TRANSLATION:

PLEURAL MESOTHELIOMA AFTER ASBESTOS DUST EXPOSURE IN BRAKE REPAIR WORK IN AUTOMOBILE REPAIR WORKSHOPS: CASE OBSERVATIONS*

Paur, R., H.-J. Woitowitz, K. Rödelsperger and H. Jahn: Pleuramesotheliom nach Asbeststaubgefährdung bei Bremsreparaturen im Kfz-Handwerk: Kasuistische Beobachtungen. *Prax. Klin. Pneumol.*, Vol. 39, pp. 362-366, 1985. Authors' affiliation: Institute and Polyclinic for Occupational and Social Medicine, Justus-Liebig University, Giessen.

ABSTRACT. The authors report on 4 cases of disease or death from diffuse malignant pleural mesothelioma in automobile repair workers. All the patients had their first contacts with the carcinogenic working material in their adolescence when performing repair jobs on brakes during apprenticeship. In three cases, meanwhile, the tumor has been recognized as an occupational disease in accordance with Federal German regulations. The disease appeared, on the average, 32 years after the first exposure to asbestos dust. It resulted in the death of the patient within only 2 to 9 months after diagnosis.

The risks involved in exposure to asbestos dust in automobile repair shops have been established for working on new brake linings and for the cleaning of brake drums from abraded dust. Measurement data are stated for typical jobs such as abrading and polishing, turning and cleaning. Risk of asbestos dust exposure has been reduced by improved technical protective measures. Hence, the asbestos dust doses measured in recent years are not readily comparable with the markedly higher exposure during the 'fifties and 'sixties.

Due to the long latency period of the disease there is a likelihood of an increase in the incidence of mesotheliomas during the next few years. This prognosis appears justified, since dust-protective measures and asbestos substitute linings have been introduced only relatively recently. The regulations governing employment of adolescents in jobs involving the handling of carcinogenic substances should receive close medical attention.

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INTRODUCTION

Brake systems and brake linings are exposed to considerable mechanical and thermal stresses in the interests of safety for the millions of motor vehicles traveling on our highways. Extreme requirements are imposed especially concerning the strength, thermal conductivity and heat storage ability as well as the most nearly constant frictional value [5]. In addition to frictional value modifiers, binders and fillers, conventional brake linings contain asbestos fibers, and these are exclusively chrysotile (so-called white asbestos). At present in the Federal Republic of Germany an annual consumption of about 10,000 tons of chrysotile for brake linings is still presumed [16].

On one hand, asbestos dust is released into the environment in the form of emissions. On the other hand such dust represents a health risk that can currently not be estimated for the vehicle mechanic. This is true for repairs on brake systems and clutches, in which abrasion and processing dust can enter the inhaled air.

In the following, case histories of 4 cases of fatal diffuse malignant pleural mesotheliomas in vehicle workers after exposure to asbestos dust as a result of abrasion and processing dust in brake repairs will be reported.

CASE PRESENTATION

1. The patient, born in 1937, began an apprenticeship as an auto mechanic at the age of 14. After completing his apprenticeship he worked in various shops until 1978. Among other things he performed maintenance and repair work on brake systems in car and truck shops. Especially in the 1950s and 1960s the work necessary for this purpose was performed without suction ventilation for the asbestos-containing abrasion and processing dust.

In May 1982 coughing and increasing chest pain developed. Beginning in September 1982 effort dyspnea was also noted. Thoracotomy resulted in the histological diagnosis of an epithelial, diffuse malignant pleural mesothelioma. Therapy had to be limited to a partial pleurectomy with partial resection of one lobe of the lung. Three months later the patient died from consequences of the tumor.

2. The automotive craftsman, born in 1932, initially worked for several hours a day on brake and clutch repair over a 3 year period during his apprenticeship beginning in 1947. The brake systems were mostly blown out with compressed air to clean them from asbestos-containing abrasive dusts. New linings were processed mechanically, i.e., ground or machined. During welding, asbestos sheets soaked in water were placed on the sheet metal to cool it, and then brushed off in dry form. An air circulation unit probably distributed the dust occurring uniformly throughout the hall. Beginning in 1950 the patient was employed as a stock clerk and a stockroom supervisor in a room adjacent to the shop. For many years the stocks of asbestos-containing brake linings were kept loose on a shelf without packaging.

