Section of Epidemiology and Preventive Medicine

President D D Reid MD

Meeting May 9 1968

President's Address

The Beginnings of Bronchitis

by Professor D D Reid MD FRCP
(*London School of Hygiene and Tropical Medicine, London*)

In talking of the 'beginnings' of bronchitis, I use the word in the sense which the Oxford English Dictionary defines as 'that in which anything has its rise or in which its origin is embodied'. I am concerned with the circumstances which favour the onset of bronchitic disease rather than the clinical detail of its 'initial or rudimentary stage'.

This will be a personal view, but one which necessarily draws much from the work of others. As an ageing academic, I can sympathize with the wistful tone of the words of James Lind which are inscribed on the wall of the medical school in Edinburgh: 'The province has been mine to deliver precepts; the power is in others to execute.' It is therefore to students and colleagues who have at some time shared my interest in the origins of respiratory disease that I am especially indebted.

Sixteen years ago, the great London smog gave new impetus to the study of chronic bronchitis. As a result, the importance of cigarette smoking, the social environment and exposure to air pollutants in the evolution of the obstructive lung diseases of adult life is now fully realized. But the relentless deterioration apparent in the later stages of bronchitis complicated by emphysema or cor pulmonale tends to fix the attention of the investigator and to engender a pessimistic fatalism about the prospects for effective prevention. We seem to have ignored the fact that the very young, as well as the old, died in the catastrophe of 1952 and to have concentrated our clinical and epidemiological research on those over the age of 40. This myopic view may have missed the true beginnings of the disease which could lie much further back in early life.

Bronchitis in Early and Late Life

My own interest in this question was aroused by our finding (Reid & Fairbairn 1958) that, compared with healthier colleagues, many postmen who were seriously disabled or died because of chronic bronchitis had an excessive liability to frequent and prolonged absence from this cause which extended back to their early 20s. This immediately raised the question: does respiratory disease in youth lead on to bronchitis in middle and late life? Pædiatric opinion is apparently in doubt about this proposition; and one long-term study has not shown that such a progression is inevitable (Harnett & Mair 1963). Certainly, the frequent clinical observation that the 'chesty' child seems to grow out of his disability is reflected in the steep decline throughout childhood in the death rate from bronchitis and pneumonia. In Fig 1, for example, this trend is seen in succeeding generations of males born in 1921, 1931, 1941 and 1951 (data from tables prepared by Case & Harley 1958, and Case *et al.* 1968). On the other hand, the bronchitic mortality experience of these generations shows that, in males, cohorts suffering relatively high death rates in childhood also had high rates from the same disease in adult life. This implies that males exposed during infancy and childhood to epidemic conditions causing heavy respiratory mortality continue to be especially predisposed to mortal lung disease throughout the remainder of their lives. Rosenbaum (1961) found similar evidence of the residual effect of childhood environment on respiratory morbidity in the higher rates of illness among National Service men recruited in the industrial areas of the North compared with the

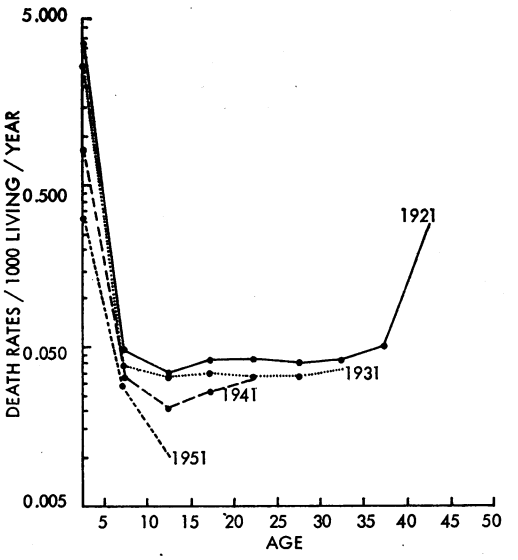

Fig 1 *Death rates from bronchitis in males born in 1921, 1931, 1941 and 1951*

rates for men from country districts even when both were serving together elsewhere.

