

Early Detection of Chronic Lung Diseases

Report on a WHO Meeting

Vienna
31 May–2 June 1978



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WORKING GROUP ON EARLY DETECTION OF CHRONIC LUNG DISEASES

Vienna, 31 May - 2 June 1978

1. INTRODUCTION

Chronic nonspecific lung diseases (CNSLD) are known to be an important cause of morbidity and mortality in adults in the European Region and in other areas of the world. The patterns of the diseases appear to vary from country to country. It is not clear how far this may be due to true differences in the frequency of their occurrence or to differences in the ways in which they are reported. But whatever the explanation may eventually be, sufficient is now known of factors implicated in the etiology of the diseases for serious consideration to be given to ways in which they may be controlled and prevented. While the major etiological factors may be the same, the approaches to control and prevention, diagnosis, care and rehabilitation will differ. There is general agreement that vigorous clinical management of patients with severe or disabling disease is often ineffective in slowing progression — hence the need to detect cases at an early stage when the progression may be halted or even reversed or, better still, to prevent the disease from developing in the first place.

The meeting of the Working Group was convened by the WHO Regional Office for Europe, in collaboration with the Austrian Government, and held on the premises of the United Nations Industrial Development Organization in Vienna.

The objectives of the meeting were:

- to advise on the identification of groups and individuals who may be most at risk of developing chronic lung diseases;
- to review and evaluate methods for the prevention and control of the diseases;
- to suggest which of the methods could form the basis for community action against the diseases;
- to identify areas needing further investigation and development.

The meeting was attended by 14 temporary advisers and 4 WHO staff members. (The list of participants is given in Annex II.) It was opened on behalf of Dr Leo A. Kaprio, WHO Regional Director for Europe, by Dr G. Lamm, Regional Officer for Chronic Diseases, who thanked the Government of Austria for its assistance. Dr J. Daimer, Deputy Director of Health, welcomed the participants to Vienna on behalf of the Federal Minister for Health and Environmental Protection.

The Working Group elected Professor H. Denolin and Professor M. Kunze as Co-Chairmen, and Professor J.R.T. Colley as Rapporteur.

2. EXTENT OF CHRONIC NONSPECIFIC LUNG DISEASES

The first requirement in investigating the extent of CNSLD is to review the existing data; as there are no reliable estimates of the prevalence of the diseases in any European or other country, data derived from routine data collection and from specific field surveys must be used.

The comparability of data obtained from such varied sources is usually limited. Only specially designed epidemiological studies, using uniform definitions and diagnostic criteria, would ensure comparability. Such criteria were proposed nearly 20 years ago by a group of British investigators,^a and were later accepted with slight modifications by WHO,^{b,c} the American Thoracic Society,^d and the Medical Research Council in the United Kingdom.^e However, the great majority of physicians seldom follow these recommendations. Hence, differences in diagnostic fashions both within and between countries remain and greatly impair the comparability of the data recorded. Even with these limitations some useful conclusions can be drawn from the available data.

2.1 Mortality from CNSLD

CNSLD are an important primary cause of death and contribute to mortality from other respiratory and cardiovascular diseases. Although the

^a *Thorax*, 14: 286 (1959).

^b WHO Technical Report Series, No. 213, 1961. (*Chronic cor pulmonale: report of an Expert Committee*).

^c WHO Regional Office for Europe. *Chronic non-specific lung diseases: report on a Symposium, Moscow*. Copenhagen, 1963 (document EURO 212).

^d *American review of respiratory disease*, 85: 762 (1962).

^e *Lancet*, 1: 775 (1965).

drawbacks of comparisons based on mortality statistics are well known, it is useful to review recent mortality statistics on CNSLD in European countries. These statistics are available for almost all countries in Europe. Rates of mortality from chronic bronchitis, emphysema and asthma in 1974 and 1975 in Europe ranged, for men, from 17 per 100 000 in France to 99 per 100 000 in the German Democratic Republic, and for women from 8 per 100 000 in Finland to 72 per 100 000 in Romania (Table 1). The percentage of deaths

Table 1. Mortality (per 100 000) from all causes and from CNSLD (bronchitis, emphysema and asthma, ICD: 490-493) by sex in European countries in 1974 and 1975

Country	Year	Males			Females		
		All causes	CNSLD	% ^a	All causes	CNSLD	% ^a
Austria	1975	1 321	32	2.4	1 238	18	1.5
Belgium	1974	1 280	41	3.2	1 105	14	1.2
Bulgaria	1975	1 114	59	5.3	950	33	3.4
Denmark	1975	1 101	39	3.5	904	16	1.7
Finland	1974	1 071	38	3.5	841	8	0.9
France	1974	1 111	17	1.6	991	9	0.9
German Democratic Republic	1975	1 408	99	7.1	1 443	33	2.3
Germany, Federal Republic of	1974	1 214	53	4.4	1 134	21	1.9
Hungary	1975	1 334	48	3.6	1 158	22	1.9
Ireland	1974	1 221	74	6.1	1 039	33	3.2
Italy	1974	1 041	48	4.6	878	21	2.4
Netherlands	1975	934	43	4.6	732	12	1.6
Norway	1974	1 085	18	1.7	897	9	1.0
Poland	1975	849	36	3.8	792	14	1.8
Portugal	1975	1 135	41	3.6	932	20	2.1
Romania	1974	949	79	8.4	871	72	8.3
Spain	1974	899	41	4.6	799	24	3.0
Sweden	1975	1 186	22	1.9	968	11	1.1
United Kingdom:							
England and Wales	1974	1 233	81	6.6	1 148	28	2.4
Northern Ireland	1974	1 204	78	6.4	1 037	31	3.0
Scotland	1975	1 285	66	5.1	1 146	24	2.1
Yugoslavia	1974	894	24	2.7	788	13	1.6

^a Proportional mortality from bronchitis, emphysema and asthma.

Source: World Health Organization. *World Health Statistics Annual, 1977*. Vol. 1: Vital statistics and causes of death. Geneva, 1977.

from CNSLD (proportional mortality) ranges in males from 1.6% in France to 8.4% in Romania, and in females from 0.9% in France and Finland to 8.3% in Romania. The lowest mortality rates for CNSLD were recorded in 1974 and 1975 in Scandinavian countries and in France, the highest at that time being in Bulgaria, the German Democratic Republic, Ireland, Romania and the United Kingdom. In all countries mortality rates for CNSLD are higher in men than in women. It seems that the within-sex variation in mortality noted above may partly be explained by different age structures of the population and by differences in diagnostic habits and coding procedures in particular countries. A more detailed study of mortality from respiratory diseases by age and sex in European countries has been published by WHO.^a

2.2 Sickness absence

Respiratory diseases are among the main causes given for absence from work in a number of European countries. These statistics reflect disease levels as well as the social and economic problems of the working population, and thus interpretation of these data is not simple.

