KALA AZAR

IN SHENSI PROVINCE OF NORTH CHINA

by

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KALA AZAR IN SHENSI PROVINCE OF NORTH CHINA.

Personal observations on 535 cases of visceral leishmaniasis, diagnosed in the kala azar outpatient department clinic of the Jenkins-Robertsen Memorial Hospital, Sian Shensi, from March 1940 to January 1942.

INTRODUCTION.

An outpatient clinic for the treatment of kala azar was started in March 1940 owing to the increasing number of cases of kala azar which were presenting themselves for treatment.

The clinic was carried on for almost two years, during which time 535 cases under my personal supervision were diagnosed by the finding of Leishmania Donovani bodies. A further 37 cases also diagnosed by the same methods during this period are omitted, as they did not come under my personal observation. Four hundred of these cases were either given a full course of treatment or died during treatment. The remaining 135 cases were lost trace of during treatment, so that no reliable estimate of the results could be made. The hospital to which the clinic was attached is a very busy one, serving a large area with few medical facilities, so that inpatient accommodation was only available for the most serious cases, and not always/

always for these. The clinic was run throughout the period under difficult war time conditions, many of the cases being drawn from a constantly changing refugee and military population. Sian was less than 100 miles from the front line throughout the period and the city was frequently bombed by Japanese planes. On two occasions there were partial evacuations of the city's population. The clinic was only a small part of a very busy hospital routine in which the author had a full share, so that a very limited part of his time was available for the clinic and practically no personal follow up work could be done. All these conditions made follow up work and estimation of final results much less satisfactory than could be desired. The clinic was carried on without the aid of any research grants, but a small proportion of the antimony compounds used was subscribed by various relief bodies. My work in the clinic was brought to an abrupt close when I was called up for active service in the R.A.M.C. in January 1942. This was unfortunate in so far as it prevented the full follow up work on an interesting series of cases treated with the aromatic amidines (May and Baker 744 or 4:4 Diamidine Stilbene dihydrochloride.) This compound was giving excellent results on short term observation, and one had hopes that long term observation would prove equally satisfactory.

Scope of Thesis. It is proposed to review kala azar in Shensi province where it was first described by me in 1941(3), to discuss 535 cases and to make comparisons with recent work on the disease in China and other parts of the world.

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HISTORICAL AND GEOGRAPHICAL SURVEY.

Kala azar was first described by Clarke in Assam in 1882[1]. Cunningham[2] was the first to see Leishman Donovan bodies in 1883, but failed to recognise them. Leishman first described Leishman Donovan bodies in 1903, and Donovan confirmed the work in India. In 1904 the first case of kala azar in China was diagnosed post-mortem by Marchand in Leipzig, on a German soldier who was invalided home from Peiping and Tsingtau in North China. Leishman Donovan bodies were found from smears of the spleen.

There are records of kala azar being diagnosed clinically in the Jenkins-Robertsen Memorial Hospital, Sian, over 20 years ago, but it is only in the last ten years that they have been confirmed by the finding of Leishman Donovan bodies. No report has been published on kala azar in Shensi, or regions to the west of Sian, until part of the present series was reported by me in the China Medical Journal[3].

Before this clinic for kala azar was started, thirty to forty cases of kala azar were diagnosed per annum in our hospital. In 1940, 197 cases were diagnosed and from January 1941 until February 1st 1942, 338 cases were diagnosed by the finding of Leishman Donovan bodies.

This great increase can be attributed to several factors. (1) There was undoubtedly an increase due to refugees and/

and soldiers from non-endemic areas, who were more susceptible to infection on coming in to an endemic area. (2) Our hospital was at this time practically the only one in Sian with supplies of the pentavalent antimony compounds available for treatment. (3) Successful treatment in the clinic and free treatment for these who could not pay, attracted fresh cases from villages in the surrounding countryside, and even from far off areas. (4) It is possible that there was also an increase in the local incidence of kala azar with the disease assuming epidemic proportions. This last factor is difficult to estimate, for the area from which we drew our cases was as large as England, and I would estimate that we were treating not more than 1% of the cases affected in the area.

Geographical and Climatic Considerations.

Manson-Bahr[1] and Wenyon[2] both state that kala azar occurs in China, north of the Yangtze river and between the coast and a line joining Peiping and Hankow. Shensi province lies north of the Yangtze river but 300 miles west of the line described. Manson-Bahr[1] accompanies this statement with a map which purports to show the "endemic area", This area extends well into the central plain of Shensi province and it is difficult to see why this district was included, since no report of kala azar in Shensi appeared in the literature before 1941[3].

Manson-Bahr[1] also mentions Canton as a possible area, quoting Schretenmayr, who described 83 cases diagnosed among soldiers in Canton during 1938. Dr O.K. Khaw in a personal communication expressed considerable scepticism about Canton being an endemic area. Chinese troops in war move over large areas of the country and Khaw is of the opinion that if these cases were kala azar, they had been infected in other areas. It is extremely doubtful if any area south of the Yangtze river is an endemic focus, although sporadic cases have occurred near Nanking and Changsha; even north of the Yangtze the disease is not endemic in all areas, there being heavily infected foci throughout, somewhat similar to the patchy distribution in the Sudan. The huge area of Szechuan lying north of the Yangtze in west China is practically free of kala azar, though one or two cases have been found in children born in Chengtu in that area. (T.H. Williams, West China Union University, Chengtu. Personal communication.)

In the course of extensive travels through west and south China in the last four years and in conversation with medical men from widely separated areas, I have not been able to find reliable evidence of endemic foci south of the Yangtze river.

The most heavily infected areas in north China are probably in Shantung, Kiangsu, Honan and Anhwei provinces, round Peiping/

Peiping, and in the central area of Shensi province, forming the basin of the Wei river, a tributary of the Yellow river. These areas comprise roughly the vast plain of north China drained, and frequently flooded, by the Yellow river. The basin of the Wei river in central Shensi, with Sian as its centre, is geographically and climatically part of this north China plain. The soil is loess and the common crops are wheat and millet. The climate is dry with extremes of heat and cold. In summer the temperature rises to 110 F. and in winter falls to 10 F. and occasionally to zero. It is an area of recurring famines from drought, and in the 1929 famine, it was estimated that one third of the population, or two million people, died of starvation.

The northern area of Shensi province is similar to the contiguous mountainous area of north Shansi, and neither of these areas is heavily infected with kala azar. South Shensi, south of the Tsinling range of mountains, is geographically part of central China (Yangtze basin) with heavier rainfall and growing rice. This area is practically free of kala azar, and I have not been able to trace a single case which developed infection there. This is curious as the contiguous and geographically similar area of Hupeh province is fairly heavily infected.

The people of central Shensi are typically northern Chinese/

Chinese in diet and habits and there are about 3% of Mohammedans. There are many dogs in the area and there is a definite sandfly season from May till July, but continuing to a lesser degree till September.

New areas of kala azar infection have been described recently in S. America, Russia and the Mediterranean basin. I would describe the central plain of Shensi as a heavily infected zone, not previously reported on.

EPIDEMIOLOGY.

The epidemiology of kala azar in Shensi conforms to type; it is sporadic, but may at present, owing to war conditions and migrating population, be assuming epidemic proportions. Since the Sino-Japanese war, subtertian malaria, and ankylostomiasis, diseases which were rarely seen before, are now becoming serious problems in this area.

The very recent work on the epidemiology of kala azar in China, started by C.U. Lee and O.K. Khaw, and carried on with enthusiasm by H.L. Chung, L.C. Feng, J.P. Lu and others, (4,5,6,7,8,9,10,11,12 and 13) goes far to prove that Chinese kala azar is similar in its epidemiology to that found in Crete, Sicily and the Mediterranean basin, and dissimilar to Indian kala azar. That is to say human and canine Leishmania coexist in Peiping.

Chung[7] and his co-workers have established the following/

following points suggesting that human and canine infection are one and the same parasite, and that the dogs form a reservoir host with a local sandfly Phlebotomus Chinensis as the probable carrier:

1. Natural infection with canine Leishmaniasis was found in 8 out of 857 apparently normal dogs in Peiping.

2. Human and canine Leishman-Donovan bodies are morphologically indistinguishable.

3. In a self contained village, Huaifang, near Peiping, treatment of all cases of human kala azar failed to eradicate the disease, which appeared again within two years. Later, 5 out of 56 dogs examined in the village showed infection with Leishmaniasis.

4. A close geographical association between human and canine infection has been shown in Peiping.

5. 5 Hamsters cured of an infection of human Leishmaniasis, were inoculated with the canine infection, three showing solid immunity and two relative immunity, whereas all the controls readily succumbed to the canine infection.

6. A volunteer cured of human Leishmaniasis proved immune to inoculation with canine infection.

7. A volunteer inoculated with canine Leishmaniasis developed symptoms indistinguishable from those of early human kala azar.

8./

8. Cross complement fixation reactions of sera of infected patients and dogs gave identical reactions with antigens prepared from L.Donovani and L.Canis.

9. Dogs, hamsters, moles and chipmonks infected with L.Donovani and L.Canis show identical pathological and histological phenomena.

10. Phlebotomus Chinensis infected by feeding on dogs suffering from Leishmaniasis, produced the disease when inoculated into hamsters and in one case infection was produced by the natural bite of an infected sandfly. There are only four other instances recorded in which Leishmaniasis has been transmitted by the natural bite of an infected sandfly(14).

It would naturally be interesting to trace a similar epidemiology in Shensi province and attempts were made to find dogs naturally infected with Leishmaniasis. Three unsuccessful examinations were made, but in the fourth dog, a very heavy infection was found, and this was confirmed by Dr T.H. Williams, Professor of Pathology at the West China Union University, Changtu. Sections from all the tissues examined from this dog showed Leishman Donovan bodies, the infection being especially heavy in the reticulo-endothelial cells of the spleen and lymph glands. This dog was found in an area highly infected with human Leishmaniasis, in the east suburb of Sian. While this one case of canine Leishmaniasis does not prove anything, it is the first step towards showing a similar epidemiology to that in Peiping.

Aetiology. There are six species of Leishmania of interest to the medical world, namely L.Donovani, L.Infantum, L.Chagasi, L.Canis, L.Brasiliensis and L.Tropica. The first three are probably the same and L.Canis is probably indistinguishable from the first three. The last two can be separated by certain serological reactions and by the pathological lesions they produce.

The local infection in Shensi is morphologically similar to L.Donovani and as the lesions it produces are these of visceral Leishmaniasis, it is probably L.Donovani. No cases of tropical sore have been seen in this area.

Transmission. In Peiping a Chinese strain of Phlebotomus called P.Chinensis is probably the commonest carrier of kala azar. Facilities of classifying the varieties of Phlebotomus in Shensi have not been available, but there is no doubt as to the existence of the sandfly in this area.

Predisposing Causes. There were 452 males (79%) in the present series and 112 (21%) females. The ordinary ratio of males to females in the hospital out-patient department is 65:35. There is probably no special significance in the lower percentage of females in the kala azar series; the treatment is comparatively expensive, and there is a tendency to neglect female children in this area when a heavy outlay of money is involved. The age groups for the series were divided into five as shown in Table 1. The area in which they were born is also indicated.

TABLE 1.

Showing the age and source of 535 cases of kala azar in Shensi province.

Age in years.	No. of cases	Percentage of the total.	source				
			Sian, east suburb.	Sian city	Shensi province excluding Sian.	Endemic areas other than Shensi.	Non-endemic areas.
0-4	128	24	30	46	43	4	5
5-9	113	21.1	15	33	40	21	4
10-14	84	15.7	6	26	30	20	2
15-19	53	9.8	1	15	24	10	3
20 and over.	157	29.3	3	7	31	57	59
Totals:	535	...	55	127	168	112	73

The over 20 group shows the largest number of cases, 157, but this is more than a five year group, so that the infant group is actually the largest, with 128 cases or 24%, and added to the second group there are 241 cases, a total of 45.1% under 10 years. It will be noted, therefore, that the majority of the cases were children, and only 29.3% were adults. The youngest patient in whom the disease was diagnosed was a child of 10 months, and the oldest a man of 54. Two other cases were over 35 years of age. Kala azar is therefore chiefly a disease of children in this area, and especially of young children. This again is similar to the Mediterranean area, and differs from India, where adults suffer more than children.

The geographical distribution of cases is also interesting, especially with relation to age. The clinic and hospital are situated in the east suburb of Sian, much the largest suburb of the city. 55 cases came from the east suburb, 127 from Sian city, 168 from the remainder of Shensi province, 112 from endemic areas in other provinces and 73 from non-endemic areas in other provinces. The high proportion of cases from other provinces in the adult group will be noted, 116 adults out of 157 or 77.9% of adults from other provinces and 59 adults out of 157 or 37,6% of adults from non-endemic areas. This accounts partially for the increase of kala azar in central Shensi area, due to the large influx of refugees and soldiers from other provinces in war time.

It also suggests that those living in an endemic area may acquire a certain immunity to the disease without actually developing it, whereas those from non-endemic areas easily succumb to infection when exposure takes place. There were patients from 21 of the 27 provinces of China, which shows how great this war time migration has been. Of the total 535 cases, 514 or 95.9% were apparently infected in central Shensi, the area under consideration. A large proportion of them, especially the children, were born in the area and had never been outside its confines. Twenty two cases or 4.1% were infected in endemic areas of neighbouring provinces, twelve in Honan, four in south Shansi, two each in Kansu, Hupeh and Shantung.

All cases in the series were Chinese with a proportion of Chinese Mohammedans. The latter seem to be equally susceptible to the disease. There was no case of congenital infection in the series. Carmichael, Low and Coke reported a case of this occurring in 1926[1]. One woman treated for kala azar, during pregnancy, gave birth to a child who was still healthy at 18 months. Her older child, born about one year before she herself developed kala azar, was diagnosed and treated for the same disease in our Hospital.

There were four instances of more than one case from one family. In three of these two members were diagnosed and treated, and in the fourth family three brothers were diagnosed and successfully treated. This family was especially interesting/

interesting from the transmission point of view, for they lived in an isolated farm about 100 years from a village. Two dogs in the house were examined for Leishmaniasis infection, one by scraping from the ear, and the other by post mortem examination, with negative results in both cases.

There was definite evidence of highly infected foci of kala azar in Sian itself and in the surrounding areas. At least one "kala azar House" as described in India was found. The dog with Canine Leishmaniasis mentioned above was found in one of the most highly infected areas.

Incubation Period. The incubation period is difficult to fix. Manson Bahr[1] quotes one case where it seemed to be only 10 days but also quotes Kirke as stating, from long experience, that it is probably 3-6 months. In one case of our own, No.325, a young man of 23 took ill when travelling from Shanghai (a non-endemic area) to Shensi, and in his case the incubation period appeared to be not more than 11 days. From the experience of this series, I would say that the incubation period is extremely variable. C.U.Lee states 20 days as a possible incubation period[15]. An attempt was made to deduce the time of year at which cases first developed symptoms of the disease, but the results were quite inconclusive, because the onset is often insidious, and many of our cases were too ignorant to give an accurate statement as to the time when their symptoms started. No month nor months showed a majority of cases.

PATHOLOGY.

Local prejudice unfortunately precluded the possibility of performing post mortem examinations on those cases which ended fatally, so that there are no pathological findings to record except in a case of canine leishmaniasis (c.f. Infra).

The first classical description of the pathology of kala azar in India was made by Christophers[16] in 1904. His findings have since been confirmed by other workers. C.H.Hu[17] has contributed greatly to our knowledge of the pathology of the disease in China in a description based on the post mortem examinations of 31 cases of confirmed kala azar in Peiping. A summary of his findings are given below.

Anatomical Changes:

Spleen. The spleen was greatly enlarged in all cases, and often weighed 1000 grams or more, even in children. In one adult male it weighed as much as 4800 grams. The consistency was generally increased, especially in the chronic cases owing to increased fibrosis. The capsule might be thickened or covered with fibrous adhesions. Infarcts, often multiple, occurred in 8 out of the 31 cases. On section, the follicles usually appeared slightly smaller than normal, and the pulp was dark red and increased in amount.

Microscopically in active untreated cases, tremendous numbers of swollen parasitised phagocyte cells were found everywhere in the pulp cords, sinuses, follicles, trabeculae, and in fibrous/

fibrous tissue round the vessels. In treated cases, and those with cancrum oris, the number of parasites was greatly reduced, or they were entirely absent. In some cases, where the parasites were absent in the usual situations, they were found in the thick periarterial fibrous tissue, and in other situations, where the blood supply was not so adequate. The proliferation of the reticulo-endothelial cells was not the only cellular response in the spleen. There was also a plasma cell reaction which was not only marked in active cases, but also in the cases which the parasites and phagocytes had disappeared. That this was not due to intercurrent infection was shown by the examination of control cases with broncho-pneumonia, but with no kala azar infection. The reticulum was thickened in chronic cases, even in children, and there was increased fibrosis, especially round the peri-follicular arteries and the trabeculae.

Liver: The liver was usually enlarged and weighed up to 4800 grams. (In my own clinical experience, the liver enlargement is marked more commonly in children than in adults.) The liver had a pale yellow surface and its capsule was stretched and smooth. The cut surface was distinctly yellow and sometimes even greasy.

Microscopically, the liver showed many parasites in the greatly distended Kupffer cells, in the phagocytes, and lying free in the sinuses. The hepatic cells in the central regions were/

were atrophied, and those at the periphery swollen. In very heavily infected cases, parasites might be found in the hepatic cells. In the chronic cases moderate increase of fibrous tissue occurred in the periportal space, so that a picture of mild biliary cirrhosis was obtained.

Bone Marrow. The bone marrow was hyper-plastic as evidenced, in adults, by the transformation of the yellow fatty marrow of the long bones to a red cellular one.

Microscopically, the marrow of a heavily infected case contained large numbers of parasitised phagocytes of the reticulo-endothelial system. In certain treated cases, where the parasites had disappeared from the spleen and liver, they might still be found in fairly large numbers in the bone marrow. As in the spleen, there was also found in the bone marrow, plasma cell infiltration, most marked in the presence of heavy infection. The reticulum of the bone marrow was greatly increased. There was also a definite increase in cells strongly resembling small lymphoid cells. Of special interest was the variation from the normal of the relative proportion of some of the marrow's cellular elements. This was noticeable in all cases, but especially striking in those in which the parasites were no longer present in the bone marrow concerned. The number of the myelocytes was increased but the metamyelocytes and leucocytes were greatly reduced, or almost entirely absent. In most cases the number of normoblasts was probably decreased.

