CLINICAL, RADIOLOGICAL AND FUNCTIONAL ABNORMALITIES AMONG WORKERS OF AN ASBESTOS-CEMENT FACTORY

H. ROBIN • E. Eloy • J. L. Edmé • J. M. Brillet • P. Frimat • D. Furon
Institut de Médecine du Travail, Lille, France

The purpose of this paper is not to report an epidemiological study but to throw light on the problems of asbestos related effects in a factory in the North of France which is mainly producing fibrocement pipes and roofing components. In this factory a population of 1800 workers had been employed in 1965 but the total number was reduced to 476 in 1987. Among the different kinds of asbestos, actinolite was never used and crocidolite unused after 1980. If several projects were already under way as early as 1952, preventive measures concerning asbestos related diseases became really effective about 1975. Notifications of occupational diseases were not frequent until 1984 but the three following years 106 cases were reported. This study relates clinical, radiological and functional abnormalities among 92 workers investigated the two last years within an expert evaluation.

SUBJECTS AND METHODS

The study was carried out in 92 male subjects with following biometric characteristics (mean and standard deviation) : age : 54 ± 9.4 years, weight : 77 ± 17.5 kg and height : 1 7 ± 0.06 m. Forty three subjects (47 p.100) were still working in the asbestos-cement factory at the time of examination and the others were recently retired. The risk exposure was 32 ± 7.3 years and it was generally the first notification of disease. Each patient was submitted to clinical, radiological and respiratory function investigations. The subjects answered a questionnaire mainly intended to specify risk exposure, smoking status, breathlessness and symptoms of bronchitis according to the usual criteria.[1] Radiological examination consisted of a chest radiograph (standard postero-anterior and lateral views) and CT scan if necessary. Respiratory function tests included lung volumes measurement, transfer factor determination and blood gas analysis. Vital capacity (VC), forced expiratory volume in one second (FEV_1 and functional residual capacity (CRF) were measured with a Gould Pulmonet III and FRC computer. Carbon monoxide transfer factor was obtained using single breath (TlCO SB) and steady-state (TlCO StSt) methods with respectively a Morgan Transfer test autolink and a SNIAS Syscomoram R. The predicted values were those of CECA[7] for lung volumes, those of BATES and all[2] for TlCO SB and DECHOUX and all[8] for TlCO StSt. Arterial blood was sampled in the patients at rest in sitting position, and analysed on a Corning 178 pH/blood gas system or an IL Meter 1306 apparatus. The predicted values for PaO_2 were evaluated according to SORBINI and all.[28]

RESULTS

In the population of 92 workers, 12 subjects had normal chest roentgenograms. Among the radiological abnormalities we found 5 pulmonary fibrosis, 9 benign pleural thickenings and 57 associated pulmonary fibrosis and non malignant pleural changes. Diagnosis of malignant pleural mesothelioma was established in 8 cases. For one patient there was a bronchial carcinoma with concomitant asbestosis and pleural plaques. The number of smokers and ex-smokers was important : 67 subjects (73 p.100). Twelve subjects (22 p.100) were suffering from symptoms of chronic bronchitis.

Asbestos-related occupational disease was recognized in 80 subjects but in our results we did not include mesotheliomas because functional respiratory investigations were not ever complete in these patients. The variance analysis showed that it was possible to express data for a population grouping together pulmonary, pleural and associated forms. In these 72 subjects, age (yr) was 55 ± 7.4 and risk exposure (yr) 33 ± 7.3 (m ± SD). Active life, smoking habits, chronic bronchitis were respectively observed in 50, 79 and 24 p.100 of the patients. Abnormal spirographic values were measured in 60 cases (83 p.100) with restrictive syndrome predominancy (58 p.100). Residual volume was always found in the predicted limits. TlCO steady-state was decreased in 76 p.100 of the group, DuCO (fractional uptake) in 50 p.100, TlCO single breath only in 29 p.100 and TlCO/VA (transfer factor by alveolar volume) in 14 p.100. Arterial hypoxemia was showed up in 35 cases (49 p.100) with PaO_2 = 9.2 ± 0.81 KPa (0.88 predicted) and mild hypercapnia was found only in 6 cases (8 p.100). The values of the respiratory function indices are listed in Table I.

