

PNEUMONIA IN GOLD COAST .

1944.

(With Observations on Broncho-Pulmonary Anatomy.)

BY

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P R E F A C E.

This is an account of pneumonia in native West African Troops who were treated at a General Hospital of 300 beds in the Gold Coast over a nine months' period from January 8th 1944, when the Hospital opened in the station. During this time the writer was physician in charge of the Medical Wards.

The paper emphasises the importance of acute respiratory disease in African Troops, compares and contrasts pneumonia with that usually seen at home and gives an account of investigations undertaken to elucidate its aetiology.

In addition, observations are made on certain aspects of pulmonary anatomy in relation to pneumonia.

I wish to thank Brigadier G.M. Findlay C.B.E., Consulting Physician in Tropical Medicine, West African Force, for his constant encouragement and stimulating suggestions. He carried out some of the animal experiments in person and helped us with others. In addition, he was responsible for putting into effect the measures which controlled the major epidemic. To Major A.R. Aidin, R.A.M.C., and to his laboratory staff, I am indebted for the large amount of pathological work so willingly executed and for the care of experimental animals; to Captain R.F. Ashwin R.A.M.C., radiologist to the Hospital, for complete co-operation and to his staff for help with photographs. I am grateful to these Officers for many happy hours spent in their respective departments.

I am pleased to acknowledge a personal communication, with valuable observations on these pneumonias, from Dr. Tom Anderson, Glasgow.

Many thanks are also due to Lieut. Colonel E.G.R. Grant T.D., R.A.M.C., Officer Commanding the Hospital, Colonel S.D. Reid, late R.A.M.C., A.D.M.S., Gold Coast Area and to Brigadier J.B.A. Wignore late R.A.M.C., D.D.M.S., West African Force, for permission to record these cases.

(T. Semple)

KUMASI  
KUM Gold C  
Gold Coast.  
Nov 44.

## I n t r o d u c t i o n .

Little stress has been laid on the incidence of common respiratory infections, especially pneumonia, in the Tropics and on the susceptibility of the African to them. The young physician is constantly struck with the high proportion of conditions similar to those seen at home. He may see, as the writer has found, more cases of pneumonia in a few months than he has seen in general hospitals at home during the whole of his medical career.

In addition, other infectious diseases of temperate climates such as chicken pox, rubella, measles and meningococcal meningitis are not uncommon. He will almost certainly be surprised at the comparatively low incidence of admissions for diseases peculiar to the Tropics.

The total number of admissions to the African Medical Wards of the Hospital during the period under review was 1947, and of these, the high figure of 1062 (54.5%) were acute respiratory infections. 733 patients were classified as suffering from pneumonia. The great majority of these (655 or 89% of the pneumonias) came from a large Primary Training Centre. Other units in the station were comparatively small.

Table I gives an impression of the relative proportion of tropical and non-tropical diseases found.

Of 15 deaths which occurred, 2 were tropical diseases (Trypanosomiasis and Sick cell crisis). Of 57 soldiers discharged

Table I

Table I. The Proportion of Tropical and Non-tropical Diseases treated in the Hospital from January till September 1944.

Total Admissions to the Medical Wards	-	1947.
Upper Respiratory Infections	-	329 (16.9%).
Pneumonia	-	733 (37.7%).
Common infectious Diseases	-	569 (29.9%).
Tropical Diseases (including the Dysenteries)	-	173 (8.9%).

from the service on medical grounds, 11 had diseases peculiar to the Tropics.

Owing to the epidemic of pneumonia which occurred during the period, the figures given in Table I rather accentuate the importance of non-tropical infections. In a similar analysis of acute respiratory conditions in African troops during a nine months' period, MacNaught and Murray-Lyon (1943) report that of 1250 admissions to the Medical Division of a larger West African General Hospital, 375 (30%) were respiratory conditions.

It is of interest to note that in the Surgical Wards of the Hospital, the proportion of tropical diseases is much higher owing to the frequency of conditions such as tropical ulcer, dracontiasis, yaws and schistosomiasis.

Table II. Quarterly Admissions to Hospital of Respiratory Infections, October 1943 till September 1944.

	<u>Oct-Dec.</u>	<u>Jan-Mar.</u>	<u>Apr-Jun.</u>	<u>Jul-Sept.</u>
Number of Pneumonias admitted.	256.	490.	187.	156.
Proportion from Primary Training Centre.	Not known	465 (93%).	156 (83%).	35 (63%).
Number of Cases of Pharyngitis admitted.	4.	155.	75.	30.



The Epidemic of Pneumonia.

In Table II, the period under review has been divided into three, each of three months and it shows the number of pneumonias admitted during these times. For sake of completeness, corresponding figures are given for the months October to December 1943, when the patients were cared for by a smaller medical unit than in the station.

It will be shown later that the general appearance and symptomatology of the African with acute upper respiratory infection is very similar to that of a European with lobar pneumonia. It seems likely that a proportion of these labelled pneumonia during October to December 1943 were indeed suffering from naso-pharyngitis. No X-Ray facilities were available to the medical unit concerned during this quarter.

Figure 1 shows the curve of the epidemic during the first four months of 1944. The peak occurs during the third week of February, when the strength of the Primary Training Centre was also at its height.

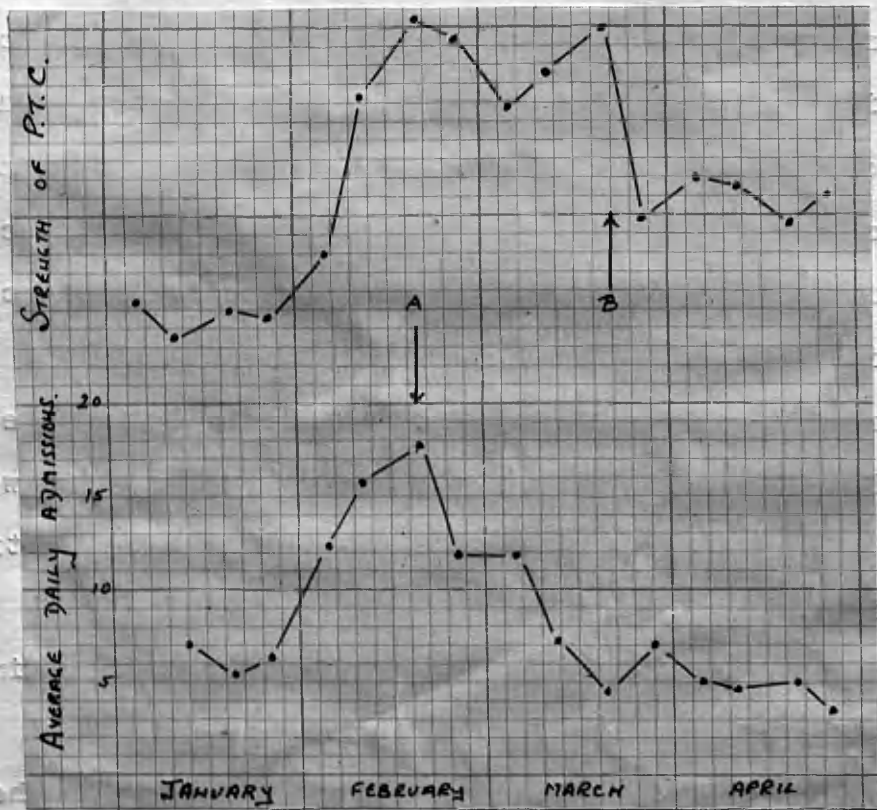
Preventive measures were put into effect at this time, on the recommendation of the Consulting Physician:

(1) Steps were taken to diminish overcrowding in the Primary Training Centre:

a. by erecting large numbers of tents (to accommodate 1000 men)

b. by immediately limiting the intake of recruits. For administrative reasons, the numerical strength of the unit did not show an immediate or significant fall.

Figure 1. Showing the Relationship between the Weekly Strength of the Primary Training Centre (top graph) and the Average Daily Hospital Admissions of Respiratory Infections (lower curve) during the Epidemic Period.



- A. Preventive measures put in operation.
- B. Two companies sent to outstations.

(new 3000)

c. Two Companies were sent to out-stations at a later date - 20th. March (Arrow B. Fig.1)

(2) A large unit sick bay was opened, where men with upper respiratory infections could be isolated for a few days. Previously such men had been excused duty and returned to bed in billets, so disseminating infection in no uncertain fashion. Medical Officers were now instructed to isolate all febrile patients and those with apparent upper respiratory catarrh.

(3) More hospital beds were made available, mainly in tents. It should be noted that these tents were used as far as possible for convalescent patients, as pneumonias in the acute stage fare very badly in such a close atmosphere.

The response to these measures was an immediate fall in the number of admissions to the hospital wards. This continued as a lysis of the epidemic during the next few weeks.

It will be shown that pneumonia during the epidemic was of rather unusual type which was invariably preceded and accompanied by a well marked upper respiratory tract infection. During the later months when the epidemic had subsided, one could detect, at least in some cases, a change in character to that of a classical pneumococcal lobar pneumonia.

Table III.

LENGTH OF SERVICE.

<u>Length of Service.</u>	<u>Pneumonia.</u>	<u>Pharyngitis.</u>
Less than 4 weeks.	445 (31%).	127 (49%).
4 - 12 weeks.	181 (25%).	75 (29%).
3 - 6 months.	47 (6%).	18 (7%).
Over 6 months.	60 (8%).	40 (15%).

### Causative Factors.

The main factors in causation and maintenance of the epidemic would appear to be:-

- (1) The exposure to infection of fresh batches of susceptible persons at short intervals.
- (2) Overcrowding in quarters in the principal unit concerned.
- (3) Seasonal Factors.
- (4) Malnutrition.
- (5) Unaccustomed exertion and protective inoculations.

### Exposure and Susceptibility.

During the first few months of the year, batches of recruits, numbering usually about 200, arrived at the Training Centre at intervals of roughly two weeks. Before the peak of the epidemic, this interval had been reduced to one week. Many of the cases occurred during the first week or two of service (445, or 61% of the pneumonias during the first 28 days of service - Table III) The majority of these men came from isolated areas in the Northern Territories of the Gold Coast. Many had never before been near a sizeable town, exposed to the infections associated with even comparatively civilised habitation; they had lived in lonely communities, each in his own enclosed native compound with his family, separated sometimes by a hundred or more yards from his neighbour.

One has the impression that the severity of the infection was in some degree related to this lack of civilisation. Literate men or those on the staff of the Training Centre were usually

found to have a simple upper respiratory infection, only occasionally necessitating admission to hospital. The recruit "whose language no one could talk" seemed always to have a well marked pneumonia.

Pagan beliefs (those other than Christian and Mohamedan) might be expected to be more common in the uncivilised tribes and it is of interest to note that they were also more frequent among patients who progressed to pneumonia.

	<u>Upper Respiratory Disease</u>	<u>Pneumonia</u>
Proportion of "Pagans"	90 (34.6%)	325 (44.3%)

The healthy African has a poorly functioning thoracic respiratory mechanism; his chest expansion rarely exceeds two inches. Probably this is largely due to climatic conditions, cold refreshing winds apparently being necessary to stimulate metabolism and inspiration. The European, likewise, finds his breathing shallow in this country, doubtless why patients with chronic respiratory disease such as asthma fare so badly in the Tropics. The African, then, may be truly "weak chested" and relatively susceptible to lung infections. Certainly, pneumonia is considered by Colonial Medical Officers to be one of the commoner ills of the people.

#### Overcrowding and Droplet Infection.

During the third week of February, when the epidemic was at its peak, the number of recruits in excess almost equalled the true establishment of the Training Centre. Only a handful of tents had been erected as extra billets and the result was serious overcrowding.

Until about a year before this, all barrack rooms in the unit were divided into small cubicles, each accommodating two men. This was intended to approximate to the African mode of living in small

individual giddas. Regimental Officers have drawn attention to the fact that pneumonia has only become a problem since the partitions were removed, leaving large spacious barrack rooms on European lines.

This seems of exceptional interest in view of recent literature which has pointed out the advantages of the cubicle type of room over large wards in preventing air infection in hospitals (M.R.C. Memorandum "The Control of Cross Infection in Hospitals")

Overcrowding is probably the more important in these native troops because droplet infection has a wider range than in Europeans. It is unusual to see a man guard a cough or turn his head away from a nearby fellow; more commonly it is sudden, forceful and directed into space, as the examining medical officer may find to his personal discomfort.

It would appear that respiratory disease is one of the major problems when shipping West African Troops to the East. Here again, below deck, we have conditions par excellence for spread by droplet infection.

#### Seasonal Factors.

Harmattan, hot, dry wind from the Sahara, affects this part of the Colony during the first few weeks of the year; the climax of the epidemic occurred when it was most intense. During this season, the inhabitants are aware of a sensation of dryness in the nose and throat and a slight reddening of the pharynx is general. It is well recognised as "the pneumonia season".

There seems little doubt that this dehydration of the respiratory mucosa - no doubt tracheo-bronchial mucous membrane is likewise affected - diminishes resistance to attack by pathogenic

organisms. Part of the respiratory defense mechanism, as it were, is put out of commission.

At this time of year, there is also quite marked variation in temperature, nights often being comparatively cool. Later in the year also, one associated smaller peaks in the curve of incidence of respiratory infections with periods when variation of temperature was more pronounced. MacNaught and Murray-Lyon (1943) discussing acute respiratory conditions in African soldiers, stress chill as a very important precipitating factor, as shown by three peaks of high incidence, associated with cold conditions, during a nine months' period.

#### Malnutrition.

Moderate degrees of malnutrition and anaemia were common among the new intakes. The diet of these men from the Northern Territories is grossly deficient in protein and Vitamin B complex. In the present series, 187 (25.5%) of the pneumonia patients showed evidence of under-nourishment and avitaminosis.

Some oedema of ankles and a dry, glossy, paved skin on the lower two thirds of the legs, were common. The skin of the scrotum might be similarly affected and sometimes the trunk had a generalised ichthyotic appearance. Glossitis and cheilosis were less frequently seen.

The improvement in general condition of such men after only a few weeks of army feeding was striking and if treated with adequate amounts of Vitamin B complex, one could see improvement within a few days.

Mild hypochromic anaemia was usual, a haemoglobin of 60% not



uncommon, and in several patients, failure to react to treatment was found to co-exist with a more severe anemia. Some of these were shown by sternal marrow examination to be of dimorphic type, nutritional in origin and reacting well to appropriate treatment with liver and iron.

#### Unaccustomed Exertion and Protective Inoculations.

Few of these recruits were accustomed to lengthy periods of work and exercise. During the first weeks, T.A.B., Tetanus Toxoid, Vaccination and Yellow Fever inoculations were administered and appreciable reactions were not uncommon.

It is clear that the less robust of these men must experience quite an ordeal during this early period of service and it is hardly surprising that they lose, at least temporarily, what little resistance they may have to respiratory infection.

Symptomatology.

The history as obtained from the African soldier is short and usually to the point; he knows when he is ill and still better when he has recovered. Leading questions have to be employed in most instances but, taking this into account, there was remarkable consistency of symptomatology during the epidemic.

Headache and pain in the chest for two to four days before admission were almost universal. The pain was substernal or across the lower anterior chest wall and was described as a dull ache. A dry cough was usual and general symptoms of fever, such as shivering, pains in the back and joints, and constipation were often described. A true rigor never occurred at any stage of the illness and this was a differentiating feature from the type of pneumonia often seen during the later months of the period under review. Rarely were sore throat and nasal catarrh volunteered but not infrequently they were admitted.

Such, then, were the symptoms during "the pre-pneumonic stage" and those given by patients who did not pass the stage of upper respiratory tract involvement.

With the onset of pneumonia, which was more or less insidious, they complained of productive cough, dyspnoea and acute inspiratory pain, localised to some area of the chest wall. In more toxic patients, pain in the liver region was common.

### S i g n s.

Pharyngitis was invariably present during the pre-pneumonic period. There was quite intense injection and oedema of pharynx and soft palate with a greenish muco-purulent discharge in the naso-pharynx. Although nasal discharge was unusual, there was always some congestion and oedema of the nasal turbinates, which can be examined with ease in the African merely by elevating the tip of his nose.

170 (23%) of the pneumonia patients had this upper respiratory inflammation alone on admission, signs of pulmonary involvement not appearing later.

Tonsillitis, catarrhal otitis and sinusitis were not uncommon. Although a mild tracheitis probably accompanied the pharyngitis, it was never an outstanding feature and laryngitis was rarely found.

With the onset of pneumonia, physical examination of the chest revealed:-

(a). The commonest picture was a patch, often easily recognised as one or more anatomical segments of a lobe, showing inspiratory crepitations, usually coarser than typical crepitus induced with or without bronchial breathing and signs listed in (b).

(b). Diminished movement, impaired percussion note and breath sounds with tenderness over a similar area. Vocal resonance might be increased or suppressed.

(c). The same features affecting areas in two lobes, often both lower lobes.

(d). Typical complete lobar consolidation which occurred in less than 15%.

Table IV. Illustrating the Lobar Areas of Jaundice and The Principal Lobe Affected. Number.

Right Upper Lobe.	65 (8.9%).
Right Middle Lobe.	78 (10%).
Right Lower Lobe.	168 (22.9%).
Left Upper Lobe.	38 (5.2%).
Left Lower Lobe.	235 (32%).
Areas in both lungs.	154 (21%).

Preponderance of Pneumonia, the Incidence Average Length of Stay in Hospital. Incidence of Average Number of Days in Hospital of Uncomplicated Cases. Jan-Mar 1944. Jul-Sept 1944.