Extramedullary Myeloid Tissue. As further evidence of myeloid proliferation, one frequently found in kala azar the formation of blood cells, outside the bones, for example in the spleen and lymph nodes. Of special interest was the formation of myeloid tissue between the skull and dura mater. This was first pointed out by Rubini[18]. This was found in 10 cases out of 24 examined. It was seen as flat patches of soft velvety tissue, chiefly on the inner surface of the frontal and parietal bones, more or less symmetrically situated. The bony tissue appeared eroded, due to the formation of new spicules of bone. Microscopically, the inner table was intact, except in a few areas where the myeloid tissue had spread through, probably with the blood vessels, and spread out mushroom-wise on the outer surface of the dura mater. This myeloid tissue had a similar cellular structure to that inside the bones. Even when parasites had disappeared from other organs, as a result of treatment, they might still be found in the marrow in fairly large numbers.

Lymph Nodes. Lymph nodes in cases with heavy infection usually contained many parasites. The nodes were more or less enlarged and, microscopically, parasitised cells were found in large numbers in the medulla, cortex and the sinuses. In treated cases the parasites were not usually found elsewhere than in some of the remote peripheral nodes. Plasma cells were present in large numbers when the infection was heavy.

Tonsils and Adenoids. In tonsils and adenoids the parasites were found in 3 out of 21 cases. In these three cases the parasites had also been found in the spleen and in other situations.

Lungs. In the lungs of heavily infected cases, the parasitised phagocytes were present in large numbers in the alveolar walls and in the connective tissue round the bronchioles and blood vessels. In cases with broncho-pneumonia, no parasitised phagocytes were found in the exudate.

Skin. Parasites were not regularly present in the skin, even though the infection was heavy. The parasitised cells were found chiefly around the small blood vessels, and among the sweat glands. The epithelial cells of these glands were not parasitised.

Testes. Infection of the testes was often heavy. The parasitised phagocytes were present in the interstitial tissue. Among these cells were scattered large numbers of plasma cells and small lymphoid cells. The interstitial cells of the testes were not infected.

Intestines. Parasitised phagocytes were frequently found in the connective tissue of the mucosa and submucosa of the intestinal tract, but no gross alteration in the appearance of the intestinal wall was ever observed. Ulcerative enterocolitis was met with occasionally, but was due to other infection and not to kala azar.

Kidneys. The presence of parasites in the kidneys, usually in moderate numbers, did not cause any important changes in the renal parenchyma.

Other Organs. Parasites might be found in the other organs of the body, usually located in the the connective tissue. In the adrenal glands they were found in the sinusoidal epithelium. The parasites were never found in the brain and meninges.

Cancrum Oris. Cancrum oris was a very common complication of kala azar cases coming to autopsy. Of the 31 cases, 18 had cancrum oris. The organisms of this lesion were fusiform bacilli and spirochaetes, and parasites of kala azar were notable by their absence. The development of cancrum oris seemed to have, in many cases, a definite parasiticidal effect. In cases receiving no treatment, or a very insufficient amount, the number of parasites found was greatly reduced following the onset of cancrum oris. (This was confirmed by clinical experience in my own series.)

Distribution of Parasites in the Body.

In general, in heavily infected cases, the distribution of parasites was widespread. In 31 proven cases kala azar parasites were found in the spleen alone, in the spleen and in other organs 12 times, in bone marrow alone 5 times, in lymph nodes alone 3 times, and by animal inoculation of the spleen once.

C.H. Hu considers that the persistent enlargement of

the spleen, when present after the disappearance, on treatment, of the proliferated reticulo-endothelial cells, is due to increase of plasma cells, reticulum, fibrous tissue and blood. The increase of plasma cells appears to persist after the phagocytic cells, and the parasites they contain are destroyed under treatment. He also considers that, in the absence of parasites, the following findings combined with the clinical features of the disease, will make the diagnosis fairly, though not absolutely, certain.

1. Splenomegaly.
2. Presence of large numbers of plasma cells in spleen and bone marrow.
3. Anaemia of secondary type.
4. Hyperplastic bone marrow, with or without extramedullary myeloid tissue formation outside the dura mater.
5. Cancrum oris.

Manson Bahr's description[1] of the pathology of kala azar in India differs in two important respects from the above. (1) He states that the hepatic cells never contain the parasites, and (2) that ulceration of the intestinal mucosa is very common and that the ulcers may often show the parasites of kala azar. The reason for this disagreement may lie in the difference between Chinese and Indian kala azar.

With reference to the pathological findings in the autopsy on a dog in the present series, the animal's liver and spleen were both enlarged and all the tissues examined showed/

showed the parasites, even the wall of a cyst from the animal's oesophagus which contained a specimen of spirocerca sanguinolenta The infection of the cells of the reticulo-endothelial systems was the heaviest I have ever seen.

Other important pathological findings. Critien[19] first showed that the stools of kala azar patients may contain Leishman-Donovan bodies, and his work was later confirmed by Mackie[20] and by Shortt and his co-workers[21]. Forkner and Zia[22 and 23] showed in two articles that viable Leishman-Donovan bodies were present in the nasal secretions, saliva and material from the tonsils of kala azar patients. They suggested the possibility of direct infection from patient to patient. Teng and Forkner[24] have also showed the presence of infective Leishman-Donovan bodies in the urine of three cases and in the prostatic fluid of one case.

CLINICAL PICTURE.

Symptomatology.

The onset of kala azar was sometimes acute and sometimes insidious. A description of the symptoms complained of is difficult as the Chinese tend to describe their symptoms in terms of their own native medicine, which bears no relationship to western medicine. For instance, the commonest complaint about infants with kala azar is that "their inside is not clear." The earliest symptom in most cases is irregular fever, often with/

with a double remission in 24 hours. This is followed by a loss of colour which resembles the cachexia of subtertian malaria. In acute cases the initial fever wears out the patient rapidly, and may suggest typhoid fever or military tuberculosis. In the insidious type the patient scarcely realises he is fevered, the appetite remaining good. C.U.Lee[15] in a masterly article on the early diagnosis of kala azar, states that the disease may be ushered in by convulsions, but I have never seen these cases myself. Cough is often present (31.4% in this series) and the spleen and liver enlarged. There are definite remissions in the fever during which the spleen may diminish in size. It is therefore not uncommon to elicit a history of fever or "malaria" three months previously, with a recent recurrence of symptoms. In the later stages the cachexia becomes more marked and the splenic enlargement may be extreme. Ulcerated and bleeding gums are extremely common (26.4% in the series) and epistaxis (24.1%) of cases. Anaemia is also progressive, and nutritional oedema not uncommon. Diarrhoea (6.45%), cancrum oris, bronchitis, pneumonia and progressive emaciation are late features of the disease.

Physical Signs.

General cachexia with a muddy complexion is the most noticeable sign on inspecting a kala azar patient. The mucous membranes are pale. Patients are generally thin with a protuberant/

protuberant abdomen, particularly in children. The spleen, and also the liver, are generally palpable. The former is firm with a marked edge like the spleen of leukaemia. The notches are easily felt and sometimes as many as four may be felt. There is often a little oedema of the ankles, and in advanced cases generalised oedema. Petechiae are not uncommon (9.4%). The gums are often ulcerated, especially in children. They bleed easily and often show a greyish line of ulceration round the roots of the teeth. This latter sign is a bad one, and is often the precursor of cancrum oris. The pulse is usually rapid and of low tension. The heart may be a little enlarged, the cardiac sounds are soft and haemic murmurs common. There are sometimes a few rhonchi in the chest at the bases. The urine may contain a faint trace of albumen, but casts are seldom found. Only one patient showed ascites in the whole series.

Blood Changes.

The blood picture is generally typical. The leukocytes are considerably reduced and the granulocytes proportionately reduced. There is a secondary anaemia, which may be intense in children. In my experience the red count is, on the average, even more reduced than the white count. The anaemia is nearly always more marked in children. Red cells may be less than a million and haemoglobin reduced to 10% or less. The average count before treatment for 470 cases in our series were as follows:

follows: W.B.C. 5141, R.B.C. 2,328,000, Haemoglobin 39.8%, colour index 0.85, ratio of white cells to red cells 1:453. The lowest blood counts recorded were: W.B.C. 1200, R.B.C. 795,000, Haemoglobin 10%.

TABLE 2.

	W.B.C. per cmm.	R.B.C. per cmm.	Hb per cent	C.I.	Ratio of W.B.C. to R.B.C.
Average counts in 470 patients before treatment	5141	2,328,000	39.0	0.85	1.453
Lowest counts noted in 470 patients	1200	795,000	10.0	-	-

Manson Bahr[1] states that the red cells are very little reduced in kala azar and the normal ratio of one white cell to approximately 600 red may be increased to 1:2000 or even as high as 1:4000. This is not the case in our series, and the figures show an even greater reduction in red cells than in white cells. Actually, the average white count appears high owing to a few cases with very high counts, due to pneumonia or other complication. Were these excluded it would be in the region of 4700 white cells per cu.mm. The average granulocyte count in this series was 59.9% of polymorphs and 2543 polymorphs per cu.mm. Unfortunately, differential blood counts were only done in 81 cases in this series and these in cases where the

leucocyte/

leucocyte counts were abnormally low. These figures are selected and therefore cannot be taken as representative. There is little doubt however that most cases of kala azar show a slight chronic granulocytopenia, the polymorph count being about 3000 per cu.mm. This statement is borne out by C. H. Huang[25] in an article on acuteagranulocytosis in kala azar.

The question of acute agranulocytosis will be considered under the question of complications, as all the cases in this series occurred during treatment. The blood platelets are reduced, generally to about 100,000 per cu.mm., as shown by C. A. Yang and K. T. Ch'en[26] and this was confirmed in our series.

Diagnosis.

The diagnosis of kala azar can only be confirmed by demonstrating Leishman Donovan bodies. These may be found by many different methods, the commonest being by splenic puncture, sternal puncture, liver puncture, lymph gland puncture and puncture of the upper end of the tibia in very young children. Less commonly they may be demonstrated in skin section, in the peripheral blood, nasal secretions, faeces and even in the aqueous humour of the eye.

In our series of 535 cases Leishman Donovan bodies were demonstrated in 452 cases (84.3%) from sternal puncture.

If/

If the sternal puncture gives negative results we follow it up with a splenic puncture. In those cases in which sternal puncture was negative (83 cases), Leishman Donovan bodies were demonstrated in the following ways: 74 cases (14%) from spleen puncture, 5 cases from both sternal and spleen puncture (0.9%), 3 cases from tibial puncture (0.6%), and in one case (0.2%) lymph gland puncture.

TABLE 3.

Diagnosis of kala azar by demonstration of the parasite (535 cases).

Method.	No. of cases	Percentage of total
Sternal puncture	452	84.3
Spleen puncture	74	
Sternal and splenic puncture.	5	
Tibial Puncture	3	
Lymph gland puncture.	1	

Spleen puncture is the most generally used method and is advocated by Napier (quoted by Manson-Bahr[1]). Sternal puncture is advocated by Lovando, H.L. Chung and D'Oelsnitz[1]. We have found sternal puncture more reliable for the following reasons:

1. In some cases the spleen is not enlarged and therefore is difficult to puncture.
2. It is not always possible to tell if tissue for examination has been obtained from splenic puncture, until the slide has been stained, whereas in sternal puncture one can tell by naked eye inspection, from the oily globules of marrow tissue. This is a great advantage in a busy clinic and obviates a repeat puncture which is a very unpleasant experience for the patient.

The risk of haemorrhage is probably less in sternal puncture. We had no fatalities nor haemorrhages from spleen puncture. There was however one most unfortunate accident from a sternal puncture in case No.342. This was done by a student with much too fine a needle. The sternum was probably punctured through/

through and through, and one of the intrathoracic vessels wounded. The child died two hours later with symptoms of internal haemorrhage. No post mortem could be obtained. If the sternal puncture gives negative results we follow it up by a splenic puncture. Liver puncture is safe, but yields a smaller percentage of positive findings, and is difficult when the liver is not enlarged. Tibial puncture is only useful in very young children where the sternum is largely cartilaginous. It is recommended by Giraud (quoted by Manson Bahr[1]). Lymph gland puncture was first advocated by Cochrane[27] in 1912 and has had a recent renewed popularity. We have found it safe, but unreliable in its results.

We employ the following technique for sternal puncture. The skin over the sternum and surrounding area is cleaned and sterilised with alcohol 70% and tinct. iod. mitis. A very large bore needle with bevelled point and stilette is used. When the stilette is withdrawn a record syringe can be fitted into the lumen. The needle and stilette are sterilised by boiling and must be perfectly dry. The syringe is dried out with ether. The least trace of water will cause the Leishman Donovan bodies to swell and make them unrecognisable. The skin and tissues down to the bone are anaesthetised with a little 2% novocaine, and the skin is punctured with a fine bladed knife to admit the puncture needle. The coarse needle is inserted down to the sternum just below the angle of Lewis

and/

and is gently screwed into the sternum at an angle of 30° to the perpendicular. As soon as it passes through the cortex less resistance is felt and the stilette is pressed home to clear the point of the needle, and then removed. The record syringe is attached and some marrow aspirated. This is spread on a slide, and if the white seed-like droplets are seen, the needle can be withdrawn and the puncture sealed with collodion gauze. If no tissue appears, a little further manipulation and aspiration almost always yields results. In very young children it is probably better to puncture the manubrium sterni where in the child there is a little more marrow. There is no need to rest the patient after the operation as one requires to do with splenic puncture. This is another advantage of sternal puncture. A very wide bore needle is employed as it is safer and less likely to be thrust into the thoracic cavity, and is also easier to aspirate the tissue through. No untoward results have been seen from sternal puncture, with the exception of the one tragedy recorded above. There is sometimes a little after pain which can be controlled by simple analgesics. The slides may be stained with Leishman's, Wright's, Romanowsky's or Giemsa's stains. As methyl alcohol has been unavailable in Sian in war time we have stained the slides, after fixing with alcohol, with a watery solution of methylene blue in sod. biborate/

biborate containing 1:10,000 eosin. This has proved an economical and satisfactory method, though the detail is not so fine as when the usual stains are used. Leishman Donovan bodies are naturally more difficult to find in the early cases, and require prolonged search. In the more chronic cases the diagnosis is sometimes very easy, the reticulo-endothelial cells being packed with the parasites. On the other hand, when cancrum oris has set in, it is a well known fact that the Leishman Donovan bodies may be extremely hard to find, and this was also our experience.

As regards the other tests for kala azar, such as the water test and the aldehyde test (serum formalin reaction, Napier[1]), we have not employed these extensively for the following reasons:

1. If they are positive, it still does not clinch the diagnosis of kala azar.
2. If Leishman Donovan bodies are not found in early cases, these tests are also often negative.
3. Any gross anaemia will give positive findings with these tests.

We have not personally tried testing the precipitations of sera of kala azar patients with 4% solution of the pentavalent antimony compounds as advocated by Napier and confirmed by Chopra.[1] These may be more reliable than the water and aldehyde tests and are said to give an indication of the suitability of the compound for/

for treatment. Laha[28] states that the antimony tests give earlier results than aldehyde tests but are less reliable. It is only effective after four months of disease.

Early Diagnosis. The early diagnosis of kala azar may be extremely difficult especially during the first two weeks of illness. The condition in the early stages may resemble the early stages of typhoid, tuberculosis, malaria, undulant fever, etc. The splenic and liver enlargement may not take place immediately. Lee and Chung[15] state that the platelet reduction does not necessarily take place early, but that the serum globulin of the blood is increased, and serum albumen decreased and that this change does take place early. The facilities were not available for this estimation in Sian.

In our own series careful and, if necessary, repeated examinations of sternal marrow and spleen tissue revealed Leishman Donovan bodies in most cases. In the latter part of the series there were only three cases in which Leishman Donovan bodies could not be found. All were treated with the pentavalent antimony compounds. Two responded to the treatment and the third did not. He was apparently a case of splenic anaemia.

Differential Diagnosis. Differential diagnosis of kala azar in the area under consideration has to be made from the following conditions. Malaria, relapsing fever, typhus fever, typhoid fever, tuberculosis of lungs, abdomen and miliary tuberculosis, Banti's/

Banti's disease, amoebic abscess of liver, and ankylostomyasis. In each case the final point of differentiation is the finding of Leishman Donovan bodies.

Malaria may simulate kala azar very closely, especially chronic subtertian infection. Liver enlargement is more common in kala azar and is not a feature of subtertian malaria. The spleen is often larger and firmer in kala azar. The leucocyte count is lower in kala azar and there is no response to quinine. The patient appears more acutely ill in malarial infection. Sternal or splenic puncture may reveal parasites of either disease.

Relapsing Fever. The patient is more seriously ill. The spleen if enlarged is soft and the patient has more a yellow tint of haemolytic jaundice than the muddy tint of kala azar. By the third day the spirochaetes are easily found, and the leucocytes not markedly reduced.

Typhus Fever. The temperature is sustained and shows no remission. The appearance of the rash on the 5th or 6th day and the onset of typhoid state gives the diagnosis, confirmed by Weil Felix reaction.

Typhoid Fever. This is difficult to differentiate from kala azar in the early stages. Diurnal rise and remission of temperature suggest kala azar. Culture of sternal puncture will reveal b.typhosus as early as the 3rd or 4th day and later the Widal reaction becomes positive.

Tuberculosis of Lungs, Abdomen and Miliary Tuberculosis.

Tuberculosis of the lungs may present a problem in differential diagnosis especially if there are no radiological facilities. The possibility of kala azar coexisting with pulmonary tuberculosis must be remembered. In children tuberculous peritonitis may simulate kala azar. Both are common infections in children from 2 till 12 years in this area.

The diffuse abdominal distension with dough-like consistency can be differentiated from the splenic enlargement of kala azar. Miliary tuberculosis in the early stages resembles kala azar, but the patient's condition deteriorates more rapidly and the leucocyte count is higher. Banti's disease or cirrhosis of the liver with splenic enlargement closely simulates kala azar at the afebrile stage. I have seen personally two cases of this condition which were treated for kala azar. The spleen is firm, the patient has the same muddy complexion, and the leucocyte count is low. Ascites is common however in late stages of Banti's disease but rare in our experience in kala azar.

Amoebic Abscess of liver resembles kala azar superficially but the high leucocyte count helps to differentiate the two conditions. Ankylostomasis bears a superficial resemblance to kala azar, but fever is not marked, the blood shows an increase in eosinophil leucocytes and the ova of the worm may be found in the stools. The infection may be co-existent with kala azar. Bilharzia infection and trypanosomiasis are not found in this area.

Course of the Disease. Kala azar, after the first febrile period, in chronic cases proceeds in a series of remissions and exacerbations. The anaemia increases and the patient eventually succumbs to the original infection, or more commonly to some complication such as cancrum oris, dysentery or pneumonia. The question as to whether a natural cure may take place is an interesting one, but it is almost impossible to produce conclusive evidence on the subject, unless a patient is diagnosed and fails to come for treatment, then presents himself later for examination and is found cured. There were many cases in this series which, having been diagnosed, received no treatment for a considerable period, and then returned. In every one the infection was still present and the patient's condition had deteriorated. On the other hand we frequently hear of cases which apparently had kala azar in childhood and had recovered. It is possible that most of them are really malaria or some other condition. It is a well known fact that in cancrum oris and sometimes in other intercurrent infections, the parasites almost disappear from the tissues, but whether it is possible that these conditions may induce a spontaneous cure is impossible to say. C. U. Lee (personal communication) believes that spontaneous cure is within the bounds of possibility. Manson Bahr (1 p.197) has said that 10 per cent of patients may possibly recover without treatment.