Levels of sickness absence or provision of sickness benefit do vary between countries. For example, sickness benefit rates among males with pneumonia, bronchitis, emphysema and asthma in England and Wales in certain age groups were between two and three times as high as the sickness absenteeism rates (a statistic measuring the same thing) in Czechoslovakia;^a in females no consistent differences were observed. In 1970-1971, in England and Wales, spells of sickness absence per 1 000 males in different age groups ranged from 18.2 to 110 days, and for females from 3.7 to 28.9 days. In 1970, in Czechoslovakia, spells of sickness absence per 1 000 for the same diseases ranged in males from 7.3 to 42.4 days, and in females from 5.4 to 23.7 days. These differences are very probably affected by differences in the criteria for claiming sickness benefit in the United Kingdom or taking sick leave in Czechoslovakia. In England and Wales short spells (less than 4 days) may not be reported, while in Czechoslovakia all spells of incapacity of one day or more are included.

The proportion of sickness absence spells for all respiratory diseases (including influenza) in relation to the total number of spells in Czechoslovakia and the Netherlands in 1973, and in Great Britain in 1974, is presented in Table 2. It can be seen that it has not been possible to assemble the data for these three countries in a uniform manner. This illustrates a further problem when attempting a comparison between data from different

^a WHO Regional Office for Europe. *Respiratory disease in Europe: report on a study*. Copenhagen, 1974 (document EURO 4905(5)).

Table 2. Spells of sickness absence due to respiratory diseases in Czechoslovakia and the Netherlands in 1973, and in Great Britain in 1974

Country	Year	Sex	Cause of temporary incapacity				
			All respiratory diseases		Bronchitis, emphysema, asthma and pneumoconiosis		Bronchitis and emphysema
			% ^a	Mean ^b	% ^c	Mean ^b	% ^c
Czechoslovakia	1973	Males and females	45.1	9.0	1.5	27.0	—
Netherlands ^d	1973	Males	12.9	19.6	—	—	—
		Females	18.5	12.9	—	—	—
Great Britain	1974	Males	35.6	—	—	—	22.3
		Females	36.6	—	—	—	12.8

^a Percentage of all causes.

^b Average duration of one spell in days.

^c Percentage of all respiratory diseases.

^d In 52% of males and in 49% of females the causes of sickness absenteeism were unknown.

Sources:

Ustav pro zdravotnickou statistiku. *ČSSR zdravotnictví*. Prague & Bratislava, 1974.

Ministerie van Volksgezondheid en Milieuhygiene, Centraal Bureau voor de Statistiek. *Vademecum gezondheidsstatistiek*. Gravenhage, Staatsuitgeverij, 1977.

Department of Health and Social Security. *Health and personal social services statistics for England*. London, H.M. Stationery Office, 1977.

countries. However, it can be seen that, of all absences, the proportion ascribed to respiratory disease differs greatly between the Netherlands on the one hand and Czechoslovakia and Great Britain on the other. In the latter countries, respiratory diseases, and more specifically those ascribed to diseases that include CNSLD, are an important proportion of total morbidity.

2.3 Permanent disability

There are few data available on causes of disability. Statistics on severe cases of disease among the working population are not really comparable

because of differing rules and regulations defining disability in different countries. In Poland^a and Romania^b in 1974, respiratory diseases (excluding tuberculosis) were the main cause of incapacity in 6% of the total number of disabled. For Czechoslovakia^c the figure in 1975 was 7%.

In the Netherlands,^d in 1974, chronic bronchitis was registered in 3% of males and 2% of females in the population aged 18 years and older.

2.4 Hospital inpatient morbidity

Information on CNSLD as the principal diagnosis was available from only four countries (Table 3). The proportion of hospital discharges for CNSLD is similar in the German Democratic Republic, Poland, Sweden and England and Wales. However, it must be remembered that the rates may be affected by different hospital admission policies and the availability of beds in each of these countries.

2.5 Outpatient morbidity

Data on visits to physicians are available only from England and Wales, Poland and Sweden. A morbidity study in selected general practices in England was performed in 1970-1971. In Poland a probability sample of patients visiting all outpatient health units throughout the country was investigated in 1967-1968. Data from these studies are given in Table 4. In spite of several differences in the methods employed in these studies, the proportions of episodes and consultations for all respiratory diseases and CNSLD registered in both studies are surprisingly similar. These data refer mainly to persons with symptoms which are severe enough to make them aware of their condition and prompt them to see a doctor, and thus must be an underestimate of persons in the community with this condition.

2.6 Epidemiological studies

Although epidemiological studies of CNSLD have usually examined relatively small population samples, the results have often given useful information on their prevalence; this can be an estimate of prevalence of those

^a Sawicki, F. et al. *Postepy higieny i medycyny doswiadczalnej*, 31: 741 (1977).

^b Ministerul Sanătății, Centrul de Calcul și Statistică Sanitară. *Breviar de Statistică Sanitară 1975*. Bucharest, 1976.

^c Ustav pro zdravotnickou statistiku. *Československe zdravotnictvi*. Prague & Bratislava, 1977.

^d Ministerie van Volksgezondheid en Milieuhygiene, Centraal Bureau voor de Statistiek. *Vademecum gezondheidsstatistiek*. Gravenhage, Staatsuitgeverij, 1977.

Table 3. Hospital discharges for chronic bronchitis, emphysema and asthma by sex in four European countries

Country	Year	Rates (per 100 000 population)		Percentage in relation to:			
		Males	Females	all discharges		respiratory diseases	
				Males %	Females %	Males %	Females %
German Democratic Republic	1971	276	122	2.3	0.8	15.0	9.7
Poland ^a	1961	87	61	1.3	0.8	10.7	10.3
	1972	230	161	2.9	1.8	19.1	18.4
Sweden ^b	1965- 1968	—	—	2.1		—	—
England and Wales	1973	182		1.8		21.1	

^a In Poland only chronic bronchitis and asthma.

^b For the Uppsala Health Region only.

Sources:

Akademie für Ärztliche Fortbildung der DDR. *Mitteilungen. Allgemeines Dokumentationsgerechtes Krankenblatt, 1971*. IX Jahrgang, Heft 2, Berlin - Lichtenberg, 1973.

Sawicki, F. et al. *Zdrowie publiczne*, 87: 501 (1976).

Smedby, B. et al. *Sluten kroppssjukvård i Uppsala sjukvårdsregion 1965-1978. Sjukhusvårdens konsumenter. Socialstyrelsen redovisar. Patientstatistik: 11*. Stockholm, 1972 (SPRI rapport 18/72).

Department of Health and Social Security. *Report on hospital in-patient enquiry for the year 1973: Tables*. London, H.M. Stationery Office, 1977.

severely affected and of those with early disease. An advantage of these studies is that the investigators usually apply similar methods and use similar basic definitions and diagnostic criteria which, theoretically, should allow comparison of their results. However, unified methods of investigation, for example by questionnaire and spirometry, are seldom followed by uniform methods of presenting the results. Suggestions for presentation of data were made some years ago by Fletcher^a and more recently by van der Lende,^b

^a Fletcher, C.M. *Bronchitis*. Assen, Royal Van Gorcum, 1961, p. 273.

