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Pneumonia in Decline

PNEUMONIA is a general term applied to inflammation of the lung whatever the cause. Although it may be a complication of drowning or of being caught in a burning building and inhaling smoke and gases, such cases are rare. Usually, when doctors speak of pneumonia, they refer to two common illnesses, lobar pneumonia and broncho-pneumonia.

Lobar pneumonia is characterized by an acute diffuse and rapidly spreading infection of one or more lobes of the lung. Broncho-pneumonia is characterized by a gradual spread of infection down the windpipe, along the bronchi and into the lung itself, it is therefore patchy and involves both lungs.

Lobar pneumonia is most commonly due to a single species of organism. Broncho-pneumonia is due to many, several of which may be present at any one time. In lobar pneumonia the bacteria are virulent invaders, often attacking a healthy man. In broncho-pneumonia, they tend to be the normal inhabitants of the mouth and throat against which the resistance of the body has fallen so that microbes, formerly harmless, become harmful.

Traditionally, diseases have been described by the effects they produce, as collections of symptoms, and changes in the tissues seen after death. The healthy lung is like a sponge. Normally it contains much air, but in pneumonia it undergoes the classic changes of inflammation except that they are seen in an exaggerated form because of the structure of the lung. It becomes red and engorged with blood so that the spaces that should contain air are filled with fluid. The affected area becomes useless making the muscular effort of breathing more strenuous. In a sense, part of the lung is drowned. The first definite reference to pneumonia in medical literature occurs in the writings of Hippocrates (c. 460–370 B.C.). In his definition of 'peripneumonia'—a term which persisted for two thousand years—were included all acute diseases of the chest accompanied by pain in the side. In the second century, Aretæus gave a clearer account together with excellent directions for the application of his remedies which remained in vogue until the beginning of this century; copious bleeding from both arms, purging, attenuant and diluent drinks, rubefacients containing mustard applied to the chest, alkaline substances such as soda given in decoction of hyssop, and, when the fever had abated, wine devoid of astringency.¹

Thomas Sydenham (1624–1689) gave the first clear account of the disease in English. Apparently he thought pleurisy was the same as pneumonia and called it "bastard peripneumonia" affecting "the stout and fat beyond adult life and those who were addicted to spiritous liquors, especially brandy".

The invention of the stethoscope by René Laënnec (1781– 1826) ultimately placed the concept of pneumonia, pleurisy and other pulmonary diseases on a sound basis. The physician could by percussing the chest, by listening with a stethoscope and by noting the great effort to breathe, state with accuracy how each lung was involved. By taking the patient's temperature, by counting his pulse and measuring his blood pressure and by listening to his heart, one can judge how much of the 'pneumonia' is due to infection and how much to blood pooling in the lungs because of the impaired efficiency of the cardiac pump.

With the great advances in bacteriology and immunology, the clinical descriptions and impressions were augmented by scientific definition. Pneumonias are now classified in terms of the bacteria or viruses that cause them. Not only does this give greater scientific exactness and definition to the disease with a much clearer guide to prognosis, but, even more important it makes possible the selection of the right drug for treatment.

The successful treatment of lobar pneumonia is certainly one of the great triumphs of modern medicine. Bronchopneumonia, with its many different bacteria, is not so readily amenable to treatment with any single anti-bacterial agent unless one is found which is effective against virtually all bacteria. Even so, only part of the illness would respond. The basic ill-health that allowed the broncho-pneumonia to develop would remain. It is perhaps something of an over-simplification to say that we nearly all die from broncho-pneumonia but certainly it occurs as a late complication of most diseases. Indeed, because of its quiet clouding of consciousness and gentle though lethal progress in the terminal stage of diseases such as cancer, it acts in some ways like a narcotic. Rather paradoxically broncho-pneumonia has been called the 'old man's friend' because of the way it causes the patient's life to draw quietly to its close.

