, &c., records and the results of urine analysis. Also I give the charts of one ordinary case of the disease, the record case book entries of two more, the charts of experimental mule cases and of one pony and one dog case, and the charts of complicated cases as occurring in the mule. These will suffice as a means of illustration of the cases to be dealt with in the inquiry, and also as a means whereby we may compare this Burma disease with Relapsing Fever as observed in man and Surra as studied by Dr. Evans in and about Dera Ismail Khan. In comparing Surra with the British Burma disease, I have had the benefit of Dr. Evans' opinion after personal examination of Burma cases. He *was* inclined to consider the two diseases as distinct, but closely allied, rather than essentially the same disease modified by difference in species of the animal affected, geographical range, and other local conditions. He *now* agrees with the latter opinion. There are some of the *symptoms which I have observed as differing markedly from those mentioned in the report on Surra.* I have seen serous effusion beneath the vulval mucous membrane, but very seldom the petechiæ in that situation as described by Dr. Evans; swelling of the sheath in males and below the sternum in females was noted in Surra, but I find

Appendix, Disease Charts, &c.

Natural cases ..	A. 39 A. 11 A. 31
Artificial case ..	XV
Record case book entries.	B. 57 B. 70
Illustrative of diagnosis	B. 54
Total records of cases.	7

the latter occurs no more in one sex than in the other, indeed, my most persistent case of it was in a stallion ; œdema of the limbs took place only in a very few of my cases, whereas it was found a frequent feature of Surra. I have never noted " a nasal discharge more or less tinged with yellow " such as was observed by Dr. Evans, nor did I find yellowness of the conjunctiva and petechiæ on it *remaining throughout the attack as reported to him ;* on the contrary, I found the mucous membranes as a rule pale in the stage of least fever, yellowish in that of active parasite invasion, and the petechiæ remarkable for their periodicity, the membranes being absolutely free from them at times, and at other phases of the disease the seat of them ; that dirty colored state of the conjunctiva complicated with the presence of purplish petechiæ which Dr. Evans noted as characteristic of Surra was constantly observed by me especially in the earlier phases of the disorder, but, although this is very significant when present, there are stages of Relapsing Fever when it is not to be found. I have never seen swelling of the sub-maxillary lymphatic glands so intense as to lead to sub-maxillary abscess although this is mentioned as taking place in the Trans-Indus cases ;* moreover I have almost constantly found the other lymph glands enlarged, a feature not noted in Surra. The fever cannot be associated in direct relation with the amount of work done by the animal before he is admitted to hospital ; work or no work, you get the characteristic thermographic tracing from an affected animal. There also is a difference with regard to the appetite of the patient, possibly in direct association with the ulceration of the stomach found so frequently in the Burma mules, but not (as autopsy shows) absolutely dependent on it. Dr. Evans, in his cases, which were not thus complicated, found that, after the primary refusal of food, the animal remained " capricious with regard to the grain," usually taking all the grass. I find that, although the mule may refuse gram when feeling very much out of sorts from height of the fever, he, as a rule, eats his food freely and his fodder excessively ; indeed he will remain eating away at the latter all day long in a surprising manner, varying it occasionally by taking a little earth from the stable floor (central passage) or by consuming his own dung. Sometimes there is extreme greediness for food, exceeding even that of the healthy mule. Also in almost every case where I examined the fresh urine I found it acid, whereas Dr. Evans always found it alkaline in the four cases which he tested. This was an important difference in results upon a question where error would not be likely to creep in. When we consider also that Surra is not specially described in association with deficiency in power of the hind limbs and with marked and regular febrile relapses, we will readily accept the opinion that the diseases are not absolutely identical. Further, in marked contrast with the almost complete absence of definite lesions observable post-mortem in the Trans-Indus cases, are the conditions noticed in Burma, which are numerous, varied, and sometimes extremely marked. We must also note that there are *differences between the Surra parasite as described and the Relapsing Fever organism* as observed by me. There are points in Dr. Evans' description of the parasite in which I am unable to confirm his observations ; thus I should describe their action on the corpuscle as rather one of tearing and pushing or dragging than of sucking. I have not been able to mark any preference for the surface over the edge of the corpuscle, and, although their meeting in pairs lengthwise to form apparently one body has been seen by me, it proved extremely rare. In stained specimens, however, I found it more frequent than I at first supposed. I preferred to examine them in the fresh blood instead of in serum obtained after the blood had coagulated, in which I failed entirely to observe them in the generality of cases without staining. I looked in vain for the " round body tapering in front to a neck " as described by Dr. Evans, for I found that their bodies are thick at one part, from which they *gradually* diminish in size in either direction, terminating in one abruptly to form a blunt end by which they adhere to the corpuscle which part is somewhat straight and rigid, whereas towards the other end they taper in such a manner as to produce a sub-spiral long part which sometimes is seen uncurled and lashed about freely like a whip. This " tail " is certainly slender in relation to the general size of the parasite, and I could neither under my highest available power ($\frac{1}{10}$th), nor from movement of the blood constituents, obtain any indications leading me to infer the presence of a colorless flagellum

* Dr. Evans at Dera Ismail Khan found the swelling of sub-maxillary gland and discharge of mucus from the nose in four cases only out of fifty.

extending from the end of the visible "tail."* I had no reason to suspect the presence of ciliæ on the head; indeed it was quite possible to understand the varied movements of the parasite without any such agency being required. I found not the slightest sign of "two fin-like papillæ on each side—one where the neck commences and one where the tail begins"—although I looked with much care at a specimen where Dr. Evans detected them. Dr. Evans and I also differed as to whether their movement can be called spiral, he maintaining the negative view and I being convinced that their movement is as much of that nature at times as can be expected from organisms with so open a corkscrew shape. I have since seen movements which are certainly spiral, when the weak and more slender organism from dog's blood is trying to drag a mule's corpuscle away from a rouleau, and I find that in dried and stained specimens the organisms retain their subspiral form of the body and markedly spiral form of the tail. However, apart from these minor points open to discussion, I contend that we have ample grounds for the view that Surra and Relapsing Fever of mules are but different manifestations of the same disease. In each inquiry the disease has been studied by an officer specially selected for the duty and able to devote time and attention thoroughly to it, and in the Rangoon investigation there have been the special advantages of Dr. Evans' researches having paved the way and indicated lines of inquiry, of longer time available, and of fewer climatic disagreeables to contend with; *he had no time to observe a single case throughout its natural course.* But there is here something more than would result from differences in powers of observation of workers and in efficiency of appliances; evidently Surra among horses in the Punjab has not quite the same symptoms as Relapsing Fever among mules in British Burma. It is possible that, as climate has a marked effect in generation of the disorder, so too it may influence their manifestations in places differing so much in climate and surroundings as Rangoon and the Trans-Indus territory, and therefore I consider we are justified for the present in considering that they bear about the same relation to one another as Anthrax among horses in India does to the "Horse Sickness" as seen in South Africa, and both to the very rare form of Anthrax seen among horses in Great Britain. Thus we may provisionally speak of the "RELAPSING FEVERS OF EQUINES" of which the British Burma disease is certainly one and "Surra" probably another, always retaining in mind that future researches will almost for certain establish the identity of the two affections; for they agree in a number of important respects, being very like one another in symptoms and in the characters of the parasite found in the blood, also in their communicability from one animal of the affected species to another by inoculation and by ingestion, and to the dog by inoculation and feeding on the flesh of the equine animals which have succumbed to the disease.

7. In some minor respects the Tonghoo disease has been found to differ from that in Rangoon, but there is an element of uncertainty here. At Tonghoo congestion of the brain and its meninges has been noted in an exceptionally large number of cases, and blood extravasations in the cranium pressing on the brain have also been found. Although not prepared to deny that in acute cases (of which there seems to have been a large proportion at Tonghoo) brain congestion is present, I am inclined to consider it rare and to accept its frequency of record at Tonghoo with reserve, for I have ascertained that no proper instruments for opening the cranium were available there, this operation, which requires special care and delicacy of manipulation, being done by a butcher with the aid of an axe. The blood extravasations may have been caused in this process, or have resulted from blows given to the head by the animal when on the ground "dying hard," or possibly be the coagulated contents of the sinuses of the *dura mater*, the arrangement of which parts in the mule is different from that in man and would hardly be familiar to a medical officer who had not specially read up veterinary anatomy. Again the diagnosis of fatal cases at Tonghoo was not always certain, and there may have been cases of true Apoplexy or Anthrax here and there intermingled with those of Relapsing Fever.

8. In comparison of the mule disease with Relapsing or "Famine Fever" of man, we find that there are a number of points of resemblance, but that the differences also

* These differences between the parasite as described by Dr. Evans and myself must be considered as possibly the result of the fact that the descriptions of the same thing observed under the microscope by independent observers seldom agree. This is simply the result of "many men having many minds," and of all microscopes not being alike in definition and penetration.

are very marked. Relapsing Fever of man is a rather rare disorder and is described as occurring in direct and immediate association with such debilitating influences as famines and imperfect nutrition. The blood contains spirilla, which, as Murchison (*Path. Trans.*, 1875, p. 317,) points out, disappear from the blood before the crisis, are absent during the intermissions and re-appear with the relapse, phenomena exactly corresponding to those noted in the mules' disease. Vandyke Carter of Bombay has conveyed relapsing fever from man to monkeys by inoculation with but six failures in twenty-one trials (*Med. Chir. Trans.*, 1880, p. 125). It has been observed that men suffer more than women (3 : 2), whereas I find that females are most liable, but not markedly so. It is most common in adolescence and early manhood, but the mule disease seems to affect animals of all ages indiscriminately. The disease of man is attributed to the following as the principal causes : scarcity of food, overcrowding, want of cleanliness, and *contagion*. I could obtain no evidence of the latter, and with regard to the former, some of my mules which died were fat; none of them are specially crowded, and their lives and persons are kept decidedly cleaner than those of the average mule. The blood, in addition to the parasites, has been noted as containing "large, colorless transparent cells," often two to four times as large as the white corpsuscles; these I have generally noted *at a certain stage* of the fever in the mules as "gelatinoid, decolorised, red corpuscles," which sufficiently expresses my views as to their nature. It seems strange to me to read in accounts of the human disease of "even a *fourth* relapse," whereas in the mule we get even a *seventeenth* in some cases; also the low mortality in man is in striking contrast with the invariably fatal result in equine patients. Season has little effect although the disease of man prevails most in winter. Relapsing fever also occurs to an extent in mules at all seasons, and specially attracts attention as an enzootic in winter and late autumn. The spirillum of man (*Spirochæte Obermeieri*) seems larger, more uniform in diameter, more markedly spiral, and less ferocious than that of the mule, which I had termed *Spirochæte Evansi* after its discoverer, but I find that he is not willing to accept the view that it is a spiral bacterium, a matter which I shall have to enter into detail about later on. I may here remark that I have never studied Relapsing Fever of man or its organism practically, and that I am in the main indebted to Quain's Dictionary of Medicine for the above facts about them, the articles being by Grimshaw and Bastian. Fever has not been specially prevalent among the transport drivers who live in lofts above the mules, nor have any cases of apparent communication of the disease from the mules to men constantly handling them occurred. I have tried inoculation of a monkey with marked success. Thus we are in a position to compare the human and mule disease on a common ground by contrasting Carter's cases of monkey disease with mine.

9. Manifestly it is not possible to verify by experiment its communicability to man, for the disease conveyed by inoculation might prove fatal after causing much disturbance to the system occurring in paroxysms extending over a long time. The following circumstances, trivial in themselves, may be alluded to as possibly links in the chain of evidence on this point. Veterinary Surgeon (First-class) Frost, when attending mules affected with this disorder, became ill; he suffered from pain in the left side and used to say he had caught ulceration of the stomach from the mules; he recovered only when he went away to district work. I also, on arrival and having attended the mules for a short time, had pain in my left side, which especially rendered laughing acutely painful; this, with an accompanying constant hunger, persisted until my trip to Tonghoo. I was examined by a doctor because I suspected pleurisy, but none was present. Subsequently, when collecting specimens of blood from one of the affected animals, I made a minute puncture accidentally on my hand; in a few days it became a star-shaped congested spot and later some fluid collected beneath the epidermis, but these phenomena ceased after remarkably resembling unsuccessful inoculation of an insusceptible subject. My monkey bit me on one occasion. The wound suppurated freely for several days, but has now healed, and I feel no ill effects from the accident. We may conclude that the British Burma disease is of the same class of disorders as "Famine Fever" of man, and that they are probably *not* exact pathological equivalents, although we require more evidence on this point.

10. In the class "Relapsing Fevers" we must also include those which have been observed and induced by Drs. Vandyke Carter and Evans and myself. Monkeys have

suffered from the affection induced by inoculation with the blood of human patients. Dr. Evans describes Surra in the camel, called also Phipri. It is considered the third stage of a progressive anæmia, in which dropsy of the abdomen and legs and rapid wasting occur. Recovery is almost hopeless, but, when it does take place, the animal remains useless for two years, its urine becoming high-colored during the hot season and natural during the cold. The usual duration of the attack is from one week to two or three months; it seldom lasts four months. The parasite lives longer by several hours in the blood of the camel than in that of the horse after it has been drawn. I find that *the Burma disease as induced in the dog* proved fatal after inducing symptoms remarkably resembling those exhibited by the mules, but with peculiar swelling of the lymph glands on either side of the face. I found no ulceration of the stomach after death (but cannot be quite sure on this point because the animal died during my absence and was much decomposed before I examined its carcass). Carter's attempts to communicate the disease of man to dogs proved unsuccessful. The parasite in the dog's blood seems more feeble than that in the mule's; it leaves the large corpuscles of the dog to tackle those of the mule, but its efforts to detach the latter from their rouleaux seem futile and ineffectual, although its movements become vigorous and markedly spiral in the effort. It seems more uniform in diameter throughout its length than that of the mule's blood. When transferred into a drop of ruminant's blood, it works its way, without the slightest difficulty, among the small red corpuscles, but never has been seen by me to touch one of them. I have seen one parasite return again and again to a small group of corpuscles, shaking each of them in the intervals between its short movements to and fro and disturbing others by violent lashings of its tail. My inoculations of a bullock with blood containing numerous and active parasites from a mule have been followed by hard swelling at and around the seat of inoculation, persisting for many days but without appreciable systemic disorder. A goat too resisted inoculation, but after intraperitoneal injection fell away rapidly in spite of excessive appetite. After death its spleen was found to be enlarged and somewhat indurated. Thus the result of this experiment was dubious. Altogether it is probable that the Indian ox and the goat enjoy immunity from Relapsing Fever.*

11. With the determination of the true nature of the British Burma disease arises a question of nomenclature. There cannot be the slightest objection to the designation "Equine Relapsing Fever" as applied to this disorder in future scientific notice of it, for it affects naturally both mules and ponies, and the thermographic tracings leave no doubt as to its being a true relapsing fever. Surra also is an equine relapsing fever in all probability, but Dr. Evans' records of cases are not sufficiently long to make this absolutely clear. He has since informed me that he had been inclined to consider the Punjab disease somewhat periodic, but the length of time available to him for the Trans-Indus inquiry did not suffice to enable him to determine the true relapsing character of the fever† (if it was so), and he considered Surra a disease different in some important respects from any other that has ever been reported on in man or beast. It will be observed that I consider the Burman disease at any rate as a close pathological ally of relapsing fever in man.

12. There are some equine diseases which ought to be examined in comparison with Relapsing Fever with a view to prevention of confusion in diagnosis in the future, such as we have reason to believe has taken place in the past. There is now no excuse for such an error, as Relapsing Fever can be diagnosed with *absolute certainty* if scientific appliances be available. Anthrax and Relapsing Fever agree in their enzootic character, in that they are due to organisms of minute size, in that they are extremely fatal, and in that they, so far as we at present know, are incurable. They are both capable of affecting animals of various species and give rise to general lesions, petechiæ of membranes, and so on, because the blood is the seat of disorder. The very high internal temperature, purple blood extravasations on the conjunctiva, sometimes diffused congestion of the bowels, also the occurrence of gelatinous deposits in various parts of the body, and occasional extravasations in the heart valves are common to

* Although Dr. Evans found camels suffering from Surra he failed to find any evidence to show that horned cattle suffered from it.

† In his report on "Surra" Dr. Evans recorded his observations in both the dog and the horse "that the parasites come and go in successive broods."

Anthrax, Relapsing Fever, and other blood diseases. It is fortunate that with such marked features of similarity we have also marked points of difference. In Anthrax the blood contains bacilli, in Relapsing Fever spirilla. In Anthrax the temperature is persistently high; in Relapsing Fever it is alternately high and low. In nine cases out of ten Anthrax is acute; in a similar proportion Relapsing Fever is of long duration. Although the differences between the two disorders are amply sufficient for diagnosis, yet there is a sufficient family likeness between these two parasitic affections to determine that they shall be studied by the same methods and to indicate that the lines of research followed and facts established in this investigation of Relapsing Fever may in the future suggest new lines of inquiry in cases of Anthrax outbreaks. The disease of the horse known as KAMRI has long been regarded as of frequent occurrence in Burma; it is possible that many cases recorded as such have been Relapsing Fever. True Kamri is a nervous disorder, having its seat in the spinal cord (unless we may accept the latest researches on the subject—those of Officiating Inspecting Veterinary Surgeon Poyser); it is characterized by a sudden or gradual paraplegia persisting apparently as a local disorder and tending rather to indefinite chronicity than to a fatal result. We never see an animal affected with Relapsing Fever sitting on his powerless hind quarters fighting violently with his fore limbs; we never see him, in trotting, roll his hind limbs in the manner distinctive of his being "gone in the loins." The loss of power in the fever patient is *general;* his staggering with the hind limbs is not from paralysis, but from sheer weakness, imperfect nutrition of their muscles on account of plugging of the blood-vessels, and impediment to their free action by the gelatinous effusion between them. Altogether it is evident that, although the two diseases resemble one another to an extent sufficient to induce some confusion in diagnosis, yet the differences are well marked and no qualified observer would commit such an error.

13. The spirillum of Relapsing Fever in man very closely, in its form and the character of its movements, resembles the *spirillum plicatilis*, which was first described by Ehrenberg, and is of common occurrence in stagnant water. This at once suggests the possibility of the two organisms being identical species and is pregnant with suggestions as to the etiology of Relapsing Fever. I have seen bodies somewhat like certain phases of the relapsing fever organism in my microscopical examinations of the stomach contents and of scrapings from the teeth of patients, as well as in the *débris* from mouth ulcers, but these bodies were so mixed with minute particles of food, also *bacilli*, *bacteria*, *infusorians*, and other low forms of life, that I was unable to derive any special information from examination of them as regards their bearings on the disease which I was studying. Microscopical examinations of water and marsh soil also gave no clue to the source whence the parasite gained entry into the organism of the mule. Examination of the air also gave negative results.

14. In seeking the cause of any disorder, we may advantageously work in two ways: Firstly, we may take all conditions, in so far as we can ascertain them, which have been acting on the affected animals and examine each in turn, considering it guilty of having generated the disorder, until proved otherwise or its harmlessness be established; or, secondly, we may, from an examination of the symptoms and post-mortem conditions, determine the pathology of the disease and then seek for all causes which might bring about such disorder and in our experience of other cases are known to have done so. In my examination of relapsing fever of mules, I at first could adopt the former method solely, but am now able to supplement my conclusions by the latter, since we may reasonably feel assured that the parasite is the actual and immediate cause of the disease—the *materies morbi*. We need not here enter further into the inquiry as to whether its agency is direct (as is most probable) or indirect through some excretum or other chemical matter or ferment thrown out by it into the blood. Suffice it that the parasite must enter the system and it must have a source whence it gains entry into the circulatory system. We know that active spirilloid organisms introduced into the alimentary canal generate parasites in the blood, and that, when thrown into the subcutaneous areolar interspaces they are also able, in a still shorter time, to produce a like effect. Experimental inquiry has also shown us that blood in which no parasites are detectable under ordinary powers in their ordinary form will, after a still longer time, generate relapsing fever. It seems that once in the blood the organisms do not leave it, but in the low stages of fever are still present

perhaps as free motile nuclei, such as are noticeable in the majority of specimens and distinguishable from the free nuclei of healthy blood only by a sort of purpose in their movements, but more probably as in a quiescent form so that they are detectable only on aniline staining.* Whether naturally the organism enters the system in the spirilloid forms, or as a micrococcus, or as an unrecognizable germ, we cannot say, and we are equally without guiding evidence as to whether its entry is effected by exercise of its eel-like activity or by its use of a special boring organ too minute for detection with ordinary microscopical powers. The size of spirilloids is not sufficiently great to exclude the possibility of their passing through the walls of normal capillaries, as do the leucocytes, but the passage may be to an extent forced, for we have evidence that the blunt extremity or "head" must be armed with some mechanism, if only a sucker, to enable it to obtain a grip on the red corpuscle; the disintegration of the latter would lead us to infer that the arrangement is something more formidable than a sucker. We know that the parasites, small though they be, can exercise a great deal of strength at the expense of the corpuscles. The amount of force expended is often greater than that which is exerted in adhesion between two corpuscles in a rouleau; it is sufficient to enable the parasites to drag the corpuscles about, shake them, distort them into various shapes, and it suffices to enable one parasite to drag two corpuscles about with ease and rapidity, also to shake a long rouleau, but not to detach it from the mesh-work to which it belongs. This strength is ordinarily exercised in locomotion, in traction, in changes of form, and in pushing a way among the corpuscles. The energy, strength, and perseverance shown by the parasites in making their way through an aggregated mass of red corpuscles should more than suffice to procure them an entry into living capillaries. The parasites in their spirilloid form, much more so in their possible germ phase of development, would find no difficulty, under the ordinary life conditions of mules, in obtaining access to the mucous membranes of the alimentary and respiratory systems; entry *viâ* the skin is not impossible especially in the presence of open wounds, blistered surfaces, &c., but we have evidence against its ordinary occurrence. The air, water, and food must therefore be looked on with suspicion as vehicles for conveyance of the organism which generates relapsing fever into the system of the victim.

15. But not only is it necessary that the organism shall gain access to the system, it is also essential that the individual invaded be susceptible to attack in order that the parasite may obtain a fair footing from which to extend its ravages. My inoculations of the bullock have thus far been valuable, in that they have shown that local irritant action by the parasite need not necessarily be followed by systemic infection. That the opposite is not the case is shown by my inoculation of the pony in which both local and general symptoms were the result. In many diseases individual peculiarities have a marked effect on the "taking" or rejection of the specific virus, but in equine patients my administrations of relapsing fever parasites have been *invariably* followed by success in communication of the disease. Animals in fat condition have succumbed as rapidly (perhaps more so) as the weak and emaciated; old and young, male and female, horse and mule, have become affected. Thus in this disease, as in anthrax, but to a less degree, predisposing causes sink into insignificance from a practical point of view in comparison with the determining influences, viz., those which have a direct influence in conveyance of the pathogenic organisms into the system, for we can alter the latter, but the former are beyond our control to a great degree, or, however much we may modify them, we cannot render the system absolutely insusceptible to invasion by any means we are yet acquainted with. Yet we cannot altogether afford to ignore predispositions on the part of the animal, and so shall have something to say about age, color, breed, and conformation after we have drawn attention to a few other matters to be taken into consideration in studying the causes of disorder. It is evident that the same cause operating on different individuals does not necessarily produce the same effect. Thus many animals exposed to a specific influence may resist it, whereas others succumb. Therefore we must not argue that, because a supposed cause is not in every case followed by an attack, it is not a true cause, nor must we, on the other hand, consider that, because a supposed cause fails to generate disease, the animal is

* In his report on "Surra" Dr. Evans says:—"The blood parasites do not disappear from the blood absolutely when it becomes difficult to find one in a drop of blood; when one brood or generation dies there are ova or spores left for the development of another brood."

resistent, for the cause may not be a true one. It is evident that there may be different determining influences at work in different cases; we prove this by our coarse experimental methods even, for in one instance the disease is conveyed by puncture of the skin and injection of blood into the subcutaneous tissue (a very unnatural determining influence unless the disease is ordinarily conveyed by mosquito punctures or leech bites), and in another instance a large number of parasites taken in with food generate the disorder, reproducing it in probably an exaggerated copy of the natural ingestion of spirilloids with food and water. From this we learn that, although we are right in considering the parasite the sole actual and immediate cause, *we would err if we stated that any one specified determining influence is at work in all cases.* Some cases may be due to food, others to water, and others to air introduced into the respiratory organs. We have no evidence opposed to the view that mosquito and leech bites and visits of flies to wounds are in some cases the determining influences by which spirilloids are brought into the system. Several of these influences may be simultaneously operative on an animal and so the actual way in which the attack was brought on in any particular case obscured; this was observable in the case of the dog "Motee." She received blood subcutaneously and ate flesh of animals which died from the fever; it is, accordingly, uncertain from which source she obtained her relapsing fever.

