CHRONIC CATARRHAL BRONCHITIS.

A CLINICAL STUDY FROM GENERAL PRACTICE.

CHRONIC CATARRHAL BRONCHITIS - A CLINICAL STUDY FROM GENERAL PRACTICE.

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BY

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Containing

A Historical, Physiological and Aetiological Review of the English Literature of Chronic Catarrhal Bronchitis with Special Reference to the Occurrence of the disease in the British Isles

and

to the Writer's Personal Experience of cases during six years in General Practice.

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C O N T E N T S.

INTRODUCTION.

As a distinct clinical entity Fry (1954) considers that chronic bronchitis is one of the most important of diseases from the point of view of national health and personal suffering. This applies both numerically and socially. It is only in general practice that it is seen in all its stages and in all the effects it produces on the individual and the family group.

The disease is in some degree responsible for over 30,000 deaths annually in Great Britain. The mortality rate from this disease in England and Wales is far and away the highest in the world. It is some thirty times that in the United States of America and statistics show that every year the death rate from bronchitis in England and Wales is much higher than in other European countries for which reliable figures are available.

The following Table shows comparative death rates from bronchitis for England and Wales and the Scandinavian countries and is published by the Committee on Air Pollution (1954).

Death Rates from Bronchitis per 100,000.

Country	Year.	Males.	Females
Denmark.	1951.	2·2	1·9
Norway.	1951	5·5	5·8
Sweden.	1951	5·0	4·0
England and Wales.	1951	107·9	62·7
	1952	83·8	42·0
	1953	91·9	47·6

According to a Report of the Ministry of National Insurance 1950, chronic bronchitis has been estimated to account for the loss of some sixteen and a half million working days a year, and this does not include the condition as it affects the housewives, who, although an important and essential working group, do not receive insurance benefits. (Fry 1954).

Other factors which make the disease one of such major national importance are its long duration, often causing trouble for twenty-five to thirty years, and the fact that each winter it causes a considerable loss of worktime. In addition, it affects principally middle-aged men in their most active and able years, which makes the absence from work of even greater significance. (Fry 1954).

The fact that it is probably largely a preventable condition adds to its importance and introduces an urgent and imperative need for further researches and actions in this field. (Fry 1954).

Professor James Mackintosh in the first John Matheson Shaw lecture of the Royal College of Physicians of Edinburgh in November 1954, discussed the opportunities for research now opening before general practitioners and mentioned chronic bronchitis as one of possible inquiries.

He said that "there is a danger inherent in the work of general practice that the very great variety of subjects for practice and thought in a day's work may lead to a superficial and desultory curiosity rather than an eagerness for study".

The importance of research is also stressed by Fry (1954) who says that in general practice the true incidence and course can be observed.

Schilling, Hughes and Dingwall-Fordyce (1955) write that the investigation of the incidence and severity of a disease in a community may depend on the clinician's skill in taking histories and observing physicial signs.

Keith Westlake (1954) thinks that it is unlikely that there will be any substantial fall in the overall mortality from "bronchitis" until the incidence of chronic bronchitis and emphysema is itself

reduced. This will depend on the discovery of all the aetiological factors involved. He stresses overcrowding, excessive smoking and atmospheric pollution as probably important factors.

Fry (1954) thinks that there is a definite relationship between climate and atmospheric pollution and the disease and points out the fact that 4,500 deaths in December 1952 in London were directly attributable to fog. He also stresses the importance of studying the possible influences of smoking, climate, atmospheric pollution and any other factors which may present themselves.

Joules (1954) defines chronic bronchitis as a cough causing increasing illness and loss of working capacity, unassociated with any specific abnormality in lung structure and usually unassociated with any specific infecting organism. Bronchitis is a cumulative disease according to Joules and each set-back - such as a severe fog, infection, or excessive exposure to dust - may lead to increased lung damage and a diminished expectation of life. Until a certain stage is reached it is reversible and good health can be restored. Steps must be taken sufficiently early to discover the factors which irritated the bronchi and maintained the cough.

Careful history-taking will often reveal a fascinating and formidable group of factors. Joules

mentions social factors concerned with place of residence and social class, infections, occupational factors, smoking, infected teeth and sinuses, allergy and gassing in the 1914-1918 World War.

In this thesis, after a study of the literature particularly of the aetiology of chronic bronchitis, some of these fascinating and formidable factors were studied and reviewed in seventy-five patients with chronic bronchitis studied in my general practice over a period of six years from 1950 up to 1955. Thus a study could be made of numerous factors in relationship to the aetiology of the disease.

It is as the cases present themselves to the general practitioner that this study is based.

This study cannot claim to be in any way complete and only approaches certain aspects on the subject of chronic bronchitis. The original part is limited to the studies carried out in general practice.

The number of cases studied is limited by the number of cases seen in general practice and it is realised that the conclusions drawn would more properly be called impressions, but is from impressions that the better understanding of disease has come.

PART ONE.

REVIEW OF THE LITERATURE.

HISTORICAL:

The term "Bronchitis" was first used by Charles Badham in the year 1808. (Howell 1951). He was Physician to the Westminster General Dispensary and his book entitled "Observations on the Inflammatory Affections of the Mucous Membranes of the Bronchiae" is still the classic treatise on this group of diseases.

Prior to the year 1808, other writers have passages which suggest that they were not unfamiliar with bronchial affections.

Hippocrates wrote that "old men suffer from difficulty of breathing, catarrh accompanied by coughing". Aretaeus, the Cappadocian, who lived in the reign of Nero, wrote several excellent clinical descriptions of pulmonary diseases, among which was one termed "pneumodes". This appears to correspond in many ways with chronic bronchitis, even to its common termination in congestive cardiac failure.

Dr. Douglas Guthrie in his textbook on the History of Medicine writes that it was prior to the year 1808 that the pulse was first accurately timed, the clinical thermometer was used to any extent and

the first account of percussion as an aid to diagnosis was written.

Sir John Floyer of Lichfield (1649-1734) first accurately timed the pulse when he introduced his Physician's Pulse-Watch which ran for one minute. James Currie (1756-1805) checked the treating of typhoid fever with cold baths by thermometry. Leopold Auenbrugger (1722-1809) had published in Vienna in 1761 a monograph of ninety-five pages which contained the first account of percussion as an aid to diagnosis.

The next great advance in the description and classification of the bronchial diseases, after the term "Bronchitis" was first used in 1808, was the publication in 1818 of "Traite de l'auscultation mediate" by René Théophile Hyacinthe Laënnec (1781-1826).

Laennec describes the sounds heard by the stethoscope, which he discovered, and also coined new terms such as crepitations, rhonchi, pectoriloquy and aegophony. In the second edition he added a detailed account of the diseases of the chest as then known.

John Mackintosh of Edinburgh, in the second edition of his book entitled "Elements of Pathology and Practice of Physic" which was published in the year 1831, draws our attention to the

fact that chronic bronchitis frequently coexists with diseases of the heart, and is a cause of dropsical affections. In his book, a discussion of the mechanism of bronchial spasm was in advance of the time.

In the nineteenth century there was overcrowding in the old towns. Hunter (1955) writes that the window tax, first imposed as a temporary measure in 1696, still served to discourage proper lighting and ventilation. Architects and builders of any houses bigger than small cottages were naturally encouraged to devise structures with as few openings as possible. In most new houses, privies, closets, passages, cellars and roofs were left without ventilation. The tax was finally repealed in 1851.

Describing social reforms in the nineteenth century and describing the evil state of the new towns with textile and metal industries Hunter quotes the following description from "Hard Times" by Charles Dickens. This book was published in 1854 and in it Dickens describes a fictitious new town of the nineteenth century called "Coketown" as follows :-

"On every side, and as far as the eye could see into the heavy distance, tall chimneys, crowding on each other and presenting that endless

repetition of the same dull, ugly form, which is the horror of oppressive dreams, poured out their plague of smoke, obscured the light, and made foul the melancholy air!!

A campaign for public health and hygiene began in 1838, and the man who took the first step was the great lawyer-sanitarian Sir Edwin Chadwick (1800-1890). By showing how the census and bills of mortality could be used to diagnose public ailments, he initiated the sanitary era of public hygiene. His inquiries included building, town-planning, sewers, water supplies, burial grounds, open spaces, lodging-houses and slums.

The clinical lectures of Graves, published in 1848, pay much attention to aetiology, climate and occupation of the patient.

According to Guthrie, the first attempt at a scientific analysis of the symptomatology of winter coughs was made by Horace Dobell at the Royal Hospital for Diseases of the Chest in London. His book, first published in 1866, discusses the mechanism of emphysema, the importance of post-nasal catarrh, the significance of family history and various methods of treatment.

Slightly later than Dobell, Milner Fothergill wrote his book on chronic bronchitis in a different style and he gave one of the most detailed clinical

presentationsof any disease to be found in the medical literature.

About this same period, Samuel Jones Gee delivered the Lumleian Lectures on "Bronchitis, Pulmonary Emphysema and Asthma" before the Royal College of Physicians in 1899.

Samuel West, who was a contemporary of Gee on the staff of St. Bartholomew's Hospital, also wrote on chronic bronchitis furnishing statistical tables of distribution, frequency and mortality in bronchitis, as well as many illustrative clinical histories in a book entitled "Diseases of the Organs of Respiration". This book was written in 1902.

Between 1884 and 1886, Comby of Paris revealed the fact that chronic bronchitis can exist alone in babies and children.

Since the twentieth century came in, numerous books and articles have been written on chronic bronchitis.

The social reforms of the twentieth century have introduced less overcrowding and congestion in housing by the building of new housing estates in places as far as is possible away from factories.

Finally one of the reforms, which is of greatest importance to the chronic bronchitic invalid, is the proposal of the principle of smokeless areas as a method of reducing smoke pollution in our industrial towns. This proposal was put forward in 1935 by Charles Gandy who is a Manchester barrister. (Leading Article British Medical Journal 1953).

PHYSIOLOGY.

From a study of the literature I have tried to elucidate the physiological disturbances and mechanisms which may be encountered in patients suffering from chronic bronchitis in the following five groups :-

1. Disturbances of the Autonomic Nervous System.
2. Special Vascular Constitution.
3. Disorders of Ciliary Action.
4. Changes in Mucus Secretion.
5. Interference with the normal exchange of gases in the lungs.

1. Disturbances of the Autonomic Nervous System :-

Samson Wright (1952) describes the respiratory centre which is subdivided into inspiratory and expiratory centres situated in the medulla and the pneumatoxic centre situated in the upper pons. He writes that the respiratory centre is responsible for automatic rhythmic respiration.

Cooper (1948) also explains that the respiratory centre possesses an inherent rhythmicity and controls the activity of the lower motor neurones supplying the respiratory muscles. It receives afferent impulses from the lungs via the vagus and these afferent fibres are sensitive to the amount of expansion of the lungs inhibiting inspiration when it has proceeded to a certain extent; expiration then follows (Hering-Breuer reflex). Other mechanisms in

regulation of breathing are not studied in this thesis.

The chief motor nerves applying the thoracic cage are the phrenic and intercostal nerves. Intrinsic lung innervation is by fibres from the vagus and the sympathetic. Vagal efferent fibres are broncho-constrictor while sympathetic fibres are broncho-dilator. Nerve fibres to and from the pulmonary blood vessels are of no particular importance, for in the pulmonary circulation the blood pressure and the velocity of blood flow are much smaller than in the systemic circulation.

J.B. Christopherson (1933) writes that, in his opinion, chronic bronchitis is fundamentally due to dominant sympathetic influences which cause relaxation of the musculature of the bronchial tree among other symptoms.

In his opinion, chronic bronchitis may be looked upon as an exaggeration of the inspiratory phase of the respiratory complex compared with spasmodic asthma which is an exaggeration of the expiratory phase.

The effects of bronchitis are seen chiefly in the medium-sized tubes according to Christopherson, where most of the pressure of the explosive cough is felt, and where the walls are relatively less well supplied with muscle. The result is that when they are frequently in a condition of relaxation, they are

liable to become permanently dilated (as in pertussis). Although knowledge of the autonomic nervous system (the only nervous supply of the lungs) has not been applied clinically to bronchitis according to Christopherson, the system of living air channels is however continuously changing in calibre, controlled by the action of the vagus and sympathetic nerves on the abundant musculature of the bronchi and bronchioles.

Roper (1942) writes that it seems therefore that the vago-sympathetic nervous supply, itself influenced by the psychological make-up and reactions of the individual, may, when disordered, give rise at one extreme to asthma and the other to bronchitis and bronchiectasis.

2. Special Vascular Constitution :-

Gorlitzer von Mundy (1954) found by experiments with cupping glasses that the skin vessels of subjects with chronic bronchitis showed a different response from those of normal subjects. The author therefore contended that there may exist a special vascular constitution by which certain subjects are predisposed to bronchitis. The author concluded that apart from an infection, thorough chill or drenching, a definite disposition or constitution is necessary for the development of bronchitis.

3. Disorders of Ciliary Action :-

Negus (1949) writes that those dealing with respiratory diseases should never lose sight of the dependence of cilia in the physiological processes on the nose and sinuses, with a corresponding danger when the mechanism is upset, the upper air passages then becoming a source of danger instead of a means of protection of the lower air tracts. In man cilia line the greater part of the respiratory tract but there are no cilia at the extreme anterior part of the nasal fossae - that is, the region in front of the inferior turbinal bodies; nor are there cilia in the air vesicles or atria of the lung. The vocal folds have a squamous epithelium.

Drying produces disordered action of the cilia as the mechanical actions of the ciliary streams are interrupted and furthermore the absence of mucus removes the protective action of lysozymes. Drying is caused by residence in overheated and undermoistened rooms, when air-conditioning is inefficient; it appears also in the dry atmosphere of Western Australia and in the high altitudes of the Himalayas.

Mechanical obstruction produces disordered ciliary action. This may be caused by a plug of viscid mucus.

Negus (1949) concludes his article by stating that if ciliary action maintains its efficiency, then there will be no penetration of the mucosa by bacteria and no strain will be thrown on the leucocyte second line of defence. In cases of chronic bronchitis there may well be disorders of ciliary action.

4. Changes in Mucus Secretion :-

Oswald (1954) writes that the term "mucus" denotes a viscid material secreted on a mucous membrane. Mucin consists almost entirely of a complex combination and mixture of proteins and carbohydrates, which vary from one type of mucin to another. In health the whole of the respiratory tract is covered with a thin layer of mucus, which is derived from the goblet-cells and mucous glands. This covering is essential for the proper conditioning of the inspired air.

Solid particles adhere to its surface, and noxious gases are largely dissolved before the air reaches the alveoli. Humidification, which takes place mainly in the nose (Negus 1952), involves the transference of about four hundred cubic centimetres of water from the mucosa to the inspired air in each twenty-four hours, in the English climate; the efficiency of this mechanism is determined largely by the thickness and consistency of the layer of mucus.

Elmes (1953) writes that the mucus catches the particles of dirt and bacteria in the air and is constantly driven along the bronchi towards the trachea and larynx by the cilia of the cells on the surface of the mucous membrane. When it reaches the larynx, the secretion with its load of dust and bacteria is swallowed and destroyed in the stomach.

Oswald (1954) thinks that the relationship between infection and bronchial mucus constitutes the essence of the pathogenesis of chronic bronchitis.

In chronic catarrhal bronchitis the volume of the secretion is greatly increased so that the film of the surface of the mucous membrane reaches appreciable thickness, and during expiration may block the lumen of the smaller bronchi. (Elmes, 1953). The inflammatory process in the mucous membrane reduces the number of cilia so that the means of moving the secretion fails and it accumulates. Small bronchi fill up and remain blocked until the secretion is removed by coughing or gravitational drainage. This stagnation of the secretions encourages the growth of bacteria so that the infection of the mucous membrane is maintained.

Bronchitic patients often have a mucoid sputum, particularly during remissions. To what extent the mucus is a result of hypersensitivity, infection, and external irritants is not clear, but

presumably all three play a part. (Oswald, 1954).

Following the infection, the bronchi become hypersensitive, being particularly susceptible to further infection and to external stimuli, and they often secrete large quantities of mucus. The hypersensitivity of chronic bronchitis is particularly related to infection, atmospheric pollution, and climatic factors. (Oswald, 1954).

External stimuli seem to be able to condition the bronchi in such a way as to render them more susceptible to infection. This view is suggested by the age of onset of symptoms which will be discussed in the appropriate section.

Thus Oswald (1954) concludes that various factors seem to operate at the onset of bronchitis, the most important being infection, external stimuli, and hereditary predisposition. Once the bronchi have become sensitised, any of the irritants, such as atmospheric impurities, tobacco-smoke, dusts, fumes, chemicals or heat, may aggravate symptoms, the most prominent of which is excessive mucus; but this effect is often overshadowed by the irritation caused by fog, dampness and cold.

5. Interference with the normal exchange of gases in the lungs :-

Elmes (1953) writes that in pulmonary disease the function of the lungs may be disturbed in three ways :-

(1) The lungs may become less elastic so that the maximum volume of air which can be drawn in with each breath (the vital capacity) is reduced.

(2) There may be interference with the movement of air in and out of the lungs, so that it takes longer to breathe in and out and the air in the lungs is mixed less efficiently with that drawn in through the bronchi.

(3) There may be changes in lung tissues which interfere with the exchange of gasses between the blood and air.

These changes vary in importance with different diseases but the general effect on the patient is similar. The ability of the lungs to oxygenate the blood and remove carbon dioxide becomes progressively reduced.

Chronic bronchitis, whether or not it is associated with other pulmonary disease such as asthma, leads to the following five changes which interfere with the exchange of gases in the lungs :-

(1) The walls of the bronchi become less rigid.

(2) The mucous membrane lining the bronchi thickens.

(3) The muscular walls of the smaller respiratory passages contract more rapidly (broncho spasm).

(4) There is unusually abundant secretion from the mucous membrane.

(5) The walls of the alveoli break.

The bronchi become unduly susceptible to further infections. These changes combine together to interfere with respiration in the following manner, mainly by interfering with the movements of air in and out of the lungs.

The walls of the bronchi are normally slightly elastic in that they are forced apart during inspiration and contract a little during expiration. Even in the normal person there is less resistance to the movement of air into the alveoli than there is out of them. In severe bronchitis the bronchial walls become so weakened that while they are normal or slightly greater than normal in width during inspiration they collapse sometimes to half this size during expiration. The thickening of the mucous membrane, the spasm of the muscular walls of the small bronchi and the increase in the amount of secretion all narrow the bronchi further and increase the resistance to expiration, more than to inspiration. This is because the resistance to the flow of air along a tube is more dependent on changes in the diameter the smaller the tube becomes.

The breaking of the alveolar walls to form abnormally large air spaces is a phenomenon known as emphysema and it is associated with chronic bronchitis and may be caused by it.

The combined effect of these changes is to slow down the rate at which the patient with chronic bronchitis can breathe in, and even more to slow down the rate at which he can breathe out.

AETIOLOGY.

The following aetiological factors have been studied in the literature on chronic bronchitis and are discussed :-

1. Constitutional Factor.
2. Age and Sex.
3. Environmental Factors -
   (a) Occupation.
   (b) Climate and Season.
   (c) Housing.
4. Habits.
5. Hereditary Predisposition.
6. Family History.
7. Social State.
8. Associated Diseases and Factors.
9. Previous Diseases.
10. Degenerative Changes.

1. Constitutional Factor :-

Scadding (1952) writes that although certain unfavourable environmental factors seem to predispose to the development of chronic bronchitis, the most important aetiological factor in most cases is constitutional.

Gorlitzer von Mundy (1954) whose results with cupping glasses have already been mentioned, also concluded that, apart from an infection, thorough chill or drenching, a definite disposition or constitution is necessary for the development of bronchitis.

2. Age and Sex :-

Oswald (1954) writes that external stimuli seem to be able to condition the bronchi in such a way as to render them more susceptible to infection. He supports his view by the age at onset of symptoms. Under the age of thirty few suffer from chronic bronchitis while the greatest number start to have symptoms from fifty to fifty-nine years according to Oswald's findings. He writes that this would be in keeping with a prolonged effect from atmospheric impurities.

Furniss (1944) has also found that the frequency of chronic bronchitis increases with age.

It should however be remembered that the disease can develop at any age depending to a certain extent also on the habits, occupation, general health and previous general and pulmonary history of the individual.

Browning Alexander (1935) believed that the larger eater and heavier drinker would be more liable to show signs of arterial degeneration and pulmonary trouble at an earlier age than the man who had been strictly moderate in his habits. He also believed that persons whose occupations had involved exposure to all weathers would undoubtedly have a greater tendency to pulmonary trouble at an earlier age than those who had been employed in sedentary ways.

Fry (1954) found that the age of onset in his cases was usually between thirty and sixty.

Men are more frequently affected than women according to Price (1952), and the mortality figures of the Registrar-General for 1946 showed a greater incidence in men as compared with women, of roughly two to one ratio. In the higher age groups, Howell (1951) recorded that the number of female deaths approached that of the males according to these mortality figures and only over the age of eighty-five that women outnumbered men.

Both Howell (1951 and Steel (1944) found a preponderance of male cases in their series. Howell had twenty-five women in a series of ninety cases. Steel studied two groups and the majority of subjects in Group A were ambulatory while Group B were hospital patients. In Group A there were fifty-three per cent. males while in Group B there were sixty-one per cent. males.

Thomas and Taylor (1943), in a series of 100 cases of allergic bronchitis however, recorded that the sex incidence was equally divided between women and men although there was a slight tendency for the majority of those cases occurring earlier in life to be in men. Of the thirty-two patients under thirty years of age only twelve were women and twenty were men, while after the age of thirty the figures were reversed with thirty-eight women and thirty men.

The observation that chronic bronchitis takes a more serious course in men was confirmed by Fulton (1953), who found that in a series of deaths from cor pulmonale in a Manchester hospital over ninety per cent. were men.

Goodman et all (1953) record that this sex difference in bronchitis may be due to hormonal or other constitutional differences so resembling

rheumatoid arthritis and coronary disease in its predilection for a particular sex. The fact, however, that this difference is not observed in the Scandinavian countries may be an argument against such an explanation. It might, however, be explained by the different work habits of men and women. Comparatively few women work for long periods in cold, rain, fog and dust; neither is there the same economic urge for them to return too early to such conditions following acute respiratory infection. Physical effort carried out during or immediately after an acute respiratory infection may also be a factor in the different sex incidence. For the most part men of social classes III. (skilled), IV. (partly skilled), and V.(unskilled) do heavier physical work than women. It is unlikely that this heavy exercise and the heavy breathing it induces may produce excessive stretching in lung tissue still abnormal as the result of recent infection. The fact that those conurbations where it is the custom for married women to go out to work show a lower sex difference than in those where most married women stay at home supports this hypothesis.

Smoking may also be a factor in producing this sex difference. (Goodman et al 1953).

If these surmises are in fact correct, one might look for a narrowing of the gap between male

and female deaths from chronic bronchitis in the next twenty years. One reason is the reduction in physical work required of the modern workman on account of increased mechanisation, while another reason is that the smoking habits of the sexes have undergone a change. (Goodman et al 1953).

3. Environmental Factors :-

(d) Occupation.

A report from the Registrar-General published in the British Medical Journal (1954) divided the community into five so-called social classes namely :-

I. Professional Classes.
II. Intermediate between I. and III.
III. Skilled Occupations.
IV. Partly Skilled Occupations.
V. Unskilled Occupations.

This classification was purely an occupational one, and took no regard of personal circumstances, income, education, and so on except in so far as these were reflected by the individuals occupation. As only one year's deaths were tabulated and as the population estimates were based only on a one per cent. preliminary sample, most of the mortality rates that were given were for the social classes. As in the 1931 census, bronchitis was

among the causes of death that showed a steep gradient of increased mortality from professional to unskilled occupations. The standardised mortality ratio increased from 33 in social class I. to 172 in social class V.

Maurice Davidson (1954) recorded that there were many employed in industry whose work was of such a nature as to constitute a serious handicap to their respiratory organs and there were various trades notorious for their association with chronic bronchial disorders.

As Young (1936) has pointed out,occupations involving exposure to inclement weather, to dust, and to risks of infection are unfavourable, as also are those involving heavy muscular strain or prolonged exertion.

Oswald, Hardd and Martin (1953) also found that the principal factors at work which the patients considered as aggravating bronchitis were changes in temperature and draughts, dampness, cold, dusts, smoke and fumes, and contracting infections from contacts.

Of these factors, dusts, smoke and fumes will be discussed in more detail now.

Oswald et al (1953 recorded that, of the formidable array of dusts,which were incriminated more than half were related to houses, coal, books

and papers. Others included wood shavings, packing materials, upholstery, paint and flour. Almost any dust seems to be a potential irritant. Possible irritants specifically mentioned by Oswald et al were asbestos, cement, cork, wool, lead, lime, marble, printer's ink, talc and chromium and these irritants had caused the patients to seek work elsewhere.

Maurice Davidson (1954) also mentioned trades where dusts might be encountered and he recorded the fur-trade, masons, miners and wool-sorters. Nevile Southwell (1946) recorded occupations involving the inhalation of dusts and fumes of silica, iron, steel, asbestos, carbon and kaolin while less commonly occupations where those employed were exposed to fumes of chlorine, ether, ammonia, nitric acid and the apparently innocuous trades such as hairdressing. In all the above occupations, Southwell recorded that the dusts and gumes caused workers liability to the development of chronic bronchitis.

Thomas et al (1943) writes about a dentist who had paroxysms of coughing whenever he buffed new dentures and this dentist had been persistently troubled with a chronic cough.

