*Brit. J. industr. Med.*, 1971, **28**, 211-216.

# Insulation workers in Belfast[1]. 1. Comparison of a random sample with a control population

WILLIAM F. M. WALLACE and JEAN H. M. LANGLANDS
Department of Physiology, Medical Biology Centre, Lisburn Road, Belfast and
Respiratory Investigation Unit, Belfast City Hospital, Lisburn Road, Belfast

**Wallace, W. F. M., and Langlands, J. H. M. (1971).** *Brit. J. industr. Med.*, **28**, 211-216.
**Insulation workers in Belfast. 1. Comparison of a random sample with a control population.**
A sample of 50 men was chosen at random from the population of asbestos insulators in Belfast and matched with a control series of men of similar occupational group with respect to age, height, and smoking habit.

Significantly more of the insulators complained of cough and sputum and had basal rales on examination. Clubbing was assessed by means of measurements of the hyponychial angle of both index fingers. These angles were significantly greater in the group of insulators.

Twenty-one insulators had *x*-rays which showed pleural calcification with or without pulmonary fibrosis; one control *x*-ray showed pulmonary fibrosis.

The insulators had no evidence of airways obstruction but static lung volume was reduced and their arterial oxygen tension was lower than that of the controls and their alveolar-arterial oxygen gradient was greater.

There is a considerable body of evidence that exposure to asbestos may lead to symptoms and signs of lung disease (Merewether, 1933-4; Wyers, 1949; Williams and Hugh-Jones, 1960b; Leathart, 1960; Bader, Bader, Tierstein, and Selikoff, 1965; Hunt, 1965; Thomson, Pelzer, and Smither, 1965). However, other factors to which asbestos workers are exposed – cigarette smoking and atmospheric pollution – have been shown to play an important part in impairing lung function. For example, Fletcher, Elmes, Fairbairn, and Wood (1959) surveyed London postmen and found that about one-third of them complained of mild exertional dyspnoea and between one-third and one-half gave a history of cough and sputum for at least three months in the year. Thus it is likely that some of the disability found in asbestos workers is unrelated to exposure to asbestos.

[1]This investigation was carried out in the Department of Therapeutics, Institute of Clinical Science, Grosvenor Road, Belfast 12.

In order to avoid overestimating the harmful effects of asbestos, comparison with a control population is desirable and the controls should be matched as far as possible with the asbestos workers, not only for such factors as age and height, but also for environment and smoking habit. The use only of controls who denied all respiratory symptoms would be quite unsatisfactory.

In the present survey we have investigated in detail 50 men selected at random from a population of insulation workers and have compared them with a control series who were closely matched for age, height, smoking habits, and general occupational group but who had minimal exposure to asbestos.

## Methods

### Selection of workers

The names and addresses of all men working as insulators in Northern Ireland were obtained from their union (membership of which is a condition for employment). The names were arranged alphabetically and numbered.

Selection was made using random number tables. Men who lived outside a radius of about 20 miles from the centre of Belfast were excluded. The home of each man selected was visited and an appointment arranged. Those who failed to attend were revisited and a further effort was made to encourage attendance. In order to examine 50 insulators, 53 men were selected. Of the three who were selected but not examined, one had suffered severe chest injury due to a fall at work and two refused to attend. Thus the 50 men examined represented 94% of those selected.

**Selection of controls**

Firstly, the names of several men whose date of birth was within a year of that of the insulator to be matched were obtained from lists of Belfast Corporation employees. These men were then interviewed in turn until one was found who matched the insulator in height to within 3 in (7·6 cm) and in smoking habit (Medical Research Council, 1960). The men selected were then asked if they would take part in the survey, the purpose of which was explained to them; only two refused and had to be replaced. Forty-one of the controls worked in the local transport maintenance department, six in the gas department (not in the gas works itself), one was a plumber, and two were student laboratory technicians. It was felt that these men had a very similar working and living environment to the insulators apart from exposure to asbestos.

