Lung function and radiographic signs of pulmonary fibrosis in oil exposed workers in a cable manufacturing company: a follow up study

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Abstract

Thirty seven workers employed for at least three years in oil impregnation of cables during 1963-83 were followed up in 1990 to study the development of pulmonary fibrosis and consequences for lung function. They had been exposed to concentrations of mineral oil vapours of 50-100 mg/m³, and concentrations of oil mist of 0.5-1.5 mg/m³. All 29 living persons were traced. For each person one control matched for age, height, and smoking habits was selected. Among 25 workers followed up with radiographic studies, 10 cases of pulmonary fibrosis were found, by contrast with one case in the control group ($p < 0.01$). Chest radiographs from 1979-80 and 1989-90 were reviewed. The profusion of small opacities increased in seven of 16 persons during 10 years without exposure. Seventeen workers had lung function tests. The bellows function (VC, FEV₁, MVV) and lung volumes (TLC, RV) did not differ from those in the matched controls ($p > 0.05$), but the carbon monoxide transfer factor (T_LCO) was decreased. The largest reduction of T_LCO (1.5 mmol/kPa/min) was found among workers exposed for 10 years or more ($p < 0.05$). Arterial blood gases were not affected at rest, but during maximum tread mill exercise, PO₂ and HbO₂ were reduced in exposed

workers compared with controls, particularly among those exposed for at least 10 years ($p < 0.05$). Exposure to low viscosity oil mist and vapour is the most plausible cause of the fibrosis. Unaffected bellows function, reduced T_LCO, and decreased arterial blood oxygen during exercise is compatible with peribronchiolar fibrosis.

A cross sectional study of 25 cable workers exposed to mineral oil aerosols and vapours was carried out in 1980. Seven cases of pulmonary fibrosis were found.¹ Six of the cases were among 14 impregnation workers, by contrast with one case among 11 workers employed in metal sheathing. In the control group of 25 workers without exposure to mineral oil, only one case of pulmonary fibrosis was found. Mineral oils used in cable impregnation are toxic to the lungs in animal experiments.² The publications concerned with oil mist include case reports of acute lipid pneumonia and pulmonary fibrosis after chronic exposure.³ No studies seem to have been done, however, on the progression or regression of oil mist fibrosis after cessation of chronic exposure.

Numerous studies have shown that pulmonary fibrosis caused by exposure to various organic agents may lead to reduced bellows function and disturbed gas transfer between alveoli and blood.³ Our study of active workers in 1980 found no reduction in bellows function, but tests of gas exchange and exercise tests were not performed. Only a few case reports of pulmonary fibrosis after chronic exposure to oil mist contain information about gas exchange and response to exercise.

The purpose of this study was twofold. Firstly, to study if progression of the pulmonary fibrosis in cable impregnation workers occurs after cessation of exposure to oil mist. Secondly, to study if exposure to cable oil disturbs pulmonary gas exchange at rest and during exercise. One hypothesis was that disturbed pulmonary gas exchange might occur in the absence of disturbed bellows function.

To evaluate the relation between exposure time, prevalence of radiographical fibrosis, and functional

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impairment in a small cohort, a matched pairs follow up design was chosen with controls for age, height, and smoking.

Material and methods

STUDY COHORT

A previous study of mortality and cancer provided a complete list of persons employed in the manufacture of cables involving oil impregnation at the Oslo plant of Alcatel STK from the start in 1920 to the end of 1979.⁴ From this list a cohort of all 37 workers employed after 1963 and working for at least three years in the impregnation department was constructed. Metal sheathing workers were not included in this follow up. The workers had been exposed to heat generated mineral oil vapour, partially condensing to oil mist. Most of the oil mist and vapour originated from low viscosity, low boiling range kerosene-like oils or from kerosene itself. The time weighted average (TWA) concentration of oil vapour has been estimated to be 50–100 mg/m³ and the oil mist concentration as 0.5–1.5 mg/m³. Exposure measurements and estimation of total exposure have been described in a previous study of pulmonary fibrosis¹ and in a report from a mortality and cancer study of cable workers.⁵ The production of oil filled cables was high during the 1960s and 1970s, decreased from 1980, and ceased in 1983. For all members of the cohort, years of exposure were calculated up to and including 1983. Median exposure time was 14 (range 3–41) years.