16. To trace out a cause of attack we must decide when it operated, that is, how long it exerted its influence before it induced the attack, and when the attack actually commenced. Unfortunately these points are both doubtful in the disease which I have been studying; the period of incubation varies with the channel through which the contagium enters the system and with the condition of the contagium at time of administration. My experiments amply prove this: incubation was longer after gastric ingestion than after subcutaneous injection of the virus, and longer still when blood with no parasites detectable was used instead of that in which they were numerous and active. Incubation in the dog was much longer than in the mule, and in the pony (and especially the monkey) it was very short. Again, in examining the outbreak as a whole, we labour under the very serious difficulty that we do not generally know when the cases commenced; this is due to imperfect diagnosis, owing to causes which I have elsewhere indicated. It is singularly unfortunate because it compels me to consider cases in relation to possible causes only by their date of fatality. This is manifestly a much less satisfactory datum than the date of attack, for the duration of cases varies very much. I have been obliged, for want of a more satisfactory method, to strike an average duration for cases and deduct it from the date of death, and in this way get a possible date of attack.

17. The fact that we have no positive evidence that the spirilloids leave the blood of affected mules is of much importance, as it may be associated with the contagiousness and curability of relapsing fever in man as contrasted with the strong evidence of non-contagiousness and non-curability of the mule disease. In man the influence of contagion can be traced in the manner of spread of the disease into unaffected districts, but the evidence which we have to hand about the mule disease is strongly opposed to the theory of its communicability by contagion; no contagious influence can be traced in its manner of spread among mules in one stable or working together. In the Somersetshire lines, D troop mules were often intermingled with those of A, B, and C troops, and had to pass through their lines daily, but, until their movement into the other stables, not one D troop mule was affected. Further, lame and non-febrile sick animals have stood in lines containing many fever patients and did not become affected although many of them had open surgical wounds at the time. They have been returned to uninvaded lines from the sick lines on recovery and have not carried the disease. There are two incidents of the outbreaks which deserve mention here. While the disease was in the Rangoon depôt lines three ponies were started (on 28th September 1884) from there to Tonghoo. One died on the way at Shwaygyin (cause unknown), another at Tonghoo shortly after arrival, the third remains alive. One mule succumbed at Tonghoo previously to their arrival and one in the third month after their arrival, so, and, especially, considering the obscurity of cause of death in two ponies, we have not here material for tracing the Tonghoo outbreak (which took place seven months later) to contagion from Rangoon. Twenty-four mules were sent from the depôt to Thayetmyo on 21st December 1883; of these, one died at Letpadan

on the way up with perforation of the stomach, the result of ulceration, and one at Nyung Hla Kyoun of "anthrax." On arrival six were sick, but seem to have recovered. These animals came from an infested locality and seem to have had the disease, and yet they did not convey it to those at Thayetmyo. On 24th March 1884 four mules were sent from the depôt to Thayetmyo and two to Tonghoo; in the former place the disease has not broken out, in the latter it has proved severe, but was commencing before the arrival of these mules. The depôt ponies which died in the 1883-84 outbreak arrived from Thayetmyo, a disease-free place, on 19th May 1883. Thus we see that the prevalence of the disease is not in direct ratio with interchange of animals from disease centres. This non-contagiousness of the mule disease may be the result of the organisms not being expelled from the system, but remaining in it until they induce a fatal result. Whereas in man profuse perspiration occurs as a critical evacuation in the attacks, no such phenomenon has been observed in the lower animals. I have not experimented with saliva of mules, which contains organisms a little like certain forms of spirilla, but have given stomach contents and fluid from the bowels through the mouth without effect. I thought it possible that the gastric ulcerations and the circumscribed congestions of the bowels were associated with elimination of parasites from the system through these organs, but the result has proved negative. Thus we have strong evidence that the disease is not communicable by contagion or infection; we are certain that it may be given by inoculation with, or ingestion of, the blood.

18. Although predisposing causes are subordinate in importance in this disease, we must give them some attention. *Breed and conformation* seem to exercise some influence. Captain Johnstone expresses the non-professional idea on this subject when he says "the small thick-set mules in general appear to enjoy almost complete immunity." This was noted at Rangoon, where the mules most like donkeys were least often affected, but, although patent to non-professional observers, this fact has limited practical value, for not only all sorts of mules, but even ponies of the country, have succumbed to the Burma disease.

19. With regard to *size*, it is a matter of constant remark that the larger mules suffer more from the fever than the smaller ones. This may be due to one or more of three causes: (*a*) The big mules are most used and have the hardest work. (*b*) Although allowed by Government more food than smaller ones, they, in feeding off the ground or out of unpartitioned mangers, generally get actually a smaller amount than their less phlegmatic small companions. Also in the distribution of gram sufficient care is not always exercised or practicable for securing to each large mule a large feed. I notice also that, as concerns fodder, the mules are all treated alike, the allowance for three fellow-workers being placed in front of the three animals and the sharpest of them obtaining an unduly large share, often, by the free use of hoofs and teeth, keeping the others away until all the tit-bits are consumed. It is evident also that, when picketed in the open, large animals will suffer most, especially they will miss their large ration when it becomes necessary to evenly distribute insufficient food or to allow animals entirely to shift for themselves. (*c*) The influence of breed and conformation may tell here, the largest mules being those which retain most of the caballine characters of their progenitors, the small ones the asinine. Although this more frequent attack of large mules has been commented on especially in connection with their use for puckal work, it certainly cannot be considered a specially marked feature of the Rangoon outbreak, in which I find 24 mules out of 47 cases were 13 hands and upwards, only 9 of them being 13·2 and upwards (*i.e.*, first-class mules). In the 3rd Native Infantry at Tonghoo of 57 cases 22 are returned as first-class mules, 32 as second class, and 3 as "not specified." Thus calculating the percentage of losses on the mules of each class mustered on the strength, we cannot consider this a marked feature of the disease, although any *special* tendency of large mules can be fully explained in the above manner.

20. *Age* does not seem to influence liability, but veteran mules have succumbed in considerable numbers. The Tonghoo returns of the 3rd Native Infantry show most deaths at the ages of 12, 18, and 20, but a fair distribution from 8 years old up to 21 years; however we have already noted the suspicious exactitude of this return as regards ages. In the Somersetshire Regiment most of the fatal cases were mules of early adult age, but both old and young also fell victims. Old animals, from the

defective state of their teeth, have been least able to utilize to the full such nutritive support as is contained in unhusked rice, and so would be weakened and rendered (perhaps) more liable to invasion by spirilloids. *Sex* influence has been marked in the 3rd Native Infantry at Tonghoo, there having been 39 cases in females against 15 in males. In Rangoon, however, mares and geldings have been affected in about equal proportions, and several stallions have died. Evidently sex has, as a rule, but little to do with development of relapsing fever in mules. *Color*, too, will not require much special notice. It is certain that in outbreaks of this disorder but few grays are left unaffected. This is difficult to explain except by the known laxity of fibre in animals of this color as compared with darker ones, also their admitted special liability to lymphatic and asthenic disorders. It is evident that no special color secures exemption from relapsing fever. In the Somersetshire Regiment I find a loss of 16 grays and 1 white against 23 browns and 5 bays—a very large proportion, when we consider the relative frequency of the lighter and darker colors. In the 3rd Native Infantry "red" and "black" mules and whites suffered most and about in the same proportion (25 per cent. of total fatalities), but everywhere mules of almost all the numerous and varied colors presented by this hybrid race died. The preponderance of fatalities among grays seems to be well established. *Physique.*—The influence of natural habit of body is obscured by the fact that the majority of patients when first observed to be affected are in poor condition as a result of undiagnosed disease. But one thing is very certain, and it materially assisted in leading to the conclusion that this disease is not merely pernicious anæmia, namely, that animals with much superfluous fat, plentiful reserves of nutriment, succumb especially to the acute form of the disorder; indeed it almost seems as if, like Anthrax, it carries off all the best mules, and that apparent strength and vigour of constitution invite attacks by the spirilloid. It may be that animals which die from the disorder when fat are not really "in condition," that, although looking comfortable and well, they are not in that state best rendering them fit for real hard work, and, from the debilitating effects of fatty heart and liver, cannot resist the severity of the fever.

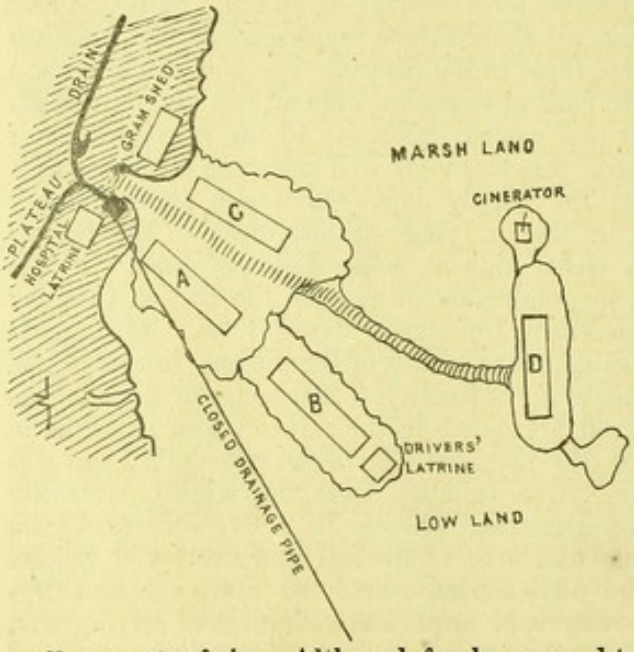
21. It is now necessary that we utilize such exact records as are available with a view to the examination of possible causes in their relations to the outbreaks as determining influences, and we shall thus arrive at some specially significant facts about the attacks which will show how certain influences have been particularly operative and are to be avoided in the future with a view to prevention. *Work* may by its excessive or defective amount predispose to the disease, severity of work must lessen the animal's constitutional resisting powers, such as, for instance, when a mule, after an exhausting day's work, has to rest during the night in a malarious place; deficiency of work has somewhat the same result by lessening the general tone of the animal's system. Also the nature of work may be such as to bring the animal within the sphere of disease influence. Much stress has, as we have seen, been laid on the effects of puckali work, because it was observed that almost all the large mules used for this purpose in the Somersetshire Regiment died. But these mules were the most predisposed as being large, horse-like, overworked, probably (as being at special work) irregularly fed, excessively exposed to cold and moisture, and almost certain to drink from the tanks from which they were bringing water for fire-engine, washing, and other purposes although it was forbidden to be drunk by mules or men. In spite of the strictest injunctions and, in some cases of neglect which were detected, severe punishment, it would obviously be impossible for a driver busy in filling a puckal to prevent his mule taking an occasional mouthful from water in which it was standing. When it is considered that for a long time the mules were working with camel puckals, each when full weighing 195 lb., and that the 390 lb. thus placed on the animal had to be carried up the steep path leading from the tanks to the men's lines for many hours in the day and that the driver would not be likely to give himself more work than he could help by only half filling these puckals each journey so as to give only the regulation load, we must conclude that hard work and exposure especially predisposed the puckal mules, although the theory of water as a cause derives a certain amount of support from the fact of these animals suffering most. In the supply depôt in 1883-84, when the disease prevailed so much among the mules in general, none of them were used for puckal work except one which remained unaffected. In Tonghoo also the influence

of puckal work as a cause of the disease was not noted. We may, therefore, fairly conclude that puckal work, *as such*, is only an indirect cause, and may safely continue to be performed by the mules, provided the animals be not overworked at it and be as much as possible prevented drinking water from the tanks.

22. *Exposure to weather* might predispose to attacks by lessening the constitutional power or determine invasion by the parasites as implying plentiful access to malaria, mud, stagnant water, bad food, and so on. I am led to consider exposure a very powerful factor in production of relapsing fever. In Tonghoo especially the mules working to and from Thandoun fell victims to the disease, for this work implied an exhausting march first through terai, then up a very steep hill for about nine miles, and finally insufficient shelter, food, and general care on arrival at Thandoun. In the Rangoon Supply Depôt outbreak the disease was preceded by a period of exposure during the height of the monsoon on the *glacis* of the pagoda and at Kokine. Early in 1884 the Somersetshire mules were sent out into the districts to collect straw, and they had hard work and a rough time of it in so far as I can make out. I have evidence of some mules dying with ulceration of the stomach when on the line of march to the frontier out-stations, but they possibly were suffering from the earlier stages of the disease when they started. Death of two mules exposed on Anthrax graves and of the hard-worked and exposed mules used for puckal work in the Somersetshire lines are evidences as to the effects of exposure, especially when combined with overwork. We may say that in every case of outbreak exposure, with its usual concomitants,—irregular food and water-supply,—has been operative previously. On the other hand, with regard to *overcrowding* as a cause, we cannot admit that it has been operative among the transport mules, which are exceptionally well housed and have lots of room. And yet in this as in other enzootics (influenza, strangles, and so on) we must admit that the aggregation of animals has some influence, for we have no evidence that the disorder has prevailed as an enzootic among ponies kept in private stables here, and we have certain evidence that it has not done so among mules and ponies let out by the Transport on hire to Government officers.

23. Stables, either by being badly placed or in an unhealthy situation, or as being deficient in protective influence on the animals housed in them, might tend to determine the appearance of relapsing fever. I can in almost all the cases which have come under my notice trace the disease to exposure, but I can in addition find distinct evidence that it also may occur from definite local centres or points. It is of great importance in relation to prevention that we should trace the disease to these fomites or local centres in order that we may avoid them in the future. The fact of the disease being enzootic would indicate some local central operative influence in each outbreak, and it has been attempted to point out *that* central influence in the consumption of water from certain tanks, but we have seen that this view will not stand exact inquiry. In the 1883-84 outbreak at Rangoon the supply depôt mules and ponies mainly suffered, only a few cases occurring in the outlying stables, those of the Native Infantry and the British Infantry on either side at some distance away. In the 1884-85 outbreak we have had a great preponderance of cases in the British Infantry lines, a very few in the depôt, none in the Native Infantry or Sapper lines. An important matter to discuss is whether this decline in frequency of the disease from a centre differing each year is due to the influence of air or locality. Advocates of the enzootic wave theory might find here a fairly good argument in their favor, and say that in 1883-84 the disease wave struck against the northern side of the pagoda hill and was diverted mainly to the west towards the Native Infantry lines, but slightly affecting the British Infantry mules in the other direction, whereas in 1884-85 it came from the south or east and thus struck on the east side of the hill and destroyed British Infantry mules, but a few traces of the wave curled round the north side and thus the depôt did not quite escape. We can very well understand that driving mist could be a carrier of minute organisms and be diffused in consequence of the physical conformation of the ground in the manner just mentioned, but we can hardly reconcile with this view of conveyance of the contagium in air the facts that in 1883-84 the sapper mules (thirty-five in number—a sufficiently large body to suffice as a test), although living in a stable close by those of the Native Infantry, escaped ; that D troop

stable of the Somersetshire lines in 1884-85, although the most exposed of four, lost none of its occupants, while the mules were dying rapidly in all the other three (it certainly contained a large proportion of the small mules and none of the first class); that ponies in Rangoon town and cantonment, which could hardly have all escaped the "wave," have not appreciably succumbed; and that *simultaneously* at places so far apart as Rangoon and Tonghoo the disease has been raging. *The balance of evidence is against the wave theory and the influence of air.* In 1883 at Rangoon, certainly, during the main prevalence of disease at the depôt, a north-east wind prevailed, to the effects of which the animals would have been exposed had they been in their lines, and to which many of them were actually for a time exposed on the east *glacis* of the pagoda, but in 1884 it was only after the main fatality had occurred that the wind veered round to the east; hitherto the Somersetshire lines had been rather out of the wind, which was mainly from the south-west, so that they lay to leeward of the pagoda hill. If we judge by the number of admissions instead of fatalities, the evidence of wind as a determining cause is even less marked. At Tonghoo the fatalities occurred mainly when the wind was from the west. Here also the disease seems to have prevailed when the wind was low; evidence from Rangoon tends also to associate high winds with less fatality. Thus sultry weather, with a stagnant state of the air, seems rather to favor development of the disease. At Tonghoo the lines are low-lying, and mist hangs over them after it has cleared away from the greater part of the remainder of the station. When I was there in December it persisted until after 8 o'clock in the morning. The outbreak in the Somersetshire lines affords us a good illustration of a local centre. Thus, out of 55 of the cases, 23 were from A troop (3/7); 17 were from B troop (2/7); 14 from C troop (1/4); and only 1 from D troop (1/55). The predominance in A troop and the practical absence of the disease in D troop seems a significant fact worthy of further investigation. We must first examine the physical geography and geology of the locality to see what special information can be derived from them. The Somersetshire mule lines are situated to E.N.E. of the elevated plateau on which the Shwe-dagon pagoda and the European Infantry lines are built. The plateau attains a very considerable height above the sea level and above that of the alluvial deposit on which Rangoon town and suburbs are situated, and which is continued to east and west of the plateau as rice land, but little above the sea level. The plateau terminates abruptly to north and east, but here and there outcrops of its component laterite form sub-plateaux and small hills projecting from the neighbouring alluvium. On these outcropping prominences are situated the mule lines, which consist in all of four stables, named after the troops which occupy them. A path to them has been formed along a natural drainage channel. To the right of this path are A and B stables, the latter on a projecting spur below the level of the former and between it the native drivers' latrine. C stable is to the left on the highest part of the sub-plateau. The path after a saddle back course terminates at a prominence on which D stable stands almost at right angles as regards its direction with the other three stables. It is practically surrounded by low-lying and marsh land and is always kept fresh and pure (if at times somewhat cold) by its exposure to all currents of air. Although freely exposed to all the vapours arising from the neighbouring marsh land and to driving mist and fog, this stable could never become the seat of stagnation of air; perhaps this was the cause of immunity of animals kept in it. Something may be attributed to good natural drainage and to the mules being of the small thick-set stamp and to the non-commissioned officer of the troop being a specially



careful man. A and B troop stables are almost parallel to a large iron pipe through which the drainage from the plateau is supposed to pass; but, as a matter of fact, during the rains a large amount of water flows from above by the path to the site of A stable; this is to an extent prevented by a receptacle cistern situated between the hospital latrine and the path on the high ground, but the brick drains leading to this are not sufficient and so the water flows over the edge of the plateau. It floods the drains of A stable and has to be constantly baled out by means of buckets from the cisterns in which they end, and this can only be done very imperfectly, so it is to be feared that admixture of animal matter, dung, urine, &c., with the remnant may render these cisterns actual nidus for development of miasma. They are quite unnecessary; increase of the natural slope to a very slight degree and removal of the cisterns would give a very efficient system of surface drainage in this and the other stables, as the natural facilities are great. Why the drivers' latrine should have been placed within a couple of yards of the end of B stable it is difficult to see; it would be much more advantageously situated if on the low land a few yards further on, and it is not always in first-rate order if I may judge from the times when I have inspected it. Its emanations cannot but be prejudicial to the mules, and its receptacles must be carried through the mule lines for disposal. Stables A and B in which the disease has prevailed most are in a direct line between two latrines; this is probably a coincidence merely, but just possibly may be a predisposing cause by its prejudicial influence on the health of the mules. Stable C is almost parallel with A and is out of the direct line of drainage. Almost parallel with stables A, B, and C is on each side a nálá with some brushwood and somewhat tempting to the natives as a latrine, for which purpose it is occasionally used. The two are mainly formed and deepened by the rush of water from the plateau, but that to the south is constantly moist from a stream formed by water trickling from the surface of one of the deeper layers of the laterite of the hill. Altogether we may sum up by saying that there are no glaring sanitary defects to which we can attribute the greater prevalence of the disease in A, B, and C stables, especially in A. There are certain minor defects which will readily admit of remedy, and part of the special prevalence must be attributed to the larger proportion of first-class mules. I am inclined to consider the deleterious influence here also to mainly reside in a stagnation of air. Microscopical examination of the air of stables in search of the cause of disease did not seem a very promising line of research as I arrived in Rangoon when the disease was already on the decline, and probably after any cause operating through the air had ceased. However, considering the possibility of infection, I deemed it right to make some examinations of the air from the sick line stable. This I did by drawing it through a plug of cotton wool in a tube and steeping the latter in pure water for some time without exposure to air. I found no development of spirilloids in the fluid, but of course this method has but little value as being rough in the extreme. In the Bedfordshire lines at Tonghoo I found that the Transport Officer noted that most cases came from one of the two stables. But all the lines at Tonghoo are defective in some important respects. They are of the usual pattern and four in number, arranged in parallel pairs. The disease has proved equally prevalent in both European and Native Infantry lines, and practically the stable conditions are the same. The ground occupied is on the line of drainage from the men's lines; it is intersected by drains, in many parts of which water is liable to stand or to overflow. There must be a great deal of marsh around the stables in wet weather. In near proximity is a jheel, which is a receptacle for filth and bazaar refuse, and into which hungry mules, turned out in numbers to the scanty grass croppings about the lines, would be sure to find their way. Followers' lines are beginning to encroach into close proximity with the mule stables, especially in the case of the Native Infantry at Tonghoo.

24. The stables supplied by Government for the mules are very superior to the average of private stables in Rangoon, but they differ in this important particular that there are no lateral walls. As a rule, private stables are closed on one side, which may or may not have a window opposite each horse's head, and the other side is generally furnished with a split bamboo chick or screen. Thus the animals are protected, not only from dew, but also from driving mist and the moist cold air of early morning, as also from certain winds which are popularly supposed to be "feverish." This closure of stables seems to be an important means of prevention of malarious disease. The private

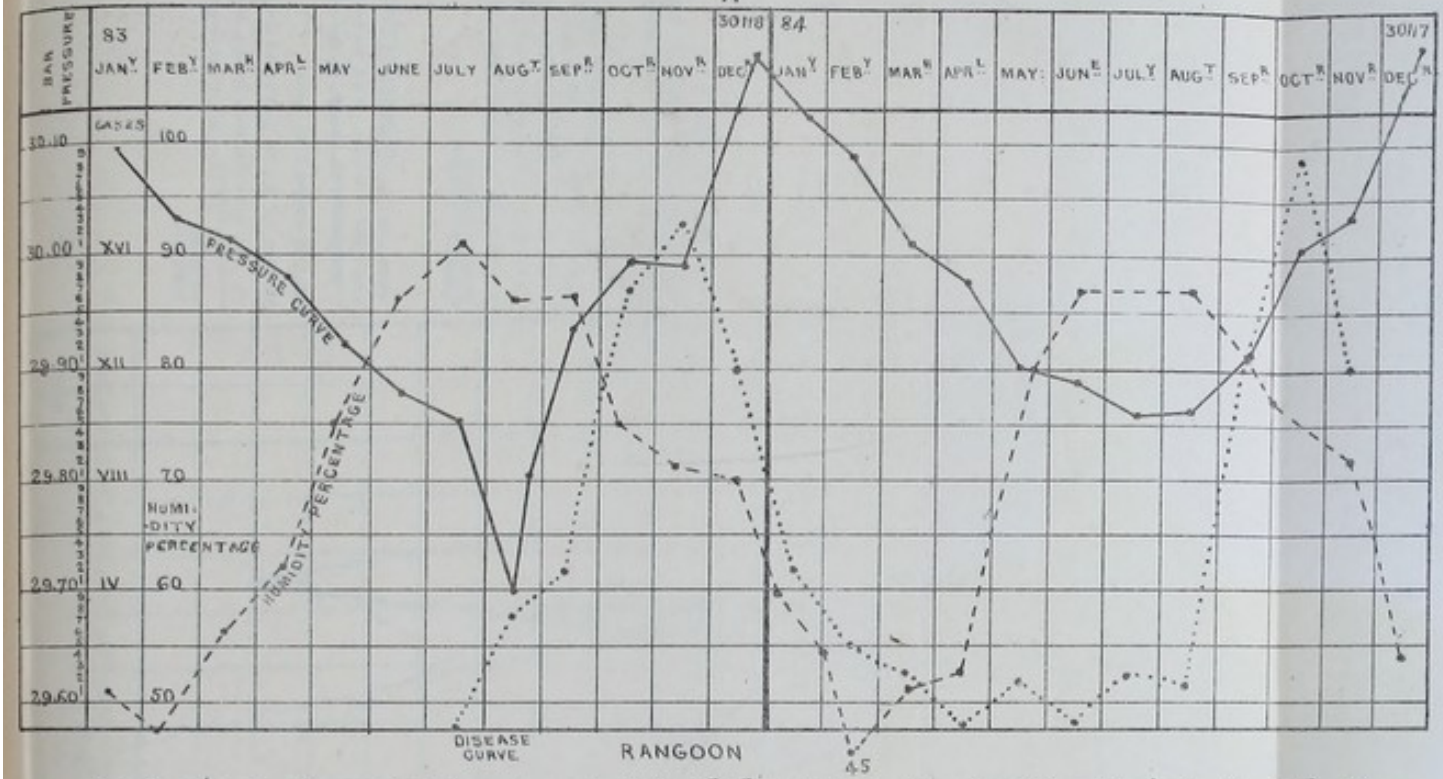
stables have boarded floors raised a little from the surface of the ground under which the earth becomes much contaminated with filth and moisture, whereas the Government stables have generally excellent asphalte floors with fair slope, surface drains, and are generally kept in excellent order. But, although the stables being open at the sides renders them less valuable as a protective means than they might be, we have ample evidence that this defect is not in itself sufficient to generate the disease, in that D stable fo the Somersetshire lines is by far the most exposed and at the same time is the most healthy. The geological formation of the plateau on which the lines at Rangoon stand is laterite ; this bluish laminated formation is somewhat clayey and tends to check percolation of water ; thus on the plateau surface there are marshy patches, and much water hangs there in monsoon time. The A troop mules used to be picketed out here in the early mornings while their stable was being washed. As far as stable management goes, the Government mules both at Tonghoo and Rangoon have been better treated than the majority of private animals in Burma. Related closely to the question of locality in its effects on the disease is the fact that it has not yet been observed at Thayetmyo or at Moulmein, and it is important to consider if those places are intrinsically healthier, especially in relation to fever, than Rangoon and Tonghoo. We must not jump prematurely to conclusions in this matter. Had we been considering it only from last year's experience, we would have included Tonghoo among the stations in British Burma healthy for mules, but we should have been sadly in error. It is necessary to wait for two or three years longer before we decide on the relative healthiness of stations for the mules ; we have not as yet sufficient evidence on which to base an opinion. Moulmein has hitherto been tested with only a very small body of mules, and we have seen that the disease has hitherto occurred only among mules collected in numbers. Thayetmyo has thus far withstood this test ; a large number of mules serving in it have remained healthy ; it is a place differing much in its climate from both Rangoon and Tonghoo. And we must remember that we have a suspicion that some animals sent there sick recovered. The balance of evidence is in favor of the view that Thayetmyo will prove a convenient station and suitable for a large number of mules. We must not forget that the Artillery mules at Tonghoo and the Sapper mules at Rangoon have entirely hitherto escaped the disease.