There was no significant difference in age, risk exposure, smoking habits, TlCO and hypoxemia between subjects suffering or not from a chronic bronchitis. However as shown in Table II they were different in VC, FEV_1 and FEV_1/VC values.

With regard to the 12 free of asbestos-related disease subjects according to chest radiograph, age and risk exposure were respectively 52 ± 6.6 and 27 ± 6.0 years. The frequency of active life, smoking habits and chronic bronchitis was the same we observed in the total population. There were functional abnormalities with decreasing in VC (0.85 ± 0.180 predicted), FEV_1 (0.79 ± 0.274 predicted), TlCO

Table I

Respiratory Function Tests in Asbestos-Related Disease (n = 72)

VC/pred	0.84 ± 0.150
FEV 1/pred	0.84 ± 0.209
FEV 1/VC	0.69 ± 0.141
TlCO StSt/pred	0.73 ± 0.307
DuCO (FuCO)	0.42 ± 0.109
TlCO SB/pred	1.03 ± 0.305
PaO_2/pred	0.97 ± 0.169

X ± SD

Table II

VC, FEV_1, FEV_1/VC Values in Asbestos-Related Disease

	A (n=17)	B (n=55)
VC/pred	0.74 ± 0.167*	0.87 ± 0.132
FEV 1/pred	0.67 ± 0.183*	0.89 ± 0.188
FEV 1/VC	0.64 ± 0.155*	0.71 ± 0.100

X ± SD. A = with chronic bronchitis, B = without chronic bronchitis. * P < 0.001

StSt (0.71 ± 0.275 predicted) and TlCO SB (0.96 ± 0.210 predicted). Hypoxemia was present in 3 cases.

DISCUSSION

Epidemiological investigations had demonstrated causal relation between asbestos exposure and non malignant pleuropulmonary pathology, mesothelioma and bronchial carcinoma.[31] Several studies attempted to determine nature and characteristics of the responsible fibres and to establish a dose-effect relationship.[25,3,26,30,11] As said in introduction our study was not an epidemiological one and an endeavour to do correlations between risk exposure and asbestos-related effects did not appear justified. Only some studies had underlined that it was very difficult to have a precise evaluation of exposure.[9,10,23,5] In fact, asbestos fibres inhalation by the workers had been varying in quality and quantity through the years. However it was possible to relate the occupational diseases to a very important dust pollution in the factory before 1975s. Furthermore the classical long delay from exposure to radiological diagnosis and the frequency of smoking habits were evident.

The repartition of the subjects according to exposure-related effects indicated few isolated fibrosis and non malignant pleural pathology with predominancy of associated pleuropulmonary disease. These findings were not surprising because the association of pulmonary asbestosis and pleural plaques, though still remaining controversial, can be shown in patients.[18,16,27,14,17] The lung fibrosis was more often a slight one on the radiographs. The frequency of "occupational bronchitis" was found important in our patients including the free of asbestos-related disease subjects.[21,19,4] It would be reasonable to suggest a synergistic effect between dust exposure and cigarette smoking to explain these results. Indeed, after retired, a number of patients saw that their symptoms had decreased or disappeared with no change in smoking habits.

Abnormalities in respiratory function tests frequently occurred without a clear relationship to put forward dyspnea. A restrictive function pattern was found in half the population, normal spirographic values in 20 p.100 and a mixed picture of restriction and obstruction in about 30 p.100 of the cases. These observations are not in opposition to the data of the literature.[12,24,22,32,13,29,6,20] Non uncommon impairment of gas exchange was indicated by decreasing in transfer factor, more frequently in TlCO steady-state and DuCO than in TlCO single breath and TlCO/VA, and attested by blood

gas analysis. This investigation detected at rest an arterial hypoxemia about one time out of two and also a mild hypercapnia in 8 p.100 of the subjects. Such disorders do not appear commonly described in predominant pleural asbestos effects but were found with the same frequency in coal miners.[15]

Bronchitis brought about alterations in spirographic values but no significative modification in transfer factor and blood gases. For the same biometric characteristics, risk exposure and smoking habits, VC, FEV_1, FEV_1/VC were more decreased in bronchitic than in non bronchitic subjects and restrictive and obstructive lung function profile was underscored.