Oswald et al (1953) recorded that fumes and smoke were another potent source of bronchial irritation. In their series some 51 males, aged

51 - 69, were exposed to gas in the 1914-1918 world war; 27 of these dated the onset of their bronchitis to gassing, and most of the remainder thought it aggravated their bronchitis, although sometimes the onset was many years after the gassing. Among other irritants were exhaust from motor cars, sulphuric acid, acetone, benzene, caustic soda, and paint spraying. The products of combustion of coal were mentioned most frequently but they are discussed in the section on climate and season.

(b). Climate and Season.

Browning Alexander (1935) recorded that in his opinion, the two essential climatic factors for the chronic bronchitic were sunshine and shelter and Cohen (1944) found in Panama that almost any deviation from a clean warm dry atmosphere seemed to be a possible source of aggravation of symptoms.

On the other hand, exposure of the patient to sudden alterations in the temperature or cold is frequently an aggravating factor and chronic catarrhal bronchitis is more common in damp and foggy climates. (Maurice Davidson 1954).

Oswald et al (1953) record that for those who are employed indoors the temperature and humidity of their place of work may well determine whether they dare risk the journey from home and those who work both indoors and outdoors are particularly

exposed to changes of temperature. Outdoor workers, and many bronchitics prefer outdoor work, are forced to regulate their activity to the prevailing climatic conditions, especially in the winter months.

Howell (1943) questioned pensioners and found that cold, fog and damp were the climatic predisposing factors in that order in a susceptible subject. Oswald et al (1953) singled out fog as the most constantly aggravating climatic factor. Wet, particularly dampness as opposed to rain, was only slightly less frequently mentioned by Oswald et al who thought that it probably accounted for the perpetuation of more bronchitis than fog because it was present so much more often in the London area. They thought the much lower incidence of aggravation by cold was significant. Many patients who were considerably upset by fog and dampness firmly maintained that they were unaffected by dry cold, a feature which might have a considerable bearing on the choice of habitat and occupation.

Goodman et al (1953) recorded that the highest death rate from bronchitis in both sexes and at all ages was experienced by those living in conurbations. There was a progressive decline in the death rate with the size of the towns, and the lowest figures were found in the rural areas.

In November 1954 the Committee on Air Pollution under the chairmanship of Sir Hugh Beaver brought out a report in which they expressed the view that there was a clear association between pollution and the incidence of bronchitis and other respiratory diseases. They found that in general it was the industrial towns liable to heavy pollution that had the highest death rates. In the heavily polluted central Clydeside conurbation in particular, the death rate from bronchitis was commonly much higher than in the rest of Scotland.

Morris Katz (1955) writes that the total amounts of contaminants discharged to the atmosphere over a city or industrial area may not vary substantially from day to day except on week ends, because it reflects the scale of all human activities in terms of industry and population. However, the mass concentration of pollutants per unit volume of air will vary widely because of metereological conditions. In clear weather and conditions of good turbulence the emissions from a multitude of sources are diluted rapidly and quickly dispersed to harmless proportions. During cloudy weather or periods of atmospheric stability, contaminant emissions are dispersed much more slowly and the concentration in unit volume increases.

An annotation on smoke and sulphur (Lancet 1955) records that the Committee on Air Pollution (1954) made clear that there were two main components in smog - namely, dirt and grit, and sulphur products.

The Committee concluded that if this (or indeed any other) Government would declare for a clean-air policy the dirt and grit could be abolished fairly quickly. An annotation on clearing the air (British Medical Journal 1955) discusses the Clean Air Bill which gives effect to the recommendations of the Beaver Report and has four main purposes :-

(1) To prohibit the emission of dark smoke from chimneys, railway engines, and vessels, subject to certain qualifications.

(2) To prohibit the installation of new industrial furnaces unless operated without emitting smoke.

(3) To require that the emission of grit and dust shall be minimized, and that new industrial furnaces burning pulverised fuel or large quantities of other solid fuel shall be provided with grit-arresting equipment.

(4) To empower local authorities by order, subject to confirmation by the Minister concerned, to declare "smoke-control areas", in which the emission of smoke from chimneys constitute an offence.

But the sulphur products present a truly difficult problem.

Sulphur dioxide is produced in the course of many manufacturing processes and Henderson and Haggard (1943) declared that the amount of sulphur dioxide permissible in an atmosphere to which individuals are exposed should not exceed ten parts per million. This, of course, is greatly in excess of any concentration found during the London fog of December 1952 (Ministry of Health Report). However Amdur, Melvin and Drinker (1953) reported evidence that sulphur dioxide breathed even in concentrations of 1 to 8 parts per million will produce shallower and more rapid respiration and an increased pulse rate and these effects were produced over periods of ten minutes in healthy subjects. It might be reasonable to suppose that the effect would be greater in those with chronic respiratory disease. J. Pemberton and C. Goldberg (1954) have recently shown that a significant correlation exists between the average sulphur-dioxide air pollution in the county boroughs of England and Wales and the mortality rates for bronchitis in men aged 45 and over. Results obtained by the Social Medicine Research Unit of the Medical Research Council agreed with these, and showed an association between the consumption of domestic coal per acre and mortality from bronchitis in males (Daly 1954).

The total quantity of sulphur dioxide discharged into our air is over five million tons a year and at present we know of no really satisfactory way of preventing its discharge. The better cleaning of coal at the pithead so as to remove as much sulphur as possible is being pressed for by the National Coal Board. It might be possible to add some material to coal which would cause the sulphur to be retained in the ashes. Fuel oil also releases sulphur into the air and the removal of most of the sulphur from fuel oil is a costly process. Likewise the washing of sulphur dioxide from the gases in large power-stations is a very expensive process although these processes are used in the Battersea and Bankside stations, though not elsewhere.

Sulphur trioxide is also present in atmospheres polluted by the burning of coal and is probably more irritant than sulphur dioxide. In the presence of moisture, aided perhaps by the surface activity of minute solid particles of fog, some sulphur dioxide is oxidised to trioxide. It is probable, therefore, that sulphur trioxide dissolved as sulphuric acid in fog droplets, appreciably reinforced the harmful effects of sulphur dioxide in the London fog of December 1952. (Ministry of Health Report).

Other contaminants of fog in London, or in any city with heavy motor traffic, are carbon monoxide and carbon dioxide. The presence of benzpyrene in city air has been reported by Waller and Doll, and by Cleme at a recent meeting of the British Association.

E. L. Collis (1955) records that Greenwall believes sulphur dioxide and sulphuric acid mist cannot be held entirely responsible for the local smog incidents, but they cannot be absolved from at least partial responsibility and further investigations of atmospheric pollutions are much needed.

In the London fog of December 1952, the irritants mainly responsible were probably those derived from the combustion of coal and its products and their lethal effects were almost wholly exercised in persons already suffering from chronic respiratory or cardiovascular disorders. By contrast with other fogs, the fogs in both 1948 and 1952 were accompanied with temperatures which remained either at or a few degrees above freezing point. Other fogs were accompanied by extremely low temperatures with minimum readings frequently showing ten to fifteen degrees of frost (Ministry of Health Report 1952).

During the period of fog in December 1952, sudden deaths from respiratory disease were more than

four times what they were during the preceding four days and the numbers continued to be high for some time.

In the Meuse Valley fog in Belgium from 1st to 5th December, 1930, when more than sixty people died, the commission concluded that sulphur dioxide and sulphuric acid were present in the fog in sufficient quantity to have caused the disaster. Later Roholm (1937) thought that acute fluorine intoxication was the cause of the disaster. The fluorine compounds were emitted by a phosphate works.

In Donora, Pennsylvania, in the United States from 27th to 31st October 1948, when 18 deaths occurred during a two week period, 17 being on the 4th and 5th days after the onset of the fog, the final conclusion of the investigators was that no single substance was responsible for the episode but that the toxic effects could have been produced by a combination or summation of the action of two or more contaminants. Sulphur dioxide and its oxidation products together with particulate matter were considered to be significant contaminants. The significance of other irritants could not be assessed.

Fry (1954) found that there was a markedly seasonal variation in his cases in general practice. The attendances of his patients with chronic bronchitis were six times more in December than in August.

Steel (1944) recorded that the cough was usually worse in the colder or changeable months (Spring and Autumn), when acute exacerbations with asthmatoid symptoms often occurred.

Otis (1905) wrote that chronic bronchitis markedly diminishes or quite disappears during the warmer months of the year to recur as the colder, damp, changeable weather of late autumn or winter returns.

Finally Price (1952) describes the course and seasonal incidence of chronic catarrhal bronchitis as starting each winter with a more or less acute catarrhal attack; each year however the summer intermission becomes shorter, until the bronchitis persists throughout the year.

(c) Housing :-

Scadding (1952) mentioned poor housing as one of the chief environmental factors predisposing to chronic bronchitis and also exposure to cold and damp at home in his opinion was a predisposing factor to the development of chronic bronchitis. Housing will be discussed in more detail in the section on Social State.

In conclusion Scadding (1952)wrote that the chief environmental factors predisposing to chronic bronchitis were poor housing, exposure to cold and damp at home and at work,and exposure at work to violent changes of temperature and to various dusts.

4. Habits :-

The habits of an individual are of importance as having some significance in prognosis as well as aetiology.

Maurice Davidson (1954) records that people who are accustomed to lives of indulgence and excessive luxury, to overfeeding, lack of sufficient exercise, with habitual confinement to stuffy overheated rooms and general pampering, are more liable to frequent colds and attendant bronchitis than their more hardy brethren who have accustomed themselves to saner modes of life under less enervating conditions.

F.H. Young (1938) also stresses the importance of reviewing the patient's habits and writes that the whole object should be to make him lead a quiet, but not sedentary life, with as few changes in temperature as possible.

According to Austin Furniss (1944), who writes about home conditions, an even temperature of about 60 - 65°F in the sitting room should be aimed at and also a temperature which does not fall below 50°F in the bedroom is about right. Neither the sittingroom should be allowed to become too hot nor the bedroom too "stuffy" although too much cold air from windows should not be allowed.

Browning Alexander (1934) records that the

chronic bronchitic imagines too often that he must be overclad. He should not sweat as this tends rather to increase a catarrhal inflammation than to diminish it. He should, however, be well clad, though attention should be paid to free ventilation, which can only be obtained if the garments are light and not too closely fitting. Maurice Davidson (1954) however emphasises that care should however be taken in small children, very old persons, persons with cardiac lesions and quiescent tuberculous lesions in the chest who are susceptible to external influences such as climate, fatigue and debilitating influences.

Browning Alexander (1934) also records that a person who is overweight and obese should keep to an obesity diet since chronic bronchitis must necessarily throw an increasing strain upon the right side of the heart.

R.A. Young (1936) records that alcoholic over-indulgence is unfavourable by promoting infection, and leading to earlier cardiac breakdown and those who suffer from alcoholic excess should try to limit their alcoholic intake. However in 1934 Christopherson and Broadbent recorded that"we may at once throw overboard gout as a forerunner of chronic bronchitis. Acute gout has almost disappeared whilst chronic bronchitis is as prevalent as ever it was".

Although it is fair to conclude that any inhaled irritant will increase the incidence of bronchitis, Scadding (1952) records that excessive smoking is a factor in some cases, and presumably acts by long-continued chemical irritation of the bronchial mucosa. R.A. Young (1936) also records that over-smoking, especially the inhaling of cigarette smoke, tends to produce a chronic cough and hawking. Smoking produces a tracheo-bronchial irritation, according to Prior (1952), and this author found that those who stopped smoking showed often a dramatic decrease in the cough and sputum production and usually a feeling of improved well-being.

Oswald (1954) is also of the opinion that tobacco-smoke is one of the factors which may render the bronchi susceptible to bronchitis. Heavy smokers nearly always have an excess of mucus in the bronchi according to Oswald, and the volume is usually diminished if the smoking is reduced or abandoned. Oswald et al (1953) found that half the smokers felt that smoking aggravated their bronchitis and that a large number had abandoned the habit with benefit. They found a significant difference between the smoking habits of the chronic bronchitics and those in a series of controls of similar age and sex distribution. There were 9% of non-smokers among the

chronic bronchitics and 20% among the controls. Oswald (1954) concludestherefore that smoking almost certainly does predispose to bronchitis.

Fry (1954) on the other hand found that no real differences were noted between the smoking habits of the chronic bronchitics and those in a series of controls of similar age andsex distribution.

Finally an investigation was undertaken by Palmer (1954) to assess the incidence of bronchitis in a group of smokers compared with a similar group of controls who were non-smokers. The patients' smoking habits were classified as follows: heavy, 20 or more cigarettes a day; moderate, 10-19 cigarettes a day; light, fewer than 10 cigarettes a day. In the pipe smokers 1 ounce of tobacco was taken as roughly equivalent to 30 cigarettes. Patients were classified as non-smokers if they had never smoked, or if they had given up smoking for a year or more before admission.

Palmer found that,in his cases, the incidence of bronchitis in the group of smokers was definitely higher in the smokers than in the non-smokers. The main factors other than smoking which were likely to predispose to a history of bronchitis were satisfactorily distributed between the groups, so that the higher incidence of bronchitis in the

smokers might be reasonably considered to be associated with smoking. The incidence of bronchitis was shown to increase markedly with increased smoking and the amount smoked. However Palmer found that the incidence of a history of bronchitis was as high as 30% in the non-smoking group in the series, and 26% of the heavy smokers gave no history of bronchitis.

Palmer concluded that these results suggested that smoking was of considerable importance in the aggravation of bronchitis, and it followed that in the management of these cases, in addition to the usual measures, they should be encouraged to give up smoking.

5. Hereditary Predisposition :-

Maurice Davidson (1954) writes that this is an aetiological element of which it is difficult to speak with scientific exactitude, but there can be little doubt that there are individuals who are born with an unusual tendency to catarrhal affections of the respiratory tract and who throughout their lives are subject to attacks of bronchitis for which no obvious specific cause can be discovered.

In some instances there is a clear history of similar trouble in parents or other near relations; at other times no such definite information is obtained, but the susceptibility is present,and in the

absence of any demonstrable aetiological features one is tempted to suppose the existence of some peculiarity, either of the tissues, or possibly of the general morphological type of the patient to which this tendency may be attributed.

Maurice Davidson writes that we are especially inclined to think that this may be the case in the chronic bronchitis associated with the barrel-shaped chest so familiar in any large medical out-patient department. In these patients the onset of symptoms at any particular period of life (and this is a most variable feature) cannot be correlated with any particular details in the patients' habits, occupation, or previous medical history, and some such explanation as the above has repeatedly suggested itself.

Oswald et al (1953) found a high incidence of bronchitis among the relations of bronchitic patients, the figure being three times as great as for controls. They recorded that their results suggested a hereditary predisposition and thought that this was probably the correct interpretation, since social and economic factors were eliminated as far as possible by making every effort, short of visiting their homes, to obtain comparable groups of patients and controls. They recorded that the part played by cross-infection within the families,

although virtually indeterminable, is unlikely to be important because of the late onset of bronchitis in most of the patients.

However Stuart-Harris (1954) considers that the high incidence of bronchitis among the relations of bronchitic patients could be explained also on the basis of family infection rather than by genetic factors. The reason given is that the occurrence of bronchitis in childhood is a well-known feature of the past history of some adult patients.

6. Family History :-

Scadding (1952) wrote that a family history of chronic bronchitis was frequently found, sometimes running through several generations and Price (1952) declared that chronic bronchitis seemed to have a special incidence in some families.

Furniss (1944) recorded also that there was frequently a tendency for catarrhal conditions and bronchitis to occur in members of the same family and wrote that some authorities felt sure that a "bronchial diathesis" existed.

F.H. Young (1938) recorded similar observations as Furniss and wrote that whether this tendency was due to "poor material" or that the tendency to catarrh was familial was not certain but he felt convinced that a "bronchial diathesis" existed.

In Howell's series of cases (1951) there was a history of bronchitis in some close relative in 45 per cent. of the patients. In 25 per cent. the father was affected while in 12 per cent. it was the mother who was affected. Other chest troubles had been present in 11 per cent. of the fathers and 8 per cent. of the mothers while 12 per cent. of brothers and sisters had suffered from pulmonary tuberculosis and 8 per cent. from bronchitis. Howell recorded that 40 per cent. of the mothers and 28 per cent. of the fathers of these patients had been completely healthy while only one man in the series had a mother and father who both suffered from bronchitis.

Fry (1954) recorded that, out of 127 cases of chronic bronchitis, a family history of chest disease was significant in some 47 instances (25 men and 22 women).

7. Social State :-

According to Goodman et al (1953), overcrowding and cross-infection, malnutrition and inability to treat early respiratory infections with enough care may all play a part as social factors in the aetiology of chronic bronchitis. In most working-class homes, it is impossible to treat a serious respiratory infection with the respect it deserves,

and the cross-infection and re-infection that ensue among many of these families is familiar to most general practitioners. Goodman et al write that the greatly improved nutrition of the children of social classes IV. and V. and the remarkable reduction in overcrowding reported by the Registrar-General (1952) in all our slum areas, may do something to reduce the excess of this disease among these social classes in the future.

Poor housing as an environmental factor has already been mentioned but it is hoped that with the reduction in overcrowding and building of new houses to replace the "slum" houses that the social state of this country will improve with a corresponding reduction in the incidence of the disease.

Dally (1931) recorded that chronic bronchitis was increasing with the progress of civilisation which tended to predispose to chronic bronchitis owing to the increase of causative factors in the air as well as to a tendency to overcrowd in places of public meeting and the closer, smaller confines of modern dwellings.

8. Associated Diseases and Factors :-

In both chronic bronchitis and bronchiectasis it seems likely that bacterial infection, while an important cause of symptoms, is essentially incidental. In chronic bronchitis the respiratory mucosa

reacts abnormally, especially in the direction of excessive secretion, to a variety of stimuli; and this abnormality predisposes to bacterial infection, which, once established, modifies symptoms and may lead to secondary morbid anatomical changes (Leading Article British Medical Journal 1953).

Marshall (1927) declared that as long as you have free movement of the chest and the lungs are freely mobile it is rare to get chronic bronchitis but if, for some reason they are not mobile, then bronchitis is almost certain to follow from infection of the stagnant secretions sooner or later. Marshall (1931) declared that there must be a definite cause for the breaking down of these elaborate defences in a case of chronic bronchitis and a careful search for this cause should be our first step when a patient comes to us for advice.

Two main features as causal factors which Marshall discussed were structural defects and infection from above.

In the majority of cases, according to Marshall, the defences fail because of some structural defect which interferes with the upward flow of the bronchial secretion. The stagnation in the majority of cases is the result of a loss of mobility in the lungs, as in the generalised emphysema of elderly subjects. In other cases there is a local

loss of mobility and this is seen when lung-tissue fails to re-expand after compression by a pleural effusion or an empyema. Bronchitis commonly occurs in lungs which have undergone fibroid change as a result of broncho-pneumonia, unresolved lobar pneumonia and chronic forms of tuberculosis. A previous attack of bronchitis may leave the mucous membrane imperfectly healed and repeated attacks of acute bronchitis will lead eventually to the development of chronic bronchitis. A severe acute respiratory illness such as lobar pneumonia or bronchopneumonia may also lead to the development of chronic bronchitis.

There is another group of cases in which the loss of movement is not due to changes in the lungs, but to fixation of the chest-wall, such as may result from severe injuries by gunshot wounds or civil accidents. Bronchitis is commonly associated with deformities of the spine; and with lateral curvature we often find signs of bronchitis in the lung lying on the concave side of the deformity, where the ribs are crowded together and immobile.

Chronic bronchitis is not always secondary to a structural defect, for even the best defences may be overcome by a prolonged siege, or in other words by a persistent stream of bacteria coming down from septic foci in the mouth, throat, nose or

accessory sinuses. According to Marshall, exogenous infection plays an important part in the causation of chronic bronchitis. Roper (1942) recorded that the acute infection of the bronchial portion of the respiratory tract might be a filter-passing virus which lowered the resistance of the mucous membrane. The patient might recover from the acute attack with the ciliated epithelium damaged although in fact partially replaced by squamous epithelium. The function was then potentially interfered with so that there was a handicap in dealing with any further attack when immunity and general resistance again became lowered, as in winter. Roper's views were shared by Steel (1944) who thought that the virus possibly activated the organisms already present and must be a potent factor in droplet infection.

Organisms such as pneumococci, streptococci, Micrococus Catarrhalis and Bacillus Friedlander are found in the sputum as a result of infection from above but the two organisms, Haemophilus Influenzae and the pneumococcus, seem to be far more important than any others in chronic bronchitis, as they are the most important pathogens according to May (1952, 1953 and 1954). It should be noted that the bacteriological findings in chronic bronchitis are not dealt with in detail in this thesis.

Chronic upper respiratory infections may thus be causative and predisposing factors in the development of the disease. Furniss (1944) recorded that in children with bronchitis quite a third of the cases showed definite evidence that the nasal sinuses were infected although infection of the tonsils did not seem to play such an important part. Nasal sinus infection seemed to be most common in the cases of bronchitis in which dilatation of the bronchi were present and in those in which acute exacerbations tended to occur.

Chronic bronchitis accompanies chronic pulmonary conditions such as asthma and emphysema. (Hatch 1930).

Roper (1942) records that asthma and bronchitis are themselves frequently associated and that both an asthma, emphysema, chronic bronchitis sequence and chronic bronchitis, emphysema, asthma sequence can frequently be observed.

Christopherson et al (1934) records that the asthmatic paroxysm occurs typically at night when the parasympathetic or vagal-system holds the reins and sympathetic stimuli are in abeyance ; whereas chronic bronchitis is primarily a daytime disease, when sympathetic influences are strongest. Furthermore chronic bronchitis is not a pyrexial disease and is often influenced quickly by such nervous factors as the stimulus of a cold draught of

air on the face or by exertion or even by the sound of hostile aircraft during war. The latter may, in another type of individual, bring on an asthmatic paroxysm and these facts suggest that much depends on the primitive, early acquired nervous poise of the patient as to the direction his disordered respiratory reaction will take.

Finke (1948) records that in children and young adults both chronic bronchitis and bronchiectasis can usually be traced to an attack of bronchopneumonia following measles, to pertussis or to laryngotracheo-bronchitis. In people over the age of forty, this type of pulmonary disease, in a large percentage of cases, originated from influenza during the pandemic of World War I. In this War, the war gases should also be considered as they also were casual agents of chronic bronchitis subsequently developing in many former servicemen. Finke believes that a considerable increase in chronic pyogenic pulmonary diseases may be expected in coming years as occurred after World War I.

Khalik (1937) records that scrofulous and arthritic children are much predisposed to chronic bronchitis and enlarged mediastinal glands, tuberculous or otherwise, pressing on the nerves and bronchi, troubling their vaso-motor innervation, and thus favouring infection are also a fairly common

cause. Other local affections of the nose, as deviation of the septum and chronic rhinitis, should be considered as causal agents in babies and children. In adults also sinus infection, nasal obstruction, polypi and dental sepsis should be studied. Corysa often initiates bronchitis and repeated attacks of coryza in a susceptible patient may initiate chronic bronchitis. Mouth-breathing predisposes also to the development of chronic bronchitis according to Price (1952).

Chronic cardiac diseases and cardiovascular lesions, such as valvular defects and arterial disease, and chronic renal diseases, such as chronic nephritis are causative and predisposing factors for the development of chronic bronchitis (Price 1952).

Lastly Maurice Davidson (1954) writes that it must be remembered that tuberculosis is one of the commonest underlying causes of what may at first seem to be a primary bronchitis whether of acute or chronic type. Moreover, the possibility of malignant disease of the lung and bronchus must not be forgotten, especially in unexplained bronchitis in persons of middle age.

9. Previous Diseases :-

Many of the previous diseases which may be possible causative and predisposing factors in the development of chronic bronchitis have already been mentioned in previous pages.

But it is important to consider these as a distinct clinical factor which requires to be carefully considered in all patients suffering from chronic bronchitis.

Howell (1951) recorded the results of his observations into previous respiratory illnesses in 53 patients with the disease.

Some 32 per cent. claimed that they had been previously healthy and 42 per cent. had never suffered from any disease affecting the respiratory tract prior to the onset of chronic bronchitis. A previous attack of bronchitis had occurred in 14 per cent. and the same number remembered having had pneumonia. Influenza was mentioned in 8 per cent., while 12 per cent. had suffered from other respiratory infections of some kind, usually pulmonary tuberculosis. As regards childhood, 77 per cent. had no history of chest trouble, but 20 per cent. remembered having bronchitis and 3 per cent. some other respiratory tract illness. The results of enquiry about coryza in childhood were unsatisfactory.

10. Degenerative Changes:-

Howell (1951) writes that in past descriptions of bronchitis and in previous discussions of its aetiology, sufficient notice has not been taken of the various degenerative alterations which advancing years bring to the respiratory tract. These

have been enumerated by Macklin and Macklin in some detail (1942) but here a brief summary is given and this contains many factors which would permit the establishment and encourage the continuation of a chronic cough.

A. Nose: 1. Aetiology of the mucosa.

B. Lungs: 1. Decline in efficiency of bronchial eliminative mechanism, including cilia and smooth muscle.

2. Sclerotic changes in bronchi, connective tissue and blood vessels, diminishing normal movement.

3. Degenerative changes in the pulmonary vascular bed.

4. Decline in efficiency of nervous mechanism in the lungs.

5. Diminished effectiveness of lymphatic drainage.

6. Reduction of Vital capacity.

7. Impairment of activity in the reticulo-endothelial system.

C. Thorax: 1. Atrophy of mucosa and lymphoid tissue in pharynx and trachea.

2. Rigidity of chest wall, calcification of cartilages.

3. Weakening of respiratory muscles, descent of the diaphragm.

4. General deterioration of circulatory efficiency.

D. General: 1. Diminished immunity values.

2. Impaired regulation of temperature.

3. Decline in efficiency of other parts of the body.

Consideration of this list will show how accumulation of the sputum, epithelial changes, altered irritability of the mucosa and diminished effectiveness of cough may be brought about by the mechanisms enumerated. When we add weakening of thoracic muscles, impaired defence organization and lowering of resistance to certain bacterial flora, some of the phenomena customary in chronic bronchitis are easier to comprehend. The benefits from heat and the maleficent effects of cold fit into line once we regard chronic bronchitis as part of a degenerative process.