25. The *influence of season* is worthy of especial attention, because of the fact that, in so far as our experience at present goes, the deaths have specially occurred in autumn and winter—in fact towards the end of the rains and in the cold weather, the latter of which should be the healthiest time for animals. But, when we look more closely into the facts of the case, we find that in most instances the disease is acquired *during the rains*, especially in animals exposed to them, but cases seem to occur occasionally and sporadically at all other times of the year. The advent of the rains in tropical countries is associated with an extensive growth and development of low organisms, especially fungi and their allies, which have been dormant during the drought and heat of the hot weather. Thus, with moisture and heat, germs of various sorts spring into activity, assuming their more active phases of development ; then, in the intervals between falls of rain each small pool becomes the centre of development of minute life, and miasm arises from marsh, jheel, and stagnant tank ; pestiferous mists prevail and active organic matter is washed down into tanks supplying drinking water. At the same time the animal constitution, enfeebled by prolonged existence in tropical heat and dryness, is rendered deficient in tone and resisting power by the sudden occurrence of moisture in excess in the atmosphere. With such fertile sources of fever germs we cannot be surprised if under-fed mules exposed in the open in the jungle during monsoon time succumb to disease. We must now examine the several elements of climate and see if we can find that any of them show marked and definite relations to the outbreaks :—

(*a*) *Barometric pressure* in the Rangoon outbreaks has been in a curiously direct relation to the number of fatal cases. This can be at once seen on reference to the Graphic Record No. 2 appended. The rise in disease curve (as deduced from the fatalities) is in direct association with the rise in barometric pressure. We obtain similar deductions from an examination of the conditions at Tonghoo.

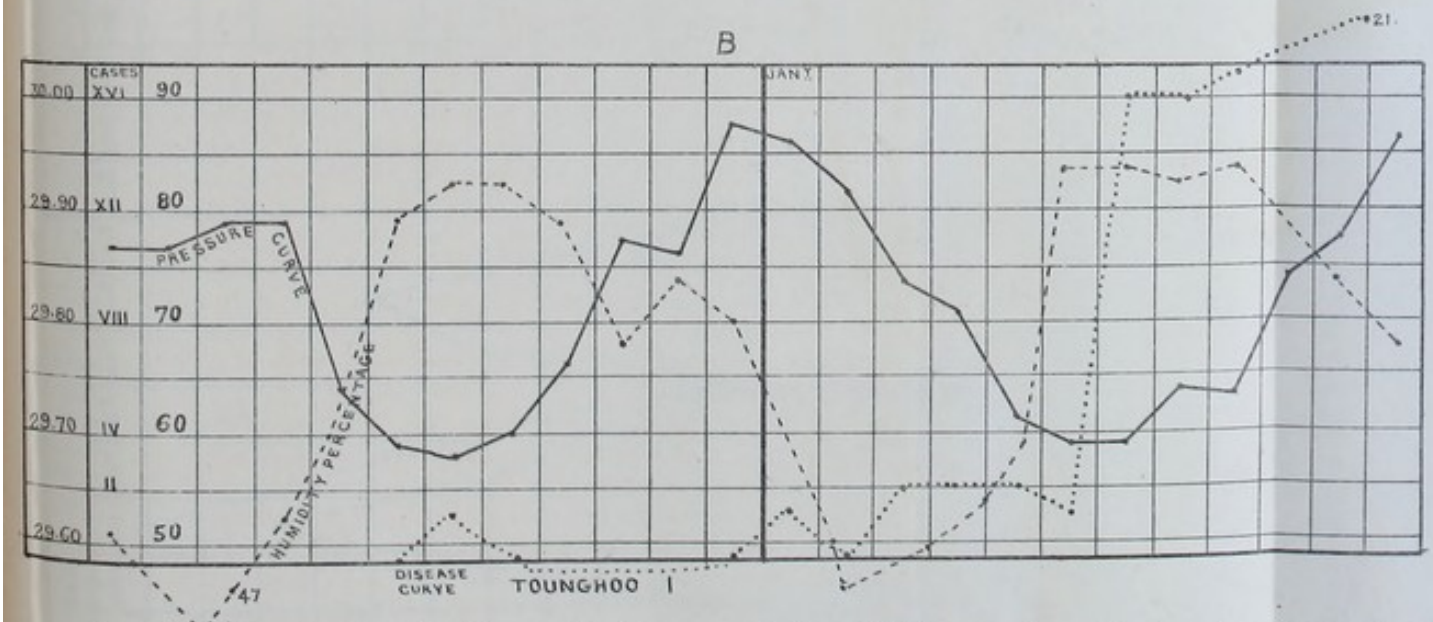
ILLUSTRATING RELATIONS OF RELAPSING FEVER OUTBREAKS TO BAROMETRIC PRESSURE & HUMIDITY.

A



Conclusions – Barometric pressure. A rise with disease curve, one month anterior to it and lasting one month longer in 1883, then declining less gradually. – 1884. Outbreak also associated with pressure rise. Humidity. Curves remarkably like those of mortality 3/4 months anterior to them.

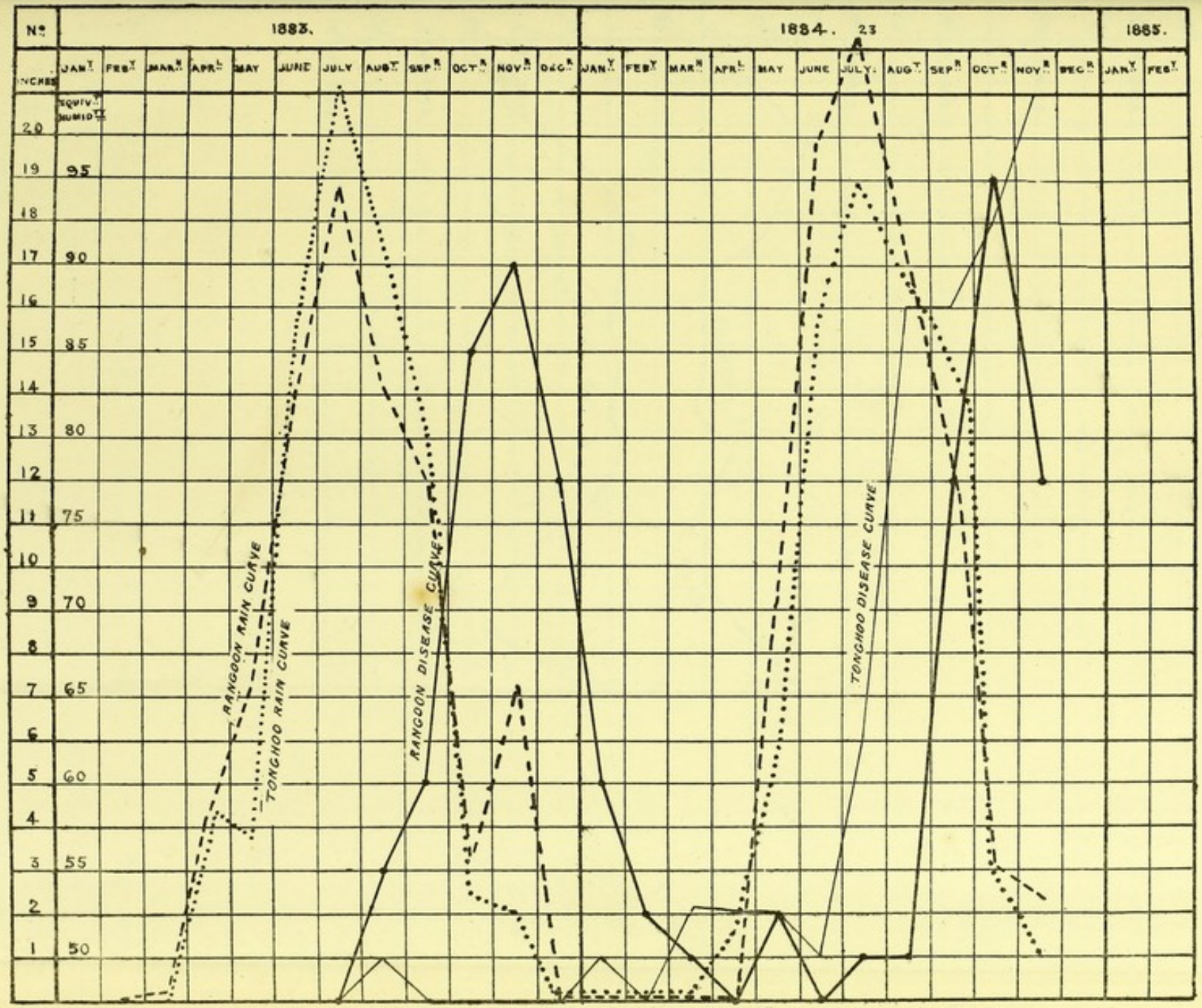
B



Conclusions. In 1884 the Barometric pressure rises with disease Curve but much more gradually. The rise of the disease Curve is associated with rise in that of Humidity percentage and the maximum of the latter is attained earlier than that of the former.

Therefore the disease fatality seems to be in direct association with a rise in barometric pressure and the disease invasion probably with increased atmospheric humidity.

ON TRANSFER BY C. SARAVANAVALOO MOO.ST AND LITHO.D BY J. SHARES GOV.T LITHO.L DEP.T CHEPAUK AUGUST 1885.



ON TRANSFER BY C. SARAVANAVALOO MOD.LT AND LITHO.D BY R. J. SALDREY GOV.T LITHO.L DEP.T CHEPAUK MADRAS JUNE 1885.

PRECIS.

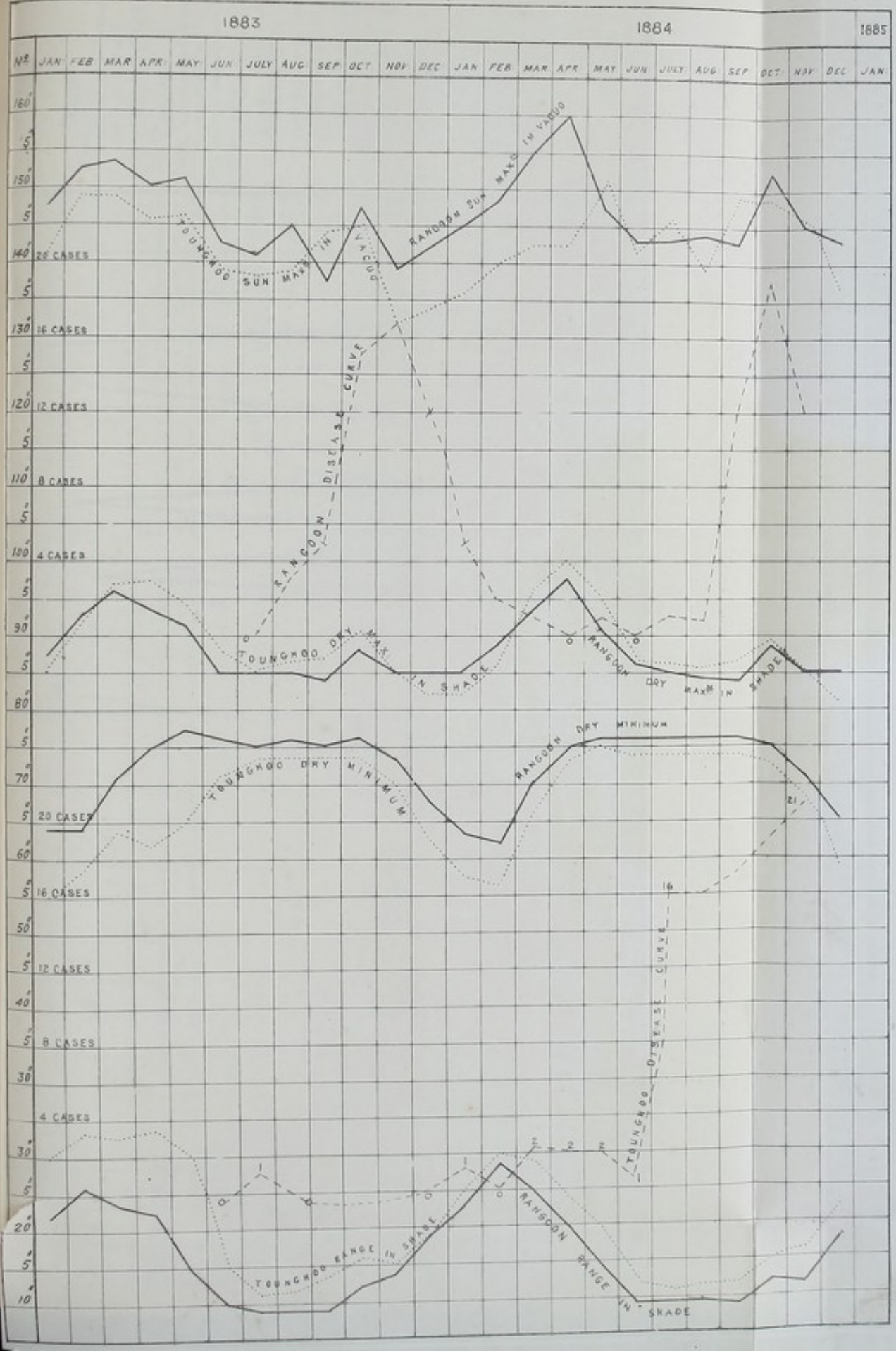
_____ CURVE PARALLEL TO THAT OF THE DISEASE BUT ABOUT 4 MOS. BEFORE IT. No. OF CASES NOT IN PROP.N TO THE AMOUNT OF RAIN.

TONGHOO RAIN CURVE OF 1884 CONFIRMS THE CONCLUSIONS TO BE DERIVED FROM THAT OF RANGOON AS FOLLOWS.

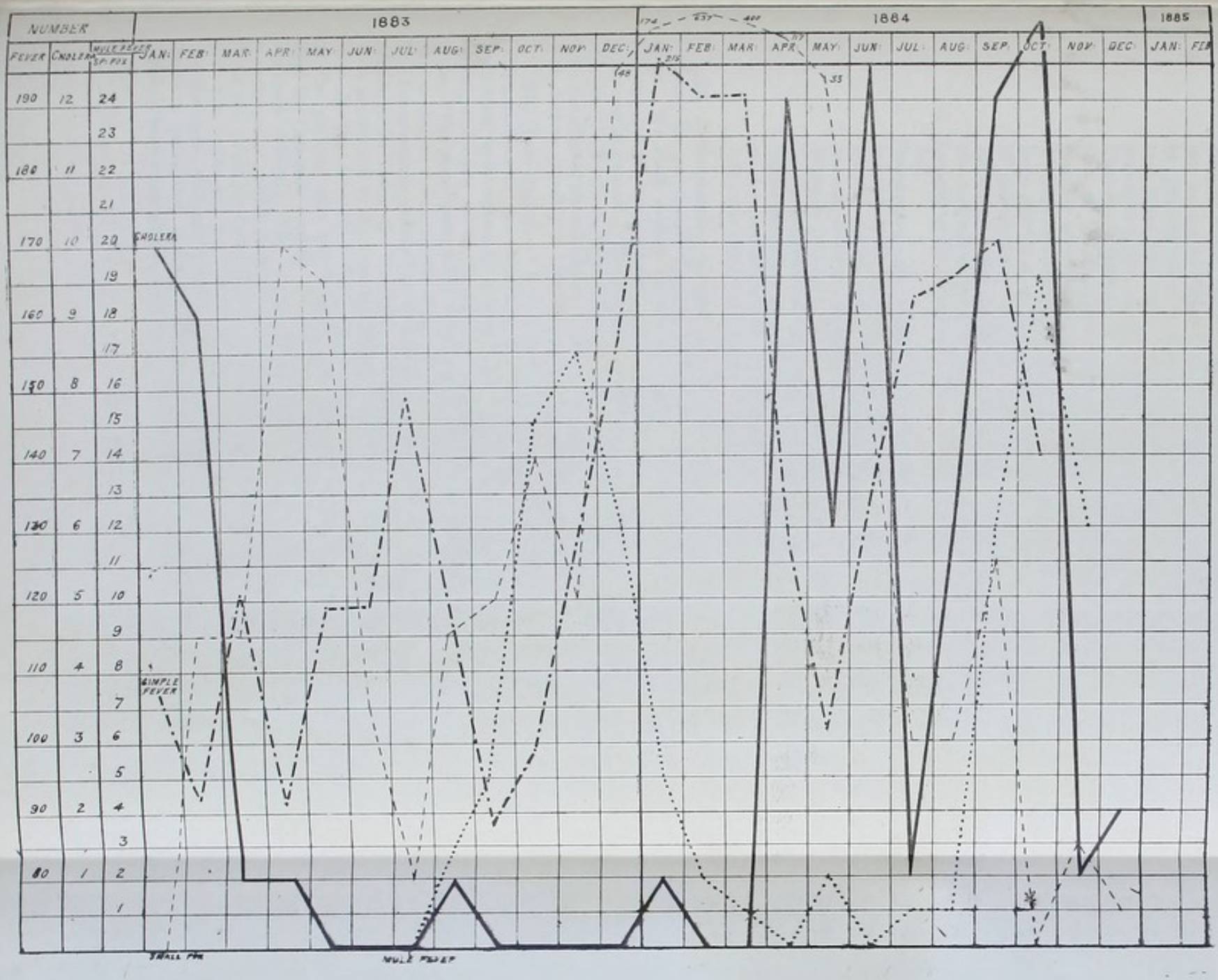
CONCLUSIONS.

1. THE PREVALENCE OF MULE RELAP-SING FEVER SEEMS IN DIRECT RELA-TION WITH THE RAINFALL, BUT THE FATAL RESULTS SOME 4 MOS: LATER THAN THE CORRESPONDING RAINFALL.
2. THE No. OF CASES & THE No. OF INCHES OF RAINFALL ARE NOT IN DIRECT RATIO.

ILLUSTRATING THE RELATIONS BETWEEN ATMOSPHERIC TEMPERATURE AND RELAPSING FEVER.



CONCLUSIONS. THE DISEASE OUTBREAKS OCCUR WHEN THERE ARE NO EXTREMES OF TEMPERATURE & WHEN THE RANGE IS SLIGHTEST.
THERE ARE NO DIFFERENCES OF TEMPERATURE BETWEEN TOUNGHOO & RANGOON SUFFICIENT TO ACCOUNT FOR THE DIFFERENCES IN DISEASE PREVALENCE.



PRECIS

SMALL POX:—

MAX: 1883. APRIL & MAY & DEC^{R}
1884. JAN^{Y} TO MAY.
(I.E. NOT WHEN MULE DISEASE PREVAILED)

CHOLERA:—

1884. PREVAILED APRIL & JUNE. ALSO IN AUG. SEPT & OCT^{R}.
(I.E. IN LATTER OUTBREAK WITH RELAPSING FEVER BUT IN OTHER QUITE INDEPENDENTLY.

SIMPLE FEVERS:—

CURVE RESEMBLES THAT OF SMALL-POX & NOT IN SLIGHTEST DEGREE THAT OF RELAPSING FEVER.

RELAPSING FEVER OF MULES:—

MONTHS OF PREVALENCE AUG^{T} TO FEB^{Y}
OF MAX: OCTOBER & NOVEMBER.

CONCLUSIONS

THE FATALITIES FROM RELAPSING FEVER IN MULES OCCUR IN NO DIRECT ASSOCIATION WITH THE GENERAL UNHEALTHINESS OF SEASON. IN 1884 IT HAS PREVAILED CONTEMPORANEOUSLY WITH CHOLERA POSSIBLY AS BOTH DISEASES ARE AFFECTED BY WATER SUPPLY.

J.H.S.

(*b*) *Rainfall* in relation to prevalence of the disease is thus far noteworthy that both at Tonghoo in 1884-85 and at Rangoon in 1883-84 and 1884-85 the rain curve has been parallel to the curve of disease fatality but three or four months antecedent to it. This is very striking (*vide* Graphic Record No. 3) and suggests that the advent of the rains and the increase and decrease of rainfall are in direct relation to the disease and probably simultaneous with the occurrence of attacks, allowing some three months for the average duration of cases (including incubation). The actual amount of rain bears no relation to prevalence of the disease, for the fall was greater in Tonghoo in 1883-84 when the disease did not prevail than in 1884-85 when it proved extremely fatal, and the total losses in the two years have been about equal in Rangoon, whereas the current season has been much wetter than that of last year. The rainfall of Thayetmyo, hitherto a disease-free place, is very low as compared with that of Rangoon and Tonghoo. It is plain that increase of average monthly rainfall means increase in the number of swamps and marshes, whereas increase in the total amount falling in the season or abnormally large rainfall at one time means increased dilution of tanks and flushing of natural water channels. We can to an extent understand why the Relapsing Fever should be in association rather with the gradual annual rise and fall of the rain curve than with unusually severe downpours.

(*c*) *Humidity of the atmosphere* holds the same relations to the disease as rainfall.

(*d*) *Temperature.*—In so far as we can judge, the disease is associated in its invasion with high minimum temperatures and low maximum, and it is on the decline when the range of temperature daily is greatest. We cannot, therefore, associate it with either excessive cold or excessive heat. It seems that exposure does not act through reduction of resisting power by the influence of extremes of temperature nor excessive diurnal range. These influences must affect the strength of constitution ; but here, again, we have evidence that condition and constitutional resisting power have but little effect for good or evil in this disease.

26. In considering the influence of season, it will be well for a moment to compare the prevalence of fevers, cholera, and small-pox of man with that of relapsing fever. We shall hereby be enabled to an extent to decide whether the seasonal influences in relation to relapsing fever of mules are special or only such as are operative in disease in general. The conclusions at which we arrive from this method of inquiry are that the fatalities from relapsing fever in mules occur in no direct association with the general unhealthiness of the season. In 1884 it prevailed contemporaneously with cholera, possibly as both diseases are affected by water-supply (*vide* Chart No. 5.)

27. *Water-supply.*—I have already shown that we can never thoroughly rely on information as to the sources of drinking water-supply for transport mules. The main facts with regard to the outbreaks are as follow:—At Tonghoo all the regimental mules are, out in the sanitary camp, and have been, in the lines, watered from wells. The water from these, although somewhat opalescent, cannot be considered bad. Water also, as far as we can judge, is not a likely source of the disease at Rangoon. The Somersetshire mules have (officially) been watered only from the Royal Lakes and certain wells during the time when the disease can have been conveyed to those which have succumbed to the current outbreak. The water of the Royal Lakes is very good, as is also that from the wells. The latter are two in number—one on the high ground of the plateau, near the hospital latrine, used for troops A and C, and the other near the Cemetery road on the low ground near the native followers' village. They are both covered. The upper one especially is liable to run short of water, on occurrence of which emergency the animals are taken to the Royal Lakes to drink. I do not think the water-supply of the Somersetshire mules has been at all to blame for this outbreak, nor do I attribute the disease at Tonghoo to that source. I quite agree with my predecessors in this inquiry that water in general, and from tanks in particular, is always open to suspicion in causation of this disorder, but I think it most often acts as such when obtained out of the ordinary routine, as when

animals are being worked and given a drink from some scanty water-accumulation. I do not think that a good full tank or a well with a fair amount of water in it will be likely ever to convey the disease; marshy pools, stagnant tanks, streams with very sluggish current, and stagnant accumulations of water whenever and wherever they occur are to be looked on with most suspicion. *A priori* we may argue that water, especially in tropical countries, is a fruitful conveyer of disease; that the spirilloids are just such organisms as we might expect to invade the system from stagnant water and marsh soil; that Lower Burma is specially the abode of marsh and malaria; and that the substitution of well water for that in tanks at the Rangoon lines has been considered beneficial. The argument that the disease died out at the depôt after substitution of well—for tank—water has been now proved unsubstantial, for the disease seems annually to practically disappear in the spring, even when no change is made in the source of drinking water. We must constantly retain in mind that we have not yet sufficiently exact evidence of when the disease begins to enable us to associate the cases directly with any special physical and climatic conditions. The water of many of the marshy tanks around the Rangoon lines is bad enough to account for any amount of disease; but here, again, we must remember that private ponies receive water from most filthy-looking sources, seldom under their owners' supervision, and they have not suffered from relapsing fever to any marked extent. We must conclude that, although it is highly probable that the drinking water is a source of the disease, in many cases we cannot point to any special water-accumulation to be shunned on this account. All stagnant water and foul tanks are to be avoided, and the use of water from wells resorted to whenever possible.