**Investigations**

The following investigations were undertaken:

**History and clinical examination** The Medical Research Council long respiratory questionnaire (1960) was used. Rhonchi and rales were sought and rales, if present, were graded 1 if confined to the axilla, and 2 if present elsewhere. Blood pressure was measured and the heart auscultated.

**Clubbing** Impressions were made of the distal one and a half phalanges of the two index fingers of each man and, from these, casts of the digits were made. Clubbing was assessed by measuring the hyponychial angle on each cast. The methods used were those described by Regan, Tagg, and Thomson (1967).

**Asbestos bodies** An early morning specimen of sputum was examined for asbestos bodies by counting under ×240 magnification the number of bodies in an area of 1 $cm^2$ in two specimens, one unstained and one stained with methyl green and pyronine to show the cellular content. The sputum was positive for asbestos bodies if one or more definite bodies were seen in either specimen.

**Chest *x*-ray** A postero-anterior film was taken in full inspiration. It was immediately checked for quality by a radiologist and, if necessary, repeated. Control subjects who had been *x*-rayed (mass radiography) in the previous six months did not have a further *x*-ray. In this way there were 31 100mm films and 19 standard chest *x*-rays for the 50 controls. Films were read by two experts in chest *x*-rays and classified under the headings given in Table 5. Because not all the control *x*-rays were full-size films, no attempt was made to use the I.L.O. classification which was used in examining the *x*-rays of the entire group of insulation workers (Langlands, Wallace, and Simpson, 1971).

The use of the 100mm films was not ideal but it was felt that to take a further chest *x*-ray of men who had recently had an *x*-ray which was considered normal could not be justified.

**Respiratory function tests** Forced expiratory volume in one second ($FEV_{1.0}$) and forced vital capacity (FVC) were recorded on a Poulton Gaensler-type spirometer (McKerrow, McDermott, and Gilson, 1960). Peak flow rate (PFR) was measured on a Wright peak flow meter (Wright and McKerrow, 1959). In these tests, the mean of the last three of five attempts was recorded. Arterial oxygen and carbon dioxide tensions ($Po_2$ and $Pco_2$) and pH were estimated by Astrup apparatus on blood obtained by puncture of the warmed ear lobe (Langlands and Wallace, 1965). Expired air was collected in a Tissot spirometer over a period of 2 minutes after washout and was analysed for oxygen using a Servomex oxygen analyser and for carbon dioxide using a modified Haldane apparatus (Campbell, 1960). Alveolar $Po_2$ was calculated using the formula given by Comroe and his colleagues (1962) at page 353, substituting oxygen for carbon dioxide, as they suggest.

Subdivisions of total lung capacity and transfer factors (Tl) for carbon monoxide in air and in oxygen were measured using the equipment described by Meade and his colleagues (1965). From the results, the diffusing capacity of the pulmonary membrane (Dm) and pulmonary capillary blood volume (Vc) were calculated by computer, using formulae given by Cotes (1965). Single breath alveolar volume was used to calculate Tl. The transfer constant (or permeability coefficient) Kl was obtained by dividing Tl by the single breath alveolar volume.

## Results

**Exposure to asbestos**

The duration of exposure to asbestos of the insulators varied from 4 to 48 years (mean ±S.D. 16·1 ± 7·5 years). Details are given in Table 1. Of the control subjects, 12 said they occasionally cut asbestos sheeting at their work and one had previously worked as an insulator for three years.

TABLE 1

EXPOSURE OF INSULATION WORKERS TO ASBESTOS

| *Years of exposure* | *No. of men* |
|---|---|
| 0-9 | 5 |
| 10-19 | 32 |
| 20-29 | 11 |
| 30-39 | 1 |
| 40-49 | 1 |
| Total | 50 |

**Adequacy of matching**

Mean differences between insulators and controls with respect to age, height, surface area, and smoking habit are shown in Table 2. The control subjects were on average 0·4 years older and 2 cm taller than the insulators. Smoking habits were similar. From prediction formulae based on age and height for lung function tests (Cotes, 1965) the controls would be expected to have a slight advantage in tests related to lung volume.