Duration/

Duration of Illness. The average duration of illness before treatment was 6½ months and the average duration from onset till death in cases who died, 8 months.

COMPLICATIONS.

There are five important complications of kala azar, namely: (1) Cancrum oris or noma; (2) Dysentery; (3) Necrosis of jaw; (4) Pneumonia and (5) Acute granulocytopoenia. The frequency of these is shown in table 4.

TABLE 4.

Complication	No.of cases	Percentage of total cases.
Cancrum oris or noma	78	14.6
Dysentery	43	8.0
Necrosis of the jaw.	16	3.0
Pneumonia	22	4.1
Acute granulo-cytopoenia	3	0.6

The importance of these five complications lies in the fact that they were responsible for the fatal issue in 41 of the 57 patients in the series who died. Each of these complications will now be considered in some detail.

Cancrum Oris or Noma.

Chu and Fan[29] in a comprehensive survey of 100 cases of cancrum oris, most of which occurred in association with kala azar, define the condition as follows: "Cancrum oris is a term used to designate a spontaneous process of gangrenous necrosis, which starts in the mucous membrane of the oral cavity and may rapidly spread to the neighbouring structures. When the term noma is used, it includes lesions of a similar nature affecting the vulva, prepuce and anal regions." All the cases listed below conform to this definition, save one, in which a similar process affected the skin over the medial side of the right leg and the dorsum of the left foot.

The pathology appears to be that of infection and thrombosis in an area where the tissue resistance is lowered by the severe anaemia so often found in kala azar. Chu and Fan[29] who were able to observe some of their cases in hospital before the onset of cancrum oris, state that the appearance of the lesion is preceded by a rise in temperature and an acute fall in the leucocytes and granulocytes in the blood. They naturally suggest that the process may be analogous to the angina of acute granulocytopoenia. Manson Bahr[1] also mentions this possibility. In the present series there were seven cases where cancrum oris was found affecting the tonsillar region or the pillars of the fauces, but in only two of these cases was there a blood picture of acute granulocytopoenia. The local lesions/

lesions of angina and cancrum oris are impossible to differentiate by clinical examination alone and it seems very possible that they are similar lesions.

Out of 78 cases of cancrum oris in the series, 49 were in the complete series and 29 in the incomplete. Of the 49 complete case records, 38 were males (77.6%) and 11 were females. These are approximately the proportions of males to females in the whole series so that cancrum oris appears to affect the sexes equally. The age groups and deaths in each group were as follows:

Birth - 4 yrs.	5-9 yrs.	10-14 yrs.	15-19 yrs.	20 and over.
18 cases	12 cases	5 cases	2 cases	12 cases
(36.6%)	(24.5%)	(10.2%)	(4.2%)	(24.5%)
10 died.	6 died.	2 died.	1 died.	6 died.

Cancrum oris was then more frequent and more fatal in the birth to 4 years group than in any other, but the mortality was nearly 50% in all age groups. In 31 cases (63.3%), the cancrum oris occurred before treatment, and in 18 cases (36.7%) during treatment, so that treatment with the antimony compounds could not be considered an important factor in the aetiology of kala azar.

Situation of Cancrum Oris Lesion. A summary is appended of the situation in which the local lesion of cancrum oris appeared in 78 cases complete and incomplete.

Cheek 36 cases. Upper lip 11 cases. Tonsils and Fauces 7 cases. Lower Lip 8 cases. Gums 4 cases. Floor/

Floor of mouth 2 cases. Upper and lower lip 2 cases. Soft palate 2 cases. Nose 1 case. Whole face 1 case. Cheek and lower lip 1 case. Lower lip and tongue 1 case. Right leg and dorsum of left foot 1 case.

The cheek was the commonest situation, the lesion starting on the mucous surface and spreading rapidly to the skin surface. When the tissues of the parotid gland or the floor of the mouth were involved, the prognosis was always bad.

Chu and Fan[29] classify their cases of cancrum oris as acute, subacute and chronic and those in our series also fell readily into these three groups. The average blood counts were - W.B.C. 5,042, R.B.C. 2,058,000, Hb. 34% before treatment. These figures are all lower than the average blood counts for the whole series.

There were 39 cases (79.4%) of acute cancrum oris, 5 (10.2%) subacute and 10 (20.4%) chronic cases in the series.

22 of the acute cases died (56.5% mortality), one of the subacute cases (20% mortality) and 2 of the chronic cases (20% mortality). Acute cancrum oris is therefore a much more fatal condition than the other two. In the subacute and chronic cases the lesion had been present for a considerable time (5 weeks to several months) and was in a quiescent form. In those patients who died, the lesion suddenly became acute again during treatment. The end results of the 49 completed cancrum oris cases were as follows:

Cured	Doubtfully cured.	Much improved.	Improved.	Improved slowly.	Moribund.	Died.
2	6	6	4	6	12	13

The total mortality in the series of 49 cases was 25 deaths (51%), and this figure is probably lower than actual fact, as many of the 29 cases who were lost track of probably died. It can be seen then that cancrum oris is a very important and a very fatal complication of kala azar, for the 25 cases who died represent 44% of all the deaths in the whole series of 400 cases. Of the 25 cases who died, 15 were males and 10 females, so that the death rate among females was higher proportionately. This is as one would expect as nutrition is definitely not so good among females and they also tend to be neglected.

Of the cases of cancrum oris, seven suffered from other serious complications. Two cases had dysentery, both of which died. Two cases had pneumonia - one with lobar pneumonia which recovered, the other with bronchopneumonia which died. Two cases suffered from acute granulocytopoenia, both of which died. One case had necrosis of jaw followed by cancrum oris. This case died.

As in the large majority of cases, the kala azar was treated with neostam (40 cases out of 49). The results of treatment with other compounds were too few for comparison. On the whole it appeared that May and Baker '744' gave the best results in cases of cancrum oris.

Treatment.

The prevention and treatment of cancrum oris is a matter of great importance. Cancrum oris is often preceded by a grey line of ulceration along the line of the gums where the teeth enter the gums. Proctor, quoted by Manson-Bahr[1], states that the energetic treatment of this condition with carbolic and spirit helps to prevent the onset of cancrum oris. Carbolic, with its well known tendency to produce gangrene of the extremities, seems to be a dangerous drug to use in the prevention of cancrum oris. Frequent cleansing of the mouth especially after food, and washing out with mild oxidising agents such as hydrogen peroxide or solution of potassium permanganate, seem more reasonable procedures for the local lesion.

General treatment however is even more important. It cannot be too strongly emphasised that early treatment of kala azar, before the inevitable anaemia is advanced, is a most important factor in prevention of cancrum oris.

Antimony therapy was persisted with in all our cases and though, as pointed out above, three chronic or subacute cases showed an exacerbation, a much larger number of cases improved with the antimonial injections.

Adjuvant methods of treatment were also employed. Small injections of novarsenobillon were used in 20 patients to combat the inevitable spirochaetal infection in this lesion.

Of/

Of these, 13 died (65%). Blood transfusion alone was employed in two cases, one of which died. Blood transfusion and novarsenobillon were used in two cases: both died. Intramuscular injections of whole blood (taken from parents) were given to two children, both of whom recovered. Intramuscular injections of whole blood and novarsenobillon were given to two other children; both died. It is obvious that no deduction as to the value of these methods of treatment can be made from so few cases, especially as the best treatments were given to the most serious cases. For example, in two of the cases quoted, the patient's condition was practically hopeless, the one from bronchopneumonia, the other from acute granulocytopoenia.

Summary of cancrum oris in kala azar.

Cancrum oris is probably the most important complication of kala azar, accounting for 44% of all deaths in the series. The importance of early treatment of the kala azar to prevent cancrum oris is stressed. Adequate nutritious food is also most important. It seems advisable to continue with antimony treatment in all cases. Injections of novarsenobillon, blood transfusion and intramuscular injections of whole blood deserve a trial. It is difficult to assess their value in the present series. In view of the fall in leucocytes and polymorphs which accompanies the onset of cancrum oris, injections of pentnucleotide, and possibly of adrenalin, as suggested later for acute granulocytopoenia, might be tried.

Dysentery.

Dysentery was noted as a complication in 43 cases out of 535, or 8%. These figures are probably lower than they should be, for the population of the area probably suffers from dysentery to the extent of about 5%. The majority of cases suffered from amoebic dysentery which is the most prevalent form in the area. A considerable number of dysentery cases were probably missed, as dysentery is almost considered a normal state rather than a disease in the summer months, and patients will often deny the condition unless it is severe. Parents too are often ignorant that their children are suffering from dysentery.

In 9 cases dysentery was considered to be contributory cause of death. The age groups of these 9 cases were:-

Birth - 4 yrs.	5-9 years.	10-14 years.	15-19 years.	20 yrs. and over.
3 deaths	1 death	1 death	1 death	3 deaths.

In two of these 9 cases, death was due to dysentery after apparent cure of the kala azar. In two other cases death was due to dysentery and relapsing fever; one of these two cases also had necrosis of the jaw. In four cases dysentery was associated with pneumonia as a further complication. Three of these died including one who also had necrosis of the jaw. Two cases also had cancrum oris and both died. It will be seen then that dysentery, in the fatal cases, was often a terminal condition/

condition associated with other major complications. In two cases however it was apparently sufficient per se to account for the fatality. These two cases were apparently cured of kala azar, but died at home later of dysentery, without having presented themselves at hospital for treatment of that disease.

Treatment. Treatment of dysentery depended on the type. Castor oil, emetine injections and dieting were employed in the amoebic cases, and sodium sulphate in the bacillary type. Treatment as outpatients was very unsatisfactory, as diets could not be controlled. The question of maintaining nutrition in kala azar complicated by dysentery is an extremely difficult one.

Summary. Dysentery is an important and sometimes fatal complication of kala azar. The incidence is probably higher than the figure (8%) given above. Most of the cases were amoebic in type. As the majority of the patients were not admitted to hospital, treatment was unsatisfactory, especially with regard to maintaining adequate nutrition.

Necrosis of Upper and Lower Jaw.

This complication occurred 16 times in the series of 535 cases (3%). Of these 16 cases 13 were in the complete and 3 in the incomplete series.

The process is similar in many respects to cancrum oris, and the former condition may easily merge into the latter and vice versa. Necrosis of the jaws is, however, a more chronic process, as one would expect in a bony lesion, and the mortality is lower than in cancrum oris. The average blood counts are all higher than in cancrum oris (c.f. below). The condition has been considered separately for these reasons. The pathological process is probably similar to that of cancrum oris, though it is questionable whether there is the same acute fall in leucocytes and polymorphs preceding the onset of necrosis of the jaws.

There were 10 males and 3 females in the series, so that the condition appeared to be relatively more frequent among males. The age groups for the 13 completed cases, with deaths in each group, were as follows:-

Birth - 4 yrs.	5-9 years.	10-14 years.	15-19 years.	20 years & over.
3 cases	6 cases	1 case	Nil	3 cases
1 death	1 death	1 death		No deaths

Necrosis of jaw was then more common in the second age group in this series. 10 cases developed before treatment, and only 3 after treatment. In 7 cases the maxilla was affected and/

and in 6 cases the mandible, the condition being invariably unilateral.

The average blood counts before treatment were: W.B.C. 6,616; R.B.C. 2,267,000. Hb. 46.7%. Only two differential blood counts were made in this complication. There were 59% polymorphs in the one and 75% in the other. These counts are higher than the average counts in cancrum oris, and W.B.C. and haemoglobin are higher than the average for the whole series. The end results were as follows:-

Cured.	Doubtfully cured.	Much improved.	Improved.	Improved slowly.	Moribund.	Died.
1	2	5	-	1	1	3

The mortality of necrosis of the jaw is therefore 30.8%.

Treatment. Early treatment of the kala azar and careful attention to oral sepsis are probably the two most important factors in preventing this complication. When the condition had developed, it was treated conservatively until the sequestra had separated. In 4 cases sequestra had eventually to be removed by operation, but in one case (No. 293) removal of sequestrum of the maxilla in a child of 5 years, apparently precipitated the onset of cancrum oris in the overlying tissues of the cheek, followed by bronchopneumonia. This child was obviously moribund when last seen. In another case in the incomplete series, a similar condition was observed in another child, but this patient was lost trace of and the end result/

result was in doubt. In the light of this experience, it seems wise to remove sequestra in children only when their general condition is very much improved.

In a second fatal case necrosis of jaw was further complicated by dysentery and relapsing fever, and in a third by dysentery and bronchopneumonia, so that in only one case were kala azar and necrosis of the jaw alone responsible for a fatal issue. This goes further to show that necrosis of jaws is not nearly so fatal a condition as cancrum oris.

Summary. Necrosis of jaw is an important complication of kala azar. Per se, it is seldom responsible for a fatal issue. It occurs in the series with equal frequency in maxilla and mandible. Sequestra should be treated conservatively in children. It appears to be commoner in the second age group (5-9 years). Anaemia and leucopoenia are less marked than in cancrum oris.

Pneumonia.

Pneumonia was recorded in 22 cases out of 535 (4.11%). There were 19 cases in the complete, and 3 in the incomplete series. Of the 19 complete cases, 15 were males and 4 females, so that the incidence was almost the same as the ratio of males to females in the series. There was no marked seasonal incidence of pneumonia cases occurring throughout the year.

Three types of pneumonia were diagnosed, namely, lobar, broncho/

broncho and hypostatic. A table is appended showing the age groups, type of pneumonia found in each group and the mortality of each type.

TABLE 5.
Incidence of pneumonia and deaths in 535 cases of kala azar.

Age in years.	No. of cases.	Type of pneumonia			Dead or Moribund
		Broncho	Lobar	Hypostatic	
0-4	6	6	0	0	5
5-9	3	2	1	0	1
10-14	4	1 (?T.B.)	3	0	2
15-20	0	0	0	0	0
20 and over.	6	0	4	2	4
Deaths	12	7	3	2	12

It will be seen that the incidence was highest in the infant and adult groups, and the mortality highest in the former. Both cases of hypostatic pneumonia died and 7 of the 9 cases of bronchopneumonia died, including one which appeared to be tuberculous, giving a very high mortality rate of 77.7% for this complication.

Lobar pneumonia appeared to be the least fatal type, with a mortality of 3 out of 8 cases, or 37.5%. Of these three lobar pneumonia cases who died, one recovered from the pneumonia, but died later from a relapse of kala azar with dysentery. Another case recovered from kala azar but died one month later from/

from a straightforward pneumonia. It will be seen then that the prognosis of lobar pneumonia complicating kala azar is not necessarily bad.

The end results of the whole 19 cases of pneumonia were as follows:-

Cured.	Doubtfully cured.	Much improved.	Improved.	Improved slowly.	Moribund.	Died.
2	2	1	2	-	1	11

Pneumonia was, then, as a whole, a less frequent, but proportionately a more fatal complication than cancrum oris. Acute granulocytopoenia (v.infra) was the most serious complication in the series with 100% mortality, and pneumonia comes next with 63.15% mortality.

Two cases of pneumonia were further complicated by cancrum oris, one - a case of lobar pneumonia - recovered, the other - a bronchopneumonia - died. Four cases of pneumonia were further complicated by dysentery, one of these four also suffering from necrosis of jaw. Three of these four cases died. One case of hypostatic pneumonia was further complicated by acute stranulocytosis and this case also died.

Treatment of Pneumonia. Treatment with antimony compounds was always stopped at the onset of pneumonia as it is well known, from experience gained with antimony tartrate injections, that antimony salts are irritating to the structures of the respiratory tract, and though the pentavalent antimony compounds are/

are not so irritating as the trivalent, they still have this action in a lesser degree. Antimony treatment can be safely resumed a week after the temperature has fallen. Treatment of the pneumonia cases was on general lines with sulphapyridine, the specific drug of choice when available. When not available, sulphanilamide was used, but appeared to have little therapeutic effect.

There was generally a good leucocytic response to the pneumonia, except in the hypostatic cases. This is interesting as it shows that in kala azar, where the white counts are generally so low, the body can still make a good response to acute infection.

Summary of Pneumonia. Bronchopneumonia is a complication very much to be feared in kala azar, especially in infants. Lobar pneumonia is a much more hopeful condition but may also be fatal, especially in adults.

The extraordinary recuperative powers of the adolescent are shown in case No. 246, an undernourished Mohammedan lad of 11 years who survived kala azar, first complicated by cancrum oris of the lower lip, and then by lobar pneumonia. He is now healthy and has put on much weight. Hypostatic pneumonia is a terminal condition, and there is naturally very little hope of recovery from this condition.

Granulocytopoenia.

Chronic granulocytopoenia is probably a fairly constant feature of kala azar. It was found in 33 cases (6.2%) of our series but differential blood counts were done in less than 20% of cases. Chronic granulocytopoenia did not seem materially to effect the prognosis in the few cases in which it was found.

Acute granulocytopoenia was found in three cases in the series - Nos. 119, 201 and 252 - all of whom died. This gives an incidence of .56% for the series.

Zia and Forkner(30 and 31) and Huang(25) in China, Shortt and Swaminath in India, and Grithi in Italy (quoted by Manson Bahr) have reported agranulocytosis in kala azar. Zia and Forkner reported 8 cases complicating kala azar and Huang(25) reported 42 cases out of 557 cases of kala azar, diagnosed in the past 10 years, an incidence of 7.6%. This incidence is much higher than in our series, possibly because their cases were all inpatients. It is possible that some of the less serious cases in our series were missed as frequent routine differential blood counts were not done. It is also possible that in some cases the granulocyte count falls before the leucocyte count. There were certainly only 3 cases with the typical angina, fever and rapidly falling leucocyte and granulocyte counts. Huang(25) considers that a count of 20% or less is necessary for a diagnosis of acute agranulocytosis in adults and 10% or less in children.

In/

In none of our cases did the granulocyte count fall below 20% so we have used the term granulocytopoenia rather than agranulocytosis, which would represent a complete absence of polymorphs.

A summary of the three cases and the blood counts is given below (Tables 6-8). Case No. 119 Tsao Chin-san, age 20, soldier by profession, weight 114 lbs., attended clinic for first time on 15/11/40 with a history of fever and enlarged spleen of 6½ months duration. The spleen was enlarged 5½ inches below the costal margin. The liver was not palpable. A few Leishman Donovan bodies were found in sternal puncture. Initial blood count was: W.B.C. 3,000; R.B.C. 2,300,000; Hb. 45%; Colour index .98. Alternate day intravenous injections of neostam (Burroughs Welcome and Co.) were started on 19/11/40 and the patient improved up till 30/11/40 by which time he had had 1.80 grams of neostam. On 30/12/40 he was not so well and the leucocyte count fell to 2,400. A further injection of .30 neostam was given. The course of the disease is hereafter shown in table 6.