28. *Diet* may predispose to fever by being defective in quantity, for an animal with an empty stomach is more liable to take specific disease than one with a full stomach; the method of preparation of food may be so defective as to prevent its nutriment being properly extracted so as to support the system and give it disease resisting power. The food of mules at Tonghoo and Rangoon at the time of attack in 1884 was open to much improvement, both as regards quantity and mode of preparation. But we cannot look upon feeding defects as having much influence on the prevalence of this disorder; they are coincident circumstances rather than actual causes of the outbreak, although we must not forget that the near ally of this disease in man is called by the very significant name "*Famine* fever."* Coarse indigestible food given to old animals with defective dental apparatus may have actually produced ulcers of the stomach and so provided an opening through which the parasites have gained ready access to the system; this effect might reasonably be attributed to prolonged use of rice-straw as fodder or of totally unprepared paddy. These irritants acting on the stomach for a long time might predispose it to undergo degenerative changes. At times the animals have been without nosebags, their gram being thrown on the floor of the stable and the mules within reach allowed to scramble for it. This would give a chance of the gram ingested being contaminated with floor impurities and possibly among them spirilloids or their spores. The floors being kept very clean must materially have lessened the liability to take the disease from this source in the case in question, but the chance remains. With regard to *fodder:* the Tonghoo mules began to fall sick after they had for at least six months received as fodder from the Commissariat only 13—15 lb. of paddy-straw per diem. This article of diet is, unless chopped, quite unsuited for mules, being irritant to the stomach and affording little nutriment, if any. It comes from land which, in accordance with the necessities of rice culture, is for many months in the year an excellent nidus for growth of low organisms, but that it is not deleteriously affected thereby would be proved by the fact that it affords excellent fodder for bullocks if we had not experimentally demonstrated that those animals take equine relapsing fever only with very great difficulty *if at all*, probably because of the small size of their red corpuscles. In Rangoon the fodder has been, during my stay, tank grass dry or tank grass wet, and previously the long rank grass collected from the broad Burmese paddy bunds was supplied in its naturally dried state (after its seeds had been shed to secure a next year's crop) as *hay!* Wet tank grass is deficient in nutritive principles in proportion to bulk, but when cut properly, only the part above water being used, is good fodder. However, the

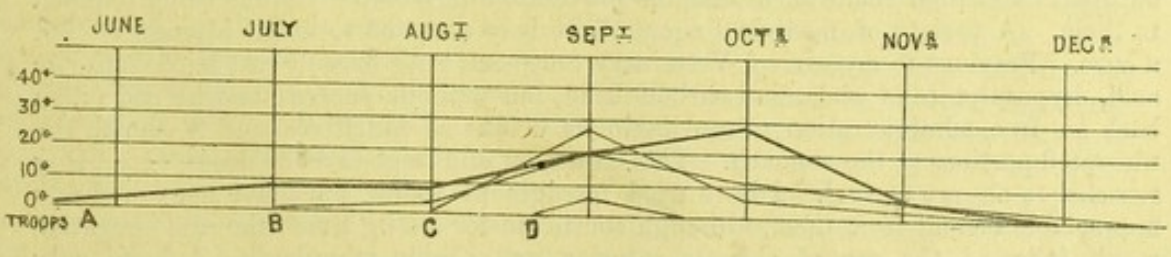
* In man also it not unfrequently occurs that well-fed individuals succumb to relapsing fever.

contractor cuts it below the water surface and thus supplies a semi-fermenting mass of which a large part is yellow semi-decayed leaves and a tough submerged stem covered with slime. This slime is just the sort of habitat a spirillum prefers. Dry tank grass is sweet to the taste, fairly nutritive, but liable to contain germs of spirilla such as have (possibly) resided in the slime which has dried on the stems. Thus, as far as relapsing fever is concerned, it is open to the same objections as the contractors' wet tank grass. Some change in the system of feeding transport mules in Burma is sadly needed and would, probably, if on correct lines, tend to lessen the prevalence of relapsing fever.

29. *Influence of attendants and general management* can have merely a predisposing effect on the development and prevalence of the fever. A driver in charge of three mules has much power over them as concerns ensuring them healthy constitutions not predisposed to disease. A careless, lazy man will allow his animals to scramble for their food, much of their fodder to be trampled under foot and contaminated with urine, the work to fall unevenly on his three mules, insufficient time for drinking, heads too tightly fastened to admit of the animals reaching their feed, and so on. Unless looked sharply after, a native may appropriate the mules' food for use of himself and family; sometimes a driver takes a dislike to one of his mules on account of its being high-spirited and troublesome, and he tries the effect of lessening its feed for the benefit of favourite mules. The eyes of the European non-commissioned officer cannot be everywhere to check such practices, and he is often compelled to be away at parades and other duties. I am inclined to consider that where a driver has lost his three mules from this disease, he is probably careless and inefficient, and that where he has lost two out of the three, he may be unfortunate merely, but is more probably inefficient. Attendants may be responsible for the actual introduction of the disease by, when on orderly or other special work, allowing their mules to drink from dirty tanks or to eat swamp grass and whatever pickings they can get from marshy places. In justice to these men I must add that, as a rule, they seem fond of their animals and the mules of them, and that in the various outbreaks it is not always the least-cared-for animals that have suffered.

30. The freedom of the Artillery mules at Tonghoo and of the Sapper mules at Rangoon may be associated with a better acquaintance by Artillery and Engineers than Infantry with the details of mule management. The officers and non-commissioned officers in the Infantry Regiments have not been given a chance in this respect. They have had no opportunity of acquiring experience in animal management, practically or theoretically, so when they have made common-sense suggestions have not been able to urge them with sufficient authority, and there has been absolutely no systematic veterinary protection for the animals nor even have adequate health records been kept.

31. The following table shows the monthly distribution of admissions to treatment of Somersetshire mules, the percentage being taken in each case on the number of mules left in the stables respectively. This method of search for the cause might be worth following further if it were based on reliable facts; but, as we have already seen that the date of admission to treatment is not, in most cases, the date of attack, we may simply notice its results without considering them as established :—



From this it would seem—

(1) The effects of the cause began to operate in July in A troop.

(2) They affected A and B in August, but A most.

(3) In September the disease raged in A, B, and C; even D also.

(4) In October it continued to an increased degree in A, was only half as frequent in B, and still operative in C.

(5) In November it still acted on A and B, most so on the latter.

All these troops were subjected to the same climatic conditions, and all up to October were watering at the Royal Lakes. We have seen why probably A troop suffered most, but have no evidence that those causes acted specially in October on A troop or specially on all the troops in September. D troop was broken up on 17th September and the mules distributed in the other three stables; *four* days after this the mule of D troop which was affected in September was admitted to treatment from B troop stable where he may have (but also probably did not) obtained the disease. The mule puckals were substituted for the camel puckals in October in so far as I can ascertain, and since then the small mules have done their share of puckal work.

9 to A troop.
21 to B do.
19 to C do.

32. After this detailed examination into the causes of the outbreak, we conclude as follows:—

(*a*) A large size, horse-like "make," and gray color seems to predispose to the disorder. Overwork, too, seems to increase liability, especially if it be associated with insufficient food of bad quality.

(*b*) Collection of mules in numbers at one place, especially when in association with a moderate uniform temperature and want of free circulation of air; also exposure to weather during monsoon time when rain is falling and the air sultry, "muggy," and stagnant; also use of food contaminated with marsh, slime, or water from marshy sources; possibly also leech and mosquito bites act as determining causes.

(*c*) The spirilloid is the sole immediate cause.

33. Treatment.—The disease caused by spirilla in man is amenable to treatment of a curative character, but hitherto the mule disease has resisted all efforts in that direction. This is a great and important difference between the human and equine disease, possibly attributable to natural expulsion of the parasites from the system of man, but not of the mule. I have hitherto utterly failed in my attempts to learn how the parasites escape from the systems of mules, and have arrived at the conclusion that we have no evidence of their doing so under ordinary circumstances. If this be actually the case, it entirely prevents our having success from adoption of the eliminant plan of treatment. Our hands are further tied by the fact that probably no improvement or reduction of the general health of a mule will render its system unfit for the support of the spirilloids; therefore the tonic system of fortifying the constitution against the inroads and multiplication of the parasite does not offer much chance of success. But one more curative means remains—the addition to the blood of some agent which will destroy the parasites or check their development. Results obtained by this method have not as yet been very promising. Our main efforts must therefore be directed to prevention, to the ascertaining by close and accurate observation of such determining causes as are most frequently present in outbreaks and their removal, or else the prevention of possible victims being exposed to them. A variety of medicinal agents have been resorted to in the attempt to find a cure. Previous to my arrival Veterinary Surgeon (First-class) Frost tried salicylic acid, hyposulphite of soda, and carbolic acid, but without success, and he had fallen back on free administration of mucilaginous drinks as nutritives and to sheath the ulcerated portions of the stomach. This treatment and large carbolic doses were being resorted to on my arrival. I have tried ferruginous tonics, but have not found any appreciable benefit from them, although continued for a long time; the same negative result followed the use of chiretta infusion and simple stimulants. I have only had glimpses of benefit in dealing with two agents—*oleum terebinthinæ* and *acidum arseniosum*. The former agent acts, not only as a stimulant and eliminative, but also as an aromatic, compensating to an extent for deficiency of aromatic principles in the fodder. In one case to which it was administered for some time the animal seemed to

regain a little strength and to exhibit less regular visitations by the parasite than usual. I think that I have noted the same result after the regular administration of white arsenic in six-grain doses daily. All I can say of these agents is that, in so far as my experiments go, they are the ones from which most hopes of good result have been obtained. I thought on one occasion that the action of ammonia in dilute solution sufficed to kill the parasites; it seemed to do so when an ammoniacal solution of carmine was added to a drop of blood in which the parasites were numerous and active, but subsequent results did not confirm this. When I administered ammonia to a patient, the case proved acute and promptly fatal. Dilution of a drop of blood with water lessens the distinctive movement of the parasites, but does not, at any rate immediately, destroy them.

34. If we look through the list of causes, we shall see that they almost all admit of counteraction. Thus I am able to draw up a list of suggestions as to measures which should be adopted with a view to avoidance of future outbreaks. The mules selected for service in Burma should be of moderate size and age, those most like donkeys being the best, and animals of gray or white color specially avoided. But the mule transport should be as limited as possible; animals indigenous to the country such as elephants, ponies, and especially Burma bullocks, being mainly relied on for military transport. Although I do not recommend any change at present, I think that a gradual alteration in distribution of the various animals of transport would be an advantage from a veterinary point of view, the mules being sent up-country as much as possible, especially to Thayetmyo, and the bullock-cart transport and use of ponies being expanded at Rangoon. Tonghoo does not seem to be a healthy place for mules or ponies, but it ought to be for elephants and is for bullocks; and we must remember that at this place the mules have not had an opportunity of showing how they would be if their food were good and sufficient and their stables in a healthy situation. Experience seems to indicate that the collection of a couple of hundred mules in one place in Lower Burma is not advisable; that their stables should be in blocks well apart from one another and in places where they will get lots of fresh air and be well above the level of neighbouring marsh land. It also indicates that the plan of hiring out Government mules and ponies is a very excellent one in British Burma; it secures individual care and attention for the animals while rendering available, in emergency, a number of useful transport beasts which are kept in condition at little cost to Government. Although exposure to weather cannot be avoided on a campaign, it can be generally avoided in peace, and its ill effects can in either case be much lessened by careful selection of camping grounds and of water for drinking purposes as well as systematic feeding of the animals. Every march into the jungle, every despatch of batches of transport animals from one station to another, and every season when communications are kept up between a central station and outposts (as between Tonghoo and Thandoun) ought to be made an opportunity for enforcement and practice of animal management in such manner as *should* be resorted to on active service and as *might*, to an extent, be if it were more generally practised. This is a matter which I feel assured might be managed much better than at present with benefit to the mules and with less serious results from their marches up-country than are now sometimes experienced. There are some stables which, as we have seen, seem to be local centres of the disease; they are probably too much shut in and liable to stagnation of air in them, and possibly also to stagnation of water in their drainage cisterns and in the soil around. It would be very beneficial if stable A in the Somersetshire lines at Rangoon were removed from its present position and thereby aggregation of animals on the sub-plateau, where it is situated, be lessened and a freer supply of air allowed to stable C. It should be removed to a more airy and open position. Failing this, something may yet be done in the way of removal of the drainage cisterns and the substitution of paved surface drains down the side of the hill and the adoption of the dry-earth system of managing the stable-flooring. The drivers' latrine should be at once removed to below the hill, and the brick drains against the gram-shed and hospital latrine should be deepened so as not to allow of overflow by the side of the pathway into A stable. The supply of movable jhamps for one side of each block of stables to be used for the exclusion of driving mist and heavy rain under the discretion of the officers in charge would be an improvement. In excep-

tionally sultry weather the mules should be picketed in the open, and the stables would benefit by the temporary evacuation. With regard to the Tonghoo lines, their bad situation might be improved and possibly be made to answer if some improvements were made in the surroundings; the drainage altered by deepening the existing channels and by cleansing of the neighbouring impure jheel, also by the removal of refuse accumulations and encroaching native huts. Each of the mules should be provided with a good jhool in order that it may be available in the event of unavoidable exposure, especially during monsoon-time, and there should be provision made for a reserve supply of nosebags being available, so that the animals may never have to feed off the ground. If all stables were, like those of the depôt at Rangoon, fitted with mangers, the saving in food for the animals would be great and the cost more than covered by the saving in nosebags, which are quickly worn out by mules and might then be kept simply as service kit. I attribute so much ill effect to exposure during the rains that I am of opinion it would be an advantage if an order were issued that whenever in British Burma mules or ponies are removed from their stables into quarantine, during the monsoon especially, cheap temporary sheds be run up for their accommodation, and that at places such as Thandoun and Kokine, where Government mules are constantly wanting accommodation, provision be made for their proper housing. With regard to watering: it seems to be generally understood that well water should be used whenever procurable, and, when it is not, large tanks with ample water in them are to be preferred, and especially those free from rank vegetation and scum on the surface. Care in selection of water and in avoidance of that of marshes and low tanks should everywhere be strictly enforced and constantly impressed on every one in charge of the mules; especially is this precaution necessary with puckal mules, and great care should be exercised that these be not overworked and that they get regularly tended and fed on their return from this duty.

35. Reserve supplies of food, both fodder and grain, should be available for immediate use, so that condemnation of a day's supply will not mean deprivation of the mules of one day's feed. The gram or paddy should be crushed; there is a large proportion of animals among the mules in Burma with defective teeth and weak digestive organs; such animals cannot make the most of uncrushed gram, and they do not digest unprepared paddy, which therefore acts as an irritant. If the gram be purchased in crushed condition, it is liable to be of inferior quality and adulterated; it would be better if there were grinding stones for crushing purposes in each large mule lines. It would be an advantage if part of the present supply of grain were supplemented by some aromatic, such as Indian oil-cake, which can be purchased in the bazaar at a cheap rate. It would supply the aromatic element, which is usually deficient in Burmese fodder, and which native horse-owners atone for by the constant administration of mussauls. It is already used in feeding the Government bullocks at Rangoon and Tonghoo, and is much liked by animals and excellent for fattening. There are so many points which might be settled with advantage on the question of feeding Government animals in Burma—the conditions as to price, quality, and procurability of foods are so different in that country from in India—that I am of opinion the whole matter might, with advantage to Government, be gone into by a committee of experts. So long as there are defects in the feeding of the animals, so long will they suffer from enzootics, such as relapsing fever and anthrax. Another cause, though an indirect one, of such losses is the want of information on animal management by those in charge of the regimental mules; they gain experience in time, but at the expense of the welfare of the mules, and it cannot be otherwise until they are instructed in such matters before being put in charge. Also, with ample stable and transport duties to occupy their attention, they are too frequently needed for drill and parades, and the mules, left to native supervision, suffer accordingly. I believe that this disease will be much less frequent when the Burma transport has adequate veterinary supervision and organization. The salootries need instruction in the most elementary points of their duties and are utterly incompetent to give advice in animal management and to cope with the most simple cases of sickness or injury, much less severe outbreaks of obscure disorder; they also need special instruction, and, until they receive it, will be of but little use. These are matters affecting the general welfare of the animals mainly, but their indirect influence on the prevalence of relapsing fever has, I feel convinced, been very

great, and correction of them will tend materially to prevention of its prevalence in the future. It would be well worth the while of Government, in the event of future outbreaks, to try the effects of change of climate by shipping affected mules in the earliest stages of the disease to Bellary, Secunderabad, or some other dry place where they could be kept isolated and treated under veterinary supervision.

36. I in one case removed blood from the heart of a mule just dead and which I had an hour before ascertained to contain some parasites, and with it conveyed the disease to another mule. The fact of the blood not having died before use lessens the value of this experiment however. I have therefore inoculated with dried blood with negative results; local abscesses, which healed readily, occurred at the seats of inoculation. A safe practical conclusion to arrive at is that the carcasses of all animals which die from this disease should be destroyed by cremation in their skins and without post-mortem examination; except when necessary for scientific purposes; it should never be needed to verify diagnosis. We are not assured that the flesh used as food by dogs conveys the disease; vultures do not seem to suffer from it, but they *may* distribute the spirilloids in their fæces.

Part III.—THE DISEASE.

The researches which have been carried out by myself supplement those of my predecessors and place us in a position to give a systematic account of relapsing fever, which must henceforth occupy no unimportant place in the list of equine diseases—indeed of diseases in general, whether of man or animals. It specially demands attention from veterinary officers serving in the East, and is very liable to occur under the conditions of active service among animals of all the mounted branches. Its slow insidious progress to sure fatality demands that we be jealously apprehensive of its occurrence in a field force and constantly on the alert to avoid it or to detect it in its earliest stages and manifestations.

2. The SYMPTOMS vary somewhat in accordance with the phase of the outbreak, the individual peculiarities of the patient, the manner in which the attack has been brought about, and possibly other conditions as yet ill-ascertained. In relapsing fever, as in most other diseases of a specific nature, those cases which occur at the commencement of an outbreak are generally the most acute and directly fatal, whereas those which occur later often succumb finally to sequelæ. This leads to corresponding differences in the symptoms and post-mortem appearances, and accordingly the phase of the outbreak is an important point to be noted in investigation and diagnosis. Still throughout all phases of the outbreak we find attacks varying in acuteness and urgency; this may generally to an extent be traced to some other disease affecting the patient concurrently with the fever, to some defect of internal organs, or to still more obscure conditions, as those of physique, digestive powers, and so on. The experiments made by Dr. Evans with surra and by myself in this investigation have enabled us to arrive at certain differences in the phenomena of the disease in accordance with species and the mode of entry of the *materies morbi* into the system. Yet with this apparent difference in special cases there runs, as an under-current, the typical disease which flows with definite course from commencement to close. There is always a difficulty in determining when the attack actually begins except in experimental cases from which we learn that the period of incubation in cases of inoculation of a mule with large numbers of the parasites subcutaneously injected is five days, in cases of administration as a drink (gastric invasion) six days, calculating from the first abnormal rise in the internal temperature, that is, the commencement of the first fever exacerbation. We have ample evidence, therefore, that this disease may be induced artificially in 4—7 days. The principal practical value of this fact lies in it affording us some assistance in tracing *particular* cases home to their immediate causes, but it is nevertheless diminished in value by the fact that it is a matter of the greatest difficulty to decide when any particular case commenced. The

The disease manifested itself earlier in my pony case than in the mules, and the period of incubation was shortest of all in the monkey; longest in the dog, in which animal it was only on 12th day the parasites appeared in the blood.

first actual febrile attack may occupy only about three days and be separated by about five days from its immediate successor—the first relapse. A brief period of the animal "being out of sorts" like this is apt not to receive much attention, especially as it may only be indicated by a want of the usual heartiness in consumption of the gram and a little abnormal heat of the mouth. People in charge of horses and mules, in India especially, think but little of a slight attack of fever, especially when (as anticipated here) it passes off in a day or two. These attacks may by non-professional observers, even careful men, be passed over again and again as trivial. The mule becomes feverish about once in every ten days, his liver seems to be out of order judging from the orange color of the membranes of the eye, but he manages to do his work, so relapse after relapse is apt to occur before the veterinary officer sees the patient. But he, too, has his difficulties to contend with in diagnosis of the disease in these earlier stages. The animal is probably brought to him after the crisis of the relapse, the day when he specially attracts attention of those in charge; but the disease is characterized by a sudden fall in body temperature after this crisis, and thus the thermometer may give a reading of but 101° F., and the records of four or five successive daily observations may show only slight variations ranging between this point and the normal. It is just at this point, too, that examination of the blood by means of the microscope fails in its diagnostic value, for at this phase of the disease the blood is free from detectable parasites. As a matter of fact, I believe it impossible to diagnose the disease *promptly* under these circumstances, to distinguish it from simple congestive disorder of the liver. If the observations with our two instruments of precision—the thermometer and microscope—be continued for five days, a diagnosis may be given; *if for ten days, a positive statement as to the presence or absence of the disorder may be made without the possibility of error.* This exactitude in diagnosis is a valuable feature in the clinical history of the disease; it may best be attained by use of the thermometer and thermographic charts and by detection of spirilloids in the blood by means of the microscope; but it is important to note that, in the absence of these instruments or of skill to use them, the number of beats of the pulse if recorded on a chart daily for ten days and a mark made against the tracing whenever the animal seems very feverish (as indicated by heat of mouth, dullness and thirst), the periodicity of the disease will be recognized, and a very fair approximate diagnosis may be made. But from the very nature of the disease it is necessary that *time* be allowed for the diagnosis except when a drop of blood under the microscope is found to contain the characteristic organisms, then the nature of the main disease can be no longer doubtful, but there is always the possibility of the case being a complicated one. During the access of the fever the membranes of the nose, mouth, and eyes are generally yellow (especially the conjunctivæ), but in the intervals they become markedly pale, that of the mouth seems to have scarcely any blood in its veins, occasionally the epithelium of the gums, inside of the lips, or tongue undergoes degeneration and separates from the deeper part of the membrane, leaving superficial circumscribed ulcers. A favorite position for these is on the margin of the lower lip, and I have seen them arranged symmetrically in pairs here and on the tip of the tongue. These must not be confounded with spear-grass ulcers, wounds received from the bit or from balling iron, twitch, or drenching bottle. It is not always possible to determine whether ulcers in the mouth of a patient are from injury or the result of circumscribed degeneration of the epithelium from the action of disease. Occasionally ulcers of this kind appear on the Schneiderian membrane where the epithelial debris remains as a yellow scab for a day or two and is then washed away by mucus; the earliest appearance of these ulcers in the nose is as though the membrane had been grazed and blood were slowly oozing; they rapidly disappear and the general character of the membrane again becomes normal. I have noted in the mouth, eyelid, and labium vulvæ large ulcers which looked as though the result of injury; they generally healed freely when treated with carbolic oil. In one or two cases I have noted an eruption of spots on the skin, especially that of the muzzle, apparently resembling in their essential nature those of the mouth and nostril. Œdema of the mucous membranes sometimes occurs; it is most frequent in the case of the conjunctiva, making the eyelids thick and the eyeball to seem sunken; the presence of this symptom renders it difficult to cause protrusion of the haw in the usual way and to determine whether or no petechiæ are present. In some cases the conjunctiva becomes bladder-like and bulges with effusion; one or both eyes and one

