INFLUENZAL PNEUMONIA:
CAUSATION AND TREATMENT *

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SYNOPSIS

The more recent epidemics of influenza have been characterized by a high death-rate among people aged 55 and over, and a decreased mortality in the lower age-groups. The danger appears to lie largely in the pulmonary complications associated with this disease; the declining mortality among the lower age-groups may be a result of the chemotherapeutic agents now available.

Two main varieties of complication of the lower respiratory tract are discussed—influenzal bronchitis and bronchiolitis, and influenzal pneumonia. The major part of the study is devoted to a description of influenzal pneumonia, its bacteriology, and methods for its diagnosis, treatment, and prophylaxis. The need for early treatment is particularly emphasized. The comparative value of various antibiotics is discussed, and courses of antibiotic and sulfonamide therapy, adjusted for conditions arising from different causative organisms, are suggested.

Influenza is still a disease with an impact upon the community which can be measured in terms of mortality; yet, compared with the historic outbreaks of 1889-90 and 1918, recent epidemics have been mild. However, even recent epidemics severe enough to affect 5%-10% or more of the population have been accompanied by an appreciable rise in the death-rate, particularly in the elderly and in spite of the general use of chemotherapeutic agents. Thus the influenza A epidemic in Holland in 1948-9 caused about 2,200 deaths during an eight-week period; moreover, an occasional outbreak with an alarming mortality has been experienced, such as that in 1951 in the town of Liverpool, which suffered an even greater mortality than in 1918. In these exceptionally virulent recent epidemics, however, almost all the deaths have occurred among those aged 55 or over, whereas as many as 50% of the deaths in 1918 occurred in the age-group 20-40. A steady fall in the death-rate in the younger

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members of the community affected by influenza has been experienced since 1938, which suggests that chemotherapy may be capable of influencing the course of the complications in these cases.

The authors' experience of recent epidemics of influenza has convinced them of the need for awareness of the therapeutic problem which may be posed, and which contrasts with the relative ease of therapy of ordinary primary bacterial pneumonia and bronchopneumonia. Also, it is important that the general practitioner should be aware of the danger signals of severe or fulminating influenzal pneumonia, particularly because so much depends upon the speed with which therapy is begun. The relative infrequency of the fulminant case is often a source of additional difficulty when, as during an epidemic, so many patients may demand attention that daily visits become impracticable. The purpose of this article is to summarize the facts known about the pulmonary complications of influenza, and to suggest therapeutic regimes which should be pursued.

Clinical Varieties of Bronchial and Pneumonic Complications of Influenza

There are two main varieties of complication of the lower respiratory tract which require recognition and differential diagnosis: (a) influenzal bronchitis and bronchiolitis; (b) influenzal pneumonia.

*Influenzal bronchitis and bronchiolitis*

Influenzal bronchitis or broncho-bronchiolitis is seen at all ages and is a serious condition only in the elderly or in those already afflicted by some chronic disease of the respiratory tract or of the heart. Mucopurulent bronchitis is thus suspected when the initial dry cough of influenza becomes productive and is accompanied by signs in the chest, such as generalized rhonchi or wheezing, and perhaps by a mild degree of dyspnoea. It varies greatly in severity but usually clears up promptly in patients with previously healthy lungs once the temperature is normal.

Bronchiolitis is suspected if the temperature remains elevated on the third or subsequent days of an attack of influenza, if the patient coughs frequently and has a mucopurulent sputum, and if examination of the chest reveals patches of fine râles at one or both lung bases. Such patients become dyspnoeic, but not excessively so; they do not complain of pleural pain, and x-ray of the chest may either reveal no abnormality or else show increased bronchial markings. Recovery even without antibiotics is usual, although several days may pass before the abnormal signs disappear from the chest. If, however, the patient has pre-existing disease such as bronchi-
ectasis or severe emphysema, or cardiac disease such as hypertensive or valvular heart disease, bronchiolitis may cause death. Certain, also, of the deaths from acute influenza which occur in elderly people are probably due to bronchiolitis rather than to an actual pneumonic process.