The follow up took place in 1990. Information about cases of cancer and causes of death in the cohort of the 37 workers were obtained from the Cancer Registry of Norway. Eight persons had died and median age at the time of death was 70 (range 38–84). Four of them died of cancer; two of whom had primary lung cancers. Occupational medical records were reviewed. One case of solvent encephalopathy was certified as an occupational disease, probably caused by exposure to kerosene vapour during the cleaning of hot production equipment.

Among the 29 living persons, seven were still at work in the company, seven worked in other companies or were unemployed, and 15 had retired with a pension. Eight persons older than 75 were only invited to have chest x ray films taken. Two retired workers aged 79 and 85 refused to participate, two persons younger than 75 agreed only to be interviewed, and four other persons refused to have physiological measurements taken. This left 25 subjects for the x ray film investigation; their median age was 58 (range 31–78). Seventeen of them also participated in the lung function and exercise tests; the median age in this group was also 58 (range 31–75).

CONTROL GROUP

For each of the 25 exposed persons who participated,

one control was selected from the company's records by pairwise matching. Controls were selected among male employees who were employed by or were receiving a pension from the company at the time of the investigation. Employees ever exposed to cable oil mist were excluded. Both office and production workers were eligible. The individual matching criteria were: same age (± 1 year); same height (± 3 cm), and same smoking habit at the time of examination.

Controls were selected stepwise. Firstly, a list of personnel sorted by birth dates was obtained. Lists of persons with the least age difference compared with the exposed subjects were produced, and persons with too large a height difference were excluded. Then the persons were contacted by telephone until one matching the smoking criterion was found. The resulting control group consisted of 19 production workers and six office employees. One subject younger than 76 and unfit for exercise testing (due to poor cooperation), was excluded and replaced; none in the exposure group had to be excluded.

EXAMINATIONS

All exposed and control persons were interviewed. All jobs held were recorded and exposure to possible lung toxic agents was asked for in detail. Smoking history was noted and the Norwegian translation of the British Medical Research Council (BMRC) questionnaire for chronic bronchitis⁶ was used to record respiratory symptoms.

The investigators responsible for the radiological and pulmonary function studies had no knowledge of the exposure state of the test subjects. New chest radiographs, including posteroanterior, lateral, and oblique views, were taken and coded according to the International Labour Office (ILO) Classification.⁷ The ILO category "small opacities," profusion 0/1 or more or the description of "basal fibrosis" were considered as positive. Later, chest radiographs of the impregnation workers taken in 1979–80 were reviewed and classified in the same manner.

Bellows function, including vital capacity (VC), forced expiratory volume in one second (FEV₁), and maximal voluntary ventilation (MVV) were determined with a low inertia, low resistance bell spirometer. Lung volumes including total lung capacity (TLC) and residual volume (RV) were determined with the He dilution technique using a Godart Expirograph and He analyser. The CO transfer factor of the lungs (T_LCO) was determined with the single breath technique by means of a Hewlett-Packard single breath diffusion system.

Pulmonary gas exchange at exercise was determined with two different tests performed on two different days. The first was based on moderate work of short duration on a cycle ergometer, with a closed circuit double spirometer system.^{8–11} The second was

based on walking to exhaustion on a motor driven treadmill using a Sensor Medics MMC Horizon system. In the cycle test CO₂ output, O₂ intake, and ventilation were recorded continuously during 20 minutes of rest, one minute of work (500 kpm; 4.9 kJ), and 20 minutes of recovery and rest. The CO₂ recovery time (CO₂RT) was determined as the time taken from the end of the exercise until the CO₂ output was reduced to the mean resting level plus 10%. Extra O₂ intake (EO₂) and extra ventilation (EV) were calculated as the total O₂ intake and ventilation for the last pre-exercise minute, one minute of exercise, and 10 minutes after exercise, minus the resting values for an equally long period (12 minutes). In the treadmill test the test subject was walking at a constant speed (80 m/min) starting with a horizontal gradient. After a familiarisation period of five minutes, the gradient was increased 1° every minute until exhaustion. In subjects reaching a grade of 12°, a further rise in exercise load was obtained by increasing the speed by 10 m/min every minute. The gas exchange was measured continuously while blood samples for blood gas determinations were withdrawn every three minutes through an indwelling catheter in the brachial artery. Arterial PCO₂, PO₂, and pH were measured using an Instrumentation Laboratory Micro Autocal pH blood gas analyser 613, and HBO₂% with a Radiometer OSM 3 Hemoximeter. Heart rate was continuously recorded with a Mingograf 7. Ventilation and expired air during exercise were analysed and O₂ uptake ($\dot{V}O_2$) and ventilation ($\dot{V}E$) were calculated. All volumes, except O₂ intake and CO₂ output, are given converted to body temperature, prevailing pressure, and water saturation (BTPS). The O₂ and CO₂ data are given at standard temperature and pressure (STPD).