or both eyelids may be affected simultaneously. I have observed serous effusion beneath the vulval mucous membrane, but petechiæ were very seldom present and, when seen, are simple, very small blood points. The irritation of these parts is, however, shown by frequent symptoms of "horsing;" indeed in mares there is almost always in this disease a little reddish brown fluid dried about the external generative orifice, which discharge persists throughout the attack. The parallel to this sexual phenomenon of mares is seen in male animals as frequent and quite uncalled-for erections of the penis, which may be due to irritation of the erection centre in the cord, but possibly also is associated with the formation of blood-clots in the erectile structure of the penis, which organ latterly in the attack remains permanently slightly protruded and turgid, or there may be embolic plugging of its vessels; sometimes slight dropsy occurs beneath the sternum, and all injuries are very liable to dropsical complications. Slight œdema of the limbs occasionally occurred in my cases, and in one acute case there was considerable anasarca, the swelling involving all the four legs and the whole under-surface of the body, and it disappeared with remarkable suddenness after the animal fell for the last time. However, throughout the disease there is not such an amount of dropsy as might have been anticipated from the depraved condition of the blood. In my cases dropsy has been the exception, not the rule. The irritation occurring in the membrane of the eye is denoted early by profuse lachrymation. The tears overflow the eyelid and trickle over the face and there dry, leaving a peculiar white saline and albuminous mark. Generally some of the surplus tears trickle through the nose, but sometimes, probably as a complication, there is an ordinary mucous discharge which may be slight or profuse. The occurrence of successive crops of petechiæ, especially in the conjunctivæ, is very characteristic of the disease. I have carefully studied the relations of these blood-spots in character, number, and time of appearance to the presence of parasites and the degree of fever. They vary much in appearance in different cases and at various times. They range from simple small spots of fresh blood or from faint very diffused extravasations of blood to extensive blood accumulations outside the vessels of a dark purple color, changing gradually to a fresher red as they clear away. This removal of the escaped blood takes place in every case and thoroughly, so much so that at times the conjunctiva looks perfectly normal and healthily moist. The spots are most readily detectable on the portion of membrane which covers the haw, but are by no means confined to there, for they invade all parts of the membrane. They are generally either very few or perfectly absent when the parasites are active in the blood, and hence it occurs that their period of invasion is generally at the crisis of the fever, and that they are numerous and well marked during the decline and least fever stages. Whether they depend on migration of the parasites or on vessels plugged by them giving way I cannot say, but, from the time of their occurrence and the purple color so often assumed by them, and in which respect they differ considerably from the petechiæ of simple anæmia, I am inclined to consider that they result from these causes. I have carefully examined, by use of the microscope, the lachrymal secretion and the gelatinous mucus which collects at the inner canthus, but have detected nothing abnormal in them, and certainly spirilloids, as such, are not present. I have not tried inoculation with them. These spots may perhaps be associated with disorder of the spleen, such as is generally found, after death, to have been present. Possibly the enlargement of that organ is indicated during life by the animal, as he sometimes does, turning his head to the left and biting over the hypochondrium. This symptom I for a long time thought was an indication of gastric ulceration until I found it present a few days before death in an animal of which the stomach proved to be not diseased. In some cases the submaxillary lymphatic glands become somewhat swollen without any lesion of mouth or nostril to account for it. I have never seen it lead to abscess of the gland nor even to a very painfully tense condition of it. I am inclined to consider this symptom due to the same causes as are in operation to produce the general engorgement of the lymph glands which I have found on post-mortem examination and markedly *before death* in my dog. On studying this symptom in relation to others, I find that it is of so unfrequent occurrence as to prevent its being considered of much importance in the progress of the case, and there are no evidences to lead to the conclusion that it is indicative of a tendency of the parasites to desert the blood channels for the lymph system in accordance with phases of their life-history. The appetite is excessive throughout the attack;

it is remarkable how an animal in a state of semi-coma will continue to eat grass almost without ceasing. In one case the patient seemed very keen for his gram, but after the first mouthful, either from pain or distaste, refused to take any more and turned away eagerly to his grass. In the human subject, pain, especially after ingestion of food and persisting until completion of digestion (or until vomition occurs), is found when the stomach is ulcerated. Sometimes, as in the case of senile ulcers of the limbs, so in gastric ulcer there is little or no pain; when present, the pain is noted as occurring *in one spot.* "It is not uncommon for the pain to increase for a few days and then gradually to subside. These exacerbations the writer has chiefly observed in very chronic cases. They probably point to an extension of the ulceration, for they not unfrequently precede hæmorrhage or perforation." There is appetite, but the patient dreads the pain resulting from feeding; there is copious flow of saliva prior to vomition; loss of strength and energy in chronic cases; pain between the last lumbar and first dorsal (?)vertebræ generally more on the left side than on the right; the bowels are constipated, there being mucus sometimes in the stools. The urine is clear and passed frequently and in considerable quantities (Fenwick). The above précis of the symptoms of gastric ulcer in man, as taken from Quain's Dictionary of Medicine, is well worthy of perusal as justifying the diagnosis of the mules' disease as ulceration of the stomach which used to be made prior to the establishment of its nature as relapsing fever. Before I proved its periodicity, demonstrated its distinguishing parasites, and proved that ulceration of the stomach is absent in some undoubted cases, I examined carefully this diagnosis of my predecessors. I found in our patients depraved appetite, the copious flow of saliva from the mouth, loss of strength and condition, and the pain on pressure over the loins and over the sciatic nerves on either side of the sacrum. The breath also was in some cases very offensive. I came to the conclusion that, although these symptoms would justify diagnosis of gastric ulcer, something else had to be dealt with, that something being blood derangement, as proved actually the case. We have already seen the bearings of ulceration of the stomach on the main disease. The urine varies in quality and quantity in different cases and, to an extent, in accordance with the phases of the disease. Sometimes it is very viscid, its specific gravity being as high as 10,40. I am not sure what is the cause of the viscidity; it is not directly associated with the presence of albumen, nor have I been able to determine its special occurrence at any one phase of the disease or in association with congestion or other disease of the kidneys. In other cases the reverse condition is found and a certain degree of polyuria is present, the urine being limpid, of a very pale yellow, and low specific gravity (10,05—10,15) and passed frequently and in considerable quantities. This also I have not been able to associate with any special pathological state. The color was not always high; on the contrary it is extremely exceptional to find it above the normal, although it is often very much paler. In almost every case the freshly-passed urine is quite clear and distinctly acid. Sometimes albumen is present, a condition I am inclined to associate in direct ratio with the breaking up of the red corpuscles as being specially present in the decline of fever, but it is by no means constantly present. I have never been able to detect sugar, bile-coloring, casts, or parasites in the urine, but rather frequently had occasion to record excess of urates and phosphates, probably in consequence of the amount of disorganisation brought about in the blood by actions of the parasites. The fæces affords no indication of disorder. I have noticed the red slime on the surface of the dung-balls mentioned by Dr. Evans, but it is not of frequent occurrence and is of minor import either in relation to diagnosis or pathology. As a rule, the dung is normal in appearance and in quantity, presenting only occasionally a reddish or chocolate color, until the last day of the disease, when it sometimes becomes pultaceous, chocolate-colored, and of most foul odour; this I am inclined to associate with the presence of circumscribed congestions of the small intestines. The state of the skin indicates the general malnutrition; it is in most cases completely devoid of the soft, bright appearance of the healthy coat, being dry, tense, dull, and harsh from an early stage of the disorder. It at length becomes "like that of a dead animal." There is then a great liability to bed-sores, and they sometimes exhibit great tardiness in healing in those animals which, having "been down," resume the standing position, the bruised portion of skin persisting in the centre of the ulcer. The skin eruption already mentioned, as seen in some cases, seems similar to that on the nasal membrane,

being simple, circumscribed, epithelial degeneration, probably as a result of plugging of the small vessels of the dermis; it disappears very rapidly and is only found in a small proportion of cases. The dry, hard condition of the skin is a characteristic feature of the chronic or subacute cases; it is associated with extreme marasmus. The wasting sets in rather suddenly after the fever has been on the animal several times. Suddenly one day it is noticed that the mule or pony is "tucked up;" his ribs show plainly, his eyepits are deep, he looks old, and there seems "nothing of him." It is at this stage the patient generally comes under professional notice; inquiry elicits the facts that lately he has been sluggish and not working with good heart; that he has been out of sorts several times recently, but not to any important degree; that he has been noticed to hang back on the collar chain in the stable; to eat earth freely and to have a most ravenous appetite for fodder. These signs are almost sufficient in themselves to stamp the case as one of relapsing fever, but the diagnosis must be assured by use of the thermometer and microscope. The movements are very languid and weak almost from the first, and the deficiency in power of the hind limbs must not be called paralysis, for no true loss of conveying power has occurred in the nerves or nerve centres, but the deficiency in motor power is, if we may judge from post-mortem results, due to blocking of the vessels of the hind limbs with blood-clot. There is often a marked deficiency in color of the supra-lumbar muscles found after death; they seem as though they had been inflamed, probably, as Dr. Evans pointed out, in consequence of excessive pack-loads to the animals and quite independently of the disease. Almost from the first the deficiency in power of free movement shows itself. When the animal is made to "stand over," it tries to obey, but catches one hind foot in the bedding and totters with the hind quarters, almost invariably recovering itself in time to prevent a fall to the ground. When the patient falls and remains recumbent broadside, as it does towards the end of the attack, it is manifesting extreme debility rather than actual paralysis, for, when the animal suffers from delirium or struggles on being touched, it will be found that some of the limbs, generally the uppermost pair, move with abnormal energy, so that the bedding soon becomes removed from beneath the animal and he is covered with bed-sores, and sometimes the skin is actually worn through on projecting points from the way in which the animal dashes himself about. The head is especially liable to such injuries; sometimes it is spasmodically drawn to one particular side, in other cases it is rigidly elevated. Generally an animal which "goes down" does not rise again, but I have records of cases in which the patient has risen after being some time recumbent persistently and has remained for a month or longer time after this constantly standing until death set in. Some patients stand and feed to the very last and then drop dead. A noteworthy indication of cachexia and the general malnutrition is occasionally to be seen in the changes undergone by the cornea in advanced cases of this disorder; it first becomes opaque and then small sub-conjunctival ulcers, like those seen in dogs suffering from distemper, may be noted. The general atony of the skin in animals which remain stretched flat on the side for some time is denoted by the undermost surface of the body being bathed in perspiration, but there is never any critical increase in the evacuations from the cutaneous glands resembling the sweating stages of the febrile exacerbation in man.

3. The very general nature of the malnutritions, the fact that practically every part of the body shares in them, at once attracts our attention to the blood as the main seat of disorder. A small drop may be obtained for examination by incision, with an abscess lancet, about a quarter of an inch long, into the thin skin of the muzzle; occasionally the animal will, by an inopportune jerk of the head, give himself rather a long slash, which, however, generally heals freely by first intention; generally blood does not flow readily from the wound, and the animal does not seem to mind it, or a small quantity of lymphy fluid only will, for some time, collect from the wound. The deficient sensibility and anæmia of the patient are noteworthy. The drop obtained is generally grumous and purple in color ("like bad claret," as it was described by a looker-on on one occasion); probably a few short delicate hairs of the muzzle become mixed with the specimen; they do not interfere with the examination, but sometimes look remarkably like embryonic filariæ, especially when curled up by contraction of the clot in which they are entangled; they taper towards each

end, most so towards the apex, and their medulla does not extend to either extremity, so that they look very like worms, for which they are apt to be mistaken. I dwell on this point because when first I saw them I thought I had made a "find," but soon decided that I was in error. A thin film of blood may now be examined under the microscope, under one-fifth inch objective, which is best for diagnostic purposes. It is at once evident that the blood is not normal. The white corpuscles are, as a rule, quite natural, but the red are much altered. Sometimes débris of red corpuscles may be observed, and the corpuscles are crenated, irregularly shrivelled, or they look half-moon shaped, this latter condition resulting from their being markedly concavo-convex. Large, irregular, filmy-looking masses, which at first look like altered white corpuscles arranged in a tessellated manner or in lines like strings of coral, turn out to be altered or decolorised *red* corpuscles. I have noted this state especially just after the parasites have left the blood when the fever is declining, but do not consider it peculiar and special to this disease, for I also find it in the blood of anæmic mules. It has been noted in the blood of human patients in relapsing fever, but I cannot recall to mind having seen or recorded it in the blood of the dog or monkey. The first change noticed in the blood elements of surra cases at Dera Ismail Khan by Dr. Evans * was *leukocytosis*, with a remarkable increase in the number of the largest granular lymphoid corpuscles, which he often saw breaking up under the microscope. Leukocytosis was not remarkable in my cases at Rangoon. The next change Dr. Evans noticed was in the red corpuscles; he speaks of a large number of shapeless particles of pale red jelly from half the size of a normal corpuscle downwards, whereas I have invariably found them larger than the normal red corpuscles. He refers to a large number of small granular particles in the serum which I also have observed; some of these look like colorless masses of débris, others are small highly refractive nuclei with an evident independent movement quite distinct from the Brownian notion, by which all such minute bodies are found to be affected. These are the "point-like bacteria" seen by Ignatovsky in man, which he found in the perspiration as well as the blood; but he observed them also in the blood of typhoid and typhus patients and in perspiration induced by pilocarpine and even in the sudor of healthy individuals. It is evident that the point-like bacteria, which I had described in my daily records, are not distinguishable from numerous other micrococci, both normal and pathological. I can, by independent observation, confirm their special prevalence in those phases of the fever when the spirilloids are absent; further, I may note that I have frequently seen nuclei like them in the blood of unaffected animals. Particularly when the corpuscles have aggregated to form rouleaux and so produced an irregular network of solid blood elements, the diagnostic characters become apparent. These are the presence of organisms of a special shape, and the fact of a special movement of the corpuscles being caused by them. The latter condition will be apparent before the former, and to the practised eye it is detectable under even very low powers, half inch for example. Quite apart from the blood currents which cause numerous red and white corpuscles to rush onwards in one direction, generally rapidly, but sometimes in an uncertain slow manner when they are entangled in side currents, also apart from the occasional rush of a red corpuscle towards the exposed end of a rouleau, there will be noted a quivering or shaking of some of the red bodies of an irregularly intermittent character. When parasites are numerous, this may at once be detected in any part of the field of vision, but sometimes we note it only after very careful attention and concentration of our vision on certain groups of corpuscles; then the parasites are not frequent. While examining the specimen it is necessary to alter the focus carefully with the fine adjustment, so as to ensure all the thickness of the layer of blood being seen; sometimes the quivering of red corpuscles is detectable only where they are massed together. I lay great stress on this quivering because it is a special feature of relapsing fever and surra, and its presence is diagnostic to such a degree that, when a large number of blood specimens are being examined in a limited time, it is sufficiently indicative to the practised eye that there are parasites present. After long examination of a specimen of slightly-quivering blood, we may at length see a minute thread-like organism with eel-like movements emerge from the mass of corpuscles and move slowly forward, or we may observe the little being apparently tugging with all its

* *Vide* paragraph 24 of his report.

FIGURES OF PATHOGENIC SPIRILLOIDS OR RELAPSING FEVER ORGANISMS

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

LENGTH OF PARAS
ESTIMATED AT 3
TIMES THAT OF
CORPUSELES.

ROUGH DRAWINGS OF THE PARASITES MADE BEFORE THE METHOD OF STAINING WAS ADOPTED. INSERTED WIT
VIEW TO ILLUSTRATING THE GENERAL IMPRESSION CONVEYED TO ME BY THEM AS SEEN IN ACTIVITY: BELOW TH
DIAGRAMS WAS A NOTE THAT THE FRAGMENTS OF CORPUSCLES WERE QUIVERING OR BEING DRAGGED HASTILY
THE FIELD OR HAVING A PARASITE HOVERING ROUND THEM.

Fig: 5.

Fig: 6.

Fig: 7.

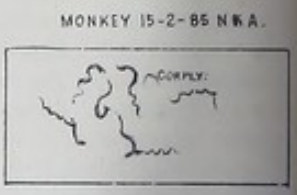


Fig: 8.

DRAWINGS FROM STAINED SPECIMENS. THE DETAILS OF INDIVIDUAL PARASITES HAVE BEEN SHOWN BUT THE FI
ARE NOT TO SCALE. AND IN THE SELECTION OF INDIVIDUALS FOR DELINEATION THERE WAS A BIAS TOWARDS TH
BEST SHOWING THE SPIRAL FORM THIS FEATURE OF THE DRIED & STAINED PARASITE HAVING BEEN DENIED.

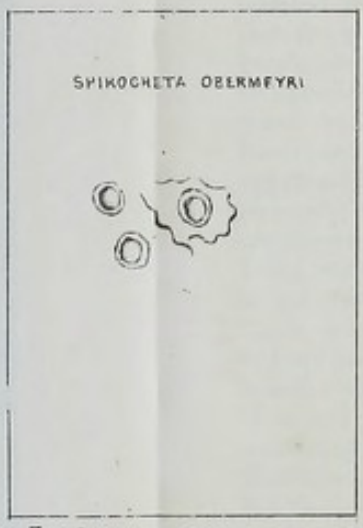


Fig: 9. AFTER ZIEGLER. FROM THE ENGLISH EDITION OF HIS "PATHOLOGICAL ANATOMY."

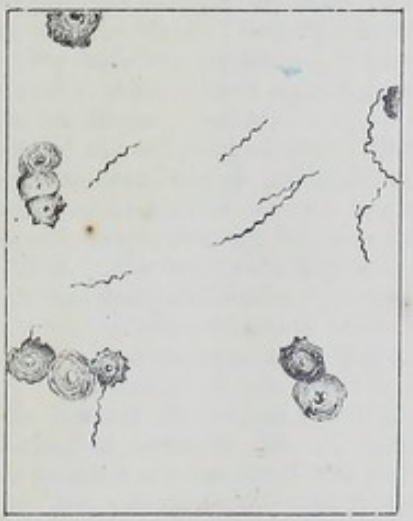


Fig: 10. HUMAN BLOOD OF RELAPSING FEVER FROM THE "PRACTITIONER." x 700 (AFTER KOCH)

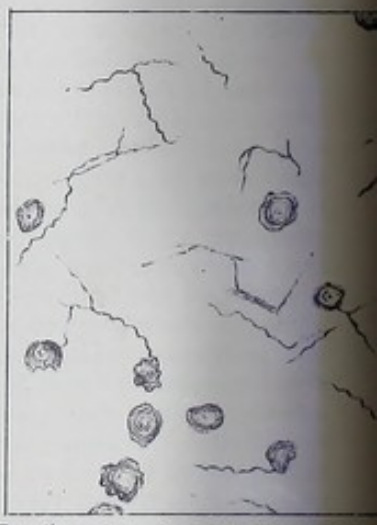


Fig. 11. BLOOD OF APE INOCULATED W
BLOOD OF PATIENT FROM WHOM FIG:
OBT'D X 700 FROM THE "PRACTITIONER" (AF

Fig: 12. ANILINE STAINED SPECIMENS AFTER THE WEIGERT-KOCH METHOD, FROM THE BRITISH BURMA OUTBREAK (BLUE)

Fig: 13. SAME AS FIG: 12. (MAJENTA)

Fig: 12 & 13. WILL PROVE OF INTEREST
THEY WERE PRODUCED BY CONDUCTOR S
FROM SPECIMENS HANDED OVER TO HIM
(WITH MY MICROSCOPE). HE PREPARED T
DIAGRAMS, WHICH MUST THEREFORE BE
AS ENTIRELY UNBIASSED TESTIMONY A
THE FORM OF THE PARASITES.

might at a red corpuscle endeavouring to detach it from its rouleau. When the parasites are "numerous and active," we see them all over the field directly we get the focus; most active in their movements, they pass from place to place by lashing their long thin bodies, by an eel-like movement, and in a vermiform manner by the propagation of a body-thickening along them like a wave. They may be seen free in the pools of serum between the rouleaux or firmly attached to a corpuscle or two which they have succeeded in appropriating and drag after them as they move backwards, spin round and round, shake and worry or occasionally push forwards with rapidity. These artificially motile red corpuscles move into and out of focus rapidly and especially attract notice. At first I thought them the heads of the parasite as they were sometimes distinctly heart-shaped, but enlarged experience very soon proved that it was not so, the heart-shaped ones being merely two corpuscles simultaneously detached from the end of a rouleau. Dr. Evans' graphic description of the actions and appearance of the surra organism applies to these to a very great extent, but not entirely. As he mentions, they always take hold of the corpuscle by their blunt ends, sometimes several at a time to one corpuscle, then they move backwards, tugging hard. I have frequently seen two or three of the parasites tugging together at one piece of corpuscle, and they often become entangled, several together, in one wriggling mass. I have never been able to observe what Ignatovsky of Odessa points out that these agglomerations may be observed exclusively at the end of the attacks and that the number of balls or bundles of the spirilloids invariably increases as the moment of critical perspiration approaches, but then we have no sudorific crisis in mules. However the bundles of spirilloids are well marked in some of my stained specimens. At different times the organisms differ in size, and I have even noted large and small side by side in the field of vision. In a number of observations we shall find them sluggish or active, large or small, ferocious or placid, altogether in every condition suitable for examination of their structure and actions. Under a powerful light they are refrangent, irregularly-curled lines; under moderate light each looks like a very thin black line actively motile in such a way as to give it a subspiral appearance, but not uniform in thickness throughout. Although, as described, they generally move in the direction of the blunt end, except when dragging at the corpuscles, a bold stroke with the lash-like tail or a doubling of the parasite on itself until the tip of the tail almost meets the blunt end followed by a sudden straightening may cause lateral movement. I have never seen them attack the white corpuscles, nor, indeed, any constituent except the red ones, which sometimes seem to be torn in pieces by two or more parasites; fragments may be seen in most specimens taken towards the end of the relapse, but we must not consider as such some corpuscles shaped like a quarter moon, which, on careful examination, prove to be unbroken red corpuscles altered in shape. At first I found that the parasites were no longer detectable in specimens after drying, and this serious difficulty presented itself until, on Dr. Vandyke Carter's advice, I adopted the Weigert-Koch method of drying in very thin layer on a cover glass. Even then they could be seen only with great difficulty, and the conclusions obtained were not reliable. I tried various staining agents and at last had complete success with aniline dyes. The effect of obtaining the parasite stained and quiet is remarkable in its modifying influence on our preconceived ideas as to its general characters. The parasite in motion is as different in appearance from when at rest as a rapidly revolving wheel is from a still one. It is only when this fact is realized that we are able to understand how it is that competent observers figure the same object so differently as they have *spirochæta obermeieri*. This will be seen by comparison of the figures of Koch and Ziegler here inserted (as kindly copied for me by Veterinary Surgeon F. Smith)—*vide* opposite page. It will be noted how in some diagrams we see organisms with well-marked spiral figure, uniform in thickness throughout and remarkably long in relation to their width. This is the idea conveyed on first sight by the equine parasite in motion. In other diagrams we find that these little beings are represented as having a much less spiral figure (although still this feature of their shape is well marked) and as markedly tapering towards each extremity, altogether looking very unlike the long spiral thread figured by the other observer. Certainly the difference between the living and the dead parasite is extremely striking. These will contrast excellently with my diagrams of the parasites of the mule taken before and after staining. The former must be received with reserve, because it is a most difficult

thing to decide on the shape of the spirilloids in movement. The way they wriggle about and constantly pass out of focus renders their delineation very trying to the patience, and any representation of them on paper purely diagramatic. The dried and stained specimens (especially the magenta ones) are most beautiful objects. I think the figures in some text-books of the spirillum, like a huge corkscrew, must be purely imaginary and deduced from ideas formed of what the parasite in motion would be like *if* it were sufficiently enlarged. If such be the case, they are very apt to mislead and require immediate correction in accordance with the latest proved facts. We shall hereafter consider the parasite further.

4. The thermometer is the guide which ranks next to the microscope in value for purposes of diagnosis. The tracings which I have obtained by careful *personal* investigation for a complete month and by reliable records under my immediate supervision for a much longer time are beautifully illustrative of the effects of the disease-producing organism on the thermogenic centre. When an inoculation has taken, the mercury rises to 106° or 105° Fahrenheit, generally in a gradual manner and with one fall in its course usually on second day ; the evening temperature is exaggerated in relation to the morning, whereby the diurnal oscillations are rendered much greater than in health. On third, fourth, and fifth days the reading is high and gradually increasing, so that usually on the evening of the fifth day of exacerbation the crisis is reached ; a rapid fall then ensues, so that on seventh morning the temperature is low and so remains for two or three days until the next rise. In typical cases there is a slight check to the abrupt fall on the evening of the sixth day. Thus a stage of the disease is completed, and these stages, varying in length from seven to ten days, may occur again and again, varying in number from three or even fewer to, it may be, as many as seventeen before death. The phase of low temperature reading cannot be designated "fever-free," because the line seldom falls to the normal ; it may, however, descend even lower, and such great oscillations indicate usually the approach of the fatal result. The maximum reading is sometimes 107° Fahrenheit or even more, but usually not quite so high. A rise above 108° Fahrenheit indicates speedy death. There are thus two conditions under which death occurs—the very high temperature (in one case I obtained a reading of 110° Fahrenheit) and the very low ; the latter is most frequent and found generally in prolonged cases. There is no other disease of lower animals in which the thermographic tracings are so neat and so specially significant. The pulse records when reproduced in the form of a tracing give us ample confirmation of the relapsing character of the fever as shown by the variations in animal heat. As the finger generally used in taking the pulse is less delicate as a medical test than the thermometer, it does not suffice for slight variations in tone or volume of the artery, but by accurate record of the number of the pulse beats we can "steady," so to speak, the results obtained by the highly sensitive thermometer and prevent our being led astray by the numerous minor surrounding influences which affect the temperature of the body. Suffice it to say that the tracing from the number of pulse beats resembles that from thermometer readings, that the pulse is sometimes dicrotous, often very small and difficult to take, but sometimes fuller and stronger than might be anticipated from the debilitated state of the animal. Records of the frequency and nature of the breathing are much less valuable. Auscultation and percussion indicate no abnormality of the lungs and pleura until within a few hours before death. When the fever is severe the breathing is quickened, and I have observed cases of double expiration and others of double inspiration. A labored oppressed breathing is generally an immediate precursor of death ; this grave symptom is associable with loss of breathing sounds at the lower part of the chest and also with a mucous râle indicative of pulmonary congestion, hydrothorax, and accumulation of froth in the trachea and bronchi. In addition to the symptoms already enumerated, I have noted others, in the cases under observation, which probably have resulted from accidental complications ; such are coughing, extreme nervousness, œdematous swellings around the arms, and external generative opening of the female due to excessive straining for removal of fæces after the animal is down ; also the formation of simple abscesses, presenting no specific characters, in so far as I could determine, on the skin. In some cases slight anæmic pulsation of the jugulars and cardiac murmurs due to the same cause may be noted.