14 (21%).	9.3	13.0
4 (5.5%).	9.1	12.0
10 (6%).	9.0	9.8
2 (5.3%).	9.1	10.0
8 (3.4%).	9.1	10.8
10 (6.5%).	9.1	15.6

Table IV illustrates the relative proportions of consolidation in individual lobes or in sequents of these lobes.

Pleural Friction, over the affected area was not unusual especially during convalescence.

Herpes Labialis, occurred only in about 5% of these infections.

Jaundice was present in 48 (6.5%) of the pneumonias, being most common, as were other toxic manifestations, in right upper lobe affections (21.5%)

#### Haematological Findings.

Polymorph leucocytosis was invariably present at all stages of the infection until resolution of the pneumonia was occurring. Even in the pre-pneumonic stage and in these patients who did not progress past that of naso-pharyngitis, it was usual to find leucocyte count of 12,000 to 20,000 per cmm., and with the appearance of pneumonia the count rose to 16,000 to 30,000 per cmm.

The high incidence of anaemia in these men has already been mentioned. Most of these were of simple microcytic type. In others iron deficiency was combined with a nutritional macrocytic anaemia to give the picture of so-called dimorphic anaemia. This was diagnosed by sternal marrow puncture. The myelogram showed a proportion of megaloblasts as seen in pernicious anaemia. It responded to treatment with liver and iron. Such patients often had severe forms of pneumonia, tending to spread to other lobes while under treatment (Figs. 13 and 14) and to be slow in resolving.

Tests for sickling were not carried out as a routine procedure and no conclusions could be drawn from the comparatively small number of examinations made. There is some evidence that Africans

who exhibited the sickle cell trait have a more severe attack of pneumonia.

Evans (1944) found that 15.5% fit men in West Africa showed this characteristic. His figure was 25% in those suffering from acute and chronic disease and the highest incidence 28.5% occurred in respiratory diseases such as pneumonia, pulmonary tuberculosis, pleurisy and lung abscess.

Findlay, in a survey of 300 soldiers in the Gold Coast, also found 15.5% of sicklers. He reports (Unpublished data 1944) that, of 1189 cases of pneumonia in West Africa tested for sickling, 117 (9.9%) were positive.

There would seem, then, to be little evidence that the sickle cell trait is associated with an increased susceptibility to pneumonia.

## The Sputum.

### Macroscopic Appearance.

During the stage of upper respiratory tract involvement, there was a greenish muco-purulent sputum derived from the naso-pharynx.

At the onset of pneumonia, the spit was mucoid and scanty; later it was muco-purulent, and with resolution, it became liquid and moderate in amount. Blood-streaking or part staining was present in less than half the cases. When copious and frothy, it seemed in the main to be saliva.

In many patients not treated with sulphonamide, and in an odd one so treated, a rather characteristic golden yellow spit was seen.

The typical rusty spit of common pneumococcal lobar pneumonia was rarely if ever witnessed during the epidemic period. Nor did one see the rose red frothy mucus or brown tenaceous varieties characteristic of severe influenza.

### Bacteriological Examinations.

These were made in early cases, later untreated ones and in treated cases. The sputum was collected in sterile stoppered bottles and special endeavour was made to obtain material coughed up from the lung.

Pneumococci were seen only in a proportion of these, seldom predominated and were never in profusion. Attempts to type such organisms by direct Neufeld method met with no success. Mice were not available for indirect typing.

Direct smear and culture invariably produced a "mixed bag" of organisms such as coagulase negative staphylococci, non-haemolytic streptococci, streptococcus viridans, pneumococci, B-subtilis group,

*N. catarrhalis*, Gram negative rods and even a Gram negative coccobacillus resembling *B. friedländer* but not identified as such. *B. influenzae* were not found in appreciable numbers.

A number of sputa were stained for the presence of cytoplasmic inclusions, with negative results.

Attempts to transmit infection to animals, with inoculations of Seitz filtered sputum, were unsuccessful.

#### Sputum in Patients with Classical Lobar Pneumonia.

During the period under review, three Europeans were admitted with pneumonia. Clinically all three were of typical lobar type with sudden onset and rigor etc.

In two of these, the sputum was rusty, the stained film was packed with gram positive cocci and culture yielded a profuse growth of pneumococci. In the third, who had blood-stained spit, pneumococci predominated but were not in profusion.

During later months, several Africans developed this type of pneumonia with classical onset and rusty sputum; here again the pneumococcus predominated. Typing sera were not available at this time.

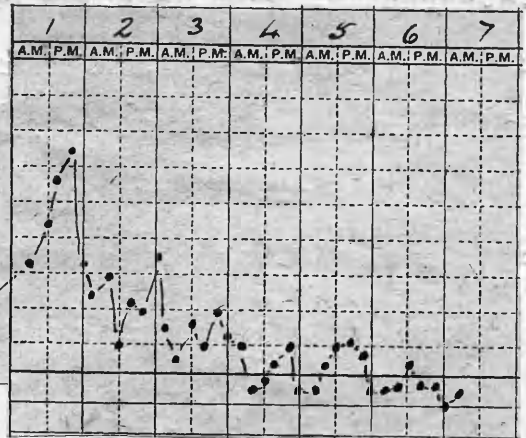
Both macroscopically and bacteriologically then, these sputa differed strikingly from those described above as typical of the epidemic.



Fig. 2.

Naso-pharyngitis.  
Lungs clear.  
No malaria parasites.

Days



Days

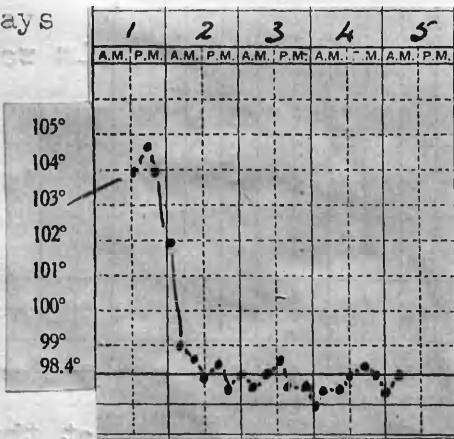


Fig. 3.

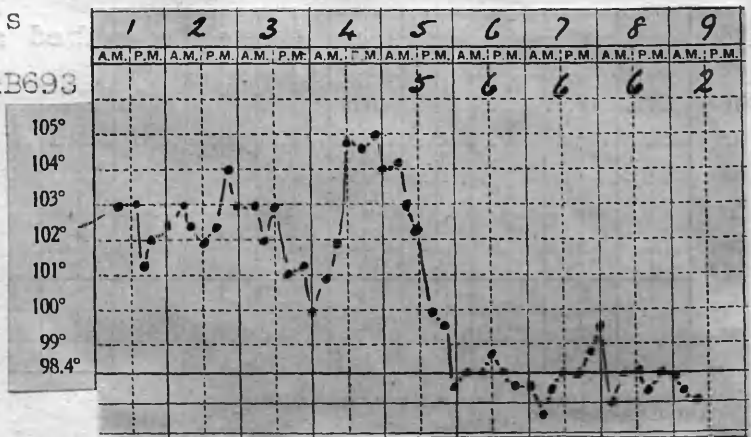
Pharyngitis. No malaria parasites. Spontaneous drop in temperature.

Days

Fig. 4.

Gms. M&B693

Pharyngitis followed by pneumonia both lower lobes. Response to sulphapyridine.



### Temperature Charts.

Pulse and respiratory rates consistently paralleled the temperature. Respiration frequency was more directly related to pyrexia than to extent of pulmonary involvement and was well marked even at the stage of upper respiratory involvement.

MacNaught and Murray-Lyon (1944) pointed out that little importance can be attached to an increased respiration rate, as this is common to all febrile conditions in Africans.

On admission with pharyngitis, temperature was found to be anything from  $100^{\circ}\text{F}$  -  $104^{\circ}\text{F}$ . If lung involvement did not occur, it usually settled to normal about the second to fourth day (Fig. 2), or occasionally more sharply on the day after admission (Fig. 3). The latter invariably occurred when appropriate sulphonamides were given.

With onset of pneumonia, temperature either remained level or rose a degree or two. (Fig. 4.). If already settled to normal, then a secondary rise occurred but this picture was rather unusual (Fig. 5). A temperature of  $106^{\circ}\text{F}$  was occasionally seen with right upper lobe involvement (Fig. 6).

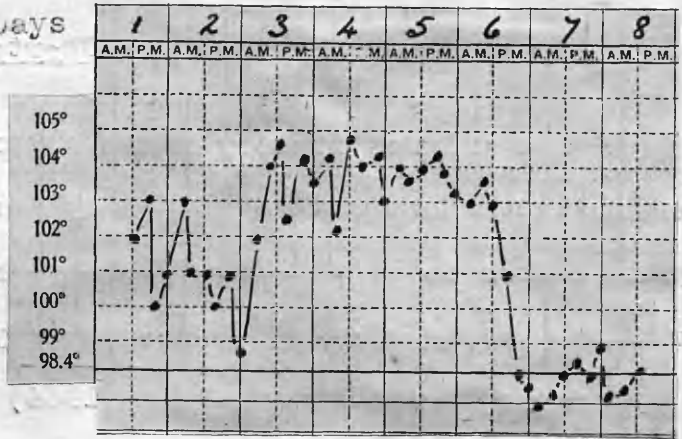
In 58 mild or moderate pneumonias, where chemotherapy was not exhibited, pyrexia diminished by rapid lysis about the fourth to sixth day (Figs. 7 and 8), sometimes even sooner (Fig. 9). Termination by true crisis was unusual (Fig. 5).

In uncomplicated cases, the sulphonamides aborted pyrexia in 24 to 48 hours, temperature either falling sharply or as a rapid lysis (Figs. 4, 10, 11 and 12).

Fig. 5.

Pharyngitis with development of pneumonia left lower lobe on 3rd day. No sulphonamide given.

Days



Days  
Gms. M&B 593

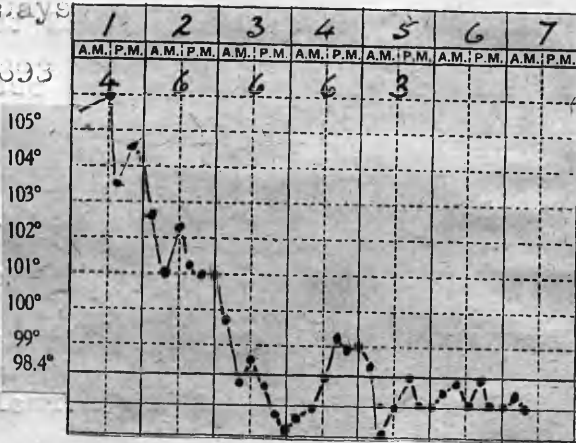


Fig. 6.

Right upper pneumonia.  
W.B.C. 32,000 per cmm.  
Jaundice.

Fig. 7.

Pneumonia left lower lobe.  
Spontaneous resolution.

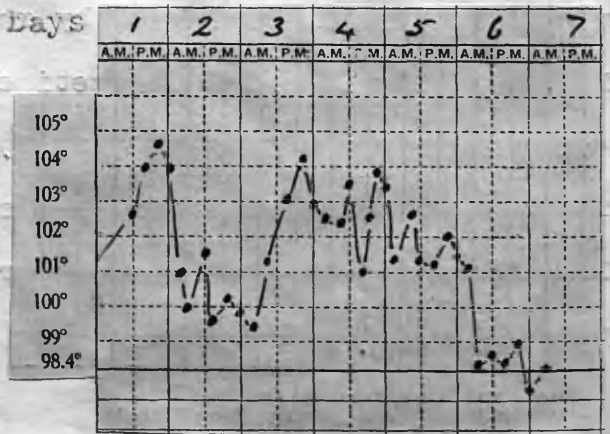
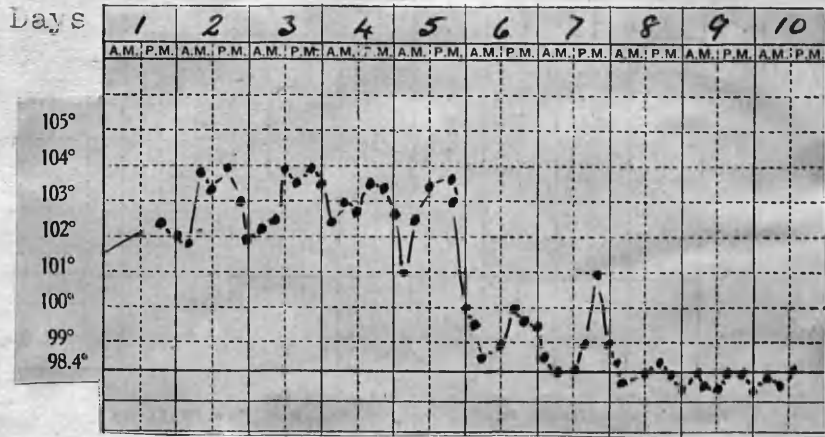


Fig. 8.

Admitted with 8 days' chest pain. Naso-pharyngitis and pneumonia left lower lobe. No sulphonamide given.



Days

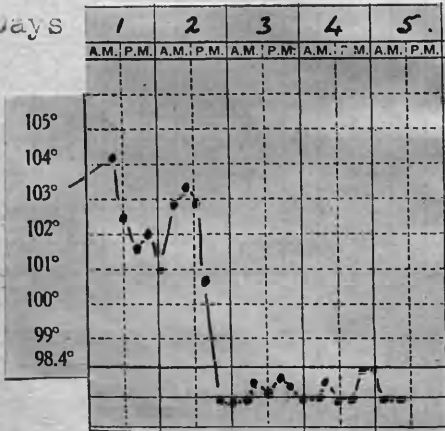


Fig. 9.

"Focal pneumonitis".  
A patch of infiltration in right middle lobe.

Fig. 10.

Pneumonia lingular segment of left upper lobe.

Days  
Gms. M&B693.

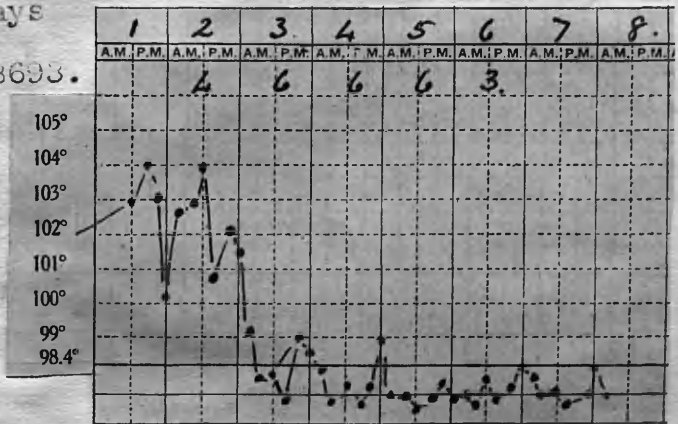


Fig. 11.

Naso-pharyngitis, sinusitis  
and pneumonia right lower lobe.

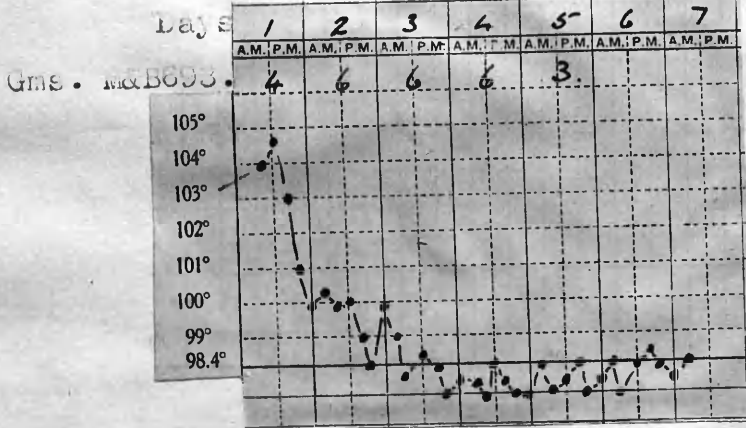
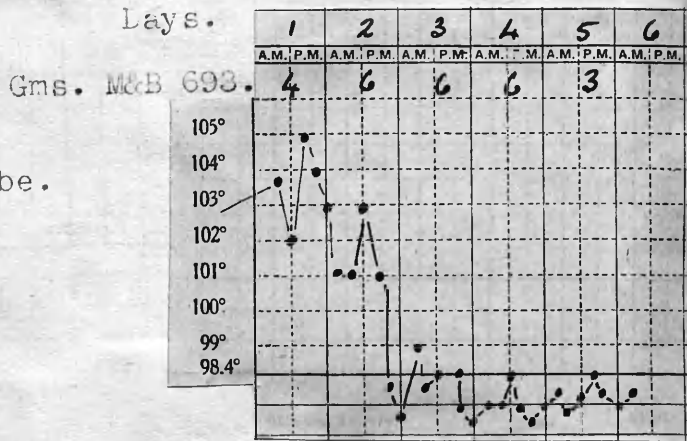
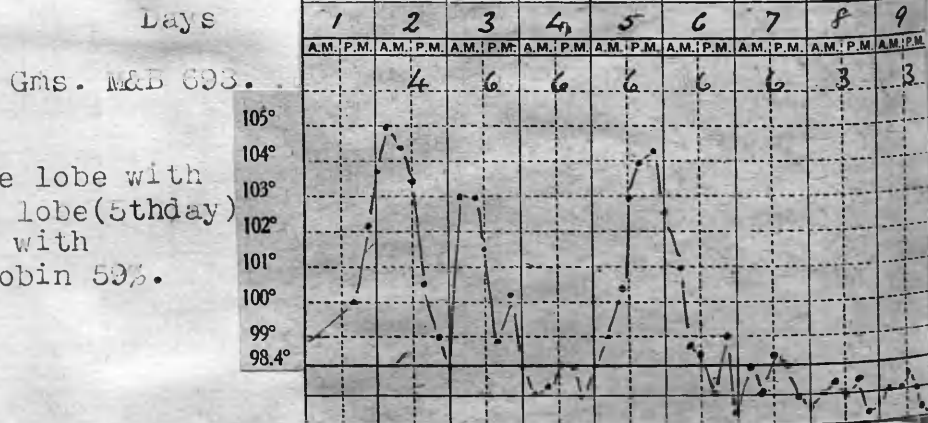


Fig. 12.