TABLE 2

PHYSICAL CHARACTERISTICS AND SMOKING HABITS

| | *Insulators* | *Controls* | *Difference* |
|---|---|---|---|
| Mean age (yr) | 39·4 | 39·8 | −0·4 |
| Mean height (cm) | 169 | 171 | −2 |
| Mean surface area ($m^2$) | 1·81 | 1·84 | −0·03 |
| Non-smokers | 13 | 11 | 2 |
| Ex-smokers | 9 | 12 | −3 |
| Smokers – grade 1 0-14 cigs./day | 5 | 6 | −1 |
| Smokers – grade 2 (15-24 cigs./day) | 13 | 12 | 1 |
| Smokers – grade 3 (25+ cigs./day | 10 | 9 | 1 |

**Symptoms and signs of chest disease**

These are shown in Table 3. More insulators than controls complained of dyspnoea, cough, and sputum; the difference was statistically significant for cough and sputum ($P < 0·01$) and almost so for dyspnoea ($P < 0·10$). In addition, the proportion of insulators with more than the mildest grade of symptoms was much higher. There was no difference in the incidence of previous attacks of bronchitis. Twice as many insulators as controls gave a history of either previous pneumonia or pleurisy or tuberculosis but the difference did not reach a significant level ($P < 0·20$). In the case of rales the difference was marked; they were noted in nine insulators and in only one control ($P < 0·02$). There was no difference in the incidence of rhonchi.

**Clubbing**

Measurements of the hyponychial angle of both index fingers are summarized in Table 4. The reason for omitting measurement on some digits was that the subject pressed the end of his finger against the base of the container in which the impression was made and the subsequent cast was therefore distorted. The insulators had a significantly greater angle in both left and right index fingers, indicating a shift from the normal towards the clubbed range.

**Asbestos bodies**

Sputum from 42 insulators was examined and asbestos bodies were seen in nine cases. No asbestos bodies were seen in 27 control sputa examined.

TABLE 3

SYMPTOMS AND SIGNS OF CHEST DISEASE

| *Clinical feature* | *Insulators* | *Controls* | P[1] |
|---|---|---|---|
| Dyspnoea[2] – | | | |
| mild (grade 2) | 16 | 15 | |
| more severe (grades 3-5) | 7 | 1 | |
| combined | 23 | 16 | 0·10> P> 0·05 |
| Cough[2] – | | | |
| grade 1 | 9 | 10 | |
| grade 2 | 24 | 8 | |
| combined | 33 | 18 | <0·01 |
| Sputum[2] – | | | |
| grade 1 | 13 | 13 | |
| grade 2 | 24 | 10 | |
| combined | 37 | 23 | <0·01 |
| Previous chest illnesses – | | | |
| bronchitis | 13 | 12 | |
| pneumonia | 7 | 5 | |
| pleurisy | 8 | 3 | |
| tuberculosis | 1 | 0 | |
| last three combined | 16 | 8 | 0·20> P> 0·10 |
| Rales – | | | |
| grade 1 | 8 | 1 | |
| grade 2 | 1 | 0 | |
| combined | 9 | 1 | <0·02 |
| Rhonchi | 2 | 2 | |

[1]P was obtained by using Billewicz (1964) paired $\chi^2$ test.
[2]The grading is that recommended in the Instructions for the use of the Medical Research Council questionnaire (1960).

**Radiological findings**

These are summarized in Table 5. Only one control

TABLE 4

HYPONYCHIAL ANGLES OF INSULATORS AND CONTROLS COMPARED

| | *Insulators (degrees)* | *Controls (degrees)* |
|---|---|---|
| Left index finger | 186·0 ± 1·263 | 181·1 ± 0·864 |
| *t* | 2·885 | |
| P | <0·01 | |
| Right index finger | 184·6 ± 1·260 | 180·4 ± 1·001 |
| *t* | 2·633 | |
| P | <0·02 | |

Left index finger 44 pairs; right index finger 45 pairs: means ± standard errors.