In the end of 1981 penetrating chest pains developed, and in May 1982, dyspnea. The histological diagnosis of a bivalent differentiated diffuse malignant pleural mesothelioma was confirmed by thoracoscopy the following month. Following chemotherapeutic treatment, death occurred in March 1983.

3. The patient, born in 1931, had been employed for a total of 18 years, since he was 14, as a mechanic in various automotive shops. About 10-20 hr a week during this time were spent on the repair of brake systems, with release of asbestos-containing abrasion and processing dust.

His illness began in July 1982 with dyspnea and chest pains. In October

the histological diagnosis of an epithelial differentiated diffuse malignant pleural mesothelioma was made by biopsy. Treatment was limited to symptomatic measures, especially pain relief. Finally up to 12 injections of morphine a day were necessary. At the age of 51 the patient died due to suicide.

4. Born in 1939, the patient, 44 years old at the time of death, began an apprenticeship as a construction mechanic at the age of 15. Beginning in 1957 he was employed as an auto and plant mechanic in a truck shop. He did not actually work on brakes or clutches himself. However, since the work on the brake linings was not spatially separated from the remainder of the shop, in view of these nearby work spaces a "bystander" exposure had to be considered probable due to the asbestos-containing abrasion and processing dusts, at least for the 11 years from 1957 to 1968.

In addition an exposure may have existed due to asbestos dust emission from an adjacent brake lining factory.

The illness began in March 1983 with chest pain symptoms. The histological confirmation of the diagnosis was carried out in October 1983 on the basis of a surgical specimen following partial resection. An epithelial, diffuse, malignant pleural mesothelioma was involved. Tele-cobalt irradiation was performed postoperatively. The patient died two months later.

The most important chronological information, especially regarding asbestos exposure, is summarized in Table 1.

DISCUSSION

Mesothelioma as a Signal Tumor of Asbestos Dust Exposure

Diffuse malignant mesotheliomas are very rare tumors in the general population. In contrast, many epidemiologic studies have shown a substantially

TABLE 1. AGE AT BEGINNING OF ASBESTOS DUST EXPOSURE, DURATION OF ASBESTOS DUST EXPOSURE, TIME ELAPSED SINCE THE FIRST ASBESTOS DUST EXPOSURE (LATENCY TIME) AND AGE AT THE TIME OF DEATH IN 4 MOTOR VEHICLE REPAIR PERSONNEL WITH DIFFUSE MALIGNANT PLEURAL MESOTHELIOMA.

Patient code	Age at beginning of asbestos dust exposure	Time of exposure (years) *		Age at time of death
		until illness	until death	
1	14	27	31	45
2	15	35	35	50
3	14	18	37	51
4	18	11	26	44
$\bar{x} \pm s$	15.3 ± 1.9	22.8 ± 10.5	32.3 ± 4.9	47.5 ± 3.5

higher expectation frequency in certain groups of individuals threatened by asbestos dust. While about 1-8 mesothelioma deaths per million inhabitants, i.e., one death per 1000-10,000 deaths in the general population are to be expected, the fraction of mesothelioma deaths in groups of individuals more heavily exposed to asbestos dust is 6-11% [7, 21, 23]. Hartmann [4] recently reported in this journal on the problem of confirming the diagnosis from the viewpoint of the pathologist.

Investigators have not yet succeeded in confirming any other important causes of this tumor in humans aside from asbestos and comparable mineral fibers. Therefore diffuse malignant mesothelioma can be regarded as a signal tumor [18, 19, 23] for asbestos dust exposure. Previous investigations, including some among patients in our polyclinic, have shown that in addition to employees from the asbestos products manufacturing industry, users of asbestos containing products are especially likely to suffer from mesotheliomas [22].