Summary. This patient showed the typical triad of symptoms associated with granulocytopoenia (acute), namely fever, angina and rapidly falling granulocyte count. The lowest granulocyte count, 682 per cu.mm., was recorded on 12/12/40 when the leucocytes were 3200. The patient's condition improved with injections of sod. nucleinate intramuscularly and one blood transfusion. The red count improved and white count returned to normal figures.

However,/

However the ulceration of the fauces continued to deepen, and he developed pleurisy with effusion on the right side. Three days before death he had a recurrence of fever and sore throat, and the following day the leucocytes had fallen slightly but the percentage of polymorphs was still high.

Case No. 201. Huang Hsiao-chen, aged 29, Government tax collector, weight 97½ lbs. Patient first attended clinic with a history of fever, cough and constipation of two weeks' duration. Spleen was 5" below costal margin and liver 1" which suggested the duration was considerably longer than two weeks. Kahn test was negative. Leishman Donovan bodies were found from splenic puncture after a negative sternal puncture. W.B.C. 3,200: R.B.C. 2,500,000; Hb. 47%. C.I. .9 plus.

Treatment by daily injections of neostam were started on 29/3/41 as the patient was very anxious to have treatment completed early and to return to work. By 5/4/41, patient had received 1.20 grams of neostam intravenously in 6 daily injections, but was feeling, and looking very ill. Progress thereafter is shown in table 7.

Summary. This case also showed the triad of fever, angina and rapidly falling granulocyte count. The granulocytopoenia was less acute than in case No. 119, and small doses of neostam were continued. Granulocytes eventually fell to 350 per cu.mm. It is possible that the heavy dosage with daily treatment at the beginning of the treatment was partially responsible for the/

the trouble. The granulocyte count improved with injections of sod. nucleinate but, as in case No. 119, the necrotic process continued and extended. The patient died of toxaemia from this, in spite of two blood transfusions, about 25 days after onset.

Case No. 252. Yao Ti-chen, aged 24, military officer; weight 119¼ lbs. Attended first on 7/5/41 complaining of fever and malaise and epistaxis of four months' duration. The spleen was enlarged 4 inches below costal margin. From sternal puncture Leishman Donovan bodies were found, and initial blood counts readings were W.B.C. 3,200 ; R.B.C. 2,100,000; Hb. 40%; C.I. 0.9. Treatment was started on 8/5/41 and alternate day injections of neostam were given. By 13/5/41 0.50 gm. of neostam had been given in 3 injections. The further course of disease is shown in table 8.

Summary. This case only showed two of the cardinal signs of acute granulocytopoenia, high fever and rapidly falling leucocyte and granulocyte counts. There was no angina throughout. The patient's granulocytes returned to normal, but he died of a low form of pneumonia which, for lack of a better term, was classed as hypostatic pneumonia. The granulocytopoenia was not so acute and though the leucocytes fell to 1,000, the granulocytes were never less than 40%. Treatment was with inj. sod. nucleinate, inj. camphor co. and one blood transfusion.

TABLE 6.

Acute granulocytopoenia - Case 119.

	W.B.C.	R.B.C.	Hb.	Poly.	S.Lymph	Lymph	Mono.	Treatment
5/12/40	3,200	-		57%	46%	1%	2%	Neostam 0.3
7/12/40	3,200	2,890,000	53%	-	-	-		Neostam 0.3
10/12/40	Patient complained of fever and sore throat. T.102.4; Pulse 128. White necrotic patches on both tonsils with red reaction round.							
	6,200			33%	58%	2%	7%	
	Throat swabs for C.diphtheriae negative. Inj, Camphor, Ol Terebinth and Creosota intramuscularly.							No Neostam.
12/12/40	3,100			22%	68%	2%	8%	
	Admitted to hospital as inpatient.							
13/12/40	3,000	2,740,000	51%	34%	57%	3%	3%	Platelets 132,980 cu.mm.
14/12/40	Spleen enlarged only ½ inch below costal margin.							
	2,800		40%		52%	3%	5%	
	Sloughs of throat separated leaving deep ulcerated cavity.							Transfusion 350 c.c.
15/12/40	2,400		-	43%	53%	1%	3%	Platelets 195,120 per cu.mm.
16/12/40	4,200	3,040,000		72%	26%	-	2%	
17/12/40	10,200	-	-	95%	4%	-	1%	
19/12/40	10,600	-	56%	-	-	-	-	
21/12/40	10,400	-	-	61%	30%	1%	1%	
	Pleurisy with effusion R.Base.							
23/12/40	Temperature rising again. Recurrence of sore throat.							
24/12/40	6,800	3,500,000	56%	77%	18%	1%	4%	
25/12/40	Ulcers of fauces and soft palate deepening. Patient is sinking.							
26/12/40	Patient died at 6 a.m. this morning.							

TABLE 7.

Acute granulocytopoenia - Case 201.

Date.	W.B.C.	R.B.C.	Hb.	Poly.	S.lymph	Lymph	Mono.	Treatment
3/4/41	2,600	-	-	56%	40%	-	4%	Neostam .30 equals 1.50 gm. total dose.
8/4/41	Patient feeling better.							Neostam .30 equals 1.80 gm. total dose.
12/4/41	Feeling worse again. Ulceration and swelling of gums.							Neostam .30 equals 2.10 gm. total dose.
15/4/41	2,400			49%	48%		3%	
16/4/41	Ulceration of right tonsillar region. Patient admitted to hospital.							
	2,400	2,800,000	49%	57%	40%	1%	2%	Inj.sod. Nucleinate
17/4/41								Inj.Neostam 2 cc. I.M. 0.10 = 2.20
18/4/41	1,600			40%	58%		2%	Inj. Sod. Nucleinate 2 cc. I.M.
19/4/41	3,200			57%	40%		3%	Inj. Sod. Nucleinate 2 cc. I.M.
20/4/41	Temperature 103.4. Pulse 110.							Inj. Sod.
	1,400			25%	69%		6%	Nucleinate 2 cc. I.M.
21/4/41	2,000			40%	56%		4%	Inj. Sod. Nucleinate 2 cc. I.M.
22/4/41	1,800			52%	46%		2%	
23/4/41	2,600	2,030,000	42%	67%	31%		2%	
24/4/41	2,200			59%	44%		1%	
	Large sloughs which have separated from fauces removed.							Blood transfusion 370 cc.
25/4/41	2,000			57%	41%		1%	
28/4/41	1,400	2,290,000	35%	50%	48%			Neostam 0.10 = 2.30 gm. total dose.
	Spleen 5" below costal margin. Ulceration extending deeply into fauces. Inflammatory swelling of right temporal region.							
29/4/41	Blood transfusion 200 cc. citrated blood.							
30/4/41	Swelling of face increasing.							
	2,000	2,280,000	44%	62%	35%		1%	Basophile.
1/5/41	Temperature subnormal; pulse rising. Patient is very ill. Spleen still 5" below costal margin. Massive necrosis in R.Pterygoid/							

Table 7. Acute granulocytopoenia.- Case 201. (Contd.)

	R. Pterygoid region. Sanguineous discharge from mouth.
6/5/41	Patient desperately ill and toxic. Petechiae all over. Temp. subnormal. Patient died at 11 a.m.

TABLE 8.

Acute granulocytopoenia - Case 252.

Date	W.B.C.	R.B.C.	Hb.	Poly.	S.Lymph	Lymph	Mono.	Treatment.
13/5/41	3,200		-	40%	57%		2%	
15/5/41								
17/5/41	Improving a little.							Inj.Neostam 0.20 = .70 gm.
20/5/41								Inj. Neostam 0.20 = .90 gm.
22/5/41	3,600							Inj.Neostam 0.30 = 1.2 gm.
24/5/41	Wt. 118¼ lbs. Spleen reduced to 2¾".							Inj.Neostam 0.20 = 1.4 gm.
29/5/41	2,800	Fever worse.		54%	44%	1%		Inj.Neostam 0.20 = 1.6 gm.
31/5/41	2,200	T.104; pulse 120. Admitted to hospital		37%	62%	1%		Pulv. Quin. Sulph. grain Inj.Sod.Nucleinate 2 cc. I.M.
1/6/41	Still high fever; patient very ill.							
	2,000	1,620,000	24%	47%	50%		5%	Nucl.2 cc. I.M.
2/6/41	1,800			46%	52%		2%	
3/6/41	1,800			50%	48%		2%	
	Friction r.base. Blood film for parasites.							None found.
	citrate blood 300 cc.							Blood transfusion 400 cc.
4/6/41	1,400	1,800,000	38%	44%	52%		4%	
5/6/41	1,000			40%	58%		2%	Neost.0.10 = 1.7 gm.
	Fine rales at l.base. Inj.Camphor co. min. 3 4 hourly.							
7/6/41	1,400		50%	48%			2%	
8/6/41	1,500		51%	48%			1%	
9/6/41	2,000	Patchy consolidation lt.base.						
				62%	34%	1%	3%	
10/6/41	Spleen 2" below c.margin.							
				70%	26%	2%	2%	
11/6/41	2,000	T.fallen to normal.						
				75%	24%		1%	
12/6/41	3,400			72%	25%		3%	
	Consolidation all over lt.base; high pitched rales rt.base.							
13/6/41	3,200			67%	25%	3%	2%	P.130.
	Recurrence of fever.							
14/6/41	3,000			64%	31%	2%	3%	
15/6/41	3,400			69%	27%		9%	
17/6/41	4,300			73%	21%		5%	Eos. 1%
	Consolidation both bases, spleen 1" below c.margin.							
19/6/41	3,800			83%	15%	1%	1%	
20/6/41	8.400							
21/6/41	6,400			77%	20%		3%	
22/6/41	Went out of hospital against advice.							
26/6/41	Patient died today.							

Discussion on Acute Granulocytopoenia.

Three cases of acute granulocytopoenia is a very small number on which to dogmatise, but this is a very rare condition. It is probably commoner in kala azar than in any other disease. Huang(25) describes two classes of the condition, one due to kala azar itself, and the other due to treatment of kala azar by the pentavalent antimony compounds. He found the incidence greater with neostibosan than with urea stibamine. All our three cases developed during the course of treatment with neostam (nitrogen glucoside of p-amino-phenyl-stibinic acid) which, we believe has not been previously reported as producing acute granulocytopoenia. In cases 201 and 252 treatment was carried on for a short period with very small doses of neostam, after development of granulocytopoenia. This was perhaps a mistake. Other treatment, as pentnucleotide was not available, was carried on with injections of sod. nucleinate, inj. camphor co., blood transfusion and quinine sulphate by the mouth. Blood transfusion seems to have very little effect on the course of the disease. Huang(25) was of the same opinion. All three cases recovered from the granulocytopoenia, and white counts and differential counts returned to normal but the angina progressed much as a cancrum oris, in the first two cases, and eventually caused death by toxaemia. The third case died of a low grade of pneumonia. The first two cases suggest that cancrum oris and acute granulocytopoenia are very closely related conditions, but

in/

in the third, no necrosis of mouth nor fauces was found. The results compare unfavourably with those of Huang[25] who records only 5 deaths in 17 cases (30%) due to antimony injections and two deaths in 20 cases due to kala azar (22%). It seems hardly possible that the use of pentnucleotide could make so much difference.

Davies and Wingfield[33] refer to remarkable results from simple injections of adrenalin in a case of agranulocytosis in kala azar. The injection was given as a stimulant, but seemed effective in stimulating the granulocytes. This method seems worthy of further trial. Their case developed acute granulocytopoenia during treatment with neostibosan.

OTHER COMPLICATIONS.

It will be appreciated that on arrival at hospital many of the cases were suffering from kala azar of some duration: in addition many of them showed evidence of nutritional deficiencies and infections not directly related to the principal disease. The following list includes therefore a mixed collection of conditions which either complicated the disease or were exacerbated by its presence.

Nutritional oedema - 35 cases. This complication was generally associated with extreme degrees of anaemia and the prognosis was bad.

Diarrhoea - 35 cases.

Acute inflammations - 30 cases. Abscess of neck 7; otitis media single 5; perisplenitis 4; ulcers of leg 3; otitis media double 3; abscess of buttock 1; cellulitis of neck 1; cellulitis of cheek 1; abscess of chest wall 1; furunculosis of legs 1; ulcers of lip 1; ulcer of cheek 1; mastoiditis 1.

Specific infectious diseases - 16 cases. Malaria 3; relapsing fever 3; chicken-pox 3; syphilis 3; whooping cough 2; smallpox 1; measles 1.

Bronchitis - 15 cases.

Pleurisy 13 - cases.

Lymph glands enlarged - 12 cases.

Skin conditions - 9 cases. Scabies 4; impetigo 2; psoriasis 1; ringworm of scalp 1; marked pigmentations of face 1.

Chronic/

Chronic inflammations - 7 cases. Nephritis 3; tuberculosis of lungs 1; tuberculous dactylitis of finger 1; tuberculosis of hip joint 1;chronic non-specific laryngitis 1.

Amenorrhoea - 6 cases.

Eye complications - 4 cases. Phlectenular conjunctivitis 3; Retinal and subhyaloid haemorrhage 1.

COMPLICATIONS FROM TREATMENT.

Vomiting after injection 180 (chiefly after neostam), diarrhoea after injection 48 (chiefly after neostam), ulcers of abdominal wall from native plasters 10, anaphylactic shock 8 (3 after urea stibamine, 5 after M & B '744'), needling of hands (native treatment to let out poison) 2, necrosis of skin of neck from antimony compounds injected outside vein 2, 1 died; abscess of buttock from intramuscular injection of neostibosan 2, abscess from native needling of abdominal wall 1, internal haemorrhage following sternal puncture 1 case, who died.

TREATMENT OF KALA AZAR.

The treatment requirements of each individual case required careful consideration. The patient's age, general condition, weight and blood picture are all important factors, as are also the presence of intercurrent disease such as pneumonia, cancrum oris, dysentery, tuberculosis and nephritis. I would say that pneumonia is the only definite contraindication for immediate antimony treatment, but in nephritis, in very anaemic children, and those with cancrum oris, one must give the treatment very gradually and carefully. Care must also be exercised where the leucocyte count is very low, especially if the percentage of granulocytes be diminished. If a variety of antimony compounds are available one must also choose the most/

most suitable for the individual case. We were never in this happy position, but solustibosan or neostibosan are probably the best compounds to start with, in a child who is very ill. The toxic reactions from these compounds is probably less, but their curative value is probably also less. One can always change over to M & B '744' or to urea stibamine later, when the child has partially recovered.

During the first few injections of the pentavalent antimonials, toxic symptoms such as vomiting and diarrhoea or even anaphylactic shock, are not uncommon, and there is often a negative phase. After the fourth injection, the patient generally improves rapidly, the fever settles and the appetite increases to almost abnormal proportions. This negative phase at the beginning of treatment is the danger period in the very ill, especially if cancrum oris has developed.

In our series treatment was all given by intravenous injections three times a week, and the course generally took about 1 month to complete. In all, over 8,000 injections were given intravenously in the clinic, and on only two occasions was any antimony spilled outside the vein. This is a great tribute to the skill of two Chinese nurses, Mr Li Chi-yü and Miss Wu Pi-hsia, who gave all the injections. In only one case was it necessary to have recourse to intramuscular injections of the pentavalent antimonials. Intramuscular injections of these salts are painful and produce a high percentage of abscesses.

The/

The following technique was developed for giving intravenous injections to young children. The child was held firmly in the lateral position, either on a high table or on its mother's knee, and the head allowed to fall down laterally. The head was firmly held in this position and the arms well controlled. The injection was made slowly with a fine hypodermic needle, into the external jugular vein. The children nearly always cried when placed in this position, and this facilitated the operation by distending the external jugular vein.

In considering the results of treatment in a condition like kala azar, where there are no standards of cure, the following arbitrary standards of results were adopted from clinical experience.

Cured. Spleen and liver no longer palpable - progressive improvement in blood counts with white count over 7,000 - progressive increase in weight - improvement in general health and good appetite - freedom from fever and other symptoms - observation for a period of at least 2 months and preferably for 6 months - total dosage considered adequate.

Doubtfully cured. Criteria as above except that period of observation was less than 2 months - spleen and liver much reduced but still palpable.

Much Improved. Case apparently proceeding satisfactorily towards cure, but spleen and liver still palpable when last examined which was at completion of treatment. Dosage adequate.

Improved/

Improved. Patient definitely improved but condition in some respects unsatisfactory, e.g. either spleen diminishing too slowly, or weight not increasing, or blood counts not progressively increasing when last seen, or total dose administered considered inadequate.

Improving Slowly. Improved, but condition unsatisfactory in more than one respect when last seen, e.g. insufficient dose and weight not increasing progressively. It is in this group that most of the recurrences occur.

Recurrence. Recurrence of fever, spleen increasing in size, weight and blood counts (especially white count) diminishing. Leishman-Donovan bodies found again after 6 weeks from completion of treatment (this last is not essential).

Moribund. Last seen in a moribund condition, but accurate information as to time of death not available. (N.B. This group is included as dead in all the mortality statistics.)

Dead. Definite confirmation of death obtained.

N.B. Films examined with negative results for Leishman-Donovan bodies after treatment are not considered essential for a "cured" result. Napier[32] has shown that the finding of Leishman-Donovan bodies after completion of treatment does not necessarily mean that the patient is not cured, and a large percentage of these cases go on to cure without further treatment. My own experience also confirms this. Taking these arbitrary standards, the end results in our 400 completed cases are shown in table 9.

TABLE 9.

The Results of Treatment of 400 cases of kala azar.

	1st 100	2nd 100	3rd 100	4th 100	400 cases	%
Cured	13	20	14	13	60	15
Doubtfully cured.	16	10	16	24	66	16.5
Much Improved.	28	26	16	32	102	25.5
Improved.	15	25	24	11	75	18.75
Improving slowly.	12	6	9	5	32	8
Recurrence (untreated)	3	4	0	1	8	2
Moribund.	1	1	9	2	13	3.25
Died.	12	8	12	12	44	11
Totals	100	100	100	100	400	100%

Total mortality = Moribund plus Died = 57 cases or 14.25%.

It is disappointing that the statistics of the later cases show little improvement from the earlier ones, apart from a reduction in those classed as "improving slowly", and as "recurrences". The mortality is comparable to that of other series published in China but is considerably higher than Napier's[32,38] Indian figures of approximately 5% after 6 months' observation. Comparison of the other figures with those of other workers is difficult, as each man has his own standards.