Influenzal bronchitis and bronchiolitis, although certainly related to influenza virus infection, are probably also accompanied by invasion by pathogenic nasopharyngeal bacteria. Throat washings or sputum taken from patients during the early days of illness constantly reveal influenza virus. On the other hand, cultivation of the sputum for bacteria usually reveals one or more common pathogens of the respiratory tract. A preponderance of *Haemophilus influenzae* is found in the most seriously affected patients and the pathogenic role of non-encapsulated *H. influenzae* in broncho-bronchiolitis seems to have been established. The fibrinous necrotizing variety of tracheo-broncho-bronchiolitis caused by pyogenic cocci (most often *Staphylococcus aureus*) is always associated with broncho-pneumonia and is described below.

**Influenzal pneumonia**

Consolidation of the lungs either may develop during the course of the febrile phase of influenza virus infection, or may follow after an interval of time which may be brief or may last for several days, and during which the patient may have made an apparent recovery from the primary attack of influenza. There is no one constant and invariable clinical picture to which the term "influenzal pneumonia" can be applied; nor can the majority of cases of influenza with consolidation be certainly distinguished from severe cases of pneumonia occurring at times when influenza virus infection is not prevalent. Nevertheless, the fulminant cases present a characteristic picture and, if for no other reason than to draw attention to these patients, the term "influenzal pneumonia" deserves to be retained.

A pneumonic process is suspected when the patient convalescing or convalescent from influenza again becomes febrile, complains of cough, dyspnoea, and pain of pleural type. Sputum, which may be mucopurulent, purulent, or frankly bloodstained, is usually evident at this stage. A leukocytosis is also frequent. Physical signs in the chest are those of frank consolidation, with dullness often suggesting a small effusion, bronchial breathing, and abundant râles. Râles may also be evident in other areas where dullness cannot be elicited, and x-ray of the chest usually shows scattered areas of relatively dense opacity resembling a bronchopneumonic process. Occasionally, and particularly in cases of pneumonia following several days after the original attack, the consolidation may be lobar in type and may affect one or several lobes.

The supervention of consolidation of the lungs in a patient during the acute stage of influenza is more difficult to recognize. In the most
fulminant of all cases, dyspnoea, bloody sputum, cyanosis, and a collapsed state of the circulation may be the main findings. Death in such cases may supervene within 24-48 hours. It is a striking fact that pleural pain may not be in evidence and that the physical signs of consolidation are frequently obscured. If pain is present, it may be retrosternal in location and experienced chiefly during coughing. Râles, which are scattered diffusely over the chest but are particularly numerous towards one or other lung base, weak breath sounds or patchy bronchial breathing, and relatively slight impairment of percussion are the only findings at first. X-rays, however, show patches of mottled opacity suggesting a broncho-pneumonia. The leukocyte count may show a leukocytosis but sometimes there is a leukopenia. Later, if the patient survives, the classical signs of consolidation may appear, and râles may lessen, except in the areas chiefly affected. However, other complications such as pleural effusion, lung abscesses, or pneumothorax may develop and cause an alteration in the signs. In any event, influenza which leads without pause into pneumonia is frequently a severe disease with a slower response to therapy than the variety of pneumonia which occurs some days after recovery from the influenza. It is important to recognize that the clinical picture and the response to treatment depend upon the bacterial species concerned in the lung invasion.