*This heading was not completed in German; something is left out. We put headings in from the information given in the text. -- *Tr. Ed.*

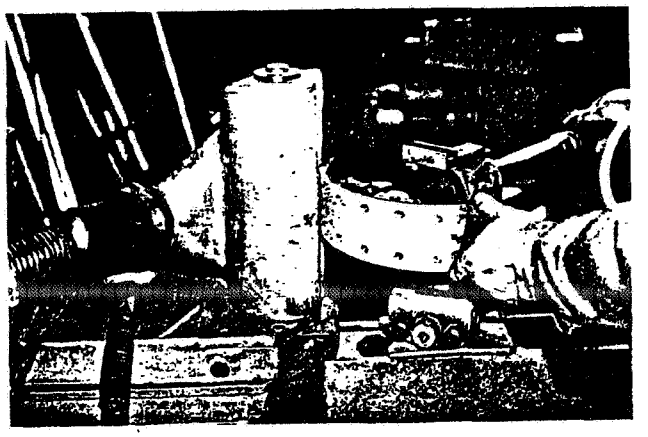

Figure 1. Regrinding an asbestos-containing truck brake lining on a grinding machine with suction of the processing dust. During grinding of the large area lining, asbestos fiber concentrations occur which can be estimated on the basis of a dust measurement covering the 5 min working process at about $5 \cdot 10^6$ chrysotile fiber/m^3 with a length of >5 µm. Thus during one breath at a volume of 1-2 ℓ, about 5000-10,000 chrysotile fibers, L >5 µm, would be inhaled. At a respiratory air consumption of 20 ℓ/min, during the regrinding of 4 brake linings on one axle observed here over a 5 min period a total of about 500,000 chrysotile fibers, L > µm, would be involved. Since the asbestos dust exposure can exceed the permissible industrial guideline concentration (IGC) in the case of long working processes despite suction, brake linings of buses and trucks are generally not reground today, but are remachined instead.

Asbestos Dust Exposure in the Motor Vehicle Repair Trade

At the end of 1982 in the Federal Republic of Germany more than 220,000 individuals were exclusively employed in the workshop area as motor vehicle mechanics, trainees or skilled or unskilled assistants [24]. Asbestos dust exposure can be anticipated here both

-- during the preparation of new coatings, and

-- during removal of the abrasive dust.

The treatment of passenger car drum brake linings is generally accomplished

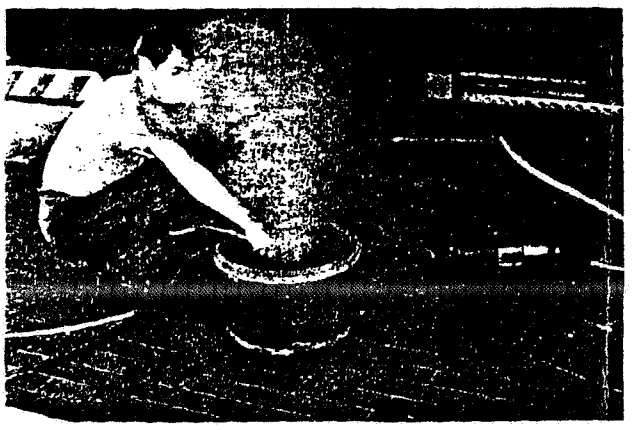

Figure 2. Blowing of the asbestos dust-containing abrasion dust out of the brake drum of a city bus with compressed air. A fine dust half mask is worn. Large amounts of chrysotile elementary fibers with a length of <2 μm are characteristic for this abrasion dust. Probe measurements performed at the mechanic's head level show that the dust cloud can contain up to about 10^9 chrysotile fibers/m^3 of all lengths on electron microscopic evaluation, although at the same time only $2 \cdot 10^6$ chrysotile fibers/m^3 with L > 5 μm are measured [8]. Blowing out or dry brushing out of brake drums was seen in approximately 1 out of every 2 shops. Because the dust release is fairly brief, the asbestos exposure is relatively low. According to optical microscopic evaluations, for each vehicle it corresponds to about 4 to $6 \cdot 10^6$ fibers/m^3 over a period of 1 min or 10^6 fibers/m^3 over 4-6 min. In the case of a respiratory air volume of 20 ℓ/min, this asbestos dust exposure leads to a count of about 80,000 to 120,000 inhaled fibers with a length L > 5 μm per vehicle.

by regrinding with brake lining grinding machinery or by hand (Figure 1). On the other hand, truck and bus brake linings are mainly processed using brake lining circular lathes. In addition to brushing out with a large or small brush, the dust is mainly removed with compressed air (Figure 2). We are currently conducting field studies, partly within the framework of a project supported by the Federal Ministry for Research and Technology (BMFT), aimed at estimating the asbestos-related health risk in motor vehicle shops. In comparison to earlier

studies [2, 5, 17], a distinct decrease in working processes involving the risk of asbestos dust exposure was found [15].