STATISTICAL METHODS

Exposed subjects and matched controls were compared in pairs with the SPSS statistical package for PC (SPSS/PC+ V3.0).^{12,13} McNemar's test was employed to compare frequencies of lung fibrosis and respiratory symptoms.¹⁴ Wilcoxon's matched pairs signed ranks test was used to compare measurements of lung function (continuous variables). Confidence intervals for differences between medians were

calculated for some function variables, using the CIA software and the Wilcoxon-based method for matched pairs.^{15,16} When the difference between observations for exposed subjects and controls was unlikely to go in more than one direction, one sided tests were used. The significance level was set at $p = 0.05$.

Results

RADIOLOGICAL FINDINGS

Table 1 shows that there were 10 cases of pulmonary fibrosis among the 25 exposed workers, by contrast with only one case among the 25 controls ($p < 0.01$). Half of the workers exposed for 10 years or more had fibrosis, and among the workers exposed for less than 10 years, three of the four with fibrosis had been exposed for nine years. The exposed workers had smoked slightly less than the controls. There were 11 present smokers in each group, eight ex-smokers among the exposed workers, and nine ex-smokers among the controls. Two of the impregnation workers with fibrosis were never smokers.

The typical description of the cases was that they had a moderate, diffuse basal fibrosis, most of them with the character of linear densities rather than rounded opacities. The ILO classification of the radiographs gave degrees of profusion of small opacities among the exposed of 0/0, 15 persons; 0/1, three persons; 1/0, three persons; 1/1, one person; 1/2, two persons; other types of fibrosis, one person. Four of the exposed persons had bilateral and one had unilateral pleural thickening. No pleural changes were calcified. The control person with a positive chest x ray film had profusion of small opacities 0/1, and bilateral pleural plaques along the chest walls. He had been handling, but never cutting, asbestos boards in a storage department since 1948.

Table 2 shows the degree of profusion of small opacities for 16 impregnation workers for whom pictures were available from both 1979–80 and 1989–90. We found increased fibrosis in seven impregnation workers, and no cases of regression.

LUNG FUNCTION

Table 3 shows pulmonary function observations for

Table 1 Radiological pulmonary fibrosis among 25 oil impregnation workers and matched controls

	Exposed workers			Controls		
	≥ 3	3–9	≥ 10	—	—	—
Subjects (No)	25	13	12	25	13	12
Age (y)†	58 (43–71)	44 (35–58)	71 (61–75)	58 (43–71)	44 (35–58)	71 (61–75)
Total smoking (pack-years)†	15 (0–5–27)	10 (0–0–24)	23 (12–32)	18 (4–0–27)	14 (0–0–20)	26 (16–47)
Present smoking (cigarettes per day)†	0 (0–15)	0 (0–14)	4 (0–15)	0 (0–14)	2 (0–12)	0 (0–15)
Lung fibrosis (No)	10**	4	6	1	0	1

** $p < 0.01$ (McNemar, one sided).

†Median value; 25–75 percentiles in parentheses.

Table 2 Radiological pulmonary fibrosis among 16 oil impregnation workers followed up for 10 years after cessation of exposure

	1989-90 No of subjects in each category						
1979-80 No of subjects in each category	Profusion*				Emphysema	Emphysema + fibrosis	Total
	0/0	0/1	1/1	1/2			
Profusion:*							
0/0	9	0	3	1	0	0	13
0/1	0	0	1	1	0	0	2
1/0	0	0	0	0	0	0	0
1/1	0	0	0	0	0	0	0
1/2	0	0	0	0	0	0	0
Emphysema	0	0	0	0	0	1	1
Emphysema + fibrosis	0	0	0	0	0	0	0
Total	9	0	4	2	0	1	16

Classification in accordance with ILO.

*Profusion of small opacities.

17 exposed workers and their matched controls. Seven of the exposed workers who were tested showed radiological fibrosis. The smoking pattern was similar among exposed workers and controls. The bellows function (VC, FEV₁, MVV), the lung volumes (TLC, RV), and the reaction to moderate standard exercise of short duration (CO₂RT, EV, EO₂) showed no tendencies for differences between the groups; T_LCO and T_LCO/TLC, however, showed a clear but not statistically significant tendency towards lower values in the impregnation workers.