The Therapeutic Attack on Pneumonia

OBAR PNEUMONIA has attracted more attention than broncho-pneumonia. The reasons for this are simple. If left untreated, the disease is characterized by a rapid and dramatic onset in previously healthy and middle-aged people. Left untreated many die, but in those who recover naturally, improvement is almost as dramatic as the development of the disease. Whenever spontaneous natural recovery occurs, through the development of specific immunity, it is probable that a similar reaction can be induced artificially. This provided a major stimulus to research. Moreover in direct contrast to broncho-pneumonia, lobar pneumonia is usually due to a single species of bacteria—the pneumococcus—so that the problem appeared to be relatively straightforward, more so than it, in fact, turned out to be.

Few infectious diseases have been studied in such detail as 'the pneumonias'. These investigations have provided much more information about the processes of bacterial invasion and resistance of the body than the study of any other group of diseases.

This fundamental work has increased understanding of the pathology of the pneumonias, but the great practical therapeutic advances sprang from systematic observation, from chance and from the methodical and routine screening of vast numbers of natural substances, extracts, known chemicals and others specially synthesized for the purpose. Much of the work was done on an entirely empirical basis. Almost without exception, the modern drugs for the treatment of pneumonia, or for the treatment of infection, achieved clinical recognition and acceptance long before their mode of action was known. Moreover, even when academic research has shown how the new drugs acted, attempts to replace the empirical search for new drugs by the application of theoretical principles have been disappointing.

The Immunological Approach to the Treatment of Pneumonia

In order to understand the immunological approach to the treatment of lobar pneumonia, it is necessary to consider a little more fully the natural history of the disease. The onset is sudden, with high fever; the patient is extremely ill because pneumococci liberate poisonous substances. In twenty to thirty per cent. of untreated cases, the temperature remains high and most of these patients die. Pneumococci multiply rapidly in the blood and fluid of the inflamed lung and may even invade the general circulation. In that event the outlook is grave. Moreover, the fluid flows by gravity from the diseased to the healthy lung, thus spreading the disease and increasing the area of drowned lung. But if the patient is going to recover naturally, the temperature falls suddenly on the fifth to seventh day of the disease and the patient, though still weak, feels remarkably better. This dramatic change is called 'resolution by crisis'.

Until the crisis the microbes are multiplying rapidly; at this stage the defence cells of the body are unable to attack them. But once the 'crisis' occurs, the whole process is reversed with the defence cells rapidly consuming the bacteria. Immunologists soon showed that 'resolution by crisis' was due to the formation of anti-bodies—immune substances—which appeared in the blood and became adsorbed onto the surface of the pneumococci. Once this happened, the defence cells of the body could engulf and digest them, a process known as 'phagocytosis'.

The next step was to inject pneumococci into animals to produce antibody and then to inject this antibody into man early in the disease. The pharmaceutical industry in the United States invested heavily in producing effective anti-pneumococci serum. Unfortunately, there are many different types of pneumococci and each requires its own antiserum. Antisera against thirty-two of these different types were made in bulk. Nevertheless, another great difficulty remained. In order to select the right serum, specimens of the sputum had to be taken from each patient so that the pneumococci could be 'typed'. Two thousand six hundred diagnostic laboratories were set up and rapid 'typing' methods devised, the aim being to get the appropriate serum into the patient as early as possible in the disease.

It was found that antibody produced in the rabbit was more

potent and more easily purified than antibody made in the horse. Millions of dollars were spent on producing immune rabbits, on serum research and on manufacturing equipment. The mortality from pneumonia began to fall. The achievement was therefore of some practical significance but unfortunately the sera were most effective in the milder types of lobar pneumonia and least effective in the most severe types. Although the practical benefits of this vast therapeutic effort were transient and relatively slight, the difficulties that were overcome were immense. Such a well-organised and brilliant attack on a disease has never been equalled.