5. The *duration of attack* varies considerably in different instances. The average of twelve cases observed by me has been over 66 days. The longest has been already 170 days on the sick list and looks pretty well still; the shortest of these cases was 17 days. Thus the disease may vary in duration from six months to one month, the period of incubation and of presence of the disease before diagnosis being allowed for. We have seen that it is difficult to fix the exact commencement of any but experimental cases, and we have shown why this is so. Generally life flickers for some time before it is extinguished, the reserve stores of fat become exhausted and the animal falls in consequence of being unable longer to support his own weight. He remains for days apparently more dead than alive, and, at length, after becoming very cold and breathing with difficulty, dies quietly; or else, towards the end of a protracted illness, the patient becomes excessively weak, dull, and heavy, with dropsy of the eyelids, pendulous lower lip, extreme labor in breathing, looking the very picture of misery. He falls, and for some time is delirious and struggles severely, especially when touched, and finally "dies hard" with a high temperature and parasites in the blood up to the last. In my most acute case I found the patient carried off while in good condition, with a plentiful reserve of nutritive matter in the way of internal and external fat, after a very short illness characterised by acute dropsy. The temperature ran up to the almost unprecedented reading 110° Fahrenheit just before the fatal result and the animal died directly from fever, his life being "burnt out" as it were.

6. The *immediate cause of death* varies from a yielding to the excessive height of the fever to the complete wearing out of the animal and exhaustion as a culmination of anæmia; not unfrequently the animal succumbs to complications rather than the disease; in others sequelæ are responsible for the fatality. Of these latter, two demand special notice in consequence of their frequency—embolism and perforation of the stomach. The latter takes place without the peritoneum being involved in appreciable inflammation and the rent through it is often extremely small; of the emboli, some are of long standing and others recently formed. In one case a blood-clot of some considerable firmness and consistency was carried in the round of the circulation until it became caught in passing into the pulmonary artery, where it became surrounded by a fresh clot. Fatty heart and enlarged liver especially weaken the animal and lessen his power of resisting the attack; thus, of all cases of fatality in this disease, the majority depend on failure of the heart's action due to fatty degeneration.

7. Post-mortem Appearances.—In marked contrast with the almost complete absence of definite lesions found post mortem in the Trans-Indus cases are the conditions noted in Burma, which are numerous, varied, and sometimes extremely marked. The carcass of an animal which has succumbed to this disorder presents normal rigor mortis; it is generally extremely emaciated and seems that of a very old individual. Gelatinous extravasations into the areolar tissue are often found; some of these are subcutaneous, their special seat being below the sternum, although not infrequently they are found elsewhere. They seem to be quite distinct from the somewhat similar lesions associated with bruises of the skin, such as result from the injuries inflicted by the animal during his delirious struggles and which may be so violent as to cause complete perforation of the integument. Favorite seats of gelatinous effusion are above the popliteal space, along the course of the sciatic nerve, and between the muscles of the thigh. The lymphatic glands are generally swollen and œdematous, especially those of the femoral space; sometimes there is a little dropsy of the belly; occasionally serous fluid has accumulated in excess in the pericardial sac; almost always there is a little liquor pleuræ above the normal. In cases of long standing the liquid contents of the stomach may have escaped into the peritoneal sac as a result of gastric perforation. I have noticed in most cases of relapsing fever a larger amount of yellow watery fluid in the stomach and small intestine than is usual in health, and that the contents of the large intestine are usually somewhat dry, although in advanced cases they may be found pultaceous, reddish in color, and most foul-smelling. The lining membrane of the small intestines is the seat of circumscribed local congestions, which may invade the mucous covering of Peyer's patches and be most marked in that situation, but Peyer's glands are not diseased. In acute cases I have seen the mucous congestion continuous throughout the anterior two-thirds of the bowel, but ulceration is never present. The usual parasites (*Strongylus armatus*, *Oxyuris*, and so on) are found in the alimentary

canal. The stomach generally presents extensive ulceration, but its pathological condition has been already noted in detail (Parts I and II). The alimentary and respiratory mucous membranes are elsewhere and in other respects free from disease with the exception of the ulcers alluded to in our notice of symptoms. These ulcers seem only to occur on those parts of the mucous membranes which are visible in the living animal, and they resemble those of the stomach in being atrophic degenerations; they are, however, evanescent, less frequent, and less numerous. The spleen is, I find, almost always enlarged and generally weighs about 4 lb. Its structure is not frequently disorganized, although in some instances there are circumscribed blood accumulations in it and on its surface. Generally there is an excess of spleen pulp, so that it pits on pressure and long retains the impression of the fingers. Microscopical examination of the fresh pulp failed to demonstrate any abnormality although on one or two occasions I have noticed an unusual preponderance of spherical highly refractive bodies generally aggregated in pairs. The liver is usually quite healthy and not nearly so frequently the seat of congestion at Rangoon as it seems to have been at Tonghoo. The kidneys are seldom free from disease; either they are in an anæmic, dropsical state, or else are markedly congested. In one instance I noted a considerable amount of extravasation on and in the under part of the right kidney as though some blood-vessel had given way. Large clots of the most varied colors may be drawn from the large vessels at the upper part of the abdomen and the hind limb; even when the investigation is made immediately after death, these are often found. The sublumbar muscles are normal; the supralumbar will generally be found atrophied. The lungs are congested in most cases, but once, where a clot prevented passage of blood to them, they were peculiarly dry and anæmic. The trachea is generally full of froth. The heart in chronic cases has gelatinous accumulations in its furrows in place of fat and a few extravasations along the course of its blood-vessels; but in only one case have I seen any beneath the endocardium, and then it was into the substance of an auriculo-ventricular valve. In all advanced cases the heart is large and flabby as having undergone extensive fatty degeneration; both sides are filled with blood with clots of various colors; thus I have seen from the four orifices of the same heart severally and respectively yellow, purple, bright red, and a greenish blue clot projecting. In all the more prolonged cases the brain and spinal cord with their meninges are decidedly sodden and anæmic; the brain is soft and looks as though it had been for some time in water; the plexus choroides of the lateral ventricles are bloodless and of a pale yellow color. In acute cases there is sometimes very slight congestion, but I have never seen this so pronounced or associated with the presence of blood-clots pressing on the brain as seems to have been the case not infrequently at Tonghoo (*vide* Parts I and II).

8. With the discovery of minute organisms in the blood arose numerous and important questions resembling those which have been so long and hotly debated concerning the bacillus of anthrax. Firstly, is the spirilloid organism a feature of the disease, or is it merely an accidental accompaniment? My researches show that it is found in all cases of the disease, is in direct and immediate association with a certain phase of the disease; thus, when the temperature is about to rise, the spirilloids become detectable, they become very numerous and active during the rise and the climax of the fever, but disappear just before the crisis; thus its presence in spirilloid form seems directly related to the degree of the fever, and its phenomena and activity seem to bear definite relations to the severity of the relapse. The features of the life-history of the organism, in so far as we are acquainted with it, place us in a position to attribute all the disease conditions of the patient to it. The inoculation of an animal with blood containing the parasites invariably produces the disease (except when the inoculated animal is a ruminant), but I have also proved that blood taken from an animal when no organisms are detectable will likewise convey the disease, but less rapidly. We have a strong chain of evidence that the parasite is the actual and immediate cause of the disease, and further that it is itself the *materies morbi*. We infer that it acts in two ways to produce the symptoms and fatal result; in its active phase it tears and drags at the red corpuscles and causes their disintegration; it passively acts by blocking up the capillaries. Careful examination of the records of cases shows that the occurrence of petechiæ on the mucous membrane especially takes place when the parasites are not detectable in the blood by ordinary methods, *i.e.*, from just before the maximum of the curve to just before the commencement of the next rise. When the

active parasites are present, the membranes are generally yellow and free from spots. Although the membranes will not be found to answer this description in all cases, the typical occurrence of spots is sufficiently frequent to warrant our considering it a fact in the pathology of the affection. In their active phases the parasites cause irritation of the membranes as is sufficiently evident from the lachrymation, profuse salivation, and slight discharge from the female generative passage, but later in their life they tend to aggregate either by two individuals uniting longitudinally to form a long one either temporarily or permanently (which is rare), or by several becoming entangled together to form wriggling masses, which Ignatovsky considers most frequent just before the crisis. Although each parasite is in diameter smaller than the smallest capillary, it is not hard to understand how several individuals wriggling together may block up an extremely narrow passage through which they are trying to pass ; hence results a more or less persistent plugging of the vessels, which becomes the more widespread because the blood in this disease has a marked tendency to form clots in the living vessels. Petechiæ, extensive extravasations, dropsies, and even ulceration are possible, indeed probable, results of such plugging of minute vessels. Ulceration of the stomach thus or otherwise induced might act as a cause of further or primary invasion by spirilloids. There can be no doubt that of two animals grazing in a marsh—one with healthy unbroken mucous membranes, the other with gastric ulcer,—the latter would be the more liable to invasion by low organisms. Although the stomach lesions seem to be part of the disease and have been found fresh and actively progressing where no other possible cause is known to have been in operation, we have to be careful in deciding that they are not due to medicine administered in the course of the attack or otherwise or to coarse improperly prepared food; since the lesion is present in cases which have not been treated and which have been fed on soft, well prepared food for some months before death, we must look on the gastric ulceration as a feature of the disease which, on minute examination, seems to result from plugging of the small blood-vessels of the stomach. Thus the lesions found ante-and post-mortem in cases of relapsing fever are such as might be, and probably are, caused by the parasite found in the blood.

9. Thus relapsing fever of mules is an invariably fatal disorder characterised by the periodical occurrence of attacks of high fever, during which a special organism, closely resembling that of spirillum fever of man, is found in the blood. The organism causes disorganization of the blood, which is to an extent repaired during the interparoxysmal phases, and thus the patient is usually able to hold up against the disorder for some time. But anæmic symptoms and lesions gradually accumulate and culminate in death from exhaustion, plugging of vessels, or gastric perforation as a result of local atrophy of the stomach. In some cases the symptoms are acute, and the animal succumbs to the height of the fever.

Part IV.—THE PARASITE.

It only remains for us to examine the natural history of the characteristic organism as found in the blood of mules suffering from relapsing fever. We may do this in the short business-like language of science.

2. General Characters—*Size.*—Varies in different cases ; usually $\frac{1}{6}$ red corpuscle in diameter and 3—6 times in length.

Shape and appearance.—In fresh state it is an eel-like body, moving from corpuscle to corpuscle, or looks like a highly motile flagellum with most varied movements appended to a red corpuscle and causing agitation of it and of those in the immediate neighbourhood—dark or refrangent according to focus. In dried state and stained is found to be very like an oxyuris on a small scale, to have a thick portion of the body and a spiral tail, the latter taking less of the dye than the former, commencing as a sudden narrowing of the body and terminating by a fine point although the whole organ is visible in the living animal and has nothing of the nature of an infusorian flagellum. The thick portion tapers in either direction from its centre. Anteriorly it terminates rather abruptly and is somewhat rigid. We have reason to believe there is some holdfast organ at this extremity. The thick part is as spiral as is compatible with its diameter. Dr. Evans having examined the live specimens at Rangoon, also the dried and stained ones I sent him, is of opinion that they are essentially the same kind

as those which he found in the surra disease at Dera Ismail Khan ; he does not think they belong to any class of spirilla, but that they occasionally take a spiral form like an eel or a serpent in contortion. He further informs me that, after examining them with a $\frac{1}{12}$ immersion lense, he found their bodies not really round, but flattened, the sides often curl in, which gives them a roundish appearance.*

Varieties.—The parasites as taken from the mule, pony, dog, and monkey differ a little in minor details of structure, such as sharpness of the "head" end, and so on ; also in strength, energy, and ferocity.

Movements.—Eel-like, lashing, vermiform ; less frequently spiral, observable especially in the parasite from the dog and when the movement is very slow. I have seen in some of my cases the very small parasites spinning round and round on a refrangent thickening apparently at about the centre of the body. Perhaps this is the early stage of spirilloids when they are in a transition from spherical to spiral or sub-spiral forms. But we have no exact evidence that spirilloids ever assume a germ form.

Action and habitat.—We have no evidence as yet of an extra-sanguinal abode of the spirilloids or of connection of the pathological with innocent spirillum forms such as are found in stagnant water, and so on, nor with the very closely allied bodies found in the blood of rats. They cause considerable destruction of the red elements of the blood, irritation of organs through which that blood circulates, and blocking of vessels with its sequelæ. Koch has cultivated spirilla of man artificially, and I notice a little growth on some of my slides.

Food and other nutritive essentials are furnished by the living blood, but what they exactly are is not established. It is remarkable that the parasites attack the red elements only ; in all my microscopical examinations I have failed to detect them in lymph, lymph glands, spleen pulp, dropsical fluids, and secretions. It would seem as if a certain temperature sufficed for their destruction, or more correctly to make them assume the undistinguished form, and that in this way the relapses are brought about.

Excretions.—In our deficient state of knowledge as to the forms assumed by the parasite, it is premature to discuss whether it is the actual presence and activity of the parasite which causes the fever and the organism which is in itself the *materies morbi*, or whether the fever and the disease depend on the excretion into the blood by spirilloid of pathogenic matter. The evidence is strongly in favor of the former view ; the fact that blood not containing appreciable spirilloids communicates the disease may be construed into the statement that we have not yet detected the parasite in it. Often, after prolonged examination of a specimen of blood, we detect just one or two parasites in it, but we almost always, when the spirilloids are not present, observe spherical nuclei with independent motile powers.

3. *Reproduction and phases of development.*—Nothing is known but this—that, some five or six days after introduction of the parasites into the subcutaneous areolar tissue and longer after ingestion through the stomach, there are many more parasites in the system than were introduced artificially. They cause local irritation at the seat of injection, as was proved in my inoculations of the bullock and monkey,† and the latter case showed that such local irritation does not involve failure of the experiment to produce systemic infection. Parasites introduced into the system therefore reproduce freely ; we have no evidence that this takes place in the lymphatic system, but rather infer that it occurs in the blood. A growth of the parasites in the blood seems to be indicated by some being larger than others. We have some indications of development in that fluid. Although, possibly, the spirilloids are viviparous, the balance of evidence seems at present in favor of their being in the non-irritant phases of the disease spherical nucleoid bodies and of the spirilloids being in due course, *i e.*, some four or five days, developed from these ; thus may be explained the longer incubation with inoculation of blood parasites not detectable, than with blood parasites free and active. Sometimes in the phase of fall of temperature masses of débris seem to have pieces of spirilloids attached to them which may be the latter degenerating. Ignatovsky found head-like bacteria, filaments of point-like micrococci which were agglutinated by means of thin stripes of a homogeneous, structureless substance, having a slow wave or pendulum-like movement, but I have never been able to find any in my cases.

---

* In paragraphs 33 and 34 of his report on "Surra" he observes "when the parasite dies slowly in serum its body spreads out and flattens."

† Inoculations made simultaneously and in the same way and with the same instruments caused in mules no local disturbance.

At present we have positively no exact evidence of the mode of exit of parasites from the body; indeed we have no proof that they ever leave the system of an affected mule. The sodden-looking congested kidneys might at first sight seem to have been rendered so by the constant passage through them of these active organisms, but the disease of those organs is nothing more than is ordinarily found as a secondary diseased state in cases of very high fever or even of prolonged debility. Microscopical examination of the urine has failed to detect anything like the blood organism. There are manifest objections to testing this fluid by inoculation. So no method suggests itself to me for settling the important point whether the animal gets rid of the parasites in the urine. If it does, then that fluid would be a fruitful means of conveyance of the disorder which would seem highly contagious, but it is not so. The liver and bowels are congested in the height of the fever, and the lining membrane of the stomach is the seat of extensive lesions, probably due to the parasites; but, in reply to the question whether the parasites are thrown off through the alimentary canal, we can only say that search for such minute beings in the contents of stomach and bowels seems hardly likely to be followed by good results, and on two occasions the gastric contents and on a third gastric and large bowel contents given *per primas vias* failed to communicate the disease. Carter's experimental inoculations of monkeys with saliva failed, but Ignatovsky's observation of motile nuclei in perspiration would seem to indicate possible ways of exit of spirilla from man, but we have as yet no data for discussing this question conclusively in the case of the mule.

4. *Affinities.*—Some light would be thrown on the changes in form of the parasite (if such actually take place) if we knew absolutely what position to assign it in the scale of living beings. There are three positions for which it presents some features of eligibility; it may be bacterian, animal, or vegetable. There are certain of the cryptogams, as, for instance, the mosses and ferns, which produce motile, highly active, free-living spores, termed antherozoids; some of these evanescently active spores may have pathogenic importance, but little light has yet been thrown on this matter by pathological research. It may be remarked that many of these antherozoids show quite as much purpose in their movements as do the organisms of relapsing fever, and, like them possibly, their production occurs at certain definite seasons, special intervals being between each brood, and during the intervals all traces of them (as such) being lost. We have no evidence that antherozoids are capable of direct production of organisms like themselves. Altogether it is probable that the organism of relapsing fever is not a vegetable form of life. There are two kinds of animals which it might be—an infusorian or a nematode. If the ciliæ considered by Dr. Evans as possibly present in the surra organism and the fin-like processes noticed by him also could be found, there would be some evidence of the infusorian nature of these parasites. But I have never found the slightest indication of, or necessity for, ciliary processes or pseudopods in the Burma disease organisms, and Dr. Lewis failed to detect them in the surra parasite; so we must consider their presence as "not proven." Next with regard to nematode characters, its extremely minute size (considerably less than that of ordinary hæmatozoa) might lead us to deny that it is a round worm, but this would be on not sufficient grounds. Its movements might be taken to be absolute proof of its animal nature, but they are not sufficient, although they, with its ferocious attacks on the blood elements, make a strong case. But there is a still stronger one in favor of their bacterian nature. Whether bacteria are animal or vegetable or neither is still unsettled, but among them we find some of a spiral form, some of which are remarkable for the characters of their movements which are like those of the spirillum of man. The similarity of the organisms observed by me in the Burma disease (and probably also of those of surra as described by Dr. Evans) to those found in man, in so far as I can judge by figures and descriptions, and the fact of their, in the same manner, producing relapsing fever, is strong evidence in favor of the close agreement in position on the scale of living beings between the *spirochæta obermeieri* and the Burman mule organism, which I therefore consider to be a spirillum but provisionally term a *spirilloid*. The similarity consists in form, habits, habitat, movements, and general appearance, as may be inferred especially from the description in the current part of the report. The question of the nature of the organisms, therefore, resolves itself into the general one of "what are spirilla?" which I shall not here discuss, but accept the general view that they are spiral bacteria.

Some doubt as to the close alliance of the mule and man organisms legitimately existed until both had been stained and examined in the quiescent state; but, now we know that the former readily take aniline dyes, are curled up subspirally after death, and are found very like the stained spirochætes of man as figured by Ziegler, I think we can have no further doubt on this point, and we cannot consider that the only relapsing fever of man differs intrinsically and radically in its probable producer from the only relapsing fever hitherto described as such in equine patients, and that both these fevers induced by similar means produce a relapsing fever of the monkey in spite of the radical and intrinsical differences in their causes! Adherence to this latter view would argue an incapability of appreciating and accepting such proofs as are generally acknowledged to be sufficient in natural history and modern pathology. Indubitably the nearest allies of these mule organisms as well as of those which Dr. Evans found in the disease called "*Surra*" are the "flagellated organisms" found in the blood of rats and some other rodents, not known to be diseased, of which Dr. Lewis says they "present many features in common with motile organisms undoubtedly of vegetable origin; on the other hand, taken as a whole, they appear to approach more closely to the forms of life usually classified as protozoa * * * *. Many believe that these organisms are zoospores, and not animalcules" (*vide* his "*Microscopic Organisms found in the Blood of Man and Animals*"—Calcutta, (1879).* Koch's cultures of spirochæta, whereby he obtained long spiral organisms, open a fresh line of inquiry, which will possibly be in the future the means by which we shall learn exactly the relations which spiral organisms of pathogenic nature found in the body bear to those similar bodies found in marshes and to spiral organisms as those of the teeth not known to be pathogenic. Hitherto these relations have been extremely problematical, but elaborate culture and working with the higher powers will in due course solve the mystery.

5. I may summarise the PRINCIPAL RESULTS OF THE INQUIRY as follows:—

Positive: (1) The unusual fatality among mules in Burma is due to a disease which is not poisoning, nor ulceration of the stomach, nor simple fever, nor simple anæmia.

(2) The disease in question is a true relapsing fever, probably the first ever recognized as such affecting equine patients.

(3) It is essentially the same as the disease described in 1880 by Dr. Evans as "surra," and only differs from it sufficiently to warrant our considering them (for the present) as local varieties of the same disease.

(4) It is allied to relapsing or famine fever of man, but more distantly.

(5) These allied disease states have been carefully compared.

(6) In the blood in each is found an organism; those described as occurring in the three disease states differ more or less markedly in various respects.

(7) Their nature has been examined, and it is concluded they are spiral bacteria.

(8) Their detection, actions, and relations to the phases of the Burman disease have been demonstrated and compared with those of the other disease states.

(9) Their relations to the affected animal are in exact accordance with the fever phases, so much so as to render the diagnosis of relapsing fever by aid of the microscope and thermometer and graphic records a matter of absolute certainty.

(10) The reasons for obscurity in diagnosis hitherto have been carefully demonstrated.

(11) The symptoms of the disorder have been carefully observed and recorded.

---

* Dr. Carter has demonstrated Lewis' rat organisms since the above was written. They certainly in actions and appearance resemble the organisms of the mules. In the rat they seem to inhabit the blood as "messmates" with the red corpuscles without exhibiting an aggressive policy towards the latter. In stained specimens they seemed to have shorter tail portions and bodies more uniform in diameter. The rats looked healthy, but no temperature records of them have been yet recorded on a chart (3rd July 1885). I have inoculated a horse with some rat's blood containing organisms, local tumefactions occurred but no systemic invasion; in fact, results similar to those obtained by introduction of the mule organism into the ox were found in this experiment. The rat, kindly sent to me by Dr. Carter, was certainly unhealthy, his toes sloughing. This may have been due to confinement, but it reminded me of my experimental monkey (18th August 1885).

(12) As also have the post-mortem appearances.
(13) The manner in which death is brought about has been determined.
(14) The influence of climate, locality, season, &c., on the production of this disease has been discussed, and some conclusions as to etiology, based on statistics, have been obtained.
(15) A number of proximate causes have been determined.
(16) Determination of the nature and causes of the disease has enabled me to suggest prophylactics.
(17) The inefficiency of certain methods of curative treatment has been demonstrated.
(18) Evidence bearing on its communication from animal to animal has been obtained.
(19) The exact relations of gastric ulceration to relapsing fever have been ascertained.
(20) The methods of a systematic inquiry into an outbreak of specific disease have been placed on record for guidance of, and improvement by, future workers.
(21) The value of modern instruments of precision for diagnosis has been vindicated.

The principal matters to be solved in the pathology of relapsing fever of mules on which light may be thrown during future outbreaks are—

(*a*) The mode of entry into, and exit from the organism, of the parasite.
(*b*) The post-mortem, developmental, and free changes which it undergoes.
(*c*) The following out of the parasite in the processes by which it enters mules.
(*d*) The means by which the hitherto invariable fatality of the disease may be changed in the future. Curative treatment.
(*e*) The exact relations between surra, relapsing fever of man and of mules; also of the organisms which give rise to them.
(*f*) The relations of pathogenic to non-pathogenic spirilla.

POSTSCRIPTUM.

1. In submitting this report, I beg to acknowledge the valuable assistance which I have received in collection of the matter contained in it from certain Government officers as follows:—

Dr. Evans, Inspecting Veterinary Surgeon.—Advice at various stages of the inquiry.
Dr. Vandyke Carter, Brigade-Surgeon, Indian Medical Department.—Advice as to methods of staining.
The Officers of the Army and Indian Medical Departments at Tonghoo.—Post-mortem records.
Inspecting Veterinary Surgeon Shaw, Indian Veterinary Department.—His report on the disease.
Veterinary Surgeon (First-class) Frost, Army Veterinary Department.—His report and verbal information and suggestions.
Veterinary Surgeon Fred Smith.—Figures of spirilla from man and *précis* of recent information on relapsing fever in man.
Lieutenant MacMahon, Madras Transport.—Verbal information, returns, facilities for investigation.
Conductor Smiles, Madras Transport.—Verbal information, figures of parasite.

