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Small opacities among dental laboratory technicians in Copenhagen

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ABSTRACT After a case of advanced pneumoconiosis occurred in a dental laboratory technician, 31 other dental technicians and 30 control subjects controlled for smoking habits, sex, and age were investigated. More technicians (55%) than controls (30%) had at least grade 1 dyspnoea ($p > 0.05$). Multiple regression analysis showed that 13 technicians who had produced dental prostheses for at least 15 years had consistently lower lung function (FVC, FEV_1, FEV_1/FVC, MEF_{50}, and DCO single breath), although the differences were not statistically significant. All mean lung function values for technicians and controls were within normal limits. Increases in MEF_{50} after breathing 80% helium and 20% O_2 failed to show small airways dysfunction among the technicians. Of the six with radiological pneumoconiosis (5 simple, 1 advanced) four had symptoms. All three biopsy specimens showed varying degrees of pulmonary fibrosis. DCO single breath was diminished in four of the six. One male dental technician had scleroderma and possibly Erasmus syndrome. Blind readings showed an increased number of suspicious chest *x* rays films (⩾ category 0/1) among older smokers and ex-smokers ($p = 0.013$) regardless of occupation. Our results support other evidence that dental technicians are at risk of developing pneumoconiosis. Therefore, adequate hygienic control of dental laboratories is indicated.

Dental laboratory technicians may be exposed to several potentially toxic materials. Non-precious metal alloys containing chromium, cobalt, molybdenum, beryllium, or nickel are used in the production of crowns, bridges, and dentures.[1 2] Liners with asbestos may be used in casting procedures and methylmethacrylate in the preparation of dentures.[3] Silica is used as a sandblasting abrasive and is also present in porcelain. A powder containing 75% silica is sometimes used as refractory investment material for casting procedures.[4] Polishing materials may contain iron, aluminium, silica, and chromium. Pneumoconioses have been described in dental technicians caused by various presumed agents: beryllium,[2 5] silica,[4 6] and metals (cobalt, chromium, tungsten, molybdenum) with or without silica.[7-11] Analysis of lung biopsy specimens has shown the presence of various metals and silica.[5 7 8 11]

Several epidemiological studies have been carried out to investigate the prevalence of pneumoconiosis and lung function abnormalities among dental technicians. Lob and Hugonnaud described suspicious chest *x* ray changes suggestive of hard metal pneumoconiosis in five out of 24 subjects.[7] Of the 70 dental technicians studied by Kronenberger *et al*, 53 had chest *x* ray films showing reticulonodular interstitial markings: 26 delicate (category 0), 25 distinct (category 1), and two pronounced (category 2).[9] The *x* ray films were read according to ILO u/c classification of pneumoconiosis, 1971. When lung function among this group of 70 was compared with 70 control subjects there was no significant difference with regard to inspiratory vital capacity, FEV_1, or DCO single breath. Only PaO_2 was significantly lower among dental technicians.[12] Eight of 178 dental technicians studied by Rom *et al* had simple pneumoconiosis (category 1/0 or 1/1).[2] The only significant difference in lung function, compared with a control group of 69, was an association between exposure-years and reduced FVC and FEV_1 for non-smoking male technicians.

A 37 year old dental technician with advanced silicosis was studied at Bispebjerg Hospital, Copenhagen.[4] After this index case, an epidemiological study of dental technicians in the greater Copenhagen area was performed. We were interested in defining the prevalence of pneumoconiosis as well as investigating the presence of small airways disease (subtle dysfunction of airway obstruction) using flow volume curves after helium-O_2 (HeO_2) breathing. An attempt was

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also made to determine which job process was particularly hazardous.

Materials and methods

STUDY SUBJECTS

Subjects (n = 31) were selected from membership lists of the Danish Dental Technicians Union using the following criteria: (1) living in the greater Copenhagen area, (2) minimum of five years employment as a dental laboratory technician with exposure to silica, and (3) no previous employment in a dusty job other than as a dental technician. The mean duration of employment was 20·1 years (range: 6–41). Five had been employed for six to nine years, 12: 10–19 years, seven: 20–29 years, and seven: 30 years or more. Control subjects (n = 30) were administrative or office workers from Bispebjerg Hospital who had never been employed in a dusty job. The group was controlled for smoking habits, sex, and age. The five ex-smokers were reclassified: three as non-smokers (<4 pack-years) and two as smokers (>9 pack-years). Cigars (5 g tobacco/cigar) and cheroots (3 g tobacco/cheroot) were translated to cigarettes (1 g tobacco/cigarette) (table 1).