PART II.

PRESENT STUDY.

OBJECT OF STUDY :

In general practice chronic bronchitis is one of the commonest diseases encountered either when visiting patients' homes or in the course of a surgery at the doctors' own consulting rooms.

It is the medical practitioner who is fortunate to have the opportunity to investigate all the aetiological factors which may be responsible for the onset of chronic bronchitis in a patient.

The object of my investigation was to ascertain these possible aetiological factors in a series of seventy-five patients suffering from chronic bronchitis and compare my results with those already noted by other observers and recorded in the literature.

It was Keith Westlake (1954), whom I referred to in my introduction, who wrote that it was unlikely that there would be any substantial fall in the overall mortality from "bronchitis" until the incidence of chronic bronchitis and emphysema was itself reduced. He said that this would depend on the discovery of all the aetiological factors involved.

Edinburgh is an ideal place for the study of the disease, since it is subject to extreme variations in climatic conditions. The photographs, inserted later in the thesis, give some impression of variation in atmospheric conditions which are experienced particularly in the autumn, winter and spring seasons of the year.

In addition to these varying climatic conditions, the district in which my general practice is carried out contains many different varieties of houses. These houses, are located in different social districts, and the patients, who live in my practice area, belong to all branches of society and are employed in diverse occupations pertaining to all the five social classes. This diversity was a great asset in recording observations as a wide field of society could be studied in the preparation of this thesis.

As a general practitioner it was possible for me to study the said state of my patients. This was accomplished by the opportunity afforded by regular visits to the patients' own homes.

As the family doctor it was also possible to study carefully the family history and enquire into the previous medical history of those patients suffering from chronic bronchitis.

Barach (1953) wrote that the diagnosis of chronic bronchitis could be established only when the wide variety of respiratory and cardiac conditions which produce cough and expectoration were excluded. Under such circumstances a patient in good general health whose complaint was limited to chronic cough might be considered to be suffering from chronic bronchitis.

None of the cases studied for this thesis had a history of cough at intermittant intervals of less than one year in duration. A Table showing the length of history of symptoms of chronic bronchitis in each case is inserted later in this thesis.

AETIOLOGICAL FACTORS INVESTIGATED.

The following aetiology factors were investigated in each of the seventy-five patients suffering from chronic bronchitis studied in my general practice :-

1. Age.
2. Occupation and Habits.
3. Climate and Season.
4. Hereditary Predisposition.
5. Factors in Onset.
6. Sex.
7. Family History.
8. Social State.

1. Age :-

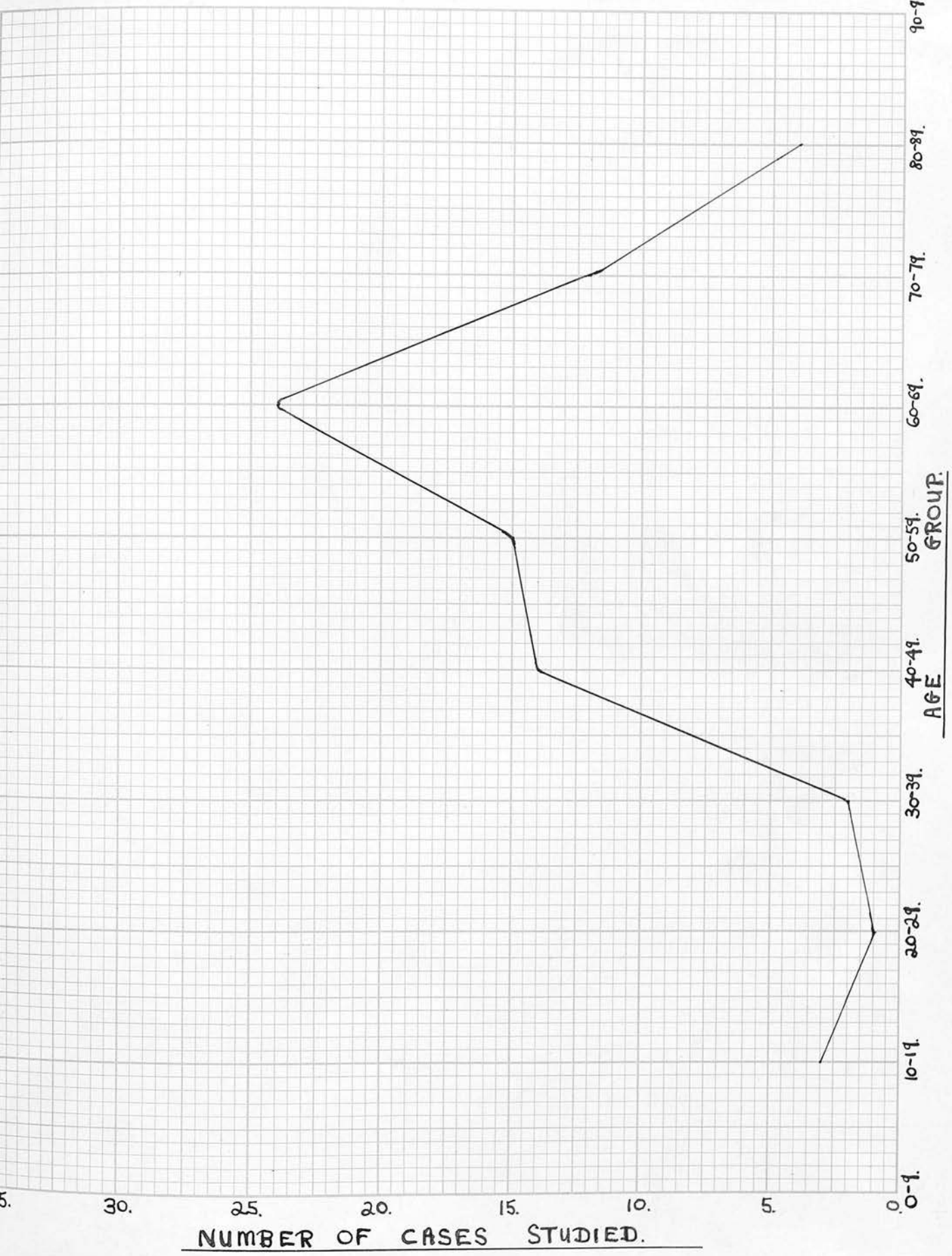
All seventy-five patients studied are grouped into the different decades of life and the age groups ranged from the second to the eighth decade of life.

The youngest boy was twelve years old while the oldest lady was eight-nine years of age.

The following Table records the number of cases studied and the percentage of cases investigated in each decade.

Age Group.	Number of Cases Studied.	Percentage.
10 - 19.	3.	4.
20 - 29	1.	1·3.
30 - 39	2.	2·7.
40 - 49.	14.	18·7.
50 - 59	15.	20.
60 - 69	24.	32.
70 - 79	12.	16.
80 - 89	4.	5·3.

The following graph illustrates the results recorded in the above Table as number of cases studied in relation to different age groups.


90-99.
80-89.
70-79.
60-69.
50-59.
40-49.
30-39.
20-29.
10-19.
0-9.
AGE GROUP.
35.
30.
25.
20.
15.
10.
5.
0.
NUMBER OF CASES STUDIED.

One of the patients died in 1953 while three patients died in 1954 and the ages recorded were those at the time of their death. Likewise the ages at the time of death were recorded for the four patients who died in 1955.

Despite these facts, it is shown from the above Table and graph that the most prevalent age group encountered in patients suffering from chronic bronchitis in my general practice in 1955 is the sixth decade. This might be considered as the results of a general practitioner in any calender year.

The last Table illustrates the individual ages of the cases :-

No. of Case	1.	2.	3.	4.	5.	6.	7.	8.
Age	80.	67.	69. Died 1955	66. Died 1955	57.	50.	76.	74.
No. of Case	9.	10.	11.	12.	13.	14.	15.	16.
Age	61.	67.	48.	50.	65.	70.	67. Died 1954.	74.
No. of Case	17.	18.	19.	20.	21.	22.	23.	24.
Age	71.	58.	48.	49.	65. Died 1954	61.	14.	56.
No. of Case	25.	26.	27.	28.	29.	30.	31.	32.
Age	46.	64.	43.	45.	43.	66.	65.	64.
No. of Case	33.	34.	35.	36.	37.	38.	39.	40.
Age	68.	45.	54. Died 1955	68. Died 1953	63.	33.	56.	70.
No. of Case	41.	42.	43.	44.	45.	46.	47.	48
Age	59.	78.	67.	59.	58.	53.	70.	49.
No. of Case	49.	50.	51.	52.	53.	54.	55.	56.
Age	55.	13.	73.	60.	64.	61.	59.	68.
No. of Case	57.	58.	59.	60.	61.	62.	63.	64.
Age	86. Died 1954	75. Died 1955.	81.	89.	67.	43.	67.	53.
No. of Case	65.	66.	67.	68.	69.	70.	71.	72.
Age	72.	72.	40.	58.	49.	61.	44.	33.
No. of Case	73.	74.	75.					
Age	46.	12.	27.					

2. Occupation and Habits:-

Each patient was questioned as to his or her present occupation or previous employment.

An endeavour has been made to divide each occupation or previous employment into one of two sections.

In the first Section I have included those who have been exposed in their present occupation or previous employment to inclement weather, dust, risks of infection, heavy muscular strain or prolonged exertion. In the second section those who have not been exposed to the above hazards and conditions at their present occupation or previous employment are included. Although housewives and clerkesses are exposed to a certain amount of dust at their work, I decided to include them in the second section as the quantity of dust to which they are exposed, in my opinion, does not merit their place in the first section.

The community has been divided into the five so-called social classes:-

I. Professional Classes.
II. Intermediate between I. and III.
III. Skilled Occupation.
IV. Partly Skilled Occupation.
V. Unskilled Occupation.

This classification is purely an occupational one, and takes no regard of personal circumstances, income, education, and so on except in so far as these are reflected by the occupation of the individual. I have not included school children in this classification.

The following Table records the results of my observations :-

Case No.	O C C U P A T I O N	Section	Social Class
1.	Retired Housewife and formerly Stationmaster's Wife	2	IV.
2.	Retired Able Seaman who was formerly on a Fishery Cruiser	1	IV.
3.	Formerly Shirt Machinist prior to retiral and up to death in 1955 Housewife.	2	III.
4.	Retired Baker. While on Active Service in 1914 - 1918 World War he suffered from effects of Mustard Gas. Died 1955	1	V.
5.	Housewife	2	IV.
6.	Skinner in Tannery	1	V.

Case No.	O C C U P A T I O N.	Section	Social Class
7	Housewife	2	IV.
8	Retired Hairdresser	1	III.
9	Housewife	2	IV.
10.	Former Rivetter in Shipyard, but since 1950 Storeman in Shipbuilding yard.	1	V.
11.	Painter	1	IV.
12.	Housewife	2	IV.
13.	Retired in 1955 and died same year. Formerly Foreman in Shipbuilding yard.	1	V.
14.	Housewife	2	IV.
15.	Died 1954. Labourer and Storeman with British Road Services. Prior to Mechanisation of latter he was a Carter.	1	V.
16.	Housewife	2	IV.
17.	Retired Draper	2	II.
18.	Bondworker since 1950 but prior to this year Paper Sorter	1	V.

Case No.	O C C U P A T I O N	Section	Social Class
19.	Dock Labourer	1	V.
20.	Housewife	2	IV.
21.	Died 1954. Tanner.	2	V.
22.	Fitter	2	IV.
23.	Schoolboy	2	-
24.	Coal Man.	1	V.
25.	Salesman up to 1941 and since then Bus Conductor.	1	V.
26.	Joiner and Builder	1	III.
27.	National Assistance Claims Investigation Officer.	1	II.
28.	Barman	2	V.
29.	Blacksmith up to 1935 and from 1943 to present date. Goods Porter from 1935 - 1943	1	IV.
30.	Retired Bodymaker (3.4.54.)	2	III.
31.	Housewife	2	IV.

Case No.	OCCUPATION	Section	Social Class
32.	Retired Clerkess (Distillery Office) 1.1.54 .	2	V.
33.	Housewife	2	IV.
34.	Charge-hand Transformer Department in Electrical Works	1	III.
35.	Died 1955. Housewife	2	IV.
36.	Died in 1953. Porter in Leith Docks for 12 years then Bondworker first as Maltsman and later worked with husks of barley. In 1914-1918 War he suffered from effects of Mustard Gas and Lewisite	1	V.
37.	Maintenance Engine Man in Gas Works	1	IV.
38.	Bus Conductor	1	V.
39.	Headmaster	2	I.
40.	Sileman in Wheat Mill 1926 - 1938. Foreman in Wheat Mill 1938 to present date.	1	V.
41.	Retired Able Seaman Merchant Navy	1	IV.
42.	Housewife	2	IV.

Case No.	O C C U P A T I O N.	Section.	Social Class.
43.	Lorry Driver previously but now Blacksmith	1	IV.
44.	Navvy work up to December 1953 but since that date he drives an electric hammer in a blacksmith's shop	1	V.
45.	Bakers' Ovensman. At Arras 1917, subject to Chlorine Gas.	1	V.
46.	Housewife	2	IV.
47.	Retired Clerk Manager (1950)	2	II.
48.	Electrician	2	III.
49.	Driller	1	V.
50.	Schoolboy	2	--
51.	Retired Night Watchman and prior to this occupation Bricklayers' Labourer	2	V.
52.	Retired Packer in Flour Mill (August 1955)	1	V.
53.	At first Dock Labourer but now Porter in Leith Docks. In 1914-1918 War he suffered from effects of Mustard Gas.	1	V.
54.	In Flour Mills as Miller 1921 - 1933 but since 1933 carried out first aid and Civil Defence work in Flourmills	2	V.

Case No.	O C C U P A T I O N.	Section.	Social Class.
55.	Safety first worker but previously Labourer	2	IV.
56.	Retired Shunter on railway	1	IV.
57.	Died 1954. Housewife	2	IV.
58.	Died 1955. Retired Fireman - Greaser in Merchant Navy	2	IV.
59.	Housewife	2	IV.
60.	Housewife	2	IV.
61.	Housewife	2	IV.
62	Housewife	2	IV.
63.	Housewife	2	IV.
64.	Housewife	2	IV.
65.	Retired railway goods' worker	1	IV.
66.	Master mechanical engineer and electric welder	1	II.
67.	School Inspector	1	I.

Case No.	O C C U P A T I O N.	Section.	Social Class.
68	Fisherman 1919 - 1935 Brewers' Worker 1935 to present date	2	V.
69	Shopworker 1921 - 1922. Housework 1932 - 1941. Packer in Ryvita Factory 1941 - 1950. Housework 1950 - to present date.	2	V.
70.	Cleaner	2	V.
71.	Labourer - Animal Feeding Stuffs	1	V.
72.	Tracer	2	II.
73.	Canteen Worker	2	V.
74.	Schoolboy	2	—
75.	Foundry Worker	1	V.

From the above Table it was found that forty-four per cent. of the patients had been exposed in their present occupation or previous employment to inclement weather, dust, risks of infection, heavy muscular strain or prolonged exertion and belonged to Section one.

	Number of Cases.	Percentage.
Section 1.	33	44
Section 2.	42	56

From the above Table the incidence of chronic bronchitis in my patients was highest in those employed in partly skilled and unskilled occupations.

The following Table showing these results does not include three schoolboys who were not classified.

Social Class Number.	No. of Cases (only 72 included)	Percentage of 72 Cases.
I.	2.	2.8
II.	5.	6.9
III.	6.	8.3
IV.	31.	43.1
V.	28.	38.9

Each patient was questioned about certain occupational hazards at work particularly with regard to dust and also about the following habits and respiratory symptoms and their effect on breathlessness and cough. Any climatic factors at work will however be considered in the next section.

As seven patients in this series did not suffer from any breathlessness, the results of only sixty-eight are recorded in factors affecting breathlessness in this section and in the next section.

Tables recording in detail the results in individual cases are inserted later in this thesis.

The following factors were investigated:-

A. Factors affecting dyspnoea:-

1. Smoking.
2. Excitement.
3. Heavy Meals.
4. Coughing.
5. Exertion.

B. Factors causing exacerbations of cough :-

1. Exertion.
2. Dust.

A. Factors affecting Dyspnoea:-

1. Smoking.

Of the seventy-five cases with chronic bronchitis, thirty-nine were non-smokers.

A series of control cases were investigated among patients reporting sick in the course of general practice and as far as possible cases were chosen who were nearly the same age as the patient with chronic bronchitis. In each case the control case and chronic bronchitic patient were of the same sex and the number of control cases investigated were also seventy-five.

Of these seventy-five control cases, forty were found to be non-smokers.

In my series there were more smokers among those suffering from chronic bronchitis than among those patients in the control group.

The following Table records the above findings :-

	Non-Smokers	Percentage
Present Series 75 Cases with Chronic Bronchitis	39	52·0
Control Series 75 Cases	40	53·3

In the series of chronic bronchitic patients it is interesting to note that one patient found that smoking a cigarette temporarily eased her breathlessness. She smoked about seven cigarettes daily usually.

Another patient found that his breathlessness was increased only if he smoked more than ten cigarettes daily.

Six of the patients with chronic bronchitis complained of an increase in breathlessness after smoking either cigarettes and/or tobacco and one of those patients smoked forty cigarettes a day and he was the only heavy smoker of this number.

Tables recording detailed results are included later in this thesis, but the following Table records the results :-

		Number	Percentage
Effect on Dyspnoea	No Dyspnoea	7	9·3
	Increase	6	8
	No effect	61	81·3
	Decrease	1	1·3

2. Excitement:-

A larger number of patients found that when they were subjected to excitement of one kind or another that their breathlessness increased in severity.

		Number	Percentage
Effect on Dyspnoea	No Dyspnoea	7	9.3
	Increase	25	33.3
	No Effect	43	57.3

As an example of the type of excitement which caused an increase in breathlessness, one lady found that a noise at her back caused an increase.

3. Heavy Meals:-

Patients were questioned as to whether they experienced any breathlessness after partaking a heavy meal. Many replied that they never or very rarely ate a heavy meal but of those who had eaten a heavy meal or were in the habit of doing so there were twenty who felt breathless after it.

		Number	Percentage
Effect on Dyspnoea	No Dyspnoea	7	9.3
	Increase	20	26.7
	No Effect	48	64

4. Coughing :-

Almost half of the patients with the symptom of dyspnoea found that coughing brought about an increase in their breathlessness.

		Number	Percentage
Effect on Dyspnoea	No Dyspnoea	7	9.3
	Increase	33	44
	No Effect	35	46.7

One patient found that after coughing his breathlessness was not increased but he experienced pain in the chest as a result of coughing. Another patient experienced both increase in breathlessness and pain in the chest as a result of coughing. Both these patients had been subject to mustard gas and one had also been subject to lewisite in the 1914-1918 World War.

5. Exertion :-

Over seventy-five per cent. of my chronic bronchitic patients found that exertion caused an increase in their breathlessness. One man of thirty-three years of age found that prolonged exertion only increased his breathlessness while another lady of sixty-seven years of age found that severe exertion only increased her breathlessness.

		Number	Percentage
Effect on Dyspnoea	No Dyspnoea	7	9.3
	Increase	58	77.3
	No Effect	10	13.3

A Graph illustrating the above findings is to be found in the section on climate and season.

B. Factors causing exacerbations of cough :-

1. Exertion:-

Half of my patients with chronic bronchitis found that exertion caused an aggravation of their cough. One man of sixty-eight years of age remarked that even the exertion of rising in the morning from the horizontal to the vertical position caused an increase in his cough.

		Number	Percentage
Effect on Cough	Increase	38	50.7
	No Effect	37	49.3

2. Dust :-

When dust is considered in relation to an individual's cough, the occupation of the person must be studied also. Dust may be a most important

factor in relation to either the onset of chronic bronchitis or the progress of the disease once it has been established.

Almost fifty per cent. of those suffering from chronic bronchitis found that dust of any kind caused an aggravation of their cough.

		Number	Percentage
Effect on Cough	Increase	35	46.7
	No effect	40	53.3

A Graph illustrating the above findings is to be found in the section on climate and season.

The following Table illustrates the occupation of those patients who found that dust caused an aggravation of their cough. It illustrates whether dust was present at the patient's work and, if present, whether it caused any effect on the patient's cough or not :-

Case No.	Sex	Occupation.	If dust present at occupation Yes or No. If present, effect on cough.	
2.	M.	Retired Able Seaman	No.	Not present at former work.
3.	F.	Former Shirt Machinist. Housewife prior to death in 1955.	No.	Not at previous work as shirt machinist, slight dust as housewife but not sufficient to be of importance.
4.	M.	Retired Baker. Suffered from effects of Mustard Gas in 1914-1918 War. Died 1955.	Yes.	Present at previous employment as worked in Bakery but changed to Confectionary Department because of dust at work aggravated cough. On Active Service 1914-1918. Subjected to Mustard Gas.
6.	M.	Skinner in Tannery	Yes.	When packing bales of wool in Tannery, a fine dry dust from wool aggravated cough.
10.	M.	Former Rivetter. Now Storeman in Shipbuilding Yard.	No.	Not as Storeman but up to 1950 as rivetter dust was present at work.
11.	M.	Painter.	Yes.	Dust particularly prevalent when cleaning down wall paper and where much house dust present. Dust aggravates cough.
13.	M.	Retired Foreman in Shipbuilding Yard. Died 1955.	No.	Dust not present at previous employment.
15.	M.	Labourer and Storeman British Road Services, carter up to mechanisation. Died 1954.	No.	Dust not present at previous employments.

Case No.	Sex	Occupation.	If dust present at occupation Yes or No. If present, effect on cough.	
16.	F.	Housewife	No.	Not sufficient in quantity to merit any importance.
17.	M.	Retired Draper	No.	Dust not present at previous employment.
23.	M.	Schoolboy	Yes.	Sawdust at carpentry at school aggravated cough.
26.	M.	Joiner and Builder	Yes.	As joiner dressing machine, which dresses the raw timber, aggravates both cough and dyspnoea and he keeps away from dressing machine when it is on. Sawdust does not aggravate cough or dyspnoea.
33.	F.	Housewife	No.	Not sufficient in quantity to merit any importance.
34.	F.	Charge-hand Transformer Department Electrical Works	No.	
36.	M.	Porter in Leith Docks for 12 years then later Bondworker first as Maltsman and later worked with husks of barley. In 1914-1918 War he suffered from effects of Mustard Gas and Lewisite. Died 1953.	Yes.	While working as Bondworker dust at work aggravated his cough. Also suffered from effect of Mustard Gas and Lewisite in 1914-1918 World War.
37.	M.	Maintenance Engine Man in Gas Works.	No.	
38.	M.	Bus Conductor.	No.	
39.	M.	Headmaster.	No.	

Case No.	Sex	Occupation	If dust present at occupation, Yes or No. If present, effect on cough.	
40.	M.	Sileman in Wheat Mill 1926 - 1938. Foreman in Wheat Mill 1938 to present date.	Yes.	As sileman, dust at work aggravated his cough.
41.	M.	Retired Able Seaman Merchant Navy.	No.	Not present at former work.
42.	F.	Housewife.	No.	Not sufficient in quantity to merit any importance.
43.	M.	Blacksmith. Formerly lorry driver.	Yes.	Dust at work causes aggravation of cough.
45.	M.	Bakers' Ovensman. At Arras in 1917 subject to Chlorine Gas.	Yes.	Flour dust at Bakery causes sneezing and wheeziness of chest but does not aggravate cough.
46.	F.	Housewife.	No.	Not sufficient in quantity to merit any importance.
49.	M.	Driller.	Yes.	Cast iron dust from drill aggravates cough.
52.	F.	Retired Packer in Flourmill.	Yes.	Dust from flour at work aggravated cough.
53.	M.	At first Dock Labourer but now Porter in Docks. Suffered from effects of Mustard Gas in 1914-1918 War.	Yes.	Grain, seed and cardboard box dust at work increases dyspnoea and aggravates cough.
54.	M.	Miller in Flourmills 1921-1933 but since 1933 first aid and civil defence work in Flourmills.	Yes.	As miller dust from flour aggravated dyspnoea but not cough from 1921-1933.
55.	M.	Safety-first worker but previously labourer.	No.	

Case No.	Sex	Occupation	If dust present at occupation, Yes or No. If present, effect on cough.
56.	M.	Retired shunter on Railway.	Yes. Dust of sulphur fumes from railway engines at previous employment (and also dust in street) aggravated cough.
62.	F.	Housewife.	No. Not sufficient in quantity to merit any importance.
65.	M.	Retired railway goods worker.	Yes. Dust of sulphur fumes from railway engines at previous employment aggravated cough.
69.	F.	Shopworker 1921-1932 Housework 1932-1941. Packer in Ryvita Factory 1941-1950. Housework 1950 to present date.	Yes. As Packer in Ryvita Factory dust from flour aggravated cough.
71.	M.	Labourer - Animal Feeding Stuffs.	Yes. Dust from animal feeding stuffs at work aggravates cough.
75.	M.	Foundry Worker.	Yes. Dust from Moulder's sand in Foundry aggravates cough.

