recurrences were seen in some of the cases, and each recurrence was only temporarily controlled by hormone treatment.

As the work progressed it became clear that, while acute symptoms were being controlled temporarily, the condition of several of the patients was definitely worse than before treatment, and that reaction was becoming more common and more severe. The investigation was stopped and the position assessed. Of the 16 patients, six appeared to have benefited (all of these had only one or two short courses); in four the results were doubtful (these had one to four courses, average three); in the other six the results were regarded as bad (these had between one and nine courses, average five). All attempts to improve the results by smaller total dosage and tapering off the dose failed.

These results indicated that the hormone treatment of lepromatous reaction is attended by considerable danger of aggravating both the complications and the underlying disease, and is therefore contraindicated. It does more harm than good.

Other Complications

One case of apparently leprous arthritis, which previously had often occurred, was apparently cured by one short course of hormone treatment.

One case of leprous elephantiasis of the legs showed marked temporary improvement followed by relapse, and further courses were followed by general reaction; the result was therefore bad.

One tuberculoid case of leprosy (the only one in the present series) came in the phase of acute tuberculoid reaction with marked inflammation of the skin lesions, of the affected mucous membranes, and of the nerves. One short course of hormone treatment brought considerable alleviation of symptoms, but further courses were regarded as undesirable in this localized and usually benign form of leprosy because of the danger of abolishing the cellular reaction which localizes the disease in such cases.

General Conclusions

While the acute manifestations of leprosy can be very readily controlled by hormone treatment, there is a grave danger of aggravating the underlying disease, particularly in those receiving repeated short courses of treatment, and even of aggravating the particular symptoms to alleviate which the hormone has been given. Thus the early results are good, and the late results too often bad.

Attempts to minimize the bad late results by modified dosage and by energetic chemotherapy during hormone treatment have met with little success. Nevertheless, in two complications of leprosy and its treatment, both of them serious, the results are striking and are usually attained with such small doses that hormone treatment is fully justified. These two complications are: (1) sulphone sensitivity, with drug fever, dermatitis, and hepatitis, and (2) acute and subacute leprous eye inflammation, in which condition the local use of cortisone appears to be effective and safe, and is to be preferred to injection.

Apart from these two conditions the use of hormone treatment of leprosy is usually contraindicated. In some cases of acute neuritis, carefully selected as showing only mild residual leprous infection after chemotherapy, one short course might be justified, but other methods of treatment appear preferable.

I am greatly indebted to Nyegaard and Co., of Oslo, Norway, for free supplies of their "nyco" brand of A.C.T.H., with which the work was started; and also to the Medical Research Council for supplies of A.C.T.H. (Armour) and cortisone (Merck). All three preparations appeared to be fully active.

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PNEUMONIAS ARISING DURING THE COURSE OF UPPER RESPIRATORY INFECTIONS

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During the period from August, 1950, to May, 1951, 393 Service men from Shorncliffe Camp reported sick with coryzal symptoms. Their ages ranged from 17 to 44. Of these cases 126 were referred to the Shorncliffe Military Hospital either because they were unfit for duty for longer than 48 hours or because they were suspected of having pulmonary complications. Of those referred, 48 were found to have abnormal chest radiographs: the seasonal incidence of these opacities is shown in Table I.

TABLE I.—Seasonal Incidence

	Virus Pneumonias	Other Opacities	Other Upper Resp. Infections Admitted to Hospital
August, 1950 ..	0	2	4
September, 1950 ..	0	1	3
October, 1950 ..	1	4	8
November, 1950 ..	0	7	8
December, 1950 ..	1	3	4
January, 1951 ..	0	8	11
February, 1951 ..	0	7	13
March, 1951 ..	1	7	4
April, 1951 ..	0	4	12
May, 1951 ..	0	2	11

Three of these cases were diagnosed as virus pneumonias. They showed marked constitutional symptoms, with a pyrexia of above 103° F. (39.4° C.), which failed to respond to combined sulphadimidine and penicillin therapy. No pathogenic bacteria were isolated from their sputa, and they demonstrated a neutropenia in association with a raised erythrocyte sedimentation rate. Two developed cold agglutinins to titres of 1/2,560 and 1/512 respectively. Serological investigations in the third case, in which cold agglutinins were not found, were negative on the fourth and tenth days for influenza A and B, ornithosis, the lymphogranuloma venereum group, Q fever, and the streptococcus MG.