Admitted with chest pain, cough  
and headache 48 hours. Intense  
pharyngitis and pneumonic  
patches in both lower lobes.  
Satisfactory response.

Fig. 13.

Pneumonia right middle lobe with  
spread to right lower lobe (5th day)  
while under treatment with  
sulphonamide. Haemoglobin 59%.



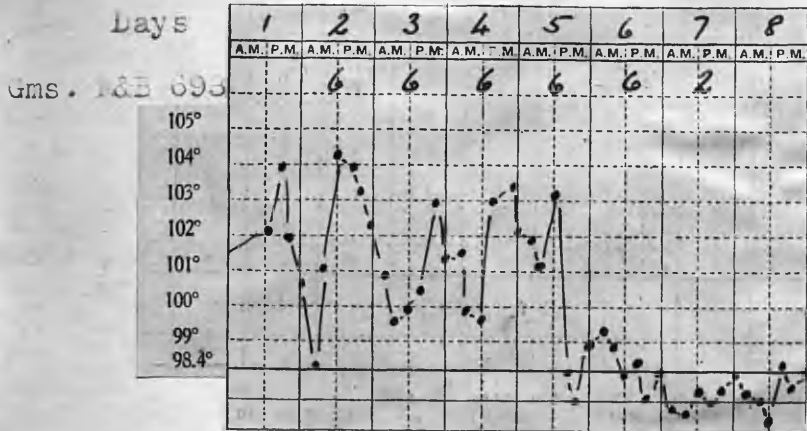


Fig. 14.

Extension of pneumonia in an anaemic patient on sulphonamide treatment. Haemoglobin 60%.  
 1st day: pharyngitis.  
 2nd day: pneumonia right middle lobe.  
 4th day: middle lobe almost clear; patch of consolidation left lower lobe.

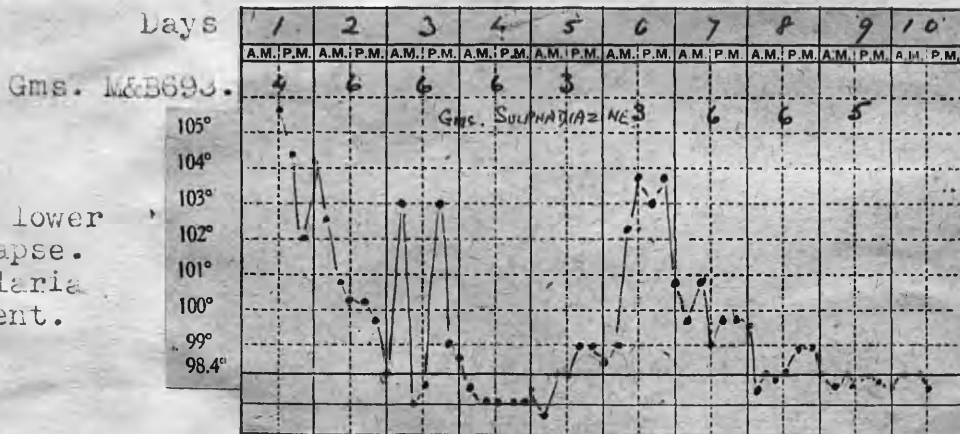


Fig. 15.

pneumonia left lower lobe with collapse. hb. 60%. No malaria parasites present.

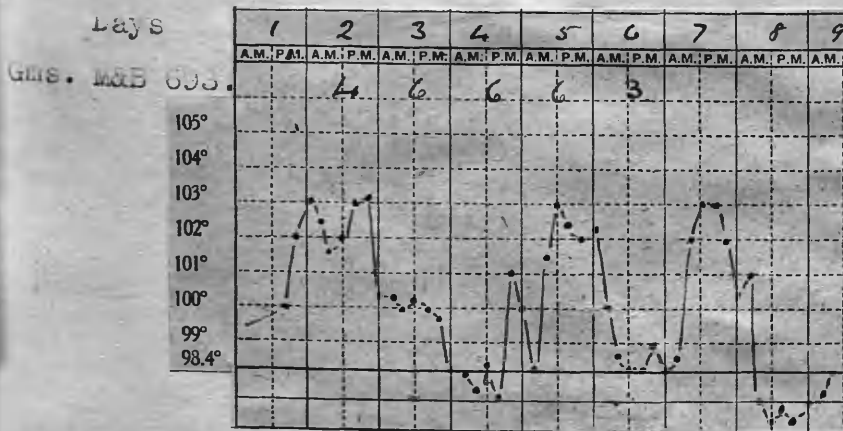


Fig. 16.

Pneumonia right middle lobe with collapse.

Days

tabs. Mepacrine.

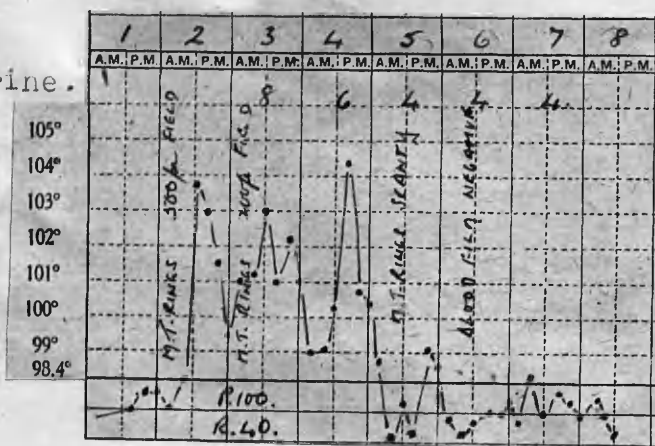


Fig. 17.

Severe M.T. malaria in an African, treated with Mepacrine.

Fig. 18.

Mild M.T. malaria in an African. resolution without specific treatment.

Days

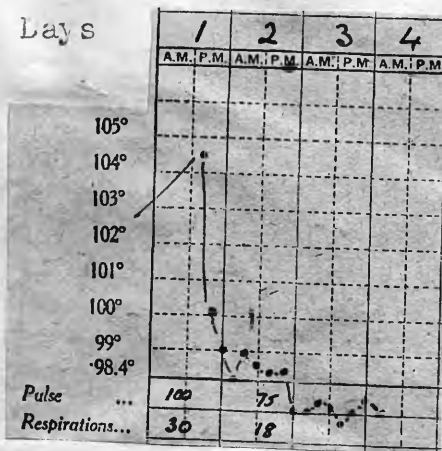


Fig. 19.

Malaria and pharyngitis. W.B.C. 14,000 per cmm. M.T. rings present.

Days

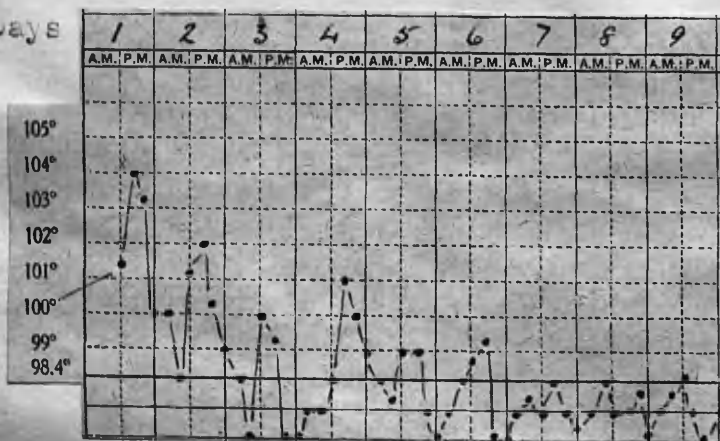
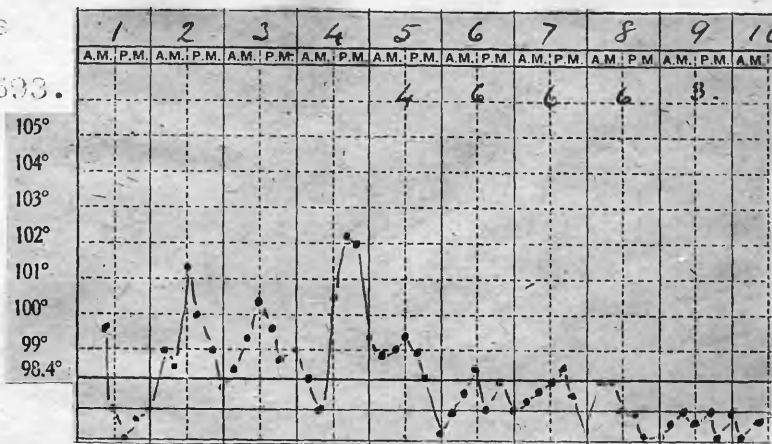


Fig. 20.

Gms. M&B 693.

Malaria and pharyngitis.  
 W.B.C. 18,000 per cmm.  
 M.F. rings present.  
 4th day: developed pneumonia  
 pectoral segment right  
 upper lobe.



Days

Gms. M&B 693

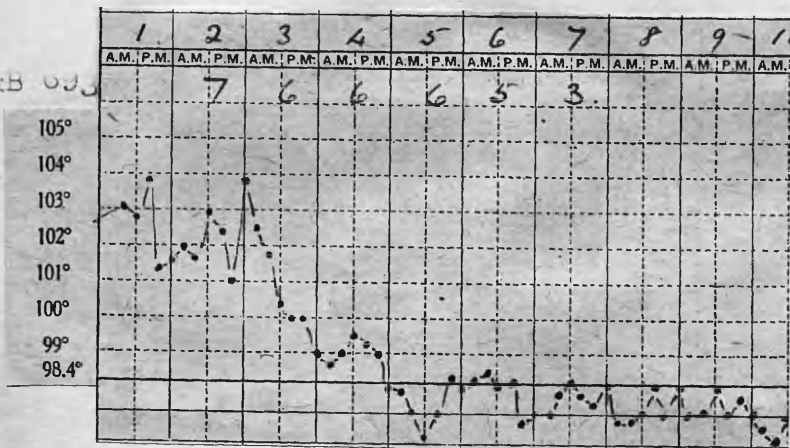


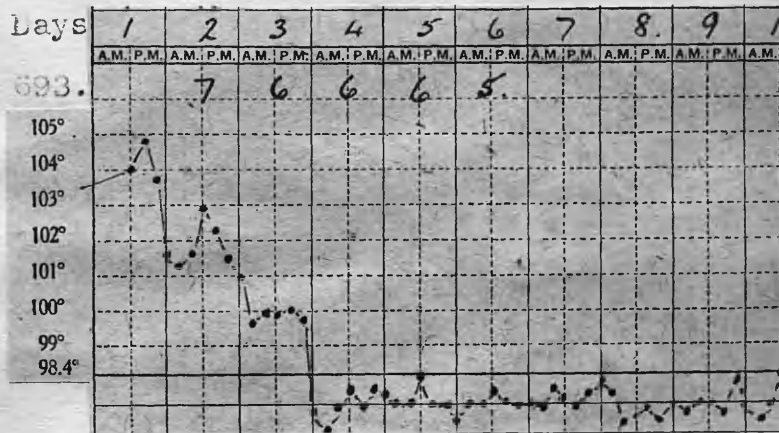
Fig. 21.

Pneumococcal pneumonia  
 right lower lobe in a  
 European.

Fig. 22.

Gms. M&B 693.

Pneumococcal left  
 lower lobe pneumonia  
 in a European.





Days  
Gms. M&B 693.

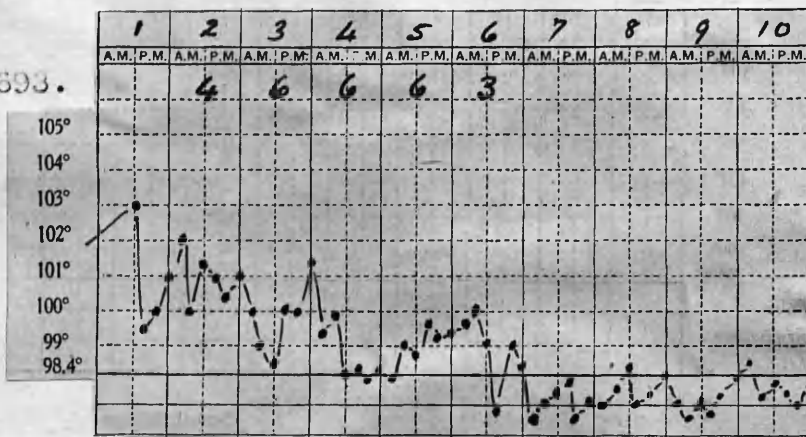


Fig. 23.

Lobar pneumonia left lower lobe and lingula  
in an African.

W.B.C. 22,000 per cmm.

Pneumococci in sputum and lung puncture.

Blood culture negative.

Figures 13 and 14 illustrate the course in several anaemic patients where spread occurred to another lobe while under treatment with sulphonamides.

Associated collapse of the pneumonic area was another not infrequent cause for failure to react to drug therapy. Two such cases are illustrated in Figures 15 and 16.

Malaria was not a frequent complication and when it did occur there was no evidence that it affected the severity of the pneumonia or its response to treatment. It always entered into the differential diagnosis of the naso-pharyngitis cases and was excluded by blood film examinations and white cell counts. Figures 17, 18, 19 and 20 show temperature charts of simple M.T. Malaria, moderately severe M.T. Malaria treated with mepacrine, malaria complicating pharyngitis and malaria complicating pneumonia.

Figures 21, 22 and 23 illustrate charts of classical pneumococcal lobar pneumonia in Europeans and in an African.

DIFFERENTIAL DIAGNOSIS.

It has already been pointed out that an increased respiration rate is common to all acute febrile conditions in Africans and one hesitates to diagnose pneumonia with certainty until there are evident physical signs of such present. From what has already been said, it will be appreciated that upper respiratory infection presents the commonest difficulty. Malaria may be difficult at the height of a paroxysm; herpes may be present; sudden drop in temperature, more rapid improvement in general well being and results of blood film examination and leucocyte count usually make the diagnosis clear.

The most difficult disease to distinguish from pneumonia is undoubtedly amoebic hepatitis. High temperature, dyspnoea, pain in the right chest, tender liver and leucocytosis associated perhaps with impairment of percussion note and breath sounds at the right base all suggest pneumonia of right lower or middle lobe; often it is failure to respond to sulphonamide which first gives a clue to the diagnosis; response to emetine in these cases is dramatic.

Radiographic Studies.

In all, 584 fluoroscope examinations were conducted, usually in slight or moderate cases soon after admission, in more severe pneumonias after several days treatment and in patients during convalescence.

Complete co-operation and harmony between radiologist and physician made these "screen sessions" most enjoyable and instructive. The former took the chair and the latter was invariably present with clinical information and for dispute and discussion.

The principal objects of the examinations were:-

1. to confirm the accuracy of clinical findings especially with regard to segmental localisation and to frequency of associated lobular collapse.
2. to elucidate the variety and extent of the pneumonia.
3. to exclude the presence of latent pneumonia in those patients who had pharyngitis, without physical signs of consolidation yet associated with well marked polymorph leucocytosis.
4. to seek a cause for delayed resolution and apparent lack of response to treatment.
5. to confirm complete resolution.

The most satisfactory deduction from the clinician's point of view was that, in the majority, physical examination alone could accurately mark out the distribution and extent of the involvement. There were two principal exceptions to this rule: in the very early stages of segmental consolidation, the only abnormal physical sign might be diminution of breath sounds over the small area and this



(a)



(b)

Plate 1. Consolidation and collapse left lower lobe. (Lateral view.)

(b) shows same outlined by pneumothorax.



(a)



(b)

Plate 2. (a) Consolidation basal segments of right lower lobe.

(b) Consolidation right lower lobe with effusion in main interlobar fissure.

was exceedingly easy to miss on clinical examination; radiographic examination was able to detect an interlobar pleural thickening or small effusion.

These studies confirmed the broncho-pulmonary segment as the unit of pneumonic consolidation, involvement of one or more such segments being commoner than that of a whole lobe. X-Ray appearances of such individual segment consolidations are described in detail in modern text books of radiology (e.g. "A Textbook of X-Ray Diagnosis by British Authors"). Further illustrations and observations on this subject will be found in the later chapter on broncho-pulmonary anatomy.

With lower lobe involvement, especially the left, fluoroscopy was found of less value than clinical examination as the heart and mediastinum would often prevent accurate delineation, even in lateral view. A good lateral film, however, usually showed this lobe up satisfactorily, especially when collapsed. (Plates 1 & 2)

One frequently hears it stated that X-Rays, in pneumonia as well as tuberculosis, invariably shows up lung disease to be more extensive than physical signs would lead one to expect. This has not been the rule with these cases. However, such a belief might arise if, <sup>only</sup> straight postero-anterior films were taken instead of views in more than one plane. With consolidation of the middle lobe, for instance, in P.A. film this would seem to involve almost half the right lung. A lateral film shows the lobe outlined in detail (Plate 4). It is becoming increasingly apparent, at least in non-tuberculous lung disease, that a good lateral film is essential for complete investigation.