^b Lende, R. van der et al. *Nederlands tijdschrift voor geneeskunde*, 119 (1975).

Table 4. Visits for respiratory disease to general practitioners in England and Wales in 1970-1971 and to outpatient health units in Poland in 1967-1968

Diagnosis	Episodes				Consultations			
	Males		Females		Males		Females	
	rate ^a	%	rate ^a	%	rate ^a	%	rate ^a	%
<i>England and Wales</i>								
Total	1 534	100.0	2 062	100.0	2 557	100.0	3 428	100.0
All respiratory diseases	402	26.2	395	19.2	603	23.6	535	15.6
Chronic bronchitis, emphysema and asthma	38	2.5	20	1.0	104	4.1	51	1.5
<i>Poland</i>								
Total	1 606	100.0	1 642	100.0	3 046	100.0	3 267	100.0
All respiratory diseases	439	27.3	366	22.3	612	20.1	538	16.5
Chronic bronchitis, emphysema and asthma	54	3.4	42	2.6	151	5.0	106	3.2

^a Per 1000 population.

Sources:

Office of Population Censuses and Surveys. *Morbidity statistics from general practice. Second national study: studies on medical and population subjects, No. 26.* London, H.M. Stationery Office 1974.

Sawicki, F. & Długasiewicz-Kopczyńska, M. *Zdrowie publiczne*, 84: 98 (1973).

and as a result it has been possible to assemble data from various studies in a reasonably uniform manner. Table 5 presents the results of selected studies covering samples of general populations in Europe. Examination of the published reports on these studies reveals that the reported prevalence of symptoms of persistent cough and sputum, an index of simple chronic bronchitis, can be compared. It can be seen that the prevalence of simple chronic bronchitis is highest in the United Kingdom, and is also high in the urban areas of Czechoslovakia. Prevalence in males in the Finnish rural area and in Yugoslavia is surprisingly high. In all countries prevalence is higher in males than in females, and in urban rather than rural areas.

Table 5. Percentage of persons with symptoms of persistent cough and sputum in selected European countries: results of epidemiological studies

Country and year of study	Area of study	Age	Males		Females	
			No. ^a	% ^b	No. ^a	% ^b
Czechoslovakia 1974	Kutna Hora (rural)	40-59	2 605	13	2 756	5
1964-1974	Several urban areas	40-64	19 876	18-32	15 640	6-14
Denmark 1958	Rønne (rural)	55-64	156	9	-	-
Finland 1961	Harjavalta (rural)	40-64	730	27	890	5
Great Britain 1958	3 urban mining communities	55-64	651	18-65 ^c	511	10-22
1955, 1956	2 rural communities	55-64	173	20-26	184	8-11
Netherlands 1964	Meppel (rural)	40-64	2 018	13	-	-
1965	2 towns	40-64	1 733	13-18	1 604	3-8
Norway 1964	urban	35-74	3 568	13	4 832	4
	rural	35-74	7 031	9	7 778	4
Poland 1968	Cracow (urban)	19-70	1 925	16	2 430	5
Yugoslavia 1969-1970	Bosnia and Herzegovina (rural)	15-64	6 117	10	6 221	3
1964	Tyzla and Remetinec (urban/rural)	35-62	11 933	25	-	-

^a Number of subjects examined.

^b Percentage of persons with symptoms of persistent cough and sputum.

^c Percentages are calculated for the groups tabulated according to place of residence, different occupational history, and occurrence of pneumoconiosis.

Sources:

Novak, M. et al. *Studia pneumologica et phtiseologica Českoslovaca*, 35: 696 (1975).

Olsen, H.C. & Gilson, J.C. *British medical journal*, 1: 450 (1960).

Huhti, E. *Prevalence of respiratory symptoms, chronic bronchitis and pulmonary emphysema in a Finnish rural population*. Copenhagen, Munksgaard, 1965.

Higgins, I.T.T. et al. *British journal of industrial medicine*, 16: 255 (1959).

Sources (contd):

- Higgins, I.T.T. & Cochrane, A.L. *British journal of industrial medicine*, **18**: 93 (1961).
- Higgins, I.T.T. & Cochran, J.B. *Tubercle*, **39**: 296 (1958).
- Lende, R. van der. *Epidemiology of chronic nonspecific lung disease (chronic bronchitis)*. Assen, Van Gorcum & Company, 1969.
- Haenszel, W. & Hougén, A. *Journal of chronic diseases*, **25**: 519 (1972).
- Sawicki, F. & Lawrence, Ph.S. *Chronic nonspecific respiratory disease in the city of Cracow*. Warsaw, National Institute of Hygiene 1977.
- Zarković, G. *Etiology of non-specific chronic respiratory illness and cor pulmonale in Bosnia and Herzegovina*. Sarajevo, Radovi Instituta za Higijenu i Socialnu Medicinu Medicinskog Fakulteta u Sarajevu, 1971.
- Kozerević, D. et al. In: *Ecology of chronic non-specific respiratory diseases*. Warsaw, PZWL, 1972, p. 178.

2.7 Conclusion

From the admittedly incomplete data presented above, it is clear that CNSLD are an important cause of illness in European countries; this also seems to be the case in other parts of the world. They are diseases affecting adults in middle and older age, and mainly men. In addition to mortality ascribed to CNSLD, the diseases are seen to interfere with work and make considerable demands on health services. The patterns of CNSLD are not uniform between countries.

Many factors, including completeness of registration, accuracy of diagnosis, coding procedures, availability of medical services, and legislation, differ from country to country, and may partly explain the differences observed. It is not possible to say to what extent the differences observed in particular countries and population groups are in part due to differences in tobacco consumption, the age and socioeconomic structure of the populations, varying exposure to occupational hazards, air pollution levels, and to other environmental factors. Added to these there are endogenous factors, whose distribution is not known, that may also influence the incidence, prevalence and sequelae of CNSLD.

The Working Group, in reviewing the available data, emphasized the continuing need for reliable data on the significance of CNSLD in terms of mortality, interference with work and as a cause of sickness disability medical care. These data were necessary as a preliminary to the allocation of resources for the control and prevention of CNSLD.

Mortality data on CNSLD in countries where they are already being collected could be investigated to determine how far the differences between countries are real or due to differences in diagnostic habits and coding practices. A few small-scale studies could be mounted with WHO support to investigate these aspects and provide models for future work. In the long

term the objective could be to attempt standardization of mortality data between countries. This would require changes in the current training of physicians and medical undergraduates.

The already very large differences in the way in which data are collected on morbidity ascribed to CNSLD, and in the type of data collected, make it unlikely that worthwhile comparisons will become possible in the near future. If it is thought necessary to make comparisons between countries, then, as with mortality studies, special small-scale investigations could be organized.