In the remaining 45 cases examination of the sputum revealed a mixed growth of throat commensals, and the course of the disease was unlike that of the virus group by reason of the minimal constitutional symptoms, the rapid response to sulphonamides, and certain other features that are described below. They were divided into two groups on the basis of the total neutrophil count, which was considered to be a more reliable guide than the total number of white cells. In Group I (17 cases) the neutrophil count was below 7,500 per c.mm., and in Group II (28 cases) it ranged from 7,500 to 25,000 per c.mm.

It is the purpose of this paper to discuss both groups, but especially Group II, with reference to aetiology and

clinical course and to suggest an approach to their treatment. The tables have been divided into the two groups on the basis of the neutrophil count.

History and Presenting Symptoms

There was no important difference in the distribution of presenting symptoms in either group. However, in Group I they tended to be less severe. The length of history averaged eight days in Group I and six days in Group II. Three clinical types of onset were recognizable: (1) *Catarrhal* (18 cases): characterized by catarrhal obstruction of the nares, cough, lassitude, sweating, and a dull frontal headache; (2) *bronchitic* (5 cases): characterized by a productive cough with the expectoration of between 1 and 3 oz. (28 and 85 ml.) of yellowish-green sputum a day and wheezing; and (3) *non-specific* (17 cases): characterized by myalgia, malaise, a trace of white sputum, and a generalized headache. Five cases could not be included in this classification; their symptoms included nausea and vomiting (four), diarrhoea (one), and sore throat (three). A proportion of both groups developed more specific symptoms referable to the chest (Table II).

TABLE II.—Incidence of Chest Symptoms

Symptom	Group I	Group II
Cough	15	24
Sputum	12	14
Wheezing	3	6
Pleuritic pain	7	14
Blood-stained sputum	3	3

Family History.—Three patients had a near relative who had suffered from pulmonary tuberculosis, and seven had a brother or sister who was described as "chesty." It would therefore seem that the family history is of little significance in this series.

Past History.—Sixteen cases gave a past history of chest disease, but none had suffered from a pulmonary infection in the year preceding admission. Those who stated that they had contracted bronchitis had remained at work throughout their illness. Table III shows the incidence of previous chest disease.

TABLE III.—Incidence of Previous Chest Disease

Disease	Group I	Group II
Pneumonia	2	4
Lymphocytic pleural effusion	2	0
Acute bronchitis (single attack)	0	3
Dry pleurisy	3	1
Recurrent winter cough	1	0

Physical Signs.—Signs confirming the presence of an upper respiratory infection were detected in 18 cases and included one or more of the following: catarrhal nasal obstruction, tonsillitis, conjunctivitis, and carious teeth. Four had tenderness over the superficial air sinuses, which, on x-ray examination, revealed no abnormality. The pyrexia, pulse, and respiration are shown in Table IV. It

TABLE IV.—Temperature, Pulse, and Respirations

	Group I	Group II
Temperature { Apyrexial	6	0
{ 98.4–101.5° F.	10	15
{ Over 101.5° F.	1	13
Pulse { 70–90 a minute	14	10
{ 91–110 „ „	2	14
{ Over 110 „ „	1	3
Respirations { 16–25 a minute	16	19
{ 26–30 „ „	1	6
{ Over 30 „ „	0	6

will be seen that there is a significant difference between the two groups, most of the patients with a pyrexia of above 101.5° F. (38.6° C.) falling into Group II. With the exception of three cases, all patients with a pulse rate of above

90 a minute are found in Group II, and the higher respiratory rates are also observed in this group. Table V indicates the distribution of the remaining physical signs. Those usually associated with consolidation were found only in Group II.