Respiratory disease in childhood and middle life can also be looked at from the international point of view. Because of differences between countries in the use of diagnostic terms such as 'bronchitis' in certifying the underlying cause of death (Reid & Rose 1964), it is best to group together the major non-malignant respiratory diseases (influenza, pneumonia and bronchitis) before comparing, as in Table 1, the mortality experience of the United Kingdom with that of our neighbours in North West Europe. The British excess over Scandinavian levels in respiratory mortality in middle age is repeated in the death rates in childhood. This consistency implies that some ætiological factor or factors in the national environment affects respiratory disease in both these age groups. On the other hand, the

Table 1

Death rates per 100,000 per annum in males from respiratory diseases
(Influenza, bronchitis, pneumonia – B30-32 in 1957–61, WHO)
(World Health Organization 1964)

	Age-specific rates		*Male:female ratios*	
Country	*45–64*	*0–14*	*45–64*	*0–14*
England & Wales	160	35	3·3	1·2
Scotland	152	37	3·3	1·2
Northern Ireland	129	40	2·4	1·3
Netherlands	39	14	2·6	1·2
France	30	23	2·2	1·2
Norway	20	20	1·4	1·2
Denmark	20	18	1·5	1·3

even closer association between the male:female ratios in national death rates and the level of these rates in the same 45–64 year age-group emphasizes the decisive influence of the predominantly masculine habit of cigarette smoking.

These data strongly suggest that some ætiological agents can affect more than one age group. In the strategy of research on the origins of respiratory disease, therefore, there is need for a broad approach, since clues derived from studies of respiratory disease in childhood may well apply in the context of adult bronchitis.

External Factors in Bronchitis at Different Ages

The way in which social and environmental factors or personal habits may act at different stages of life is clearly seen in the mortality statistics of this country. Within England and Wales, for example, there is, as shown in Table 2, a similar rural-urban gradient in bronchitis mortality in males in infancy and in the age group 45–64 years. Some urban factor, perhaps air pollution, is thus especially important at those stages of life. There is, however, a reversal of this gradient in the age-group 5–14; and the Registrar-General has suggested that this is due to a greater exposure of

Table 2

Death rates per 100,000 per annum from bronchitis (ICD 500-2) in England & Wales 1959–63
(Registrar-General: 1961 Area Mortality)

	Age group (years)			
	0–4	*5–14*	*15–44*	*45–64*
Conurbations and urban areas of 100,000+	16	0·4	2·2	75
Urban areas of 50,000 – 100,000 and <50,000	13	0·5	1·9	61
Rural districts	11	0·8	1·4	43
Male:female ratio	1·3	1·2	1·8	5·1

children in country districts to vegetable and other allergens. Even at the risk of a charge of the most blasphemous heresy, I would venture that it is more likely that this reversal is similar to that observed in the specific infectious illness such as measles. The country child often first encounters infection on going to school and suffers the heaviest mortality then rather than earlier. In the city, on the other hand, there has been a removal by death in infancy and early childhood of the most susceptible and at least partial immunization of the remainder, so that the death rates after age 5 are lowered. Minor respiratory infections apparently follow the same pattern. Lidwell & Somerville (1951), surveying in a country village, found peak rates for the common cold in school children whereas we, in Paddington in Central

London, found the highest rates in pre-school children more widely exposed to infection at an earlier age in the congested city (Brimblecombe *et al.* 1958). If repeated infections are important in initiating bronchitis, these differences in exposure could explain the changing urban-rural differences in bronchitis mortality in children.

As age increases, the gap between the rates for rural and urban areas widens; and it is reasonable to suppose that this is the result of the cumulative effect of urban elements such as air pollution on an ageing population. More clear cut is the indication of the effect of cigarette smoking given by the male:female ratio which, as Table 2 shows, starts from a small masculine excess in mortality and begins, after the age of 15, to rise steeply.

Table 3

Bronchitis death rates in the county boroughs of England & Wales in 1950–63 and environmental conditions
(Zero-order correlation coefficients for 79 county boroughs)

	Age group (years)		
	0–1	*1–14*	*45–64*
Percentage in Social Classes IV & V	+0·37	+0·04	+0·59
Air pollution grading ●	+0·30	−0·08	+0·72
Persons per room	+0·25	−0·04	+0·52
Persons per acre	+0·11	−0·09	+0·39

● Waller classification: *see* Douglas & Waller (1966)

The possible effects on bronchitis in different age groups of factors, such as domestic overcrowding and air pollution, which may affect the spread or severity of respiratory infections can be sought in comparisons of the mortality experience in the county boroughs of England and Wales. Table 3 gives the zero-order correlation coefficients which indicate the degree of association between these factors and bronchitis death rates at different ages in the period 1950–63 in the areas for which data were available. Those factors related to respiratory mortality in adults, such as air pollution, Social Class distribution and domestic overcrowding, appear to have a similar influence on bronchitic mortality in infancy, but none on the death rate among children. In both these age groups, however, the number of deaths in the period in many boroughs is small and the rates too unstable to justify either a more sophisticated analysis or very firm conclusions.