Most epidemiological studies have concentrated upon etiological aspects of CNSLD rather than natural history. Their design is thus not usually suitable for obtaining national estimates of the prevalence or incidence of CNSLD. Furthermore, as noted earlier, the format used for publication of findings has rarely permitted worthwhile comparisons between studies. There seems, however, little justification for carrying out national prevalence studies of CNSLD, although, as discussed in the next chapter, further studies of etiology and natural history do seem justified.

3. NATURAL HISTORY

3.1 Definition of chronic lung disease

As with any disease that has a wide spectrum of severity and where progression from minor to major disease may or may not occur, difficulties can arise in precisely "defining" its natural history. Chronic lung disease is no exception. As noted in the preceding section, taking mortality ascribed to CNSLD does not guarantee an accurate measure of the true extent of mortality due to these diseases. In part, this is due to a lack of uniformity in the terms used to describe this group of conditions, which may lead to underestimates of their frequency. This problem is not confined to the recording of cause of death, but applies throughout the clinical field and also in respiratory physiology.

These problems prompted the WHO Regional Office for Europe and the European Society for Clinical Respiratory Physiology to agree on a joint programme aimed at establishing comprehensive definitions for the most commonly used terms in diseases of the lung. This led to the publication in 1975 of *Nomenclature and definitions in respiratory physiology and clinical aspects of chronic lung diseases* in *Bulletin de physiopathologie respiratoire*, Volume 11, 1975, pages 937-959.

The definitions of CNSLD, chronic bronchitis, emphysema and asthma given in this publication are worth quoting here.

(a) *Chronic nonspecific lung disease (CNSLD)*: A general term used to describe the group of conditions in which there is chronic sputum production and/or shortness of breath at rest and/or on exercise.

Comment: There is a transition between the forms in which cough and sputum predominate and the ones in which wheeze and/or shortness of breath are the most important features. Thus in individual cases CNSLD may reflect mainly either chronic bronchitis (with or without airways obstruction) or bronchial asthma or emphysema or any of these conditions in combination. All these types may be acutely or chronically complicated by infections and described accordingly: e.g., infected chronic bronchitis, infected asthma, infected emphysema. Fibrosis, bronchiectasis or other diseases of the lung may be present as associated conditions. The clinical pattern is diverse and lung function data should be considered in this light.

(b) *Chronic bronchitis*: A persistent increase of bronchial secretion.

Comment: For epidemiological purposes may be defined as a productive cough at some time of the day for at least three months in two consecutive years. According to the degree of airway obstruction, one may distinguish two forms of the disease: nonobstructive chronic bronchitis and chronic bronchitis with airways obstruction.

(c) *Emphysema*: A pathological condition of the lungs characterized by an increase beyond the normal size of the air spaces, distal to the terminal bronchioles with destruction of the walls.

Comment: The term emphysema is frequently misused in clinical medicine and clinical diagnosis is often uncertain. Emphysema may be suspected in patients with dyspnoea accompanied by functional changes such as hyperinflation, decrease of transfer factor, loss of elastic recoil, with or without hypoxaemia at rest.

(b) *Bronchial asthma*: A disease characterized by acute attacks of shortness of breath induced by different agents or by exercise, accompanied by clinical signs of bronchial obstruction, totally or partially reversible between the attacks.

Comment: Corresponds to a sudden increase of airway resistance of immunological or nonimmunological origin. Must be differentiated from acute pulmonary oedema of cardiac origin, pulmonary emboli with bronchoconstriction, stenosis of the upper airways. May also occur in chronic bronchitis.

These definitions are essentially based upon clinical observations, including tests of lung function.

The Working Group agreed that, while their deliberations would not include bronchial asthma, they would include CNSLD, chronic bronchitis and emphysema. As CNSLD is a general term describing a group of conditions and would thus include the other two terms, it is used extensively in the rest of the report.

3.2 Natural history of CNSLD

3.2.1 Pathology

In chronic bronchitis there are two major structural changes in the airways:

- (a) mucous gland hypertrophy, concentrated in the more central bronchi and leading to hypersecretion of mucus;
- (b) stenotic and inflammatory lesions of the small conducting airways less than 2 mm in diameter.

In North America, these lesions, which are characterized by mucous plugging, parietal inflammatory cell infiltration and scarring of the bronchioles, when isolated, have been named “small airway disease”.

In emphysema, there are two major changes defining the main types of emphysema:

- (a) centrilobular emphysema, which is characterized by localized destructive changes in the respiratory bronchioles of the acinus. This form of emphysema is associated with an inflammatory process located predominantly in respiratory and terminal bronchioles;
- (b) panlobular emphysema, characterized by a diffuse degenerative process with distension of the alveolar ducts and alveoli and structural changes in the supporting connective tissue framework and of the capillary bed of the lung.

3.2.2 Relation of pathological changes to clinical symptoms and functional abnormalities

Chronic bronchitis: Mucous gland hypertrophy and hypersecretion of mucus usually result in chronic sputum production and predispose to the development of recurrent bronchial infections. The term “simple chronic bronchitis” refers to these structural and clinical changes and usually does not imply any morbidity or disability; it is often observed in smokers. The term “chronic obstructive bronchitis” is used when mucus hypersecretion is associated with an obstructive syndrome.

The major site of obliteration of the airways seems to be in the small conducting bronchioles less than 2 mm in diameter. Numerous peripheral airways need to be damaged and obstructed before significant symptoms and physiological abnormalities, such as increased airways resistance, occur.

Emphysema: Several comparative anatomical functional studies have demonstrated the relationship between morphological emphysema and the

decrease in the lung diffusing capacity. Additionally, a loss of lung elastic recoil could indicate the presence of a significant degree of emphysema. These two tests have been shown to be capable of detecting pulmonary emphysema in asymptomatic patients.

It became apparent in the discussions that, while progress was being made in increasing understanding of the pathological basis for CNSLD, there was scope for further work. The Working Group noted that little was known of the pathological changes present in the early stages of CNSLD. This seemed an area that would benefit from the application of some of the newer techniques such as scanning electron microscopy, transmission electron microscopy and histochemistry.

3.2.3 *Clinical aspects*

A considerable amount of research has been conducted into the clinical features of CNSLD, their symptomatology, pathophysiology, and more particularly, their etiology. While there is general agreement that smoking, particularly cigarette smoking, is the major single factor in etiology, other factors such as air pollution, childhood chest illnesses and personal susceptibility may also contribute.

As indicated earlier, CNSLD do not constitute single disease entity but show a wide spectrum of severity and rate of progression. There is general agreement that severity can range from, on the one hand, no more than chronic cough and no other disability, to severe disability resulting eventually in death. Furthermore, progression in some persons may be slow, while in others it can be rapid, so that over the space of a few years an affected person can change from having only minor symptoms to being severely disabled.