Sulphonamides

For many years before this great immunological onslaught, research workers had been following the lead of Ehrlich, in searching for drugs that would kill bacteria without harming the host. For over half a century a major break had eluded them. But the year was now 1937.

Two years earlier Domagk's paper on "The Behaviour of Prontosil towards Streptococci" had been published from the laboratories of a German pharmaceutical firm. Subsequently Colebrook at Queen Charlotte's Hospital in London had shown that Prontosil could cure puerperal fever. Trefouël in Paris had shown the activity of Prontosil was due to sulphonamide. A British pharmaceutical firm was making and screening vast numbers of sulphonamides for anti-bacterial activity. One, sulphapyridine, was to become famous as M. & B. 693. It proved to be effective against pneumococci, meningococci, gonococci and other organisms. M. & B. 693 was so valuable in the treatment of pneumonia that the very expensive and, in terms of mortality, relatively ineffective serum therapy to which so much effort and money had been devoted, was outdated before it had ever become established. Although the amount spent on the discovery of M. & B. 693 had been great, it was comparatively inexpensive to manufacture and easy to use. Serum therapy had cost approximately £40 per patient and required highly skilled bacteriologists for typing pneumococci. The result was that M. & B. 693 was used world-wide, providing effective treatment at a greatly reduced cost.

Even though M. & B. 693 was a great advance, it was soon

to be superseded by newer and better drugs. The pharmaceutical industry turned them out in rapid succession, sulphadiazine, sulphathiazole, sulphadimidine, sulphafurazole, sulphacetamide and many others, designed for different purposes, such as the treatment of intestinal infections. As a result of this massive effort each new 'wonder drug' had a short life and slipped rapidly into history. With each new drug, however, the so-called 'therapeutic index' of the sulphonamides increased. That is to say, they became safer and more effective.

Just as the sulphonamides brought the age of pneumococcal antisera to an end, they in turn were soon to be replaced. The discovery of penicillin and the flood of new work on antibiotics was soon to bring a further revolution in the treatment of lobar pneumonia. The sulphonamides had been merely bacteriostatic; they prevented bacteria multiplying. Penicillin opened up the way to a new family of drugs, the antibiotics, many of which kill bacteria. Today the sulphonamides are seldom used in the treatment of pneumonia, although they are still of great value in other diseases.

Penicillin

The early story of penicillin is well-known, from the day the mould first fell upon a culture plate in Fleming's laboratory in Paddington, through the long and unsuccessful attempts to extract and purify the antibiotic, to the re-birth of the project in the hands of Chain and Florey.

In 1941 the leading pharmaceutical manufacturers in Great Britain formed the Therapeutic Research Corporation to solve the great problems to be faced in producing penicillin in bulk. *Penicillium notatum* was a mould which grew poorly, and had to be cultured on the surface of liquid medium. Moreover, the yield of antibiotic was greatly influenced by the composition of the media. Six hundred culture bottles were needed to provide sufficient antibiotic to treat one patient. The cost was astronomical. Because of wartime conditions, and the complexity of the problem, the American pharmaceutical industry was called in to help. They invested over $\pounds7$ m. on the development of penicillin production.

With a new mould, *Penicillium chrysogenum*, it was possible to develop a deep culture technique, using vast vats. Penicillin became very cheap and readily available to all in most of the countries of the world. Penicillin not only provided the greatest therapeutic weapon in the history of medicine, it opened up a new approach in the search for better drugs.* A great new research programme began resulting in the discovery of the broad-spectrum antibiotics.

Measuring the Benefit

Every doctor who has lived through the tremendous therapeutic advance of the last quarter of a century knows that the treatment of many types of pneumonia has been revolutionised. However, attempts to provide statistical data to support this experience or to express it in quantitative terms inevitably oversimplify.

The clinical picture of pneumonia is not so precise as certification of death might suggest. A patient suffering from broncho-pneumonia is usually ill from another cause, and the pneumonia develops gradually and undramatically, causing weakness and stupor until death ensues. It complicates many diseases: measles, whooping cough, bronchitis, influenza and even the common cold. It may follow anaesthesia, or occur in patients immobilized in plaster because of fracture of their bones.