**Bacteriology of Influenzal Pneumonia**

Bacteria believed to be concerned in the pneumonic process are found in the sputum in cases of influenzal pneumonia, and all fatal cases of influenza, except possibly some of those in the aged or enfeebled, are attributable to bacterial action. Pneumococci predominate in the spu­ta of patients who develop pneumonia some days after the onset of influenza. Staphylococci of the ordinary pyogenic variety occur in a lesser percentage of cases, but particularly in cases of pneumonia concurrent in time with the influenza virus infection. In view of the normal relative infrequency of staphylococcal pneumonia, and the fact that pneumococcal infection is the predominant cause of ordinary non-influenzal pneumonia, attention deserves to be drawn to the increase in staphylococcal infection which occurs during an epidemic of influenza. Thus 104 (80%) of 130 cases of pneumonia in Sheffield in non-influenzal periods between 1947 and 1951 yielded pneumococci in the sputum. During these periods *Staph. pyogenes* was found in the sputum in only seven instances. During two periods of prevalence of influenza virus A-prime infection—January to March 1949, and January to March 1951—166 cases of pneumonia yielded 114 instances (68%) of pneumococcal infection. Also, during these influenzal periods 33 patients yielded staphylococci either alone or with pneumococci.
During the 1949 influenza A epidemic in Rotterdam, Bruins Slot\(^1\) observed 37 cases of pneumonia, 17 of which were caused by \textit{Staph. aureus}. Fifteen instances of serologically confirmed influenza virus A-prime infection were found among the latter cases. Similarly, one of us (J.M.) has personally observed 25 cases of staphylococcal pneumonia unassociated with primary septicaemia, 18 of which were superimposed on influenza virus A or B infection. It is the authors’ opinion, therefore, that the occurrence of severe staphylococcal pneumonia, characterized pathologically by a fibrinous necrotizing inflammation of the tracheal and bronchial epithelium and purulent bronchopneumonia, is a sign of the existence of influenza virus A or B infection.

Unlike experience in 1918, pneumonia caused by the haemolytic streptococcus is relatively rare at present, and organisms such as \textit{Klebsiella pneumoniae} have not been more prominent at times of influenza epidemics than during normal periods.

The exact role of the influenza virus infection in relation to influenzal pneumonia is difficult to discern. There is no doubt of its occurrence but no one can be sure whether the virus infection is limited to the pharynx or whether, as the authors believe, epithelial lesions occurring in the trachea and bronchi are due to virus rather than bacterial action. It is unlikely, except in cases of severe and fulminant pneumonia, that the influenza virus plays a role in continuing pulmonary infection, and thus treatment directed towards the bacterial component is usually adequate. It is by no means certain, however, that this conclusion is valid for particularly virulent epidemics such as that of 1918. The potentially pneumotropic property of influenza virus cannot be ignored, and some strains of influenza virus may multiply more readily in the human lung than others. Since the first isolation of the influenza viruses in 1933, however, few variations in pulmonary complications in different epidemics which could be attributed to the virus have been encountered. The complications of influenza B appear to be the same as those of influenza A.

\section*{Diagnosis}

Clinical methods alone may fail to differentiate many cases of influenzal pneumonia because the picture often resembles that of ordinary bacterial pneumonia. However, as these patients are also those who respond most readily to therapy with, for example, penicillin or the sulfonamide drugs, no harm results from incomplete diagnosis. The same cannot be said for the cases of influenzal staphylococcal pneumonia, or, indeed, for severe instances of any variety of influenzal infection. The problem of

\footnotetext[1]{Bruins Slot, W. J. (1950) \textit{Ned. Tijdschr. Geneesk.} 94, 3438}
recognition of the latter cases is, however, a considerable one, first, because such patients should be treated at as early a stage of the disease as possible, and second, because bacteriological assistance and facilities for radiological examination may not be available in the home. The clinical features which should suggest the possibility of pneumonia in patients with either the symptoms of influenza (headache, shivering, myalgia) or a history of recent recovery from influenza are as follows:

(1) **Age.** The probability of complications increases at ages over 50, and patients of 60 and over require to be watched and repeatedly examined. Nevertheless, some cases of the most fulminant form of staphylococcal pneumonia occur in young and middle-aged adults, so that no age-group can be considered as exempt from this complication.