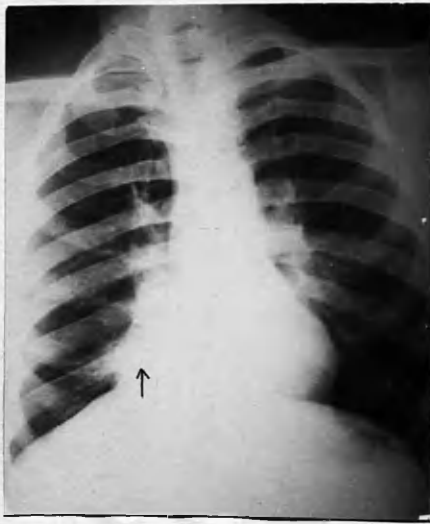
Plate 3. Pneumonia lingular segment of left upper lobe.



(a) Antero-posterior. (b) Right lateral view.

Plate 4.

Right middle lobe consolidation.



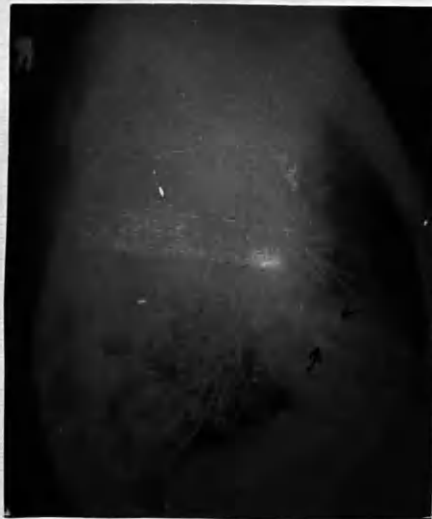
(a)



(b)



(c)



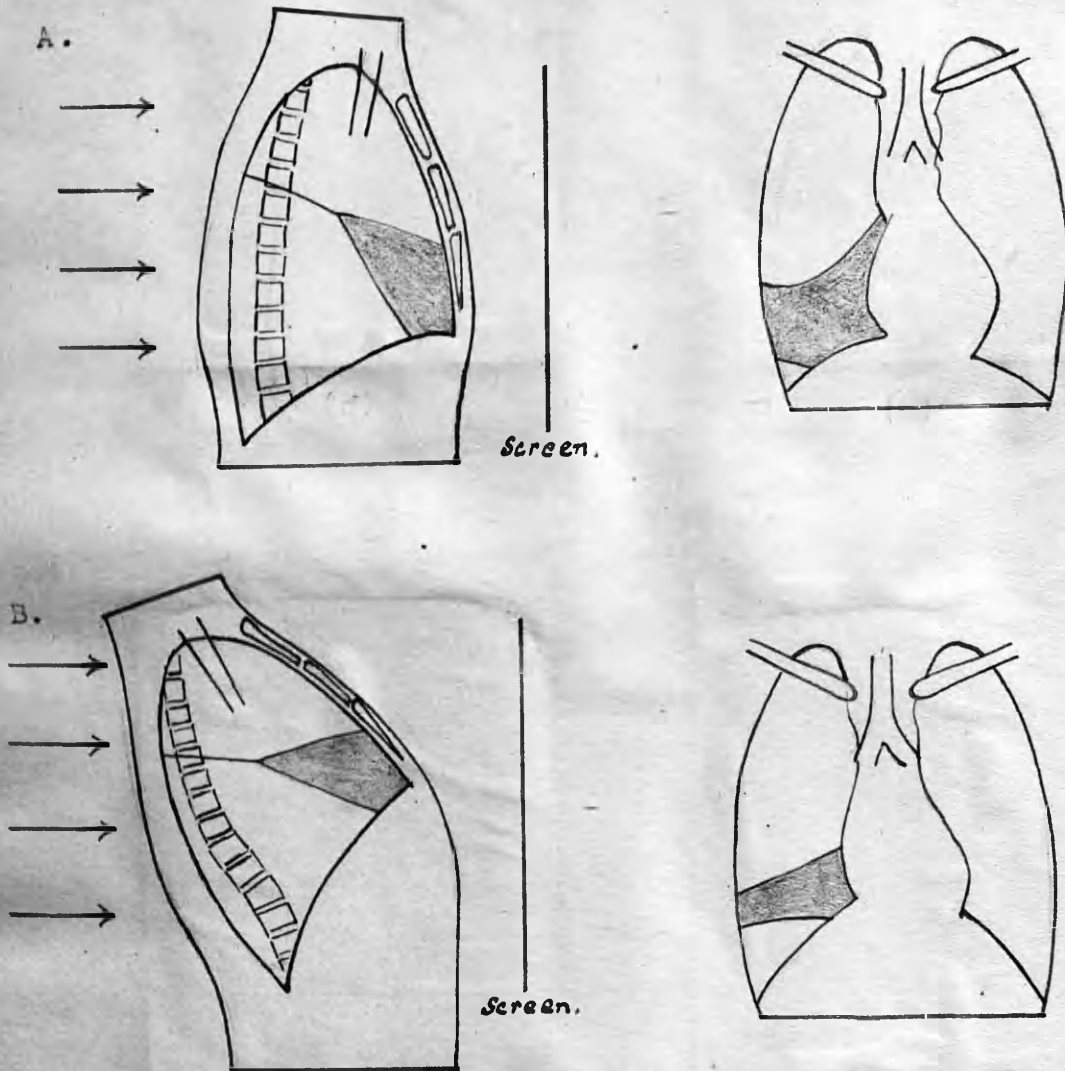
(d)

Plate 5.

- (a) Collapse antero- medial segment right middle lobe.  
(b) Right lateral bronchogram of same: arrow points to blocked bronchus.  
(c) Expansion of segment a few days later with disappearance of shadow.  
(d) Lateral bronchogram now shows right middle lobe bronchi well filled.



DIAGRAM 1. Fluoroscopy of Right Middle Lobe.  
Position of Extreme Lordosis.



A. Normal upright Fluoroscopic position. The middle lobe is seen as a shadow occupying the lower third of the right chest.

B. Position of Extreme Lordosis. The middle lobe is now seen as a narrower, clear-cut shadow separated from the right hemi-diaphragm by a definite space.

In studying this area of the right middle lobe by fluoroscopy, it is helpful to make use of the position of extreme lordosis - "Kreuzhohlstellung". An area of density in the lower third of the right lung field may be due to involvement of middle lobe, basic segments of lower lobe, pleuro-pericardial disease, thickened interlobar pleura or interlobar effusion. With a lateral film, it will often be seen to be confined to the middle lobe and, on screening, this is most easily demonstrated by the lordotic view. It will be seen (Diagram I) that the right middle lobe takes on a characteristic narrow shape with a clear space between it and the diaphragm (King 1939)

It was interesting to note signs of partial collapse in the early cases of pneumonia e.g. a patch of compensatory emphysema (Plate 11) slight crowding of ribs (Plate 6), a small amount of cardiac retraction (Plate 11), a deflection or convexity of an interlobar septum or a slight raised and hesitant diaphragm.

This patch of compensatory emphysema, although related to atelectasis, should not be confused with localised emphysema caused by a check valve bronchial obstruction (Maxwell 1940). In the former, the neighbouring parenchyma is emphysematous; in the latter, the parenchyma of the affected lobe or segment is affected.

The diminution of lung volume, for which the emphysema endeavours to compensate, has been a matter of fairly common observation in pneumonic conditions and was noted by Davies, Hodgson and Whitby (1929) in a review of 119 cases of pneumococcal



(a)

(b)

Plate 6.

(a) Consolidation and collapse lingula of left upper lobe.

Note also the crowding of ribs as evidence of early collapse of right upper lobe.

(b) Resolution and expansion 7 days later.

pneumonia. Terrell, Robertson and Coggelshall (1933), demonstrated it clearly in experimental work on dogs. By many, however, it has been thought to occur only during the resolving stage when absorption of inflammatory exudate would leave the alveolar walls more closely approximated.

In the pneumonias we are now considering, it would seem clear that an element of collapse may be present from the first and the writer thinks this was also the case in pneumonias seen in British soldiers at home. He subscribes to the view, <sup>held</sup> by Coryllos that this early atelectasis follows the blockage of bronchi or bronchioles by plugs of mucus. It will be remembered that Coryllos went so far as to advocate bronchoscopic suction in early pneumonia when there was evidence of associated collapse.

It should be noted that the diminution of lung volume seen in these cases is not the picture of acute massive collapse. In the latter, a large bronchus is obstructed and the radiological appearance is unmistakable. The moderate or slight degree of collapse is most probably due to a mere peripheral block e.g. by exudate in the respiratory bronchioles and alveolar ducts, coincidental with or even preceding the outpouring of exudate into the alveoli. Rapid absorption of the small quantities of the fluid remaining in the alveoli at this stage would lead to a uniform atelectasis, radiologically indistinguishable from the true consolidation.

The evidence of collapse was most commonly noted in affection of the left lower lobe, which is in keeping with the well known fact that the chronic pulmonary disease, especially bronchiectasis



Plate 7.

Consolidation dorsal segment right lower lobe.

(usually the result of unresolved atelectasis) has this area as its favourite site. In a follow up study of 400 cases of bronchiectasis at Massachusetts General Hospital, Perry and King (1940) noted involvement of the left lower lobe in 44%. Of 166 lobectomies performed for bronchiectasis, Tudor Edwards (1939) reported that 92 (55.4%) involved the left lower lobe. During 1940 - 1941, the writer investigated 24 cases of bronchiectasis in British troops; 15 (62.5%) of these affected this lobe.

### Central Pneumonia.

A study of the segmental anatomy of the lungs (see later section) shows that there is only one broncho-pulmonary segment - the cardiac lobule - which has not a surface adjacent to the chest wall (Diagram IV). Consolidation of this segment might accurately be termed a central pneumonia. No such involvement, however, was recognised in the present series. The term has too frequently been used by the clinician to describe a patient in whose chest he cannot locate physical signs of consolidation, who looks the picture of pneumonia and does indeed, at a later date, show typical signs of pneumonic consolidation. Experience with such cases has shown that more careful examination will often demonstrate an area, albeit minute, of diminished air entry and that radiological examination in more than one plane will show up the patch to be peripheral. The P.A. film will often demonstrate a "central pneumonia" or "hilar" consolidation when a lateral view shows it to be, in fact, an involvement of the dorsal or apical segment of the lower lobe which has been superimposed on the hilar shadow. (Plate 7).



Plate 8.

Large effusion (empyema) in right horizontal interlobar fissure.



Plate 9.

Small effusion in left interlobar fissure. This resembles a consolidation of anterior basilar segment of left lower lobe.

These observations lead the writer to agree with Ude, that "central pneumonia" is in most cases a misnomer.

Fluoroscopy was of special value in demonstrating interlobar pleurisy and small collections of fluid. A P.A. film may not differentiate with certainty an interlobar effusion from segmental consolidation in an adjoining area. An effusion in the upper interlobar fissure may simulate consolidation of the lower part of the upper lobe while a collection in the main fissure may be difficult to distinguish from middle or lower lobe consolidation. The lateral view clearly differentiates these conditions. (Plates 4,5,8 and 9) Localized pleural fluid also, can be accurately localised by views in different planes.

A radiographic study of these African chests confirms one's impression that they have had very little in the way of previous infection and dust irritation with which to contend. They have virgin lungs like those of an infant, with clear parenchyma and absence of those hilar shadows and pronounced vascular markings which we <sup>are</sup> accustomed to consider normal in Europeans. Recently the writer mistook a radiograph of an African soldier for that of a European and later found that the patient had previously been a regular in the French Army and had spent ten years of his service in the industrial towns of France!



P A T H O L O G Y.

Four deaths occurred in the series, a mortality of 0.55%.

All, however, were admitted during the first three months of 1944 and they represent a death rate of 0.82% for cases seen during that time.

Case I was a man aged 24 years, with two weeks service, who died after 10 days in hospital. Severe continuous hiccough had lasted 5 days.

At Post Mortem, the lungs showed grey hepatisation of both lower lobes. Several small suppurative lesions were seen at the base of the right lower lobe, adjacent to the diaphragmatic pleura. Trachea and bronchi showed fairly intense congestion of mucous membranes.

Microscopic Findings. Some of the alveoli were nearly empty, others showed a coarse fibrinous reticulum. There were tiny haemorrhagic areas, infiltrated with polymorphs. The alveolar walls were thickened with congested and distended capillaries. A section from the base of the right lower lobe showed an abscess cavity about 3 mm in diameter. The picture was that of a resolving lobar pneumonia with abscess formation.

Case II. A Private aged 55 years, with one week's service, was admitted with pneumonia of both lower lobes. He was seriously ill from admission and did not respond to full courses of sulphapyridine (39 grams) and sulphathiazole (30 grams). The sputum did not yield pneumococci. R.B.C. 2,700,000 per cmm. Hb 48%, W.B.C. 30,000 per ccm. He died 17 days after admission. (4th day)

Post Mortem Findings: The left lower lobe showed red hepatisation, the right upper and lower lobes resolving grey hepatisation. The right middle lobe was aerated but appeared to be in the first stage of resolution of a similar process. A thick greenish gelatinous material covered, and was adherent to, the right pleural surfaces. It was not fluid or purulent and seemed most like an old-standing tuberculous pleurisy. Tracheal and bronchial mucosae were only slightly inflamed.

Microscopic Findings: In parts, the alveoli were almost empty, in other places, they still contained fibrin and polymorphs. The alveolar walls were thickened. Vessels were not congested and there were few red cells to be seen. In one part, there was a cavity bordered by dense masses of polymorphs. Resolving lobar pneumonia with minute abscess formation.

Case III. This soldier, aged 17 years, had 7 days service and was admitted with a note from unit medical officer "Temperature 99.4° F. Collapsed on parade. Pharyngitis. Chest normal". Temperature was

102.5°F on admission. He walked from the ambulance and was only slightly distressed and dyspnoeic. Marked naso-pharyngitis. Signs of pneumonic consolidation right upper lobe posteriorly. He died 7 hours after admission.

At Post Mortem, there was consolidation of the posterior segment of right upper lobe and a smaller patch in the right lower lobe. A layer of soft mucoid material was present over the surface of the right lung. The tracheal mucosa was only slightly inflamed.

Microscopic Findings: The alveoli were packed with red blood cells and polymorphs. In parts, the cellular congestion was so great that alveolar walls were difficult to distinguish and in other places, there were tiny collections of polymorphs suggesting early foci of suppuration. Conclusion: Severe early lobar pneumonia.

Case IV. A Private aged 25 years with 4 weeks' service, was admitted with generalised bronchitis. Temperature 103°F. Treated with course of sulphapyridine. Two days later there was consolidation left lower lobe and next day, pneumonia right upper and middle lobes. 5th day. White cell count 15,000 per cmm. Sulphapyridine discontinued at 23 grams. Sulphadiazine course started. 6th. day. Little change. Signs of fluid left base. 7th. day. Improved in morning with normal temperature. In afternoon, vomited and died suddenly.

Post Mortem Findings: Straw coloured fluid was present in left pleural cavity. The pleura was thick, yellowish and adherent like an old empyema. There was consolidation of left lower lobe and grey hepatisation of right upper and middle lobes.

To summarise.

1. The four deaths occurred in recruits with less than a month's service.
2. All showed pathological lesions consistent with a diagnosis of lobar pneumonia.
3. Three showed evidence of old pleural disease.
4. Suppurative changes in the lung were present in three cases.
5. One patient died as a result of continuous hiccough lasting five days and probably related to suppurative lesions in the lung adjacent to the left hemi-diaphragm.

Another death was associated with well marked anaemia.

The third patient presumably died from a severe, toxic right upper lobe pneumonia.

The fourth had sudden cardiac failure on the seventh day of extensive bilateral pneumonia which did not respond to sulphonamides.

## COMPLICATIONS.

Serious complications were remarkably infrequent, 611 (83%) of the pneumonia patients were discharged from hospital by the 14th day after admission. Only five patients were discharged from the service on medical grounds with sequelae of this pneumonia. Three of these had healed empyema, one had fibrosis and collapse following resolution of an abscess and empyema and the other had unresolved consolidation with a severe and resistant anaemia.

EMPYEMA. Five cases occurred, two during the epidemic and three in later months. Pneumococci were isolated from four of these and the fifth was sterile after large doses of sulphonamide. Three recovered after open drainage. One was associated with an abscess of left lower lobe and both conditions cleared with repeated aspiration and postural measures, leaving some residual fibrosis and collapse. The other was loculated and cleared with similar conservative measures.

ABSCCESS. One abscess of right upper lobe was seen during the epidemic period. Its relation to the prevalent pneumonia was doubted. In another patient, the sudden expectoration of three ounces of pus suggested evacuation of an abscess, but there was no evidence on X-Ray examination immediately after. In yet another case, there was evidence of cavitation on X-Ray of a right upper lobe pneumonia, which resolved satisfactorily with routine treatment.

PLEURAL FLUID. A small sterile pleural effusion, showing a preponderance of polymorph leucocytes, seemed to be a not infrequent accompaniment. It was found on X-Ray as a faint haze in the

costo-phrenic sinuses or by exploratory aspiration of the pleural cavity while anaesthetising the chest wall for lung puncture.

INTERLOBAR EFFUSIONS. These were usually small collections of fluid, found on a routine fluoroscopic examination, in the interlobar fissure adjacent to a consolidated segment. In nearly every case, this resolved rapidly, did not appreciably lengthen the man's stay in hospital but sometimes accounted for a lack of immediate response to treatment. Thickening of the interlobar septum was a still more common finding. The incidence of pleural involvement, recognisable on X-ray examination as thickening or actual fluid collection was in the region of 12%. One case of pneumococcal interlobar empyema was seen and this cleared with repeated aspiration.