It is clearly impossible to examine the whole field of mortality where pneumonia may have played a part or to apportion precisely the share between primary and secondary causes of death. In this study the discussion is confined only to mortality where pneumonia was certified as the primary cause of death.

Although this restriction simplifies the discussion, there is a further problem in interpreting mortality rates.^a Many factors may simultaneously influence the rise or fall of a disease, although some are clearly more significant than others. The impact of improved medical care is difficult to measure precisely, but with pneumonia, mortality rates for most ages fell abruptly and substantially following the introduction of

^{*} Bacteria are broadly classified into two groups, designated Gram positive or Gram negative. Penicillin is effective against most Gram positive organisms, but is ineffective against those that are Gram negative. Unfortunately, broncho-pneumonia is frequently caused by the latter.

modern drugs. Given abrupt and substantial falls, the margins of error become of small importance. Mortality from pneumonia was falling gradually prior to the development of modern chemotherapy. The effect of drugs on mortality rates may be measured by the abrupt changes which took place over this long-term trend. Pneumonia. Standardised Death Rates. Ages 15-64. England and Wales. 1861-1960.

Sources: Registrar General-Decennial Supplement, 1921, Part III (1933). Decennial Supplement, 1931, Part III (1952). Statistical Review of England and Wales, Part III (1955). Statistical Review of England and Wales, Part I (1954-60). Note: Decennial Averages 1961-1950; Annual Rates 1931-60.



The Epidemiological Picture

THE three principal epidemiological features of the disease are that its prevalence follows the cyclical rises and falls in influenzal epidemics³; that like many respiratory diseases the incidence tends to be higher in towns and lower in rural areas⁴; and that there is a regular rising gradient in the disease from upper to lower socio-economic groups⁵.*

Clearly these three features may be related. Where influenzal or other predisposing infections affect prevalence of the disease, the probability of infection is greater in cities or with crowded working and living conditions. Such factors may also act independently on the epidemiological pattern. The geographical distribution of influenza varies from one outbreak to another, being affected by the port of entry and the pattern of communications within the country. Atmosphere pollution in industrial cities predisposes the inhabitants to respiratory diseases, while poor living conditions can deny a patient the chance of careful nursing during fulminating respiratory infections.

In all but the lowest age groups, mortality from pneumonia rose steadily during the second half of the nineteenth century. Apart from the exceptionally high rates associated with the influenzal pandemic of 1918/19, rates fell gradually from the beginning of the century. Throughout this gradual rise and fall, the relative experiences of different age groups, particularly in middle-age remained remarkably uniform. The rate may have risen as an increased proportion of the population came to live in towns, and fallen with improved social conditions. (*Figs.* 1, 2, 3.)

^{*} The differences between conurbations, urban, semi-urban and rural areas are, however, not regular. Consultations per thousand population (urban 38 · 1, semiurban 40 · 4 and rural 35 · 6)*. The relative incidence (in terms of mortality) can vary with different ages, and sexes.

which different ages, and serve. The inverse relationship between social class and incidence is more distinct and regular. Consultations per thousand rise from 15 in the highest social class to 49 for the lowest social class. (Males aged 15-64)⁸.

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Pneumonia. Standardised Death Rates. Ages 0-14. England and Wales. 1861-1960.

Sources: Registrar General-Decennial Supplement, 1921, Part III (1933). Decennial Supplement, 1931, Part III (1952). Statistical Review of England and Wales, Part III (1955). Statistical Review of England and Wales, Part I (1954-60). Note: Decennial Averages 1861-1930; Annual Rates 1931-60.



Pneumonia. Standardised Death Rates. Ages 65 or more. England and Wales. 1861-1961.