The Epidemic Behaviour of Upper and Lower Respiratory Tract Diseases

When death rates are, as in adolescence, too low to give adequate indices of the frequency of respiratory disease, data on morbidity are a useful alternative. Despite its limitations, the Hospital In-Patient Enquiry is a potential source of information that has been largely neglected. Yet an analysis of the discharge rates will show that, as in international mortality rates, the range of these rates for bronchitis from low rates in East Anglia to peak rates in Liverpool is the same for children as adults. Moreover, as Fig 2 shows, the rates for upper respiratory tract infections such as otitis media in childhood are high in the same areas where admission for bronchitis is especially frequent among adults. Among men examined before National Service, Lee (1957) found similar

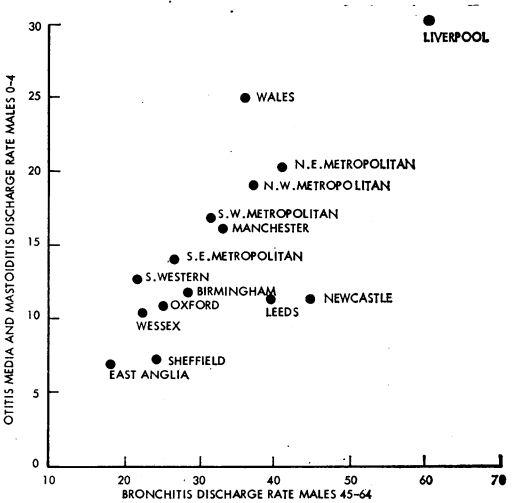

Fig 2 *Regional discharge rates per 10,000 per annum for middle ear disease in males aged 0–4 and for bronchitis at ages 45–64 (Hospital In-Patient Enquiry 1960–64)*

geographic differences in the prevalence of chronic middle ear disease. We may reasonably suppose that all these forms of respiratory morbidity are adversely influenced by some factor, perhaps the poor social environment and high air pollution levels, common to the industrial areas in Northern England. This impression is reinforced by the suggestive concordance seen in Fig 3 between peaks of pollution recorded in nearby Kew and the onset of otitis media among the schoolchildren in Southall included in the Medical Research Council trials of ultraviolet air sanitation in classrooms. Similarly, the Social Class gradient which is so characteristic of adult bronchitic mortality was evident in the consultation rate for such middle ear disease in the Registrar-General's survey of general practice (Logan & Cushion 1958). It seems, then, that the uniformity in response to environmental conditions in both upper and lower respiratory disorders means that the investigation of causes common to several age groups can be as sensibly based on respiratory morbidity in general as on bronchitic mortality in particular.

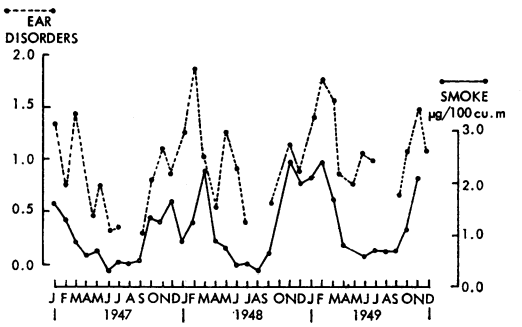

Fig 3 *School absence rates per 100 child-months from earache, mastoiditis and otitis media in Southall, Middlesex, and smoke pollution levels recorded at Kew four miles away*

Field Surveys of Bronchitis in Childhood

These analyses of routinely recorded data suggest the need to view bronchitis in the setting of respiratory disorder as a whole, particularly among the young where the effects of other ætiological factors are not obliterated by the dominant influence of smoking. Again, school populations are widely representative, and, through the school health service, readily accessible. For these reasons, we decided to conduct surveys in collaboration with our colleagues in the public health services of upper and lower respiratory tract disorders among children living in the wide range of circumstances found in this country. Dr M Wahdan was responsible for the field work in the first of these carried out from our Department at the London School of Hygiene in 1962 (Wahdan 1963, Reid 1964). The object was to examine random samples of boys and girls aged 11 years living in the very different conditions of urban Sheffield and the rural Vale of Glamorgan, in Wales. Some 600 children were examined in Sheffield and 400 in Wales. They were measured and weighed and the ears, nose and throat examined. The Wright peak-flow meter and the McKesson Vitalor were then used to assess ventilatory function in terms of the maximum flow rate and the volume expired during the first second of forced expiration. The mother then completed a standard questionnaire dealing with past respiratory illness and present disabilities such as discharge from the ears or snoring, and with the social and domestic circumstances of the family. The response from mothers and children was, at around 97%, excellent.