TABLE 5

RADIOLOGICAL FINDINGS

| *X-ray grade* | *Insulators* | *Controls* |
|---|---|---|
| 1 Normal | 12 | 37 |
| 2 Possibly abnormal | 17 | 12 |
| 3 Pleural calcification only | 1 | 0 |
| 4 Abnormal – ? early asbestosis | 6 | 0 |
| 5 'Fibrosis' – ? atypical asbestosis | 5 | 1 |
| 6 Asbestosis | 9 | 0 |
| Total | 50 | 50 |

Combining grades 3 to 6, $P < 0.001$ (Billewicz (1964) paired $\chi^2$ test).
Because some of the control *x*-rays were 100mm films, the I.L.O. classification was not used.

had definite abnormality. His *x*-ray suggested the presence of diffuse fibrosis not typical of asbestosis. He gave no history of exposure to asbestos or other dusts and no explanation for the abnormal *x*-ray was found.

In marked contrast, 21 of the 50 insulators had definitely abnormal *x*-rays. Of these, 14 showed definite *x*-ray evidence of fibrosis and one showed pleural calcification only. The fact that 31 of the 50 control *x*-rays were 100mm films could have biased the comparison since these films were clearly from a supposedly normal population. It is, however, felt that any bias from this source could account for only a very small part of the marked difference between the two groups in the incidence of definite abnormality recorded jointly by two experienced observers of chest *x*-rays.

**Lung function tests**

A comparison of the mean results for controls and insulators is given in Table 6. The predicted differences shown are calculated from formulae given by Cotes (1965), apart from the Kl (McGrath and Thomson, 1959) and Dm and Vc (Hamer, 1962).

In the tests of static lung volumes the insulators had smaller volumes than the controls in all cases. For residual volume (RV) and total lung capacity (TLC) differences were statistically significant and greater than predicted. In the tests of dynamic lung volumes, however, the insulators and controls showed no significant differences. The insulators' $FEV_{1.0}/FVC\%$ was slightly less than the controls and their mean PFR was the same as that of the controls, although their predicted value was slightly less. In tests which provide an indication of the efficiency of the lungs with respect to gas exchange the insulators had poorer results. For arterial $P_{O_2}$ and alveolar-arterial oxygen difference ($A\text{-}aP_{O_2}$)

TABLE 6

LUNG FUNCTION TESTS

| *Test* | *Insulators* | *Controls* | *Difference Mean (S.E.)* | P | *Predicted difference due to age and height*[1] |
|---|---|---|---|---|---|
| *Static lung volumes* | | | | | |
| IC (l.) | 2·74 | 2·78 | −0·04 (0·11) | 0·70 > P > 0·60 | |
| ERV (l.) | 1·43 | 1·57 | −0·14 (0·10) | 0·20 > P > 0·10 | |
| VC (l.) | 4·17 | 4·36 | −0·19 (0·13) | 0·20 > P > 0·10 | −0·10 |
| RV (l.) | 1·55 | 1·78 | −0·23 (0·11) | < 0·05 | −0·07 |
| TLV (l.) | 5·71 | 6·16 | −0·45 (0·17) | < 0·01 | −0·19 |
| *Dynamic lung volumes* | | | | | |
| $FEV_{1.0}$ (l.) | 3·05 | 3·20 | −0·15 (0·13) | 0·20 > P > 0·10 | −0·07 |
| FVC (l.) | 4·22 | 4·35 | −0·13 (0·13) | 0·30 > P > 0·20 | −0·10 |
| $FEV_{1.0}/FVC$ % | 72·4 | 73·6 | −1·2 (1·9) | 0·60 > P > 0·50 | 0·2 |
| PFR (l./min) | 492·4 | 492·5 | −0·1 (16·4) | 1·00 > P > 0·90 | −6·1 |
| *Tests related to gas exchange* | | | | | |
| Minute volume (l.) | 8·73 | 8·05 | 0·68 (0·55) | 0·20 > P > 0·10 | |
| $V_D/V_T$ | 38·9 | 35·4 | 3·5 (2·1) | 0·20 > P > 0·10 | |
| Tl (ml/min/mmHg) | 20·0 | 21·1 | −1·1 (0·7) | 0·20 > P > 0·10 | −0·6 |
| Kl (ml/min/mmHg/l.) | 4·45 | 4·54 | −0·09 (0·12) | 0·50 > P > 0·40 | 0·02 |
| Dm (ml/min/mmHg) | 33·8 | 34·0 | −0·2 | 0·90 > P > 0·80 | 0·2 |
| Vc (ml) | 43·0 | 48·8 | −5·8 | 0·10 > P > 0·05 | 0·1 |
| $A\text{-}aP_{O_2}$ (mm) | 24·0 | 15·5 | 8·6 (2·7) | < 0·01 | |
| $P_{O_2}$ (mm) | 85·6 | 93·4 | −7·9 (2·0) | < 0·001 | |
| $P_{CO_2}$ (mm) | 35·3 | 35·7 | 0·4 (1·1) | 0·70 > P > 0·60 | |