TABLE V.—Distribution of Physical Signs

Physical Signs	Group I	Group II
Tubular breathing	0	6
Whispering pectoriloquy	0	5
Bronchophony	0	3
Impaired percussion note	2	6
Impaired air entry	2	3
Pleural friction rub	2	3
Crepitations	10	28
Rhonchi	9	6
No physical signs	2	0

X-ray Appearances.—Postero-anterior and lateral films were taken on admission, 48 hours later, again when the patient became afebrile, and a fourth immediately before discharge. The lesions consisted of a patchy coarse mottling, merging at the periphery, with normal lung markings. Where two or more segments were involved the central portion tended to become more homogeneous, though never approaching the density of lobar pneumonia. Although there was no mediastinal displacement, in three cases in Group I the mottling assumed a triangular shape with the apex at the hilum, strongly suggesting segmental atelectasis. Pleural fluid was seen in 10 cases—in three as a trace at the costophrenic angle, in six in the interlobar fissures, and in one it was considerable. This last case, together with five others, occurred in Group II, the remainder in Group I. Table VI shows the segmental involvement, and the pre-

TABLE VI.—Segments Involved (Nomenclature after Foster Carter)

Right	Cases	Left	Cases
Lower lobe:		Lower lobe:	
Posterior basic	11	Posterior basic	12
Middle basic	4	Middle basic	2
Anterior basic	3	Anterior basic	4
Cardiac	6	—	—
Dorsal	6	Dorsal	5
Total	20	Total	23
Middle lobe:		Lingula:	
Anterior	3	Superior	1
Lateral	0	Inferior	1
Total	3	Total	1
Upper lobe:		Upper lobe:	
Apical	1	Apico-posterior	1
Postero-lateral	2	—	—
Antero-lateral	1	Antero-lateral	2
Total	3	Total	3

dominance of the lower lobes should be noted. The frequency of multisegmental involvement is shown in Table VII. Those cases in which more than one segment was involved fall largely into Group II.

TABLE VII.—Multisegmental Involvement

No. of Segments Involved	Group I	Group II
1	15	15
2	2	10
3	—	1
4	—	2

Pathology.—A specimen of sputum was collected by percussion postural drainage before the beginning of chemotherapy. Two further specimens were collected in a similar manner at daily intervals. All three were examined as direct smears by Gram and Ziehl-Neelsen stains, and by culture on blood-agar plates, and Dorset's egg and Löwenstein-Jensen media. White blood counts were made on the first and third days, and subsequently as indicated by the clinical condition. Specific agglutination tests were not performed

on Groups I and II. In a large proportion of cases in Group II cold agglutination tests proved negative on the tenth day.

Results

Table VIII shows the organisms isolated from the sputa of the two groups and from throat swabs taken from healthy soldiers. There was no significant difference in the distri-

TABLE VIII.—*Bacteriology of Sputa and Normal Throats*

Organism	Sputa		Normal Throat Swabs
	Group I	Group II	
<i>Pneumococcus</i>	3	4	17
Beta haemolytic streptococci	3	5	10
Alpha haemolytic streptococci	9	8	20
Non-haemolytic streptococci	6	8	18
Coagulase-negative staphylococci	5	9	1
Friedländer's bacillus	1	0	0
<i>Neisseria catarrhalis</i> and pharyngis group	14	21	19
<i>Candida albicans</i>	1	0	0
No. of cases	17	28	20

bution of the organisms in either group. Where pneumococci were isolated they occurred in a minority with other throat commensals.

The white-cell count, expressed as the absolute number of lymphocytes, monocytes, basophils, and eosinophils, did not differ from the normal as proposed by Osgood *et al.* (1939). The absolute neutrophil counts are shown in Table IX.

TABLE IX.—*Distribution of the Neutrophil Count*

Neutrophils per c.mm.	Group I	Group II
1,500 to 3,500	5	
3,500 " 5,500	5	
5,500 " 7,500	7	
7,500 " 10,000		13
10,000 " 25,000		15

Discussion

Before this series can be considered as a clinical entity certain other causes of pneumonia must be excluded. From the past history it seems improbable that we were observing cases of unresolved pneumonia associated with bronchiectasis. The clinical picture and sputum cultures likewise excluded pulmonary tuberculosis. The absence of a predominant growth of pneumococci in the sputum and the radiological appearances differentiated the cases from bacterial lobar pneumonia. Moreover, in Group I the neutrophil count remained within normal limits.