At last in the absence of typical radiological changes, asbestos workers were not free of probably linked to dust exposure functional abnormalities.

Our study reported pathological effects in relation to an important asbestos exposure. At present time, with preventive measures, the risk has become less worrying in asbestos-cement factories. However environmental measurements remain fundamental for defining safe working dust levels to avoid occupational diseases.

REFERENCES

1. American Thoracic Society.: Chronic bronchitis, asthma and pulmonary emphysema. A statement by the Committee on diagnostic standards for non tuberculosis respiratory diseases. *Am. Rev. Respir. Dis.* 85:762-768 (1962).
2. Bates.D.V., Christie.R.V.: *Respiratory function in disease* 1 vol., 556 p. Ed. W.B. Saunders Co., Philadelphia (1964).
3. Becklake M.R.: Asbestos-related diseases of the lung and other organs: their epidemiology and implications for clinical practice. *Am. Rev. Respir. Dis.* 114:187-227 (1978).
4. Becklake M.R., Gibbs G.W, Arhirii M.I.: Health effects of exposure to asbestos in manufactoring processes. *Final Report Project* 605-1173-73, Health and Welfare. Canada (1979).
5. Berry G., Gilson J.C., Holmes S., Lewinsohn H.C., Roach S.A.: Asbestosis : a study of dose-response relationships in an asbestos textile factory. *Br. J. Ind. Med.* 36:98-112 (1979).
6. Boulier A., Fabre J., Lemenager J., Caillard J.F., Vergnaud M.C., Petiot J.F.: Comparaison entre les donnees de l'exploration fonctionnelle respiratoire et les images radiologiques pulmonaires. A propos de 106 sujets exposes a l'amiante. *Rev. Fr. Mal. Respir.* 8:211-218 (1980).
7. C.E.C.A.: *Aide-memoire pour la pratigue de l'examen de la fonction ventilatoire par la spirographie.* 2ème Ed., Commission des Communautes Europeennes, 1 vol., 127 p. (1971).
8. Dechoux J., Pivoteau C.: La capacite de diffusion alveolo- capillaire (sa mesure chez le sujet normal et le silicotique) *Rev. Tuberc.* 24:267-282 (1960).
9. Doll R.: Mortality from lung cancer in asbestos workers. *Br. J. Ind. Med.* 12:81-86 (1955).
10. Enterline P.E., Decoufle P., Henderson V.: Respiratory cancer in relation to occupational exposures among retired asbestos workers. *Br. J. Ind. Med.* 30:162-166 (1973).
11. Finkelstein M.M., Vingilis J.J.: Radiographic abnormalities among asbestos-cement workers. An exposure response study. *Am. Rev. Respir. Dis.* 129:17-22 (1984).
12. Fournier-Massey G., Becklake M.R.: Lung function in relation to chrysotile asbestos mine and mill workers of Quebec. Appendice: Epreuves de fonction respiratoire. *Bull. Physiopath. Respir.* 6:661-667 (1970).
13. Fournier-Massey G., Becklake M.R.: Pulmonary function profiles in Quebec asbestos workers. *Bull. Physiopath. Respir.* 11:429-445 (1975).
14. Fridrikson H.V., Hendenstrom H., Hillerdal G.: Increased lung stiffness in persons with pleural plaques. *Eur. J. Respir. Dis.* 62:412-424 (1981).
15. Guerrin F., Robin H., Boulenguez C.: Parametres respiratoires chez les mineurs pneumoconiotiques du bassin houiller Nord- Pas de Calais. *Bull. Physiopath. Respir.* 11:569-588 (1975).