Fig 4 shows one set of results relating the prevalence of ear disease to Social Class and area. There is, in both Sheffield and rural Wales, a Social Class gradient in middle ear disease similar to that which is the most distinctive feature of adult bronchitis mortality; and, within each Social Class, the frequency of ear disorder is higher in the urban area. Moreover, these Social Class and rural-urban gradients are most marked in the more serious form of aural infection where there is either perforation or chronic discharge.

The sensitivity of children to differences in presumptive air pollution levels was also evident in the low rates for repeated attacks of chest disease in rural Wales and the old and new residential areas of Sheffield and the high rate for the industrial part of the city where pollution is worst. The lung function tests also showed a clear gradient between rural Wales and industrial Sheffield. The findings of this pilot enquiry have now been amply confirmed by a comprehensive enquiry in the different parts of Sheffield by Lunn and others (1967).

A different approach has been taken by Douglas & Waller (1966). As part of the continuing observation of the cohort of children born in 1946, Dr Douglas asked school medical officers to examine and interrogate children in his sample now living in different parts of the country. The residential histories obtained allowed the classification by Mr Waller of presumptive exposure to pollution according to the prevailing levels of pollution in each of the areas concerned. As a result, they were able to show a consistent increase in the frequency of bronchitis with increasing pollution. But, unlike the workers in Sheffield, they did not detect any gross excess of upper respiratory infection in the more polluted areas.

Another line has been followed in the study by Professor Holland and Dr Colley of the frequency of respiratory symptoms in all members of over 2,000 families living in two areas of North London (Colley & Holland 1967). They showed that, in both the mothers and children in families living in these areas, the frequency of symptoms

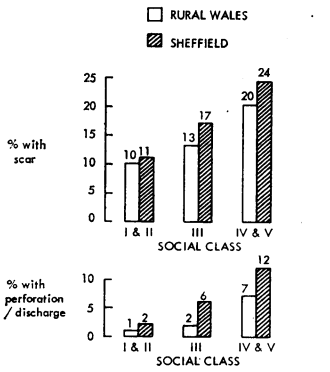

Fig 4 *Percentage prevalence of ear disease by Social Class in rural Wales and Sheffield*

such as winter morning cough differed between the two districts. Among the fathers, differences associated with Social Class or occupation and with smoking seemed to mask any geographic differences that might be present.

More recently, Dr Colley and I have been extending the earlier studies involving the collaboration of academic department and medical officer of health much more widely throughout the country (Colley & Reid 1969). The methods of clinical examination and interrogation of mothers were much as before but the larger number of school health service doctors helping in the survey meant that special care had to be given to the definition of clinical standards and to the training and testing of observers. The plan of the survey called for the examination of 500 boys and 500 girls between the ages of 6 and 10 drawn randomly from the school populations in each of 11 areas of both England and Wales. These areas were selected to represent industrial cities with high levels of air pollution, e.g. Newcastle upon Tyne and Bolton, each matched by towns of similar size where the population might be just as crowded but where pollution levels were known to be lower, e.g. Reading and Bristol. For each of these towns, a rural area was selected for survey from the same county so that the climatic conditions might be roughly similar but urban circumstances of life would be absent. As before, the restriction of the age range to 6–10 years was designed to eliminate all but the most precocious habitual cigarette smokers and thus to bring out more clearly any effects of exposure to the various elements in the urban or domestic environment.

The Urban and Social Environment in Childhood Bronchitis

In the English areas, where about 8,000 boys and girls were examined, clear Social Class and rural-urban gradients in respiratory illness were found, as shown in Fig 5. A history of repeated attacks of

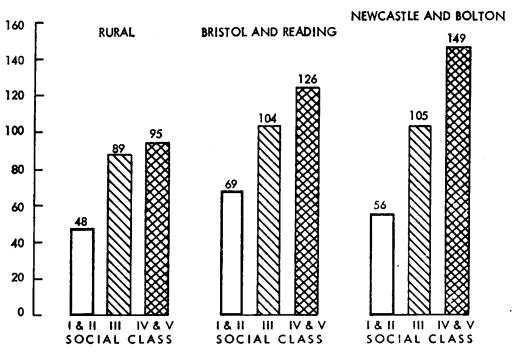

Fig 5 *Age-adjusted morbidity ratios (%) for prevalence of persistent cough. (Overall rates for England and Wales=100%)*

Table 4

Peak flow rates (litres/minute) adjusted for age, height, weight and area differences by Social Class and respiratory disease history

	History of bronchitis and/or pneumonia	
Social Class	*−ve*	*+ve*
I & II	233	224
III	231	219
IV & V	228	219

either bronchitis or pneumonia has, even at this early age, the beginnings of a Social Class gradient which is particularly obvious in the industrial towns of Newcastle and Bolton where air pollution levels are higher. Similar trends are evident in the prevalence rates of cough throughout the day.