Of the thirty-five patients who found that dust of any kind aggravated their cough, eighteen told me that dust was present either at their present place of work or at their previous employment. Sixteen of the latter found that dust aggravated their cough. Two men were subjected to Mustard Gas while on Active

Service in 1914-1918 war and another man suffered from effects of both lewisite and mustard gas in this war. Another man was subject to chlorine gas in this war and in all four cases the result of their subjections to different varieties of gas in active service was probably a contributory factor in the aetiology of the disease.

Total Number		Number of Cases.	Percentage.
35.	Dust at present or past employment.	18	51.4
	If dust present at work or past employment, dust aggravated cough.	16	45.7

3. Climate and Season :-

According to studies made in the literature chronic bronchitis is one of the diseases in the development of which climate may be an environmental factor.

The following factors were investigated :-

A. Factors affecting dyspnoea :-

1. Draughts.
2. Fog.
3. Cold Weather.
4. Damp.

B. Factors causing exacerbations of cough:-

1. Cold Air.
2. Fog.
3. Smoke.
4. Wet.

C. Factors diminishing cough :-

1. Dry Weather.
2. Warmth.

A. Factors affecting dyspnoea:-

1. Draughts:

A comparatively small number of chronic bronchitis patients found that when they were exposed to draughts that their dyspnoea was increased. Only thirteen patients in my series found that exposure to draughts increased their dyspnoea.

		Number	Percentage
Effect on Dyspnoea	No Dyspnoea	7	9.3
	Increase	13	17.3
	No Effect	55	73.3

2. Fog :-

The district in Edinburgh in which my general practice is located is situated near the vicinity of the estuary of the river Forth. The area is prone to fog and sea mist all the year round but particularly in the autumn, winter and spring seasons of the year.

The results of the effect of fog on both dyspnoea and cough in my series are particularly interesting in view of the prevalence of this atmospheric condition in the area.

It was found that more than half of the patients interrogated stated that their breathlessness was increased by the presence of fog in the atmosphere around them.

		Number	Percentage
Effect on Dyspnoea	No Dyspnoea	7	9.3
	Increase	43	57.3
	No Effect	25	33.3

3. Cold Weather :-

Again the district in Edinburgh in which my general practice is located is particularly prone to cold east winds particularly in the winter and autumn seasons of the year.

Quite a number of my patients found that when they were subjected to cold weather that their

breathlessness was increased.

		Number	Percentage
Effect on Dyspnoea	No Dyspnoea	7	9.3
	Increase	24	32
	No Effect	44	58.7

One patient found that cold weather did not increase his breathlessness but made him feel "choked". Two patients were particularly affected in frosty weather and one patient told me that when the weather changed from warm to cold, even to a slight extent, that his breathlessness increased.

4. Damp :-

Finally in the autumn and spring seasons of the year, dampness is particularly prevalent in the area in which my general practice is situated. This is associated with fog and cold weather in the area next to the Firth of Forth. The dampness appears as rain and east wind haar which penetrates the atmosphere.

Almost fifty per cent. of patients found that damp increased their breathlessness and this was a larger proportion than those affected by cold weather.

		Number	Percentage
Effect on Dyspnoea	No Dyspnoea	7	9.3
	Increase	34	45.3
	No Effect	34	45.3

A Graph illustrating the above findings is found later in this section.

B. Factors causing exacerbations of cough :-

1. Cold Air :

Each patient was questioned as to the time of day that his or her cough occurred. The majority of my patients found that their cough occurred mainly in the morning, mainly at night, or mainly both night and morning.

The atmosphere changes temperature and is colder in the morning and at night, and this cold air appears to aggravate the patients' cough as can be seen by my findings.

One man however found that his cough was diminished in cold air while three patients found that their cough was only aggravated when the cold air was frosty.

Chronic bronchitis usually starts in the winter months when the air is cold, and only after some years does the cough affect the individual in the warmer summer months. The seasonal incidence in my cases is depicted at the end of this thesis in a Table.

		Number	Percentage
Frequency of Cough.	Constantly	5	6·7
	Rarely	0	0
	Mainly Night	12	16
	Mainly Morning	23	30·7
	Variable	16	21·3
	Mainly Night and Morning	16	21·3
	Mainly Morning but Variably	1	1·3
	Mainly Afternoon	2	2·7

		Number	Percentage
Effect on Cough.	Increase	35	46·7
	No Effect	39	52
	Decrease	1	1·3

2. Fog :-

Over fifty per cent. of the patients found that fog aggravated their cough.

		Number	Percentage
Effect on Cough	Increase	43	57·3
	No Effect	32	42·7

3. Smoke:-

When patients were questioned about the effect of smoke on their cough, it was considered that smoke could be present in the atmosphere from numerous sources including fumes from cigarette smokers and pipe smokers.

Two patients complained of cigarette fumes aggravating their cough while two others found that the fumes from a pipe smoker irritated their cough.

Almost fifty per cent of my patients found that smoke from any source aggravated their cough.

		Number	Percentage
Effect on Cough	Increase	34	45.3
	No Effect	41	54.7

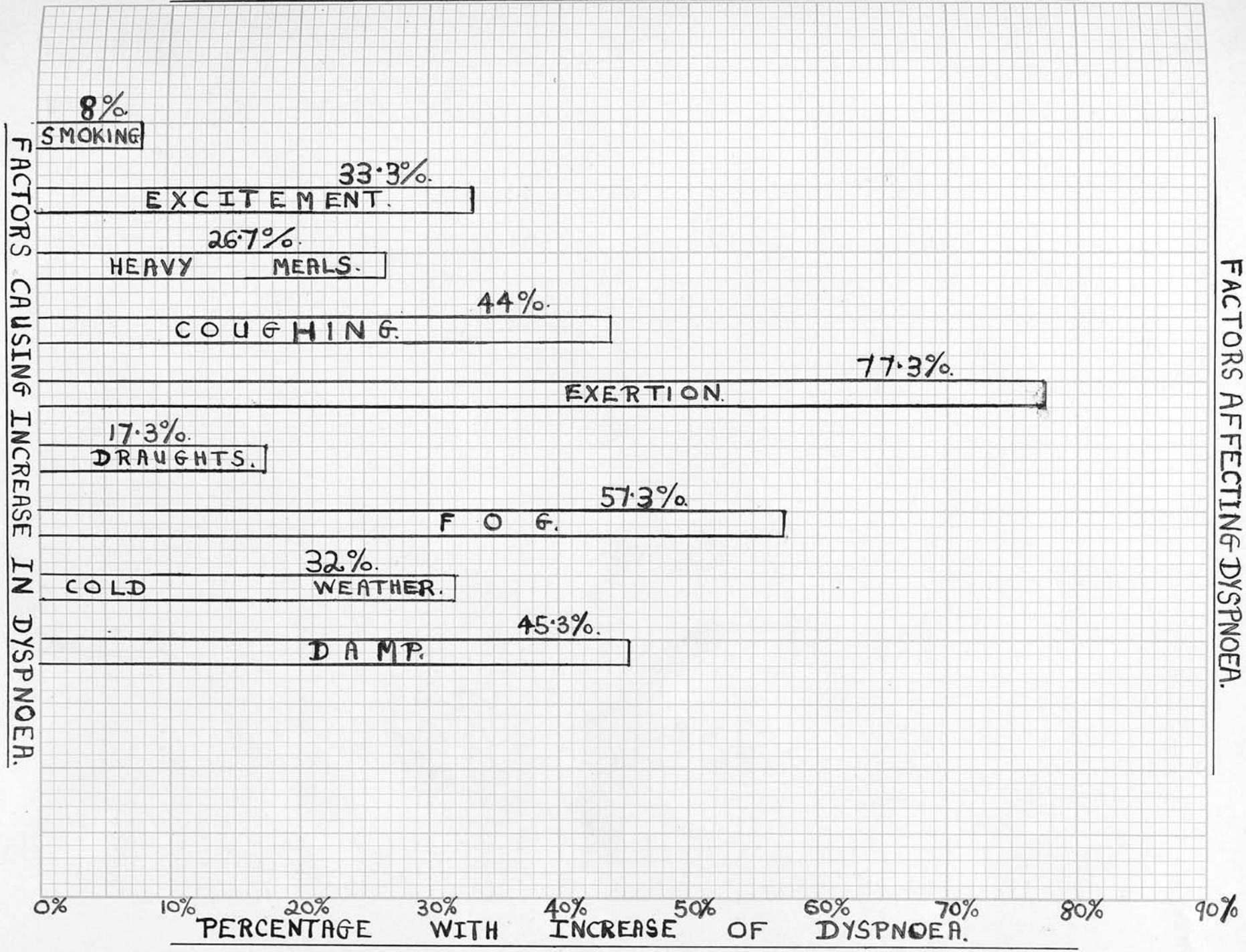
4. Wet :-

In comparison with other climatic factors, a smaller percentage of my chronic bronchitic patients found that their cough was aggravated when they were exposed to wet weather.

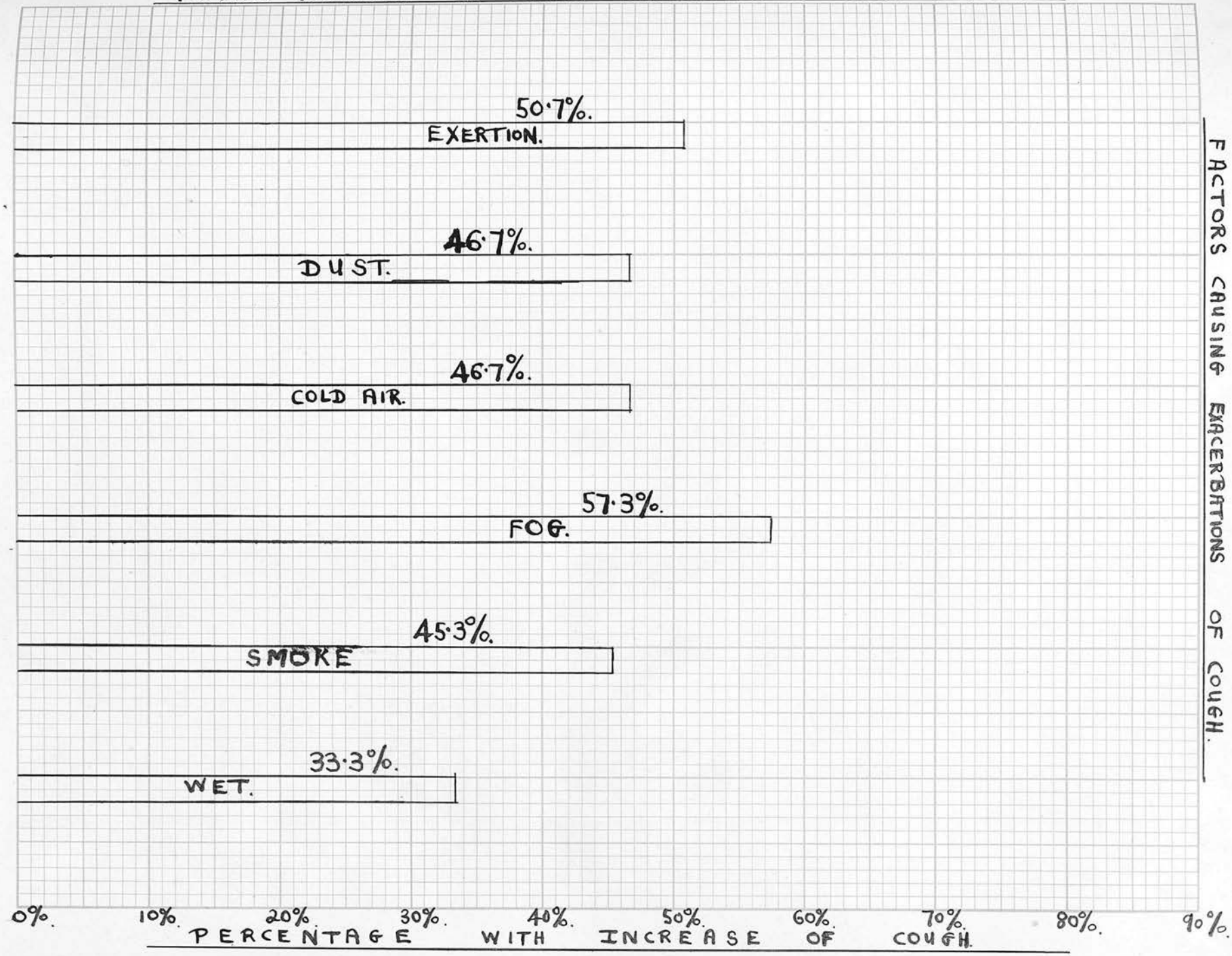
		Number	Percentage
Effect on Cough	Increase	25	33.3
	No Effect	50	66.7

The following two graphs illustrates the results of the factors affecting dyspnoea and those causing exacerbations of cough which have been studied in both sections :-

TOTAL NUMBER OF CASES INVESTIGATED SEVENTY FIVE.



TOTAL NUMBER OF CASES INVESTIGATED SEVENTY FIVE.



C. Factors diminishing cough :-

Dry weather and warmth both caused a diminution of cough in over forty per cent. of patients.

1. Dry Weather :-

One man noticed that it was cold or warm day weather which caused a diminution in his cough.

		Number	Percentage
Effect on Cough	Diminution	35	46.7
	No Effect	40	53.3

2. Warmth :-

Three patients found that their cough was increasedin warm atmospheric conditions.

		Number	Percentage
Effect on Cough	Diminution	37	49.3
	No Effect	35	46.7
	Increase	3	4

Experience has shown that the collection and measurement of the sputum does not present insurmountable difficulties. Alstead (1940) wrote that the quantity of the secretion might be determined by volume or by weight. For my observations in general practice I only measured the quantity of sputum by volume and the photograph inserted later in this thesis will help to illustrate the following description of the apparatus used.

After Alstead (1940), a number of heat-resistant glass test-tubes were used and each test-tube

measured about 8 x 1½ inches. These tubes were able to be boiled and sterilised periodically. Each tube was calibrated for volume by markings made at intervals representing 10 mils from 170 mils to 10 mils.

A block of wood with edges about 4 inches long was used as a stand for each test-tube and the tube was inserted into a hole 3½ inches deep, drilled to be just wide enough to receive it.

The block and its contained tube was then placed at the patient's bedside on a table or if the patient was up on a suitable table in the living room.

Alstead (1940) recorded that, although there was some admixture of saliva with the sputum, observations showed that it was less than might be supposed since the sufferer from chronic bronchitis mobilised the mass of sputum and ejected it without retaining it in the mouth for more than a second or two. The amount of saliva in the mouth at this time was no more than was necessary to moisten the buccal mucosa. Furthermore, such contamination as occurred was roughly from day to day. This was important, because in studying the course of bronchitis as indicated by the output of sputum, we were mainly concerned with changes (increase or decrease) rather than with absolute values.

The amount of secretion was recorded as the output for twenty-four hours and both the quantity of

fluid secretion and the quantity of froth were recorded for each of the four cases daily during the months when observations were undertaken.

All four patients kept a daily record also of the atmospheric weather conditions.

A survey could then be made of the daily sputum quantity and its relationship to the changing seasons of the year from winter to the early summer months.

A record was made in all four cases of the therapy administered and any alterations in that therapy.

Unfortunately two of the patients died during the time that these observations were being undertaken but the other two patients definitely showed a decrease in the daily quantity of their sputum when the warmer weather gradually superseded the cold winter days in the months of April and May.

The following graph illustrates the sputum records in these latter two cases and the period of observation chosen for this graph is from the end of March to May in 1955. Observations in one of the cases were discontinued at the beginning of May because the patient felt so much better in his general health and his sputum quantity was so low that I

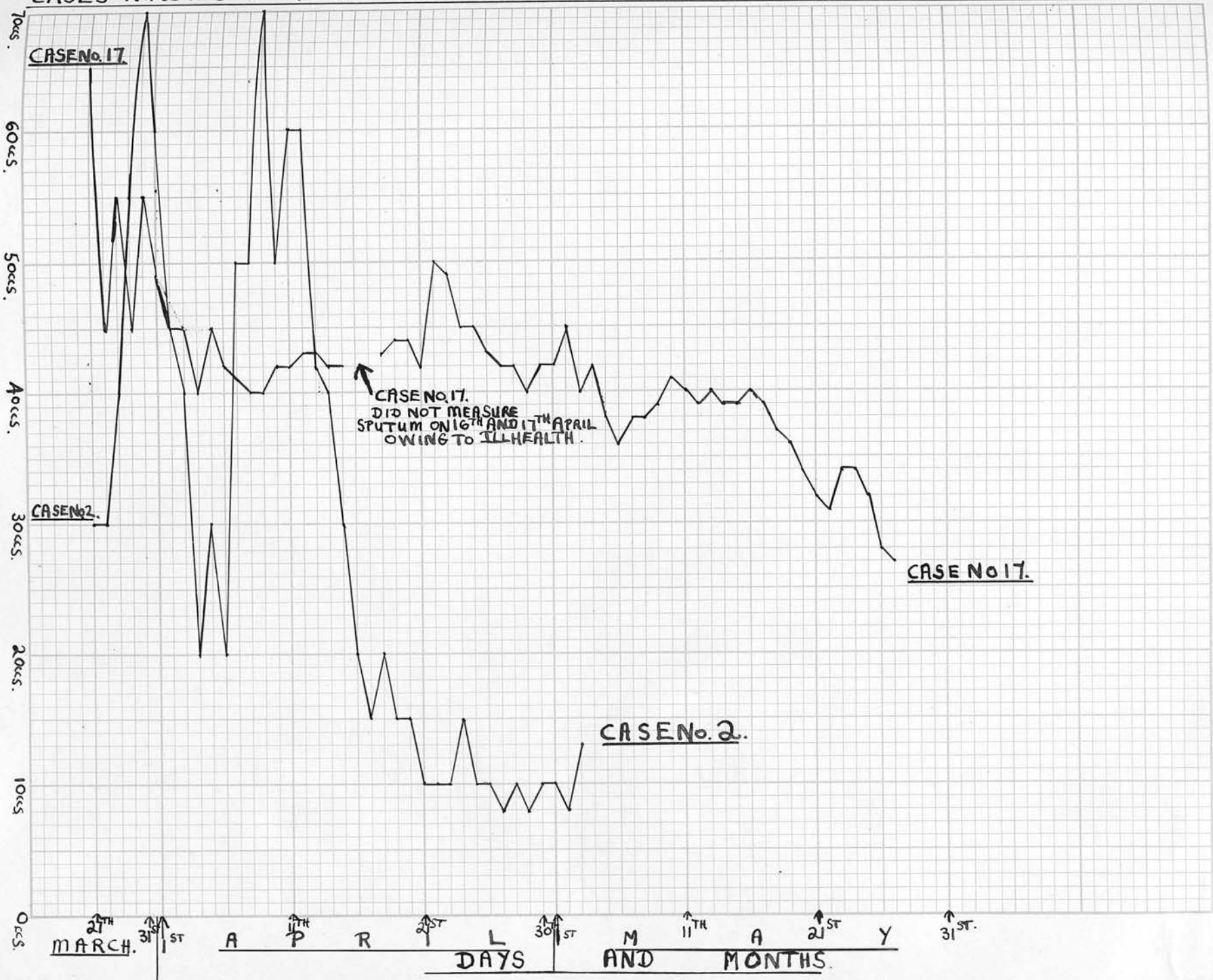
thought he would gain far more by heliotherapy than by continuing these readings. This patient has continued in good general health and a low sputum output throughout the summer months.

Tables recording these results in all 4 cases and illustrating the seasonal incidence in all 75 cases will be found later in this thesis.

CASES No. 2 AND No. 17 OF THESIS - CASE No. 2 DISCONTINUED FOR HELIOTHERAPY ON 4TH MAY.

SPUTUM MEASUREMENTS FROM 27TH MARCH TO 27TH MAY 1925.

DAILY TWENTY FOUR HOUR SPUTUM QUANTITY MEASURED IN CUBIC CENTIMETRES.



4. Hereditary Predisposition :-

An investigation was undertaken into the incidence of chronic bronchitis among the relations of the seventy-five chronic bronchitic patients in my series of cases.

A control series of seventy-five patients with no personal history of chronic bronchitis was chosen from patients reporting sick in my practice and as far as possible comparable age groups with the chronic bronchitic patients were chosen. Each control case belonged to the same sex as the chronic bronchitic patient.

Although some patients among the chronic bronchitic series had relatives who had suffered from "bronchitis", the following Table shows the number of chronic bronchitic patients with relations who suffered from "chronic bronchitis".

Patients with Chronic Bronchitis with History of Chronic Bronchitis among Relations.	Number	Percentage
Case Nos.2, 3, 5, 9, 10, 13, 15, 16, 17, 18, 19, 20, 25, 26, 31, 32, 33, 37, 39, 40, 44, 46, 47, 50, 52, 55, 57, 62, 65, 70, 72, 74.	32	42·7

A Table illustrating details of the relatives with this disease is shown along with respiratory illnesses among relatives in the section on Family History.

Of the seventy-five patients in the control series, only seven had relatives with chronic bronchitis.

	Number	Percentage
Patients in control series with History of Chronic Bronchitis among Relations	7	9.3

From these two Tables it is shown that patients with chronic bronchitis had a far greater incidence of a similar disease in their family history than the control series which suggested hereditary predisposition.

5. Factors in Onset :-

There was a gradual development of the disease in all the cases studied over variable periods of time but in no case was the history of development less than a year in duration.

In certain cases however it was impossible to ascertain any particular factor which caused the onset or predisposed to the development of the disease apart from repeated attacks of acute bronchitis.

In the following cases however, the gradual development of the disease was accompanied by attacks of bronchial asthma.

Patients in which development of disease accompanied by attacks of Bronchial Asthma.	Number	Percentage
Case Nos. 1. 5. 11. 12. 18. 20. 21. 26. 28. 33. 35. 38. 46. 62. 65. 69.	16	21.3

In two cases Nos. 22 and 23 the disease followed a previous history of bronchial asthma and it appeared that bronchial asthma was certainly a factor in the onset.

Nine patients had a history of respiratory disease, apart from acute bronchitis, at the onset or during the course of chronic bronchitis or prior to the onset of the disease. From the duration of symptoms of chronic bronchitis, it was found that a history of respiratory disease, other than acute bronchitis, was responsible for the onset of disease in four of the cases and in another case an attack of pneumonia preceded the onset of the disease by four years.

The following Table illustrates my observations :-

Case No.	Duration of Symptoms of Chronic Bronchitis Years	Other Respiratory Illnesses and Year.	Time in relation to course of Chronic Bronchitis.
16.	44.	Broncho Pneumonia in 1911.	At onset of Disease.
18.	2.	Right Apical Pneumonia in 1954.	During Course.
27.	6.	Pneumonia in 1919 and 1927. Left sided Pneumonia in 1954.	Prior to onset of Disease during Course.
28.	25.	Pneumonia in 1930. Pneumonia in 1954.	At onset of Disease. During Course.
41.	3.	Left sided Pneumonia & Pleurisy 1952.	At onset of Disease.
54	6.	Left Basal Pneumonia 1955.	During Course.
56.	31.	Broncho Pneumonia in 1949.	During Course.
68.	9.	Left sided Pleurisy in 1946.	At onset of Disease
70.	2.	Left sided Pneumonia in 1943.	Prior to onset of Disease.

Eleven of my patients had a history of upper respiratory tract infection and nine of these patients informed me that an attack of coryza always preceded the onset of a further attack of bronchitis.

Four of my patients were subjected to war gases in the 1914-1918 World War and these cases have already been discussed in the Section on Occupation and Habits.

Four patients attributed fevers to the development of the disease since, in each case, the respective fever directly preceded the onset of the disease and was a responsible factor in its onset.

Case Number	Fever.
17.	Influenza.
25.	Scarlet Fever.
61.	Influenza.
74.	Whooping Cough.

One patient informed me that he had suffered from symptoms of the disease since 1944 and that these symptoms had been immediately preceded by an accident to his chest in the same year. As the result of a fall he had fractured his left scapula and suffered from concussion.

Finally an elderly lady blamed exposure for the onset of her symptoms in 1940. In that year she fractured her right leg but had to wait for one hour in the snow-laden street for an ambulance to take her to hospital for treatment.

6. Sex :-

The great majority of patients in my series belonged to the male sex as can be seen by the following Tables :-

	Male.						Female.					
Number 46.			Percentage 61·3				Number 29.		Percentage 38·7			
Case No.	1	2	3	4	5	6	7	8	9	10	11	12
Sex.	F	M	F	M	F	M	F	M	F	M	M	F
Case No.	13	14	15	16	17	18	19	20	21	22	23	24
Sex.	M	F	M	F	M	F	M	F	M	M	M	M
Case No.	25	26	27	28	29	30	31	32	33	34	35	36
Sex.	M	M	M	M	M	M	F	F	F	F	F	M
Case No.	37	38	39	40	41	42	43	44	45	46	47	48
Sex.	M	M	M	M	M	F	M	M	M	F	M	M
Case No.	49	50	51	52	53	54	55	56	57	58	59	60
Sex.	M	M	M	F	M	M	M	M	F	M	F	F
Case No.	61	62	63	64	65	66	67	68	69	70	71	72
Sex.	F	F	F	F	M	M	M	M	F	F	M	F
Case No.	73	74	75									
Sex.	F	M	M									

7. Family History :-

An investigation was made into the incidence of respiratory illness among the relatives of those cases in my series suffering from chronic bronchitis. Over sixty per cent. of these patients informed me that they had relatives with a history of respiratory illness.