MEDICAL QUESTIONNAIRE AND TESTS

A self administered questionnaire containing standardised work histories, the WHO standardised dyspnoea questions,[13] and the British Medical Research Council's standardised chronic bronchitis questions[14] was sent to all participants and subsequently checked in a personal interview. All participants had full sized chest *x* ray films classified according to the 1980 International Classification of Radiographs of Pneumoconioses[15] by four readers (2 chest physicians, 1 radiologist, 1 specialist in occupational diseases) in a blind fashion.

Hewlett-Packard's pulmonary calculator system 47804A with pneumotach was used to measure flow volume curves and DCO-single breath (SB) controller 47305A to measure diffusing capacity (transfer factor). Daily calibration was performed using separately atmospheric air and a mixture of 80% helium and 20% O_2. The best of a minimum of three flow volume curves was chosen. To test density dependence, flow volume curves were measured immediately after three vital capacity inspirations of 80% helium and 20% O_2 (HeO_2). FVC breathing air and FVC breathing HeO_2 had to agree within 5%.[16] DCO-SB was performed before HeO_2 breathing because He was also used in DCO-SB calculations. Maximum expiratory flow volume (MEF) curves with air and HeO_2 were used to measure flow at 50% FVC (MEF_{50} air and MEF_{50} HeO_2). The percentage difference (ΔMEF_{50}) was calculated as follows:

$$\Delta MEF_{50} = \frac{MEF_{50}\,HeO_2 - MEF_{50}\,air}{MEF_{50}\,air} \times 100$$

Volume of iso flow could not be measured as data were derived on line by computer analysis. Predicted values (tables 2 and 3) were obtained from Knudson *et al*[17] (spirometry) and Kanner and Morris[18] (DCO-SB). Statistical methods included the one tailed *t* test, multiple linear regression, and the chi-squared test.

Table 1 *Comparison of dental technicians and control subjects*

	Control subjects	*Dental technicians*
Men	18	18
Women	12	13
Smokers	23	23
Non-smokers	7	8
Mean pack-years for smokers	24·0	23·3
Mean age (years)	43·2	42·1
Mean height (cm)	172·6	172·9
Mean weight (kg)	71·0	70·1

Results

SYMPTOMS AND LUNG FUNCTION

There were no significant differences between dental

Table 2 *Description of six dental technicians with simple pneumoconiosis*

No	*Age*	*Total years employed (years producing chromium-cobalt prostheses)*	*Sex*	*Smoking pack-years*	*Pulmonary function values (% prediicted)* FVC	FEV$_1$	FEV$_1$/FVC	DCO-SB (ml/min/mm Hg)
1	60	38 (5)	M	53	3·29 (73)	2·05 (58)	0·62 (78)	17·35 (69)
2	39	14 (10)	M	32	5·39 (115)	2·75 (75)	0·52 (63)	22·58 (84)
3	48	23 (23)	M	50	3·71 (71)	2·75 (66)	0·74 (92)	11·34 (37)
4	41	17 (17)	M	8	3·40 (83)	1·98 (61)	0·58 (70)	15·43 (65)
5	61	41 (35)	F	45	3·50 (112)	2·00 (80)	0·57 (70)	12·37 (61)
6	46	29 (29)	M	45	3·75 (80)	2·53 (67)	0·68 (83)	22·79 (83)
Mean ($\bar{x}$)	49	27 (20)		39				

Table 3 *Multiple regression analysis of estimated difference in lung function between control subjects and dental technicians (DT) with < 15 years or ≥ 15 years employment with production of prostheses (all types). The mean lung function of DTs with ≥ 15 years employment is arbitrarily set at 0*