16. Hagestrand I., Seifert B.: Asbestos bodies and pleural plaques in human lung at autopsy. *Pathol. Microbiol. Scand.* 81:457-460 (1973).
17. Hillerdal G.: Nonmalignant pleural disease related to asbestos exposure. *Clin. Chest. Med.* 6:141-152 (1985).
18. Hourihane 0.B., Lessof L., Richardson P.C.: Hyaline and calcified pleural plaques as an index of exposure to asbestos. A study of radiological and pathological features of 100 cases with a consideration of epidemiology. *Br. Med. J.* 1:1069-1074 (1966).
19. Lebowitz M.D.: Occupational exposures in relation to symptomatology and lung function in a community population. *Environ. Res.* 14:59-67 (1977).
20. Lemenager J., Raffaelli C., Letourneux M., Sesboue B., Boulier A.: Atteintes pleurales et retentissement fonctionnel de l'asbestose chez 380 sujets exposes. *Presse Med.* 14:1462-1464 (1985).
21. Mc Donald J.C., Becklake M.R., Fournier-Massey G., Rossiter C.E.: Respiratory symptoms in chrysotile asbestos mine and mill workers of Quebec. *Arch. Environ. Health* 24:358-363 (1972).
22. Muldoon B.C., Turner-Warwick M.: Lung function studies in asbestos workers. *Brit. J. Dis. Chest.* 66:121-132 (1972).
23. Peto J., Doll R., Howard S.V., Kinlen L.J., Lewinsohn H.C.:. A mortality study among workers in an English asbestos factory. *Br. J. Ind. Med.* 34:169-173 (1977).
24. Regan, G.M., Tagg, B., Walford, J., Thomson, M.L.: The relative importance of clinical, radiological and pulmonary function variables in evaluating asbestosis and chronic obstructive airways disease in asbestos workers. *Clin. Sci.* 41:569-582 (1971).
25. Selikoff I.J., Lee D.H.K.: *Asbestos and disease.* Academic Press Ed. 1 vol, 549 p. New-York (1978).
26. Selikoff I.J., Hammond E.C., Seidman H.: Mortality experiences in insulation workers in the United States and Canada—1943-1976. *Ann. N.Y. Acad. Sci.* 330:91-116 (1979).
27. Sheers G.: Asbestos associated disease in employees of Devonport Dockyard. *Ann. N.Y Acad. Sci.* 330:289-294 (1979).
28. Sorbini C.A., Grassi V., Solinas E., Muiesan G.: Arterial oxygen tension in relation to age in healthy subjects. *Respiration.* 25:3-13 (1968).
29. Tourreau A., Molina C., Cheminat J.C., Roche G., Gourgand A.J., Bedu M.: Bilan fonctionnel respiratoire chez les ouvriers de l'amiante a propos de 200 observations. *Rev. Fr. Mal. Respir.* 6:312-315 (1978).
30. Wagner J.C.: *Biological effects of mineral fibres.* World Health Organization, IARC. Scientific publications n°30. INSERM. Symposia Serie, vol. 92, 1001 p. Lyon (1980).
31. Weill H., Turner-Warwick M.: *Occupational lung diseases. Research approaches and method.* M. Dekker Inc. Ed. 1 vol. 504 p. New York (1981).
32. Zedda S., Aresini G., Ghezzi J., Sartorelli E.: Lung function in relation to radiographic changes in asbestos workers. *Respiration* 30:132-140 (1973).

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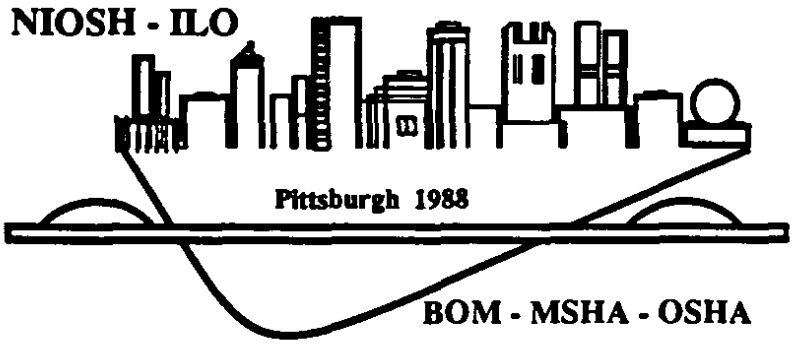

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