The great variation in clinical features, severity and rate of progression makes a precise description of the natural history of CNSLD impossible at present. However, there have been several epidemiological studies that have included follow-up of populations over a number of years and give some indication of the progression and prognosis in this disease. The Working Group considered the background papers prepared for the meeting in discussing this aspect of natural history. Differing views were expressed on the interpretation of the few studies that had been conducted on the natural history of CNSLD. The general view was that there was insufficient evidence to distinguish discrete syndromes, for which there was both a specific pathology and clinical findings that had different prognoses. One study, where working men had been followed up for eight years, had yielded the suggestion that two syndromes could be distinguished: a hypersecretory syndrome, i.e., chronic phlegm production, which had a good prognosis, and an obstructive syndrome (i.e., air flow obstruction), which led to chronic disability and death.

These ideas are set out in more detail in Annex I. The Working Group discussed this and other evidence on natural history. It was generally agreed that:

- (a) there was insufficient evidence at present to be able to state firmly that specific syndromes could be identified;
- (b) the basis for susceptibility to CNSLD was not understood;
- (c) the reasons for CNSLD progressing in some and not in others were not known;
- (d) as a consequence further studies along the lines referred to in Annex I were needed on the identification and prediction of who would or would not have disease that progressed.

These major gaps in our understanding of the natural history of CNSLD have consequences as far as possibilities for prevention and methods of early detection are concerned (see sections 4-7). The Working Group considered that further epidemiological studies were needed to clarify those aspects of natural history that remained in doubt. This could only be done by conducting further longitudinal studies on populations of adequate size; these populations would need to be followed for sufficient time to allow evolution of the disease to occur. Furthermore, as well as taking account of the more obvious factors, such as smoking habits and occupational exposure, others such as socioeconomic influences, respiratory disease experience in childhood and early adult life, and immunological aspects would also need to be considered in such studies.

4. EARLY DETECTION

The main reason for attempting to detect persons at an early stage in the development of CNSLD is the hope that measures may be used to slow down or even arrest progression of the disease. The Working Group discussed the techniques that might be used to detect early disease, with emphasis on their use in populations.

4.1 Methods for detection of early CNSLD

4.1.1 *Questionnaires*

Extensive use of questionnaires has been made in studies of the etiology of CNSLD and considerable experience has been gained in their development

and application in population studies. Two questionnaires have gained widespread acceptance: the British Medical Research Council's (MRC) questionnaire on the etiology of chronic bronchitis, and the EEC questionnaire that was developed for studies of CNSLD in occupational groups.

Some doubts were expressed about the validity of questionnaires in the early detection of CNSLD. The experience with the MRC questionnaire suggested that it was good at detecting phlegm production and thus made the actual collection of phlegm from subjects unnecessary. It was less good at detecting early airways obstruction, as indicated by responses to questions on dyspnoea. In spite of some reservations, there was general agreement that questionnaires were an essential component of any programme whose objective was the detection of early disease. The Working Group considered that, in preference to having many different questionnaires, the MRC and EEC questionnaires should remain the basic questionnaires, although it might prove necessary, where indicated, for additional questions to be included.

Further study was needed to establish the validity and sensitivity of questionnaires in the context of the early detection of CNSLD. In some populations their use might be impossible, for example, owing to the absence of suitable terms to describe early symptoms.

4.1.2 *Physical and clinical measurements*

Clinical examination. This examination, including recording of chest signs, was discussed. Such examination might be useful for establishing a clinical diagnosis, but was quite unsuitable for application in populations. Firstly, a doctor would be required to conduct such an examination, and secondly, major interobserver biases had been found in these assessments. They were therefore not discussed further.

Chest X-ray. In some special circumstances chest radiology might be necessary, for example, if pulmonary tuberculosis was thought to be prevalent in the population being investigated. However, as a method for the early detection of CNSLD it had no place, as changes in the chest radiograph only occurred in persons with advanced emphysema.

Laboratory tests. Tests of the immune system, such as RAST, and of the lack of α -1 antitrypsin, were considered either too nonspecific or would only detect a fraction of those at risk. Furthermore, they were expensive to perform. They were thus not suitable for population studies.

Respiratory function. The Working Group discussed the place of tests of respiratory function in the early detection of CNSLD and suggested techniques that could be suitable. Certain criteria were stated as being necessary for any lung function indices that were to be used for the early detection of functional abnormalities. These were:

- (a) that the index is sensitive: i.e., the ratio of signal to noise should be relatively high;
- (b) that the index is specific for a certain type of disease;
- (c) that the index is reproducible: under the same circumstances the method yields the same results;
- (d) that the index requires relatively simple apparatus, can be applied under a large variety of measuring conditions and needs relatively little cooperation of the subject.

With these criteria in mind, the Working Group reviewed the various respiratory indices and methods that might be suitable. The discussion that followed was at times highly technical. Some general conclusions emerged. There were no respiratory indices at present that could, with certainty, be stated to be wholly suitable for the early detection of CNSLD. General agreement existed that tests of (a) ventilatory function, and (b) gas mixing were those most likely to contribute to early detection.

Ventilatory function. Measurement of ventilatory function in terms of forced expiration, and not measurement of spontaneous breathing, was stated to be the most appropriate. Specific measurements included the following time-averaged indices:

- (a) forced expiratory volume in one second (FEV_1);
- (b) maximum mid-expiratory flow (MMEF);

and the following instantaneous flow indices:

- (a) maximum (peak) expiratory flow (MEF_{max} ; V_{max});
- (b) maximum expiratory flow when 25% of the FVC has been exhaled (MEF_{25} ; V_{25}).

The following technical specifications in relation to these measurements are relevant:

- (a) measurements should be performed at the mouth, using spirometry or pneumotachography;
- (b) the equipment must be suitable for dynamic measurements and adequate calibration must be available;
- (c) visual display and/or recording of the breathing manoeuvre should be made, if possible, in order to detect technical failures;
- (d) the tests need to be carried out by well-trained technicians;
- (e) the index represents the highest value of at least three technically satisfactory expirations.

It was pointed out that the equipment for performing these tests was widely available and the measurements themselves were routine in many pulmonary function and diagnostic laboratories. As a group, these tests were valid and had a high level of reproducibility.

Gas mixing. Tests that measure gas mixing in the lung were next discussed. Gas mixing can be severely disturbed in CNSLD, and changes can thus occur in the early stage of the disease.

When the technical facilities are available for these measurements, the index of ventilatory capacity, discussed above, is completed by an index which reflects the intrapulmonary mixing of gases. The index of choice appears to be ΔN_2 : the slope of the alveolar plateau (phase III) of the nitrogen concentration, expressed as percentage N_2 per litre expired volume.

The following technical specifications are relevant:

- (a) expired gas volume is measured at the mouth; the nitrogen concentration is measured by gas ionization or mass spectrometry;
- (b) adequate calibration of the nitrogen meter is needed;
- (c) the volume-gas concentration curve is recorded;
- (d) phase III is quantified by the slope of the line of best fit drawn through the alveolar plateau, beginning at the point when 70% of the vital capacity remains to be expired.
- (e) the index represents the mean of at least two consecutive estimations.

Other respiratory measurements were discussed, for example, closing volume, and for various reasons were all considered unsuitable for detecting early changes in respiratory function.