Prognosis./

Prognosis. The prognosis in kala azar is hard to assess. As has been pointed out by Napier[32], early cases may be just as resistant to treatment as the late ones, but I believe myself that the early cases are less liable to cancrum oris than the late cases, who have already become grossly anaemic. There is no doubt that cancrum oris, pneumonia, dysentery and other intercurrent infections make the prognosis grave, and that the mortality is highest below 4 years of age. With treatment with the pentavalent antimony compounds, recovery can be hoped for in nearly 90% of cases.

The average blood counts before and after treatment based on the complete record of 268 cases is given below for comparison with other figures.

	White Blood Count	Red Blood Count	Haemoglobin
Before Treatment	5,342	2,456,000	41.4%
After Treatment	7,547	3,140,000	49.7%
Percentage Increase	41.75%	27.8%	20.0%

Treatment with Various Antimony Compounds. Four compounds were used. Three could be classed as pentavalent salts, and the fourth as an aromatic diamidine.

A. Pentavalent. (1) Neostam i.e. nitrogen glucoside of P-amino phenyl stibinate (Burroughs, Wellcome and Co.) used in 4% solution intravenously. Makes a yellow-brown solution. Makers recommend 3 grams per 100 lbs. We found a higher dosage necessary especially in children. 281 cases treated entirely with this/

this compound. Vomiting and diarrhoea very common after injections. Post injection shock not common.

TABLE 10.

Results of treatment of 281 cases of kala azar with neostam.

	No. of Cases.	Percentage of total.
Cured	46	16.5
? Cured	38	13.6
Much improved	71	25.3
Improved	53	18.6
Improving slowly	21	7.5
Recurred	9	3.2
Moribund and died	43	15.3
	281	

Average Blood Count	White Blood Count.	Red Blood Count.	Haemoglobin.
before treatment	5,167	2,334,000	42.1%
after treatment	7,481	2,797,000	49.6%
% increase	44.59%	17.3%	17.93%

Average weight before 59.87 lbs. After 53.05 lbs. Percentage decrease 5.31%.
Average dose per 100 lbs weight = 4.77 grams.
Average decrease in spleen = 2.46 inches. In 102 cases spleen and liver could no longer be felt, i.e. 36.3%.

(2) Urea Stibamine. Urea and P-amino-phenyl Stibinic acid (Bramachari Calcutta). Percentage of antimony is not stated. Dosage recommended is 2/3 of corresponding dose of neostam or neostibosan (personal communication Peiping Union Medical College). Shock/

Shock symptoms with pallor, constriction of chest, and rapid feeble pulse were all common, and were severe in three cases. It is a lighter colour than neostam or neostibosan.

Results in 12 cases.

Cured 3 cases = 25%. ?Cured 2 = 16.66% Much improved 2 = 16.66%
Improved 3 = 25%. Died 2 = 16.66%.

Average Blood count	White Blood Count
before treatment	5,473
after treatment	9.150
% increase	66.8%

Weight before 52.8 lbs. After 57.4 lbs. Percentage increase = 8.0%

Average dose per 100 lbs = 1.9 grams.

In seven cases liver and spleen reduced to normal = 58.3%.

Average decrease in size of spleen = 1.77 inches.

(3) Neostibosan. Dimethyl amine p-amino phenyl stibinate. It is manufactured by Bayer, and is said to contain 40% of antimony. It gives a dark brown solution in water. The reactions from neostibosan are very much less than from the other compounds, but one occasionally sees vomiting and diarrhoea after injection. All the patients treated with compound bought the drug themselves at high prices, therefore they were wealthier patients and consequently better fed.

Results in 35 cases.

Cured 7 = 20%. ?Cured 11 = 31.4%. Much improved 10 = 28.6%. Improved 1 = 2.8%. Improved slowly 3 = 8.6%. Recurred and requiring further treatment - nil. Moribund or died 3 = 8.6%.

	White Blood Count	Red Blood Count	Haemoglobin.
Average blood count before treatment	6,330	2,490,000	43.7%
after treatment	9.440	2,866,000	51.55%
Percentage increase	47.37%	15%	18.00%

Average dose per 100 lbs = 4.44 grams.

Average weight before = 74.1 lbs. After = 80.2 lbs.
Percentage increase 8.23%.

Average decrease in spleen = 2.81 inches.

In 18 cases the spleen and liver could no longer be felt = 51.4%.

(4) M & B '744'. This is 4:4 Diamidine Stilbene Di-hydrochloride. It is one of the aromatic diamidines. It was described by Professor Warrington Yorke in 1940[34] and its use was described by Kirk and Sati[35] in 1940.

We were fortunate in securing 25 grams for trial through Dr C. C. Chesternan. Unfortunately we could get no literature as to the dosage, and had to find this out by trial. We found that a .60 gram total dose was necessary for a child, and 1.20 grams for an adult. The initial dose used was 0.05 gram for an adult and 0.02 for a child. M & B '744' is less soluble than the other compounds. It is a white powder with a greyish-yellow tinge, and makes a faintly yellow solution. We used it in a 2.5% solution in warm distilled water. Shock was not uncommon, with a sense of constriction of the chest, flushing of the face and conjunctivae, and rapid feeble pulse. This was severe in 5 cases. Kirk and Sati[35] describe the same syndrome and attribute/

attribute it to stimulation of the para-sympathetic. These cases were the last treated in the series, and there was not time for full follow-up.

Results in 20 cases.

Cured 1 = 5%. ?Cured 9 = 45%. Much improved 9 = 45%. Died 1 = 5%.

	White Blood Count.	Red Blood Count.	Haemoglobin
Average Blood Count before treatment	7,083	2,252,000	41%
after treatment	8,311	2,660,000	51%
Percentage increase	17.2%	13.99%	24.4%

Average dose per 100 lbs = 1.77 grams.

Average weight before = 53.2 lbs. After = 55.10 lbs.
Percentage increase 3.58% (due to short follow-up).

Spleen no longer palpable in 11 cases = 55%.

Average decrease in size of spleen = 2.81 inches.

A comparison of the four drugs used is not very valuable owing to the very small series with urea stibamine, neostibosan and M & B '744', but it is given for what it is worth in tables 11 and 12.

TABLE 11.

Comparison of results of treatment of kala azar with various drugs.

	No.of cases	Cured %	? Cured %	Much Imp. %	Imp. %	Imp. Slowly %	Re-curr-ence %	Moribund and died. %
Whole series	400	15.0	16.25	25.25	19.5	6.75	2.25	15.00
Neostam	281	16.4	13.5	25.3	18.6	7.5	3.2	15.3
Urea stibamine	12	25.0	16.66	16.66	25.0	-	-	16.66
Neostibosan	35	20.0	31.4	28.6	2.8	8.6	-	8.6
M & B '744'	20	5.0	45.0	45.0	-	-	-	5.0

N.B./

N.B. The remaining 52 cases were treated with more than one compound. The cure figures for M & B '744' are abnormally low because there was insufficient follow-up to justify the term cure.

TABLE 12.

Dose and effects of various drugs used in treatment.

	Av.dose per 100 lbs	% incr. WBC	% incr. RBC.	% incr. Hbg.	% incr. Wgt.	% Cases with spleen no longer palpable.	Av.Reduct. of spleen in inches
Neostam	4.77 gms	44.59	17.33	17.93	5.31	36.3	2.46 in.
Urea stibamine	1.9 "	66.80	-	-	8.00	58.3	1.77 "
Neostibosan	4.44 "	47.37	15.00	18.00	8.23	51.4	2.81 "
M & B '744'	1.77 "	17.2	13.99	24.4	3.58	55.0	2.99 "

Analysis of Results of Different Drugs Used.

As pointed out before, the neostibosan cases were a wealthier class and better fed. Had time been available for a full follow-up, I believe the M & B '744' results would have far surpassed the others. Our impression was that neostibosan was the least effective of the products and the least toxic.

I would use the various drugs in the following order of preference: (a) M & B '744', (b) urea stibamine, (c) neostam, (d) neostibosan reserved for initial treatment in very ill children. Struthers and C. C. Lin[36] report favourably on solustibosan/

solustibosan (a 2% solution of antimony hexonate, Bayer product) and states that it is even less toxic than neostibosan. We have not been able to obtain supplies of this drug for trial, but Struthers and Lin's report suggest it is the most suitable compound for the very young and for very toxic cases. Stephenson[37], in reporting an epidemic of kala azar in the Sudan with a mortality of 80% and a high incidence of cancrum oris, states that a patient with cancrum oris has more chance of recovery when treated with urea stibamine than with other compounds; I believe that M & B '744' would give equally good, if not considerably better results.

RECURRENCES.

The Problem of Recurrences.

Recurrences are a considerable problem in the treatment of kala azar. In a disease like this, where there is no single criterion of cure, and a case can only be considered cured when observed over a long period, it is questionable whether these should be considered recurrences or relapses. I have used the term recurrence as these cases appeared to be well and then, at a later period, presented themselves with definite symptoms of kala azar, though they appeared to have had adequate treatment in the first instance. The consensus of opinion is that such cases are recurrences, and that there is no such thing as re-infection once cure of kala azar has been established. This is my own opinion. This is the opinion of as great an authority

as/

as L. E. Napier[38].

There were 30 such recurrences in this series. There were also 11 cases who had been insufficiently treated outside the clinic, and who came to us for further treatment. These latter were not considered true recurrences. The age groups of the 30 recurring cases were as follows:-

Birth to 4.	5-9 years.	10-14 years	15-19 years	20 years and over.
2 cases.	3 cases.	3 cases.	2 cases.	20 cases.

There were 26 recurrences among males and only 4 females. There were 14 recurrences in the first 100 cases treated, 9 in the second 100, 3 in the third 100 and 4 in the fourth 100.

It was early considered that the doses recommended by the manufacturers of neostam was inadequate for children, and in consequence the dose was materially increased early in the series, with the result that recurrences among children were few. Later it was considered that the dose for adults was also insufficient, and this was also increased, so that the recurrence rate was steadily reduced until it reached 3 or 4 per 100 which seemed to be an irreducable minimum. The average dose per 100 lbs weight of the various compounds used was: neostam 4.77 grams, neostibosan 4.44 grams, urea stibamine 1.9 grams and M & B '744' 1.77 grams. The amount of pentavalent antimony required for the cure of any one case is impossible to estimate, and every case must be considered individually. By materially increasing/

increasing the total dose above that recommended, we seemed to cut down the recurrence rate considerably, without any harm to the patient. Though more extravagant in the use of the drugs, I believe this method was well worth while, as even when supplies are limited, as ours were, owing to war conditions, it is more satisfactory to treat a smaller number adequately than a large number inadequately.

The final results of recurrences were as follows: 18 cases were re-treated with even larger total doses, and were apparently proceeding satisfactorily towards cure. 3 died, and the remaining 9 were in the process of being re-treated, or had been advised further treatment, but had not attended for this. It must be stressed that, if the first course fails to bring about a cure, the patient inevitably becomes antimony resistant, and requires at least 50% more than the original total dose for cure, and also that a much longer period of follow up is necessary in these cases. One case received as much as 8.8 grams of neostam and only then was apparently cured (case No. 80).

The causes of recurrence were considered to be as follows: insufficient dosage 15 cases, insufficient dose and failure to report for follow up, 5 cases, antimony resistant 3 cases, intermitted treatment and consequent antimony resistance 2 cases, insufficient dose and opium habit 1 case, malnutrition and failure to report for follow up 1 case, malnutrition/

malnutrition and chronic dysentery 1 case, malnutrition and necrosis of jaw 1 case, tuberculosis of chest and peritoneum 1 case. 28 recurrences were after neostam, 1 after neostam and urea stibamine combined and 1 after urea stibamine alone.

Apart from insufficient dosage, the main causes of recurrence appear to be as follows:

1. Cases who are naturally antimony resistant and require exceptional dosage.

2. Cases who intermit treatment, and so become antimony resistant.

3. Malnutrition and wasting intercurrent disease such as tuberculosis, chronic dysentery, necrosis of the jaw and opium addiction.

Early treatment is not necessarily effective in preventing recurrence, but efficient follow up is most important in catching the recurrent cases early when they are still amenable to treatment. All our cases were instructed to report 4 times at fortnightly intervals. On each occasion they were weighed and the liver and spleen measured, and repeat blood counts done on two occasions. If the spleen and liver were not palpable at the end of this time, and weight and blood picture were improving satisfactorily, patients were dismissed and told to report immediately if there were any recurrence of symptoms. If their condition was not satisfactory at the end of two months, they were told to continue reporting at fortnightly, or shorter intervals, until apparently cured. 268 cases reported for follow up on 744 occasions. 132 cases failed to report.

Many/

Many of the latter were from far away districts (up to 400 miles distant). An attempt to visit those who did not report was made, but owing to shifting population and insufficient personnel, it was not very satisfactory. Follow up by letter enquiry was not possible as many of the people were illiterate, or had no postal address. It was only by constant exhortation that any follow up was obtained. The average duration of disease before treatment was 6½ months but this did not, I believe, materially affect the recurrence rate, though undoubtedly it affected the mortality.

MORTALITY.

Analysis of Causes of Death. In 51 of the 57 deaths kala azar was considered to be the primary cause of death. In the remaining 6 cases this was not so. The causes of death in these 6 cases were as follows: 2 cases due to dysentery and anaemia after apparent recovery from kala azar; 1 case from relapsing fever, dysentery, anaemia and cancrum oris after apparent recovery from kala azar; 1 case due to relapsing fever, dysentery, anaemia and necrosis of the jaw after apparent recovery from kala azar; 1 case due to pneumonia and anaemia after kala azar apparently cured; 1 case due to internal haemorrhage following sternal puncture, after kala azar treated and doubtfully cured.

The causes of death in the 51 cases primarily due to kala azar are given in table 13.

TABLE 13.

Analysis of the Ultimate Cause of Death in 51 Fatal Cases of Kala Azar.

7 cases due to anaemia.

19 cases due to anaemia and cancrum oris.

2 cases due to anaemia, cancrum oris and dysentery.

2 cases due to anaemia, acute granulocytopoenia and cancrum oris.

1 case due to anaemia, cancrum oris and bronchopneumonia.

1 case due to anaemia, necrosis of upper jaw and cancrum oris.

1 case due to anaemia and necrosis of jaw.

1 case due to anaemia, dysentery, bronchopneumonia and necrosis of jaw.

1 case due to dysentery and hypostatic pneumonia.

2 cases due to anaemia and bronchopneumonia.

1 case due to anaemia, bronchopneumonia and diarrhoea.

1 case due to anaemia, acute granulocytopoenia and hypostatic pneumonia.

1 case due to anaemia, tuberculosis of hip and tuberculous bronchopneumonia.

1 case due to anaemia, dysentery and bronchopneumonia.

1 case due to anaemia and dysentery.

1 case due to anaemia and lobar pneumonia.

1 case due to lobar pneumonia (recovery), and relapse of kala azar.

2 cases due to anaemia and malnutrition.

2 cases due to anaemia, diarrhoea and malnutrition.

1 case due to anaemia, purpura and malnutrition.

1 case due to mania and uraemia.

1 case due to anaemia, necrosis of tissues of neck following injection of pentavalent antimony around the ext. jug. vein.

44 cases were definitely known to have died and 13 were moribund when last seen. These latter are included as deaths in the above table. Of these 57 patients who died, 45 were males and 15 females, so that the mortality rate among females was proportionately higher than the number of females in the series. This is again easily explained by the poorer nutrition, and adverse conditions under which the females live.

The age groups of the patients who died were as follows:

Birth to 4 years.	5-9 years.	10-14 years.	15-19 years.	20 yrs. & over.
25 = 43.2%	8 = 14%	6 = 10.3%	2 = 3.5%	16 = 28%

Comparison of these figures with table 1, showing the distribution of kala azar according to age, makes it clear that the first four years of life was much the most fatal period in the series, the percentage mortality being almost twice the percentage incidence. In the adult group the percentage mortality is almost equal to the percentage incidence, whereas between 10 and 19, and especially between 15 and 19, the mortality percentage is very much lower than the incidence. This is as one would expect, as the very young succumb readily to infection, while the adolescent group have the greatest recuperative powers.

The average duration of disease, before treatment was instituted, was 7 months in the fatal cases, and the period of onset of symptoms till death average 8 months.

The average blood counts before treatment in the fatal cases were:-

White Blood Count.	Red Blood Count.	Haemoglobin.
5,654	2,056,000	36.6%

The white count is higher than the general average for the series, probably due to the fact that there were more children in the fatal cases, and the white blood count is higher in children, even when suffering from kala azar. It is interesting to note in this connection that all the cases of acute granulocytopoenia were in adults. The red count and haemoglobin were lower than in the whole series, which is interesting as anaemia is undoubtedly one of the crucial factors in kala azar. Again, the red counts and haemoglobin in children with kala azar tend to fall lower than those of adults with kala azar and this accounts partially for the low average.

43 of the fatal cases were treated with neostam, 2 with urea stibamine, 1 with M & B '744', and the remaining 8 cases with a combination of various compounds. Seven cases died after only one injection and six after the second injection, which means that these cases were practically moribund when first seen.

Discussion on Mortality.

It will be seen that the great majority of deaths were not from kala azar alone, but from cancrum oris, pneumonia, dysentery, acute granulocytopoenia and other intercurrent conditions. Actually, only 10 cases could be said to have died/

died of uncomplicated kala azar, that is, less than 20% of the fatalities. As pointed out above, the anaemia produced by the kala azar infection is the crucial point, especially in young children, and they consequently succumb readily to intercurrent disease. From this point of view, I believe, that though the earlier cases are often just as resistant to treatment as the late ones, yet the mortality must be higher among the late cases where anaemia has had time to become serious.

31 cases actually presented themselves for treatment with developed cancrum oris, and 16 of these died. I am therefore convinced that early treatment would lower the mortality rate.

CONTROL AND PREVENTION OF KALA AZAR.

This is the most important problem of all. Napier demonstrated that kala azar could be practically eradicated in areas in India by mass treatment of the disease. A similar attempt reported by H. L. Chung[9] in the village of Huaifang near Peiping, failed, probably owing to the presence of infected dogs, which constituted a reservoir host. Chung believes that further mass treatment with extermination of the infected dogs may eradicate the disease.

Shensi province is a vast virgin field for such an experiment and it is my earnest hope that such work may eventually be started there in the days of reconstruction after the war.

SUMMARY OF THESIS.

535 cases of confirmed kala azar treated in Sian, Shensi are recorded, and the history, epidemiology, pathology, diagnosis, complications and treatment of the disease with the relevant literature are reviewed.

The following opinions are expressed from observation of the cases.

1. That Shensi province is an endemic focus of kala azar, and that with war conditions the disease may be assuming epidemic proportions.

2. That immigrants into an endemic area of kala azar are probably more susceptible to the infection than the natives, especially when the former are undernourished refugees and soldiers.