This reduction is attributable to the use of model-correct manufactured parts which is customary today. As a result, the working processes on brake linings that were previously necessary, such as cutting to size, breaking of edges, and drilling of rivet holes, have been eliminated. Whereas rotary grinding was previously common, see Figure 1, today remachining of the brake linings is preferred, at least in the case of trucks and buses.

The asbestos dust dosimetry is of principal significance in the risk estimation. The dust measurements that we performed for this purpose show that in the case of a sampling time of between 0.5 and 3 min in the case of blowing out of brake units, peak concentrations of up to $20 \cdot 10^6$ chrysotile fibers/m^3 a length >5 µm occur in the breathing area of the employees, see Figure 2. The asbestos dust exposure in the case of the characteristic working processes of the cleaning of brakes and the processing of the brake linings is preferentially described by the mean value of the asbestos fiber doses, determined by optical microscopy, as the product of the measured fiber concentration and the duration of sampling [8]. For passenger cars, both during blowing out and during grinding the product of the sampling time and the concentration is observed at about $4 \cdot 10^6$ fibers/$m^3 \cdot$ min corresponding to a 4 min concentration of 10^6 fibers/m^3. For trucks and buses this product amounts to about $6 \cdot 10^6$ fibers/$m^3 \cdot$ min for blowing out, $8 \cdot 10^6$ fibers/$m^3 \cdot$ min for machining, and $14 \cdot 10^6$ fibers/m^3 for grinding. These fiber doses are regarded as low in comparison to the currently permitted maximal asbestos dust exposure upon maintaining the industrial guideline concentration of 10^6 fibers/m^3 per working shift for 480 min, or $480 \cdot 10^6$ fibers/$m^3 \cdot$ min.

Electron microscopic analyses sometimes give higher asbestos fiber dose values. Especially in abraded dust, more than 99% of all chrysotile fibers having a length of <5 μm are seen. Because their diameter is too small, these fibers are not accessible to routine optical microscopic detection [17]. Thus exposure to asbestos-containing respirable dusts exists both during the processing of new linings and during unprotected removal of the abraded dust, for example with compressed air. Since chrysotile detection was also accomplished for most fibers in the abrasion dust using refined analytical methods (energy-dispersive x-ray microanalysis, electron diffraction) [16, 17], it is no longer possible to assume complete mechanical or thermal transformation of the asbestos fiber fraction of the linings, e.g., to forsterite [5], see legend to Figure 2.

Consequences of Asbestos Inhalation in the Auto Repair Trade

Epidemiologic investigations on the consequences of asbestos inhalation in the auto repair trade were already published in the 1970s by the working group of Selikoff [10]. In more than one-fourth of the individuals employed for at least 10 years, appreciable radiologic changes in the lungs or pleura in the sense of at least a questionable beginning asbestosis were described. Boillat et al. [1] in 30 mechanics employed in the auto brake business observed only one case of a radiologically questionable beginning asbestosis.

A cross sectional study performed in West Germany in the early 70s as a pilot study on 116 motor vehicle mechanics who had been engaged for more than 10 years in the repair of brakes, among other things, presented findings in two instances in which asbestos inhalation consequences in the sense of asbestosis had to be assumed with the degree of probability necessary for the area of legislated accident insurance. However, in both cases obesity and chronic bronchitis

with compression effect and increases in peribronchitic marking were present as competing causes [20].

Within the framework of our BMFT research project, more reliable results are now being sought with an expanded examination program on a larger group. Mesotheliomas in conjunction with friction layer processing have already been reported in the international literature. Thus Otto [13] published the case of a 50-year-old mechanic who developed pleural mesothelioma after years of installing frictional layers. An additional case of illness was described by Langer [9]. A 55-year-old man, who mainly repaired brake systems as a motor vehicle mechanic from the age of 19, died from a diffuse pleural mesothelioma. Likewise Greenberg et al. [3] published a case of mesothelioma in a motor mechanic.