The presence of β haemolytic streptococci in the throats of healthy persons has been noted by Opie *et al.* (1921), while Heffron (1939) thought Friedländer's bacillus to be a common inhabitant of the nasopharynx. In view of the work of Conant (1945) the isolation of a few colonies of *Candida albicans* from one case was not regarded as of pathological significance.

Streptococcus viridans has rarely been incriminated in the production of pulmonary lesions. However, Solomon and Kalkstein (1943) described five cases, with four deaths. They emphasized the severity of the condition and the multilobar involvement. None of our cases fulfilled these conditions. We therefore believe the isolation of these organisms to be of no diagnostic importance. Although specific agglutination tests were not performed, the clinical picture was unlike that of the virus pneumonias summarized by Joules (1950). In Group I the constitutional symptoms were minimal, the temperature was normal or below 101.5° F. (38.6° C.), and the response to sulphadimidine or postural drainage alone occurred within 48 hours. There was a distinct contrast to the picture presented by the three cases of virus pneumonia. Group II, which was associated with a neutrophil leucocytosis and a prompt response to sulphonamides, was also totally dissimilar to the common virus pneumonias. In the more recent cases of the series, moreover, cold agglutinins were negative.

Verco (1949) summarized the radiological findings in virus pneumonias. He emphasized the frequency of hilar enlargement and the rarity of pleural fluid. None of our cases demonstrated hilar enlargement, whilst 10 were associated with pleural fluid. We do not, therefore, consider that we were treating cases of virus pneumonia.

The association of abnormal chest radiographs with upper respiratory diseases has been noted by many workers, including Clement (1935), Bowen (1935), Kornblum and Reimann (1940), Ackermann (1941), Kennedy (1943), Reimann (1947), and by Trail *et al.* (1944), who reported similar cases in Royal Air Force personnel.

The exact mechanism evoked in the production of these lesions was not fully appreciated till the publication of Ramsay and Scadding's paper in 1939. Fourteen of their 21 cases were of insidious onset, with minimal symptoms and few physical signs. The x-ray appearances consisted of "opacities of medium rather homogeneous density, with a tendency to coarse mottling." They concluded that the "localized consolidations" were due to aspiration arising in the course of upper respiratory infections. Scadding (1946, 1948, 1950) has further contributed to our knowledge of this condition and has recommended the term "aspiration pneumonia." He believes the aetiology in these cases to be a mildly infected segmental atelectasis.

Group I closely corresponds to this picture described by Scadding and others. From the description of Group II it will be seen that it differs from Group I more by its severity than in any essential feature. Factors that would influence the severity of an aspiration pneumonia would include the nature of the inhaled material, previous respiratory tract damage, and the duration of bronchial obstruction.

Examination of Table VIII shows that similar organisms were obtained in mixed culture from both groups. There is no reason to believe that they were of different pathogenicity.

Consideration of Table III shows that there is no significant difference in the incidence of past chest disease that might have impaired the defence mechanisms and promoted massive aspiration. We believe, therefore, that the greater severity of Group II depends upon a more sustained bronchial obstruction. Since aspiration of mucus need not take place concurrently with the onset of prodromal symptoms, the shorter history obtained in Group II does not invalidate this proposition.

Robertson (1943), considering the defensive mechanisms that combat aspiration, mentions the ciliary action of the bronchial mucosa and the cough reflex. Since the efficiency of the ciliary action depends on the integrity of the mucus layer (Proetz, 1934), upper respiratory infections may themselves be responsible for impairing the ciliary action and promoting aspiration. That aspiration does occur has been demonstrated by Amberson (1937), who introduced iodized oil into the noses of sleeping patients. The following morning he was able to demonstrate radiologically that it was within the bronchial tree.