4.2 Populations to be investigated

4.2.1 *General populations or specific groups*

Although CNSLD had been found to be a relatively common condition in many of the populations so far investigated, the Working Group considered that programmes for early detection should concentrate upon populations that were thought to be particularly at risk in relation to the disease, rather than study general population groups. Any detection programme was bound to require considerable resources, and by concentrating upon those populations most at risk the numbers investigated would be smaller and the resources needed fewer.

The Working Group identified three population groups that, on the basis of their exposure to etiological factors, were at risk of developing CNSLD and might thus have an excess of persons with early manifestations of CNSLD. These population groups were:

- (1) current cigarette smokers;
- (2) persons exposed to occupational air pollution;
- (3) persons exposed to other environmental hazards, such as general air pollution.

A further group were persons who had early manifestations of CNSLD.

Finally, the Working Group drew attention to evidence which suggested that adverse childhood respiratory experience was relevant to the onset of earlier manifestations of CNSLD in adult life, and suggested that particular attention should be paid to children with such experience.

5. POSSIBILITIES FOR PRIMARY PREVENTION

It was clear from the discussion on methods for early detection of CNSLD that none of the techniques could be applied without further, and in some cases extensive, development. The Working Group proceeded to consider what scope there might be for the prevention of CNSLD. First discussed was primary prevention of CNSLD. Primary prevention was defined as either the removal from or reduction of exposure to a factor or factors known to produce the disease before manifestations of the disease appear.

5.1 Primary prevention

The most important of the known factors in the etiology of CNSLD is, without doubt, the smoking of tobacco, particularly in the form of cigarettes. Other factors, such as general air pollution and air pollution specific to certain occupations, do appear to have an effect, but this is much less significant than the effects of tobacco smoking. Other factors may contribute to the development of CNSLD. These include respiratory disease in childhood and innate susceptibility to respiratory pathogens and irritants. All these factors may act independently or in combination with each other.

5.1.1 *Detection of those at risk of developing CNSLD*

The preceding section reviewed methods for the early detection of CNSLD and the populations that might be investigated. In primary prevention

a different approach would be used; its aim would be to identify those persons who are exposed to a factor or factors, and then if possible, to identify those in whom CNSLD is most likely to develop.

Estimating exposure may be simple. For example, measurement of smoking in terms of the amount smoked, the type of cigarette, the pattern of inhalation and so on, has been successfully and reliably carried out in many studies. Both the MRC and EEC questionnaires include a section on smoking habits and could be used as the basis for identification of smokers. In contrast, estimates of occupational exposure to air pollution are far less certain, as little reliable data exist concerning air pollution levels to which workers are exposed in specific occupations. Furthermore, even within a specific occupation, the work environment and processes may vary greatly, so that no air pollution level could be defined as typical for that occupation. The best that can be done would be to obtain an occupational history from workers and identify those jobs with a probable high exposure to air pollution. Both the MRC and EEC questionnaires have included questions on occupational history, from which crude estimates of exposure might be obtained.

A similar problem arises in estimating exposure to general air pollution. While it may be possible to list all the places where a person has lived from birth, and the time spent in each place, the associated air pollution data may not be available.

One conclusion seems inescapable, namely, that the only reliable data on exposure which could be readily obtained are those relating to cigarettes and other tobacco products.

A potential further refinement would be the ability to detect persons at risk of developing CNSLD if exposed to one or more of the factors already discussed. At present there is no reliable way of detecting such susceptible persons in the general population.

5.1.2 Scope for reduction or elimination of exposure to etiological factors: smoking

The Working Group considered the feasibility of smoking control and particularly what had so far been achieved in this direction. It was emphasized that, although smoking had been prevalent for a long time, attempts at control were a relatively new development. In fact, work on smoking control could still be considered as being in a pioneering stage. It was thus hardly surprising that smoking control programmes had resulted in a mixture of success and failure. It was too early to be specific about what the various components of such programmes might be, but it was suggested that they should serve at least four main objectives:

- (1) creating a widespread understanding of the reasons for smoking control;

- (2) preventing the onset of smoking;
- (3) changing smoking behaviour;
- (4) establishing a social climate orientated towards nonsmoking.

Objective (1) was a necessary preliminary and formed the basis for all the others. Objectives (2) and (3) were the core of the programme, while (4) was a longer-term objective that was needed to preserve and sustain the achievements gained under (2) and (3).

It is clear that a smoking control programme must contain a wide variety of activities to cover all these and other subsidiary objectives. Although the Working Group was discussing smoking control in the context of CNSLD, any such programmes would necessarily include statements about the risks of lung cancer and cardiovascular disease resulting from smoking. It was noted that programmes which were primarily directed at changing the smoking habits of adults would also have an impact upon the attitudes and smoking behaviour of children. However, as it was so important to discourage children from taking up the habit, there was a need for health education to be given to them both in the preschool period and during their school years.

There was agreement that health professionals had an important role to play in smoking control programmes. Attention needed to be drawn to their potential in providing antismoking education whenever the opportunity arose. Training and encouragement in this work would be necessary.

The Working Group recognized that a majority of smokers would probably be able to give up smoking following exposure to a minimum-intervention procedure. A limited number of hard-core smokers might need a more intensive programme. It was probably here that smoking withdrawal clinics could have a useful role.

Some smokers might be unable to give up the habit, and in such cases advice was needed on ways of modifying their smoking behaviour in an attempt to lessen its risks. This could take the form of, for example, switching to brands yielding lower amounts of noxious substances, taking fewer puffs and leaving longer butts.

In efforts to achieve the four objectives for smoking control discussed earlier, there was a need for governments, organizations, both national and international, and the individuals concerned to make this proper contribution.

5.1.3 General and occupational air pollution

The contribution of air pollution to the etiology of CNSLD is, as noted above, probably not large in comparison with that of smoking. The practicability of reducing general air pollution by community action has been amply demonstrated, for example, in the United Kingdom. Here legislation was enacted that greatly reduced the levels of smoke that were allowed to

be emitted from coal fires. This was achieved by treating coal so that less smoke resulted when it was burnt, by introducing modified grates, and by increasing the use of gas and oil as fuel for domestic and industrial heating. There is evidence that the resulting dramatic reduction in air pollution has been accompanied by a reduction in mortality from CNSLD.

While successes have undoubtedly been achieved in reducing general air pollution, less is known about the levels and types of air pollution in specific occupations, and, except in a few notable cases, little progress has so far been made in removing or limiting such exposure. While there is clearly scope for development, this may be limited by lack of knowledge concerning air pollution levels and lack of willingness on the part of industrial enterprises to spend money on necessary changes. The inability to identify susceptible persons would also make it more difficult to suggest that persons should change to jobs where air pollution exposure levels were lower.

6. POSSIBILITIES FOR SECONDARY PREVENTION

In contrast to primary prevention, secondary prevention has the objective of identifying persons who already have manifestations of the disease and, by reducing or removing exposure to the relevant etiological factors, coupled, where appropriate, with medical treatment, halting progression of the disease or in certain circumstances causing it to regress.