COLLAPSE. Clinical and radiological evidence of early collapse has already been referred to as a common feature of these pneumonias. In most cases, the area of the lung rapidly expanded but in a few, persistence of the collapse accompanied failure of response to sulphonamide and delay in resolution. This was commonest in left lower lobe affection.

It was treated with large doses of emetic cough mixture combined with postural measures. The patient was instructed to cough into a receptacle on the floor at the opposite side of his bed from the lung affected. This was designed to produce frequent movement rather than true postural drainage, for which such "intermittent tilting is quite inadequate."

DELAYED RESOLUTION. The principal causes for failure to respond to chemotherapy and for delay in resolution were:-

(1) Associated collapse. (2) A small pleural effusion. (3) Anaemia  
 (4) Underlying chronic lung disease such as bronchiectasis or a  
 tuberculous focus.

MALARIA: This disease hardly need be considered in the study of  
 pneumonia in these adult natives, although the hospital was  
 situated in a hyperendemic malarial zone. Scanty M.T. malaria  
 parasites were present in blood films of a few patients but the  
 condition had no deleterious effect on the course of the pneumonia  
 and antimalarial drugs were never prescribed.

Simpson (1944) and MacNaught and Murray-Lyon (1943) describing  
 pneumonia in West African troops, also formed this conclusion.  
 These writers, finding only six positive blood films in 375 patients  
 with respiratory infections, compared the picture with that of  
 Europeans, in whom any acute infection seems to allow latent  
 malaria to manifest itself and call for specific drug therapy.

PNEUMOCOCCAL MENINGITIS. Three fatal cases of pneumococcal  
 meningitis arose during the epidemic period. Not one of these,  
 however, was associated with pneumonic consolidation, so their  
 inclusion in a list of complications is hardly warranted.

JAUNDICE. The high incidence of this complication (6.5%) is  
 probably the most striking feature of these pneumonias. It is, of  
 course, quite out of the usual British experience at home.

Jaundice occurring with pneumonia is usually considered to  
 be of toxic variety and there is evidence that this was the case  
 in the present series.

Upper lobe pneumonia, especially right-sided has long been

recognised as a specially severe form of the disease with very high temperatures, delirium etc., and this was our experience. Right upper lobe consolidation had an extraordinarily high incidence of jaundice (21.5%): this was not the case with the left upper lobe variety.

In the writer's experience, the degree of enlargement and tenderness of the liver (which was an almost constant finding in the acute stage) was a sound indication as to the severity of the pneumonia. It was always well marked in the icteric cases. Jaundice was never seen in these patients with upper respiratory inflammation alone.

Findlay (1944) records the incidence of jaundice as 59 (4.8%) in a series of 1225 pneumonias occurring in West African Troops and discusses the probable aetiology. 117 (9.9%) showed the sickle cell trait but only 11 of these were jaundiced. He concludes that sickling is not the primary cause of jaundice but may be a factor in certain cases. He excludes infective hepatitis, cirrhosis and primary carcinoma as likely causes and points out that there is no evidence of haemolysis in these pneumonia patients.

Assuming then that jaundice is due to the action of bacterial toxins in the liver, one must explain why it is so much commoner in Africans than Europeans. One probable reason, already noted, is the high incidence of upper lobe infections. This has also been reported by MacNaught and Murray-Lyon (1944) in West African Troops and by Raper (1944) in East Africa.

Findlay points out that pneumonia among recruits is always characterised by a higher prevalence of jaundice than in seasoned

troops. He postulates that the increased liability of the African recruit to jaundice is correlated with the deficient diet on which he has lived before joining the service. It will be remembered that 61% of our patients had less than four weeks' service and a large proportion showed evidence of malnutrition and avitaminosis.

The importance of protein in normal liver metabolism is a matter of fairly recent recognition. Marshall (1944) reminds us that the incidence of jaundice is inclined to rise in times of economic stress, with consequent dietary insufficiency; he adds, very rightly, that in places such as West Africa, where the staple diet is low in protein, the incidence of all types of liver disease is very high. Certainly, in Gold Coast, it is common to find some degree of hepatic cirrhosis, usually microscopic, at routine autopsies.

### T R E A T M E N T.

The drug of choice was sulphapyridine and this was given to 629 (86%) of the pneumonia patients. The drug was started on the day of admission in 482 of these.

Reasons for preference were:-

1. the therapeutic effect equalled that of the newer sulphonamides.
2. it was in more plentiful supply.
3. toxic effects or even unpleasant symptoms were most unusual.

The standard course given was 25 grams by mouth, starting with 2 grams and followed by 1 gram four-hourly. In very toxic patients, an initial intravenous dose of 1 gram might be given or

2 grams orally to be continued for three or four doses. One had no hesitation in prolonging the course to 35 grams or even 40 grams in severe or resistant cases.

In a very few, whose temperature showed no response after three or four days treatment, the drug was changed to sulphthiazole or sulphadiazine with seemingly good effect.

A smaller total dosage, usually 20 grams, was administered in very mild pulmonary cases or in those patients (50 were so treated) who showed only respiratory signs. None of the latter developed lung involvement, evidence that the drug had an effect in aborting pneumonia. In pharyngitis cases temperature invariably settled within a few hours of commencing the drug. With tonsillitis, sinus and ear complications however, it appeared to have much less effect.

Sulphadiazine, in similar dosage by mouth, was given to 46 pneumonia patients and sulphathiazole to a smaller number.

One did not note any difference in therapeutic effect as compared with sulphapyridine.

In the treatment of gonorrhoea, it is claimed that intramuscular injection of a suspension of 1 gram sulphapyridine given on four successive days, has an effect comparable to that of an oral course of 25 grams of the drug. This method of treatment was tried out in six patients with pneumonia. Apart from adding considerably to the discomfort of the patient, it had no effect whatsoever, either on the temperature or on the course of the disease.

Ancillary methods of treatment were simple. Careful nursing with a back rest during the short acute stage, copious fluids and



alkalis and nocturnal sedative during the first 48 hours, seemed all that were necessary. Fresh air was ensured in open-ended and open-windowed wards. Oxygen was used only in acutely distressed patients. These measures ~~alone~~ were used in 58 mild and moderate cases of pneumonia who had no sulphonamide. Pyrexia subsided rather slowly two or three days after one would have expected with chemotherapy but in the majority of these, the length of time before final resolution was little different from similar cases treated with sulphonamide.

The patients were not confined to bed longer than they wished and in the majority of cases, they were getting up to the latrine the day after admission.

For a short time at the height of the epidemic, hospital beds were in such demand, that one was obliged to return the uncomplicated pneumonia patient to his unit on completion of the sulphonamide course, on the fifth or sixth day. Such men were excused duty for several days. 186 patients were thus discharged from hospital on or before the seventh day of admission and all these had resolved, or almost so, in this short time. Not one returned with an immediate relapse. Eight were re-admitted within five or six weeks with what was apparently a fresh pneumonia infection.

It will be understood then, that this rather unusual form of epidemic pneumonia was of relatively mild type. In later months, when a proportion at least of the cases were of typical pneumococcal lobar variety, the average period before complete resolution and the duration of stay in hospital were considerably longer (Table IV).

The African tolerates sulphapyridine extremely well; unpleasant symptoms or toxic signs are practically never found.

No cases of agranulocytosis, urinary suppression, haematuria or apparently related serous pleural effusions were encountered and there was certainly no evidence that the drug retarded resolution.

What was apparently a haemolytic anaemia occurred after 65 gramp sulphapyridine (given in two courses) in a lad aged 19 years. His nutritional state was below par and obvious signs of avitaminosis were present. Sickling tests were negative. Haemoglobin reached 25% level. He reacted well, however, to blood transfusion, liver and iron, and his pneumonia, which had been resistant to treatment, slowly resolved.

The African soldier is a model patient, without a shade of anxiety as we know it in many European patients. The very few exceptions one can remember, have occurred with nursing orderly patients or other educated men. If he suffers any unpleasant effects from sulphonamide, one rarely hears him voice any complaint. As Simpson (1944) states; "he is either well or ill; there is no half way". Although it is the first time he has ever slept on a bed or between clean sheets or has been properly looked after when sick, he only appreciates this until the time when he considers he is fit for duty, which is usually some days before the medical officer agrees to discharge him from hospital.

SPECIAL INVESTIGATIONS.Hirst Test.

Samples of Blood serum were taken during the epidemic period from twenty pneumonia patients about the fourth and fourteenth day of the disease. These were sent by air to the Emergency Vaccine Laboratory for influenza virus serological reactions.

Lieut. Colonel Francis reported that one of the pairs of sera showed a significant rise in influenza B., virus agglutinins and one other was probably significant for influenza A virus.

Gold Agglutinins.

Recent literature has drawn attention to the high content of cold agglutinin in the blood serum of patients suffering from atypical or "virus-type" pneumonia. (Peterson, Ham and Finlaid, 1943).

Using the technique described by Turner (1943), tests were carried out on the sera of early and convalescent patients in an endeavour to demonstrate their presence.

There was uniform absence of agglutinin in significantly high dilutions.

Animal Experiments.

Intranasal inoculations of Seitz filtrate of saline nasal washings of patients into monkeys, guinea-pigs and mongooses, failed to demonstrate a virus.

Using a similar technique in mice, however, Brigadier Findlay got a broncho-pneumonia with two such samples. The first was obtained directly, the other after one blank mouse passage. This strain was passaged successfully into a second lot of mice.

Other experiments, with negative results, included intranasal inoculation of Seitz filtrate of sputum and of post mortem material into similar animals. Direct intra pulmonary inoculation of monkeys with unfiltered material obtained from the pneumonia consolidation by lung puncture also failed to reproduce the disease.

#### Lung Puncture.

It was felt that the African, like children, might have difficulty in expectorating from the lowermost respiratory passages, a possible explanation for failure to isolate organisms other than normal upper respiratory commensals.

The appearance of the sputum however, hardly warranted this view and certainly in the later weeks, when patients with sudden onset and rusty sputum of typical lobar pneumonia were seen, one had no difficulty in isolating the pneumococcus.

Puncture of the lung, or aspiration biopsy, was instituted in an endeavour to isolate the offending organism directly from the diseased area.

**TECHNIQUE:** The area of consolidation is localised by clinical examination and preferably confirmed by fluoroscopy. The chest wall over the area is anaesthetised with novocain as for pleural aspiration. A large bore sterile needle, three inches long, with stilette, is then inserted through the chest wall and pleurae into the consolidated area. The stilette is withdrawn and the needle pushed gently to and fro for half an inch. Gentle suction with a Record syringe may be applied during withdrawal of the needle. If more than gentle suction is used or if it be applied too soon, blood is usually aspirated and dilutes the exudate. The fluid obtained should fill only the needle and should not appear in appreciable quantity in the barrel of the syringe. Films are made and culture plates inoculated by blowing the fluid out of the needle with the syringe.

It was realised that the procedure was not without danger but only once a small haemoptysis was induced; it lasted five minutes and amounted to about an ounce. In one pneumococcal lobar

pneumonia so investigated a loculated empyema appeared later. This was the only evidence of infection possibly conveyed to the pleura. No other complication occurred.

The procedure was carried out in 28 patients with well marked pneumonic consolidation, who had not yet received treatment with sulphonamide.

In 18 of these, a satisfactory inflammatory exudate was obtained. Films were stained by Gram and Giemsa and showed numerous pus cells, red cells and tissue cells.

Definite pathogenic organisms were not isolated by this method from the pneumonias associated with upper respiratory infection. Four smears showed scanty organisms of *B. subtilis* group; one showed numerous yeast cells. Cultures produced organisms of *B. subtilis* group in six patients (including four seen on direct smear) and in one or two others, isolated colonies, presumably contaminants, included coagulase negative staphylococci, yeast cells and coliform organisms.

A strikingly different result was obtained when six typical pneumococcal lobar pneumonias were thus investigated. These men had rusty sputum containing pneumococci and the illness had a sudden onset with rigor. In four of these, a satisfactory inflammatory exudate was obtained on puncture and in each, pneumococci were grown on culture. In three of these the organisms were seen on direct smear of the exudate.

During production of local anaesthesia for this minor procedure, an endeavour was made to aspirate any small quantity of fluid present in the pleural cavity overlying the pneumonia.

This was successful in five instances, when five or ten cubic centimetres of a turbid fluid were obtained. Polymorph leucocytes predominated in stained films, no organisms were seen but in two fluids, *B subtilis* was obtained on culture.

Blood Culture.

This examination was not done as a routine. Sterile cultures were invariably obtained in the limited number of cases so investigated.

DISCUSSION.

Investigation and observation of this pneumonia epidemic has failed to reveal with certainty the exact aetiology of the disease. The story of the epidemic and the universal presence of upper respiratory inflammation suggests some form of droplet infection which could be either viral or bacterial.

It in some ways it resembled ordinary pneumococcal pneumonia; e.g. leucocyte counts between 12,000 and 24,000 per cmm, the lobar and lobular distribution and the excellent response to sulphonamide. Against this belief, there was the characteristic insidious onset with nasopharyngeal infection for two to four days before the appearance of pneumonia, the lack of rusty sputum and the failure to demonstrate pneumococci by standard methods. The low fatality rate (0.55%) is striking; one would expect this to be much higher in pneumococcal pneumonia affecting such undernourished primitive natives who presumably lacked previous exposure to such infection.

Anderson et alia (1942) have shown from a study of 1,940 pneumococcal pneumonias treated in Glasgow 1938 - 42, that the sulphonamides are most effective in the middle age group, within which our patients were included (all were between 18 and 40 years, mostly 20 - 30 years of age). The fatality rate in this age group in their series (1007 patients) was 2.98%.

During later months of the period under review, when the major epidemic had subsided, several cases of typical pneumococcal pneumonia were seen. These patients had a sudden onset of illness

with rigor, rusty spit containing pneumococci and four out of six lung punctures performed, yielded pneumococci. These then, differed markedly from the form of disease seen during the epidemic.

Simpson (1944) studying Lobar Pneumonia in African Soldiers (West Africa November 1941 to July 1942), found that of 255 pneumonias, 159 were classical pneumococcal lobar type. He classifies the remaining 66 as "Atypical Pneumonia" and used the term to designate "all forms of pneumonia which do not conform clinically with typical lobar pneumonia- it is to be clearly understood that virus pneumonia is not referred to". He found that these "atypical pneumonias" were prevalent up to May and then most assumed typical lobar form. It seems not unlikely that the writer is referring to a condition similar to, if not identical with, our epidemic pneumonia.

During the first three months of 1944, when the epidemic was at its height there was a smaller epidemic occurring, among African railway labourers, some sixty miles from the station. 200 men with classical lobar pneumonia were admitted to the civil hospital. Sputum was invariably rusty and contained large numbers of pneumococci. The onset of each case was sudden and response to sulphonamides excellent. There were six deaths.

It would seem certain that our pneumonias during the epidemic were not of typical pneumococcal type; certainly not Type I or II, which are usually typed without difficulty and produce characteristic rusty spit. A number of <sup>pneumococcal</sup> pneumonias do have a preliminary cold or upper respiratory catarrh and this is commoner with Group 4, which is also responsible for more typing failures (Anderson 1944).



The few purulent complications seen (Empyema rate 0.68%) were pneumococcal; three of the empyemas occurred in patients who had frank pneumococcal pneumonia and it is possible that the others were related to similar pneumonias occurring as isolated cases during the epidemic.

Type III pneumococcal pneumonia is nowadays considered to be an endogenous infection, encouraged by a precedent lowering of resistance to pneumococcal invasion. It may be that we have been seeing one of similar nature, a primary virus upper respiratory infection lowering the resistance of the patient to the ravages of such a secondary invader. Judging by the comparative mildness of the disease, the organism directly responsible for the pneumonia must be of low virulence or one to which the patient is normally well accustomed.

The only organism isolated fairly frequently from sputum, lung puncture and pleural exudate, was of B. subtilis group. This organism, like the pneumococcus, is included in the group of "sputum commensals". It is the Hay bacillus, is usually considered non-pathogenic and is a frequent contaminant on culture media. According to Axenfeld (Muir and Ritchie), it may produce infection of the eye with resulting iridocyclitis and panophthalmitis and subcutaneous inoculation may produce a lethal effect in mice and guinea-pigs if large doses of cultures are used. The possibility that this was the organism directly concerned with the pneumonia infection, however, is not considered likely.

In an epidemic of pneumonia where the causal organism cannot be demonstrated, one must exclude Primary Atypical Pneumonia of virus origin. Recent extensive literature on the subject demonstrates certain points of similarity to this pneumonia seen in West Africa. Thus Drew, Samuel and Ball (1943) describe three modes of onset:

1. like influenza with headache, shivering and generalised pains followed by cough.
2. upper respiratory infection with coryza, sore throat, retrosternal pain and cough.
3. cough and pleuritic pain.

The sputum is mucoid, occasionally blood-stained and contains organisms normally seen as commensals; pharyngitis is common; the picture is difficult to distinguish from influenza.

Young, Storey and Redmond (1943), in an article with similar conclusions, show that repeated cultures fail to reveal convincing evidence of secondary bacterial invasion and blood culture is invariably negative. They find the white cell count normal in the early stages but later rising to 11,000 - 20,000 per cmm. Meekins, also Haight and Trolinger, allude to the insidious onset with cough, malaise, headache and non-pleuritic chest pain and to physical signs which consist of suppressed breath sounds in the early stages and rales later. Currell and Cowan (1943), reporting an epidemic of primary atypical pneumonia of unknown aetiology in an army camp in Louisiana, found that other respiratory illnesses occurring in the unit showed identical symptomatology with the pneumonia cases and concluded that the

Table V.