	Mean estimate of lung function		
Parameter	*Control group (n = 30)*	*DTs with < 15 yrs (n = 18)*	*DTs ≥ 15 yrs (n = 13)*
FVC (l)	0·11	0·25	0
FEV_1 (l)	0·21	0·15	0
FEV_1/FVC (%)	3·73	0·76	0
MEF_{50} air (l/sec)	0·47	0·48	0
DCO-SB (ml/min/ mm Hg)	2·61	3·60	0

All p values > 0·05.

technicians or control subjects in respect of the prevalence of chronic bronchitis or dyspnoea. Eight of the 30 (27%) dental technicians and six of the 28 (21%) controls had chronic bronchitis whereas 16 of the 29 (55%) dental technicians and eight of the 27 (30%) controls had grade 1 dyspnoea ($p > 0·05$).

The results of the lung function studies are presented in table 3. Multiple regression analyses were controlled for age, height, sex, and smoking habits. The 13 dental technicians who had been employed at least 15 years producing all types of prostheses (gold or chromium cobalt) had consistently lower lung function. Nevertheless, no significant differences were found. The mean improvement in flow after HeO_2 breathing ($\triangle MEF_{50}$) was 13% (1 SD = 14·75) for all dental technicians and 14% (1 SD = 15·95) for all controls. All mean lung function values were within normal limits.

X RAY FINDINGS

Four of the 31 dental technicians and one of the 30 control subjects had small opacities (≥ category 1/0) with agreement among three of the four readers ($p > 0·05$). The chest *x* ray films were read independently in a blind fashion. It was accepted that one of the three positive readings for one dental technician was 0/1.

Table 4 *Smoking habits and suspicion of small opacities (≥0/1) among 31 dental technicians and 30 control subjects without regard to occupation*

	Normal	*Suspicion of small opacities*	*Total*
Smokers and ex-smokers	27	22	49
Non-smokers	11	1	12
Total	38	23	61

p = 0·0219.

Table 5 *Age and suspicion of small opacities (≥0/1) among 49 smokers and ex-smokers, dental technicians, and control subjects without regard to occupation*

Age	*Normal*	*Suspicion of small opacities*	*Total*
16–39	19	6	23
40–62	10	16	26
	27	22	49

p = 0·013.

There was a significantly increased number of smokers and ex-smokers with suspicious chest *x* ray films compared with non-smokers (table 4). An *x* ray film was classified as suspicious if one or more of the readers classified it as ≥0/1 (blind readings). In a similar manner one could demonstrate a significantly increased number of suspicious *x* ray films among older smokers and ex-smokers without regard to occupation (table 5).

Of the four dental technicians with small opacities (agreement among 3/4 readers) two were symptomatic: subjects 1 and 6 in table 2. Subject 4 had an advanced pneumoconiosis (3/3 r B) for which he received compensation. Subject 5 had a positive ANA, abnormal bronchoalveolar lavage (23% lymphocytes), increased lung gallium-67 index: right = 0·51 and left = 0·53 (normal 0·28),[19] and a transbronchial lung biopsy showing non-specific lung fibrosis with some birefringent particles observed by polarised light microscopy. Two other dental technicians had radiological pulmonary fibrosis diagnosed by the radiology department (with only one or two of our readers classifying the *x* ray films as ≥ 1/0). Subject 2 had a spontaneous pneumothorax. Lung biopsy specimens taken during pleuroscopy showed PAS positive and iron positive particles within alveolar macrophages and some thickening of the alveolar walls. Subject 3 had diffuse scleroderma with advanced fibrosis, proliferation of blood vessels, and some birefringent particles shown by open lung biopsy. Table 2 gives the smoking habits, work histories, and lung function test results for the six dental technicians with radiological pulmonary fibrosis. Diffusing capacity was diminished in four of the six but none had radiological changes of emphysema.

The six dental technicians with simple pneumoconiosis (19·4%) were mainly employed in the production of chromium cobalt prostheses (table 2). None of the 12 dental technicians who had never worked at this process had simple pneumoconiosis. Of the eight workers with less than 10 years employment producing chromium cobalt prostheses ($\bar{x} = 5$), one had simple pneumoconiosis (12·5%). Of the 11 workers with $\geqslant 10$ years experience producing these prostheses ($\bar{x}$ = 19·6), five had simple pneumoconiosis (45·5%).