Sources: Registrar General—Decennial Supplement, 1921, Part III (1933). Decennial Supplement, 1931, Part III (1952). Statistical Review of England and Wales, Part III (1955). Statistical Review of England and Wales, Part I (1954-60). Note: Decennial Averages 1861-1930; Annual Rates 1931-60.



Mortality rates fell dramatically in 1939; the fall was greatest in the middle-aged groups, less in the younger groups and slight for the elderly. The sulphonamides became widely available in that year and it is reasonable to attribute this abrupt improvement to their effect, particularly on lobar pneumonia, which explains the greatest benefit for the middleaged. (*Fig.* 1.)

There was a further marked change in the mid-1940s, which affected all age groups, but was most significant in the young, becoming increasingly less with age. These changes may be attributed to the introduction of penicillin. Presumably each new effective antibacterial drug reduces the mortality from those bacteria against which it is most effective. If so, one would expect the improvement to accelerate but then to revert to the original rate of decline. There is some suggestion that this is happening in the old and the young. (*Figs.* 2, 3.) These groups seem to be less affected by the broad-spectrum antibiotics introduced in the late 1940s and early 1950s. During the period covered by the introduction of modern drugs, mortality for most age groups fell more rapidly than ever before. Nevertheless, in spite of the highly satisfactory general picture, the adverse effect on mortality of the influenzal years 1943, 1947, 1951, 1957 and 1959 can be clearly seen. (*Figs.* 1, 2, 3.)

The abrupt decline in mortality associated with the introduction of modern drugs means that premature death rates are between one-tenth and one-fifth of the levels prevailing in the early 1930s. Had the rates declined only gradually at the speed of the long-term trend, premature mortality rates would be no more than one-third to one-half lower than these levels. (*Tables* A, B.)

The irregular decline in death rates has altered the relative risk for different age groups and the comparative sex ratios of mortality. The early adult age groups have benefited relatively more than others. Death risks now, therefore, change more rapidly with age. The greater fall in lobar pneumonia mortality among men has also partially levelled up the sex differences below late middle-age. (*Figs.* 4, 5.)

From the mid-1940s, death rates late in life have risen substantially, particularly for ages over 75 years. (Fig. 3.) This rise is due entirely to broncho-pneumonia, and could be explained in either of two ways.

Firstly, the increase may reflect no more than a change in certification habits. The rise coincides with a decline in certification of death from old age and senile decay. Before a specific treatment of broncho-pneumonia existed, the disease could well have been accepted as the natural end of life. If a change in certification habits lies behind this rise, pneumonia death rates among the elderly should shortly level off as a more exact diagnosis covers the whole population.

An alternative explanation is that since pneumonia and other respiratory mortality in late middle age has declined, the elderly population contains a far higher proportion of people with 'weak chests' than formerly. Before the therapeutic and prophylactic use of antibiotics, these people would probably have died in their fifties or sixties. Drugs have postponed the inevitable, and the vulnerable person survives until he succumbs later in life to broncho-pneumonia. The rise in numbers of deaths in old age from pneumonia is of the same order as the numbers saved in late middle-age ten or fifteen years earlier. If the second explanation is correct, pneumonia death rates in Changes in Pneumonia Mortality by Age and Sex: 1956-60 Rates per cent. 1931-35 Rates: England and Wales. Source: Registrar General. Statistical Review-(various years).

	Actual Rates		Projecting 1901–35 trend	
Ages	Male %	Female %	Male %	Female %
0-4	18.6	17-9	59.1	52.6
5-14	14.6	14.5	56.7	67.6
15-24	15.0	17.4	51.7	51.4
25-34	11.7	17-0	57.6	60.6
35-44	11.8	17.4	64.5	64.5
45-54	21.0	25.9	69.8	62.3
55-64	48.5	41.9	67.7	61.8
65-74	90.1	67.6	69.7	65.9
75+	181.6	147.2	63.8	88.3

old age should continue to rise far higher as a greater proportion of vulnerable persons survive beyond their three score years and ten.