(2) **Previous history of a staphylococcal infection,** such as a furuncle or skin infection in the patient or other members of the family.

(3) **Previously existing chronic disease,** such as diabetes, bronchiectasis, chronic bronchitis, emphysema, and all forms of heart disease.

(4) **Persistent high fever** on the third or fourth day of the disease.

(5) **Occurrence of dyspnoea, cyanosis, chest pain, and a productive cough.** Any or all of these point to the probability of a chest lesion, but the degree of subjective dyspnoea may be slight compared to the rise in respiration-rate, and chest pain may be central in situation rather than in the usual lateral location of a pleural pain. Sputum may not be raised at all in the most gravely ill patients, but a purulent, blood-streaked, or frankly bloody sputum are usual in any of the varieties of influenzal pneumonia, so that the patient should always be asked to cough in the presence of the doctor in order to permit inspection of any material which may be expectorated.

(6) **Frank signs of consolidation** may exist, but cases of influenzal pneumonia may exhibit extensive radiological changes although the clinical signs are equivocal or even suggestive only of a diffuse bronchitis. Patches of weak breath-sounds and abundant râles may therefore be of greater significance than the absence of bronchial breathing or of dullness.

(7) **Existence of a leukocytosis in excess of 14,000 total leukocytes per mm$^3$** is in favour of a bacterial complication. A leukopenia may, however, be found in severe bacterial influenzal pneumonia, so that, as with so many of the other signs, a negative finding does not rule out the possible existence of a pneumonic process.

(8) **Occurrence of a feeble rapid pulse, low blood pressure, cold extremities, and sweating** may indicate peripheral circulatory failure which occurs in the severest clinical grades of pneumonia. Thus, the patient with influenzal-staphylococcal pneumonia may resemble superficially a case of myocardial infarction with resultant pulmonary oedema and shock-like state.
In addition to the observation of purely clinical findings, the most helpful step is to examine the sputum bacteriologically. A simple film of sputum stained by Gram’s stain will often reveal the existence of staphylococcal pneumonia, for in this condition vast quantities of staphylococci are nearly always present. Cultivation of the sputum is, however, essential if the predominant organism is to be identified, and particularly if the sensitivity of the bacterial species to antibiotics is to be ascertained. Fortunately, the majority of cases of staphylococcal pneumonia are still initially caused by penicillin-sensitive organisms, although resistant strains may appear after therapy with penicillin. Film and cultivation will also reveal pneumococci, or haemolytic streptococci, or *H. influenzae*. The use of special selective media for the latter, or of mouse-inoculation for the detection of pneumococci, is not likely to be of assistance from the standpoint of therapy although it is necessary for exact bacteriological diagnosis.

Radiological examination is a further essential step in the differential diagnosis of influenzal pneumonia and will, of course, be carried out as a matter of routine in hospital practice. For the benefit of practitioners who are unable to obtain facilities for radiological examination, or who are treating the patient at home, it may help to point out the existence of relatively severe cases of influenzal bronchiolitis which may closely resemble pneumonic cases for a brief period. The essential difference is that the bronchiolitic cases (who show no gross radiological abnormality) may undergo rapid remission of symptoms and signs. Such patients should probably be regarded as pneumonic cases, if a radiological examination is unobtainable, and should be treated accordingly.

Finally, the sequelae of influenzal pneumonia which include lung abscess, pleural effusion or empyema, various degrees of atelectasis, and even rarely pneumothorax, require careful attention in spite of previous chemotherapy. Every effort should be made to obtain the admission to hospital of patients whose conditions fail to resolve within a reasonable period of time (5-10 days), as the differential diagnosis from chest disease such as pulmonary tuberculosis or bronchial carcinoma requires radiological, and possibly bronchoscopic, investigation.