An earlier section covered the methods that could be used to detect early CNSLD and populations that might be investigated. It is necessary here to discuss again how information on exposure to etiological factors should be obtained, to examine the effects of removing or reducing exposure, and finally, to consider the likely benefits of medical treatment.

In the previous section it was concluded that estimates of the exposure of individuals to cigarettes and other tobacco products could be readily obtained, whereas there are major problems in estimating exposure to air pollution in general and to occupational factors in particular.

When data on exposure are available, it may not always be possible to decide, where more than one factor is present, the relative contribution each makes to the disease. The factors may have been exerting effects, alone or in combination, over a long period before manifestations of disease appear. This leads one to consider what effects the removal of exposure to a factor might have.

Clinical impressions, now backed up by well-based evidence, indicate that, in smokers who have phlegm production, ceasing to smoke can lead to a reduction or even disappearance of phlegm. There is also evidence that the rate of loss of lung function in persons with early obstructive disease slows

down when smoking is given up.^a These findings should encourage doctors and patients alike to understand that giving up smoking is an important component in the clinical management of persons with early CNSLD. Doctors should advise all such patients to cease smoking and should support them in this action.

On the other hand, when obvious airways obstruction is present, such changes may be irreversible, whatever is done to remove exposure to etiological factors. Here, however, there may be worthwhile gain if, with such action, the disease does not progress. The same comments could be made about occupational air pollution exposure, where there is less evidence on the effects of removing individuals from such exposure. This is an important area for further research. The Working Group concluded that it was not possible to state with certainty to what extent the removal of exposure to etiological factors would lead to a reduction in CNSLD.

6.1 The role of medical treatment

The Working Group next discussed the contribution medical treatment might have in limiting the progression of CNSLD or causing regression.

6.1.1 *Antibiotics*

There was no general agreement on the place of antibiotics in the management of even established CNSLD. The rationale for their use is based on the observation that in some acute exacerbations, when there may be fever or increased sputum production which may be purulent, bacterial infection can be present. This has led to the widespread use of antibiotics, particularly broad-spectrum antibiotics, not only to treat these exacerbations but as long-term prophylaxis. Little evidence is available, from well-based studies, that in most circumstances antibiotics contribute materially to limiting these episodes. While there are specific and limited circumstances when they should be given, antibiotics do carry risks, for example in producing antibiotic-resistant strains, and furthermore they are expensive.

The Working Group could not decide on the precise role antibiotics should have in treating early CNSLD, but agreed that there was a need for further controlled clinical trials to assess the use of antibiotics in CNSLD, particularly in the early stages of the disease.

6.1.2 *Bronchodilators*

These drugs are used to facilitate bronchial patency. Little evidence is available on their long-term effectiveness, particularly in early CNSLD.

^a Fletcher, C. & Peto, R. The natural history of chronic airflow obstruction. *British medical journal*, 1: 1645 (1977).

6.1.3 *Nonsteroid anti-inflammatory drugs*

These may have a place in reducing bronchial oedema and improving bronchial patency.

6.1.4 *Proteolytic enzymes*

These have been used to reduce the viscosity of bronchial secretions, thus leading to improved mucociliary clearance. Their use is not without the risk of adverse side effects.

6.1.5 *Cystein derivatives*

These are known to favour the synthesis of mucins rich in sialic acid which may lead to improved mucociliary clearance. These derivatives also have an anti-inflammatory action on the bronchial mucosa.

As with antibiotics, little is known of the precise place any of these drugs have in the treatment of early CNSLD. Again, the Working Group drew attention to this lack of knowledge and suggested that rigorous assessment of them was needed in the management of CNSLD.

6.2 **Breathing exercises**

The possible role of these in treating CNSLD was discussed. Although a case could be made for their use both in improving the general efficiency of breathing and in clearing local areas of atelectasis, there was little evidence to indicate that they offered any but the smallest benefit in established CNSLD. If it was thought possible that breathing exercises had a beneficial effect in early CNSLD, then this treatment required to be rigorously assessed.

6.3 **Rehabilitation programmes**

Such programmes appear to be widely used for persons with established and disabling CNSLD. Evidence for their benefit is almost wholly lacking and studies are required to assess their role, if any, in the management of this group of diseases.

The Working Group concluded that there was insufficient evidence to make any firm statement on the effects of treatment in altering the natural history of CNSLD when detected in the early stages, and reiterated the need for randomized controlled trials, of adequate size, to assess these treatments. It was recognized that there was a need to educate doctors in the management of CNSLD; this applied particularly to general practitioners, who provided most of the medical care for patients with CNSLD. The results of

randomized controlled trials of various treatments should be brought to the notice of all doctors and others involved in the management of these diseases.

7. ORGANIZATIONAL ASPECTS OF PRIMARY AND SECONDARY PREVENTION

The Working Group discussed ways in which the primary and secondary prevention of CNSLD might be organized.

As noted earlier, a major component in any programme would be a continuing effort to persuade people not to start smoking, and, for those who already smoke, to give up or at least reduce the amount they smoke or change their smoking habits. The way such programmes are conducted will vary from place to place. However, there is a need for much greater effort on the part of governments and of national and international organizations to support the development, evaluation and coordination of such programmes. These problems have been considered in more detail by the WHO Expert Committee on Smoking Control.^a Further support is needed, particularly by national governments, for any action to reduce general and occupational exposure to air pollution. This is an area in which trade unions and workers should be encouraged to take an interest. Workers should be made aware of the need to take steps to maintain their health, for example, by not smoking.

Reference has already been made to the combined effects on health of smoking and exposure to occupational air pollution. This danger needs to be brought to the attention of employers, trade unions, workers and health authorities, as well as persons working in the industrial health services. Collaboration will be needed between these groups if prevention is to be seriously pursued. As a start, antismoking advice and the restriction of smoking at work could be implemented quickly at little cost.

Secondary prevention would require the detection of persons with early manifestations of disease, with a view to offering such persons advice and support on limiting their exposure to adverse factors, and to prescribing appropriate treatment. It is clear that the detection of such persons would probably involve the screening of populations or groups characterized by their smoking and/or occupational and general exposure to air pollution. The Working Group was not aware that any such specific screening programme had been set up, and considered it essential that the need for screening programmes to detect early CNSLD should be adequately considered before their use was recommended.

^a WHO Technical Report Series, No. 636, 1979 (*Controlling the smoking epidemic: report of the WHO Expert Committee on Smoking Control*).

Various criteria have been proposed for judging whether screening for a disease should be advised. Some of these criteria were discussed by the Working Group in relation to CNSLD and are set out below, with comments.

(1) The disease to be screened for is important. In an earlier section attention was drawn to the extent of mortality ascribed to CNSLD. Morbidity, and in particular disability, is sufficient to place the disease among the most common affecting adults.

(2) The disease is latent at a stage, i.e., a stage when only minor changes are present. If respiratory symptoms denote early stages then this criterion is satisfied.

(3) The natural history is understood. Knowledge of this is incomplete at present.