Dm and Vc were analysed in reciprocal.
[1]Cotes (1965); McGrath and Thomson (1959) (Kl); Hamer (1962) (Dm and Vc).

the differences were significant ($P < 0.001$ and $P < 0.01$ respectively).

In summary, the insulators had, on average, lungs which were smaller and less efficient at gas exchange than the controls but air flow was unaffected.

## Discussion

The validity of the present comparison of insulators with matched controls depends on two main assumptions – firstly, that the sample of insulators was representative and, secondly, that the control series was appropriate. As the entire population of insulation workers was eventually examined (Langlands *et al.*, 1971) it is possible to compare the present sample with the complete population (Table 7). The sample examined appears to be representative.

TABLE 7

PRESENT SAMPLE OF INSULATORS COMPARED WITH ENTIRE POPULATION

| | *Present sample* | *Entire population* |
|---|---|---|
| No. examined | 50 | 252 |
| No. not examined | 3 | 20 |
| % Examined | 94 | 93 |
| Age (yr) | 39·4 | 40·5 |
| Height (cm) | 169 | 169 |
| Exposure (yr) | 16·1 | 16·4 |
| Smoking habit | | |
| 0 | 13 (26%) | 40 (16%) |
| ex | 9 (18%) | 31 (12%) |
| grade 1 | 5 (10%) | 50 (20%) |
| 2 | 13 (26%) | 80 (32%) |
| 3 | 10 (20%) | 51 (20%) |

The controls closely matched the insulators in age, height, and smoking habit. As skilled tradesmen they were of similar social class. Some of the controls had occasional contact with asbestos and one had worked as an insulator for three years. This contact with asbestos probably reflects the widespread use of the material. The exclusion of a control subject because of contact with asbestos would probably not be justified since contact with asbestos has been shown to be very common in Belfast where Elmes and Wade (1965) found asbestos bodies in the lungs of 20 to 25% of men at necropsy.

Thus the comparison between insulators and controls appears valid and should reveal the effects of exposure to insulating asbestos as distinct from the effects of living and working in an industrial environment.

The features which were commoner in the insulators and may be presumed to be due to exposure to asbestos were a history of cough and sputum and the presence of rales, increased hyponychial angles, *x*-ray signs of pleural calcification and/or pulmonary fibrosis, a reduction in static lung volume, a reduced arterial oxygen tension, and an increased alveolar-arterial oxygen difference. These are the features generally associated with asbestosis. The clinical findings of dyspnoea, cough, sputum, clubbing, and rales have been commented on repeatedly (Oliver, 1927; Seiler, 1928; McShane, 1930; Merewether, 1933-4; Lynch and Smith, 1935; Lanza, 1938; Wyers, 1949; Knox, 1958; Perry, 1963; Leathart, 1964; Hutchinson, 1965). In the present study, dyspnoea was commoner in the insulators but the difference was not quite significant at the conventional level of $P < 0.05$. Measurement of the hyponychial angle has been shown by Regan *et al.* (1967) to be a satisfactory index of clubbing. It is of interest that their mean result for normal fingers (187·0) was greater than the present results in insulators (184·6, 186·0) which, in turn, were significantly greater than the present results in controls (180·4, 181·1). Diagnosis of asbestosis by *x*-ray appearances has been shown to be uncertain in the individual case (Williams and Hugh-Jones, 1960a). However, in the present comparison of groups of individuals there was a very clear difference, with only one abnormal *x*-ray in the control series and 21 in the exposed series. A reduced arterial oxygen tension and a raised alveolar-arterial oxygen difference have been reported by a number of workers (Wright, 1955; Williams and Hugh-Jones, 1960b; Bjure, Söderholm, and Widimsky, 1964). Reduced lung volumes without airway obstruction were also found. These are also consistent with the present study.