**Treatment**

The essential difficulty in the treatment of influenzal pneumonia is the need to begin therapy as early as possible, which in practice means that it will often have to be started before a bacteriological diagnosis concerning the causative bacterial species is available. Unfortunately, the standard dosage and method of treatment for bacterial (pneumococcal) pneumonia—which, in most countries, is based upon penicillin with or
without the addition of sulfonamides—has proved regularly effective only in the pneumococcal types of influenzal pneumonia. The staphylococcal cases require much more energetic and early treatment with penicillin and, in the authors’ experience, may even then prove resistant to therapy. Experience with the various antibiotics derived from the *Streptomyces*—chloramphenicol, aureomycin, and oxytetracycline—is still inadequate for a firm statement to be made concerning their merits in comparison with penicillin. In infections with *H. influenzae*, however, these drugs are effective, and they should also be used in cases infected with penicillin-resistant strains of *Staph. pyogenes*. In any event, it is necessary to stress the fact that the sulfonamide compounds such as sulfadiazine and sulfadimidine, if used alone, are effective in a much lower proportion of cases of influenzal pneumonia than with ordinary bacterial pneumonia. Their routine use will therefore cause delay in the institution of therapy in precisely those patients whose need for effective antibiotic treatment has existed from the onset of the pulmonary complication. For this reason, their routine use is deprecated. It is doubtful whether much is gained by combining sulfonamide therapy with other agents such as penicillin. On the other hand, combined antibiotic therapy with penicillin and streptomycin may perhaps avoid the emergence of penicillin-resistant staphylococci, although experience with such therapy is still inadequate. Combined therapy with other antibiotics is as yet experimental in nature, and recommendations cannot be made at present. There is no particular advantage to be gained by administering antibiotics by inhalation.

The following regime is suggested for patients in whom a definite clinical diagnosis or a presumptive diagnosis of influenzal pneumonia has been made. After the initial clinical examination, aided when possible by radiological examination, the following steps should be taken:

(1) Obtain specimen of sputum, stain by Gram’s method a sample thoroughly washed in physiological saline, and set up cultures on blood-agar. Rapid antibiotic-sensitivity tests on the predominant species in the sputum can be carried out by cultivating the sputum on plates with discs containing different antibiotics. If no sputum is available but the patient presents the aspect of a mild or moderately ill case of pneumonia, treat as for pneumococcal infection. If the patient is severely ill, treat as for staphylococcal infection.

(2) If pneumococci predominate in the sputum, treat with penicillin by intramuscular injection of 50,000-100,000 units of ordinary aqueous penicillin every four hours (daily dosage 300,000-600,000 units). If preferred—but only in mild or moderately ill cases—procaine penicillin may be given as 300,000 units intramuscularly twice daily (600,000 units per day), either

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1 Oxytetracycline is the non-proprietary name for Terramycin (see *Chron. World Hlth Org.* 1953, 7, 41).
alone or with 100,000 units of ordinary sodium penicillin. Treatment should be continued for at least 14 days.

(3) If staphylococci predominate in the sputum and the patient is severely ill, penicillin may be given by intramuscular injection at the rate of 1,000,000 units initially, followed by 500,000 units every four hours. If the patient is desperately ill, 1,000,000 units every two hours may be given for the first 12 hours, followed by a lower rate of dosage. This regime may be modified in staphylococcal infections with a lesser degree of clinical severity, but the daily dosage should still be of the order of 1,000,000-2,000,000 units.

(4) If no bacteriological facilities are available, the clinician should be guided by the response to treatment with penicillin at the standard dosage of 50,000-100,000 units intramuscularly every four hours, but should use the higher scale of dosage for all fulminant cases.