Table 4 shows that the peak oxygen uptake ($\dot{V}O_{2max}$) and heart rate on a tread mill were essentially the same in exposed workers and controls, whereas

the peak ventilation ($\dot{V}E_{max}$) was higher in impregnation workers ($p < 0.05$). Also the median $\dot{V}E_{max}/MVV$ (0.67) was higher in the impregnation workers than in the controls (0.61) ($p < 0.05$). No definite difference in $\dot{V}E_{max}/\dot{V}O_{2max}$ was found, however, between the two groups. The arterial blood gases were the same in both groups at rest (table 5). At maximum exercise, however, PO₂ and HbO₂ were clearly lower among the exposed despite their higher ventilation (table 4) and a tendency to lower PCO₂. Also pH at maximum exercise was slightly lower ($p < 0.05$) among the exposed workers.

The findings on lung function were further analysed with regard to the influence of time of exposure. Only T_LCO, T_LCO/TLC, and PO₂ and HbO₂ at maximal exercise showed differences dependent on duration of exposure. Table 6 shows that the values for these parameters were lower in workers exposed for at least 10 years (long term exposed) as compared with controls, but not in those with a shorter exposure time. The calculated difference between medians¹⁶ of T_LCO among the long term exposed compared with controls was 1.5 mmol/kPa/min (92.2% confidence interval (92.2%CI) -0.1 to 3.2). The difference between the medians of T_LCO/TLC among exposed subjects *v* controls was 0.21 mmol/kPa/min/l (92.2% CI 0.14-0.31). The long term exposed workers tended to have smoked less than matched controls; medians were 22.5 *v* 39.3 pack-years.

Impregnation workers reported respiratory symptoms more frequently than controls. The median number of positive answers to eight main questions from the BMRC questionnaire was two *v* one ($n = 21$, $p < 0.05$). Among seven workers below the age of 76 and exposed for 10 years or more four showed radiological pulmonary fibrosis. In the group of seven workers, the median number of positive answers was three *v* zero among the controls ($p < 0.05$). Four of

Table 3 Lung function among 17 oil impregnation workers 75 or younger and matched controls

	Exposed workers	Controls
	Median (25-75 percentiles)	Median (25-75 percentiles)
Age (y)	58.0 (42.5-65.0)	58.0 (42.5-65.5)
Height (cm)	173 (171-182)	176 (172-182)
Total smoking (pack-years)	15.0 (3.5-27.5)	19.5 (11.6-39.3)
Present smoking (cigarettes per day)	7.0 (0.0-17.5)	3.0 (0.0-15.0)
VC (l)	4.46 (3.80-5.86)	4.46 (3.99-5.34)
FEV ₁ (l)	3.41 (2.71-4.16)	3.52 (3.18-4.41)
MVV (l/min)	156 (100-180)	160 (147-184)
TLC (l)	7.10 (5.89-7.65)	6.47 (6.01-7.64)
RV (l)	1.90 (1.60-2.36)	1.80 (1.61-2.31)
RV/TLC (%)	29 (25-33)	27 (25-31)
CO ₂ RT (min)	7.0 (5.5-9.0)	7.0 (6.0-8.0)
EV (l)	46.0 (36.5-61.5)	48.0 (41.0-57.0)
EO ₂ (l)*	1.54 (1.31-1.81)	1.67 (1.58-1.81)
T _L CO (mmol/kPa/min)	8.70 (6.90-10.30)	10.10 (7.55-11.00)
T _L CO/TLC (mmol/kPa/min/l)	1.19 (1.07-1.59)	1.38 (1.19-1.58)

*n = 16.

No differences were significant at the 0.05 level (Wilcoxon, one sided).

the exposed *v* one control gave a positive answer to the question of breathlessness ($p = 0.05$). Exposed workers reporting breathlessness showed a tendency towards an increased $\dot{V}E_{\max}/MVV$ ratio; median values were 0.78 compared with 0.62 among the matched controls. This ratio did not differ between exposed workers without breathlessness and controls; median values were 0.61 and 0.60 respectively.

Table 4 Oxygen uptake, ventilation, and heart rate at maximum exercise among 14 oil impregnation workers and controls

	Exposed workers	Controls
	Median (25–75 percentiles)	Median (25–75 percentiles)
$\dot{V}O_2$ (l/min)	2.45 (2.02–2.89)	2.52 (1.87–3.10)
$\dot{V}O_2$ (ml/kg/min)	30.2 (22.3–36.5)	27.9 (25.0–35.3)
$\dot{V}E$ (l/min)	91* (66–116)	85 (55–107)
Heart rate	164 (151–182)	166 (149–176)

* $p < 0.05$ (Wilcoxon, one sided).