3. That the dog may be a reservoir for leishmaniasis infection in Shensi and that phlebotomus is common in the area.

4. That cancrum oris, pneumonia, dysentery, and acute granulocytopoenia are the most important and fatal complications of kala azar in the area, and that the pathogenesis of cancrum oris may be closely related to that of acute granulocytopoenia. Three cases of acute granulocytopoenia, developing during the treatment of kala azar with neostam, are reported in detail.

5./

5. That cases of kala azar which have been cured are not liable to reinfection.

6. That the finding of Leishman-Donovan bodies on completion of treatment does not necessarily mean that the case is not cured.

7. That the mortality of the series was 14.25% but only one fifth of these died of uncomplicated kala azar, and that the mortality could probably be reduced by earlier treatment.

8. That the presence of anaemia is a critical factor in prognosis.

9. That M & B '744' promises to be one of the most useful drugs for the treatment of kala azar, but that solustibosan or neostibosan are probably safer drugs to use for the initial stages of treatment in very ill children.

10. Finally, attention has been drawn to the importance of H. L. Chung's work on the epidemiology of kala azar in the Peiping area. The application of this knowledge, with an effective scheme of treatment and follow-up, shows the way to the possibility of effective kala azar control in the Shensi area.

A P P E N D I X .

Photographs of advanced cases of kala azar are shown in plates I and II.

Appended is a list of the 400 completed cases of kala azar in the series, giving names, age, sex, treatment, important complications and results. The following abbreviations are used:-

N = neostam; Ns = neostibosan; U.S. = urea stibamine; 744 = M & B '744'.

Dosage is given in grams. Age is given in years and fractions of years, except when followed by an 'm' which indicates months.

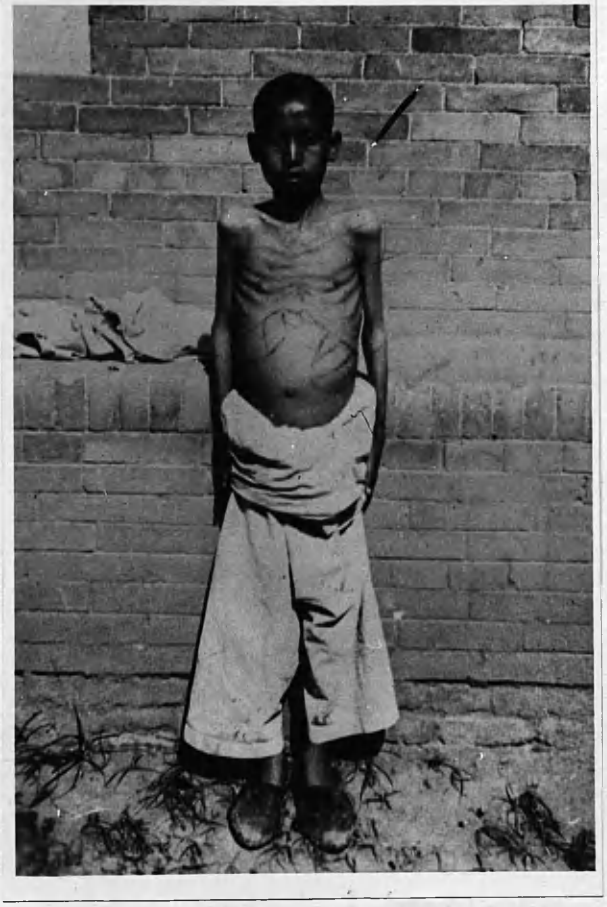
The following abbreviations are used to record the end results:

C = Cured. ?C. = Doubtfully cured. M.I. = Much improved.
I = Improved. I.S. = Improving slowly. Mo. = Moribund. D.= Died.

The following abbreviations are used for the commoner complications:-

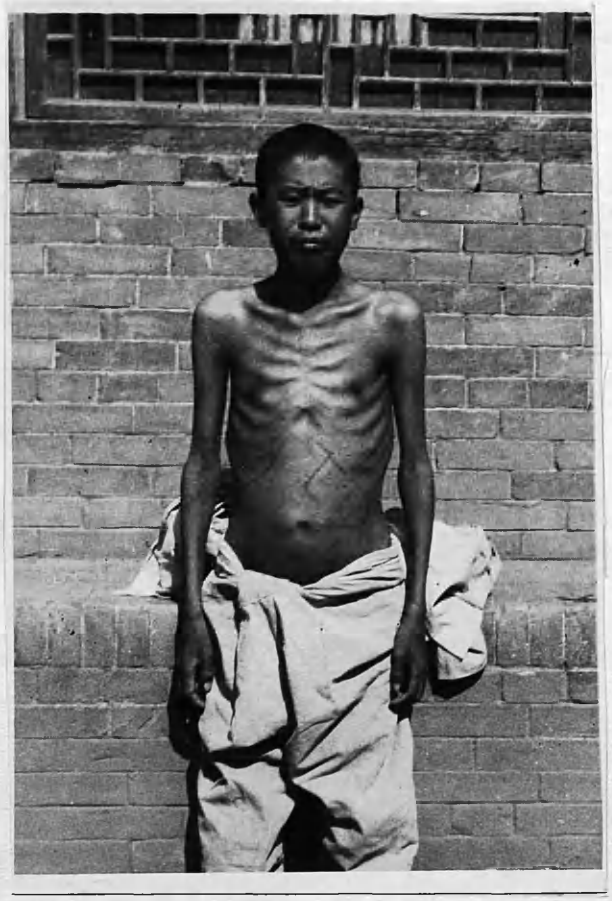
C.O. = Cancrum oris. Dy. = Dysentery. Di. = Diarrhoea.
C = Cough. G. = Gingivitis. E. = Epistaxis. P. = Pneumonia.

PLATE I. Advanced kala azar. Case 360.



Kao Yang To, aged 18 years; weight 57¼ lbs. Photographed on 18.10.41 after 1.45 gm. of neostibosan.

PLATE II. Advanced kala azar. Case 284.



Huang Lung, aged 27 years. Soldier. Recurrence after 3.0 gm. of neostam. Photograph taken on 18.10.41 during second course after an attack of pneumonia from which he recovered. Died 31.10.41 of recurrent kala azar with intense anaemia.

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RECORDS OF 400 FULLY TREATED CASES.

No.	Name.	Age & Sex.	Treatment Total Dose		Important Complications	End Results
1.	Wang Hsiao Hai	3m/M	N.1.35		G.	I.S.
2.	Ma Hsien	5/M	N.1.05		G.O.acute	D.
3.	Chang Hsiao Hai	1m/F	N.2.60		Abscess buttock	?C.
4.	Kuo Yu Chen	9/F	N.1.25		G.Dy.	I.
5.	Wang Kuan Shang	29/M	N.300			M.I.
6.	Wang Tan Wa	7/M	N.1.20		Lobar P.	C.
7.	Cheng Tzu Hsuan	23/M	N.3.85		E.	M.I.
8.	Sun Tun Wa	9/F	N.1.50		Bronchitis	?C.
9.	Sung Han Chang	21/M	N.3.30		E.G.Faruncul-osis.	M.I.
10.	Li Ya Ping	3/-	N.3.10		G.Albuminuria	?C.
11.	Kuo Hsi	2/F	N.0.85		Dy.C.	M.I.
12.	Chang Ai Yu	3/F	N.0.75			I.S.
13.	Hsieh Yu Hsien	3/F	N.1.00	U.S.0.6	Di.C.	I.S.
14.	Fan Yu Chen	4/F	N.1.55		Dy. Anaemia oedema.	?C.
15.	Hu Kuang Nai	6/M	N.1.05		Anaemia	I.S.
16.	Hou Feng Yin	3/F	N.0.70		C.O.Dy.Anaemia	Mo.
17.	Lu Yeh Nang	30/M	N.3.40		C.	?C.
18.	Ning Kuan Hua	18/F	N.3.20		Dy. Anaemia Amenorrhoea.	C.
19.	Wang Kuo San	8/M	N.1.30		C.	I.S.
20.	Li Hai Ch'ing	30/M	N.8.15		G. Anaemia.	I.
21.	Teng Fu Ch'ing	21/M	N.5.25	U.S.0.7	Nausea.	Recur-rence.
22.	Chao Ping Chi	8/M	N.1.10	Ns.1.3	C.O. Anaemia.	D.
23.	Wang Li Shih	20/F	N.0.80		Dy. C.O.	D.
24.	Chang Hsien Ching	21/F	N.3.20		Pregnant	?C.
25.	Chang Ju I.	31/M	N.2.90		E.C.Anaemia	M.I.
26.	Sun Yuan Wa	4/M	N.0.05		Acute C.O. Anaemia.	D.
27.	Li I Ying	16/M	N.1.65			I.
28.	Huang Chin Hai	20/M	N.3.20		E.G.Anaemia	M.I.
29.	Hsu Yin Te	17/M	N.2.00		E.G.	C.
30.	Li Yun	8/M	N.1.50		C. Anaemia, Ulcers of face.	I.
31.	Wang Pu Sheng	2/M	N.0.75		C.	I.
32.	Chow Heng Yin	2/F	N.0.50		G.Anaemia mar-asmus.	D.
33.	Ho Men Hsien	10/F	N.3.10	U.S.0.15		?C.
34.	Chang Kou Wa	8/M	N.1.05		C.O.Oedema	I.S.
35.	Yang Hsing Hua	25/F	N.2.65	U.S.0.20	C.O.	?C.
36.	Li Kuo Chen	25/M	N.2.60	U.S.0.20	G.Anaphylaxis.	Recur-rence.
37.	Hsia Hua Min	36/M	N.2.40	U.S.0.60	Di. Pleurisy	M.I.
38./						

No.	Name.	Age & Sex.	Treatment Total Dose	Important Complications	End Results
38.	Sun Pen Nien	4/M	N.0.80 U.S.0.10	C.O.	M.I.
39.	Chow Ta Li	20/M	N.2.60 U.S.0.60	E. Malaria, Anaemia.	M.I.
40.	Li Tu Sheng	1/F	N.0.40 U.S.0.68		I.S.
41.	Hsu Pao Pao	7/M	N.0.85 U.S.0.30	G.Di.Pleurisy with effusion	D.
42.	Hsu Shih Chen	21/M	N.2.35 U.S.0.40	E.	M.I.
43.	Jen Chao Hai	11m/M	N.0.50 U.S.0.10	C.	I.S.
44.	Fu Chieh Shih	5 /M	N.0.50 U.S.0.425	C. Anaemia	M.I.
45.	Meng Chung Wa	14/M	N.1.80 U.S.0.85	E. Dy.G.Anaemia	?C.
46.	Kao Chuan Pao	8/M	N.0.50 U.S.0.60	G.	I.S.
47.	Chang Chow Wa	8/M	N.0.30 U.S.1.15	Anaphylaxis	C.
48.	Lo Wei Chih	5/M	N.1.50 U.S.0.475	Mild Smallpox	M.I.
49.	Feng Kuan Ying	26/M	N.0.90	Chr.Granulo-cytopoenia.	D.
50.	Tung Wen Chieh	21/F	N.0.75 U.S.1.60	G.	C.
51.	Chen Shih Wa	15/M	N.0.50 U.S.1.35	E. Dy.	I.S.
52.	Che Hung Chun	17/M	N.0.90 U.S.1.35	E.	C.
53.	Teng Hsi Tou	17/M	N.0.45 U.S.2.25	E. G.	C.
54.	Wang Yung Te	4/M	N.0.20 U.S.0.575	C. O. E.	I.
55.	Chiu I Sun	30/M	N.0.45 U.S.1.75	G. E.	M.I.
56.	Huang Chu Chiang	6/M	N.S.0.85	Anaemia	I.
57.	Jen I Shih	25/M	U.S.1.95	Anaemia P.	I.
58.	Wang K'ai	28/M	U.S.1.95	Diurnal Fever	C.
59.	Feng Hsiung Lin	30/M	U.S.1.80	Anaemia Di.	I.
60.	Liu Chun Ting	33/M	U.S.2.10	Anaemia.Scabies	M.I.
61.	Tan Yin Wu	28/M	U.S.0.15	Oedema. Dy. Hypostatic P.	D.
62.	Li Hsing Chun	15/M	U.S.1.35	Ulcers of lip	C.
63.	Chang Tsui O	4/F	U.S.0.725	Di.	C.
64.	Keng Li Min	1½/M	U.S.0.60	Dy.	M.I.
65.	Peng Chin Tang	29/M	U.S.2.05		?C.
66.	Yang Chu Wa	16/M	U.S.1.45	Anaemia	C.
67.	Lou Chao Ti	6/F	U.S.0.70	Necrosis of Jaw Measles.	?C.
68.	Sun Chiu	15/F	U.S.0.75 Ns.0.80		M.I.
69.	Ho Chin Feng	3/F	U.S.0.50	Acute C.O.	D.
70.	Huang Hsin An	7/M	U.S.0.725	G.	M.I.
71.	Wu Su Shih	20/F	U.S.0.75 N.0.4	Dy. Diurnal fever.Anaemia	M.I.
72.	Chen Te San	28/M	U.S.0.9 N.0.8	Anaemia. Dy.	I.
73.	Liu Hsing Han	27/M	U.S.0.975 N.3.2	Diurnal fever.	M.I.
74.	Tan Pei Liang	26/M	U.S.0.70 N.1.7	E.	M.I.
75.	Ma Hung Shuang	23/F	U.S.0.825 N.2.15	Dy. Anaemia, Oedema, Amenor-rhoea.	I.
76.	Liu Chen Yu	28/M	U.S.0.75 N.2.0	Anaemia. E. Scabies.	M.I.

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No.	Name.	Age & Sex.	Treatment Total Dose	Important Complications	End Results
77.	Yen Chang Hsuan	8/M	U.S.0.175 N.1.25	C.O.Oedema Dy.	I.
78.	Tao Chiang Wen	20/M	N.3.30	G.	M.I.
79.	Mu Ling	20/M	U.S.0.25 N.2.40	Lymph glands enlarged.	I.
80.	Chao Wei Sheng	28/M	U.S.0.10 N.8.80		C.
81.	Shih Ching Jun	10/F	N.2.60 '744' 1.5.	G.	M.I.
82.	Wang Lin Hsueh	7/F	N.2.10	Anaemia Dy. C.	M.I.
83.	Ai Chao Shih	40/F	N.3.00	Anaemia. Loss of weight.	C.
84.	Yang Tzu Shuo	13/M	N.2.45	G. Dy.	I.
85.	Hang Cheng Hung	23/M	N.7.90	Anaemia albuminuria.	?C.
86.	Chien Hsun	20/M	N.9.00		M.I.
87.	Ma I Sheng	12/M	N.2.50	E. C.	I.S.
88.	Shih Kang Shan	5/M	N.2.50	Dy. G.	M.I.
89.	Tuan Ko Liang	20/M	N.0.25	Anaemia E. Di.	D.
90.	Wu Yu Shih	3/F	N.1.70	Anaemia	?C.
91.	Hu Kuo Hua	28/M	N.2.95	Anaemia. E.	I.S.
92.	Li Yun Ai	11/F	N.1.75	Relapsing fever Dy. Anaemia, Necrosis of jaw	D.
93.	Niu Tien Ling	10/M	N.2.35	G.	I.
94.	Li Hsueh Hsin	20/M	N.3.00	E. Diurnal fever.	M.I.
95.	Chow Tien Fu	9/M	N.2.60	E. G. Lymph glands enlarged.	?C.
96.	Chang Shu Yuan	29/M	N.3.00	G.	Recurrence.
97.	Tung Hsiang So	4/F	N.2.00	Anaemia	M.I.
98.	Tung Erh Nu	2/F	N.0.95	Anaemia.Broncho-pneumonia.	D.
99.	Tsui Wan Lu	21/M	N.4.70	Anaemia E. G.	?C.
100.	Kuo Wu Shih	21/F	N.9.00	E. Anaemia	?C.
101.	Hsiao Kan	26/M	N.2.90	Di. Diurnal fever.	M.I.
102.	Hsu Shen Yang	11/M	N.2.95	Anaemia	C.
103.	Chen Yu Kui	18/M	N.3.10	Anaemia G.	M.I.
104.	Wang Hsiao Tien	2/M	N.2.05	C. Anaemia Oedema of face.	C.
105.	Wang Fang So	12/M	N.2.85	C.E.G.Anaemia	C.
106.	Hsu Pei Ya	26/M	N.1.85	C.O.	D.
107.	Wang Hsin Min	31/M	N.7.20	Anaemia. Opium addiction.	M.I.
108.	Wang Chung Lin	24/M	N.7.50	E. C. Anaemia	?C.
109.	Liu Yang Wa	10/M	N.2.45	Anaemia G.	M.I.
110.	Chen Min Wa	1½/M	N.1.55	Anaemia	I.
111.	Kuo Chen Chu	5/F	N.2.00	Anaemia	M.I.
112.	Li Li Wa	7/M	N.2.20	Anaemia	C.
113.	Wang Chi Sheng	5/M	N.2.45	Anaemia G. C.	I.

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No.	Name.	Age & Sex.	Treatment Total Dose.	Important Complications	End Results
114.	Chang Chi Hai	12/M	N.1.10	Anaemia. Ring-worm of scalp.	D.
115.	Shih Chin Hung	6/M	N.2.20	Anaemia. C. Hands needled	C.
116.	Wei Chen Tsai	20/M	N.8.00	Anaemia E.	C.
117.	Tsao Kuang Ai	5/M	N.2.35	Dy.	?C.
118.	Wang Kao Shih	21/F	N.1.50	Anaemia C.O. Oedema.	D.
119.	Tsao Chin San	20/M	N.2.70	Anaemia.Acute granulocyto-poenia. C.O.	D.
120.	Chow Shuang Lan	10/F	N.7.60	Dy.	I.
121.	Fu Shuang Ho	1½/M	N.2.05	Anaemia	M.I.
122.	Tsao Hsueh Chin	1½/F	N.1.90	Anaemia.	C.
123.	Ma Pei Niee	19/M	N.3.00	E. Mastoiditis Pleurisy.	I.
124.	Chang Tebg Hsi	1½/M	N.2.05	Whooping Cough. Anaemia.	M.I.
125.	Liu Hsi Sheng	1/M	N.1.75		?C.
126.	Chang Hsiao Tang	22/M	N.3.00	G. Pleurisy.	M.I.
127.	Wang Sui Tsang	9/M	N.2.60	G. E.	M.I.
128.	Sun Chi Sui	9/M	N.2.60	E. Lymph glands en-larged.	I.S.
129.	Li Kou Wa	16/M	N.3.10	C. Anaemia.	I.S.
130.	Lei Meng Wa	4/M	N.2.00	Anaemia. C.	M.I.
131.	Ma Man Tang	19/M	N.2.90	E. G.	M.I.
132.	Chang Tzu Hai	13/M	N.2.60	C.	M.I.
133.	Hsieh Tao Sheng	23/M	N.3.00	Dy.	M.I.
134.	Wang Ching Chu	8/M	N.2.40	C.	?C.
135.	Yang Su Sheng	8/M	N.2.60	E. G. C.	?C.
136.	Sun Ai Fang	3/F	N.2.00	Anaemia. C.	M.I.
137.	Chao Pao Pao	3/M	N.2.00	Anaemia. C. O.	I.
138.	Hsia Hsiao San	12/F	N.2.80	C.	M.I.
139.	Wang Chung Hsueh	5/M	N.2.20	Anaemia. C. Impetigo Di.	I.
140.	Chang San	26/M	N.5.95	E.G.	C.
141.	Ku Hsia Hsin	34/M	N.3.60	Syphilis.	M.I.
142.	Wang Chu Wa	9/M	N.4.10	E. C. Lymph glands enlarged.	I.
143.	Wang Ching Chung	3/F	N.2.00	Anaemia. Necrosis of jaw.	M.I.
144.	Hsing Chan Wa	4/M	N.1.60	Anaemia. Dy. Broncho-P. Nec. of jaw	D.
145.	Yang Kou Sheng	14/M	N.3.00	E. G.	C.
146.	Liu Chang Nu	8/F	N.2.70	Anaemia. G.	M.I.
147.	Wang Sun Lien	16/F	N.2.80		I.
148.	Hsieh Chi Wa	11/F	N.2.10	Anaemia. G. Chronic Granulocytopoenia.	M.I.