A Comparison of Distribution of the Present Series  
of Pneumonias with that of 200 Cases of Primary Atypical  
Pneumonia at Fort Eustis, Virginia.

Distribution of Consolidation.	Primary Atypical Pneumonia 200 Cases.	Present Series 735 Cases.
Basal Consolidation.	81%.	76%.
Right Lung.	39%.	41.7%.
Left Lung.	39%.	37.3%.
Both Lungs.	22%.	21%.
Right Upper Lobe.	11%.	8.9%.

illnesses might be milder infections of the same specific nature. The distribution by lobes was the same as pneumococcal pneumonia but they claimed that "differentiation was usually possible". Inoculation of sputum, naso-pharyngeal washings and blood into guinea-pigs, mice, chick embryos and ferrets in attempts to isolate the causal virus were uniformly unsuccessful. Joules (1943) states that atypical pneumonia may show the same signs and approximately the same mode of onset as pneumococcal pneumonia; "lung puncture should be used before asserting that consolidation is non-pneumococcal, because chemotherapy, which most patients will have had, may sterilize the sputum but possibly not the lung!"

These then, are findings not dissimilar to these in the present epidemic, and Table V shows that the distribution of these pneumonias closely resembled that of 200 cases of **Primary Atypical Pneumonia** reported by Campbell et alia (1943)

Reading the literature on atypical pneumonia, one is struck with the varying descriptions of symptoms and signs and one reaches the conclusion that it is not a single disease entity which is being described but rather a number of related conditions. "A virus has been isolated from some cases by using the cotton rat or the mongoose but the disease is probably a group of conditions caused by different aetiological agents" (Brown 1943). Thus Dingle and his co-workers restricted the diagnosis of atypical pneumonia to those in which there was definite radiological evidence of a lung lesion and in which pneumococcal pneumonia could be excluded.

There are various points in the descriptions which distinguish

the condition clearly from our series of pneumonias. Thus there would seem to be uniform agreement that primary atypical pneumonia does not respond to sulphonamides. Pulse rate remains low in relation to temperature, and respirations are rarely above 30 per minute and never embarrassed (Meakins) Relative bradycardia and normal respiratory rate were described by Haight and Trolinger. The late leucocytosis described by Young, Storey and Redmond is not found in other reports. Thus, in the paper by Meakins, the leucocyte counts were relatively low, below 10,000 per cmm., in 70% of cases and in Currell and Cowan's series, below 12,000 per cmm., in 95%. All writers stress the paucity of physical signs as compared with the extensive X-Ray findings. "Atypical pneumonia differs from ordinary pneumonia by its usually mild character, few physical signs, relative leucopenia, absence of bloody sputum, hazy appearance on X-Ray and lack of response to sulphonamides" (Brown 1943). Infectivity would appear to be comparatively low. Radiological appearances are of various types: (a) a hazy, soft, string-like infiltration extending out from the hilus onto the periphery, usually of the lower lobes; (b) a dense, circumscribed, fan-like shadow; (c) a diffuse veil-like shadow occupying about two thirds of a lobe and simulating atelectasis but with no shift of the mediastinum; it may resemble that of certain types of lobar or broncho-pneumonia but the density is rarely as marked and circumscribed as in pneumococcal pneumonia.

In the present series of cases, X-Ray showed varying degrees of density always lobar or lobular in distribution with associated physical signs of consolidation comparatively well marked at some stage of the illness.

Tenderness of chest wall is not mentioned in the literature of atypical pneumonia.

Peterson, Ham and Finland (1943), also Turner have drawn attention to the high content of cold agglutinins in serum of patients suffering from primary atypical pneumonia. As already mentioned, there was uniform absence of such agglutinins in our cases.

A three to five day fever, with malaise, headache, general aches and pains and with pulmonary complications in a proportion of the cases, is in keeping with a diagnosis of influenza. Most of the patients complained of pains round the front of the chest as a predominating symptom. Although nasal and pharyngeal signs with a cough and scanty spit were invariably present, the African seldom complained of these, being more concerned with the manifestations of general illness - "headache, fever and pains in the chest".

That the infection was due to a true influenza virus is unlikely. Of twenty pairs of sera examined, only two showed a significant rise in influenza virus agglutinins.

The symptoms, as experienced in Europeans working in close

working in close contact with the patients, were those of common cold with pharyngitis predominating; not one of these developed a chest complication.

As Stuart-Harris (1941) points out, the future may reveal the existence of not two but a whole cluster of influenza viruses possessing different serological and pathogenic properties, yet showing in common the capability of attack upon the mucous membrane of the upper and lower respiratory tract.

Donnelly et alia (1944) have recently described a minor epidemic of influenza A infection in an R.A.F. unit. Pneumonia complications and leucocytosis were uncommon. They noted that sulphapyridine had definitely assisted the drop in temperature - "being normal in each case before a total of 8 grams had been given". This was our experience with the drug, even in the simple cases of pharyngitis.

Until, recently, it was thought that secondary pneumonia is necessarily diffuse and broncho-pneumonic in type. Increased use of radiography has shown that mere localised single areas of consolidation are quite common. Thus Kennedy (1943) found such abnormal shadows in 100 people during mass radiography. 74 of these had coryza or admitted symptoms of common cold within the preceeding 7 - 14 days. The distribution of the shadows (Right lung 49. Left lung 30. Both lungs 21) was that expected from a descending infection (Compare with Table V). He found evidence of collapse not uncommon in these areas and ascribed the shadows to "a low grade alveolar infection or deficient aeration caused by swelling of the bronchial mucosa". Ramsay and Scadding (1939) describe a transient localised

consolidation arising in the course of a catarrhal infection of the respiratory tract. They term this "benign circumscribed pneumonia" and say that it produces few symptoms and is commonly found on routine radiological examination.

Lobar pneumonia has been considered, for practical purposes, synonymous with pneumococcal pneumonia. E.C. Smith (1945) has shown that in children in Nigeria, cases of pneumonia from which staphylococcus aureus was isolated, were of lobar type. Too often, he said, the aetiological significance of pneumococci, isolated from the sputum and typed, is taken for granted. In other words, a few pneumococci in the sputum, even if they belong to a pathogenic group, may be an incidental finding. Staphylococcal pneumonia has long been recognised as a complication during influenza pandemics; minute abscess formation was seen in the lungs of two of our patients who came to autopsy but the severe suppurative and necrotising lesions characteristic of staphylococcal infection did not occur.

E. Davis (1944) has described the pulmonary complications seen during a recent influenza epidemic in England, November 1943. "White cell counts were essentially the same as in primary lobar pneumonia. The sulphonamides had no obvious effect in at least 12 of the 17 cases ..... Concurrently with the influenza epidemic, primary lobar pneumonia became much more frequent..... It may be that a number of these cases were really influenzal pneumonias, but there was not enough clinical evidence for so regarding them".

That a filtrable virus spread by droplet infection was the causative agent in the primary respiratory infection of the present epidemic, is supported by the extreme infectivity of the condition



and by the successful transmission of disease to mice with Seitz filtrate of saline nasal washings of patients. The very early and pronounced leucocytosis suggests that secondary infection by organisms, presumably already present as normal inhabitants on the inflamed mucosa, took place almost immediately. Onset of pneumonia was usually delayed for a few days and this also was apparently due to one or more similar organisms, indistinguishable from those of the normal flora of the respiratory mucosa.

The reason for localisation of pneumonia to a certain individual lobe or segment of a lung has always been to the writer a matter for speculation. The frequency of early clinical evidence of collapse in these pneumonias suggests that deficient aeration, caused by swelling of the mucosa or blockage, by a mucus plug, of the bronchus supplying the segment, produces a state of affairs conducive to infection by organisms already present in the finer air passages of the affected area. Kaunitz (1940) postulates that upper respiratory infection may be complicated by a localised "atelectasis" which, when infected, produces a pneumonia. It is well known that such multiplication of organisms does take place in a lobe or segment collapsed by more chronic disease or foreign body. It may be that a similar mechanism plays a part even in the early stages of primary pneumococcal lobar pneumonia.

It was of great interest to see a similar contagious pneumonia which affects cattle in the Northern Territories of the Gold Coast and in other Colonies of West Africa. Stewart (1943) describes it as a disease caused primarily by a virus which "pathogenises" one or several of the organisms, already present as usual inhabitants

of the mucosa but which, in itself is comparatively benign. The actual contagious pneumonia in cattle is preceded by a general coryza, the effect of the virus per se."

A small localised area of pulmonary inflammation was a fairly frequent finding during the epidemic. Physical signs consisted of scanty rales confined to a small area or segment of a lobe, without evidence of true consolidation and with little to be seen on X-Ray. Often, the patient could localise the patch accurately by pain and tenderness. Nomenclature in this type of case is difficult; by the older clinicians, it was termed "a patch of congestion" or even "threatened pneumonia". Neither of these make a satisfactory diagnosis on an Army, or indeed on any other hospital case sheet. The term pneumonitis has been revived in recent years but unfortunately has been used to describe a variety of different conditions such as primary atypical pneumonia or even capillary bronchitis. One would like to see the term "focal pneumonitis" reserved for the above type of localised inflammatory parenchymal lesion.

The infrequency of both early and late complications of pneumonia in Africans, can be explained in part by their mobility and activity during the illness. It is unlikely that bronchial mucous plugs will remain in situ for any length of time when the patient is walking about the ward on the day after admission! This can surely be a lesson to us. Now that sulphonamides have almost abolished the dreaded risk of heart failure occurring later in the course of the illness, we should encourage our younger pneumonia patients to move about or at least do simple postural exercises in bed as soon as the more acute stage has passed. Unfortunately, nurses

are still taught to care for these patients on old fashioned lines, thus increasing the risk of chronic pulmonary disease. Breathing exercises alone are probably of less value, having the effect of encouraging lung expansion only if the air passages are clear. Indeed it is conceivable that more harm than good might come of forced breathing before the mucous plug has been expelled. Coryllos, in America, has advocated bronchoscopic aspiration in these cases of pneumonia showing evidence of collapse and although the method is considered somewhat drastic, one can believe that in skilled hands it might benefit some of these patients.

C O N C L U S I O N .

The epidemic was primarily one of upper respiratory infection, spread by droplet infection, aided by overcrowding and probably due to a virus.

In Europeans and in seasoned African soldiers, the illness did not progress beyond this stage of naso-pharyngeal inflammation, with symptoms akin to those of common cold or a mild influenza.

In the majority of recruits the condition went on to pneumonia. The principal reasons for this difference in susceptibility were an appreciable degree of malnutrition in these men and the fact that they lacked previous exposure and consequent immunity - truly they were "raw recruits". Other factors such as anaemia, fatigue, chill and inoculations are suggested.

This pneumonia had certain points of resemblance to typical pneumococcal lobar pneumonia and to primary atypical pneumonia of virus origin but sufficient contrasting features were found to distinguish it from each of these.

The segmental distribution, failure to isolate specific organism and various other features suggest that parenchymal infection was caused by an organism or organisms, normally found as commensals on the upper respiratory mucous membrane, which had gained access to the lower respiratory passages by way of an inflamed mucosa. We are thus reminded of the essential continuity of the whole respiratory mucous membrane.

Well-marked leucocytosis and most satisfactory response to sulphonamide favour a bacterial, rather than a viral, agent in

the production of lung consolidation.. Yet the only organism grown significantly often on culture of sputum, lung puncture exudate and pleural fluid was of *B. subtilis* group, which is usually considered a contaminant. In a susceptible person, such organisms might be expected to become pathogenic in an area of lung improperly aerated. Other writers have produced evidence to show that diminished air entry in a lung segment may occur during the course of an upper respiratory infection, by swelling of the mucosa of a finer bronchus or by plugging with tenaceous mucus. In these pneumonias, there was both clinical and radiological evidence of such early associated alveolar collapse.

The epidemic bears a striking resemblance to a smaller one described by Shore and Passmore (1943). 54 Indian soldiers were admitted to a hospital in the Middle East with pneumonitis associated with upper respiratory infection. Polymorph leucocytosis was well marked, being as high as 61,000 per cmm., in one of the cases. No special organisms were isolated from sputum, blood or lung puncture. The epidemic occurred in the cool season, when the nights were cold and days warm and dry.

THE SEGMENTAL ANATOMY OF THE LUNGS IN RELATION TO PNEUMONIA.

As physician and bronchoscopist in a thoracic surgery unit the writer was specially interested in anatomy of the lungs and bronchi, and experience with these pneumonias has helped to enhance his knowledge of the subject.

Lipiodol bronchographic studies made during preoperative pulmonary investigations and bronchial injection of post mortem lung specimens demonstrated that the lungs are a collection of easily recognisable and fairly constant broncho-pulmonary segments, each supplied by a twig from the bronchial tree. This segment or lobule seems indeed to be replacing the lobe as the surgical unit and this necessitates a thorough knowledge of segmental anatomy if foreign bodies or disease such as bronchiectasis, abscess or atelectasis are to <sup>be</sup> accurately localised before operation.

Three hundred years ago, anatomists made a study of the bronchi by filling the air passages with fusible metal and removing the lung tissue by corrosion, but no detailed description was given until that of Aeby (1882). In 1889, William Ewart, physician and pathologist in the Brompton Hospital, using the old method of fusible metal casts, gave a minute and accurate account of the ramifications of the tracheo-bronchial tree.

Bronchoscopy, first demonstrated by Killian (1897) was further developed by Chevalier Jackson after 1904. The study of

bronchography began in 1917 when Waters, Boyne-Jones and Rowantree made X-Ray studies in living animals with 10% iodoform in olive oil; the following year Jackson studied the human tracheo-bronchial tree by X-Rays after injection of dry bismuth subcarbonate powder into the lower passages; Sicard and Forestier (1922) are usually considered the pioneers of modern "lipiodol" bronchography, their original prescription of 40% iodine in poppy seed oil being still that in common use to-day.

Despite these advances in visualising bronchial pathology, it was not until the advent of modern thoracic surgery some 10 years later that an interest in the normal anatomy of the bronchial tree was revived. Kramer and Jones (1932) demonstrated the division of the lung into segments, each supplied by a well defined bronchus. Nelson (1934) in a short article on Postural Drainage of the lungs, gave a concise account of the course and nomenclature of the bronchial branches and his diagrams are frequently referred to and reproduced to-day. Churchill and Belsey (1939) gave a similar account with special reference to bronchography and the surgery of bronchiectasis, their description and diagrams being in the main, deduced from lipiodol studies in the living patient.

More recently Foster-Carter (1942) published details of a method of producing corrosion casts of the bronchial tree, using celloidin instead of fusible metal, and before coming to West Africa, the writer made studies using a similar technique.

Briefly, the method consists of the instillation, into the air passages of a freshly excised lung, of a solution of used X-Ray film in acetone. The lung is then suspended in a jar containing

concentrated sulphuric acid which, in the course of a few days, corrodes the organic tissue, leaving a rather beautiful solid cast of the bronchi. The chief difficulty is that air bubbles frequently break the continuity of the finer branches and that the cast in itself is so extremely delicate.

Foster-Carter found that his casts brought to light certain points which were at variance with previous accounts and he undertook further investigations of dissections and bronchograms in order to supply the omissions and clarify obscurities in former descriptions. He published his results (1943) as "An Outline of Bronchial Anatomy".

The present series of pneumonias has demonstrated to the writer that a knowledge of the segmental anatomy of the lungs is of importance to the physician in charge of non-surgical pulmonary cases as well as to his surgical colleague. As already mentioned, one has been struck with the essential segmental distribution of these pneumonic consolidations, studied clinically and radiologically. An opportunity thus arose for the further study of broncho-pulmonary anatomy.

In the writings referred to above, the authors are agreed on most points as regards the anatomy of the tracheo-bronchial tree. Minor differences of opinion arise with regard to nomenclature and to branches of the upper lobe bronchi. In a modern "Textbook of X-Ray Diagnosis" by British authors, the description shows rather more variation. These differences are well illustrated in approximate reproductions of one diagram from each of these articles, portraying a lateral view of the left bronchial tree. (Diagram II).





(a) Antero-posterior. (b) Right Lateral.  
Plate 10.

Pneumonia axillary area right upper lobe. Lateral view shows pectoral segment slightly involved.

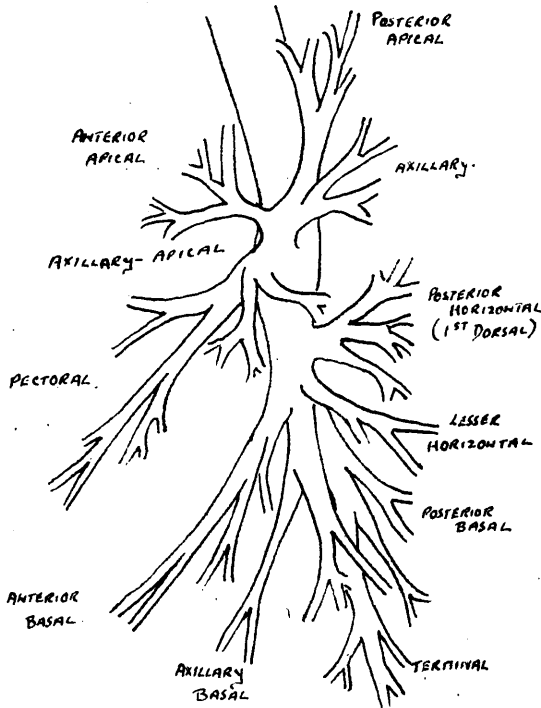


(a) Antero-posterior. (b) Lateral.  
Plate 11.

