PNEUMONIA IN HOSPITAL*

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It is impossible, after dealing with cases of pneumonia in hospitals for over thirty years, not to start the subject with a backward glance and to realize how greatly the problem has changed in that time. I suppose that in my house physician days and for the next dozen years or so most pneumonias were treated in hospital, and amongst these were many cases in the first half of life. Now, and I speak with a view to learning something from my audience, I imagine that very many of these cases are treated at home and that only social difficulties, age and drug resistance or some other factor such as heart disease, make it necessary to send the case to hospital.

In the old days classification of the pneumonias was simple. Most of the younger ones were pneumococcal 'lobar' pneumonias and many of the old ones were bronchopneumonias. If influenza came along, then we had a crop of influenzal bronchopneumonias. Treatment was even simpler: the nurses did it during the day and hypnotics and nurses during the night. The main problem was to decide when the heart needed the support of digitalis and whether alcohol was required. As many of the older patients in the districts in which I worked in my youth were more than somewhat addicted to alcohol, a bellicosity and a tendency to delirium tremens was not uncommon and these tendencies were somewhat mollified by alcohol, though they became extremely rampant at night. Hypnotics were a problem particularly in such cases, in the cyanosed, and towards the crisis in lobar pneumonias, for none of these tolerated morphia well. Paraldehyde and bromide and chloral were chiefly administered and human restraint was often needed until these became effective. At one hospital we had a very stout and powerful night porter who might have been elected for this very purpose, and when things got out of hand in the ward an urgent message was sent to the front hall and then began one of the earliest advances in the treatment of pneumonias—like the advance of some heavy officer of the law against a malefactor. My Australian friends, when I was at the Brompton Hospital, used to recount the story of an athletic pneumonia who, before the crisis of his complaint, bolted across the lawns in front of the hospital, was brought down by a rugger tackle before he entered the main road, and brought back to make an uneventful recovery. Perhaps one can hardly refer to this as pneumonia in hospital. I personally, now very rarely see the old-fashioned lobar pneumonia with its sudden onset of malaise and shivering followed by chest pain, rusty pneumococcal sputum and dyspnoea; running a defined course of increasing toxemia for about a week and then terminating by crisis. When I was trying, in 1940 and 1950, to classify pneumonias by physical signs, sputum test, blood virus tests and X-rays, I only had one probable case out of 150 cases.2

To turn to more recent advances and to skip over the pneumococcal typing and appropriate serum injections for lobar pneumonia, we come to the drugs of the classes on which we still rely. First, the sulphonamides starting with sulphadiazine (1937), which was of little use in pneumonia, and then in 1938 sulphapyridine (M. & B. 693), that nauseous and cyanoxing drug which was effective against pneumococci and streptococci and which I think, first robbed pneumonia of much of its terrible reputation as 'the old man's friend.' At last the illness was cut short before it reached the toxic heights which had led to the mental aberrations to which I have referred and to the often inevitable ending in heart failure. Rashes, haematuria and a tendency to agranulocytosis were not infrequent with this drug and its toxic tendency and a wartime civilian population many an shift work in the factories, still made it necessary to treat many of the pneumonias in hospital. Sulphathiazole, with its less toxic tendency but still fairly marked depressant effect on the white cells and tendency to produce erythema nodosum was its successor. Sulphadiazine (1940), sulphasemethazine, sulphasomidine (1942), sulphasomide or Elkosin (1943), and sulphafurazole or Gantrisin (1947) have followed—increasingly effective drugs, the last two especially less toxic and less acetylated than their predecessors and yet somehow less regarded because of the advent of the antibiotics headed by penicillin, which we have had in fairly constant use since the end of the war.

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Fig. 1.—Four cases of right lower lobe consolidation of similar type but associated with different infections.

(a) Male—38 years. Influenza B secondarily infected with haemolytic streps and pneumococci. (CFT titre 1:2.)
(b) Male—33 years. Primary atypical pneumonia. (Cold agglutinin titre 1:512.)
(c) Female—34 years. Psittacosis pneumonia with enlarged right hilar glands.
(d) Male—21 years. Unclassified pneumonia following sore throat with staph. aureus in sputum.
Fig. 2.—Two similar fluffy and reticular bilateral consolidations.

(a) Male—60 years. Influenza A with secondary B. coli and Proteus Vulgaris infection.
(b) Female—38 years. Primary atypical pneumonia (Cold agglutinin titre 1 : 2048) and gross haemolytic anaemia (Hb. 40%).

Fig. 3.—Two "ground glass" shadows in right lower lobe.