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No.	Name.	Age & Sex.	Treatment Total Dose.	Important Complications	End Results
149.	Chu Te Hsiang	15/M	N.3.00	Anaemia.Bronchitis.	C.
150.	Chang Chi Hsueh	2/F	N.2.00	Di. C. Otitis media	C.
151.	Chin Chien Tu	1¼/M	N.0.60	Anaemia.G. C. O.	D.
152.	Chang Su Fan	5/F	N.2.20	C.	C.
153.	Tien Tien Pao	2/M	N.2.00		C.
154.	Wu Yin Ti	2/F	N.2.00	C. Anaemia. Di.	I.S.
155.	Liu I. Tsan	35/M	N.5.00	Anaemia. E.	?C.
156.	Han Shuang Te	14/M	N.2.80	Anaemia.	C.
157.	Li An Kuo	12/M	N.2.60	Anaemia. Pyorrhoea.	?C.
158.	Chang Nu Nu	6/M	N.2.20	G. Anaemia. Bronco-P.	I.
159.	Ma Wa	7/M	N.2.40	G. C.	I.
160.	Chang Tsai Hsia	1½/M	N.2.00	Anaemia.	C.
161.	Li Tsui Men	7/M	N.2.00	G. Anaemia. C. Chr. Granulocytopoenia.	?C.
162.	Ma Hai	12/M	N.2.60	Lymphocytosis.G. C.	I.
163.	Tung Pu Chin	17/M	N.3.00	C. Di.	M.I.
164.	Wang Ming Te	25/M	N.3.00		I.
165.	Yang Man Liang	14/M	N.2.95	E. C. Pyorrhoea.	I.
166.	Ching Fan Liang	3/M	N.2.20	Bronchitis. Anaemia. Di.	C.
167.	Fei Chia Tzu	18/M	N.3.00	E.G.Bubo. Syphilis.	I.
168.	Pai Tai Tai	19/F	N.3.00	C. Anaemia. E. Amenorrhoea.	I.
169.	Jen Chih Hsin	8/M	N.2.60	E. Lymph glands enlarged.	M.I.
170.	Chia Tseng Lu	22/M	N.3.55	C. E. Pyorrhoea	I.S.
171.	Chiao Tzu Chung	19/M	N.3.00	Anaemia. Oedema. Pleurisy.	I.
172.	Chang Chia Kuan	1¼/M	N.0.30	Anaemia. Acute C.O.	Mo.
173.	Han Chin Wa	6/M	N.2.30	Anaemia. C. G.	I.
174.	Yu Kuo Hua	15/M	N.3.05	E. C. Lymph glands enlarged.	C.
175.	Yang Hsu Hsu	7/M	N.2.20	Dy.	I.S.
176.	Chia Fan Ting	2/M	N.2.00	Anaemia. G.	I.
177.	Ma Ming Chang	16/M	N.3.70	Anaemia. E.	Recurrence.
178.	Ma Tzu Hsu	33/M	N.7.00	E. C. Anaemia	C.
179.	Wei Ting Hsi	8/M	N.2.20	C. G.	I.
180.	Tao Fu Min	21/M	N.3.00	E. C.	Recurrence.
181.	Chu Wa	13/M	N.3.00	E. C. G. Malaria	C.
182.	Ma Hai Yun	35/M	N.3.00	G. Anaemia	I.
183.	Su Chi Lin	26/M	N.5.85	Anaemia	I.
184.	Han Yin Te	35/M	N.3.20		I.
185.	Shih Ying Chow	7/M	N.2.10	Bronchitis. Anaemia	I.
186.	Sun Chu Ma	7/M	N.3.65	E.Anaemia. Oedema	1.
187.	Chao Sui Chiang	15/M	N.2.85	E.Anaemia. Impetigo	M.I.
188.	Hsia Ma Lien	18/M	N.3.00	C. E.	M.I.
189.	Yang Chih Kuei	5/M	N.4.00	C. E.	Recurrence.
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No.	Name.	Age & Sex.	Treatment Total Dose.	Important Complications	End Results
190.	Chang Tung Sheng	10/M	N.2.80	C.T.B.Dactylitis Cellulitis leg.	M.I.
191.	Hu Sheng Jung	4/M	N.2.00	E. G. C. Otitis media	M.I.
192.	Li Sun Hsia	8/F	N.2.20	G. Subacute C.O.	?C.
193.	Cheng Yun Ting	28/M	N.3.00	Chronic Di. C.	Recurrence.
194.	Tung Pu Yun	16/M	N.3.05	E. G. C.O.	I.
195.	Chang Yin Te	9/F	N.2.15	Anaemia. Oedema	C.
196.	Sung Pen	1¼/M	N.0.55	Anaemia. Cough. E.	D.
197.	Yuan Hsi Yuan	2/F	N.0.25	Anaemia. C.	D.
198.	Chao Shu Men	2/F	N.2.00	G. Bronchitis	M.I.
199.	Fu Ping An	5/M	N.2.00	C.	?C.
200.	Hsu Miao Ta	20/M	N.3.00	C. E.	I.S.
201.	Huang Hsiao Chen	29/M	N.2.40	Anaemia Ac.agranulocytosis. C.O.	D.
202.	Wang Mu Sao	7/M	N.2.00	C. G.	M.I.
203.	Chang Yin Men	10/F	N.3.00	E. C. Anaemia	C.
204.	Tso Yin Chen	2½/M	N.1.85	Chronic C.O. Anaemia	I.S.
205.	Han Ching Yu	13/M	N.2.80	C. E.	C.
206.	Liu Hsi Men	7/M	N.0.15	Anaemia. Acute C.O.	Mo.
207.	Yao Chi San	2/M	N.0.40	Anaemia. Acute C.O. Anaemia.	Mo.
208.	Chang Yin Kuan	5/M	N.2.00	G. E. Chr. Granulocytopoenia.	I.
209.	Tang Hung	31/M	N.2.80	C. E. G.	I.
210.	Liu Su Mao	54/M	N.0.10	Anaemia.Mania.Uraemia	D.
211.	Liu Kuang Wa	4/M	N.0.60	Anaemia.Acute C.O.	D.
212.	Wu Ming Ching	30/M	N.3.00	Anaemia. C.	I.
213.	Teng Chia Chiu	34/M	N.5.00	Anaemia E. G.	C.
214.	Li Chin Mao	1½/M	N.0.25	Anaemia.Broncho-P. Di. Oedema.	M.O.
215.	Lo Ken Sheng	4/M	N.1.80	G. C.	C.
216.	Wang Shou Jen	10/M	N.2.60	Anaemia.E.Chicken-pox Hands needled.	C.
217.	Tsai Chiu Min	1½/M	N.1.60	Anaemia C.	I.
218.	Chen Chio Ting	2/F	N.2.00	Anaemia. C.O. C.Di.	?C.
219.	Kuo An Kang	1½/M	N.1.65	Anaemia C.O.	M.I.
220.	Chang Shen Tao	26/M	N.3.10	Chr.Granulocytopoenia C.O.	M.I.
221.	Liu Chien Hsuan	25/M	N.3.3	C.	I.
222.	Chen Chin Liang	3/M	N.2.00	Anaemia	?C.
223.	Wang Fu Hsi	31/M	N.3.00	Anaemia. Necrosis of maxilla.	?C.
224.	Wang Chang Chuan	16/M	N.3.00	Relapsing fever. Dy. Anaemia. Acute C.O.	D.
225.	Chang Hsin Nien	6/M	N.3.30	E.G.	M.I.
226.	Hsieh Ching Shou	14/M	N.3.00	E.Lymph glands enlarged	?C
227.	Li Hsin Chen	2/F	N.2.00	Anaemia. G.	I.S.
228.	Sun Kuo Pao	5/M	N.2.20	Anaemia	I.

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No.	Name.	Age & Sex.	Treatment Total Dose.	Important Complications.	End Results.
229.	Wang Ming So	2½/M	N.I.80	Dy. Anaemia.	D.
230.	Pang Kuang Hui	15/M	N.3.00	E. Oedema	I.S.
231.	Liu Tzu I	5/F	N.0.15	Anaemia. Acute C.O.	Mo.
232.	Liu Shuang Hsi	6/M	N.2.00	Anaemia C.	I.
233.	Kuo Wen Tien	17/M	Ns.0.75 N.0.50	Anaemia. Oedema	?C.
234.	Chow Yu Tien	5/M	N.1.9 Ns.1.1	C. Otitis media.	I.
235.	Chang Chu Hua	25/M	N.7.00	E. G. Subacute C.O.	?C.
236.	Chih Ping Wa	½/M	N.1.50	Anaemia C.	C.
237.	Wang Hsiao Kou	2/F	N.1.50	G.	C.
238.	Tsao Chin I	14/M	N.3.00	Anaemia.E. G. Pleurisy with effusion.	I.
239.	Yang Sun Shih	18/M	N.3.00	G.	?C.
240.	Ma Mu Sha	6/M	N.2.40	Anaemia. Malaria.	C.
241.	Tao Tai Liang	2/M	N.0.05	Anaemia. Broncho-P.	D.
242.	Shih Huan Yu	18/M	N.3.10	G. Chicken-pox	?C.
243.	Teng Chien Hou	6/M	N.2.00	Anaemia. G. C. Necrosis of jaw.	I.
244.	Shih Yu Fang	5/F	N.2.00	Anaemia.	M.I.
245.	Li Chiu Lan	4/F	N.1.75	Anaemia. C. Oedema.	I.
246.	Pao Hei Wa	11/M	N.2.40	Subacute C.O. Anaemia. Lobar P.	C.
247.	Kuo Ying Hsi	3/F	N.1.90	Anaemia.	C.
248.	Tsai Hsiao Chung	10/F	N.2.60	Anaemia. G.	I.
249.	Sun Chang An	3/M	N.1.80	C. Anaemia E.	M.I.
250.	Chu Wai Chuan	3/M	N.2.00	Anaemia. Subacute C.O.	C.
251.	Kou Kuei Sheng	4/M	N.1.80	C.G.Abscess of neck.	I.
252.	Yao Ti Chen	24/M	N.1.80	Anaemia. Ac.Agranulo-cytopoenia. Hypostatic P.	D.
253.	Tung Tse Hsuan	32/M	N.4.50	C. Diurnal fever.	I.
254.	Yuan Yu Yin	28/M	N.4.00	E.	?C.
255.	Chen Shao Kang	28/M	N.3.00	C. Diurnal fever. Anaemia.	I.
256.	Huang Yun Chiang	9/M	N.2.50	C. Anaemia.	I.
257.	Chen Lai Hsuan	5/M	N.2.20	Anaemia. Ulcers of abdominal wall.	M.I.
258.	Chao Ping Yin	18/M	N.3.00	E. G. C.	M.I.
259.	Han Chang Wa	17/M	N.2.80	E. C.	I.
260.	Tuan Man	6/F	N.0.75	Acute C.O. Broncho-P.	Mo.
261.	Lo Chuan Shui	1¼/M	N.1.50	Anaemia. Bronchitis.	I.
262.	Chu Pei Yao	7/M	N.2.40	G. Anaemia. Pleurisy	?C.
263.	Kang Li Wa	3/F	N.1.80	C. E.	?C.
264.	Huang Shang Lin	1¾/M	N.1.75	Anaemia	I.
265.	Ma Hsing	7/F	N.0.05	Anaemia. Acute C.O.	Mo.
266.	Jung Lien Yung	1½/F	N.1.75	Anaemia. Di. Whooping cough.	?C.
267.	Wu Chang An	3/M	N.1.80	C. Anaemia.	C.

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No.	Name.	Age & Sex.	Treatment Total Dose.	Important Complications.	End Results.
268.	Wu Su Sheng	4/M	N.0.05	Acute C.O. Anaemia.	Mo.
269.	Wang Hsi Chien	10m/M	N.0.35	Anaemia. Necrosis of skin of neck.	D.
270.	Ma Tzu Chiang	13/M	N.3.00	Pyorrhoea. C.	?C.
271.	Chiang Hung Hsi	30/M	N.3.00	E. C. Pyorrhoea	I.S.
272.	Sun Ming Chi	5/M	N.2.60	G. Anaemia	C.
273.	Ho Fu	37/M	N.4.50		M.I.
274.	Sun Hsuan Lai	4/M	N.2.00	C.	M.I.
275.	Wu To Te	2/M	N.0.45	Anaemia	D.
276.	Wang Sun Ming	34/F	N.3.20	Opium Addiction. Anaemia.Menorrhagia.	I.S.
277.	Chia Yueh Ming	11/M	N.3.00	G. Anaemia	?C.
278.	Chi Hsiao Men	5/F	N.2.10	C. G. Anaemia.	M.I.
279.	Wang Chiao Shih	24/F	N.3.00	Anaemia. Marasmus.	D.
280.	Shen Fu Tang	9/M	N.2.75	G. E. Ulcer of cheek.	M.I.
281.	Chiang Pei Ching	24/M	N.3.60	E.	C.
282.	Ching Te Wa	12/F	N.2.85	E. Anaemia	I.S.
283.	Lei Hung Liang	13/M	N.3.00	C. E. G. Di.	M.I.
284.	Huang Lung	27/M	N.5.00	Lobar-P. Anaemia. Oedema.	D.
285.	Wang Te Hsiao	23/M	N.3.00	Pulmonary Tuberculosis.	I.S.
286.	Wang Meng Shih	32/F	N.3.05	Anaemia	I.
287.	Li Wen Tsan	22/M	N.3.10	E. Angina of throat.	I.S.
288.	Chang Ping Shen	18/M	N.3.00	E. C. Anaemia	M.I.
289.	Li Tso Jen	30/M	N.3.50	C. E. Hiccough	I.
290.	Li Chuan	7/F	N.3.00	C. G. Lobar-P.	M.I.
291.	Yuan Chu Wa	6/M	N.2.25	E. C. Anaemia	C.
292.	Shih Huan Tsao	11/M	N.0.05	Anaemia. Acute C.O. Oedema.	Mo.
293.	Lou Ying Wa	5/F	N.2.10	Anaemia. Necrosis of maxilla. C.O.	Mo.
294.	Wang Tai Tai	34/F	N.3.15	Anaemia E. Opium addiction	I.
295.	Shih Sui Ko	21/M	N.3.75	C.O. Anaemia. Oedema. Ascites.	I.S.
296.	Wang Shih Jui	23/M	N.3.50	C. Pyorrhoea	I.
297.	Ho Wen Ying	1½/F	N.1.50	G.	?C.
298.	Sung Fu Yin	16/M	N.3.00	C. G.	M.I.
299.	Chen Chin San	30/M	N.3.50	C. Anaemia.	I.S.
300.	Yang Tzu Ying	22/M	N.1.35	Anaemia. E. C. Malnutrition.	D.
301.	Wang Pao Shan	20/M	N.3.00	E. C. Dy.	Recurrence.
302.	Kao Man Liang	6/M	N.2.10	E. Ulcer of abdominal wall.	C.
303.	Chang Niu Wa	5/F	N.2.00	E. C.	I.
304.	Kao Tu Wa	13/M	N.3.00		M.I.
305.	Tsan Lu	6/M	N.2.90	E. G.	I.
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No.	Name.	Age & Sex.	Treatment Total Dose.	Important Complications.	End Results
306.	Liu Feng Wa	3/M	N.1.80	Anaemia. G.	I.S.
307.	Sun Hsi Cheng	12/M	N.3.00	Anaemia	?C.
308.	Hsu Feng Feng	11/F	N.3.00	Anaemia. Subacute C.O.	M.I.
309.	Yao Shu Chin	1½/F	N.1.60	C. Anaemia.	C.
310.	Lu Cheng Wa	10/M	N.3.00	G.	?C.
311.	Chang Kung Chang	8/M	N.2.80	Necrosis of mandible.	M.I.
312.	Li Jiu Wu	22/M	N.3.10	Anaemia. Di. C.	I.
313.	Yen Shu Hsia	15/F	N.3.00	Anaemia. Malnutrition.	M.I.
314.	Hou Kuang Jen	1¾/M	N.2.00	Anaemia. Di.	I.
315.	Sun Hsiao Lan	3/F	N.2.00	Anaemia. Vomiting.	M.I.
316.	Wang Chung Lin	13/M	N.0.15	Anaemia. Acute C.O.	Mo.
317.	Chang Chang An	4/M	N.0.20	Anaemia. Acute C.O.	D.
318.	Chang Chu Yin	4/F	N.1.80	Anaemia. C.	M.I.
319.	Cheng Te San	30/M	N.4.00	E. C. Syphilis	I.
320.	Chen Te Cheng	20/M	N.3.00 '744'0.05	Di. Acute C.O.	D.
321.	Yang Kuan Hsin	4/M	N.1.80	Anaemia E. Di.	?C.
322.	Ning Wu Chien	10/M	Ns.0.20	Anaemia T.B.Hip joint T.B. Broncho-P.	D.
323.	Wang Chung Yu	7/M	N.5.40	Anaemia. Necrosis of jaw. Dy.	I.S.
324.	Chang Jui Yuan	5/M	N.2.00	Anaemia. E. C. Necrosis of maxilla.	M.I.
325.	Hsu Chien	23/M	N.4.00	Diurnal fever.	C.
326.	Cheng Wei	27/M	N.3.50	Anaemia C. G.	M.I.
327.	Wang Li Lin	5/M	Ns.2.15	E. G. Anaemia. C.	C.
328.	Yen Ya Ching	28/M	N.3.00 Ns.0.80	G. Anaemia. Malnutrition.	I.
329.	Liu Wang	10/M	N.3.00	Anaemia. G. Subacute C.O.	?C.
330.	Li Ta Ching	3/F	N.1.90	Anaemia. G. C.	M.I.
331.	Kuo Pao Tsang	5/M	N.0.15	Anaemia. Oedema.	D.
332.	Han Ming Sheng	3/M	N.1.80	Anaemia. G. C. Broncho-P.	D.
333.	Li Wen Jui	10/F	N.0.05	Anaemia. Lobar-P.	D.
334.	Chen Chi Nu	5/M	N.2.00	Anaemia. C.	C.
335.	Shih Lin Hua	14/M	N.3.00	C. E. Dy.	C.
336.	Fu Sheng An	11/M	Ns.3.10	Di.	M.I.
337.	Li Hung Jang	3/M	N.1.80	Anaemia. C. Necrosis of jaw.	M.I.
338.	Cheng Chen Shih	18/F	N.3.00	Scabies.	I.
339.	Shih Yen Ming	14/M	Ns.3.00	E. C. Pyorrhoea.	C.
340.	Chou Fu Min	14/M	Ns.3.00	Anaemia. C.	M.I.
341.	Chen Shui Kui	7/M	Ns.2.20	Anaemia. C.	C.
342.	Hu Mao Hsueh	7/M	Ns.2.40	Anaemia. C. Int.Haemorrhage following sternal puncture.	D.
343.	Wu Wei Ling	27/M	Ns.4.00	Pigmentation of face. Dy. Pyorrhoea.	C.
344.	Kao Hsiao Pang	6/M	Ns.2.40	E. C.	M.I.