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A

B

Changes in Pneumonia Mortality by Type, Age and Sex: 1956–60 Rates per cent. 1931–35 Rates: England and Wales. Sources: Registrar General. Statistical Review—(various years).

-	Lobar Pneumonia		Broncho and Unspecified Pneumonia	
Ages	Male %	Female %	Male %	Female %
0-4	4.7	6.7	21.0	19.3
5-14	4.8	5.5	21.0	20.0
15-24	5.4	5.9	34.4	33.3
25-34	4.4	7:3	26.1	28.4
35-44	5.6	8.0	22.3	28.7
45-54	10.8	11.9	34.7	38.1
55-64	23.9	18.8	75.0	58.2
65-74	42.0	31.4	120.0	84.6
75+	87.4	60.6	209.4	178.0

Lobar Pneumonia Death Rates by Age and Sex. England and Wales. 1931-35 and 1956-60. Source: Registrar General. Statistical Review-(various years).



Broncho and unspecified Pneumonia Death Rates by Age and Sex. England and Wales 1931-35 and 1956-60. Source: Registrar General. Statistical Review-(various years)



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Economic Profile of Pneumonia

THE great improvement in the medical picture of pneumonia has radically altered the economic effects of this disease. The number of premature deaths has fallen considerably and incapacity caused by the illness is substantially reduced.

Mortality

If the decline of death rates per million is translated into numbers of deaths, it represents the saving of 19,400 lives of persons under 65 years a year. At the mortality rates of the early 1930s, premature deaths would currently be 25,400 a year. In fact they currently number approximately 6,000. (*Figs.* 6, 7, 8.)

If 1931-35 rates had remained constant, total premature deaths would have risen in all age groups. Not only is the population larger but, particularly with the high birth rate since the war, it also contains a higher proportion of persons of vulnerable ages. Despite the increased number of infants, fewer have died.

On the long-term trend alone the number of deaths in all ages below 65 years would currently have fallen only to 15,500 giving an *expected* saving of 9,900 lives a year. The difference between this and the actual saving, 19,400, results from abrupt improvement in premature mortality dating from the late 1930s. The use of modern drugs has thus given an *additional* saving of 9,500 lives a year.

This saving of life has been cumulative. If the death rates of the early 1930s had not declined a total of 320,000 persons now alive would have died from pneumonia. Of this number 150,000 would have been expected to be saved on the longterm decline in the disease. The remaining 170,000 have survived because the disease yielded to effective treatment. These estimates are calculated from the annual numbers saved, less



Annual number of Pneumonia Deaths. Ages 15-64. England and Wales. Quinary averages 1931-60. Source: Fig. 1.



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Annual number of Pneumonia Deaths. Ages 65 or more. England and Wales. Quinary averages 1931-60. Source: Fig. 3.



death risks later in life as each year's group of survivors grows older. The *additional* survivors currently out-number the *expected* survivors because the improvement over the longterm trend was greatest with younger age groups.

The earnings of these survivors give an indication of the economic gain flowing from this saving in life. Assuming they enjoy the same employment conditions as the rest of the population, and taking manual workers' earnings as the basis for the estimate, annual earnings amount to $\pounds 26m$. for the *expected* survivors and $\pounds 43m$. for the *additional* survivors. This represents the loss of personal income which would have been suffered each year had there been no improvement in pneumonia mortality.

The central economic issue involved in mortality, however, is the effect it can have on the age composition of the population. The population can be divided into two broad groups, supporters and their dependants. Supporters are men aged 15 to 65 years and women aged 15 to 60 years. Their dependants are children and the elderly. A change in mortality from a disease can alter the balance between the groups.

The expected and additional survivors present a contrasting picture when analysed into these age categories. The expected survivors include more dependants than supporters. The additional survivors include approximately equal numbers of each. (Table C and Fig. 9.)