(a) Male—21 years. Influenza B with secondary pneumococcal and strep. viridans infection.
(b) Male—36 years. Unclassified pneumonia in previously bronchitic subject.
I, personally, have never given up the sulphonamides and have used Elkosin, a most admirable drug, in conjunction with penicillin as the sheet anchor of my treatment of pneumonia during the last nine or ten years. My present practice is to start a patient on crystalline penicillin in doses of 500,000 units eight-hourly for three or four days, and after an initial dose of 2 g. of Elkosin I keep this drug going in doses of 1 g. four-hourly up to a total dose of 40 g. The sputum is obtained as soon as possible and examined for organisms and their sensitivity. One usually is already beginning to appreciate the presence of a resistant organism by the time it is signalled by the bacteriologist, but I find that the great majority of pneumonias will respond to this type of treatment.

It is of some comfort to me and to those like myself who have never forsaken the sulphonamides, to note the various compounds of penicillin and sulphanilamide which have come on the market. I use procaine penicillin in doses of 600,000 units, 12-hourly, in the latter half of what is usually about eight days of active therapy for a reasonably responsive pneumonia. It is more convenient for the nurses and more comfortable for the recipient, but I have not found it built up the penicillin blood level fast enough in the early stages. Indeed, for a very bad pneumonia or one in a feeble subject, I often start with 1,000,000 units of crystalline penicillin.

Before I leave the sulphonamides I must not forget to say a word about the long-acting varieties such as Orisulf (sulphaphenazole) and Lederkyn (sulphamethoxypridazine). There is no doubt that these are highly effective drugs in doses of 0.5 g. twice or even once daily, as will be seen from the table, and quickly produce and maintain a good steady concentration in the blood. At present I use them, only in conjunction with penicillin or other antibiotics, in twice-daily doses and for the milder pneumonias. As will be seen from the table the lowest concentrations of the twice-daily sulphonamides compare favourably with the comparable concentration of the four-hourly drugs, but the latter presumably show higher peaks of blood level soon after absorption. If with the long-acting sulphonamides we can give a really effective oral penicillin, the home treatment and prevention of pneumonia will be easy indeed, and especially if one thinks back to the dark days and nights that one remembers 30 years ago and less.

The other antibiotics, streptomycin, chloramphenicol, tetracycline, oxytetracycline and erythromycin, all have their place but I hold them in reserve until my bacteriologist colleague tells me that they should be used and then I use what is appropriate, generally in doses of 250 g. four-hourly. The one exception to these rules is streptomycin which I give in doses of 1 g. daily, and which I use together with penicillin from the start in pneumonias, often in old subjects, who are too ill to take sulphonamides.

I do not use neomycin because of its bad repute, and I have not yet found it necessary to use ristocetin or novobiocin for staphylococcal infections. I feel that it is important to rest these drugs and erythromycin for the resistant organism, particularly resistant Staphylococcus aureus, or we may be left in the near future without any adequate therapeutic weapon against such infections.

You may say to me at this point, 'Why are you blindly treating all the varieties of pneumonia by one method at the start? 'Surely you should differentiate between them and vary your treatment accordingly.' My answer to this is twofold. First, that one cannot lose time in waiting for bacterial tests; secondly, that all the staphylococcal infections are resistant to the antibiotics, sulphonamides and the problem in them is to prevent or kill off the bacterial secondary invader. Finally, that treatment is itself for this reason some means of differentiating between viral and purely bacterial pneumonias. The therapeutic problem in secondarily infected virus cases is...
different from treating purely bacterial pneumonias. One must strike the offender with the right therapeutic weapon under bacteriological guidance, give nature all the other support she needs and hope that all will be well. Oxygen, pain relievers, digitalis for those with heart disease, and most important, nursing care, are the main supports which in hospital we add to the drug therapy. There is no need to say much about these, but we do use tents for our anotic pneumonias and we do find that many of the older patients fibrillate rapidly during their pneumonias, but that as a result of their treatment, including digitalis, normal rhythm is restored by the time they leave hospital.

As to the virus pneumonias, the most numerous are the influenzal ones, A, A2 and B, in the various epidemics, and sometimes we have two varieties in one epidemic. In them the great menace is the superinfection of a Staphylococcus aureus infection which rapidly extends a terrible ulcerative bronchitis and bronchiolitis and a haemorrhagic destruction of the lung. It seems that in bad influenzal pneumonias the patient is, as it were, sensitized to the infection—the illness gallops forward, the Staphylococcus aureus is received with open arms though not with open bronchioles, and the patient is overwhelmed by the toxaemia within 48 hours. Fortunately, not many cases are as bad as this and with vigorous antibiotic therapy against the secondary invaders and usually with sulphonamides, oxygen and digitalis as well, the battle is slowly won.