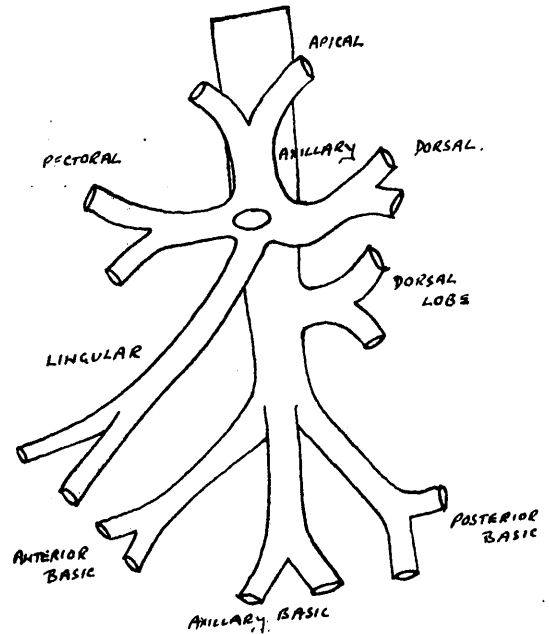
Pneumonia axillary area right upper lobe. In lateral view, the pectoral segment is seen to be clear, but the posterior segment is probably involved. Note mediastinal retraction as evidence of partial collapse right upper lobe.

DIAGRAM II. Left views of tracheo-bronchial tree (lateral)  
as portrayed in previous articles.

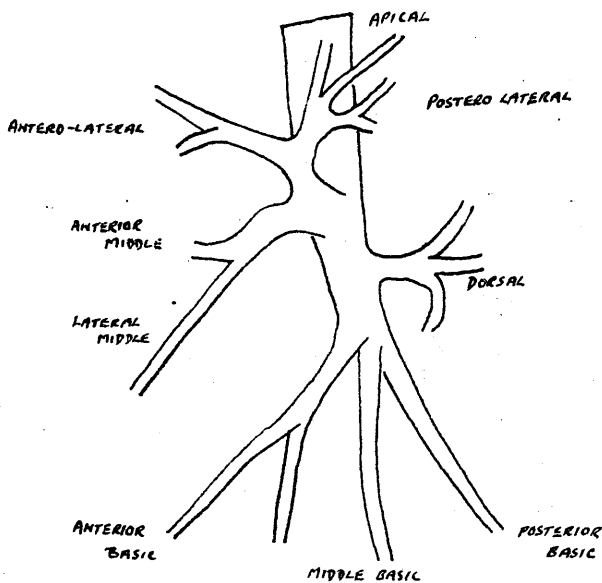
SHANKS, KERLEY AND TWINING



CHURCHILL AND BELSEY.



FOSTER-CARTER



NELSON.

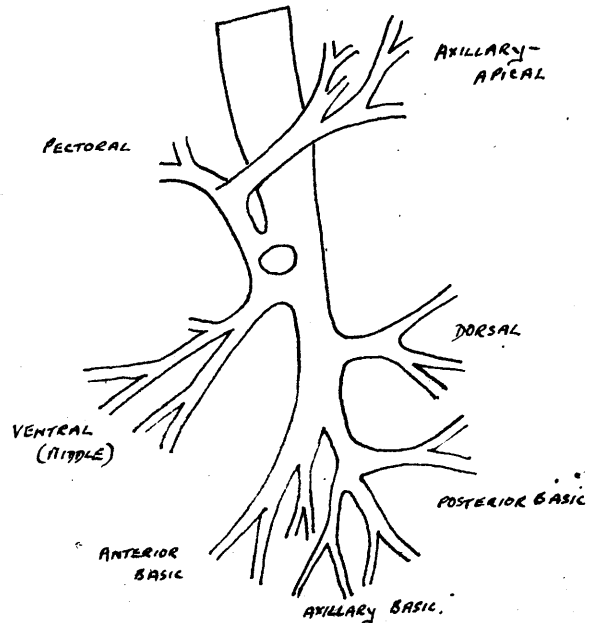




Plate 12.

Pneumonia axillary area left upper lobe.

Note the calcified Ghon Primary Complex in right lower lobe.

intranasal technique with fixed posture.



- (a) Upright position for preliminary local anaesthesia. The anaesthetic (Anethaine 2%) is inserted into the nostril from the blunt end of a Record syringe while the tongue is firmly held in the extruded position.



- (b) Lateral posture for filling the whole right bronchial tree. Iodised oil is inserted in the same manner as the local anaesthetic.

Plate 14. Complete Delineation of the Tracheo-Bronchial Tree  
in Two Stages.



(a) Antero-posterior.

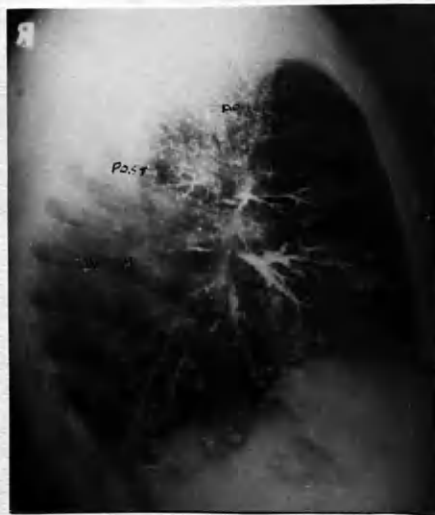


(b) Left Lateral.

(a)&(b) Left lateral bronchogram showing normal upper lobe branches and bronchiectatic lower lobe.



(c) Antero-posterior.



(d) Right Lateral.

(c)&(d). Complete filling of right bronchial tree.  
Note the normal "leafy tree" appearance.

It will be seen that three are agreed upon the course of the bronchi of the lower lobes and middle lobe (or lingula) but all are at variance as regards the branches of the upper lobe bronchus. Nelson shows the bronchus, after giving off the standard lingular branch, dividing into two, the anterior supplying the pectoral segment and the other again dividing into apical and axillary branches. Churchill and Belsey depict four branches, pectoral, apical, dorsal, and axillary and point out in smaller diagrams on variations, that the axillary branch may originate from apical or even the lingular bronchus. Foster-Carter states that the axillary branch does not normally exist in the human lung, the axillary area being supplied by lateral twigs from the anterior, posterior and apical branches. He showed in his former article on the subject that other important variations occur.

In the present pneumonia series, consolidation of nearly every individual segment described in Foster-Carter's paper, was met and confirmed radiologically. In several instances of upper lobe infection consolidation seemed to affect only the axillary area of the lobe, or, at most, axillary and pectoral areas (Plates 10, 11 and 12); one wondered if perhaps, Nelson and Churchill and Belsey were not correct in postulating a separate axillary bronchus and segment.

To clarify this obscurity it was decided to make further examinations of living and post mortem bronchograms and of upper lobe dissections.

It is probable that this difference of opinion by authoritative workers on a point which would seem easily discernable, is in part due to the fact that upper lobe branches are rarely fully delineated in

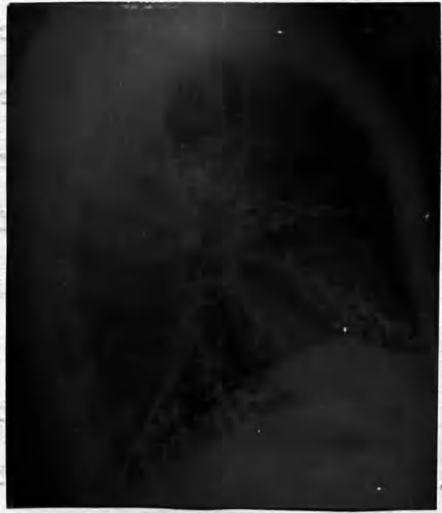


Plate 15. Normal right bronchogram.

Plate 16. Normal right bronchogram, lateral view.



Plate 17. Right bronchogram showing sacular bronchiectasis.

Plate 18. Left bronchogram showing fusiform bronchiectasis of left lower lobe.

a lipiodol bronchogram. For some years, the writer has used the method of intra-nasal lipiodol instillation with fixed posture. (Plate 15). Some bronchograms thus obtained demonstrate that complete upper lobe filling and, in fact, complete bronchial delineation of a single lung is possible in most cases by this procedure. (Plates 14 - 18). Owing to the relative density of the upper mediastinum however, the branches of the upper lobe bronchi are seldom so clearly seen as those in the lower chest in the lateral view.

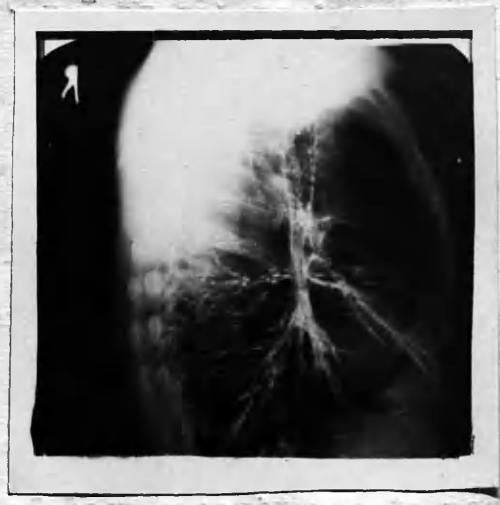
There are three most important points, well recognised yet worthy of stress, in bronchography: each lung should be iodised separately with an interval of some days between (c.f. Plate 19): an endeavour should be made to fill all segmental bronchi of the lung at one time: a good lateral film is most helpful and should never be omitted.

While bronchoscopy supplies most accurate information on the anatomy of the trachea and main bronchi, it is only in the case of the lower lobes that one can visualise the orifices of the segmental bronchi (although the modern retrograde bronchoscope does enable one to see a little way into the upper and middle lobe bronchi.).

Bronchographic studies with material excised at post-mortem or at operation are most easily performed with lung or lobe slightly hardened in form. Either lipiodol or bismuth emulsion can be used (Plates 20 & 23).

Dissections are instructive but photographic reproduction is unsatisfactory (Plate 24).





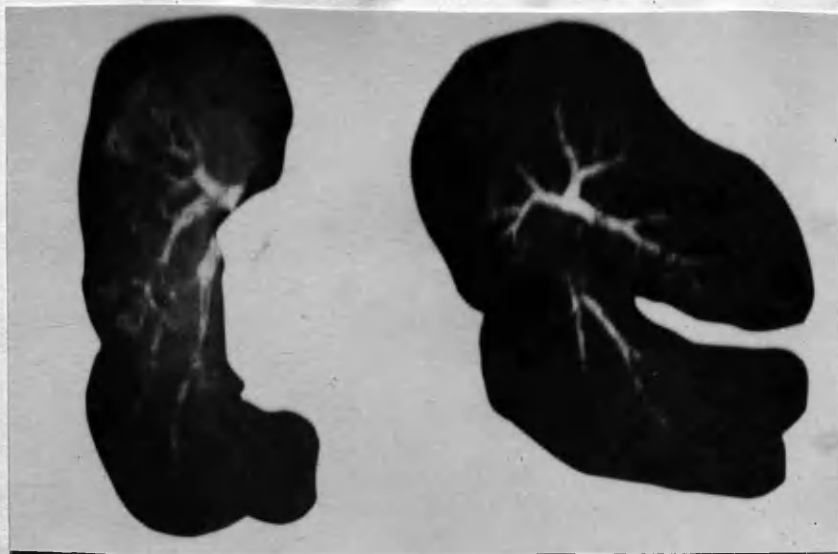
(a) Antero-posterior.

(b) Right Lateral.

Plate 19.

Bilateral bronchogram. Note the confused picture obtained in lateral view, owing to overlapping of bronchi.

Plate 20.



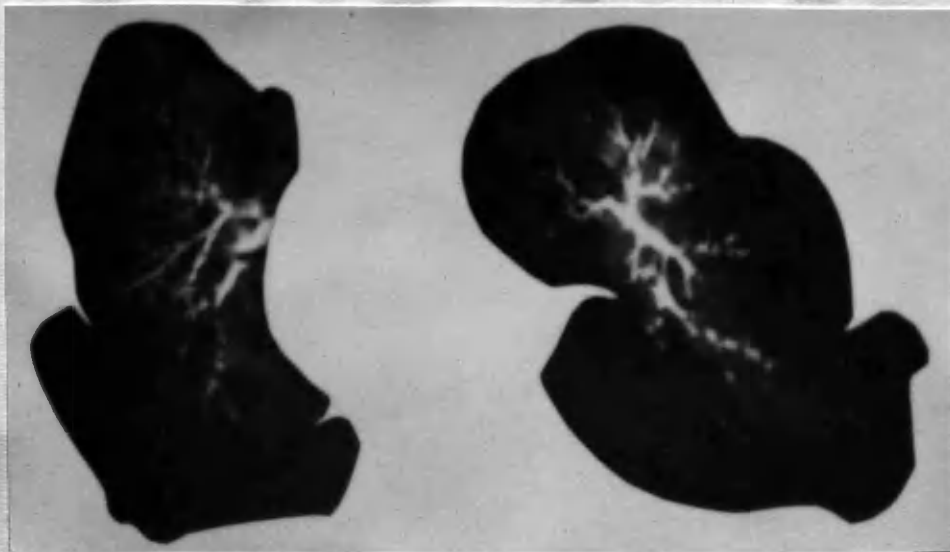
Antero-posterior.

Lateral.

Plates 20 & 21.

Two autopsy specimens of right upper and middle lobes, injected with Lipiodol. Note in each case the three segmentary branches of the eparterial bronchus and the two twigs of the middle lobe bronchus.

Plate 21.



Antero-posterior.

lateral.

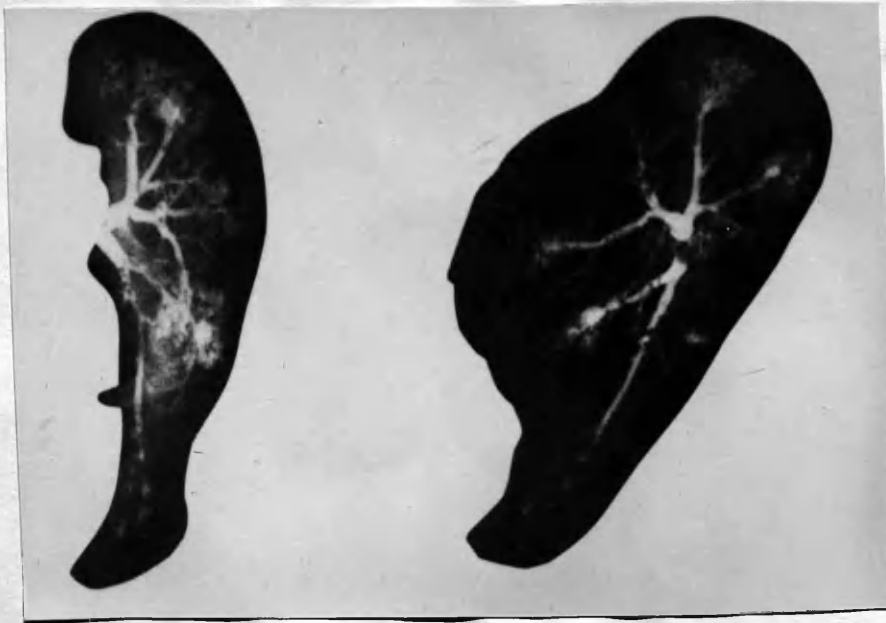


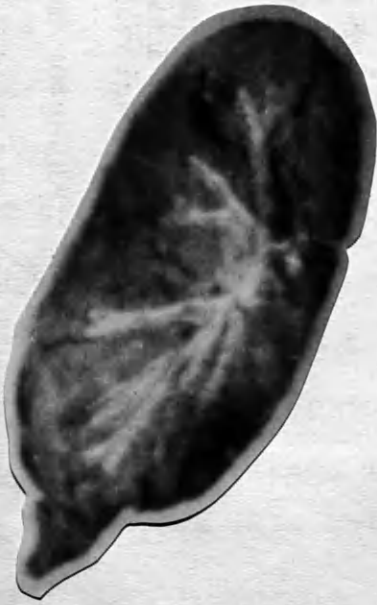
Plate 22.

Post-mortem Lipiodol bronchogram of left upper lobe and lingula. Note the three main branches of the upper lobe bronchus, pectoral, apical and posterior and the two lingular branches. The axillary area in this case is mainly supplied by a branch of the pectoral bronchus.



Plate 23.

Two antero-posterior post-mortem bronchograms, each showing the pectoral and lingular bronchi arising from a common stem.



Right Upper Lobe.