75 Cases suffering from Chronic Bronchitis.

	Number	Percentage
Cases with Family History of Respiratory Illness	47	62·7
Cases with no Family History of Respiratory Illness	28	37·3

Seventy-five control cases were chosen at random from patients reporting sick with no history of chronic bronchitis. These patients were interrogated when they came to the surgery or were questioned when they were visited at their homes. The control case was of the same sex and as far as possible of the same age as the chronic bronchitic patient. Only thirty-seven per cent. of the control series reported a history of respiratory illness among relatives.

75 control cases with no history of Chronic Bronchitis.

	Number	Percentage
Cases with Family History of Respiratory Illness	28	37·3
Cases with no Family History of Respiratory Illness	47	62·7

The following Table applies only to those patients suffering from chronic bronchitis in my series of cases who had relatives with a history of respiratory illness and it illustrates the respiratory disease from which the relative suffered :-

Case Number.	Relative with Respiratory Illness and Respiratory Disease.
2.	Grandfather and Grandmother had Chronic Bronchitis and Bronchial Asthma. Daughter suffers from Chronic Bronchitis, Bronchial Asthma and Pulmonary Tuberculosis. His cousin's daughter has Bronchial Asthma. His youngest son had Bronchial Asthma up to age of sixteen.
3.	Her elder sister died of Chronic Bronchitis after two years' course. Her younger sister died of Bronchial Asthma.
5.	Her husband and elder brother both suffer from Chronic Bronchitis. Her son had Bronchitis, March 1953. Two of her cousins suffer from Chronic Bronchitis and Bronchial Asthma.
6.	Wife had Bronchitis and left-sided Pleurisy in April 1954.
9.	Her mother had Chronic Bronchitis. Her husband suffered from left-sided Pneumonia in May 1955. Her son suffered from Acute Bronchitis in 1952 and 1955.
10.	His mother had Chronic Bronchitis. Younger sister has Chronic Bronchitis. Elder brother died of Pulmonary Tuberculosis following Malaria.
13.	Wife (now widow) suffers from Chronic Bronchitis.
14.	Her daughter had Pneumonia.
15.	One of his elder sisters developed Bronchial Asthma in early childhood but later developed and died of Chronic Bronchitis.
16.	Two of her elder brothers had Chronic Bronchitis.

Case Number.	Relative with Respiratory Illness and Respiratory Disease.
17.	His father suffered from Chronic Bronchitis and Bronchial Asthma. His brother suffered from Chronic Bronchitis and Bronchial Asthma and died of Pulmonary Tuberculosis. His wife had Acute Bronchitis in January 1954. One sister had Bronchial Asthma and died of Pulmonary Tuberculosis while another sister suffers from Bronchitis at intermittent intervals.
18.	Her husband suffers from Chronic Bronchitis. Her son had laryngitis and bronchitis in January 1953.
19.	His sister-in-law suffers from Chronic Bronchitis and Bronchial Asthma.
20.	Her brother-in-law suffers from Chronic Bronchitis and Bronchial Asthma.
24.	His father died of Bronchopneumonia.
25.	His grandmother suffered from Chronic Bronchitis.
26.	His sister suffers from Chronic Bronchitis. His daughter suffers from attacks of Bronchial Asthma.
31.	Her husband had Chronic Bronchitis.
32.	Her mother had Chronic Bronchitis.
33.	Her husband suffered from Chronic Bronchitis for 35 years and her mother-in-law had Chronic Bronchitis. Her elder brother also suffered from Chronic Bronchitis. One sister had Pulmonary Tuberculosis.

Case Number	Relative with Respiratory Illness and Respiratory Disease.
35.	Her daughter suffered from attacks of Acute Bronchitis until she emigrated to Sydney in Australia and since living in Australia she has suffered from no attacks of Bronchitis or Coryza. Her grand-daughter suffered from Bronchitis with Bronchial Spasm in July 1954.
36.	His sister had Bronchitis.
37.	His grandmother (on mother's side) had Chronic Bronchitis. His uncle had Chronic Bronchitis and Bronchial Asthma and died of Cor Pulmonale. One brother has Chronic Bronchitis and Bronchial Asthma.
39.	Aunt on his father's side had Chronic Bronchitis. Uncle on father's side had Bronchial Asthma.
40.	His mother suffered from Chronic Bronchitis.
41.	His youngest daughter developed Bronchial Asthma at age of 2 following Whooping Cough and suffered from Bronchial Asthma up to the age of 15. His son had Pulmonary Tuberculosis.
42.	Her grandfather and aunt both had Bronchitis.
43.	His grandson has occasional attacks of Acute Bronchitis.
44.	His wife suffers from Chronic Bronchitis and his brother-in-law also suffers from Chronic Bronchitis.

Case Number	Relative with Respiratory Illness and Respiratory Disease.
45.	Two of his younger brothers died of Pulmonary Tuberculosis.
46.	Her uncle on mother's side (older brother of mother) died of Chronic Bronchitis. Her mother had Bronchitis in infancy.
47.	His father had Chronic Bronchitis and died of Pneumonia.
48.	His elder sister died of Pulmonary Tuberculosis. His elder brother suffers from Pulmonary Tuberculosis.
50.	Uncle (father's eldest brother) died of Chronic Bronchitis and Bronchial Asthma. Uncle (father's 2nd eldest brother) has Chronic Bronchitis and Bronchial Asthma.
51.	Two of his brothers died in infancy from Bronchitis.
52.	Her brother had Chronic Bronchitis and Pleurisy and died of Pneumonia. Her sister suffers from Chronic Bronchitis. Her father died of Pneumonia.
55.	Younger sister and brother-in-law both suffer from Chronic Bronchitis.
57.	Her mother suffered from Chronic Bronchitis.
62.	Her mother suffers from Chronic Bronchitis.
63	Her sister suffers from Bronchial Asthma.
64.	Three children out of family of five children (eldest - boy, third child - boy, and youngest-girl) have had attacks of Bronchitis and eldest - boy three attacks. Other two children in her family no attacks.

Case Number.	Relative with Respiratory Illness and Respiratory Disease.
65.	His mother suffered from Chronic Bronchitis from ages 36 to 75.
68.	His second son suffered from Pulmonary Tuberculosis.
70.	Her mother had Chronic Bronchitis.
71.	His youngest son had Bronchitis at the age of 3. Eldest son had Bronchitis in 1955.
72.	Her mother had Chronic Bronchitis.
74.	His mother had suffered from Chronic Bronchitis for the last two years.

8. Social State :-

Each patient in my series was questioned about the type of house in which he or she lived.

Nearly seventy-five per cent. of them lived in tenement buildings and thirty-six per cent of those who occupied tenements lived in overcrowded areas. There was no overcrowding in the other varieties of houses.

The following Table shows the results of these investigations in the seventy-five cases with chronic bronchitis :-

TYPE OF HOUSE.

	Tenements				Flatted Villas.	
	Ground Flat	1st Flat	2nd Flat	3rd Flat	Upper Apartment	Lower Apartment
Number	12.	14.	19.	10.	7	3
Total Number	55.				10.	
Percentage	73·3				13·3	

	Semi-Detached Villas.	Semi-Detached Houses.	Eventide Homes.	Prefabricated Houses.
Total Number	6	2	1	1
Percentage	8	2·7	1·3	1·3

OVERCROWDING.

Number living in Overcrowded Areas.	Type of House	Percentage of Cases Investigated.
27.	All lived in Tenements.	36.

PART III.

CONCLUSIONS.

1. A review of the aetiology in each individual case is important and any aggravating factor should be dealt with appropriately.

2. A definite disposition or constitution is necessary for the development of chronic bronchitis which may be looked upon as an exaggeration of the inspiratory phase of the respiratory complex.

3. The relationship between infection and bronchial mucus constitutes the essence of the pathogenesis of the disease since the volume of the secretion is greatly increased and the inflammatory process in the mucous membrane reduces the number of cilia so that the means of moving the secretion fails and it accumulates. To what extent mucus is the result of hypersensitivity, infection and external irritants is not clear but presumably all three play a part.

4. Hypersensitivity of chronic bronchitis is particularly related to infection, atmospheric pollution and climatic factors. Fog was found to aggravate the respiratory symptoms in patients with the disease in this series far more than any other single climatic factor although damp appreciably affected respiratory symptoms also. There was a reduction in the

daily quantity of sputum in those cases investigated between February and May and this illustrated the beneficial effect of cleaner, warmer and drier air on the individual with the disease. Adverse weather and little sunshine prevailed throughout the summer of 1954 and six patients in my series had attacks of bronchitis in this summer and these patients normally only had attacks in the winter months. Dry weather and warmth diminished the cough in nearly half the patients in my series.

Those who are fortunate enough to be able to do so should spend the winter in a warm dry climate and those who are unable to go away should endeavour to keep to an even temperature and remain indoors in damp foggy and windy weather in the winter months. Elderly people who live in a town or near the east coast and cannot afford to go abroad or to the south coast of England should endeavour to spend the winter in the country away from the east coast with country relatives if they are fortunate enough to have the latter.

5. The age of onset of the disease depends on the habits, occupation, general health and previous general and pulmonary history of the individual and the sex affected is predominantly male.

6. Occupations involving exposure to inclement weather, to dust and to risks of infection as well as those involving heavy muscular strain or prolonged exertion are unfavourable for those who suffer from the disease. Exertion aggravated the respiratory symptoms in over half of my series while nearly all those who were exposed to dust at their present or previous employment found that it aggravated their respiratory symptoms. Those engaged in unsuitable occupations which expose them to extreme variations of temperature, to dusty atmospheres or to inclement weather, should endeavour to change their occupations. The majority of cases in this series were employed in partly skilled and unskilled occupations.

7. Smoking, excitement, heavy meals, coughing and exertion aggravated breathlessness in certain cases in this series. Patients with chronic bronchitis should be advised to reduce or stop smoking and lead quiet but not sedentary lives. They should avoid heavy meals and should diet if they are overweight. Over exertion should also be avoided and the patient should be provided with a cough sedative.

8. The high incidence of the disease among relatives of those with chronic bronchitis suggests a hereditary predisposition. Chronic bronchitis seems to have a special incidence in some families.

9. The development of the disease in a number of cases in this series was accompanied by attacks of bronchial asthma. A severe acute respiratory illness preceded the onset of chronic bronchitis in some cases and careful treatment of acute respiratory illness is essential if the onset of chronic bronchitis is to be avoided. In all cases with the disease upper respiratory tract infection should be investigated and treated as chronic upper respiratory tract infections are causative and predisposing factors in the onset of the disease. Respiratory complications following fevers and exposure to war gases may be predisposing factors in the development of the disease, and a considerable increase in chronic pyogenic pulmonary diseases may be expected in the coming years following the second World War. Two mothers in this series found that the disease developed after the birth of children. Exposure, shock and chronic pulmonary, cardiac and renal disease and structural defects should all be considered as possible causative and predisposing factors.

10. Many of the patients studied in this series lived in tenements and a number of these tenements were overcrowded. A person who suffers from chronic bronchitis should be given priority for ground flat accommodation if he lives in the second or third flat of a tenement and those who live in overcrowded

tenements should be given priority either for other tenement accommodation or lodgement in new housing schemes.

New housing schemes should be built as far distant from factory smoke as possible and new tenements should be built with sufficient space between them to permit fresh air and sunshine to enter the rooms on all flats.

It is hoped that with the introduction of the Clean Air Bill that there will be a reduction in the high incidence of chronic bronchitis in this country.

PART IV.

SUMMARY OF CASE-HISTORIES.

In each case only the relevant clinical details are included as they affect the contents of this thesis.

Each case was clinically examined in detail and diagnosed as a case of chronic bronchitis from the clinical history and findings.

Certain hospital reports are included in the description but only those reports which apply to the subject matter of this thesis are included.

Case No. 1. Age 80. Widow.

Occupation: Retired housewife who lived in Eventide Home.

History: Gradual onset with recurrent attacks of bronchitis over the last 18 years which occurred both summer and winter and disease was accompanied by attacks of bronchial asthma. She had no family history of chest illness. She did not smoke. Worry and other mental anxieties, excitement, fog, damp, coughing and exertion all increased dyspnoea. Her cough was variable in frequency and cold air, fog and wet all aggravated it.

Hospital Reports: X-Ray of chest in 1953 showed nothing of note.

Case No. 2. Age 67. Male.

Occupation: Retired Able Seaman who was on a fishing cruiser.

History: He had no history of chest illness up to February 1952 when he developed acute bronchitis following acute coryza and since 1952 he had gradually developed chronic bronchitis. Attacks of bronchitis were always preceded by coryza and persisted both summer and winter. The disease was accompanied by attacks of bronchial asthma. His grandfather and grandmother had bronchial asthma and chronic bronchitis and his daughter had chronic bronchitis, bronchial asthma and pulmonary tuberculosis.

His cousin's daughter had bronchial asthma while his youngest son had bronchial asthma at the ages of 15-16. He lived in the third flat of a tenement but backroom of flat was penetrated at times by smoke from a near-by chimney stack situated just outside the window. In his former occupation, he was subject to varying climatic conditions and inclement weather. He did not smoke. Changes of atmosphere and damp both increased his dyspnoea and wheeziness while fog and exertion also increased his dyspnoea. His cough occurred mainly in the morning and cold air, fog, exertion, wet and dust aggravated his cough while dry weather diminished it. He also suffered from hypertension.

Hospital Reports: He was treated in Royal Infirmary in November 1952 for acute exacerbation of bronchitis and bronchospasm. An X-Ray of his chest in 1952 revealed emphysema.

Case No. 3. Age at death in 1955 was 69. Widow.

Occupation: Housewife but previously shirt machinist.

Cause of Death: Chronic Bronchitis and Myocarditis.

History: Acute Bronchitis developed in February 1951 but since February 1952 she had repeated attacks of bronchitis with productive cough, dyspnoea and attacks of wheeziness of chest in the winter and also in the summer. Her elder sister died of chronic bronchitis while her younger sister died of bronchial

asthma. She did not smoke. Excitement, heavy meals, fog, coughing, damp and exertion all increased her dyspnoea while smoke from the fire increased both her "wheeziness" and dyspnoea. Her cough occurred mainly in the morning and at night. Fog, exertion, smoke, wet and dust all aggravated her cough, while dry weather and warmth diminished it. Hospital Reports: In February 1953 she was treated for acute exacerbation of bronchitis in Leith Hospital. An X-Ray of her chest in 1954 showed emphysema which was moderate generalised and probably right basal bronchiectasis.

Case No. 4. Age at Death in 1955 was 66. Male.

Occupation: Retired Baker.

Cause of Death: Acute cardiac failure and chronic bronchitis.

History: While on active service in 1917, he was exposed to mustard gas which affected his respiratory system and eyes and he was blind for 9 days. He also worked in bakery from 1908, apart from war service, up to 1940 at hot ovens and high temperatures in the bakery but in that year he developed first attack of acute bronchitis and these attacks continued every year at intermittent intervals at first in winter only but since 1951 the attacks occurred throughout both summer and winter. He

continued working in hot atmospheres at bakery until 1943 but in that year he had to change to confectionary department owing to severity of bronchitis. He had no family history of chest illness. He lived in an overcrowded tenement on the top flat. He stopped smoking in February 1953 but previous to this date he used to smoke one ounce of tobacco a fortnight. Excitement, heavy meals, fog, cold weather, exertion, excess heat and high winds all increased his dyspnoea and he complained of a pain in his chest after coughing. His cough occurred mainly in the morning or at night and cold air, fog, exertion, smoke, dust and high winds all increased his cough.

Hospital Report: X-Ray of his chest in 1952 revealed emphysema.

Case No. 5. Age 57. Female. Married.

Occupation: Housewife.

History: Thirty years ago she developed first attack of acute bronchitis and gradual onset over last 30 years accompanied by attacks of bronchial asthma. Attacks occurred between June and September up to 1938 but since 1938 attacks came on both winter and summer. Her husband and elder brother both had chronic bronchitis and two cousins had bronchial asthma and chronic bronchitis while her son had acute bronchitis in March 1955. She did not smoke. Excitement, draughts, heavy meals, fog, cold weather,

damp, coughing and exertion all increased her dyspnoea. Her cough occurred mainly in the morning and cold air, fog, exertion and wet all increased her cough.

Hospital Report: X-Ray of her chest in 1950 revealed gross emphysema and further X-Ray of her chest in 1954 showed changes consistent with chronic bronchitis and emphysema.

Case No. 6. Age 50. Male.

Occupation: Skinner in tannery.

History: For the last 5 years, he had suffered from repeated attacks of bronchitis which occurred in winter and spring seasons. His wife had pleurisy and bronchitis in 1954. He lived in upper apartment of a two-storey flatted villa and smoked 15-20 cigarettes daily. Fog, coughing and exertion all increased his dyspnoea. His cough occurred mainly in the morning and fog, dust at work, and exertion all aggravated his cough while warmth diminished it.

Hospital Reports: X-Ray of his chest in 1954 revealed bronchitis and emphysema. Barium meal X-Ray in the same year showed a deformed duodenal cap without any active ulcer present.

Case No. 7. Age 76. Female. Married.

Occupation: Housewife.

History: Gradual onset of productive cough accompanied by bronchial spasm mainly in the winter

at intermittent intervals since 1943 was her present history. She had no family history of chest illness and she did not smoke. Excitement, heavy meals, fog, damp, coughing, exertion and very cold weather all increased her breathlessness. Her cough was variable in frequency and exertion and very cold weather increased her cough while warmth decreased it.

Hospital Report:- X-Ray of sinuses in 1953 showed gross mucosal thickening of both antra. An X-Ray of her chest in 1954 showed fibrotic tubercular lesion at both apices and marked increase in both hilar shadows which were mainly vascular and probably represented the effects of a cor pulmonale.

Case No. 8. Age 74. Male.

Occupation: Retired Hairdresser.

History: He had been subject to attacks of productive cough and dyspnoea occurring at intermittent intervals both summer and winter for the last 5 years. He had no family history of chest illness. He lived in the third flat of a tenement. He smoked a pipe and smoked 2½ ounces of tobacco a week. Damp increased his breathlessness. His cough was variable in frequency but he was uncertain as to factors which caused its aggravation.

Hospital Report: An X-Ray of his chest in 1951 showed some degree of bronchitic change throughout both lung fields.

Case No. 9. Age 61. Female Married.

Occupation: Housewife.

History: In 1932, she developed acute bronchitis and up to 1950 she suffered from occasional attacks of bronchitis. Since 1950 she had repeated attacks of bronchitis with productive cough and dyspnoea only in the winter. Her mother had chronic bronchitis and her husband had left-sided pneumonia in 1955 and her son had acute bronchitis in 1952 and 1955. She lived in 1st flat of a tenement. She did not smoke. Fog, damp weather and exertion all increased her breathlessness. Her cough occurred mainly in the morning. Fog, wet weather and exertion all aggravated her cough while dry weather and warmth diminished it. She suffered from psoriasis.

Hospital Report: X-Ray of her chest in January 1953 showed an increase in the lung markings consistent with chronic bronchitis.

Case No. 10. Age 67. Male.

Occupation: Up to 1950 he was a rivetter in a shipyard but since 1950 he has been a storeman in a ship-building yard working in a welding shop.

History:- In 1944, he fell from a staging at his work and suffered from concussion and fractured left scapula for which he was treated for 2 months in Leith Hospital. Since this accident he had suffered from repeated attacks of bronchitis occurring both

summer and winter. Older brother died of Pulmonary Tuberculosis after Malaria and mother had chronic bronchitis while his younger sister had chronic bronchitis. He lived in the 3rd flat of a tenement. He did not smoke. Excitement, fog, damp, draughts and exertion all increased his breathlessness. His cough occurred mainly in the morning and exertion, smoke, wet, fog and dust all aggravated his cough.

Hospital Reports: Bilateral radical antral operations were performed at Leith Hospital in April 1953. An X-Ray of his chest in February 1952 showed a heart shadow within normal limits but he had an old fracture of one of his ribs on the left side of the chest. This X-Ray also showed that both lungs were essentially emphysematous and had appearances compatible with chronic bronchitis.

Case No. 11. Age 48. Male.

Occupation: Painter.

History: At the age of 10 he had three attacks of acute bronchitis. At the age of 23, he suffered from acute bronchitis and for the last 25 years he had attacks of bronchitis, with productive cough and dyspnoea, occurring at intermittent intervals both summer and winter. He had no family history of chest illness. He smoked an ounce of tobacco a week and 4 - 7 cigarettes a day. After heavy meals

he felt breathless. Dust, which was present at his work when cleaning down wall paper or house dust, aggravated his cough. His cough affected him mainly in the afternoon or early evening and was accompanied by a tightness of the chest.

Hospital Report: X-Ray of his chest in May 1954 revealed no abnormality.

Case No. 12. Age 50. Female. Married.

Occupation: Housewife.

History: For the last 2 years she had a productive cough at intermittent intervals occurring both summer and winter. She was subject also to attacks of bromchial asthma and suffered from dyspnoea. She had no family history of chest illness. She did not smoke and lived in the ground flat of a tenement. Exertion increased her breathlessness while fog increased both dyspnoea and "wheeziness". Her cough occurred mainly in the early morning and changes of atmosphere aggravated her cough while cold weather diminished it.

Hospital Report: X-Rays of her chest and sinuses in June 1954 were negative.

Case No. 13. Age at death in 1955 was 65. Male.

Occupation: Retired foreman in ship-building yard - retired in 1955.

Cause of Death: Chronic Bronchitis, emphysema and ischaemic heart-disease.

History: Since July 1953 he had attacks of bronchitis with productive cough and dyspnoea which occurred both summer and winter. He also had hypertension. His wife had chronic bronchitis. He smoked 20 cigarettes daily up to 7 years ago when he stopped smoking for the rest of his life. Draughts, fog, damp and exertion all increased his breathlessness. His cough occurred mainly at night and was aggravated by fog, exertion, smoke and dust but was diminished by warmth and in dry weather.

Hospital Reports: An X-Ray of his chest and sinuses in October 1953 showed slight mucosal thickening of both antra and in his chest nothing apart from a small calcified focus at the right base. He developed a pertrochanteric fracture of his left femur without displacement as a result of a fall and this was treated in the Royal Infirmary by Smith Peterson nailing, the day after his admission, on 24th May 1955.

Case No. 14 Age 70. Widow.

Occupation: Housewife.

History: In 1940 she fractured her right leg and had to wait in the street covered with snow for one hour before the ambulance arrived to remove her to hospital. She developed acute bronchitis and since this year she was subject to attacks of bronchitis, with productive cough and dyspnoea,which occurred

only in the winter months from 1940 to 1953. In 1954 she had attacks of bronchitis also in the spring and in 1955 her bronchitis persisted throughout both summer and winter. Coryza usually preceded all her attacks of bronchitis. Her daughter had pneumonia in December 1953. She lived in a particularly overcrowded tenement on the 2nd flat. She did not smoke. Excitement, draughts, fog, cold weather, damp, coughing and exertion all increased her breathlessness. Her cough occurred mainly night and morning. Cold air, fog, exertion, smoke and wet all aggravated her cough while dry weather and warmth diminished it. She had myxoedema and angina of effort for which she received treatment at the Royal Infirmary in 1953.

Case No. 15. Age at death in 1954 was 67. Male.
Cause of death: Bronchopneumonia and uraemia.

Occupation: He was a carter up to mechanisation of British Road Services and after this he was a labourer and storeman with British Road Services.

History: In 1933 he developed acute bronchitis and since then up to his death in 1954 he was subject to attacks of bronchitis at first only in the winter but in the last 4 years of his life the attacks persisted throughout both the summer as well as the winter months. His elder sister died of chronic bronchitis which was preceded by bronchial asthma which developed in early childhood. He smoked 10

cigarettes a week. Excitement, heavy meals, fog, cold weather, damp, coughing and exertion all increased his breathlessness. His cough occurred mainly in the morning and at night. Cold air, fog, exertion and dust all aggravated his cough while dry weather and warmth diminished it. He died in Leith Hospital from bronchopneumonia and uraemia on 11th October 1954.

Case No. 16. Age 74. Female Single.

Occupation: Housewife.

History: At the age of 30 she developed bronchopneumonia and since this illness she had repeated attacks of bronchitis only in the winter months over the last 44 years. Two elder brothers had chronic bronchitis. She did not smoke. Exertion increased her breathlessness. Her cough was variable. Smoke and dust aggravated her cough, while dry weather and warmth decreased it. She also suffered from angina pectoris since 1954.

Case No. 17. Age 71. Male.

Occupation: Retired Draper.

History: While in the Merchant Navy in 1916 he developed influenza at the age of 32 and this was followed by pneumonia for which he was treated in hospital for 13 weeks. Since 1916 he suffered from bronchitis which first affected him after the above illness and came on in attacks only in the winter

months up to 1933. Since 1933 however the attacks of bronchitis persisted throughout summer and winter months. His father and brother both suffered from chronic bronchitis and bronchial asthma and the brother died of Pulmonary Tuberculosis. One sister had bronchial asthma and she died also of Pulmonary Tuberculosis. Another sister occasionally had attacks of bronchitis while his wife had acute bronchitis in January 1954. He did not smoke. Heavy meals, cold weather, coughing and exertion all increased his dyspnoea. His cough was variable in frequency and was aggravated by cold air, exertion, dust and wind.

Hospital Reports: X-Ray of his chest in May 1954 revealed emphysema and X-Ray of his sinuses in May 1954 revealed no abnormality. He had a transverse colostomy performed on 12th August 1955 for a carcinoma of the rectum.

Case No. 18. Age 58. Female Married.

Occupation: Up to 1950 she was a paper sorter but since 1950 she had been a bondworker.