That these histories of bronchitis and pneumonia in young children should not be ignored is clear from Table 4 where it appears that, even after adjustment for differences in physique and place of residence, those with such a history have significantly lower peak flow rates than those without. Moreover, there is a consistent if not significant downward trend in both groups between Social Classes I & II and IV & V. As a result, the bronchitic child of a semi-skilled or unskilled labourer has a peak flow rate 6% below that of a non-bronchitic child in a professional family even after his lower height and weight have been taken into account.

Some Consequences for Further Studies

In our study of the problem of adult bronchitis, we have been mesmerized by the desperate clinical problems of the old and depressed by our failure to influence either their smoking habits or, by antibiotic or other therapy, to halt pulmonary deterioration. We have not, I believe, sufficiently considered the implications, as regards prevention, of the likelihood that, despite the improvement apparent in adolescence, the bronchitic child *is* father to the bronchitic man. If, however, I have given the impression that an innate weakness of the respiratory system leads inexorably and inevitably to adult bronchitis, you must put these overtones of Calvinistic predestination down to my own Presbyterian beginnings. On the contrary, I believe that there are grounds for reasoned optimism and that this Section should be no place for prophylactic nihilism. There is evidence, for example in our own studies of British and Norwegian migrants to the United States (Reid *et al.* 1966), *see* Table 5, and in Dean's observations of their experience in South Africa (Dean 1965), that the British race is not doomed by its genetic constitution to die of the British disease. When people move from this country, particularly in childhood, to the more

Table 5
Average annual age-adjusted death rates from chronic non-specific lung disease per 100,000 (ICD 502, 526 & 527·1)

Norwegian-born living		*British-born living*		
In Norway	*In USA*	*In UK*	*In USA*	*American-born living in USA*
10	9	125	23	24

favourable conditions of the United States, they smoke no less, but the death rate from chronic lung disease among them falls remarkably. Similarly, the experience of successive generations in this country suggests that the rising standard of living does not breed a race of respiratory weaklings who survive infancy but die later in life.

As yet we know little about the precise nature of the locality, social and domestic environment to which these studies point as the beginnings of bronchitis. There is thus much need for the multilateral approach to the problem by the various disciplines represented in the membership of this Section. Does air pollution or cold aggravate respiratory disease by reducing the resistance of the respiratory tract to secondary invasion? Does the special susceptibility of Social Classes IV and V lie in some innate poverty of physique, or in the inadequacy of diet or crowded homes? And which comes first, the poor lung function or the repeated attacks of respiratory infection? Or are both poor function and recurrent illness simply different manifestations of an inadequate physical endowment? How can we obtain the short-term benefit of immunization by minor infection without incurring the long-term risks of permanent lung damage caused by more serious complications? The answers can come only from long-term surveys.

Of special importance to prevention is the prospect that the early detection of those susceptible to respiratory illness could lead to special care in minimizing the effects of exposure to infection and, above all, to dissuade them from beginning to smoke. North Sea gas may reduce the aggravating effect of cold and air pollution on the respiratory tract of the next generation but the seriousness of smoking among children was recently emphasized by the study of Holland & Elliott (1968). I hope that they are now more enlightened at Eton than they were in the plague year of 1603 when a certain Tom Rogers said: 'All the boys were obliged to smoak in the school every morning, and that he was never whipped so much in his life as he was one morning for not smoaking.'

In all this, the school health service could play as vital a part as it has done in the preliminary research. Whatever our special skills, our aim as epidemiologists concerned with prevention must be to observe and to measure the influence of each factor that promotes bronchial disease especially in its beginnings. As William Petty, the grandfather of vital statistics, put it, our most urgent needs are those numerical indices of places and of health 'which are a better Judge of Ayres than the conjectural notions we commonly read and talk of'.

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Meeting January 9 1969

A discussion was held on **Pertussis Vaccines.** The opening speakers were Dr S Polakoff and Dr F T Perkins.