McDonald et al. [11] counted the number of mesothelioma cases which occurred during several years in North America and the auto repair sector at a total of 11. Nicholson et al. [12] estimate that 115 mesothelioma cases can be expected in the USA in 1987 among the 1.1 million motor vehicle mechanics (1975)

Details of Our Own Case Observations

Among the 4 patients whom we observed with diffuse malignant pleural mesotheliomas, the first occupational contact always took place at the age of 14 to 18 years ($\bar{X} \pm s = 15.3 \pm 1.9$ years), i.e., always at a young age, see Table 1. The following should be considered for occupational protection of young individuals: Peto [14] determined from epidemiologic data that the incidence of mesothelioma at a given asbestos fiber dose increases by the third or fourth power as a function of time since the first asbestos dust exposure, i.e., the risk duration. This depends on whether the exposure was short term, i.e., took place within a few years, or was continuous.

TABLE 2. HISTOLOGY, THERAPY AND SURVIVAL TIME IN FOUR MOTOR VEHICLE WORKERS WITH DIFFUSE MALIGNANT PLEURAL MESOTHELIOMA.

Patient No.	Histological differentiation	Therapeutic measures	Survival time (months) after beginning of symptoms	Survival time (months) after confirmation of diagnosis
1	epithelial	partial pleurectomy, partial lobectomy	8	3
2	bivalent	chemotherapy	16	9
3	epithelial	symptomatic	11*	8*
4	epithelial	partial resection, Telecobalt irradiation	9	2
$\bar{X} \pm s$			11.0 ± 3.6	5.5 ± 3.5

*Death by suicide.

Using the example of a school contaminated with spray-on asbestos, Peto calculated that an asbestos dust exposure between the ages of 12 and 18 can already lead to one mesothelioma among 100,000 students at a concentration of only 3000 fibers/m^3. The number of mesotheliomas would increase in a linear fashion at higher concentrations. This fact should lead physicians to carefully note and apply the specifications of the youth employment protection laws for activities involving exposure to carcinogenic asbestos materials. The duration of the occupational asbestos dust exposure of our patients was between 11 and 35 years ($\bar{X} \pm s = 22.8 \pm 10.5$ years). At that time the processing of the frictional layers was performed in the typical fashion. Thus the abrasion dust was generally removed with compressed air, without suction. The result was especially high fiber concentrations. One patient (No. 4), as a vehicle electrician, worked for 11 years under exposure through adjacent workplaces in the

sense of a so-called bystander exposure. As early as 1976 Rohl et al. [17] pointed out that several minutes after blowing out a brake drum with compressed air at a distance of up to 25 m, an increased asbestos concentration was still detectable by optical microscopy. This should be taken under consideration in occupational medicine, since in individual instances the inhalation of relatively low dust doses can apparently cause a mesothelioma.

The mesothelioma cases which we presented among 4 auto repair workers appeared after a latency time of 26-37 years ($\bar{X} \pm s = 32.3 \pm 4.9$ years). In a larger, comparable group of patients, the median value for the latency time was 30 years. We speak of the so-called 30 year rule [22].

Regardless of therapeutic measures applied, in all cases the disease resulted in death within a short time, see Table 2.

In the literature, the survival times from the onset of symptoms are likewise given as only about 10 months for half of the patients [6].

In 3 patients, the occupational disease recognition process was started as a result of the Occupational Disease Report to Item No. 4105 of the Occupational Disease Ordinance, legally precribed for each physician ("Asbestos-induced pleural and peritoneal mesothelioma"). In one instance the report was made by the health insurance agency. Up to the present time 3 of the 4 mesothelioma cases have been recognized as compensable cases of occupational cancer. However, the so-called lifetime recognition of compensation did not take place. In the fourth case the findings of the Workers' Association are not yet complete.

Our case histories show that illnesses due to diffuse malignant mesothelioma are also to be expected in the motor vehicle repair trade. However, more accurate estimation of the risk does not seem possible, on one hand because of selection effects and on the other hand because of the expected unreported cases.

It should be emphasized again that the low fiber doses measured today can not necessarily be considered representative of the workplace situation 20, 30 or 40 years ago. Instead it is necessary to ask whether the high fiber concentrations demonstrated in Figure 1 for the regrinding of frictional layers even if suction is used, together with other processing methods used much more frequently in the past, have not led to a much greater asbestos dust exposure.

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