History; In 1950 she had acute bronchitis. Since 1953 she had repeated attacks of bronchitis which occurred throughout both summer and winter months. She was subject to attacks of bronchial asthma also and had right apical pneumonia in 1954. Her son had laryngitis and bronchitis in February

1953. She did not smoke. Excitement increased her breathlessness. Her cough was variable in frequency and smoke increased her cough while warmth decreased it. She had to lift heavy objects and required to climb stairs at her work at the bond warehouse.

Hospital Report: X-Ray of her chest revealed a right-sided apical pneumonia which she had in April and May 1954, but another X-Ray of her chest at the end of May 1954 showed almost complete resolution of the pneumonia and all sputum specimens proved negative for tubercle bacilli.

Case No. 19. Age 48. Male.

Occupation: Dock Labourer.

History: For the last 2 years he suffered from attacks of bronchitis with productive cough and dyspnoea at intermittent intervals only in the winter months. His sister-in-law suffered from bronchial asthma and chronic bronchitis. He smoked 4 ounces of tobacco a week but was advised to reduce the quantity. Smoking increased his breathlessness and smoke from pipe smoking also increased his cough. Fog, cold weather, coughing and exertion all increased his breathlessness. Fog, exertion and sweating all aggravated his cough which occurred mainly night and morning. He developed a left facial palsy of lower motor neurone type in 1953. Dry weather and warmth decreased his cough.

Hospital Report: An X-Ray of his chest in February 1954 was negative.

Case No. 20. Age 49. Female. Married.

Occupation: Housewife.

History: At the age of 37, she had an attack of acute bronchitis. Since the age of 37 and the acute attack she had gradual development of chronic bronchitis with attacks of bronchitis and bronchial asthma at intermittent intervals occurring both summer and winter. Her brother-in-law had chronic bronchitis. She did not smoke. Attacks of bronchial asthma caused dyspnoea and sometimes in warm weather she felt breathless. Her cough was variable but attacks of bronchial asthma increased her cough. She was treated for anaemia in 1952 and 1953.

Hospital Report: An X-Ray of her chest in 1951 while she was an inpatient in the Royal Infirmary showed a vertical type of heart shadow and the hilar shadows were prominent, with increased linear markings radiating towards the bases. This might have indicated a degree of bronchiectasis and there was some emphysema generally.

Case No. 21. Age at death in 1954 was 65. Male.

Occupation: Tanner.

Cause of Death: Cerebral haemorrhage, congestive cardiac failure and chronic bronchitis.

History: At the age of 3 he fell into a dirty pool and swallowed some of the water from the pool. Since this incident he was subject to attacks of bronchitis at intermittent intervals and he gradually developed chronic bronchitis. At first his attacks came on both winter and summer from ages 3 to 15 but from the age of 15 to 64 he was only subject to bronchitis in the winter months. In the last year of his life the attacks persisted throughout summer as well as winter months. He had no family history of chest illness. He did not smoke during the last 5 years of his life but previous to this time he smoked 20-25 cigarettes a day. Coughing and exertion both increased his breathlessness. Cold air, exertion and smoke increased his cough which was variable in frequency while dry weather and warmth decreased it. He suffered from hypertension in 1954.

Case No. 22. Age 61. Male.

Occupation: Fitter in large engineering works.

History: Since 1944 he was subject to attacks of bronchial asthma which started when he was working in black-out conditions at the engineering works. Following development of bronchial asthma, of which he suffered the last severe attack in 1951, he had his first attack of acute bronchitis in 1951 and had repeated attacks of bronchitis and bronchial spasm

each winter up to 1954. In 1955 attacks came on both in the winter and the summer months. He had no family history of chest illness. He lived on 3rd flat of a tenement. He did not smoke. Draughts, cold weather and exertion all increased his breathlessness. He was uncertain as to what factors aggravated his cough but when he had bronchial spasms he found that this aggravated his cough. He suffered from auricular fibrillation in 1954. He went for his holidays every July to Shiel Bridge near the Kyle of Lochalsh which benefited his chest condition particularly in 1955.

Hospital Report: An X-Ray of his chest in May 1954 was negative.

Case No. 23. Age 14. Male.

Occupation: Schoolboy.

History: At the age of 4 he developed bronchial asthma from which he suffered for the last 10 years. He first suffered an attack of acute bronchitis in 1952 and since then he had recurrent attacks of bronchitis with productive cough every winter except for 1954 when attacks persisted throughout summer as well as winter. He had no family history of chest illness. Fog, coughing, exertion and attacks of bronchial asthma increased his breathlessness. His cough occurred mainly at night and in the morning and sawdust at carpentry at school, exertion and smoke all aggravated his cough.

Hospital Report: A report from the Ear, Nose and Throat Department at the Royal Infirmary in 1952 showed that he had definite allergic signs in his nose which linked up with his chest condition

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Case No. 24. Age 56. Male.

Occupation: Coal Man.

History: Chronic Bronchitis started with acute bronchitis 23 years ago and since first attack he was subject to recurrent attacks of bronchitis characterised by productive cough and dyspnoea on exertion at intermittent intervals in the winter only for the last 23 years. In 1955 he stopped carrying heavy loads and since working mainly in the office he noticed an appreciable improvement in his chest symptoms. His father died of bronchopneumonia. He lived in a particularly overcrowded tenement. He smoked 40 cigarettes a day and was recommended to reduce or stop smoking altogether in 1954. Smoking, fog, damp, coughing and exertion all increased his breathlessness. His cough occurred mainly in the morning and fog and lifting heavy loads both aggravated his cough. Dry weather and warmth both decreased his cough.

Hospital Report: An X-Ray of his chest in March 1954 revealed no abnormality.

Case No. 25. Age 46. Male.

Occupation: Salesman was his work up to 1941 but since 1941 he was employed as a bus conductor.

History: At the age of 7 he developed Scarlet Fever which was complicated by acute bronchitis. Since this illness he gradually developed chronic bronchitis with recurrent attacks of bronchitis at intermittent intervals over the last 39 years. Attacks of bronchitis were always preceded by coryza and up to the age of 24 occurred only in the winter but in the last 22 years have persisted throughout summer and winter. His grandmother had chronic bronchitis. He smoked up to 4 cigarettes daily. Cold weather and excessive heat made him feel choked but not breathless but exertion increased his breathlessness. He was uncertain as to the factors causing exacerbations of his cough which was variable in frequency.

Hospital Report: An X-Ray of his chest in 1951 revealed chronic bronchitis with some associated emphysema.

Case No. 26. Age 64. Male.

Occupation: Joiner and builder.

History: His house was bombed and after being bombed out of his house he developed acute bronchitis. For the last 8 years he suffered from attacks of bronchitis at intermittent intervals accompanied by attacks of bronchial asthma. Attacks of bronchitis occurred only in the winter months except

in 1954 when attacks persisted throughout both summer and winter months. His sister had chronic bronchitis and daughter suffered from bronchial asthma. He smoked 1 ounce of tobacco a week. Worry, heavy meals, fog, cold weather, damp, coughing, dust from dressing machine and exertion all increased his breathlessness. His cough occurred mainly at night and in the morning, and cold, air, fog, exertion, wet and dust all aggravated his cough while dry weather and warmth decreased it. At his work dust from dressing machine, which dressed the raw timber, caused both increase of dyspnoea and cough, but the sawdust at his work did not affect either cough or dyspnoea.

Hospital Report: In 1954 an X-Ray of his sinuses was negative while an X-Ray of his chest revealed gross emphysema in the month of April.

Case No. 27. Age 43. Male.

Occupation: National Assistance Claims Investigation Officer; previous to this employment in Fire Service.

History: While in the Fire Service he had no chest illness and at the ages of 7 and 15 he had pneumonia. Six years ago, after leaving Fire Service, he developed bronchitis and he had recurrent attacks of bronchitis since then which occurred in the winter months except for 1954 when attacks

persisted throughout summer and winter. He had left-sided pneumonia in 1954. He had no family history of chest illness. He ceased smoking in April 1954 but prior to this time he smoked 10 cigarettes daily. Heavy meals, cold weather, damp, coughing and exertion all increased his dyspnoea. His cough occurred mainly in the morning and was aggravated by smoke and wet.

Hospital Report: An X-Ray of his chest in May 1954 showed emphysema and mild chronic inflammatory changes at the right base.

Case No. 28. Age 45. Male.

Occupation: Barman.

History: At the age of 20 he had pneumonia in 1930. Since then he gradually developed chronic bronchitis with attacks of bronchitis in the winter only from 1930 to 1952 but in the last 3 years the attacks persisted throughout summer and winter. While in Bombay he developed bronchial asthma in 1943 and he was invalided out of the Royal Navy in December 1944 with a fractured os calcis and bronchial asthma. He had attacks of bronchial asthma and suffered from dyspnoea for the last 12 years. He had right basal pneumonia in February and March 1954. He had no family history of chest illness. He smoked 6 cigarettes daily. Excitement, draughts, fog, damp, coughing and exertion all increased his breathlessness.

His cough occurred mainly in the morning and at night. Fog, exertion, smoke, wet and attacks of bronchial asthma all caused exacerbations of his cough and once when in the surgery he found that the smell of floor polish in the surgery caused marked "wheeziness" of the chest and aggravated his cough.

Case No. 29. Age 43. Male.

Occupation: He was a blacksmith prior to 1935 and from 1943 onwards but between 1935 and 1943 he was a goods' porter.

History: At the age of 16 he developed acute bronchitis and for the last 27 years he has gradually developed chronic bronchitis with attacks of bronchitis characterised by cough and dyspnoea at intermittent intervals in the winter months only but in 1953 he did up his house and he suffered from dyspnoea constantly for 6 months. He had no family history of chest illness. He lived in a particularly overcrowded tenement. Since 1949 he stopped smoking but prior to this year he smoked 20 cigarettes daily and 3 ounces of tobacco a week. Exertion increased his breathlessness. His cough was variable and fog and smoke aggravated his cough while warmth diminished it.

Hospital Report: In 1953 an X-Ray of his chest and sinuses was negative.

Case No. 30. Age 66. Male.

Occupation: Retired Bodymaker. He retired on 3rd April 1954.

History: At the age of 57, he had acute bronchitis and since then he had repeated attacks of bronchitis with productive cough and dyspnoea which occurred in winter months only over the last 9 years. Attacks of bronchitis were always preceded by upper respiratory tract infection. He had no family history of chest illness. He smoked 2 ounces of tobacco a week but prior to 1945 he used to smoke 5 ounces of tobacco a week. His cough occurred mainly in the morning and no factors affected his dyspnoea or aggravated his cough. Even the dust at his former employment did not affect his chest symptoms.

Hospital Report: An X-Ray of his chest in December 1954 was negative.

Case No. 31 Age 65. Widow.

Occupation: Housewife.

History: She gradually developed chronic bronchitis over the last 4 years since 1951 when she had an acute attack of bronchitis. Since 1951 she was subject to attacks of bronchitis in the winter months only. She had no history of dyspnoea. Her husband had chronic bronchitis. She did not smoke. Cold air increased her cough which occurred mainly at night. She had a gastrectomy performed in 1935 and

since 1950 she was treated for iron deficiency anaemia.

History: An X-Ray of her chest in December 1953 was negative.

Case No. 32. Age 64. Female. Single.

Occupation: Retired Clerkess. She retired on 1st January 1954.

History: In 1951 she had an acute attack of bronchitis and for the last 4 years she was subject to attacks of bronchitis at intermittent intervals occurring throughout summer and winter months. These attacks were always preceded by upper respiratory tract infection and particularly coryza. Her mother suffered from chronic bronchitis. She did not smoke. Her cough occurred mainly in the morning and apart from attacks of coryza she knew of no other factor which aggravated her cough.

Hospital Report: An X-Ray of her chest in May 1953 was negative.

Case No. 33. Age 68. Widow.

Occupation: Housewife.

History: At the age of 3 she had acute bronchitis which recurred every winter until she was 7 years old. Since 1933, when she had a recurrence of acute bronchitis, she had attacks of bronchitis at intermittent intervals every winter and in the summer of 1954 she had bronchitis also as well as in the winter.

At the age of 33 she suffered from bronchial asthma but she had no recurrence of the latter until 1951 when an attack of bronchial asthma lasted one week and since 1953 she had periodic attacks of bronchial asthma. Her husband had chronic bronchitis for 35 years and her husband's mother also suffered from chronic bronchitis. Her elder brother had bronchitis and her sister had pulmonary tuberculosis. She used to stay in the 2nd flat of a tenement near the sea but since removing to 1st flat of tenement further away from the sea her chest condition improved. She does not smoke. Excitement, draughts, heavy meals, fog, damp, coughing and exertion all increased her breathlessness. Cold air, fog, exertion, smoke, wet and dust all aggravated her cough. She had osteo-arthritis of her right hip joint,

Hospital Report: An X-Ray of her chest was negative and X-Ray of the sinuses showed slight muco-sal thickening of both antra in November 1953.

Case No. 34. Age 45. Female Single.

Occupation: Charge-hand in transformer department of electrical engineering firm.

History: In early childhood she had bronchitis and in 1940 she had acute bronchitis. Since 1940 she had recurrent attacks of bronchitis every winter. She had no family history of chest illness. She did smoke 6-7 cigarattes daily. Excitement,

coughing and exertion all increased her breathlessness. Her cough was variable in frequency and was aggravated by dust but diminished by dry weather and warmth.

Hospital Report: An X-Ray of her chest in November 1953 was negative.

Case No. 35. Age at death in 1955 was 54. Female Married.

Occupation: Housewife.

Cause of Death: Massive pericardial effusion.

History: She had recurrent attacks of bronchitis for the last 22 years which occurred only in the winter months and were accompanied by attacks of bronchial asthma. Coryza always preceded these attacks which were characterised by productive cough and dyspnoea. Her daughter had bronchitis until she emigrated to Australia but since living in Sydney she had no bronchitis or coryza. Her grand-daughter had bronchitis with bronchial spasm in July 1954. She did not smoke. Fog increased her breathlessness. Her cough was variable in frequency and she was uncertain as to factors aggravating her cough. She developed Banti's Syndrome in the last few years of her life.

Case No. 36. Age at death in 1953 was 68. Male.

Occupation: He was a porter in Leith Docks for 12 years but after this he worked as a bondworker first as a maltsman and later with husks of barley.

Cause of Death: Cerebral Thrombosis following extra-pyramidal lesion caused by cerebral arteriosclerosis.

History: During the 1914-1918 World War he was twice subjected to mustard gas and lewisite. He developed chronic bronchitis with recurrent attacks of bronchitis both summer and winter from 1951 until his death in 1953. These attacks were characterised by productive cough and increasing dyspnoea on exertion. His sister had bronchitis. He smoked 1 ounce of tobacco a week. Excitement, heavy meals, fog, damp, coughing and exertion all increased his dyspnoea. His cough occurred mainly at night and exertion, smoke, dust and wind all aggravated his cough. There were large quantities of dust in the atmosphere where he worked with husks of barley. Smoking helped to make his cough more productive. In 1951 he had auricular fibrillation and hypertension.

Hospital Reports: In 1951 X-Rays revealed atheromatous changes in the aorta and gross osteo-arthritic changes in the spine and X-Ray of his chest showed emphysema and bronchitis. An X-Ray of his sinuses in 1953 showed slight diminution of air entry of right frontal sinus. An X-Ray of his chest later in 1953 showed possibly some basal bronchiectasis also.

Case No. 37. Age 63. Male.

Occupation: Maintenance engine man in gas works.

History: For the last 23 years he had frequent attacks of acute bronchitis characterised by productive cough and dyspnoea which occurred both summer and winter. These attacks were always preceded by coryza. His grandmother on his mother's side had chronic bronchitis and an uncle died of cardiac failure after chronic bronchitis and bronchial asthma. One of his brothers had chronic bronchitis and bronchial asthma. He smoked 10 - 12 cigarettes daily and drank occasionally about 1 pint of beer but if he smoked and drank in excess he noticed that his breathlessness increased in severity. Fog, cold weather, damp, coughing and exertion all increased his breathlessness. His cough was variable in frequency and was aggravated by cold air, fog, exertion, smoke and dust while dry weather and warmth diminished his cough.

Hospital Reports: X-Rays of his chest in January and December 1953 were both negative.

Case No. 38. Age 33. Male.

Occupation: Bus conductor.

History: He had recurrent attacks of bronchitis since 1951 which occurred only in the winter of that year but persisted throughout both summer and winter of the years 1952 to 1955. He was

afflicted also with bronchial asthma and in 1952 he had Besnier's Prurigo. He had no family history of chest illness. He smoked 1 ounce of tobacco a week. Warm moist weather, draughts, heavy meals, fog, cold weather, damp and prolonged exertion all increased his breathlessness which was also aggravated by attacks of bronchial asthma. Cold air, fog, exertion, dust and warm moist weather increased his cough, which occurred mainly morning and night, while warmth and dry weather decreased it.

Hospital Reports: He had a submucous resection of nasal septum performed in December 1953. An X-Ray of his sinuses in September 1953 revealed an opacity of right maxillary antrum and X-Ray of chest at same time was negative.

Case No. 39. Age 56. Male.

Occupation: Headmaster.

History: He developed his first attack of acute bronchitis in 1952 and since then he had recurrent attacks of bronchitis which occurred only in the winter months except for the summer of 1954 when he had attacks of bronchitis. An aunt on his father's side had chronic bronchitis while an uncle on his father's side had bronchial asthma. He stopped smoking in 1949 but prior to this year he smoked 20 - 25 cigarettes a day and 1 ounce of tobacco a week. Excitement, fog, and exertion all increased his

breathlessness. Cold air, fog, smoke and dust all increased his cough which was variable in frequency. He had hypertension in 1955 and had a haematemesis from a duodenal ulcer in the same year.

Hospital Report: An X-Ray of his chest and sinuses in December 1953 was negative.

Case No. 40: Age 70. Male.

Occupation: He was a sileman in a wheat mill from 1926 to 1938 and was a foreman in a wheat mill from 1938.

History: He gradually developed chronic bronchitis with attacks of productive cough and dyspnoea only in the winter over the last 3 years since 1952. His mother had chronic bronchitis. He did not smoke. Cold weather, damp and coughing all increased his breathlessness. Cold air, fog, exertion, smoke, wet and dust all aggravated his cough, which occurred mainly in the morning, while warmth and dry weather diminished his cough. He was subject to the inhalation of appreciable quantities of dust while he worked as a sileman.

Hospital Report: An X-Ray of his chest in January 1954 was negative.

Case No. 41. Age 59. Male.

Occupation: Retired Able Seaman in Merchant Navy.

History: He had bronchial asthma from ages 1½ to 21 when he developed measles and since the latter disease he had no further attacks of bronchial asthma. He had left-sided pneumonia and pleurisy in October 1952 and since then he was subject to recurrent attacks of productive cough and dyspnoea at intermittent intervals for the last 3 years. These attacks came on only in the spring and autumn from 1952 to 1953 but in 1954 and 1955 they were present in both summer and winter months. He had bilateral pleurisy in 1953. His youngest daughter had bronchial asthma which followed whooping cough at the age of 2 and also ceased to afflict her at the age of 15. His son had Pulmonary Tuberculosis. Until a few years ago he lived in the 3rd flat of a very steep tenement but he lived now in a prefabricated house. He smoked 1 ounce of tobacco a week. Smoking, heavy meals, fog, and damp all increased his breathlessness. Fog and dust increased his cough, which occurred at night mainly, while cold air, dry weather and warmth decreased his cough. He was crippled with severe rheumatoid arthritis.

Hospital Report: X-Rays of his chest in November 1952 and March 1953 showed resolution of the inflammatory lesion at the left base of his chest.

Case No. 42. Age 78. Female. Single.

Occupation: Housewife.

History: She was subject to attacks of bronchitis, at intermittent intervals, characterised by productive cough and dyspnoea for the last 6 years. Attacks came on only in winter from 1949 to 1953 but from 1953 to 1955 she was subject to attacks of bronchitis throughout the summer and winter months. A grandfather and aunt both had bronchitis. She did not smoke. Fog, cold weather and coughing all increased her breathlessness. Her cough occurred mainly at night and was aggravated by cold air, fog, wet and dust.

Hospital Reports: An X-Ray of her sinuses in November 1953 was negative while an X-Ray of her chest at the same time revealed calcified glands at the left hilum.

Case No. 43. Age 67. Widower.

Occupation: He was a lorry driver prior to his present occupation as blacksmith.

History: He had his first attack of acute bronchitis in 1950 and over the last 5 years he suffered from cough and dyspnoea which came on during both summer and winter months but came on more frequently in the last 2-3 years. His grandson had occasional attacks of bronchitis. He smoked 10 cigarettes daily. Exertion increased his breathlessness. His cough occurred mainly in the morning and at night and was aggravated by exertion, dust and smoke and also by warmth.

He suffered also from hypertension and obesity. He was on obesity diet.

Hospital Reports: X-Ray of his chest in 1953 revealed appearances consistent with hypertension. X-Ray of his chest and sinuses in July 1954 showed a wide aortic arch with catarrhal changes at lung bases especially the right base and opaque antra on both sides.

Case No. 44. Age 59. Male.

Occupation: He was employed in navvy work up to December 1953 but since then he drove an electric hammer in a blacksmith's shop.

History: He developed acute bronchitis in 1950 and since then he was troubled with recurrent attacks of bronchitis which lasted 5-6 weeks at each attack. These attacks came on only in the winter. His wife and brother-in-law both suffered from chronic bronchitis. He smoked 5 cigarettes a day. Heavy meals, cold weather, coughing and exertion increased his breathlessness. The latter was also aggravated by smoke from the fire in the blacksmith's shop. Cold air, fog, exertion and smoke from fire in the blacksmith's shop all aggravated his cough. His cough occurred mainly ay night and in the morning and was diminished by dry weather and warmth.

Hospital Reports:- An X-Ray of his chest in February 1954 showed evidence of chronic bronchitis

and kyphosis of the chest. He had a duodenal ulcer and X-Ray barium meal in February 1954 showed some deformity of the duodenal cap.

Case No. 45. Age 58. Male.

Occupation: Bakers' ovensman.

History: In the 1914-1918 World War, he was subjected to chlorine from a high explosive gas shell at Arras. Since then he had recurrent attacks of bronchitis from 1917 for the last 38 years and these attacks recurred throughout summer and winter. In 1952 he started to suffer dyspnoea on exertion. Two younger brothers died of Pulmonary Tuberculosis. He lived in the 3rd flat of a tenement. He smoked 10 - 15 cigarettes a day. Fog, damp and exertion all increased his breathlessness. Cold air, exertion, wet and dust all aggravated his cough which occurred mainly in the morning. At the bakery flour dust caused sneezing and wheeziness of his chest. An X-Ray of his sinuses in April 1953 was negative while an X-Ray of his chest at the same time showed calcified tubercular lesions at both apices but no obvious active disease.

Case No. 46. Age 53. Female. Married.

Occupation: Housewife.

History: Since the birth of her second child 28 years ago she has gradually developed chronic bronchitis which was characterised by attacks of

bronchitis at varying intervals of time throughout the summer and winter. This development was accompanied by bronchial asthma. Her mother had bronchitis in infancy and an uncle on her mother's side died of chronic bronchitis. She lived in 3rd flat of a tenement. She did not smoke. Attacks of bronchial asthma, excitement, heavy meals, fog, coughing and exertion all increased her dyspnoea. Her cough occurred mainly in the morning and was aggravated when she had attacks of bronchial asthma and by frosty cold air, exertion, smoke, wet and dust but was diminished during dry weather and in warm atmospheres.

Hospital Report: An X-Ray of her chest in May 1955 showed some hazy shadowing in the right upper zone but the appearances were not those of pulmonary tuberculosis but resultant upon bronchial asthma and chronic bronchitis according to the report.

Case No. 47. Age 70. Widower.

Occupation: Retired clerk manager.

History : He had his first attack of acute bronchitis in 1948 and since 1948 he had intermittent attacks of acute bronchitis at first only in the winter months but since 1953 attacks were present throughout summer as well as the winter months. He had a ductal hyperplasia of right breast treated by

mastectomy in 1953 and developed a ductal hyperplasia of left breast in 1955. In July 1955 he developed a swelling of face and neck which was accompanied by palpable cervical and axillary glands. He was admitted to Leith Hospital on the 23rd August 1955 for radiotherapy for carcinoma of chest revealed by X-Ray with secondary spread.

His father had chronic bronchitis and died from pneumonia. Up to 1953 he smoked 2½ ounces of tobacco a week and 4-5 cigarettes a day but since 1953 he only smoked 9 cigarettes daily and ceased pipe smoking. Exertion increased his breathlessness. Lying down, cold air and fog all aggravated his cough while smoking helped him to expectorate the sputum. His cough occurred mainly at night though at times it affected him constantly. Dry weather and warmth decreased his cough.

Hospital Reports: X-Ray of his chest at Leith Hospital on 15th August 1955 showed an extensive opacity in the right upper zone with widespread emphysema in the other parts of the lungs.

Case No. 48. Age 49. Male.

Occupation: Electrician.

History: He had first attack of acute bronchitis in 1924 and for the last 31 years he had attacks of bronchitis with productive cough at first only in the winter but from 1948 attacks occurred

throughout summer and winter. He had dyspnoea on exertion for the last 4 years. He had a duodenal ulcer and was awaiting surgical treatment for this disability. His cough troubled him mainly in the morning. His elder brother had pulmonary tuberculosis. He lived in the 3rd flat of a tenement. Up to September 1954 he smoked 12 - 15 cigarettes daily but since that date he only smoked 10 cigarettes daily. Draughts, fog, cold weather, damp and exertion all aggravated his dyspnoea. Cold air, fog and wet all aggravated his cough while dry weather and warmth decreased it.

Hospital Report: An X-Ray of his chest in 1949 showed emphysema and there was a calcified nodule in the right mid-zone compatible with an old Koch focus and lung markings were rather heavy in both lower zones.