Discussion

There is now sufficient evidence to conclude that dental technicians are at risk of developing pneumoconiosis.[20] Apparently silica, beryllium, chromium, or cobalt may be causative agents. Our index case used Multivest powder (50% quartz and 25% cristobalite) as a refractory investment material for casting procedures.[4] He used as much as 6 kg daily for six years without ventilation. Percutaneous lung biopsy showed iron positive material that could not be identified by light microscopy and advanced interstitial fibrosis. Unfortunately lung tissue was not studied in either our index case or the subjects in the epidemiological study by scanning electron microscopy to define its particle content.

A precise exposure history for our dental technicians was difficult to define. Most were employed at small laboratories where it was necessary to grind and polish as well as produce prostheses. Thus there was a mixed exposure to silica and methyl methacrylate in addition to various metals. Unfortunately, no air sampling was undertaken to define exposures quantitatively.

We were able carefully to match our study subjects with a control group (table 1). Almost twice as many dental technicians (55%) had at least grade 1 dyspnoea compared with the controls (30%). This difference, however, was not statistically significant, possibly due to insufficient numbers. Rom *et al* could not show excess symptoms among dental technicians.[2] Study subjects with a minimum of 15 years producing prostheses had consistently lower lung function (table 3). There were, however, no significant differences. All mean spirometric parameters and DCO-SB for controls and the exposed group were normal as in Rom's study.[2]

Measurements of maximum expiratory flow (MEF) while breathing gases of varying density (air versus HeO_2) has shown small airways dysfunction in smokers.[21] Flow volume curves after HeO_2 breathing have been used to investigate the effect of hair spray[22] and cotton dust[23] on small airways. Nevertheless, the pathological correlation with density dependence of MEF may be poor.[24] The mean per cent increase in flow after HeO_2 breathing ($\triangle$ MEF_{50}) was almost identical in our two groups: dental technicians (13%) and controls (14%). Thus we could not show the presence of small airways disease using this technique. The procedure used in measuring MEF immediately after HeO_2 breathing was extremely difficult. It was often difficult to obtain FVC breathing air that agreed with 5% of FVC breathing HeO_2. We agree with Knudson *et al* that HeO_2 breathing with $\triangle$ MEF_{50} is not a suitable screening tool under field survey conditions.[16]

We were not able to show a statistically significant increase in the prevalence of simple pneumoconioses ($\geqslant$1/0) in dental technicians (4/31 = 12·9%) compared with controls (1/30 = 3·3%) with agreement among three or four readers. A prevalence of 12·9%, however, is much higher than is found in the general population ($\leqslant$3·7%).[25] Our dental technicians had an increased relative risk of developing small opacities: 12·9/3·3 = 3·9.

Surprisingly, the prevalence of suspicious chest *x* ray films ($\geqslant$0/1) was significantly higher among older smokers (tables 4 and 5) without regard to occupation. This observation among smokers is in opposition to the accepted opinion that cigarette smoking does not produce radiological pulmonary fibrosis,[26] although there may be synergism between asbestos and cigarette smoking.[25] These data, however, should be interpreted cautiously as "suspicious chest *x* ray films" is an uncertain evaluation despite blind readings.

Of the six dental technicians with simple pneumoconiosis (subject 4 had also advanced changes) four were symptomatic (table 2). All three lung biopsy specimens showed varying degrees of fibrosis. The pattern of fibrosis and its specific aetiology could not be determined. The production of chromium cobalt prostheses, however, appears to be the most hazardous job process. The Erasmus syndrome (association of scleroderma and pneumoconiosis) has been described in a dental technician[10] and it is possible that subject 3 had this syndrome. It is important that all products used in dental laboratories are properly labelled and that dental technicians are well informed about the potential hazards. The experience now gathered concerning pneumoconiosis among dental technicians indicates the need for effective ventilation of these laboratories and the substitution of dangerous materials when possible.

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