Discussion

AETIOLOGY AND DEVELOPMENT OF PULMONARY FIBROSIS

Several authors have described the occurrence of lung fibrosis in workers after chronic exposure to oil mist and vapour.^{17–22} The workers had been exposed to different mineral oil products—namely, lubricating oils, steel rolling oils, paraffin, or commercial grade kerosene. Lushbaugh *et al*²³ and Wagner *et al*²⁴ both found experimental pulmonary fibrosis in animals after long term inhalation of different mineral oils. So, it is generally accepted that mineral oils are capable of producing a fibrogenic response in the lungs. In the present study, however, asbestos and smoking are possible confounders.

Although exposure to oil is the dominant exposure in oil cable impregnation, low level occupational exposure to asbestos is likely to have occurred. Asbestos or talc containing asbestos was never used as a raw material in cable production in the impregnation or neighbouring departments. Background exposure to asbestos is probable, as chrysotile and amosite asbestos have been identified in insulation of

Table 5 Arterial blood gases at rest and at maximum exercise among 14 oil impregnation workers and matched controls

	Exposed		Controls	
	At rest	At maximum exercise	At rest	At maximum exercise
pH	7.42 (7.38–7.42)	7.32* (7.27–7.34)	7.40 (7.39–7.41)	7.33 (7.30–7.34)
PCO ₂ (kPa)	4.83 (4.69–5.09)	4.88 (4.25–5.52)	4.95 (4.76–5.24)	5.02 (4.56–5.31)
PO ₂ (kPa)	11.4 (9.7–11.9)	10.5* (9.4–11.6)	11.3 (10.8–12.6)	11.1 (10.4–12.7)
HbO ₂ (%)	96.6 (95.1–97.3)	93.9** (92.5–96.0)	96.6 (96.1–97.5)	95.8 (94.8–96.5)

Values are medians; 25–75 percentiles in parentheses.

* $p < 0.05$; ** $p < 0.01$ (Wilcoxon, one sided, exposed workers *v* controls).

Table 6 T_LCO at rest and arterial O_2 parameters at maximum exercise among oil impregnation workers and matched controls in relation to duration of exposure

		Exposed workers		Controls
	No of subjects	Exposure time (y)	Median (25–75 percentiles)	Median (25–75 percentiles)
Age (y)	10	3–9	44.0 (36.8–59.5)	44.0 (36.8–59.5)
	7	≥ 10	65.0 (59.0–72.0)	65.0 (59.0–72.0)
Total smoking (pack-years)	10	3–9	10.2 (0.0–20.0)	14.3 (1.7–20.0)
	7	≥ 10	22.5 (14.6–34.5)	39.3 (18.1–57.0)
Present smoking (cigarettes per day)	10	3–9	3.5 (0.0–20.0)	1.5 (0.0–13.5)
	7	≥ 10	14.0 (0.0–15.0)	14.0 (0.0–20.0)
T_LCO (mmol/kPa/min)	10	3–9	9.50 (8.03–11.63)	10.40 (8.38–12.00)
	7	≥ 10	6.80* (5.70–8.80)	7.90 (7.30–10.50)
T_LCO/TLC (mmol/kPa/min/l)	10	3–9	1.35 (1.17–1.72)	1.48 (1.26–1.67)
	7	≥ 10	1.06* (0.89–1.14)	1.20 (1.03–1.50)
PO ₂ (kPa)	8	3–9	10.9 (9.1–11.5)	11.1 (10.1–12.4)
	6	≥ 10	9.7* (8.8–10.5)	10.7 (9.5–11.4)
HbO ₂ (%)	8	3–9	95.1 (93.2–96.4)	96.0 (95.4–97.2)
	6	≥ 10	93.3* (92.0–94.3)	95.1 (93.4–96.0)

* $p < 0.05$ (Wilcoxon, one sided, exposed workers *v* controls).

steam pipelines and one lead sheathing press. As nearly all impregnation workers had the same low level exposure to asbestos, this material does not permit a multivariate analysis of the influence of exposure to asbestos. Seven of 25 controls had been exposed to low levels of asbestos, however, whereas only one case of fibrosis occurred in this group. If asbestos inhalation alone were responsible for the occurrence of pulmonary fibrosis, a higher prevalence of pleural changes would be expected than was seen among the exposed workers (20%). In conclusion, background exposure to asbestos may have contributed to the radiographic pulmonary fibrosis, but is unlikely to have been the main cause.