Case No. 49. Age 55. Male.

Occupation: Driller.

History: While in the infantry at Callender he contracted acute bronchitis as a result of sleeping out on hills exposed to cold damp weather and this attack lasted for 4 weeks in December 1940. Since 1940 he had repeated attacks of bronchitis which only occurred in the winter mainly between December and March. He was discharged from the Army in 1942 because of chronic bronchitis. He had no family history

of chest illness. Up to July 1953 he smoked 12 cigarettes daily but since then he only smoked 2 cigarettes daily up to January 1955 when he stopped smoking altogether. Smoking, excitement, heavy meals, fog, damp, coughing and exertion all increased his breathlessness. Cigarette smoking, eating food, cold air, fog, exertion, wet and dust from cast iron off the drill at his work all aggravated his cough which occurred throughout day and night when he had attacks of bronchitis. Cold dry weather and warmth both decreased his cough.

Hospital Report: An X-Ray of his chest in 1949 showed evidence of bronchitis and an X-Ray of his sinuses in 1955 was negative.

Case No. 50. Age 13. Male.

Occupation: Schoolboy.

History: He had an attack of acute bronchitis in October 1953 and since then he had recurrent attacks of bronchitis in the winter months only. His uncle, an elder brother of his father, died of chronic bronchitis and bronchial asthma, while another uncle, who was also a brother of his father, had chronic bronchitis and bronchial asthma. He lived in the 1st flat of a tenement in a very overcrowded tenement. Exertion increased his breathlessness. Laughter, fog, exertion and smoke all aggravated his

cough, which occurred mainly at night, while dry weather and warmth diminished it.

Hospital Report: In January 1955 an X-Ray of his left antrum showed that it was opaque while an X-Ray on his chest was negative.

Case No. 51. Age 73. Male.

Occupation: He was a retired night-watchman but previous to that job he was a bricklayer's labourer.

History: He developed chronic bronchitis which started 4 years ago with acute attack of bronchitis. He was repeatedly subjected to attacks of bronchitis both in the summer and winter months over the last 4 years with productive cough and dyspnoea on exertion. He himself thought that worry was a factor starting first attack in 1952. Two of his brothers died in infancy from bronchitis. Up to 7th December 1954 he smoked 3-4 cigarettes daily but after this date he stopped smoking. When he did smoke he found that it increased his dyspnoea. Draughts also increased his dyspnoea and since he draught-proofed all the doors in his house he suffered less dyspnoea. Heavy meals, coughing and exertion all increased his dyspnoea. Cold air and dampness increased his cough which occurred mainly in the morning.

Case No. 52. Age 60. Widow.

Occupation: She was a packer in a flour mill but retired in August 1955.

History: Her first attack of acute bronchitis occurred in 1935 and for the last 20 years she had attacks of bronchitis characterised by productive cough and dyspnoea on exertion at intermittent intervals both summer and winter. Her brother had pleurisy and chronic bronchitis and died at the age of 62 from pneumonia, Her sister had chronic bronchitis while her father died at the age of 46 from pneumonia. She lived in the ground flat of a very overcrowded tenement. She smoked 10 cigarettes daily. Excitement, fog, damp, cold weather, coughing and exertion all aggravated her dyspnoea. Changes of atmosphere, cold air, fog, exertion and flour dust at her previous employment all aggravated her cough. Her cough troubled her mainly in the morning and at night. She had hypertension.

Hospital Report: X-Rays of her chest and sinuses in May 1955 showed changes consistent with chronic bronchitis and chronic mucosal thickening of her left antrum.

Case No. 53. Age 64. Male.

Occupation: He was a porter in Leith Docks but prior to this he was a dock labourer.

History: In 1917 he suffered from mustard

gas poisoning and in 1925 he had his first attack of acute bronchitis. Over the last 30 years he developed chronic bronchitis with repeated attacks of bronchitis at first only in the winter months but in latter years his attacks had come on in the summer months as well as winter months. He found that if he worked with dust such as grain or seeds that he was particularly liable to bronchitis. He had no family history of chest illness. He did not smoke. Excitement, fog and exertion increased his breathlessness. Dust from cardboard boxes, grain or seeds and smoke from a cigarette both increased his breathlessness. His cough occurred mainly at night and was aggravated by fog, dust from cardboard boxes or grain or seeds and smoke from a cigarette. He had a deviation of nasal septum.

Hospital Reports: In 1942 an X-Ray of his chest revealed a chronic bronchitic process and X-Ray of his chest in January 1955 revealed chronic bronchitic changes in his chest while X-Ray of sinuses revealed mucosal thickening of his left antrum.

Case No. 54. Age 61. Male.

Occupation: He was a miller in flour mill from 1921 to 1933 but since 1933 he carried out first aid & civil-defence work in flour mills.

History: He had his first attack of acute bronchitis in 1949 and for the last 6 years he had

attacks of productive cough and dyspnoea at intermittent intervals only in the winter months. He had left basal pneumonia in June 1955. He had no family history of chest illness. He lived in the 3rd flat of an overcrowded and very high tenement and only had the rays of the sun entering his rooms for a limited time of day owing to the height of the opposite slum building and narrowness of the street between the tenements. He smoked 20 cigarettes daily. Frosty cold weather, fog, smoke, wet and dust all aggravated his cough while frosty cold weather, damp and exertion increased his dyspnoea. His cough was variable in frequency and was decreased in dry weather and warm atmospheres.

Hospital Reports: An X-Ray of his chest in November 1953 was negative while an X-Ray of sinuses at same time revealed slight mucosal thickening of both antra. An X-Ray of his chest in June 1955 after his pneumonia revealed an incompletely resolved inflammatory lesion at the left base of his chest. Another X-Ray of his chest in August 1955 showed some improvement of the left base but resolution was still incomplete.

Case No. 55. Age 59. Male.

Occupation: He was a safety-first worker but previous to this employment he was a labourer.

History: He suffered from rheumatic fever in 1917, 1919 and 1926. Since 1951, when he had first attack of acute bronchitis, he was subject to repeated attacks of bronchitis which occurred only in the winter months except for the summer of 1954, when his cough and dyspnoea affected him during the summer months also. His younger sister had chronic bronchitis and brother-in-law also suffered from chronic bronchitis. He smoked 20 cigarettes daily but was advised to reduce his cigarette daily number. Excitement, heavy meals, fog, cold weather, damp, coughing and exertion all increased his breathlessness. His cough occurred mainly in the morning and was aggravated by cold air, fog, exertion, smoke, wet and dust but diminished by dry weather and warmth.

Hospital Reports: He had a perforated peptic ulcer operation in 1926 and cholecystectomy performed in Leith Hospital in May 1955 following acute cholecystitis.

Case No. 56. Age 68. Male.

Occupation: Retired Shunter.

History: In 1924 he had his first attack of bronchitis and for the last 31 years he had attacks of bronchitis characterised by a productive cough at intermittent intervals mainly in the winter although occasionally in the summer. He had no history of

dyspnoea and no family history of chest illness. He smoked 2 ounces of tobacco a week but was advised to reduce his quantity of tobacco per week. His cough occurred mainly at night when he sat near the fire. Cold air, wet and an east wind aggravated his cough. Dust in the street and dust of the sulphur fumes from railway engines at his previous work aggravated his cough. He was under treatment for iron deficiency anaemia. He had bronchopneumonia in 1949.

Hospital Report: An X-Ray of his chest in July 1954 showed the changes of chronic bronchitis.

Case No. 57. Age at death in 1954 was 86. Female Married.

Occupation: Housewife.

Cause of Death: Cardiac Failure.

History: At the age of 47 she suffered from an acute bronchitis attack and for the last 39 years of her life she had repeated attacks of bronchitis in the winter only which started in 1915 when she came to Edinburgh after leaving England. Her attacks were characterised by productive cough and dyspnoea. She also suffered from acute cholecystitis in 1944 and 1954. Her mother had chronic bronchitis. She did not smoke. Draughts, fog, cold weather, damp, excitement, coughing and exertion all increased her breathlessness. Fog and wet increased her cough which was variable in frequency.

Case No. 58. Age at death in 1955 (17th January) was 75. Male.

Occupation: Retired fireman greaser in Merchant Navy.

Cause of Death : Carcinoma of the stomach.

History: Since 1914 he had been subject to attacks of bronchitis with cough and dyspnoea at intermittent intervals at first only in the winter months but for the last 6 years of his life the attacks persisted throughout summer and winter months. He had a right inguinal hernia operation in 1954 and developed carcinoma of the stomach from which he died in January 1955. He had no family history of chest illness. He lived in the ground flat of a very overcrowded tenement. He smoked 30 cigarettes a week. Draughts, fog, damp and exertion all increased his breathlessness. Fog, exertion and smoke all aggravated his cough which occurred mainly in the early morning, but his cough was decreased in dry weather and warmth.

Case No. 59. Age 81. Female. Married.

Occupation: Housewife.

History: In March 1954 she developed acute bronchitis and since then she nearly constantly suffered from a productive cough which was accompanied by dyspnoea and symptoms persisted throughout both summer and winter. In 1952 she was treated for iron

deficiency anaemia and she continued under treatment for this disability up to 1955. She was treated at Royal Infirmary and at home in 1955 for cholecystitis, aneurysm of the aorta, aortic stenosis and incompetence and cardiac failure. She had no family history of chest illness. She did not smoke. Fog, cold frosty weather, damp and exertion all increased her breathlessness. Her cough occurred mainly at night and was aggravated by fog, cold frosty weather, exertion, smoke of tobacco and wet but was decreased in dry weather and warmth.

Case No. 60. Age 89. Widow.

Occupation: Housewife.

History: In 1941 she had a right-sided hemiplegia and 5 years later in 1946 she had dysarthria and dysphagia. Since 1946, when she had her first attack of bronchitis, she had recurrent attacks of bronchitis in the winter only for the last 9 years. She had congestive heart failure in 1952. She had no family history of chest illness. She did not smoke. Fog, damp and exertion all increased her dyspnoea. Her cough was variable in frequency and she was uncertain as to the factors which aggravated her cough.

Case No. 61. Age 67. Female. Married.

Occupation: Housewife.

History: In 1950 she had influenza and following this she developed acute bronchitis and since 1950 she has had repeated attacks of bronchitis in the winter only and in March 1950 she had wheeziness of the chest. She had hypertensive cardiac failure in 1953 and myocardial infarction in 1954. She had no family history of chest illness. She lived in a 1st flat of a particularly overcrowded area. She smoked 3 cigarettes daily. Excitement, such as a noise at her back, fog, cold weather, coughing and exertion all increased her breathlessness. Cold air and exertion aggravated her cough which occurred mainly in the morning and at night. Dry weather and warmth decreased her cough.

Case No. 62. Age 43. Female. Single.

Occupation: Housewife.

History: She had whooping cough and measles as a child but had no respiratory complications following these illnesses. She came to Edinburgh from Aberdeen in 1935 and 10 years later in 1945 she developed her first attack of acute bronchitis. For the last 10 years she had repeated attacks of bronchitis accompanied by attacks of bronchial asthma which occurred in the winter only but for the last 3 years the attacks persisted with cough and dyspnoea throughout the summer as well. Her mother had chronic bronchitis. She did not smoke. Attacks of bronchial

asthma, fog, damp and exertion increased her breathlessness. Fog, dust, cold wind and changes of atmosphere aggravated her cough which was variable in frequency though often occurring in the morning. Warmth and dry weather both decreased her cough.

Hospital Report: An X-Ray of her chest in October 1954 showed moderate emphysema of both lungs but no active underlying lung disease.

Case No. 63: Age 67: Widow.

Occupation: Housewife.

History: In 1936 she had her first attack of bronchitis which followed coryza and since this year she had repeated attacks of bronchitis which always followed coryza in the winter except for 1954 when she had bronchitis in the summer also. Her attacks had not been so severe since leaving Shetland 8 years ago. She occasionally suffered from dyspnoea on going upstairs. She had a duodenal ulcer in 1936. Her sister suffered from bronchial asthma. She did not smoke. Severe exertion aggravated her breathlessness. Her cough occurred mainly in the morning and was aggravated by fog. She had herpes zoster of her left arm in January 1955.

Hospital Report: An X-Ray of her sinuses in July 1954 revealed slight mucosal thickening of both antra and an X-Ray of her chest at the same time

revealed widening of the aortic arch but no pulmonary lesion or cardiac enlargement.

Case No. 64. Age 53. Female. Married.

Occupation: Housewife.

History: Since the birth of her first child 30 years ago, when she had her first attack of bronchitis, she developed chronic bronchitis with recurrent attacks of bronchitis with cough and dyspnoea. These attacks came on only in the winter months. She has a family of five children and her eldest child who was a boy had bronchitis three times while her third child also a boy and youngest child who was a girl, both also had bronchitis. She lived in the 1st flat of a very overcrowded tenement. She smoked 7 cigarettes daily which she stated eased her breathlessness but she was to try and stop smoking altogether. Fog, cold weather, damp, coughing and exertion all aggravated her dyspnoea. Her cough troubled her mainly at night and in the morning and was aggravated by plugs of sputum, cold air, fog and exertion but was diminished by dry weather and warmth. In 1955 she suffered from hypertension. She had an infected upper canine tooth removed in July 1953 and suffered from Herpes Zoster in March 1952.

Case No. 65. Age 72. Male.

Occupation: Retired railway goods' worker.

History: At his work he was at the age of

20 a railway platelayer and he became a porter and signalman up to the age of 54 when he became a railway goods' porter up to his retirement in 1942. He had acute bronchitis in 1942 and had to leave the railway on account of his chest condition. At his previous work he was exposed, particularly during night shifts, to changeable and draughty weather. He himself told me that heavy work, draughty conditions at work and changeable weather at night shifts were responsible in his opinion for the onset of his chronic bronchitis. He was subject to frequent attacks of coryza from 1903 to 1942. Since 1942 he suffered from recurrent attacks of bronchitis with productive cough and dyspnoea and during the last 13 years also he had attacks of bronchial asthma. The attacks of bronchitis and bronchial asthma came on during both summer and winter months. His mother had chronic bronchitis for many years between the ages of 36 and 75. He lived in a rather overcrowded tenement. He stopped smoking 11 years ago. Attacks of bronchial spasm, heavy meals, damp and excitement all increased his breathlessness. Dust, present at his former employment in the form of sulphur fumes from engines, and warmth both increased his cough which occurred mainly at night.

Hospital Reports: An X-Ray of his chest in July 1954 showed some general increase in lung

markings consistent with chronic bronchitis. There was no cardiac enlargement but some dilatation of the ascending aorta. An X-Ray of his sinuses at the same time revealed a completely opaque right antrum with possible erosion of the lateral wall and the radiologist advised further investigation to exclude tumour. He was found to have a large number of polypi on both sides of the nose on further investigation and a radical antrostomy was performed followed by radium insertion into antrum and deep X-Ray therapy. A histological report of the biopsy performed showed only chronic sinusitis.

Case No. 66. Age 72. Male.

Occupation: Master mechanical engineer and electric welder.

History: He gradually developed chronic bronchitis which started as an acute attack of bronchitis 16 years ago. Since then he had repeated attacks of bronchitis which were always preceded by coryza and which occurred throughout summer and winter months. He experienced some dyspnoea on exertion for the last 3 years. He had no family history of chest illness. He did not smoke. For 50 years he dealt with ships' boiler repairs which were carried out inside hot boilers and in the winter particularly he came out of the boilers into the cold air outside. Draughts, exertion, coughing and changes of atmospheric

conditions all increased his breathlessness. His cough was constantly present when he had attacks of bronchitis and was aggravated by fog, draughts, changes of atmospheric conditions, severe exertion and smoke from tobacco fumes. His cough was diminished in dry weather and warmth. He had a haematemesis from a duodenal ulcer in February 1954 and a gastrectomy was performed in May 1954 which was unfortunately followed by phlebo-thrombosis of his legs. In January 1955 he had dilatation carried out for urethral stricture and in February 1955 he had physiotherapy for osteoarthritis of left hip joint.

Hospital Reports: An X-Ray of his chest in February 1954 showed bronchitic change with some fibrosis at the right base and X-Ray barium meal at the same time revealed a duodenal ulcer with diverticulosis of the descending colon.

Case No. 67. Age 40. Male.

Occupation: School Inspector.

History: He had an acute attack of bronchitis in 1945 and since then he had attacks of bronchitis characterised by cough sometimes productive but no dyspnoea for the last 10 years. These attacks of bronchitis were always preceded by coryza and only occurred in the winter months. He had no family history of chest illness. He smoked 10 cigarettes daily. Smoking, cold air, fog, smoky atmospheres

and wet all increased his cough which occurred mainly in the morning and was aggravated when he was confronted with sudden changes of atmospheric conditions. Dry weather and warmth both decreased his cough. In his work as a school inspector he travelled to various parts of Scotland and was confronted with varying temperatures and atmospheric conditions in doing his work.

Hospital Report: X-Rays of his chest and sinuses in February 1955 were negative.

Case No. 68 : Age 58. Male.

Occupation: He was a fisherman from 1919 to 1935 but from 1935 he was a brewers' worker and worked in brewery as an engineer's mate in an ammonia compressor room.

History: In 1946 he had left-sided pleurisy and he developed acute bronchitis. Since 1946 he had recurrent attacks of bronchitis characterised by cough sometimes dry and sometimes productive for the last 9 years. These attacks of bronchitis occurred in both summer and winter months. He had dyspnoea on exertion for the last 9 years. His second son had Pulmonary Tuberculosis. He lived in the 3rd flat of a tenement in a particularly overcrowded area. He smoked 10 cigarettes daily. Fog and exertion both increased his breathlessness. The exertion of rising in the morning aggravated his cough which occurred mainly in the morning except for acute exacerbation

in February 1955 when his cough troubled him at night and in the morning.

Hospital Report: An X-Ray of his chest in March 1955 showed low diaphragms consistent with emphysema and a little calcification of the aorta but otherwise no abnormality.

Case No. 69. Age 49. Female. Single.

Occupation: She was a shopworker from 1921 to 1932 and a packer in a ryvita factory from 1941 to 1950 otherwise she did housework.

History: She had attacks of bronchitis and bronchial asthma at intermittent intervals over the last 5 years which occurred throughout the year though attacks were more prevalent and severe in the winter months. She had attacks of hay fever in the summer months particularly after the neighbours cut their grass lawns. Her attacks of bronchitis were characterised by a cough which was productive and wheeziness of her chest and usually followed coryza and lasted for 3 weeks. She had no family history of chest illness. She lived in the 2nd flat of an overcrowded tenement. She did not smoke. Fog, heavy meals, damp feet, exertion and attacks of bronchial asthma all increased her breathlessness. Cold air, exertion, fog, excitement, smoke, wet and dust from the flour in the ryvita factory all aggravated her cough.

Hospital Report: An X-Ray of her chest in February 1955 was negative.

Case No. 70. Age 61. Widow.

Occupation: Cleaner.

History: In 1943 she had left-sided lobar pneumonia and in 1953 she developed acute bronchitis and she had attacks of bronchitis with productive cough but no dyspnoea in the winter months since 1953. Her mother had chronic bronchitis. She smoked 3-4 cigarettes a day. Cold air and exertion aggravated her cough which occurred mainly in the morning while dry weather and warmth decreased it.

Hospital Report: An X-Ray of her chest in February 1955 showed no abnormality.

Case No. 71: Age 44. Male.

Occupation: He was a labourer who worked with animal feeding stuffs.

History: After demobilisation from the Army in 1945 he developed acute bronchitis and suffered from recurrent attacks of bronchitis in the winter months mainly, except for 1955 when he had attacks also in the summer months for the past 10 years. One attack of bronchitis was particularly severe in November 1951. His youngest son had bronchitis at the age of 3 while his eldest son had bronchitis in 1955. He lived in the 2nd flat of an overcrowded tenement. He smoked 10 cigarettes daily. He had no history of

dyspnoea. Cold air at bedtime, fog, smoke and dust from the animal feeding stuffs at his work all aggravated his cough which occurred mainly at night. Dry weather and warmth both decreased his cough.

Hospital Reports: X-Rays of his chest in March and April 1955 revealed no evidence of intrapulmonary disease.

Case No. 72. Age 33. Female. Married.

Occupation: Tracer.

History: In January 1953 she developed a severe attack of acute bronchitis and for the last 3 years she had repeated attacks of bronchitis which came on at intermittent intervals throughout the summer and winter. These attacks were characterised by a cough which was sometimes productive and sometimes non-productive and also dyspnoea. She had a severe attack of bronchitis in January 1954. Her mother had chronic bronchitis. She smoked 5-15 cigarettes daily. Coughing and exertion both increased her breathlessness. Close atmospheres increased her cough which occurred mainly in the morning while dry weather and warmth decreased her cough. She was under treatment for iron deficiency anaemia from 1951.

Hospital Reports: X-Rays of her chest and sinuses in December 1953 were negative.

Case No. 73. Age 46. Female. Married.

Occupation: Canteen Worker.

History: In 1945 she had acute bronchitis and for the last 10 years she had recurrent attacks of bronchitis with productive cough and dyspnoea on exertion which occurred both in the summer and winter. She had no family history of chest illness. She lived in a tenement in a very overcrowded street. She smoked 6 cigarettes daily. Smoking, excitement, cold weather, damp, fog, coughing and exertion all increased her breathlessness. She was uncertain as to the factors causing exacerbations of her cough which occurred very often however after lunch when attacks of bronchitis came on. At her canteen work she had two gas fires at her back and central heating in the winter months and customers insisted on closed windows so that she came out from the hot and close atmosphere of the canteen to the cold air. She had a total hysterectomy with removal of the appendages in May 1955 and suffered from angioneuritic oedema and iron deficiency anaemia in 1954.

Hospital Report: An X-Ray of her chest in January 1953 was negative.

Case No. 74. Age 12. Male.

Occupation: Schoolboy.

History: At the age of 6 he had whooping cough and since this illness he had recurrent attacks

of bronchitis both in the summer and winter months. The cough was usually a dry cough and he had no history of dyspnoea. He had a very mild degree of bronchiectasis in his right mid zone in January and February 1952 and sinusitis in the same year. His mother had suffered from chronic bronchitis for the last 2 years. He lived in the ground flat of a very overcrowded tenement. Fog, exertion and smoke aggravated his cough which occurred mainly at night when he had attacks of bronchitis, while dry weather and warmth decreased his cough.

Hospital Reports: An X-Ray of his sinuses in February 1952 showed diminution in normal translucency and on the left side more apparent changes compatible with some degree of infection. X-Rays of his chest in July 1952 and October 1953 and January 1955 were negative though X-Ray of his sinuses in January 1955 revealed an opaque left antrum.

Case No. 75. Age 27. Male.

Occupation: Foundry Worker,

History: Previous to his employment as a foundry worker he served with the Royal Navy for 5 years in which he was in submarine service. Since leaving the Royal Navy and entering his present employment in 1953 he had recurrent attacks of bronchitis at repeated intervals with attacks of 3 weeks duration for a whole year in 1953 and 1954 with

recurrences of productive cough in 1955. Attacks of bronchitis persisted throughout summer and winter months. He also suffered from dyspnoea on exertion. He had no family history of chest illness and had no previous history of chest illness. He lived in an overcrowded tenement whose walls were damp and this tenement was situated in an area which was very susceptible to fog and mist from the sea. He smoked 20 cigarettes daily but was advised to stop smoking altogether. Exertion of lifting objects or running aggravated his breathlessness. Smoke from cigarette fumes and dust from moulder's sand at the foundry aggravated his cough which was variable in its frequency.

Hospital Report: X-Rays of his chest and sinuses in July 1954 were negative.

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TABLE I. Length of History of Bronchitis and Seasonal Incidence of Attacks of Bronchitis.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12
Length of History of Bronchitis in years.	18	3	4	15	30	5	12	5	5	11	25	2
Season when attacks of Bronchitis.	S & W	S & W	S & W	W 1940-51 W & S from 1952.	S only 1925-38 W & S from 1939.	W & April	Mainly W.	S & W	W	S & W	S & W	S & W

Case No.	13	14	15	16	17	18	19	20	21
Length of History of Bronchitis in years.	2	15	21	44	39	2	2	12	62
Season when attacks of Bronchitis.	S & W	W only 1940-53 Spring & W 1954 S & W 1955.	W only 1933-50 W & S 1951-54	W	W only 1916-33 W & S from 1934	S & W	W	S & W	W & S 1892-0 W only 1905- W & S 1954.

Case No.	22	23	24	25	26	27
Length of History of Bronchitis in years.	4	3	23	39	8	6
Season when attacks of Bronchitis.	W only 1952-54 S & W 1955.	W only 1953 & 1955. W & S 1954	W	W only 1916-33 W & S from 1934	W only 1947-53 and 1955. W & S 1954.	W only 1949-53 and 1955. W & S 1954.

W = Winter. S = Summer.

BLE I. (Contd) Length of History of Bronchitis and Seasonal Incidence of Attacks of Bronchitis.

se No.	28	29	30	31	32	33	34	35	36	37	38	39
ngth of History Bronchitis in ars.	25	27	9	4	4	22	15	22	2	23	4	3
ason when Attacks of Bronitis.	W only 1930-52 W & S 1953-55	W	W	W	S & W	W only 1933-54 W & S 1955	W	W	S & W	S & W	W only 1951 W & S from 1952.	W only 1952-1953 W & S 1954.

se No.	40	41	42	43	44	45	46	47	48	49
ngth of History Bronchitis in ars.	3	3	6	5	5	38	28	7	31	15
ason when Attacks of Bronitis.	W	Autumn & Spring 1952-1953 W & S from 1954	W only 1949-53 W & S 1954-55.	S & W	W	S & W	S & W	W only 1948-52 W & S from 1953	W only 1924-47 W & S 1948-55	W

ase No.	50	51	52	53	54	55	56	57	58	59	60	61
ength of History Bronchitis in ears.	2	4	20	30	6	3	31	39	40	1	9	6
ason when Attacks of Bronitis.	W	S & W	S & W	W only but in recent years S & W	W	W only 1951-53 W & S 1954.	W mainly S occasionally	W	W only 1914-49 W & S 1950-54	S & W	W	W

W = Winter. S = Summer.