Cornwell *et al.* (1930) injected a suspension of virulent pneumococci into the trachea of dogs. The severity of the lesion depended more upon the viscosity of the vehicle than the dose of the bacteria. This was probably due to a greater degree of atelectasis produced by the more viscid medium. They also noticed that when morphinization was utilized as a method of anaesthesia the lesions produced were of greater severity. As morphine is a powerful cough depressant its administration would prevent expectoration and so favour a greater duration of bronchial occlusion. From this evidence it would seem that the treatment of the "common cold" with Dover's powders or codeine tablets is unscientific and not without possible danger.

Fleming (1941) found that a raised carbon dioxide tension materially assisted the culture of pneumococci. Such a condition may be present in a collapsed segment of lung and assist the growth of the entrapped bacteria. This would be reflected in the neutrophil count, the pyrexia, the pulse

and respiration rates, and the extent of the radiological opacity. We believe that this explains the greater severity of Group II, and suggest that if treatment is inadequate the condition may progress to bacterial lobar pneumonia superimposed upon the absorption atelectasis.

Treatment

It should be more widely recognized that even such upper respiratory infections as the common cold can give rise to segmental atelectasis and that this may progress to a moderately severe pulmonary involvement. Treatment should be directed to the prevention of atelectasis by deep-breathing exercises, promotion of cough, and the avoidance of cough depressants. Particularly we deprecate the use of Dover's powders and codeine phosphate.

In Group I the aim of treatment must be to prevent sustained bronchial occlusion, and to this purpose the same measures will apply. In addition postural drainage should be instituted and expectoration assisted by simple inhalation therapy. If pleuritic pain is present, on no account must the respiratory excursions be restricted by the application of strapping. However, it is essential that this pain should be promptly relieved, because it may cause diminution of voluntary movement and suppression of the cough. For this purpose we have used pethidine, 100 mg. orally, or have sprayed the chest with ethyl chloride local analgesic. If these measures are employed early, and vigorously carried out, admission to hospital or additional chemotherapy will not be required.

In Group II chemotherapy will be required, for in addition to bronchial occlusion there are infection, consolidation, and a neutrophil leucocytosis. All our cases responded to sulphadimidine.

Conclusions and Summary

During the winter of 1950-1 393 Service men reported sick with coryzal symptoms.

Forty-eight of these presented with abnormal chest radiographs.

Forty-five cases were thought to be due to aspiration from the upper respiratory tract. They were divided into two groups on the basis of the neutrophil count. Group I had a normal white count, while Group II was more severe and was associated with a leucocytosis. Evidence is presented to suggest that Group II resulted from a more sustained bronchial occlusion and might have progressed to bacterial pneumonia.

A scheme of treatment has been described: in no circumstances must the cough reflex be depressed.

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FRIEDLÄNDER'S PNEUMONIA

A REPORT ON SIX CASES

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In 1882 Carl Friedländer described the large Gram-negative capsulated bacillus which he found at necropsy in several cases of pneumonia. He regarded it as the cause of lobar pneumonia. This was disproved, and for a time it was thought to be of no pathogenic significance. Weichselbaum (1886) established that it was the infecting organism of a small percentage of cases of pneumonia. Series of cases with reviews of the literature have been published by Solomon (1937), Bullowa et al. (1937), Perlman and Bullowa (1941), and Hyde and Hyde (1943).

We wish to draw attention to a type of pneumonia which is not affected by penicillin. In the absence of specific therapy, cavitation can occur in a few days. In spite of advances in treatment the prognosis remains serious.

Case 1

Five days before admission a man aged 57 got a severe wetting on the golf course. That night he felt shivery. The next day pain in the right side of the chest, worse on coughing, was noticed. The cough became more persistent and productive.

On examination he looked extremely ill, but was rational and co-operative. His temperature was 102.4° F. (39.1° C.). There was dullness at the right base, where numerous crepitations were heard, and the voice sounds were increased. The sputum was yellow and sticky. The initial chest film revealed consolidation, involving most of the right lung. The sputum yielded a growth of Friedländer's bacillus. This was stated to be streptomycin-sensitive but penicillin-resistant.

On his first day in hospital, before the result of the sputum examination was known, the patient was placed on sulpha-

FIG. 1.—Case 1. One year after acute attack. Multiple small cavities in right upper lobe. Surrounding fibrosis. Appearance substantially unchanged for 10 months.