Several workers left the impregnation department in 1980, and exposure gradually ceased towards 1983 when production was stopped. None of the workers had been exposed to fibrogenic agents after leaving cable impregnation. Despite this, we found an increase in radiological fibrosis in seven of 16 workers and no cases of regression. This suggests that the type of pulmonary fibrosis acquired during this work may be progressive.

CONSEQUENCES FOR LUNG FUNCTION

As in the previous study,¹ we found no evidence of poorer bellows function, but T_LCO and T_LCO/TLC were decreased among the oil exposed workers, compared with controls, and this effect increased with the duration of exposure. The difference could not be explained by age, height, or smoking, as the pairs were matched with respect to these variables.

Oxygen uptake and lactate concentrations at maximum exercise were not different from controls, indicating that the effects on lung function lead to a marginal reduction of the work capacity in most of the exposed workers.

INFLUENCE OF SMOKING

It has been reported that smoking alone can produce diffuse pulmonary fibrosis.²⁵ In the present study the long term exposed workers tended to have smoked less than controls (tables 1 and 6). Thus smoking alone is unlikely to explain the radiological findings. As practically all the exposed workers were ever smokers, however, the possibility exists that smoking has contributed in some way to the development of fibrosis. This has been shown to be the case for smoking and asbestos.²⁶

It is established that smoking leads to lower diffusing capacity,²⁷ and one might speculate that an even greater difference in T_LCO would have been found, had the exposed workers smoked as much as the controls. Compared with the data of Cotes,²⁷ the controls of the long term exposed workers had lower values of T_LCO than expected for their age. This may be due to the fact that the seven controls for the long term exposed workers were heavy smokers (median

value: 39.3 pack-years) with an estimated group average of 17.5 cigarettes a day during their adult lifetime.

RELATION BETWEEN PULMONARY FIBROSIS,

FUNCTIONAL CHANGES, AND CLINICAL IMPLICATIONS

Most cases of fibrosis were classified according to ILO as 1/1 or lower and 10 of the 17 exposed subjects who were tested for lung function had no radiographic signs of fibrosis. Therefore it is not surprising that VC and FEV₁ showed no definite differences between exposed workers and controls; T_LCO and PO₂ at exercise are probably more sensitive indicators of fibrosis in the small airways.

At maximum exercise, arterial oxygenation was affected, although the long term exposed workers ventilated more than controls. The combination of essentially unaffected bellows function, reduced T_LCO and normal arterial blood gases at rest, and a definite fall in arterial oxygen combined with increased ventilating demands during exercise can be explained by peribronchiolar fibrosis, or loss of pulmonary capillaries, or both. This is a picture similar to that described by other authors. Weill *et al*²⁸ reported three cases of non-occupational lipid pneumonia after chronic exposure to mineral oil. One of the patients had pulmonary function tests. These showed no airflow obstruction, but pronounced ventilatory restriction, reduction in gas transfer, and lowered arterial oxygen saturation after exercise. Studies of asbestos exposed workers have shown obstruction of the small airways not detected by ordinary measurements of bellows function, indicating peribronchiolar fibrosis.²⁹

In this study, the prevalence of respiratory symptoms was increased among impregnation workers, as compared with controls. Eight exposed workers reported breathlessness; four of them, including three with pulmonary fibrosis, had lung function tests. These four had an increased $\dot{V}E_{max}/MVV$ ratio. This indicates that although the bellows function was unaffected, a moderate functional impairment of the small airways may be accompanied by respiratory symptoms. Routine spirometry testing of workers exposed to oil mist may, therefore, be insufficient in future health surveillance.

Conclusion

The high prevalence of pulmonary fibrosis together with the influence of the duration of exposure suggest a causal relation between oil impregnation work and pulmonary fibrosis. The most plausible cause of the fibrosis is exposure to low viscosity oil mist and vapours. Smoking and industrial exposure to asbestos from heat insulation may have contributed to the radiographic changes, but it is unlikely that these factors alone are responsible. Follow up chest radio-

graphs indicate that the fibrosis is progressive after cessation of exposure. The hypothesis that disturbance of the alveolocapillary gas transfer might occur in the absence of disturbed bellows function was confirmed. Both reduced CO transfer capacity of the lungs and lowered arterial oxygenation during exercise were found, despite increased ventilation. These changes are compatible with peribronchiolar fibrosis or "small airways disease."

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