TABLE I. (Contd.) Length of History of Bronchitis and Seasonal Incidence of Attacks of Bronchitis.

Case No.	62	63	64	65	66	67	68	69	70	71	72
Length of History of Bronchitis in years.	10	18	30	13	16	10	9	5	2	10	3
Season when Attacks of Bronchitis.	Mainly W & S	W only from 1945-52	W & S 1936-1953 1954.	W	S & W	S & W	W	S & W	S & W	W only W & S 1945-54 1955.	S & W

Case No.	73	74	75
Length of History of Bronchitis in years.	10	6	3
Season when Attacks of Bronchitis.	S & W	S & W	S & W

W = Winter. S = Summer.

TABLE II. Smoking - Effect on Dyspnoea.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Effect on Dyspnoea	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Non-Smokers	*	*	*	*	*		*		*	*		*	*	*	
Case No.	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
Effect on Dyspnoea	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-
Non-Smokers	*	*	*		*	*		*				*		*	
Case No.	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45
Effect on Dyspnoea	N.D.	N.D.	-	-	-	-	+	-	-	-	+	-	-	-	-
Non-Smokers	*	*	*		*				*	*		*			
Case No.	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60
Effect on Dyspnoea	-	-	-	+	-	-	-	-	-	-	N.D.	-	-	-	-
Non-Smokers	*				*	*		*				*		*	*
Case No.	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75
Effect on Dyspnoea	-	-	-	E	-	-	N.D.	-	-	N.D.	N.D.	-	+	N.D.	-
Non-Smokers		*	*		*	*			*					*	

\+ = Increase of Dyspnoea. - = No Effect on Dyspnoea. N.D. = No Dyspnoea.
* = Does not Smoke. E = Eased Dyspnoea.

TABLE III. EXCITEMENT - Effect on Dyspnoea.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Effect on Dyspnoea	+	-	+	+	+	-	+	-	-	+	-	-	-	+	+	-	-	+
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37
Effect on Dyspnoea	-	-	-	-	-	-	+	-	+	-	-	N.D.	N.D.	+	+	-	+	-
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56
Effect on Dyspnoea	+	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	+	N.D.
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75
Effect on Dyspnoea	-	-	-	+	-	-	-	+	-	N.D.	-	-	N.D.	N.D.	-	+	N.D.	-

TABLE IV. Heavy Meals - Effect on Dyspnoea.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Effect on Dyspnoea	-	-	+	+	+	-	+	-	-	-	+	-	-	-	+	-	+	-
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37
Effect on Dyspnoea	-	-	-	-	-	-	+	+	-	-	-	N.D.	N.D.	+	-	-	+	-
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56
Effect on Dyspnoea	-	-	+	-	-	+	-	+	-	-	+	-	+	-	-	-	+	N.D.
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75
Effect on Dyspnoea	-	-	-	-	-	-	-	+	-	N.D.	-	+	N.D.	N.D.	-	-	N.D.	-

\+ = Increase of Dyspnoea. - = No Effect on Dyspnoea. N.D. = No Dyspnoea.

TABLE V. Coughing - Effect on Dyspnoea

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Dyspnoea	+	-	+	-	+	+	+	-	-	-	-	-	-	+	+	-	+	-	+
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Dyspnoea	-	+	-	+	+	-	+	+	+	-	-	N.D.	N.D.	+	+	-	+	+	-
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Dyspnoea	-	+	-	+	-	+	-	+	-	-	+	-	+	+	-	-	+	N.D.	+
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Dyspnoea	-	-	-	+	-	-	+	-	+	N.D.	-	-	N.D.	N.D.	+	+	N.D.	-	

TABLE VI. Exertion - Effect on Dyspnoea.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Dyspnoea	+	+	+	+	+	+	+	-	+	+	-	+	+	+	+	+	+	-	+
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Dyspnoea	-	+	+	+	+	+	+	+	+	+	-	N.D.	N.D.	+	+	-	+	+	+
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Dyspnoea	+	-	-	-	+	+	+	+	+	+	+	+	+	+	+	+	+	N.D.	+
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Dyspnoea	+	+	+	+	+	+	+	-	+	N.D.	+	+	N.D.	N.D.	+	+	N.D.	+	

+ = Increase of Dyspnoea. - = No Effect of Dyspnoea. N.D. = No Dyspnoea.

TABLE VII. Exertion - Effect on Cough.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Cough	-	+	+	+	+	+	+	-	+	+	-	-	+	+	+	-	+	-	+
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Cough	-	+	-	+	-	-	+	-	+	-	-	-	-	+	-	-	+	+	+
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Cough	-	+	-	-	+	+	+	+	-	-	+	+	-	+	-	-	+	-	-
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Cough	+	+	-	+	-	-	+	-	-	-	+	+	+	-	-	-	+	-	

TABLE VIII. Dust - Effect on Cough.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Cough	-	+	+	+	-	+	-	-	-	+	+	-	+	-	+	+	+	-	-
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Cough	-	-	-	+	-	-	+	-	-	-	-	-	-	+	+	-	+	+	+
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Cough	+	+	+	+	+	-	+	+	-	-	+	-	-	+	+	+	+	+	-
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Cough	-	-	-	-	+	-	-	+	-	-	-	+	-	+	-	-	-	+	

\+ = Increase of Cough. - = No Effect on Cough.

TABLE IX. Draughts - Effect on Dyspnoea.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Dyspnoea	-	-	-	-	+	-	-	-	-	+	-	-	+	+	-	-	-	-	-
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Dyspnoea	-	-	+	-	-	-	-	-	+	-	-	N.D.	N.D.	+	-	-	-	-	+
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Dyspnoea	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	N.D.	+
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Dyspnoea	+	-	-	-	-	-	-	-	+	N.D.	-	-	N.D.	N.D.	-	-	N.D.	-	

TABLE X. Fog - Effect on Dyspnoea.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Dyspnoea	+	+	+	+	+	+	+	-	+	+	-	+	+	+	+	-	-	-	+
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Dyspnoea	-	-	-	+	+	-	+	-	+	-	-	N.D.	N.D.	+	-	+	+	+	+
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Dyspnoea	+	-	+	+	-	-	+	+	-	+	+	-	-	+	+	-	+	N.D.	+
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Dyspnoea	+	+	+	+	+	-	+	-	-	N.D.	+	+	N.D.	N.D.	-	+	N.D.	-	

\+ = Increase of Dyspnoea. - = No Effect on Dyspnoea. N.D. = No Dyspnoea.

TABLE XI. Cold Weather - Effect on Dyspnoea.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Dyspnoea	-	-	-	+	+	-	-	-	-	-	-	-	-	+	+	-	+	-	+
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Dyspnoea	-	-	+	-	-	-	+	+	-	-	-	N.D.	N.D.	-	-	-	-	+	+
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Dyspnoea	-	+	-	+	-	+	-	-	-	+	-	-	-	+	-	+	+	N.D.	+
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Dyspnoea	-	+	-	+	-	-	+	-	+	N.D.	-	-	N.D.	N.D.	-	+	N.D.	-	

TABLE XII. Damp - Effect on Dyspnoea.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Dyspnoea	+	-	+	-	+	-	+	+	+	+	-	-	+	+	+	-	-	-	-
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Dyspnoea	-	-	-	-	+	-	+	+	+	-	-	N.D.	N.D.	+	-	-	+	+	+
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Dyspnoea	-	+	+	-	-	-	+	-	-	+	+	-	-	+	-	+	+	N.D.	+
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Dyspnoea	+	+	+	-	+	-	+	+	-	N.D.	-	-	N.D.	N.D.	-	+	N.D.	-	

\+ = Increase of Dyspnoea. - = No Effect on Dyspnoea. N.D. = No Dyspnoea.

TABLE XIII. Cold Air - Effect on Cough.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Cough	+	+	-	+	+	-	-	-	-	-	-	-	-	+	+	-	+	-	-
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Cough	-	+	-	-	-	-	+	-	-	-	-	+	-	+	-	-	-	+	+
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Cough	+	+	D	+	-	+	+	+	+	+	+	-	+	+	-	+	+	+	-
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Cough	-	+	-	+	-	-	+	-	+	+	-	+	+	+	-	-	-	-	

TABLE XIV. Fog - Effect on Cough.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Cough	+	+	+	+	+	+	-	-	+	+	-	-	+	+	+	-	-	-	+
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Cough	-	-	-	-	+	-	+	-	+	+	-	-	-	+	-	-	-	+	+
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Cough	+	+	+	+	-	+	-	-	+	+	+	+	-	+	+	+	+	-	+
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Cough	+	+	-	-	+	+	+	-	+	+	-	+	-	+	-	-	+	-	

\+ = Increase of Cough. - = No Effect on Cough. D = Decrease of Cough.

TABLE XV. Smoke - Effect on Cough.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Cough	-	-	+	+	-	-	-	-	-	+	-	+	+	+	-	+	-	+	+
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Cough	-	+	-	+	-	-	-	+	+	+	-	-	-	+	-	-	+	+	-
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Cough	+	+	-	-	+	+	-	+	-	-	-	+	-	-	+	+	+	-	-
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Cough	+	+	-	-	-	-	-	-	+	+	-	+	-	+	-	-	+	+	

TABLE XVI. Wet - Effect on Cough.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Cough	+	+	+	-	+	-	-	-	+	+	-	-	-	+	-	-	-	-	-
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Cough	-	-	-	-	-	-	+	+	+	-	-	-	-	+	-	-	-	-	-
Case No.	39	40	41	42	43	44	45	46	47	48	49	59	51	52	53	54	55	56	57
Effect on Cough	-	+	-	+	-	-	+	+	-	+	+	-	+	-	-	+	+	+	+
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Cough	-	+	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	

\+ = Increase of Cough. - = No Effect on Cough.

TABLE XVII. Dry Weather - Effect on Cough.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Cough	-	D	D	-	-	-	-	-	D	-	-	-	D	D	D	D	-	-	D
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Cough	-	D	-	-	D	-	D	-	-	-	-	-	-	-	D	-	-	D	D
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Cough	-	D	D	-	-	D	-	D	D	D	D	D	-	-	-	D	D	-	-
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Cough	D	D	-	D	D	-	D	-	D	D	-	-	D	D	D	-	D	-	

TABLE XVIII. Warmth - Effect on Cough.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Effect on Cough	-	-	D	-	-	D	D	-	D	-	-	-	D	D	D	D	-	I	D
Case No.	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38
Effect on Cough	-	D	-	-	D	-	D	-	-	D	-	-	-	-	D	-	-	D	D
Case No.	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57
Effect on Cough	-	D	D	-	I	D	-	D	D	D	D	D	-	-	-	D	D	-	-
Case No.	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	
Effect on Cough	D	D	-	D	D	-	D	I	D	D	-	-	D	D	D	-	D	-	

D = Diminution of Cough. - = No Effect on Cough. I - Increase of Cough.

TABLE XIX

Sputum Measurements.

Date Month February	Climate	Case No. 4. 24 Hr. Sputum ccs.	24 Hr. Froth ccs.	Therapy	Case No. 3. 24 hr. Sputum ccs.	24 hr froth ccs.	Therapy.
	Mild & Dry						
1	Fog & Rain night	30	5	Ephedrine in the	6	2	
2	Mild & Damp	35	5	morning + Amino-	6	2	Throughout
3	Dry & Cold	35	5	phylline in the	6	2	month
4	Cold & Foggy	50	10	morning + Codeine	6	2	on
5	Cold & Damp	50	10	Linctus at Bed-	6	2	Ephedrine
6	Cold & Dry	100	10	time. Changed	6	2	in
7	Snow & Cold	70	10	on 5.2.55 to	6	2	morning
8	Cold snow & rain	50	5	Ephedrine Tid. and	6	2	(½ grain)
9	Cold & Dry sun	80	10	Aminophylline Bid.	5	1	+
10	Cold & Dry	50	10	and Penicillin daily	5	1	Aminophylline
11	Dry and snow later	50	10	Injections from	6	2	0.1 Gram Bid.
12	Snow & Sleet	50	10	7.2.55 to 10.2.55.	6	2	and 0.2 Gram
13	Cold & Dry	70	10		6	2	at Bedtime
14	Cold & Damp	80	5		5	2	+
15	Cold & Dry	70	5		5	2	Codeine Linctus
16	Cold & Dry	80	10		5	2	at
17	Snow & Cold	90	5	Started on 17.2.55	5	2	Bedtime
18	Snow & Cold	80	10	Sulphatriad and on	5	2	+
19	Snow & Frost	60	5	18.2.55 Dihyro-	6	2	Sodium Amytal.
20	Snow & Frost	40	5	streptomycin	6	2	
21	Snow & Frost				6	2	
22	Snow & Frost	Patient became very			5	2	
23	Snow & Frost	ill and died on 22.2.55.			5	2	
24	Snow & Frost	No sputum measurements			5	2	
25	Snow & Frost	after 20.2.55.			6	2	
26	Snow & Frost				5	2	
27	Snow & Frost				5	2	
28	Rain cold wind				6	2	

TABLE XIX. (Continued). Sputum Measurements.

Date 1955 Month February	Climate	Case No. 2. 24 hr. Sputum ccs.	24 hr. Froth ccs.	Therapy	Case No. 17. 24 hr. Sputum ccs.	24 hr. Froth ccs.	Therapy
1	Mild & Dry Fog & Rain night	30	10	Amesec 1 Capsule	50	10	Ephedrine
2	Mild & Damp	35	10	Tid. +	50	7	½ grain Tid.
3	Dry & Cold	35	10	Neo-Epinine Com-	58	5	Neo-Epinine Com-
4	Cold & Foggy	20	8	pound Solution No.2	57	5	pound Solution
5	Cold & Damp	70	10	Inhalations as re-	50	15	No. 2 as required
6	Cold & Dry	30	6	quired and Lobel-	50	10	throughout month.
7	Snow & Cold	40	5	iae mixture Tid.	55	7	On 16.2.55 started
8	Cold Snow & Rain	35	5	+ Mist. Brompton	58	8	on
9	Cold & Dry Sun	40	5	at Bedtime.	50	7	Aminophylline
10	Cold & Dry	45	5	Penicillin daily	54	6	0.1 Gram Bid. and
11	Dry & Snow later	50	20	Injections from	50	8	0.2 Gram at Bed-
12	Snow & Sleet	50	10	31.1.55 to 3.2.55.	50	4	time.
13	Cold & Dry	50	20	Stopped Lobeliae	55	5	Penicillin daily
14	Cold & Damp	60	13	Mixture on 13.2.55.	Owing to se-		from 16.2.55. to
15	Cold & Dry	40	10		vere Dyspnoea		22.2.55.
16	Cold & Dry	60	10		unable to mea-		
					sure sputum		
17	Snow & Cold	70	12		60	10	
18	Snow & Cold	40	5		55	10	
19	Snow & Frost	50	30		57	5	
20	Snow & Frost	60	13		50	7	
21	Snow & Frost	50	5		50	5	
22	Snow & Frost	50	10		48	7	
23	Snow & Frost	45	15		42	12	
24	Snow & Frost	50	10		45	12	
25	Snow & Frost	40	10		43	7	
26	Snow & Frost	50	20		47	6	
27	Snow & Frost	40	5		47	7	
28	Rain & Cold Wind	35	15		45	9	

1955 TABLE XIX. (Continued). Sputum Measurements.

Date	Climate	Case No. 3			Case No. 2.			Case No. 17.		
Month March		24 Hr Sputum ccs,	24 Hr Froth ccs,	Therapy	24 Hr Sputum ccs.	24 Hr Froth ccs.	Therapy	24 Hr Sputum ccs.	24 Hr Froth ccs.	Therapy
1	Rain & Wind	6	2	As February	30	10	As February	48	7	As February but stopped Aminophylline Sulphaphthalidine for Diverticulitis of Sigmoid Colon.
2	Dry sun but fog night	6	2	But changed from Sodium Amytal to Soneryl on 19.3.55.	40	20	started Penicillin Inhalations BID. + Postural coughing on 11.3.55 and 15.3.55 respectively. Total 750,000 I.Us. Penicillin.	47	8	
3	Cold & Fog	5	2		20	20		47	9	
4	Dry & Cold	6	2		30	5		48	5	
5	Cold & Showery	6	2		30	5		47	5	
6	Cold & Damp	6	2		30	20		48	6	
9	Wet & Snow	5	2		40	10		46	7	
8	Damp & Fog	6	2		50	10		47	5	Started Penicillin Inhalations on 24.3.55.
9	Dry & Sun	6	2		50	5		47	5	
10	Dry sun but fog night	6	2		60	5		45	7	
11	As 10, severe night.	5	2		55	5		45	8	
12	Fog 11-12th	5	2		35	5		46	4	
13	Dry & Sun	6	2		40	10		47	-	
14	Dry & Sun	6	2		35	15		45	5	
15	Cold & Dry	5	2		25	10		45	5	
16	Cold & Showery	5	2		30	20		47	6	
17	Dry Sun Cold	5	2		42	10		46	7	
18	Snow sleet rain	6	2		25	50		47	5	
19	Cold & Dry	Patient died on 21.3.55 suddenly. No sputum measurements between 18.2.55 and 21.3.55.			15	5		47	5	
20	Cold & Snowy				10	5		46	7	
21	Cold & Snowy				15	3		45	6	
22	Cold & Rain				5	-		47	6	
23	Cold & Snow				10	2		46	5	
24	Fog & Cold &				5	3		53	10	
25	Damp 24 & 25				20	5		60	20	

1955 TABLE XIX. (Continued). Sputum Measurements.

Date Month March	Climate	Case No. 2. 24 Hr Sputum ccs.	24 Hr Froth ccs.	Therapy	Case No. 17 24 Hr Sputum ccs.	24 Hr Froth ccs.	Therapy.
26	Cold & Dull	30	10	Restarted	60	15	Finished Penicillin
27	Fine & Dry	30	10	Penicillin	65	5	Inhalations on 1.4.55.
28	Cold & Dry	30	8	Inhalations	45	15	Total Penicillin
29	As 28 & Sun	40	10	on 31.3.55.	55	-	600,000 I.Us.
30	As 29	55	10	Total Peni-	45	13	
31	As 30	70	10	cillin	55	10	
April							
1	Dry & Sunny	60	18	Stopped	49	10	
2	Cold & Dry	45	10	Penicillin	45	5	
3	Dry & Sunny	40	15	Inhalations	45	5	
4	Dull & Cloudy	20	10	on 3.4.55.	40	7	
5	Mild & Dry	30	10		45	7	
6	Dry & Sunny	20	10		42	7	
7	Damp & Frost	50	12		41	8	
8	Showers	50	5		40	7	
9	Dry & Sunny	70	10		40	6	
10	Mild	50	5		42	7	
11	Dry	60	10		42	6	
12	Cold & Dry	60	5		43	8	No sputum readings on 16th
13	Showers	42	18	On 14.4.55	43	6	or 17th April.
14	Warm & Dry	40	3	getting out	42	7	
15	Sunny	30	2	walks + Helio-	42	7	
16	Warm	20	2	therapy.	No Reading.		

1955 TABLE XIX. (Continued) Sputum Measurements.

Date Month April	Climate	Case No. 2. 24 Hr Sputum ccs.	24 Hr. Froth ccs.	Therapy	Case No. 17 24 Hr Sputum ccs.	24 Hr Froth ccs.	Therapy
17	Warm	15	3		No Reading		Started on 18.4.55.
18	Warm	20	2		43	6	
19	Fine & Warm	15	2		44	7	Lobeliae Mixture Tid. +
20	Fine & Warm	15	3		44	7	
21	Cold & Dry	10	-		42	10	Mist. Brompton at Bedtime +
22	Cold & Dry	10	1		50	10	
23	Cold & Damp	10	2	Reduced on	49	7	getting out to
24	Warm	15	-	25.4.55 Amesec	45	7	garden for Helio-
25	Dull & Variable	10	-	to 1 Capsule	45	6	therapy.
26	Variable	10	1	only if re-	43	6	
27	Rain & Cold	8	-	quired and Neo-	42	5	
28	Warm & Dry	10	-	Epinine Com-	42	7	
29	Dry & Sunny	8	-	pound Solution	40	4	
30	Cool & Windy	10	-	No. 2 as re-	42	5	
				quired only.			
May							
1	Showers	10	2		42	6	
2	Cool & Dull	8	3		45	5	
3	Showers	13	2		40	6	
4	High Wind	Discontinued for Helio-therapy.			42	8	

1955 TABLE XIX. (Continued) Sputum Measurements.

Date Month May	Climate	Case No. 17. 24 Hr. Sputum ccs.	24 Hr. Froth ccs.	Therapy.
5	Showers	38	5	Barium Enema Examination showed
6	Showers	36	7	Obstructive Lesion just distal
7	Fine	38	5	to the Pelvi-Rectal junction
8	Showers	38	6	from which Neoplasm cannot be
9	Rain	39	7	ruled out.
10	Showers	41	6	
11	Fine	40	6	
12	Wet	39	7	
13	Showers	40	6	
14	Showers & Cold	39	5	
15	Cold	39	6	Admitted Leith Hospital for
16	Showers & Cold	40	6	Sigmoidoscopy so Readings discon-
17	Cold & Wet	39	5	tinued.
18	Cold	37	6	
19	Showers	36	5	
20	Showers	34	6	
21	Showers	32	5	
22	Showers	31	6	
23	Fine	34	6	
24	Fine	34	5	
25	Fine	32	6	
26	Fine	28	7	
27	Fine	27	8	

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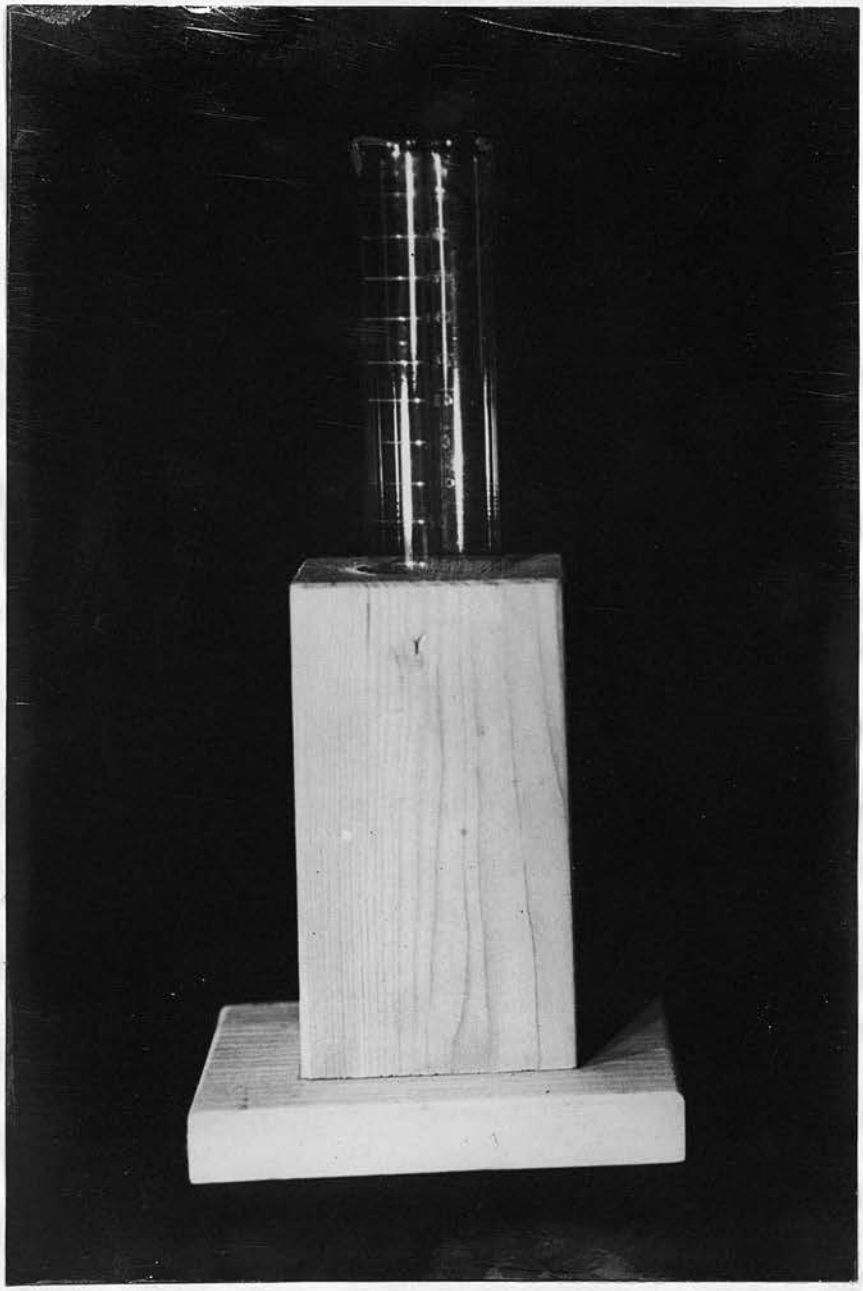
EDINBURGH - A CLEAR DAY.



CALIBRATED TUBE FOR SPUTUM MEASUREMENT
IN WOODEN BLOCK.

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