If looked at simply in terms of numbers, since pneumonia causes greatest mortality at the extreme of life its decline has increased the dependency burden. To reduce this burden six supporters would need to be added for every four dependents. The significance of this increase in dependents is however slight. The pneumonia survivors number about seven for every thousand of the total population, producing an increase in the dependency ratio of four parts in every thousand.

If the question of supporters and dependants is considered in terms of earnings rather than of numbers a more exact measure is obtained of the way national income is affected. Also the differences between the additional and expected survivors become more distinct.

The additional survivors include a very high proportion of male earners. Their greater earning capacity more than compensates for the large number of child dependants, if income per head is compared against a group which has the same sex and age composition as the whole population. Also the additional survivors' income per capita is almost half as high again as a comparable figure for the expected survivors.* (Fig. 9.)

The distinguishing feature of both groups of pneumonia survivors is that a large number of the dependants are children.

^{*} Annual income per capita amounts to £173 for the expected and £251 for the additional survivors. If the survivors had the same age and sex compositions as the total population, the per capita income would be £243. These estimates are lower than average national income per head as they are calculated on manual workers' earnings. This basis was selected because of the socio-economic pattern of pneumonia mortality. Since figures are not available the esti-mates are not weighted to reflect socio-conomic gradients. The estimates do take into account unemployment, retirement and, for youths, girls, men and women, the earning experiences of different age groups. (Sources: Registrar General. Statistical Review of England and Wales, Part 11. Population 1960; Census Figures for In-dustrial States. Ministry of Labour The Length of Working Life of Males-Great Britain 1959 et al.)

Cumulative 'Expected' and 'Additional' Pneumonia Survivors and Total Population. Percentage Age Distribution by Sex: England and Wales, 1956-60.

Source: Figs. 6, 7 and 8. Registrar General. Statistical Review of England and Wales 1960, Part II, Tables Population (Table A5).

		'Expected' Survivors %	'Additional' Survivors %	Total Population %
Male	Child Dependants	40	36	24
	Supporting Ages	49	58	66
	Aged Dependants	11	6	10
Female	Child Dependants	40	38	21
	Supporting Ages	35	45	59
	Aged Dependants	25	17	20
Persons	Child Dependants	40	37	23
	Supporting Ages	43	53	62
	Aged Dependants	17	10	15

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C

Age and Sex Distribution of total number 'Expected' and 'Additional' Pneumonia survivors, and total population, England and Wales, 1960. Source: Vide Table C.



If the survivors are looked at in terms of potential earnings, then the expected decline of the disease promises no alteration to the dependency burden while the additional saving in life resulting from the development of modern drugs promises a potential easing of this load.* (*Fig.* 9.)

Incapacity

The effective treatment of the disease has halved the loss from incapacity among the working population. Spells of incapacity due to pneumonia currently commencing each year in the working population number 32,000 involving a loss of 2.09m. working days.⁶ Since figures were compiled on a uniform basis under the post-war National Insurance Acts, absence has tended to fall since there have been fewer cases of shorter durations—influenzal years excepted. (*Fig.* 10.)

The greater gain is the improvement over the 1930s. Strictly comparable figures do not exist, but using data from pre-war industrial sickness insurance schemes, it would seem that in the early 1930s an attack of pneumonia meant a loss of eighteen to nineteen weeks' work.[†] Now the loss averages nine to ten weeks with half the patients returning to work after only seven weeks.^{*}

This reduction in incapacity is equivalent to a saving of $\pounds 3.8m$. per annum. The National Insurance funds benefit to the extent of $\pounds 1.5m$. and the balance represents savings to patients or their employers. The saving to the insurance funds is in the region of twice the estimated total cost falling on the National Health Service for domiciliary care of pneumonia among the working population.§

^{*} The present value of future earnings per capita, discounted at 5 per cent. single rate works out to $\pounds4,502$ for the expected and $\pounds5,184$ for the additional survivors. If the survivors had the same age and sex composition as the total population, the capita value would be $\pounds4,507$. (Source: see footnote on page 24.)