I am also much indebted to the General Officer Commanding British Burma Division and to the officers commanding outstations and regiments, also to the Deputy Surgeon-General to the Forces in British Burma for assistance and support.

2. I have the honor to request that, if this report be printed by order of Government, I be permitted to correct the proof-sheets, and that a few copies be given me for distribution to the professional journals, as the disease is one which is but little known to the veterinary profession.

P.S.—Receipt of a copy of Dr. Vandyke Carter's valuable work on spirillum fever in man, *since the above report was written*, has suggested a few other points of inquiry. I have already explained to the Inspecting Veterinary Surgeon why I have, with the exception of one correction of Dr. Carter's statements, not made more use in this report of Dr. Carter's special researches, *i.e.*, the internal evidences of independence in my report, which goes over a ground similar to Dr. Carter's, will render my paper valuable for comparison on many points with the elaborate production of the distinguished Bombay professor.

(Signed) J. H. STEEL, V.S.,
Army Veterinary Department.

SUMMARY OF MY DIARY OF WORK DONE AND PRECIS OF METHODS ADOPTED IN THE INQUIRY.

16*th November* 1884.—Arrived at Rangoon with instructions to investigate the nature of a fatal epizootic of an obscure nature among transport mules. Reports on the subject by Veterinary Surgeon (First-class) R. Frost, Army Veterinary Department, and Inspecting Veterinary Surgeon Shaw, Indian Veterinary Department, being placed in my hands. Of these I had made a *précis* and had from them obtained much information as to the line of primary action. An equipment of medicines, instruments, appliances, &c., supplied on emergent indent from the Madras Medical Stores including Reagents for preservation of specimens, microscope (a very inferior instrument, having, only 1-inch, ½-inch, and ¼-inch powers, and of which I made little use), urinometer, clinical thermometer, post-mortem appliances, &c.

17*th November* 1884.—After official visits to the Executive Transport Officer, Veterinary Surgeon (First-class) Frost (who was away in the districts), and reporting arrival in writing, examined all animals under treatment and commenced careful clinical study of the disease. This I repeated morning and evening daily until my departure for Tonghoo on 20th December. The routine consisted in taking the temperature, three minutes per mule (four in severe cases; observation repeated in every case of doubt); pulse felt; respirations, condition of membranes, ejecta, &c., noted. Admission and diagnosis of fresh cases. Urine analyses of all specimens obtainable continued systematically at first, but subsequently only in special cases. The following are *Specimens of the Clinical Records in the form adopted*:—

Morning of 28th, November, Friday. Stable Temperature 64° F. at 8 a.m.

Case.	Temperature.	Respiration.	Pulse.	Remarks.
1	104·4°	12, much disturbed.	54, very weak.	Blood extravasation in left nostril; mucous membrane of eye good color, a few spots; ulceration of mucous membrane of mouth and tongue; a foul smell from mouth.
2	104·2°	18	78, very small.	.....
3	100·6°	16, belly very full.	42	Membranes pale. .....
4	104·6°	14	76	Membranes yellowish; of eye slightly spotted.
5	103·8°	12	72	.....
6	99·8°	8	..	Urine scanty.
7	99·2°*	12	42	Spots in left eye.
8	102·4°	10, belly full.	54, very weak.	Conjunctiva very nasty looking, spotted; degenerating epithelium in nostril; eruption on the upper lip, very dull; hanging head; fell several times.
9	102·°	18	..	.....
10	101·9°	32	..	.....

* Thermometer not inserted deep, due to resistance.

Evacuations, where not otherwise recorded, normal. Urine collected under supervision in country jelly-pots and examined as to the following points:—

Urine of 29th November.

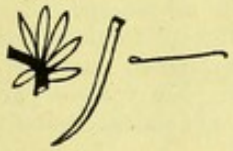
Case.	Temperature.	Specific gravity.	Color, &c.	Reaction.	After standing one night.
1	75°F	1,020	Viscid, yellow, clear ..	Very slightly acid ..	No sediment.
2	74°F	1,020	Viscid, light yellow, clear ..	Acid	Do.
3	74°F	1,010	Pale yellow, watery, clear.	Acid	Very pale.
4	74°F	1,013	Pale brownish dirty yellow, some sediment	Acid	A slight mucous sediment.
5	75°F	1,034	Thick, yellow	Acid	Milky.
6	None obtained.	..	....	....	....
7	75°F	1,040	Clear, yellow	Neutral	Upper stratum reddish.
8*	80°F	1,020	Thick strong-smelling dirty-looking brownish yellow ..	Strongly alkaline ..	Copious sediment.
9	75°F	1,030	Viscid, yellow, slight sediment	Slightly acid ..	No sediment.
10	75°F	1,030	Viscid, clear, bright yellow..	Acid	No sediment.

* I found on investigation that this urine was not quite fresh.

Urines Nos. 7, 8, 9, and 10 showed on test copious albumen ; no sugar nor bile constituents detected.

The following is a Specimen of my further investigations of urine :—

Morning of 27th November.

	Case.	I. Treat with HNO_3 and boil.	II. Allow to stand in Watch Glass.	III. Add KHO.
	1	Slight sediment, mainly mucous crystals.	No change	Needle-shaped crystals and flocculi.*
Uric and Hippuric Acids.	2	Coagulum	Slight milkiness ..	Flocculi.
	3	Precip. deep yellow; amorphous crystals.	No change	Slight deposit.
	4	 crystals, a few ..	Do.	Slight amorphous deposit.
	5	Dark brown deposit; amorphous granular.	*Nil*	Creamy change ; spicular crystals and flocculi.
	6	*Nil*—A red brown color ..	*Nil*	Slight flocculi.
	7	Coagulum, dark	 crystals, a few; also others.	Flocculi.
	8	Copious granular amorphous deposit.	No change	Granular crystaline deposit.
Hippurates.	9	Coagulum, granular ..	A little cloudiness ..	Flocculent deposit.
	10	Deep color, feathery crystals ..	Do. ..	Almost solidified ; flocculent coagulum.

The thread test, murexide test, and others were also resorted to, but not as a matter of routine, only for the settlement of dubious points.

At first, while the clinical features of the disease were being ascertained, the blood was not examined daily in each case, but the microscopes were used freely in examination of evacuations of various kinds, but without any substantial results. I also similarly examined the various articles of diet, water collected from wells and drinking tubs, soil, &c., but with no substantial benefit.

Later in the inquiry my methods changed somewhat, and I adopted a different form of table for clinical record, of which the following is an example :—

Saturday, 17th January 1885, *Temperature* 71° *at* 8 *a.m.*

No.	Temperature.	Respiration.	Pulse.	Parasites.	Membranes.	Urine. Specific gravity.	Urine. Reaction.	Characters.	Remarks.
5	100	18	48	None detected ..	Slight spots ..	..	..	....	(As the urine was not at this time being specially examined, it was less carefully selected and elaborately tested than earlier in the inquiry.)
7	99	16	48	One seen ..	Spots	10·50	Alk. ..	Slight cloud.	
12	102	24	72	Many nuclei ..	Membranes yellowish.	10·40	Acid ..	Clear viscid.	
13	103	20	52	Many ; active, free or adherent.	Membranes yellowish lachrymation.	..	..	....	
14	99	22	52	Some tessellated arrangement.	Slight ulceration near tushe, left side upper jaw.	10·30	Acid ..	Pale yellow, very cloudy.	
15	Obiit nocte.	..	..	None in blood ..	....	10·40	Slight acid	Very thick dark color.	
16	100	24	48	Many mulberry-shaped spherical bodies ; corpuscles look like spores.	....	..	..	....	
17	100·5	48	40	None detected ..	....	..	..	....	
18	98	32	40	Do. ..	Spots both eyes ..	..	..	....	
19	101	20	60	Do. ..	Spot left eye ..	10·40	Alk. ..	Slightly yellow, slight cloud.	
Pony.	98·5	16	36	Do. ..	....	10·30	Alk. ..	Light yellow, slight cloud.	
Bull.	101·5	24	60	Do. ..	....	10·36	Acid ..	Clear watery.	
Goat.	104	..	136	Do. ..	....	..	..	....	Nasal discharge.
Dog.	102	..	145	Many blood crystals.	....	..	..	....	....

General observations, work done, &c.—Post-mortem examination of No. 15 (D 181). The only ordinary appearances of relapsing fever were clots in the large vessels, the heart fat gelatinous, its muscular substance fatty, slight fluid in chest. Froth in trachea, some lesions of the lining membrane of the stomach obscured by the effects of the irritant and chemical action of the ammonia used in treatment which were marked on stomach, oesophagus, and bowels. Lungs actively inflamed. Kidneys and brain (with its membranes) congested.

My methods of inquiry into causes are explained in the text. All data are from the Returns in the *British Burma Gazette*, which I collected, tabulated, and drew up in the form of Graphic Records. A certain amount of literary work, apart from the actual drawing up of the report, has been carried out. This consisted in digestion and précis of various Government records, researches as to the literature of the subject and possible solutions of the problem as to the nature of the disease in periodicals and text-books—medical and veterinary. I am much indebted to Mr. Frost for (in addition to the numerous other ways in which he assisted me in my work at Rangoon) placing his library at my disposal. I claim for my exact record of the influence of physical and climatic causes on the production of the disease a sound practical value. The tendency in study of diseases due to micro-organism seems to be to allow such causes to be obscured by attention to the vital conditions of the parasite. I have tried to apply the older, practical, and the more recent biological methods to the investigation of etiology of equine relapsing fever.

The post-mortem examinations were all made as soon after death as practicable. The ordinary routine was adopted. Abnormal parts were placed at once into Müller's or Goulard's fluids, or into solutions of spirit, or else examined immediately under the microscope. Such rough semi-diagramatic illustrations of the conditions found as I was competent to make were carried out for illustration of the report. The principal fluids and tissues examined microscopically were as follow:—Blood, urine, abdominal and thoracic serum, lymph from various parts of the body, spleen pulp, heart muscle, gastric walls, conjunctival petechiæ, abdominal contents, lymphatic gland, kidney. The results will be found embodied in the report.

Such portions of the carcasses as were not consumed by vultures (among which animals no special fatality was observed in consequence) were carefully consumed by fire. The place of post-mortem examination was carefully disinfected and cleaned.

All observations made in the sick lines were entered in a register and record case book in the usual official manner. From the records a thermographic chart (giving full details of the case) was prepared. From each individual case pathological deductions were obtained and recorded. Many a result casually mentioned in the body of the report represents a week's careful work.

My clinical observations were carried out by me in full detail for the first month of the inquiry; subsequently, except during my absence at out-stations, the records were every morning made under my personal supervision; generally also in the evening I attended to make observations and supervise. This discipline was relaxed somewhat towards the last when the main facts had been ascertained and special details had to be followed out. My assistants, as trained by me, attained a very creditable amount of manipulative skill. All experimental cases were treated clinically in the same way as natural cases. My subjects were several old and worn-out mules, one pony, one bullock, one monkey, one dog, and one goat. The principal experiments were —

Mules—(*a*) Twice administration of gastric contents from affected mule through the mouth.
(*b*) Subcutaneous injection of blood of a mule in the active fever phase mixed with water.
(*c*) Gastric ingestion of a considerable amount of similar blood.
(*d*) Inoculation with a mixture of water and virulent blood thoroughly dried.
(*e*) Inoculation with blood in which no parasites were detectable on microscopical examination, but from an affected patient.
(*f*) Inoculation with blood from the heart of a recently dead patient taken before coagulation.
(*g*) Inoculation with blood of a feverish animal drawn directly from the jugular and at once thrown into the subcutaneous tissue.
Pony—(*h*) Transmission of disease by inoculation with blood of a feverish mule (spirilloids present).
Bullock—(*i*) Unsuccessful inoculation with spirilloid containing blood from a mule (only local lesions resulted twice).
(*j*) Unsuccessful injection of spirilloid containing blood into the peritoneal sac.
Goat—(*k*) Unsuccessful inoculation with spirilloid containing blood from a mule (twice).
(*l*) Injection of similar blood into the peritoneal sac; result dubious.
Dog—(*m*) Inoculation by subcutaneous injection of spirilloid containing blood.
Monkey—(*n*) Inoculation by subcutaneous injection of spirilloid containing blood.

A number of minor experiments were performed.

I was enabled, from the Tonghoo Death Reports, to supplement my Rangoon records with those of cases at Tonghoo.

The following are examples of my digests of these reports :—

"Case 38—*23rd November* 1884.—*President*, Lieutenant-Colonel Foord, 3rd P.L.I.; *Members*, Lieutenants Bryant, R.A. and Jackson, 2nd Bedfordshire Regiment, and Surgeon Williamson.

Evidence of Inspecting Officer (2nd Bedfordshire Regiment.)

History.—Mule of 2nd Bedfordshire Regiment, well and strong until a fortnight ago; was admitted with usual symptoms; staggering and evidently suffering great pain in head; for two days has appeared unconscious and violent.

Post-mortem Appearances.—Body emaciated; large intestines slightly inflamed throughout, small healthy; stomach mucous membrane covered with small nonpenetrating circular ulcers; kidneys both slightly congested; heart healthy; apex of right lung slightly tubercular; brain healthy.

Case 39—*5th December* 1884.—*President*, Captain Campbell, 2nd Bedfordshire Regiment; *Members*, Lieutenants Bryant, R.A., and Jackson, 2nd Bedfordshire Regiment.

History.—Mule of 3rd P.L.I. for more than two months unfit for work; on 24th November seized with symptoms of the prevalent disease; treated in accordance with instructions from Rangoon; died at 7 A. M. on 5th.

Post-mortem Appearances.—Stomach inflamed and eroded; lungs congested; heart enlarged; brain slightly congested; other organs healthy.

I have in all 50 such records of cases, more or less full. I have found them most valuable. My official correspondence has comprised—

(*a*) Indents and other papers connected with supplies.
(*b*) Progress reports made to you periodically.
(*c*) Official letters reporting movements, requirements in pursuit of the inquiry, &c.
(*d*) Detailed report of the investigation to Inspecting Veterinary Surgeon Evan's office, with appendices, &c.
(*e*) Scientific correspondence on the disease with Dr. Carter and others.
(*f*) Reports after visits to the out-stations of British Burma.
(*g*) Report on salootries; also official correspondence in connection with the veterinary management of the British Burma Transport during my stay in Burma.

The last not associated with the inquiry into relapsing fever, but in accordance with my original and subsequent instructions from the office of the Inspecting Veterinary Surgeon.

The following visits have been made in performance of the duties entrusted to me in British Burma, and especially to determine points for my report :—

	Days.
1. To Tonghoo, 20th December 1884 to 2nd January 1885	13
2. Thayetmyo, 17th to 23rd February 1885	6
3. Moulmein, 3rd to 6th March 1885	4
Total ...	23

The following periods were occupied in the journey to and from Burma :—

Journey from Bangalore to Rangoon *viâ* Madras and Calcutta, 2nd to 16th November 1884	14
Journey from Rangoon to Bangalore *viâ* Madras and Coast ports, 11th to 22nd March 1885	11
Total ...	25
Actual number of days spent in Rangoon	92
Total period of service with the British Burma Transport and in the inquiry	115
Total period occupied in special service	140

Among the various duties performed by me during my stay in Burma have been castration of Government mules and instruction of salootries and transport officers in the method of performance of the operation, operations on elephant, a number of miscellaneous operations on Government animals, advice and suggestions as to animal management at the different stations, examinations of silos, grass lands, and the question of forage supply in general (which has demanded and received much notice from me), inspections of freshly arrived cattle, treatment of medical and surgical cases, advice in cases of suspected or actual farcy, and so on.

The actual details of daily work have been dealt with in my periodical progress reports to the office of the Inspecting Veterinary Surgeon, Madras Army.

P.S.—A considerable amount of time during the last month or so of my stay in Burma was occupied in staining the specimens of parasite-containing blood for preservation, future inquiry, and despatch to experts. I retain a number of these specimens and shall make use of them; also my experimental monkey, which is not yet dead. My dog, pony, and several mules died from the effects of inoculation. A few cases remained at Rangoon for experimental treatment. I slaughtered the goat for post-mortem examination.

(Signed) J. H. STEEL, V.S.,
Army Veterinary Department.

SPECIMEN CASES OF RELAPSING FEVER IN MULES.

[True extract from Record Case Book.]

Case I—B. 57.

Month.	Day.	Time.	Rectal Temperature.	Respiration.	Pulse.	Details.
September.	17	..	..	..	..	Admitted to sick lines.
October.	21	..	..	..	..	Sent out apparently well; soon fell off his feed; again began to eat and was given double feeds until this date when he was re-admitted.
November.	10	..	..	..	..	....
,,	17	..	..	..	..	Staggering considerably; has lost much flesh. Cease carbolic treatment.
,,	18	7 a.m.	..	18, forced.	54, good	Near hind leg a little swollen; rolls a great deal when in stable.
,,	,,	5 p.m.	106·2°	36, irregular in expiration, deep.	96	Visible mucosæ very pale.
,,	19	7 a.m.	106·6°	18	66	Temperature at nostril 100·4°; membranes a little yellow; fæces normal.
		2 p.m.	106°	..	..	....
		5 p.m.	105·4°	42	84	Ulceration inside right labium vulvæ perhaps due to injury, for fell twice during the day and is now staggering much. Attempts to resent temperature-taking.
,,	20	8 a.m.	102·8°	42	60	....
		4 p.m.	104°	24	72	....
,,	21	M.	103·9°	18	72	Membranes pale yellowish; hind legs more filled, especially the off.
		E.	103·6°	36	60	....
,,	22	M.	98·6°	15	44	Very many blood-spots in eyes; fresh in left, purple in right; mouth and limbs cold; much inclined to eat bedding; discharge from vulva; "Horsing" (?).
		E.	100·4°	31	58 (unequal).	....
,,	23	M.	99·8°	23	52	Red spots still present on conjunctiva; won't eat food off ground; dung good.
		E.	..	..	..	....
,,	24	M.	99·5°	13	52	Dung good; conjunctiva pinkish and spotted; off feed.
		E.	101·1°	16	54	Dung dark-colored and plentiful; free lachrymation; a small ulcer on left labium.
,,	25	M.	100·5°	12	48 (weak).	Urine acid.
		E.	103·2°	11	56	Dung good; left eye spotted and lachrymose.
,,	26	M.	103·5°	12	72 (very small)	Spots continue in eye; "broken" expiration.
		E.	105·2°	14	60	Urine neutral, much uric and hippuric acids.
,,	27	M.	104·3°	12	72	Membranes still spotted; still off feed; incipient ulceration of mouth.
		E.	105·6°	13	66	Urine acid, clear, yellow; sp. gr. 10·22.
,,	28	M.	104·4°	12 (much disturbed).	54 (very weak)	Blood extravasation left nostril; conjunctiva a few spots; ulceration of mouth and tongue.
		E.	106·2°	17	84	Mouth very foul; dung darkish; lying down a good deal; urine albuminous.
,,	29	M.	104·2°	12 (deep).	60 (good tone)	A little bloodiness of left nostril, mucous membrane of eye much better; still off feed; urine viscid, clear, yellow, slightly acid; sp. gr. 10·20.
		E.	101·8°	16	54	Ulceration of vulva healing; nostril scab shed and a small ulcer left.
,,	30	M.	99·6°	16	48 (small)	Fresh-looking spots in eye, lachrymation; ate a little feed; abrasion left nostril, a spot in the right.
		E.	102·2°	16	60	....
December.	1	M.	101·6°	16	60	Spot in left nostril.
		E.	102°	12	54 (small)	....

CASE I—B 57—*continued.*

Month.	Day.	Time.	Rectal Temperature.	Respiration.	Pulse.	Details.
December.	2	M.	100·4°	8	60	Ulcer left nostril; eats much bedding, but no feed.
		E.	102·4°	12	60	Urine viscid, clear, acid; sp. gr. 10·30.
,,	3	M.	101·8°	12	60	Ate whole feed; membranes healthy.
		E.	105·1°	12	60	Urine strong smelling, alkaline, cloudy; sp. gr. 10·16 (probably not fresh).
,,	4	M.	103·8°	12	60	Membranes look healthy.
		E.	104·6°	16	66	....
,,	5	M.	105·4°	16	66	Two symmetrical ulcers on tip of tongue.
		E.	105·7°	12	84	Urine neutral, clear, yellow; sp. gr. 10·24.
,,	6	M.	104·2°	15	60	Much "tucked up," swelling near hind leg; many large purple spots left eye.
		E.	104·4°	16	66	....
,,	7	M.	100·6°	16	48	Two sluggish ulcers, one in front of each ear; petechiæ left eye.
		E.	101·8°	16	60	....
,,	8	M.	99·1°	10	48	Blood-spots continue in left eye, looks very dull.
		E.	102·6°	14	52	....
,,	9	M.	100·4°	16	84 (small)	Slight ulceration of lip, very dull; slight spots right conjunctiva.
		E.	101°	16	60	....
,,	10	M.	100·4°	18	54	Left eyelid œdematous; mucous discharge and much lachrymation.
		E.	104°	12	66	....
,,	11	M.	103·2°	13	60	....
		E.	103·2°	14	60	....
,,	12	M.	102·9°	15	66	Numerous parasites in the blood.
		E.	104·0°	19	84	Parasite in blood; lying down and looking at left side.
,,	13	M.	98·0°	39	100	Got down at 6 A.M.; incipient ulceration of cornea; circulation irregular; small vaginal blood-spots; parasites in blood; died at 10 A.M.

AUTOPSY (at 5 P.M.).—Good death stiffening. Lymphatic vessels and glands engorged. Gelatinous material between some of the muscles, such as those of the hind legs. Some serum in pleural and peritoneal sacs. In spite of the emaciation of the animal a good deal of internal fat. No petechiæ or blood extravasations. Liver weight, 7¼ lb.; organ apparently quite healthy. Stomach with contents, 14 lb. Its cuticular part normal; its villose a little congested, the organ full of well-masticated matter in active digestion. Œsophagus throughout healthy; a few circumscribed local congestions of the small bowel, not at Peyer's patches; terminal portion of the ileum has its mucous layer flocculent, and reddish. Large intestine well filled, healthy; rectum contains a few hardish dung-balls with reddened mucus in spots on surface; abdominal aorta contains a clot from beginning to end; spleen weighs 2¾ lb., enlarged; heart without blood, 3¾ lb., fatty, choked with blood in various colored clots ranging from green to deep maroon. This gives the organ a very peculiar and striking appearance. The right side is the main seat of distension. Left lung congested and its vessels choked with green clots. The lymphatic glands at anterior thoracic opening very large. Cervical great lymphatic conspicuously full. Trachea contains much froth. Brain anæmic and a little soft. Adrenals somewhat congested. Kidneys quite normal. Discharged —died.

CASE XIII—B. 70.

September 27*th.*—Admitted to treatment.

October 8*th.*—Returned to duty.

October 17*th.*—Re-admitted with fever; entirely off feed; also "giddy."

November 17*th.*—Extreme emaciation; lying recumbent, broadside, apparently more dead than alive. Has numerous nasty bed-sores on both sides. Is lying on a bed rendered most offensive by urine accumulation, covered by a net to keep crows and vultures away. Reported to have been down now about six days; passes some dung occasionally and frequently some strong-smelling urine. Has lately received carbolic doses (which cease). Has been supported with conjee, but not with much hopes of benefit, for it is considered that "once they get down they seldom rise again." Mouth most foul and filled with sordes. Conjunctiva of left eye sloughing. A very nasty and neglected looking wound above this eye, and there is an opacity of the centre of the cornea. Occasionally the uppermost legs quiver.

November 18*th* (7 *a.m.*)—Pulse 60, of fair tone; respirations 18 (labored); near hind leg cold; the other limbs warm.

November 18*th* (5 *p.m.*)—Temperature 105·2° F.; pulse 66; respirations 18, deep; some dark-colored urine passed; anus irritable.

November 19*th* (7 *a.m.*)—Pulse very indefinite and irregular, 64·80; constantly stretching and quivering the legs, and inclining the muzzle to the *right* side even after turned over. Right

conjunctiva looks better; left cornea quite opaque, and the side which is undermost is always bathed in sweat. A fair dropping of hardish dung, coated with dark viscid material, mucus and bile, very foul smelling; respirations 72, labored. Temperature at rectum 105·1° F., at nostril 100·6° F.; visible mucous membranes moist, of good color.

November 19th (2 *p.m.*)—Rectal temperature 106° F.

November 19th (5 *p.m.*)—Rectal temperature 107° F.; pulse 60; passed a lot of soft, chocolate-colored fæces, very strong smelling; was turned over and rubbed dry; more urine passed.

November 20th (2 *a.m.*)—Died.