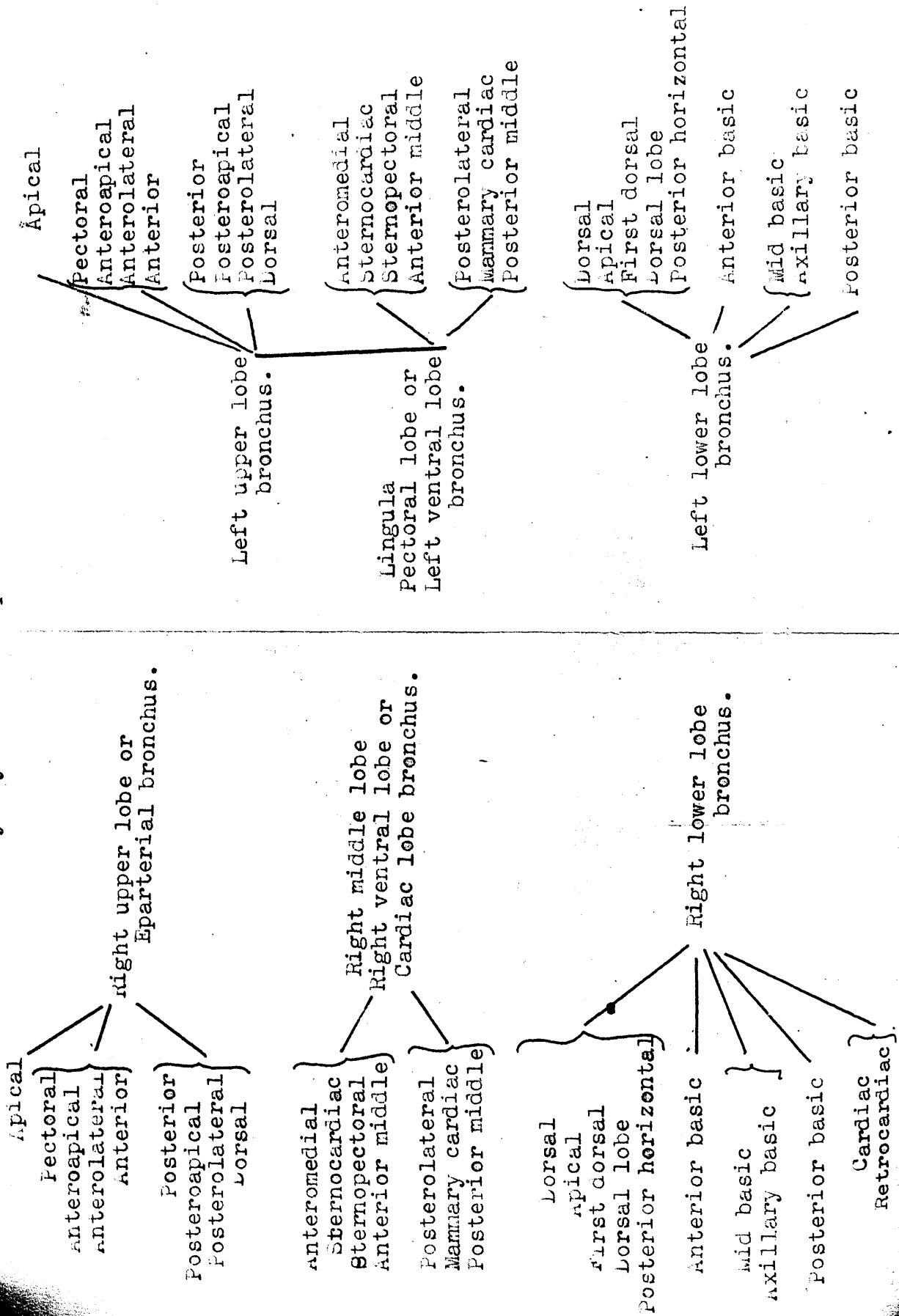


Left Upper Lobe and Lingula.  
(The arrow points to the orifice  
of an axillary twig from the  
pectoral bronchus.)

Plate 24.

Lissections of the upper lobes. The bronchial branches  
have been outlined with paint.

Figure 24. Nomenclature of the lobar and segmental bronchi with synonyms found in previous literature.



Investigation by the writer on these lines has confirmed Foster-Carter's belief that the axillary segmental bronchus, as such, does not normally occur and that the "axillary area" is supplied by twigs from pectoral, dorsal and apical bronchi. One of these twigs is usually considerably larger than the others and it may branch from any one of the three segmental bronchi; in the case of the left upper lobe bronchus, as Churchill and Belsey demonstrate, it may even originate from the lingular bronchus. Sometimes it may branch off so close to the main upper lobe bronchus as to seem a separate bronchus in itself. The fact that pneumonia not infrequently affects the pectoral segment and axillary area together suggests that in these cases, it is a branch of the pectoral bronchus; this arrangement was seen in two of the upper lobe specimens dissected.

The lesser posterior horizontal bronchus portrayed by Shanks, Kerley and Twining is inconsistent and was seen once in ten consecutive pairs of lungs dissected. Usually it is represented by a superior branch of the posterior basic bronchus.

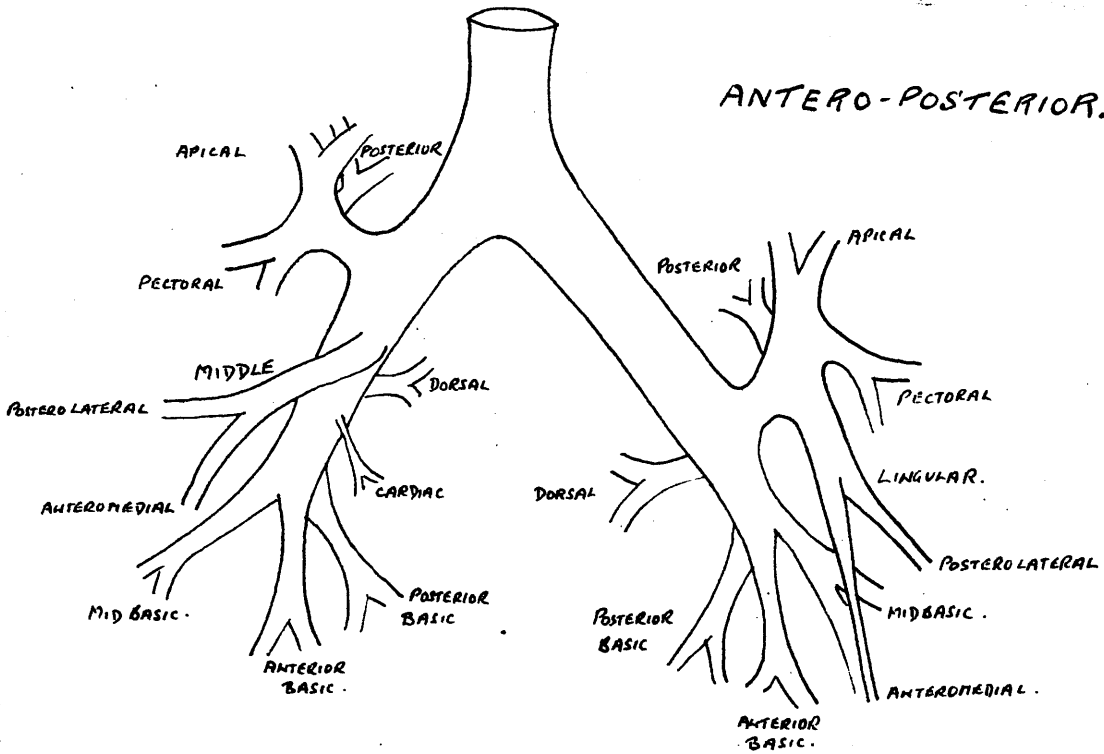
Variations in bronchial anatomy described in the literature and differences of opinion on the subject have concerned mainly the branches of the upper lobe bronchus. These can be summarised and reconciled as follows: the left upper lobe bronchus has four branches, apical, dorsal, pectoral and lingular; the former pair and the latter pair may arise from common stems (Plate 23.); the axillary area is supplied by twigs from some or all of these branches, the major twig coming from any one, frequently the pectoral bronchus.

These remarks apply to the right upper lobe bronchus with the

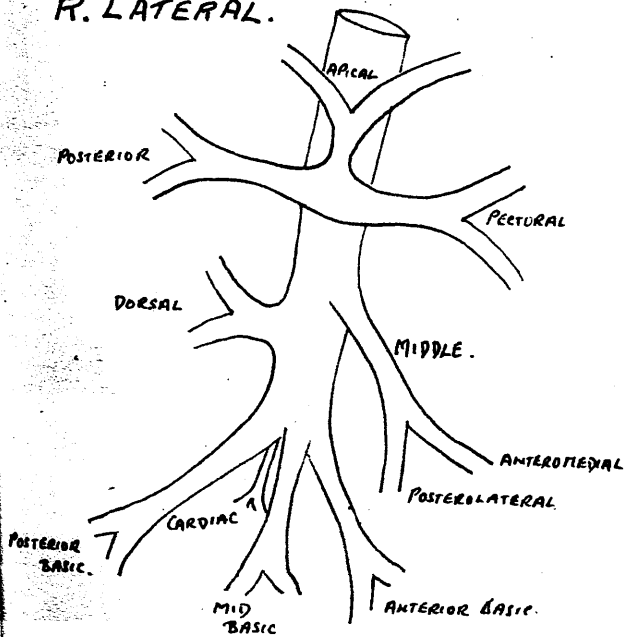
DIAGRAM III. The tracheo-bronchial tree.

Schematic diagram with suggested nomenclature.

ANTERO-POSTERIOR.



R. LATERAL.



L. LATERAL.

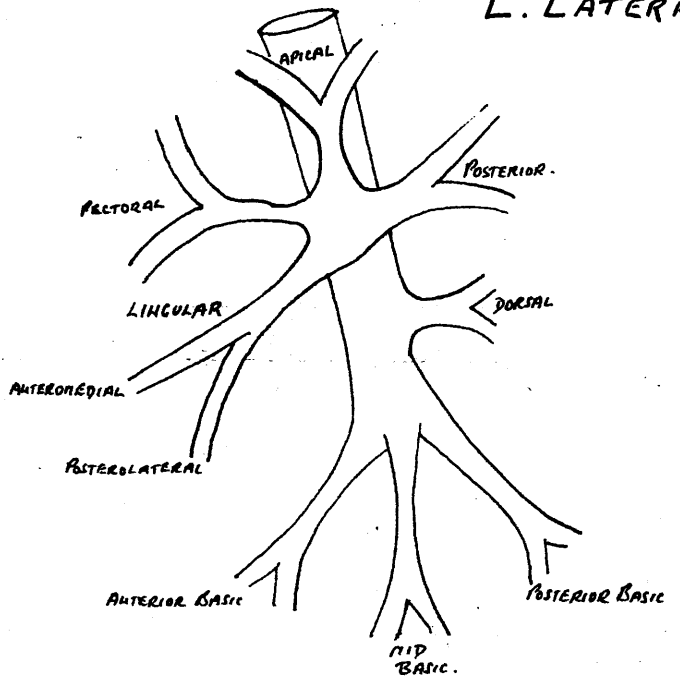


Plate 25. The Surface Markings of the Broncho-Pulmonary Segments.



Right Lung Anterior.



Right Lung Posterior.



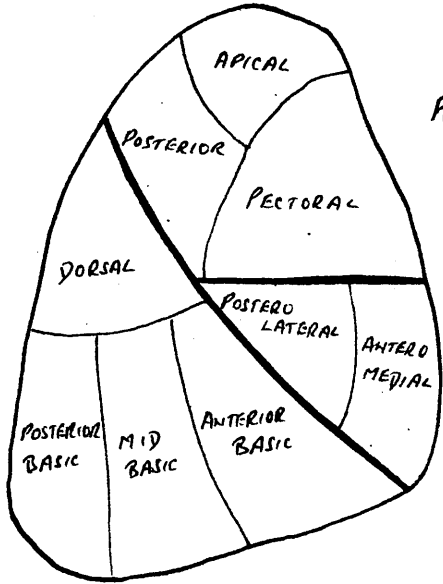
Left Lung Anterior.



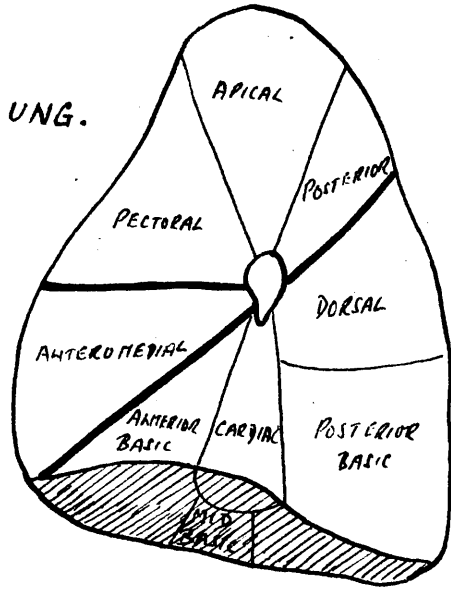
Left Lung Posterior.



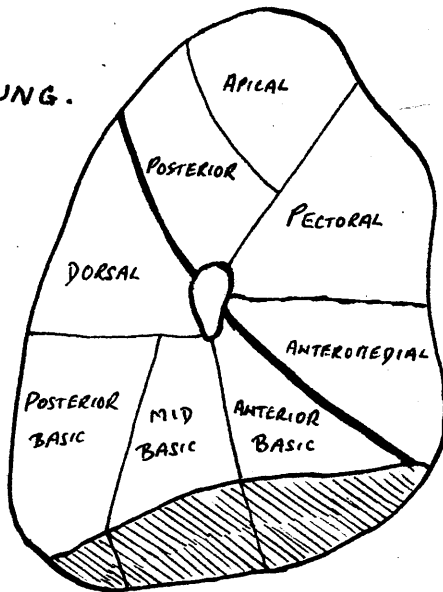
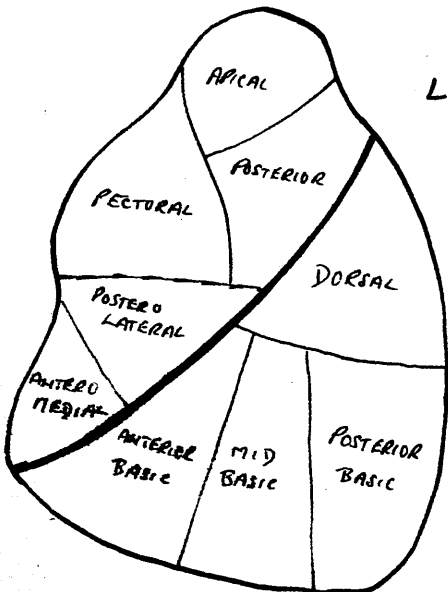
DIAGRAM IV. The Broncho-pulmonary Segments.



RIGHT LUNG.



LEFT LUNG.



exception that the middle lobe bronchus replaces the lingular branch.

While anatomy of the tracheo-bronchial tree has been studied in detail and segmentation of the pulmonary lobes is well understood and illustrated in modern radiological textbooks, (An Outline of X-Ray Diagnosis by British Authors (Reprint 1943) Lewis, London), it is surprising that one can find no description in the literature of the surface markings of the individual segments.

Clinical examination of these patients with pneumonia affecting a single segment, combined with radiological confirmation, has produced an understanding of this surface anatomy, probably more accurate than could be obtained by other methods of investigation. These photographs (Plate 25.) illustrate the impressions so gained. The surface markings have not a very accurate definition as the size of the segments relative to each other varies slightly in different lungs; in addition, physical signs related to one segment may encroach slightly on the border of an adjacent segment.

It will be seen that a knowledge of the surface anatomy of the lung lobes combined with diagrams of their segmentation (Diagram IV) allows one to map out the surface markings of the individual segments without much difficulty. Familiarity with standard nomenclature is the next essential. Too often one hears of "Pneumonia at the left base" or "Pneumonic consolidation of middle zone of right lung". Students should be taught precision.

in such descriptions - e.g. "consolidation of posterior basic segment of the left lower lobe".

Figure 24 lists the synonymous terms used in previous descriptions of these segments and illustrates the urgent need for uniform terminology.

The present nomenclature is submitted as probably the simplest and most descriptive and in Diagrams III and IV, is shown applied to drawings of the tracheo-bronchial tree and bronch-pulmonary segmentation.

A knowledge of segmental pulmonary anatomy is of some practical value in the diagnosis and treatment of such pneumonias. The middle of each of these surface segments is a site of election for the early appearance of physical signs and the stethoscope should not be allowed to miss one of these areas in examination during the invasive stage. As mentioned elsewhere, some collapse of the segment was a common associated finding and accurate postural measures necessitating such a knowledge, combined with emetic doses of expectorant mixture were of great value in hastening resolution.

S U M M A R Y.

1. The paper relates an epidemic of respiratory infection, associated with a high pneumonia incidence, which occurred in a West African Primary Training Centre. 733 pneumonia patients were treated in the hospital during a nine months' period January till September, 1944.

2. Aetiology is discussed; the condition is compared and contrasted with other recognised forms of pneumonia: clinical, radiological, pathological studies and animal transmission experiments are described.

3. Observation and investigation suggest that the infection was primarily by a virus, spread by droplet infection, related to that of the common cold and of influenza but not identified as such.

4. Pneumonia occurred as a secondary infection, apparently bacterial, in susceptible recruits, mostly primitive natives, many of whom showed evidence of malnutrition, avitaminosis and anaemia. The organism responsible for this secondary infection was not identified with certainty: it is suggested that one or more organisms normally present in the bacterial flora of the upper respiratory mucous membrane gained access to the lower passages via an inflamed mucosa: a degree of collapse of the affected area of lung was recognised as an early feature and it is thought that this deficient aeration was responsible for localisation of the pneumonia to that particular lobe or segment.

5. Response to sulphonamides was excellent. Complications were rare. Mortality rate was 0.55%.

6. A striking feature was the high incidence of associated jaundice (6.5% of the pneumonias). Possible explanations for this are discussed.

7. Such an epidemic is less likely to occur when barrack rooms are divided into cubicles, each accommodating two or three men.

8. A modern method of nursing younger pneumonia patients is advocated, encouraging moderate exercise to abolish lobular collapse and so eliminate the prospects of chronic pulmonary disease.

9. Nomenclature of the minor forms of pulmonary inflammation requires revision and standardisation.

10. The segmental or lobular distribution of this pneumonia is stressed. Anatomy of the tracheo-bronchial tree and broncho-pulmonary segments is discussed with special reference to nomenclature. Dissection and bronchographic studies, conducted with a view to clarifying certain differences of opinion on anatomy, especially of the upper lobes, are described and illustrated.

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