(4) Suitable tests are available to detect early stages. This is true as far as chest symptoms are concerned, but information on the suitability of other tests, e.g., tests of respiratory function, is incomplete.

(5) There is an accepted treatment. In early CNSLD the cessation of smoking may result in a lessening or disappearance of symptoms; in more advanced disease there is doubt as to the effects of treatment.

(6) Facilities are available for diagnosis and treatment. In many countries these facilities exist, but they may not be adequate to cope with the extra work generated by a screening programme.

(7) The costs of case-finding are to be adequately balanced in relation to other medical care expenditure. These costs cannot be estimated at present and need to await pilot screening studies.

(8) Case-finding is a continuous process. This would be linked to costs and facilities and implies a long-term commitment to screening.

The Working Group noted that, while certain of these criteria were satisfied, others were not. In particular, the natural history was not fully understood, the validity of tests for the early detection of airways obstruction was uncertain, and the effects of reducing environmental exposure and treatment in the early stages of the disease were unknown. These reasons were more than sufficient for the Working Group to recommend strongly that population screening should not be introduced. Only when more was known about the above aspects would pilot studies of screening become justified.

8. CONCLUSIONS AND RECOMMENDATIONS

1. The available evidence indicates that tobacco smoking is the most important of the known causes of CNSLD and that the cessation of smoking in the early stages of the disease can slow down or even arrest its progression. The prevention of CNSLD should therefore include the following measures.

(a) United Nation's agencies dealing with health education, together with governmental and nongovernmental organizations active in this field at both national and international levels, should play an increasingly significant role in smoking control programmes.

(b) Even though smoking control programmes aimed at changing the habits of adults automatically have an impact on the attitudes and smoking behaviour of children, special health education programmes directed at children should start at preschool age.

(c) Smoking education programmes should include statements about the risk to smokers not only of counteracting lung cancer, but also their developing CNSLD and cardiovascular disease.

(d) Members of the health professions, who have an important role in smoking control, should recognize that antismoking education is an important component of the management of CNSLD. In their routine contacts with patients who smoke, even with those who show no symptoms of respiratory disease, appropriate advice on stopping smoking should be given. Advice should be given to those who have special difficulties in giving up the habit on ways of modifying their smoking behaviour so as to reduce the health risks involved.

(e) Those members of the health professions who smoke should consider their responsibility to set an example by giving up the habit. If they are unable to do this, they should at least refrain from smoking in front of their patients.

(f) The social climate should be modified towards decreasing the acceptability of smoking in order both to reduce smoking and to ensure the right of nonsmokers not to be inconvenienced by tobacco smoke at their places of work and in public places.

(g) Air pollution and other environmental factors contribute to the development of CNSLD; consequently, governments should continue to promote any action aimed at reducing air pollution in general and in specific industries.

- (h) As smoking results in significant health hazards when combined with exposure to adverse environmental factors in many occupations, workers should be made aware of their own responsibility in helping to maintain their own health by not smoking. In addition, employers, trade unions, health and other authorities should recognize the possible consequences of such combined exposure and collaborate in the protection of workers' health by providing antismoking advice, introducing restrictions on smoking at places of work, and by reducing levels of exposure to potentially hazardous occupational factors.
2. Routinely collected data on CNSLD may be of value in defining the extent, severity and impact on health services, and on work, of this group of conditions. However, these data are often inadequately analysed, and further efforts should be made to use them more fully so that health and other authorities are better equipped to plan for the control and prevention of CNSLD.
 3. Since the diagnosis and management of patients with CNSLD is usually carried out by the primary health care physician, adequate training should be given, where needed, in this connexion.
 4. There is a lack of knowledge about the natural history of CNSLD, particularly concerning the reasons for the progression of the disease in some persons and not in others. Longitudinal studies which take into account both childhood respiratory experience and that in early adult life are needed.
 5. The validity of currently available tests for the early detection of airways obstruction is uncertain. Research is urgently needed to develop techniques for investigating ventilatory capacity and intrapulmonary mixing of gases.
 6. The value of therapeutic methods in the management of CNSLD is also uncertain, and there is a need for adequate evaluation of the wide range of drugs currently being used. This can best be done by means of randomized controlled trials. The same is true with regard to rehabilitation programmes for those more severely affected or disabled by CNSLD.
 7. General population screening should not be introduced at present, in view of doubts as to whether, with current available methods, the early stages of CNSLD can be detected and whether treatment can influence the course of these diseases.
 8. As there are a number of aspects of CNSLD that require further research, WHO should support, develop and coordinate appropriate activities. These could include training programmes on the etiology, diagnosis and management of CNSLD, publicity on the role of smoking and air pollution, further studies on natural history, and the development of methods for the early detection of CNSLD.

Annex I

HYPOTHESIS ON CNSLD BEING TWO MAJOR SYNDROMES

R. Peto^a

These ideas stem from the work by Fletcher and co-workers as reported in 1976.^b They are summarized below and set out in the table.

Chronic airflow obstruction and chronic phlegm production are conditions which affect different anatomical sites within the lung, and either condition may quite commonly be present without the other. Although tobacco smoking is an important cause of both conditions, chronic phlegm production does not greatly predispose individuals to the more rapid development of early chronic obstructive lung disease. Finally, the relationship of smoking to the two conditions is qualitatively different. Chronic phlegm production may start within a few years of starting smoking, and may cease within a year or two if the smoker gives up the habit. In contrast, in non-asthmatic smokers the development of chronic airflow obstruction is spread over decades, and, once present, the airflow obstruction will not disappear if smoking ceases.

There is thus reason for keeping chronic phlegm production ("hypersecretory syndrome") and chronic airflow obstruction ("obstructive syndrome") conceptually separate.

Once they are separated, it is simple to recognize that the chief object of preventive medicine should be to prevent chronic airflow obstruction rather than to prevent chronic phlegm production. This is because chronic obstruction disables or kills people while, in the absence of chronic obstruction, chronic phlegm production does not, but merely predisposes to infective episodes which are usually a nuisance rather than a danger, given the standards of nutrition and medical management typical of developed countries. Although affected individuals may have either of these conditions, some may have both.

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^b Fletcher, C. et al. *The natural history of chronic bronchitis and emphysema*. Oxford University Press, 1976.

Table. Distinctive characteristics of "hypersecretory syndrome" and "obstructive syndrome"

Name proposed by Fletcher et al., 1976	Main clinical findings	Chief pathological cause	Future effects	Effect of removing stimulus
1. Hypersecretory syndrome	Chronic phlegm production and/or recurrent clinical infections	Mucous gland hyper- trophy in the large airways	Not serious ^a	Often remits
2. Obstructive syndrome	Chronic dyspnoea; airflow obstruction	Many qualitatively different changes in the periphery of the lung (emphysema, small airways disease, etc.)	Chronic disability and ultimate death	Usually irreversible

^a Not serious (given the standards of nutrition and medical management of acute infections typical of developed countries) except as an indicator of exposure to noxious agents which may affect other sites.

Annex II

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