(5) If penicillin-resistant staphylococci are reported in the sputum, or the patient fails to respond within 72 hours of initiation of therapy, then a change should be made to a different regime. Oxytetracycline or aureomycin is preferred by the authors because oral therapy (which may be impracticable in severely ill patients who have difficulty in swallowing) can be supplemented by intravenous therapy with the same agent. The requisite daily dosage is still under study, but 4 g daily by mouth should be given for at least 10 days, with lesser daily dosage if intravenous injections are substituted. Treatment may have to be given for at least three weeks or more. It seems unlikely that any advantage will be obtained by combining either of these antibiotics with penicillin or streptomycin.

(6) *H. influenzae* infections should be treated with penicillin in high dosage (4,000,000 units daily in adults) or preferably with chloramphenicol, aureomycin, or oxytetracycline at the rate of 0.5 g every six hours.

(7) Infections with *K. pneumoniae* should be treated with streptomycin, either alone or with the addition of sulfadiazine or sulfadimidine. If no response is obtained, chloramphenicol, aureomycin, or oxytetracycline may be tried, but the response cannot be predicted.

(8) *Streptococcus haemolyticus* infections may be severe. Optimal treatment has not been studied, but the authors suggest the same dosage of penicillin as in staphylococcus infections.

Ancillary treatment in addition to antibacterial therapy is obvious. Oxygen is of chief value in patients with abundant bronchial secretion and deep cyanosis. It is unnecessary as a routine measure. Peripheral vascular failure may be helped by the use of cortisone and later of adrenocorticotrophic hormone but antibacterial treatment should also be given, and experience is so far totally inadequate for firm recommendation. The use of drugs such as digitalis is not, in the authors’ view, of critical importance unless auricular fibrillation or congestive heart failure co-exist.
In the latter instance, and also in patients with abundant bronchial secretion, diuretics of the mersalyl (mercury salicyl-allylamide-o-acetate of sodium) variety may assist.

**Prophylaxis**

Mass prophylaxis of bacterial complications in influenza is impracticable and probably undesirable, because of the possible encouragement of resistant bacterial species or of superinfections with organisms such as fungi, especially *Candida albicans*. Prophylaxis is more reasonable, however, in patients with influenza who are known carriers (nasal or skin) of staphylococci, or who have had a recent infection with staphylococci, in diabetics, and in patients with chronic respiratory-tract or cardiovascular disease. Thus, patients with chronic bronchitis, bronchiectasis, or possibly chronic nasal sinusitis or otitis media, are candidates for the development of a superimposed bacterial infection of the bronchioli and the lung. It is difficult, at present, to lay down rules for the guidance of those who desire to attempt prophylaxis. Sulfonamide compounds are probably prophylactic against pneumococcal or haemolytic streptococcal infections. It is not these organisms, however, but staphylococci and *H. influenzae* which are most dangerous to the subjects already suggested above. Penicillin might be effective prophylactically against staphylococci but there is always the risk of causing the emergence of penicillin-resistant strains. Penicillin in normal dosage is prophylactically ineffective against *H. influenzae* infections. Less risk appears to exist in the case of chloramphenicol, aureomycin, and oxytetracycline at a level of 2 g orally a day, and these agents are capable of exerting an action against all the bacteria concerned. If used, however, these agents should be given for a period not in excess of a week, and careful watch should be kept for possibly harmful effects, especially with chloramphenicol in regard to the development of inhibition of the bone-marrow.

**Mobilization after Uncomplicated Influenza**

The elderly patient convalescing from influenza is in a debilitated and weakened condition. The respiratory-tract epithelium has probably not returned to a completely normal state until three to four weeks after the acute illness. It is therefore desirable to urge caution before return to normal occupation, and avoidance of exposure to inclement weather or overcrowded public places.
RÉSUMÉ