[†] Pre-war incapacity figures are derived from statistics compiled by the Department of Health for Scotland Reports on Incapacitating Sickness in the Insured Population of Scotland (1931-2 annually to 1936-7). The figures were adjusted to allow for the higher death rate of the early 1930's as, in the nature of things, the period between onset and death is shorter than that between onset and recovery.

[§] The estimated cost to the Insurance Fund, $\pounds 1.5m$, is based on an earner with two dependants receiving benefits at rates current in 1960. The estimate for general medical services is $\pounds 0.4m$, calculated from the proportion of consultations over pneumonia (among the working population) of total consultations. (Logan, W.P.D. and Cushion, A.A. Morbidity Statistics in General Practice Vol. 1.) The cost of drugs, $\pounds 0.3m$, is an estimate for nine week's supply per case.

Pneumonia Incapacity. Spells Commencing and Working Days Lost by Sex per Thousand Working Population. Great Britain. 1954-55 to 1959-60.

Source: Ministry of Pensions and National Insurance: Report for the Year 1961, Table 39, Cmnd. 1764.



The saving to the individual patient has greater social significance. Unless he is covered by a comprehensive sickness insurance scheme, the extra nine weeks of pneumonia would have meant a further loss in earnings of almost £80. The longer an illness lasts the greater a family's difficulties will be. The loss not only multiplies with duration but changes in nature. Savings are consumed, hire purchase instalments are defaulted and rent falls in arrears.^{*} A long spell of sickness can leave a residue of economic disability hampering the chance of full recovery. Drugs shorten the disease and help take pneumonia out of this class of illness.

The reduction of pneumonia mortality and morbidity brings far-reaching social advantages for there are few aspects of life the disease might not touch. The death of a young mother makes no impact on national income figures, but the social impact is great. In the region of 2,500 marriages would have ended each year through the death of the wife before she reached her sixty-fifth birthday had pneumonia mortality not improved. Of this number about one-quarter would have ended after less than ten years of marriage.

The Present Position

WENTY-FIVE years ago, the boldest physician could hardly have dared to hope that so much could be achieved so rapidly. Premature mortality has been reduced four-fifths. It seems probable that the effect of drugs on mortality diminishes with age. A far greater number of people are now living their normal span of active life while the time spent in old age has not lengthened. Contrary to the popular impression, the effect of modern medicine is not simply to increase the economic burden of an aged population. The increased proportion of dependants due to reduced pneumonia mortality results mainly from a greater number surviving in childhood.

But even now, with 6,000 premature deaths a year from pneumonia there is room and scope for further advances in therapy. Pneumonia still accounts for one in ten deaths of infants and children.

Some authorities consider that there is an irreducible minimum of premature pneumonia mortality, but that is the view of the pessimist. Hope for further improvement rests on the possible development of antibiotics capable of dealing with those organisms responsible for broncho-pneumonia, and resistant to existing drugs. Also, because of the marked effect on pneumonia mortality of virus epidemics, there is a need for greater research into such virus infections as influenza, measles and the common cold which predispose their victims to pneumonia.

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Cover The illustration is Laënnec's first stethoscope taken from his book De l'auscultation médiate (Paris 1819).

The English translator, John Forbes, made one of the most famous false prophesies in dealing with the stethoscope. He doubted whether the invention would be adopted generally-"It must be confessed that there is something even ludicrous in the picture of a

grave physician formally listening through a long tube . . . as if the disease within were a living being that could communicate its condition to the sense without."

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Office of Health Economics

THE Office of Health Economics was founded in 1962 by the Association of the British Pharmaceutical Industry with the following terms of reference:

- 1. To undertake research to evaluate the economic aspects of medical care.
- 2. To investigate, from time to time, other health and social problems.
- 3. To collect data on experience in other countries.
- 4. To publish results, data and conclusions relevant to the above.

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