Without the control group it would have been possible, from the radiological abnormalities, to suspect the presence of asbestosis in the population of insulators. Because a control group was used, it was possible to prove that the radiological abnormalities in this population were associated with symptoms, signs, and abnormalities of lung function. It must be pointed out that the average disturbance in lung function compared with the controls was relatively mild despite the large excess of abnormal chest *x*-rays. In addition there was only a small excess incidence of dyspnoea. This suggests that much of the dyspnoea was due to factors other than asbestos and is in keeping with the conclusion of Langlands and her colleagues (1971) that smoking was associated with a greater impairment of lung function than was *x*-ray abnormality.

## Conclusions

The present study suggests that the following features are useful in the diagnosis of asbestosis in

a population – an abnormal chest *x*-ray, the presence of cough, sputum, clubbing (assessed by measuring the hyponychial angle), and rales, and lung function tests which show a decrease in static volume, a reduced arterial oxygen tension, and increased alveolar-arterial oxygen difference but no evidence of airways obstruction. Because abnormalities in these respects may occur fairly commonly in any industrial population and because techniques vary somewhat from laboratory to laboratory, use of a control series is important.

This investigation was suggested and encouraged by Professors O. L. Wade and P. C. Elmes. Assistance in questioning and examining the men was given by Mr. J. Collins, Mrs. M. Simpson, Miss A. Stevenson, and Miss S. Wheeler. Specimens of sputum were examined by Miss D. P. Bell. Chest *x*-rays were taken under the supervision of Dr. J. O. Y. Cole and read with his assistance and that of Professor Elmes. The computer programme was written by Dr. J. D. Merett. Employers, trades union and individual men co-operated willingly. One of us (W.F.M.W.) was supported by a Royal Victoria Hospital Research Fellowship and a Research Fellowship from the Chest and Heart Association. We wish to thank Miss M. Wilkinson for typing the manuscript.

## References

Bader, M. E., Bader, R. A., Tierstein, A. S., and Selikoff, I. J. (1965). Pulmonary function in asbestosis: serial tests in a long-term prospective study. *Ann. N.Y. Acad. Sci.*, **132**, 391-405.

Billewicz, W. Z. (1964). Matched samples in medical investigations. *Brit. J. prev. soc. Med.*, **18**, 167-173.

Bjure, J., Söderholm, B., and Widimsky, J. (1964). Cardiopulmonary function studies in workers dealing with asbestos and glasswool. *Thorax*, **19**, 22-27.

Campbell, E. J. M. (1960). Simplification of Haldane's apparatus for measuring carbon dioxide concentration in respired gases in clinical practice. *Brit. med. J.*, **1**, 457-458.

Comroe, J. H., Forster, R. E., Jr., DuBois, A. B., Briscoe, W. A., and Carlsen, E. (1962). *The Lung: Clinical Physiology and Pulmonary Function Tests*, 2nd ed. Year Book Medical Publishers, Chicago.

Cotes, J. E. (1965). *Lung Function: Assessment and Application in Medicine*. Blackwell Scientific Publications, Oxford.

Elmes, P. C., and Wade, O. L. (1965). Relationship between exposure to asbestos and pleural malignancy in Belfast *Ann N.Y. Acad. Sci.*, **132**, 549-557.

Fletcher, C. M., Elmes, P. C., Fairbairn, A. S., and Wood, C. H. (1959). The significance of respiratory symptoms and the diagnosis of chronic bronchitis in a working population. *Brit. med. J.*, **2**, 257-266.

Hamer, N. A. J. (1962). The effect of age on the components of the pulmonary diffusing capacity. *Clin. Sci.*, **23**, 85-93.