345./

No.	Name.	Age & Sex.	Treatment Total Dose.	Important Complications.	End Results
345.	Wang Hung Yin	7/M	N.2.40	C. E. Phlectenular Conjunctivitis.	M.I.
346.	Li Yu Pei	1½/M	Ns.1.80	C. Anaemia. E. Di.	M.I.
347.	Chen Tzu Chang	35/M	Ns.4.00	Anaemia. Retinal & Sub-hyaloid haemorrhages.	C.
348.	Hu Chen Chung	3/M	Ns.2.00	E. Anaemia.	?C.
349.	Chang Kun Lan	12/F	Ns.3.00	Dy. Anaemia.	C.
350.	Sung Lien Cheng	7/M	N.2.20	Anaemia. Chr.C.O. G.	I.
351.	Pien An Hai	1¼/M	Ns.2.25	Anaemia. G. Chr.C.O.	?C.
352.	Wu Shui Hsueh	4/M	Ns.2.00	Anaemia. G.	I.
353.	Huang Miao	26/M	Ns.4.00	Anaemia. Died of Lobar-P.	D.
354.	Yang Wu Fa	4/M	Ns.1.20	Anaemia. G.	D.
355.	Wang Hsueh Chien	7/M	Ns.2.40	Anaemia. C.	?C.
356.	Chen Hsin An	6/M	N.2.20	G. E. Oedema	C.
357.	Liang Hsiu Yuan	11/M	Ns.3.00	C. Anaemia	I.
358.	Meng Tsang	8/M	Ns.3.00	Anaemia. Necrosis of jaw.	C.
359.	Ting Keng Lu	8/M	N.2.40	Anaemia. E.	M.I.
360.	Kao Yang To	18/M	Ns.4.50 N.0.20	C. Chr. Dy. Malnutrition. E.	M.I.
361.	Li Ping Shen	14/M	Ns.3.00	E.	?C.
362.	Tseng Wa	1¾/M	N.0.20	Anaemia. Dy. Died of Dysentery.	D.
363.	Wang Wu Wa	15/M	N.4.00	Anaemia. G.	M.I.
364.	Liu Hsiao Wu.	6/M	Ns.0.20	Anaemia. Acute C.O.	Mo.
365.	Kao Hai Tang	10/F	'744'1.20	E. G.	?C.
367.	Nan Yu Wa	9/F	'744'1.20	C. Anaemia. Abscess of neck.	M.I.
368.	Wang Pao Yu	10/F	'744'1.20	E. G.	?C.
369.	Tung Chien Wu	37/M	Ns.3.90	E. Anaemia. Pleurisy.	?C.
370.	Huang Chia Ling	37/M	Ns.1.60	Anaemia. Faruncle.	?C.
371.	Wu An Sheng	8/M	Ns.3.00	Anaemia. G.	I.
372.	Chang Chi Lui	25/M	Ns.3.90	Anaemia. E. G.	?C.
373.	Li Tzu Ying	18/F	Ns.4.00	E. Amenorrhoea.	?C.
374.	Fu Yu Yu	3/M	'744'0.80	Anaemia.	?C.
375.	Hsiao Chia San	13/M	'744'1.50	C. Anaemia.	C.
376.	Chang Mao Ting	24/M	Ns.5.00	E. C. Abscess of abdominal wall from 'needling'.Lobar-P.Dy.	?C.
377.	Chang Tsung Fa	5/M	'744'0.65	C. Anaemia	M.I.
378.	Chang Yu Men	4/M	'744'0.025	Anaemia. Bronchitis.	D.
379.	Chang Chuan Wa	1/M	Ns.2.00	Anaemia. E. G. Broncho-P.	?C.
380.	Kao Chiao Huan	4/M	'744'0.40	Anaemia. C.	?C.
381.	Wu Tsung Hui	6/F	'744'0.65	Anaemia.G.	?C.
382.	Chang Chen Tien	21/M	'744'1.25	Anaemia. E. G. Necrosis of skin of leg	?C
383.	Peng Chiang	28/M	'744'1.20	Anaemia. E.	M.I.

384./

No.	Name.	Age & Sex.	Treatment Total Dose.	Important Complications.	End Results.
384.	Li Tien Yu	15/M	'744'1.20	Anaemia. G.	M.I.
385.	Hou Fu Hsien	7/F	'744'0.60	Anaemia. G.	?C.
386.	Yang Tsui Ting	11/F	'744'1.00	Anaemia. G.	?C.
387.	Liu Shen Ying	20/F	Ns.4.00	Anaemia. Amenorrhoea.Di.	M.I.
388.	Han Hsiao Lin	4/M	Ns.1.80	Anaemia. G. E. C.	M.I.
389.	Chou Fu Chieh	19/M	Ns.5.00	Anaemia. Pyorrhoea.	M.I.
390.	Ku Shou Wa	19/M	'744'1.30	Anaemia. C. E. Pyorrhoea	M.I.
391.	Chen Chien Hsien	32/M	Ns.3.65	Anaemia. C. Di. Pleurisy	M.I.
392.	Hou Tzu Te	24/M	Ns.5.00	Anaemia. C.	M.I.
393.	Li Chen Ching	5/M	'744'0.70	Anaemia. Acute C.O.	?C.
394.	Chou Chen Tung	10/F	'744'1.55	E. Anaemia. Chr.Granulocytopoenia.	M.I.
395.	Yang Hsiang Hsiang	11/F	'744'1.00	Anaemia	M.I.
396.	Chiang Chien Hsueh	20/M	'744'1.00	Anaemia. C. G.	M.I.
397.	Chao Hang Chen	28/M	Ns.5.00	Anaemia. G. Di.	I.S.
398.	Chen Hsing Shen	7/M	'744'0.60	Anaemia. Subacute C.O.	M.I.
399.	Wang Ting Su	23/M	N.0.80 '744'0.10	Anaemia. Necrosis of maxilla. Orbital Cellulitis.	D.
400.	Wang Shu Ying	24/F	N.3.30	Anaemia C. Amenorrhoea, Oedema.	?C.

Records of 135 cases who did not complete their treatment are also given below.
Photographs of Cases 284 and 360 are submitted (Plates I and II).

RECORDS OF 135 INCOMPLETE CASES.

1.	Li Keng Hsien	22/M	N.0.80	
2.	Wu Chih Hai	2/F	N.0.25	G. C.O.
3.	Teng Shu Yuan	22/M	N.1.40	C.
4.	Li Chen Tu	27/M	N.0.80	C. E.
5.	Hsi Hsueh Wen	45/M	Nil	
6.	Han Yao Kung	16/M	N.0.60	G.
7.	Tsui Chu Wa	2½/F	N.0.05	C.O.
8.	Wang Fu Tang	8/M	N.0.40	G.
9.	Chang Hsien Yen	28/M	N.0.50	Di.
10.	Wei Tang We	23/M	N.0.20	C. Pyorrhoea.
11.	Wang Chun	26/M	N?0.80	C. Chr. Laryngitis.
12.	Wang Huo	13/M	N.0.10	Anaemia. C.O.
13.	Huang Ching Hua	19/M	U.S.0.15	E. C. G. Lymph glands large.
14.	Tsao Ching San	20/M	U.S.0.35	G.
15.	Su Sheng Ping	1/M	N.0.15	Anaemia. C.O.
16.	Liu Nien Shang	11/M	U.S.0.30	G. Oedema.
17.	Sung Hsiao Hai	7/M	U.S.0.025	Di. Anaemia. C.O.
18.	Li Tang Chen	3/M	N.0.20	C.

19./

No.	Name.	Age & Sex	Total Dose	Complications
19.	Yao Chun Yu	37/M	U.S.0.05	Diurnal fever.
20.	Wang Hsiao Hai	3/M	U.S.0.075	G. Dy. Oedema, Albuminuria. No casts.
21.	Yu Min	28/M	N.0.30	G.
22.	Chang Chi Wa	6/M	Ns.0.15	Anaemia. Bronchitis Nephritis.
23.	Chao Fu Wen	16/M	Ns.1.15	Oedema. Lymph glands enlarged.
24.	Wang Ling	10/F	N.0.80	Dy. Chr.Granulocytopoenia.
25.	Liu Hu	7/M	N.0.50	Anaemia. Dy. G.
26.	Chang Mai Shih	3/M	Ns.1.80	Anaemia. Oedema.
27.	Hsiu Pei Shen	4/M	N.0.80	Di.
28.	Ho Niu Tu	4/M	N.0.15	Dy. C. Oedema.Petechiae.
29.	Wang Yun Lai	21/M	Ns.0.30	Anaemia. Pyorrhoea.
30.	Chen Chung Yu	38/M	N.0.10	Anaemia.
31.	Tung Ma Wa	4/M	Ns.0.10	Dy. C. Anaemia.Petechiae
32.	Chiao Ying Hsia	22/M	Ns.0.10	G.
33.	Yao Su Chin	16/F	Ns.2.00	Amenorrhoea. C.
34.	Chang Ke Pin	18/M	Nil.	C. Chr.Granulocytopoenia
35.	Shuo Sun Tao	5/M	Ns.0.20	C.
36.	Lo Te Jang	2/M	N.1.05	Anaemia. Di.
37.	Chiao Tien Hsi	16/M	Ns.0.50	Anaemia.
38.	Chen Kuang Lin	4/M	Ns.0.70	Anaemia. C.O.
39.	Chiao Yung Feng	18/F	Nil.	
40.	Yuan Feng Shih	26/F	Ns.1.55	Anaemia.
41.	Fang Po	10/M	Nil.	Anaemia. C.O.
42.	Wang Pao Min	9/M	N.1.20	Anaemia. C.O.
44.	Chu Ming Ching	6/M	N.0.15	Anaemia.
45.	Mao Nu	12/F	Ns.1.05	Anaemia. Di.
46.	Ming So	7/F	N.0.25	C.
47.	Fan En Shan	6/M	Ns.1.20	C. Anaemia.
48.	Liu Ping Kang	27/M	Ns.0.60	E.G.
49.	Chiao Chang Chien	10/M	N.1.35	E.C.
50.	Li Hai	10/M	N.0.10	Chr.Dy.
51.	Fu Yu Ai	6/F	N.1.20	C. Oedema.
52.	Yin Chuan Lin	30/M	N.0.10	C.
53.	Chu Hsin Lo	2/M	N.0.05	Anaemia.
54.	Wei Yin Hsiang	20/M	N.0.50	Anaemia. Oedema.
55.	Chung Hui	1/M	N.1.35	C.
56.	Ho Pao Te	10/M	N.0.05	Anaemia. G.
57.	Lan Chuan Kuang	12/M	N.2.30	C. Lymph glands enlarged
58.	Chu Pao Wa	13/M	N.1.70	Bronchitis.
59.	Yang Hsu Ting	11/M	N.0.10	E. Chr.Granulocytopoenia
60.	Liu Hsiao Kou	2/M	N.0.05	C. Anaemia. E.
61.	Yin Hsiao Nu	32/M	Nil.	G.
62.	Hsi Ping Te	25/M	N.0.10	Pyorrhoea.
63.	Chang Lin	8/M	N.0.30	Anaemia.
64.	Cheng Wei Ai	2/M	Nil.	C.O.

65./

No.	Name.	Age & Sex.	Total Dose.	Complications.
66.	Pi Tzu Lan	4/F	N.0.35	C.O.
67.	Tsai Jang	9/F	N.0.30	C. Anaemia.
68.	Chao Hsiao Pao	16/M	N.2.10	E. Dy. Pyorrhoea.
69.	Li Wen Min	14/M	N.0.60	Anaemia. C.O.
70.	Pei Chuan Li	18/M	N.2.25	Anaemia. Perisplenitis Constipation.
71.	Ma Li	18/M	N.2.50	C. E.
72.	Liang Yung Shan	10/M	N.0.55	Anaemia. Oedema. E.
73.	Sung Chin Pin	4/M	N.0.65	Anaemia. Dy. Cellulitis of cheek.
74.	Tsao Chin Wa	10/M	N.0.30	Anaemia.
75.	Chu Yu Shen	3/M	N.1.10	Anaemia.
76.	Li Cheng Chien	4/M	N.0.65	Necrosis of jaw. C.O. Dy. Oliguria.
77.	Lou Tsuan Tsuan	4/F	N.1.60	C. Di. Anaemia.
78.	Ching Yang Lan	9/F	Nil.	Anaemia.
79.	Huang Shuang Hsi	6/M	N.1.50	C. Anaemia. G. Dy.
80.	Shao Pei Tao	4/M	N.0.85	G. Anaemia. Di.
81.	Tsai Wen Cheng	26/M	Nil.	Anaemia. Ankylostomiasis.
82.	Cheng Tien Hua	17/M	N.0.15	Chr.C.O. Necrosis mandible. Oedema.
83.	Fu Hui Hsi	6/M	N.1.35	Subacute C.O. Anaemia.
84.	Niu Wu Yuan	8/M	N.0.95	Anaemia. C.O.
85.	Wang Chung Hai	10/M	Nil.	Anaemia. C.
86.	Wang Ching Chu	3/M	N.0.55	Anaemia. G. C.
87.	Jen Yueh Men	7/F	N.0.05	Chr. C.O.
88.	Chao Chu Lan	6/F	Nil	Anaemia.
89.	Keng Hsiao Hsin	10/F	N.0.40	Anaemia. E. Necrosis of mandible. C.O.
90.	Sun Yu Chien	7/M	Nil	Anaemia. E. Dy.
91.	Liu Tung Li	27/M	Nil	Anaemia.
92.	Chang Te Ming	7/M	N.0.15	C.O. Anaemia.
93.	Ti Chung Wa	6/M	Nil	Anaemia.
94.	Chinag Tzu Liang	26/M	Nil	C. Anaemia. Pyorrhoea. Oedema.
95.	Wang Pei Ling	4/M	N.0.25	Anaemia. Double otitis media. C.O. Dy.
96.	An Feng Men	8/F	N.0.15	Anaemia. C.O.
97.	Wu Tien Hsiang	10/M	Ns.0.75	C. E.
98.	Liu Chen Ping	35/M	N.0.05	Petechiae. Necrosis of tonsillar region.
99.	Yeh Jui Chiao	40/M	Nil	Dy. Psoriasis.
100.	Yen Chia Hsuan	13/M	Nil	E. G. C.
101.	Lui Chang Tzu	24/M	Nil	E.
102.	Tse Yun Chang	26/M	Nil	Anaemia.
103.	Han Tzu Hsing	2½/M	Ns.0.05	Anaemia. C.O.
104.	Chen Chu Chi	1½/M	Ns.0.05	Anaemia. C.

105./

No.	Name.	Age & Sex.	Total Dose.	Complications.
105.	Li Chu Chen	30/M	Ns.0.60	E. C. Pyorrhoea.
106.	Teng Keng Yang	12/M	Ns.0.10	C. G.
107.	Chen Chen Hsing	22/M	'744'0.45	
108.	Pai Wen Wen	9/M	Nil	Necrosis of mandible.
109.	Chao An Min	11/M	Ns.0.75	Anaemia. Chr.Granulocyto-poenia. Oedema.
110.	Shih Ping Ho	17/M	'744'0.15	G. Necrosis of maxilla.
111.	Hsi Te Ping	21/M	Nil	G. Dy.
112.	Ma Ke Chien	6/F	'744'0.45	G. C.
113.	Chang La Sheng	12/M	'744'0.275	Necrosis of mandible. C.O. Oedema.
114.	Kuo Keng Wu	8/M	Nil	Anaemia.
115.	Ho Chun She	8/M	Nil	Anaemia. G.
116.	Tsai Hsien Chu	30/M	Nil	Anaemia. G.
117.	Han Chin Lien	2/F	N.0.05	Anaemia. C.
118.	Chang Hung Yen	3/M	'744'0.25	Anaemia. G.
119.	Hsieh Ken Ping	4/M	'744'0.25	Anaemia. C.O.
120.	Li Hsien Hsi	5/M	N.0.85	Anaemia. Di. C.O. G. Otitis media. Oedema.
121.	Shih Te Hou	10/M	N.1.35	Anaemia. C.O. Di. G.
122.	Chu Su Chin	5/F	N.1.50	Anaemia.
123.	Feng Chao Hsing	5/F	N.0.10	Anaemia. C.O.
124.	Lo Ping Yin	21/M	N.3.00	Anaemia. Lobar-P.
125.	Chang Yueh Shui	14/M	N.1.10	Anaemia. G.
126.	Wang Fan Sheng	7½/M	N.1.35	C. Anaemia. C.O.
127.	Chen Chung Tang	31/M	N.0.30	Anaemia. C.O.
128.	Chang Hsing Hua	2/M	N.0.15	Anaemia. C.O.
129.	Liu Yu Wa	10/M	N.0.65	Anaemia. G. Oedema.
130.	Sun Nu Nai	2/M	N.0.15	Anaemia G. C.O.
131.	Chang Hu Lai	1½/M	N.1.10	Anaemia. Oedema. Broncho-P.
132.	Li Chin Lan	4/M	N.1.75	Anaemia. Bronchitis.Oedema.
133.	Ho Hsiao Jung	11/M	N.1.35	Anaemia. E. C. Chr.Di.Oedema.
134.	Tsai Hsiao Chung	9/M	N.1.20	Anaemia. Necrosis of jaw.
135.	Liu Chih Te	14/M	N.1.60	Anaemia. C. E.