Les récentes épidémies de grippe ont été moins meurtrières que celles de 1889-90 et 1918-19. Elles ont affecté cependant 5-10% de la population et ont causé une mortalité élevée dans certaines régions, en particulier aux Pays-Bas en 1948-49 et à Liverpool en 1951. Ce sont les sujets âgés de plus de 55 ans qui en ont été les principales victimes, alors qu’en 1918, 50% des décès avaient été observés dans les groupes d'âge de 20-40 ans. La baisse de la mortalité chez les jeunes adultes, constatée depuis 1938, suggère que la chimiothérapie a atténué peut-être la gravité des complications grippales. Quoi qu'il en soit, le praticien doit rester sur ses gardes et se méfier des formes foudroyantes de pneumonie grippale; pour être efficace, le traitement de telles complications doit être entrepris avec une extrême célérité. L'objet de cet article est de résumer les connaissances sur les complications pulmonaires de la grippe et de proposer des thérapeutiques adéquates.

La bronchite et la bronchiolite d'une part, la pneumonie grippale d'autre part sont les principales complications. Les premières ne sont graves que chez les sujets âgés ou affectés de maladies chroniques de l'appareil respiratoire ou du cœur. La pneumonie grippale, au contraire, peut prendre une allure foudroyante, chez les malades de tous âges, et entraîner la mort en 24-48 heures. Bien qu'elle ne soit guère définie par des caractères cliniques spécifiques, elle doit garder une individualité diagnostique, en raison de la gravité qu'elle peut présenter.

Les auteurs énumèrent les types bactériens que l'on trouve dans les expectorations et qui paraissent intervenir dans le processus pathologique (pneumocoques, staphylocoques, *H. influenzae*, *Kleb. pneumoniae*). L'augmentation de la fréquence des staphylocoques dans les affections pulmonaires, en période d'épidémie de grippe, est à souligner. Il est difficile de préciser le rôle que joue le virus grippal dans la pneumonie grippale. On ne sait si l'infection virale est limitée au pharynx ou si, comme le pensent les auteurs, les lésions épithéliales de la trachée et des bronches sont dues au virus plutôt qu'aux bactéries.

Le traitement est compliqué par le fait qu'il doit être entrepris aussitôt que possible, c'est-à-dire souvent avant que le diagnostic bactériologique soit posé. Le traitement standard de la pneumonie bactérienne par la pénicilline associée éventuellement aux sulfamides n'est efficace à coup sûr que contre la pneumonie grippale due aux pneumocoques. Les formes à staphylocoques demandent un traitement par la pénicilline plus énergique et plus précoce et peuvent même, d'après l'expérience des auteurs, y être réfractaires. Le chloramphénicol, l'auréomycine et l'oxytétracycline (Terramycine) ont donné des résultats satisfaisants dans les infections à *H. influenzae* et ont été utilisés avec succès dans les cas d'infection à *Staph. pyogenes* résistants à la pénicilline. Les sulfamides sont moins actifs dans la pneumonie grippale que dans la pneumonie bactérienne ordinaire. Leur emploi dans les cas courants ne ferait que retarder l'application des antibiotiques. L'adjonction de sulfamides aux antibiotiques ne paraît pas augmenter l'efficacité du traitement. Les auteurs décrivent en détail la posologie qu'ils recommandent pour le traitement de la pneumonie grippale dans les cas où le germe infectieux a été identifié, ainsi que dans les cas où, faute de laboratoire permettant de préciser la nature de l'infection, il faut parer au plus pressé. Lorsque les pneumocoques dominent dans les crachats, on donnera 300.000-600.000 unités de pénicilline aqueuse par jour, durant 14 jours au moins. Dans les cas graves, ou dans ceux où le staphylocoque est prédominant, la posologie sera de 1-2 millions d'unités par jour, par voie intramusculaire. L'oxytétracycline et l'auréomycine seront employées si des staphylocoques résistants à la pénicilline ont été décelés dans les crachats ou si après 72 heures l'organisme n'a pas répondu au traitement. Une thérapeutique d'appont — oxygène, cortisone, digitale, mersalyl — peut être indiquée suivant les cas. La prophylaxie n'est guère à conseiller, en raison du risque d'apparition de souches résistantes.