Hunt, R. (1965). Routine lung function studies on 830 employees in an asbestos processing factory. *Ann. N.Y. Acad. Sci.*, **132**, 406-420.

Hutchinson, J. E. M. (1965). Symposium on asbestosis – introductory lecture. *Newcastle med. J.*, **29**, 12-16.

Knox, J. F. (1958). The pneumoconioses and their control – asbestosis in the manufacture of asbestos textiles. *J. industr. Nurses*, **10**, 167-179.

Langlands, Jean H. M., and Wallace, W. F. M. (1965). Small blood-samples from ear-lobe puncture: a substitute for arterial puncture. *Lancet*, **2**, 315-317.

——, ——, and Simpson, Marion, J. C. (1971). Insulation workers in Belfast. 2. Morbidity in men still at work. *Brit. J. industr. Med.*, **28**, 217-225.

Lanza, A. J., ed. (1938). In *Silicosis and Asbestosis*, p. 60. Oxford University Press, London.

Leathart, G. L. (1960). Clinical, bronchographic, radiological and physiological observations in ten cases of asbestosis. *Brit. J. industr. Med.*, **17**, 213-227.

—— (1964). Asbestos: a medical hazard of the 20th century. *Occupational Health*, **16**, 119-131.

Lynch, K. M., and Smith, W. A. (1935). Pulmonary asbestosis. III: Carcinoma of lung in asbestos-silicosis. *Amer. J. Cancer*, **24**, 56-64.

McGrath, M. W., and Thomson, M. L. (1959). The effect of age, body size and lung volume change on alveolar–capillary permeability and diffusing capacity in man. *J. Physiol. (Lond.)*, **146**, 572-582.

McKerrow, C. B., McDermott, M., and Gilson, J. C. (1960). A spirometer for measuring the forced expiratory volume with a simple calibrating device. *Lancet*, **1**, 149-151.

McShane, C. (1930). Asbestosis – a thesis submitted to the Queen's University of Belfast for the degree of Doctor of Medicine.

Meade, F., Saunders, M. J., Hyett, F., Reynolds, J. A., Pearl, N., and Cotes, J. E. (1965). Automatic measurement of lung function. *Lancet*, **2**, 573-575.

Medical Research Council (1960). Standardized questionnaires on respiratory symptoms. *Brit. med. J.*, **2**, 1665.

Merewether, E. R. A. (1933-4). A memorandum on asbestosis. *Tubercle (Lond.)*, **15**, 69-81, 109-118, and 152-159.

Oliver, T. (1927). Clinical aspects of pulmonary asbestosis. *Brit. med. J.*, **2**, 1026-1027.

Perry, K. M. A. (1963). In *Chest Diseases*, ed. K. M. A. Perry and T. Holmes Sellors, vol. I, p. 552. Butterworths, London.

Regan, G. M., Tagg, B., and Thomson, M. L. (1967). Subjective assessment and objective measurement of finger clubbing. *Lancet*, **1**, 530-532.

Seiler, H. E. (1928). A case of pneumoconiosis. Result of the inhalation of asbestos dust. *Brit. med. J.*, **2**, 982.

Thomson, M. L., Pelzer, A. M., and Smither, W. J. (1965). The discriminant value of pulmonary function tests in asbestosis. *Ann. N.Y. Acad. Sci.*, **132**, 421-436.

Williams, R., and Hugh-Jones, P. (1960a). The radiological diagnosis of asbestosis. *Thorax*, **15**, 103-108.

—— and —— (1960b). The significance of lung function changes in asbestosis. *Thorax*, **15**, 109-119.

Wright, B. M., and McKerrow, C. B. (1959). Maximum forced expiratory flow rate as a measure of ventilatory capacity. *Brit. med. J.*, **2**, 1041-1047.

Wright, G. W. (1955). Functional abnormalities of industrial pulmonary fibrosis. *Arch. industr. Hlth*, **11**, 196-203.

Wyers, H. (1949). Asbestosis. *Postgrad. med. J.*, **25**, 631-638.

Received for publication July 21, 1970.