Post-mortem examination (*after five hours.*)—A good death stiffening; gelatinous effusion in areolar tissue in front of chest, between muscles of thigh, and at upper part of sternum, and here and there subcutaneously. Œdema of the lymph glands. A large quantity of milky fluid in peritoneal sac. A fair amount of serous fluid in the chest. Enormous clots in the large blood-vessels, mainly yellow, but with purplish accumulations of red corpuscles on the surface; portal vessels full. Bowels; here and there patches of vessels full of blood and slight extravasations of corpuscles, sufficient throughout the small intestines as a whole to account for the nature of the contents of the large bowel. A fair amount of serum-like fluid in the ileum. Glands of the bowel free from disease. Large intestine containing foul-smelling, grumous, coffee-colored fluid, but its walls healthy and no parasites present. A dark deposit on under-surface of left kidney and much congestion of that organ. Right kidney much congested and œdematous. Urine, specific gravity reading post-mortem 10·35; no deposits. Spleen perhaps a little large and full, otherwise no abnormality detected. Liver normal, of good size, free from parasites. Stomach presented special and characteristic ulceration of its cuticular portion; *one perforation*. Mucous membrane somewhat yellow. Contents, only a little rice, the rest having escaped through the perforation into the belly. Right lung deeply congested, left less so; some blood extravasations on the surface, and some along the course of intercostal veins. Heart a little fatty; extra-vasations along its vessels, otherwise normal. Trachea full of froth; tongue, fauces, &c., healthy. Brain membranes normal; a fair amount of arachnoid fluid. Brain substance anæmic and looks sodden Spinal cord (carefully examined microscopically) no disease. Discharged—died.

Case XII—B. 54 (Illustrative of diagnosis of the disease.)

Dec. *9th*, Admitted as a fresh case. Reported as very weak and in poor condition.

		Temp.	Respr.	Pulse.		Temp.	Respr.	Pulse.
	morning	101·4° F.	...	48	Evening	101·8°	30	42
„	10*th* „	100·4° F.	12	42	„	102·4°	...	...
„	11*th* „	99° F.	36	48				
„	21*st* „	104·4° F.	20	64	(by Corporal Hill).			
„	22*nd* „	102·8° F	20	48				
„	23*rd* „	102·2° F.	20	48				
„	24*th* „	97·6° F.	20	40	Very cold; eye-membranes spotted; down.			
„	25*th* „	102·2° F.	27	52	Still down.			

Dec. 26*th*, died at 1 p.m. Autopsy showed disease of the cuticular portion of the stomach, extensive ulceration and yellow degeneration, the muscular coat being exposed in two places; villous portion healthy. Discharged—died. (This is a case which was not properly diagnosed at first, because it came under notice when the pulse, &c., were fairly normal. More prolonged inquiry would have shown the relapse which was just ending on 21st December.)

SUMMARY OF MEDICAL HISTORY OF THE EXPERIMENTAL MONKEY.

The patient was a young female monkey of the common Burma variety. It received spiril-loid containing blood beneath the skin of the inner side of the thigh on 2nd February 1885, and scratched the parts somewhat after administration. On the third day after inoculation the parasites were numerous and active in the blood. A rise of temperature occurred on the evening of the second day; some swelling took place at the seat of inoculation. A reddish discharge from the vulva was one of the earliest events following inoculation.

The red corpuscles seemed to become much shrivelled under the action of the parasites; the patient suffered from high fever. On the morning of the fifth day after their first appearance the parasites became non-detectible and remained so for four days.

The patient was brought over to India and suffered from numerous relapses; became very thin and weak, and with a haggard, drawn, expression of countenance.

After the second month marked deficiency in power in the legs set in, and later the feet began to swell. Ulcers of large size formed on them and burst; the bones of the foot became exposed; finally the upper eyelids became œdematous and the animal died after a few hours of coma and slight delirium.

Autopsy.—Gave no marked positive results. The lungs were a little petechiæted on their surface, and their was *no* ulceration of the gastric mucous membrane.

J. H. S.

THERMOMETER CHART.

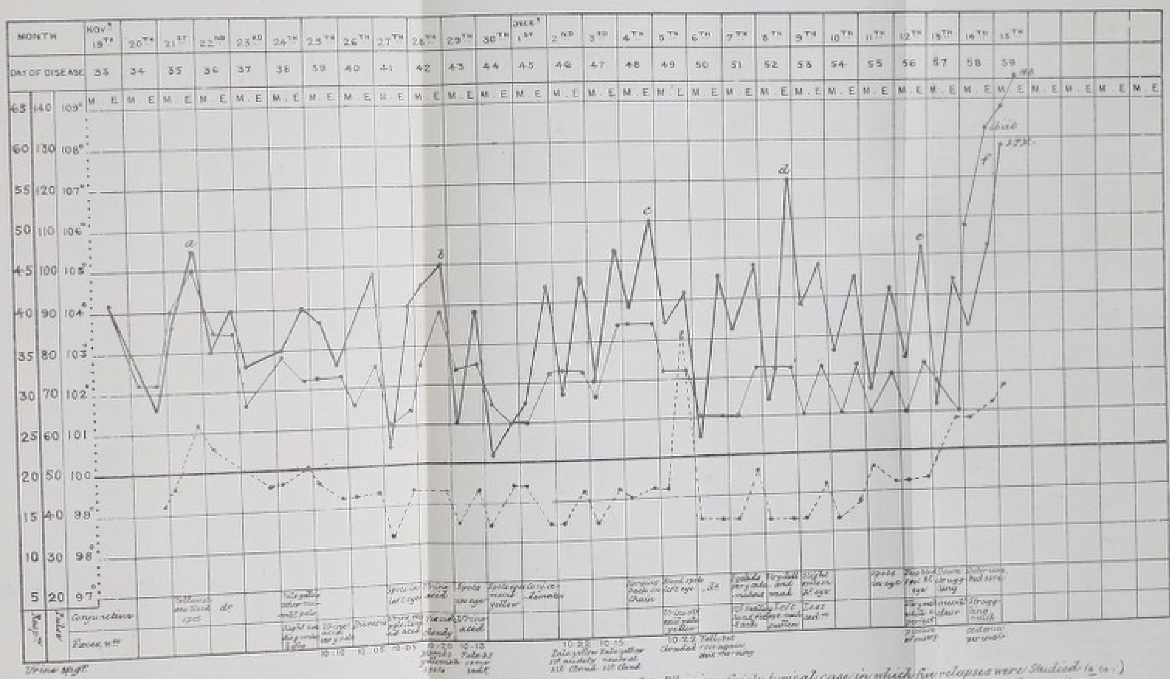
2[nd] SOMERSETSHIRE REGIMENT TRANSPORT MULE

STATION RANGOON

RELAPSING FEVER

Case-book No. 2 Letter A No. 39 Age 10 Colour Lt. Bay Sex G Disease (Gastric Typhoid) Date of Admission 17th Octr. 1884 Date of Discharge 15th Decr. 1884 Result Died.

N.B. In these Charts the thick continuous line represents the Temperature Curve; the thin continuous line the Pulse Curve and the discontinuous line the Curve of Respiratory efforts per minute.



Autopsy: Much gelatinous greenish material thrown out between muscles of neck & abdomen. No surplus fluid in belly. Bowels, in only one place, the seat of circumscribed congestion. Numerous entozoa in bowels. Kidneys both diseased, right one whitish & glistening, in its cortical portion especially. Stomach extensively ulcerated and losing its epithelium. The clotting of blood in the small vessels of the ulcers very well marked. Villous portion much thickened throughout the greater part of its extent. Spleen enlarged, wt. 4 lbs.; Liver studded throughout with miliary calcareous spots, wt. 11½ lbs.; clots in vessels not marked; lymphatic glands engorged with lymph; Brain & its membranes normal; spinal cord not examined. Trachea contd. froth; Lungs congested; Heart fatty, no petechiæ nor extravasations; some clear fluid in chest.

Remarks. This is a fairly typical case in which five relapses were studied (a to e). A happy observation two days before death showed the presence of parasites.

THERMOMETER CHART

TRANSPORT SUPPLY DEPÔT

STATION RANGOON

Case book No. 58 Letter ...... No. ...... Age Abt 12 yrs Colour Br. Sex M Disease Artificially induced Relapsing Fever Date of Admission 17th December 1884 Date of Discharge 31st Jany 1885 Result Died.

RECORD No. XV

EXPTL. CASE No. 1

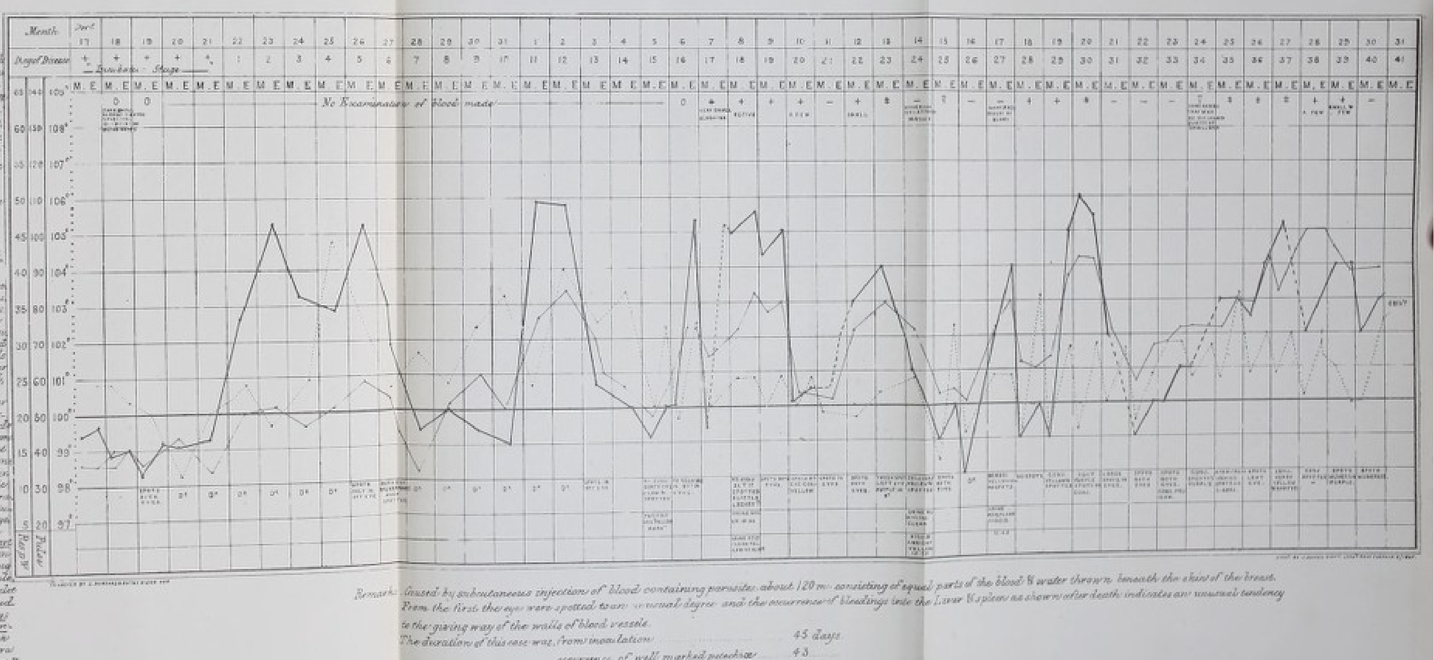
HISTORY OF THE CASE

An oldish thin male, of which the normal temperature ranged between 98°F & 100°F. The temperature began to rise the fourth day after inoculation and a very typical attack of relapsing fever occurred. There being some five relapses & the characteristic symptoms well developed.

The fatal result took place on the morning of 41st day.

POST MORTEM APPEARANCES

Cougar of long standing in ... vessels. Gelatinous extravasations between thigh muscles. Lymphatic glands not much enlarged, much sero sanguineous fluid in belly. Diffused peritoneal congestion. Bowels normal. Stomach; particular ... somewhat ulcerated, wrinkled as it were, with ... bluish black matter in the furrows. Villous ulcerated in spots & lines towards pylorus, looking as if the membrane had been rent, but apparently only degeneration. ... parts inflamed, many ... present but ... Spleen; one old ... & one recent blood ... Liver, somewhat congested, a large blood tumour on ... surface of right lobe. Heart much gelatinous matter in furrows & blood extravasations along ... its structure a little pale. A small old & large recent clot in pulmonary artery (immediate cause of death). Lungs rather sodden. Brain & spinal cord normal. Some dark ... coloured fluid in pleura & pericardium ... blood extravasation in right supralumbar muscle. Kidneys congested. Other organs normal.



Remarks. Caused by subcutaneous injection of blood containing parasites about 120 m. consisting of equal parts of the blood & water thrown beneath the skin of the breast. From the first the eyes were spotted to an unusual degree and the occurrence of bleedings into the Liver & spleen as shown after death indicates an unusual tendency to the giving way of the walls of blood vessels.

The duration of this case was, from inoculation .... 45 days

occurrence of well marked petechiae 43

first rise in Temperature 40

No appreciable local phenomena resulted at the seat of inoculation.

The method in which death was brought about is exceptional, the accident of the blood clot considerably shortened the case.

THERMOMETER CHART.

A CASE OF RELAPSING FEVER COMPLICATED WITH SUBACUTE PNEUMONIA

2ND SOMERSETSHIRE REGIMENT TRANSPORT MULE

STATION RANGOON

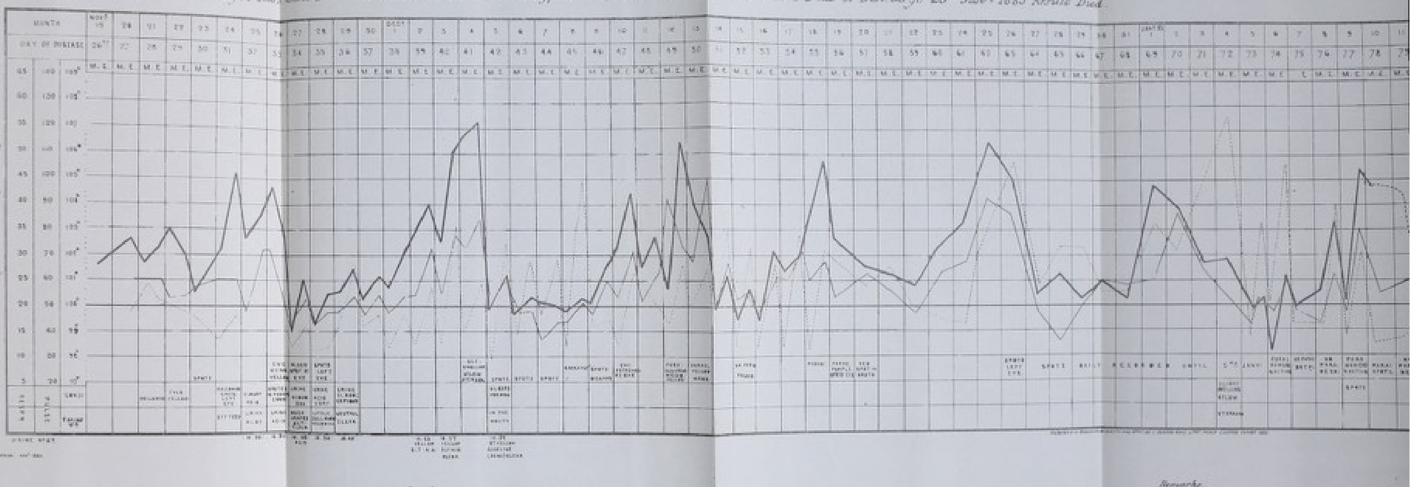
Case book No. VII Letter A No. II Age 9 Years Colour [illegible] Sex Stallion Disease Gastric Typhoid (Sic) Date of Admission 24th Octr. 1884 Date of Discharge 25th Janry. 1885 Result Died.

Details of History.

[illegible]

P.M. Appearances.

[illegible]



Remarks

(1) [illegible]

(2) [illegible]

(3) [illegible]

(4) [illegible]

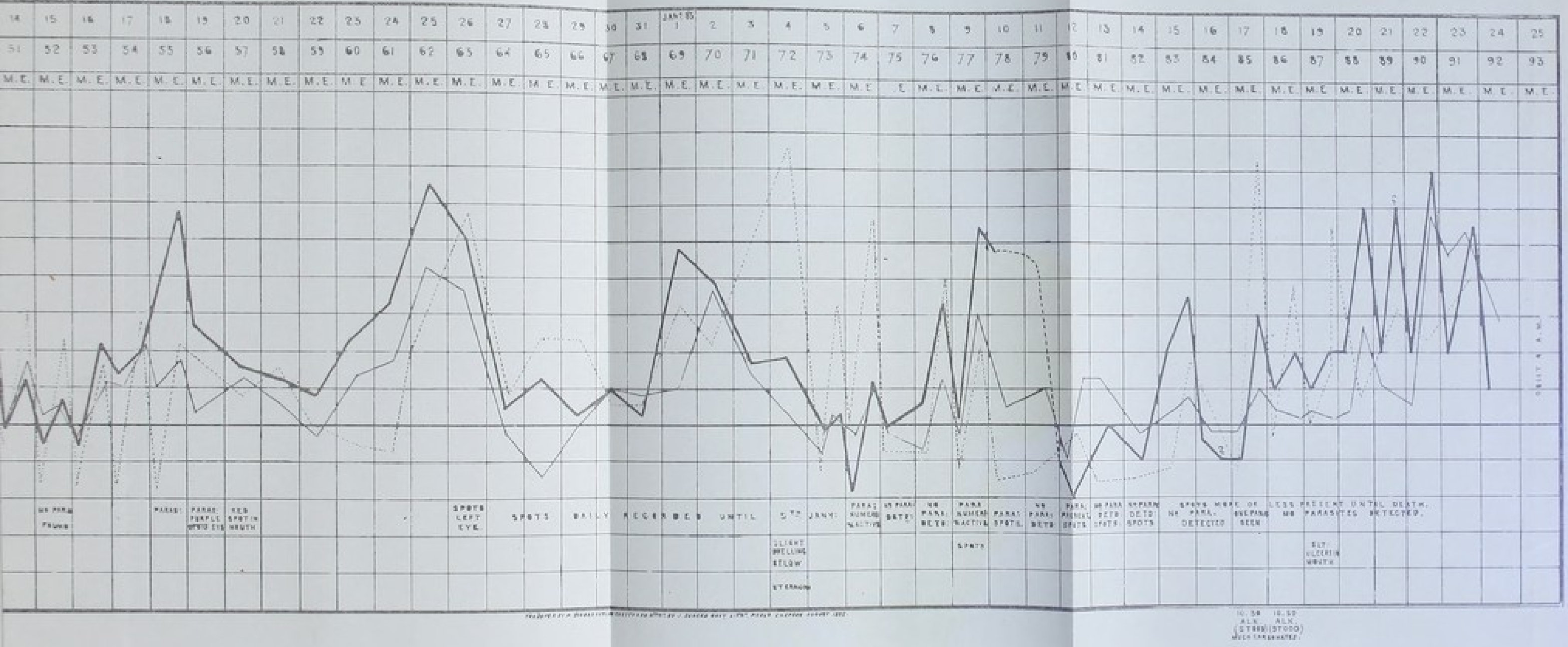
(5) [illegible]

(6) [illegible]

Remarks.

MONIA

n 24th Octr 1884 Date of Discharge 25th Jany 1885 Result Died.



Remarks,

...ments became

... and is strange,

...imens at

...ature curve

...ast.

A Case of Relapsing Fever Complicated with Liver Disorder.

THERMOMETER CHART.

2ND SOMERSET SHIRE REGIMENT TRANSPORT MULE

STATION RANGOON

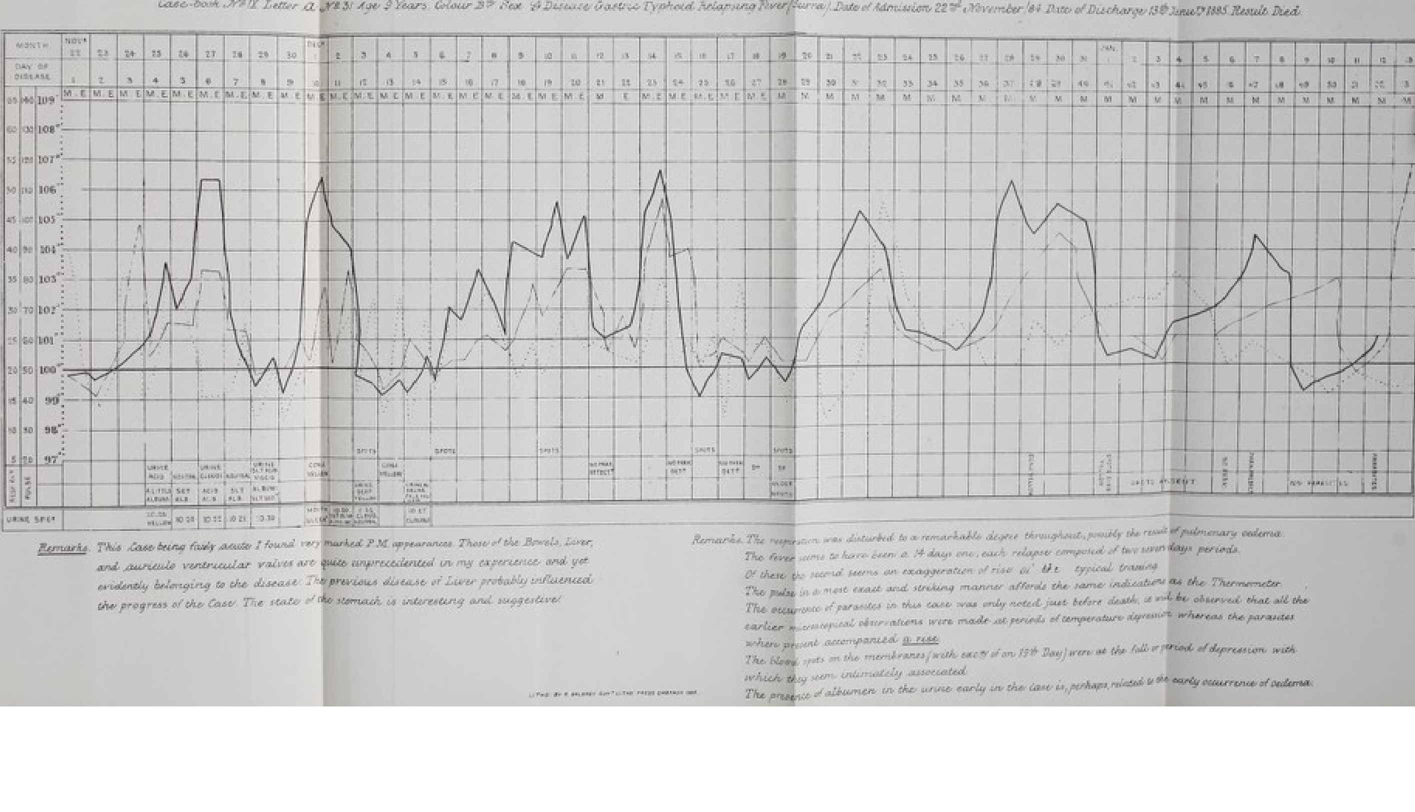
Case-book No. IX Letter a No. 31 Age 9 Years. Colour Bn Sex ♀ Disease Gastric Typhoid Relapsing Fever (Surra). Date of Admission 22nd November '84 Date of Discharge 13th January 1885 Result Died.

Details of History

…s and legs were swollen from …t first and the chan. oedematous: …ly occurrence of ulceration in …e mouth indicated its asthenic …aracter. Swelling of the penis …1st December showed the drop- …sical tendency, as also did the …ardia of neck and perineum shortly …fore death.

P.M. Appearances

…ensive gelatinous deposits. Heart …tty weight 4 lbs 6 ozs. Blood …staves in auric. vent. valves. …ous in chest and belly. …ng, right, congested and oedema- …tous. Lymph glands much enlarged. …cous layer of small intestine exten- …sively congested throughout its pos- …terior 1/3, the Peyer's patches being …e main seat. A film of coagulum co- …vering the surface of the membrane. …ver weight 11 lbs 20 ozs., congested, …s studded with calcareous spots. …pleen enlarged, weight 2 lbs 1 oz. …tomach slight unimportant ulcera- …tion of long standing and practical… …s of cuticular coat. It looks …s tho' the muscular coat had been …osed but again had become cover- …d. Extensive clots in heart and …arger vessels. Brain anaemic.



__Remarks.__ *This Case being fairly acute I found very marked P.M. appearances. Those of the Bowels, Liver, and auriculo ventricular valves are quite unprecedented in my experience and yet evidently belonging to the disease. The previous disease of Liver probably influenced the progress of the Case. The state of the stomach is interesting and suggestive.*

Remarks. The respiration was disturbed to a remarkable degree throughout, possibly the result of pulmonary oedema.
The fever seems to have been a 14 days one, each relapse composed of two seven days periods.
Of these the second seems an exaggeration of rise of the typical tracing.
The pulse in a most exact and striking manner affords the same indications as the Thermometer.
The occurrence of parasites in this case was only noted just before death; it will be observed that all the earlier microscopical observations were made at periods of temperature depression whereas the parasites where present accompanied a rise.
The blood spots on the membranes (with exc. of on 19th Day) were at the fall or period of depression with which they seem intimately associated.
The presence of albumen in the urine early in the case is, perhaps, related to the early occurrence of oedema.

Lithd. by … Govt. Litho. Press … 1885