But nearly all virus pneumonias run a longer course and fever than the average bacterial cases, sometimes for two or three weeks. If suppuration occurs then, of course, the fever is more hectic and more prolonged, but generally this is rare, and empyemases—which once used to cause at least of hang-over of fever and toxaemia in the wake of pneumonia—have practically ceased to exist. In my first week at Edgware (Redhill), early in 1938, I had nine empyemases in my bedded male ward; I have only had one, a sterile case, in my 70 or so beds in the past five years. I also only had one sterile empyema of the 159 pneumonias investigated in 1949 and 50, 12 years in which influenza was rife. It is easy sometimes to think that an empyema is developing in the influenzal pneumonias who have prolonged fever with dullness and very weak breath sounds at one base, but in these cases the inflamed lung tissue is often entirely free for the signs. In any pneumonia in which such signs persist over a long period and fluid is found on tap, it behoves us to think bronchial carcinoma underlying and to get a bronchoscopy done.

Even without an underlying carcinoma, a partial lobar aetlectasis may persist after pneumonia, particularly in older people, in those previously bronchitic, and in those enfeebled by the illnesses. Whenever a chest is slow to regain its normal expansion after pneumonia, breathing exercises are advisable and I would say that in an ideal world all convalescent pneumonics should do them, particularly diaphragmatic exercises.

The other two types of virus pneumonia, which I have met less often, are the type associated with a rising titre of cold agglutinins in the blood, the so-called 'primary atypical pneumonia of unknown aetiology,' and the ornithosis-psittacosis group. The first type, if one admits that is a single type, may not show very gross signs of consolidation but has an influenzal type of onset often with a very severe headache, shivering and malaise followed by chest pain. The conjunctivae may be injected and a severe haemolytic anaemia sometimes develops. The Hb. may fall quite quickly to 40 per cent. These cases are often sporadic and are at least as likely to appear, in my experience, in warm seasons as in cold. Psittacosis pneumonia is less frequent, but I have seen two cases in one family when the ban on parrot imports was lifted and I have picked up occasional cases by routine C.F.T. observations since then. Two such cases were encountered out of 159 pneumonias whilst C.F.T. observations were being made during 1949 and 1950. The initial malaise is often very severe, the headache troublesome, some photophobia often present and haemorrhagic sputum is not infrequent. Otherwise it is not difficult, apart from C.F.T.'s, to mistake these cases for influenzal infections.

All these virus infections of the lungs, and this is particularly true of influenza, seem to have a kind of two-pronged onset. First, the familiar symptoms of 'flu' with upper respiratory symptoms, shivering, general aching and extreme malaise and asthenia. Then a few days or so after these symptoms of general systemic infection, infection may settle in the lungs with a second rise of temperature, pleural pain, cough and sputum. I always feel that if our patients would not try to 'soldier on' during or soon after the initial infection, but would spend a week looking after themselves, we should see less of these dangerous virus pneumonias that cause so much anxiety. Probably nearly every virus pneumonia has some secondary infection.

One last point that has struck me in the younger and less complicated viral pneumonias is that these are more toxic and cyanotic than one would expect for the degree of lung involvement and for the amount of dyspnoea that they show. They are often remarkably hypotensive; the white count
too, is often not raised, but the leucocyte count in younger virus pneumonias almost always rises with secondary infection and the signs are modified in accordance with the type and extent of that infection. Some influenzal pneumonias secondarily infected with pneumococci will, for instance, give classical signs of lobar pneumonia. We have greatly changed the picture of the pneumonias by our treatment, but their classification is no easier, perhaps more difficult, than 20 years ago.

Apart from special tests, I have to admit that I find it difficult to say straight away if a case is of virus origin or not. In an influenzal epidemic it is easier because the cases do tend to follow a type. But even then cases occur often which look like viral pneumonias and yet cannot be proved as such by the C.F.T.s. It is tempting to say that many such cases are viral, especially if their white count is not raised, if their sputum does not contain pathogens and if they are slow to respond to sulphonamides and antibiotics. But if one does this after excluding rarer causes, such as choriomeningitis, varicella, Cocksackie and glandular fever viruses, one has to stipulate that there are unclassified viruses which affect the lungs—not, I think, an entirely unjustifiable assumption, and only recently two para-influenza viruses (HA1 and HA2) have been isolated.

79 out of my 159 tested cases were suspected but not proved, to have viral infections and were left unclassified. It is probable that in these, in a large proportion of pneumonias of the present day, a viral infection paves the way for pneumococci to develop. It is also even possible that in the presence of a viral infection the mouth of meningals acquire a degree of pathogenicity.

Radiologically partial consolidations in which the shadow is 'ground glass,' or in which reticular peribronchial thickening is associated with small fluffy shadows, is some evidence, though not conclusive, of virus pneumonia. The illustrations in the text underline the difficulties distinguishing recognized virus pneumonias from unclassified consolidations of the lungs.

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REFERENCES
2. JENNINGS, G. H. (1952), Brit. med. J., i, 123.