EFFECTS OF INHALATION OF AIRBRASIVE POWDER

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WITH the introduction of the airbrasive technic for preparation of cavities in teeth, it was realized that both patient and operator would be exposed to airbrasive powder, which, according to the manufacturer, was of the following composition: $Al_2 O_3$, 94.56 per cent; $Si O_2$, 2.23 per cent; Fe O, 0.41 per cent; Ca O, trace.

Numerous reports expressing a wide variation of opinions as to the effect of the inhalation of alumina dust have appeared. Many of the animal experiments reported are inconclusive and the clinical and autopsy findings, although suggestive of specific changes, do not present positive agreement as to the toxicity of alumina.

Because inhalation of any dust, especially one containing silica, may cause pneumoconiosis and because of the conflicting reports on the effect of alumina dust and the presence of silica in the airbrasive powder, it seemed desirable to pursue this study.

Industrial dusts are commonly classified into 3 groups based upon tissue reaction to the dust[1-3]: (1) dusts which are absorbed without appreciable tissue alteration or damage; (2) dusts which remain inert within the living tissues, neither being absorbed nor causing proliferative changes; and (3) dusts which irritate and initiate cellular proliferation, fibrosis, and retrograde changes. Groups 1 and 2 are referred to as harmless dusts and Group 3 as harmful dusts, but all types of dusts may interfere with normal respiration and cause bronchial irritation or obstruction if they are inhaled in large enough quantities. The present knowledge of the biologic effects of aluminum-oxide dust is based upon animal experimentation, clinical findings in human beings, and a few autopsy reports.

Since Arnold[4] introduced the technic of exposing animals to dust in dust chambers, this method has been widely used for investigation of the biologic effects of various types of dust. Most of these investigations have been concerned with the effect of various types and mixtures of silica and other related industrial dusts, but few experiments have been reported on aluminum-oxide and hydroxide.

Haynes[5] exposed guinea pigs in dust chambers to $Al_2 O_3$. His animals demonstrated pulmonary fibrosis after 9 weeks of exposure. The fibrosis was

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progressively more severe in those having longer exposure. The fibrosis decreased in the animals after exposure was terminated and was minimal in those sacrificed after a long period. Denny, Robson, Irwin[6] exposed rabbits to aluminum powder for 14 months. The lungs contained 270 to 1,200 mg. of powder, but the animals showed minimal clinical and histologic change. Jötten and Eickhoff[8] reported the most extensive dusting experiment using aluminum-oxide. Dusting was heavy and most of the animals died of dust exposure. Marked pulmonary changes were produced, but the findings were not consistent for all animals. The observed changes were thought to be due to "aluminosis." The changes were similar to those described in human beings by Kahlan.[23] Injection studies with $Al_2 O_3$, reported by Gardner and Cummings[10] showed the aluminum-oxide was phagocytized and incapable of producing fibrosis after 2 to 3 years.

Human beings have been studied by Sutherland, Meiklejohn and Price,[12] Doese,[13] Goralewski,[14-18] Shaver and Riddell,[19] and Shaver.[20] Sutherland, Meiklejohn, and Price and Doese could find no specific changes produced by alumina, but Goralewski, Riddell, and Shaver demonstrated definite clinical and roentgenologic findings. Autopsy findings attributed to alumina were described by Kirch[22] and Kahlan.[23]

The mechanism of irritation in the various types of pneumoconiosis is not well established. The chronicity and the nonspecificity of the pneumoconioses and the individual variation in response from one species to another, and from one organ to another, variation to response to the particle size, to chemical and physical characteristics of the material, to type of exposure, to length of exposure, are all important factors in the study of this group of diseases. A well-controlled experimental study or clinical observation requires a consideration of all these factors which makes such investigations very difficult. Furthermore, an interpretation of the materials which are influenced by so many variable factors will tend to be subjective, especially with reference to clinical observation, and to the evaluation and selection of limited pulmonary fields suitable to prove or disprove the theories of the observer. The reported experimental investigations with $Al_2 O_3$ are of limited significance, mainly due to unsatisfactory correlation of the numerous mentioned variants. Aluminum-oxide with particle size larger than 1 to 3 microns seems to be relatively inert in guinea pigs and rabbits. Pulmonary lesions of aluminosis were experimentally produced by Jötten and Eickhoff in rabbits, but one cannot be sure that the dust which they utilized was pure aluminum-oxide, since it was factory dust, which had not been analyzed. The same objection may be made to the autopsy cases of aluminosis where the individuals had been exposed to factory dust, and the autopsy cases which showed a relatively high percentage of silica present, so no conclusion can be made as to the specific effect of the aluminum-oxide. One very important factor is present in all of the reported cases of aluminosis—the small particle size. The cases of aluminosis in German factories developed after the particle size of the dust was reduced 10 times and the particle size of the fumes in the bauxite factory was also extremely small.

It was found by van Wijk and Patterson[30] that about 25 per cent of particle size 0.2 micron is eliminated by breathing. About 80 per cent of particle size 2 micron is eliminated and at particle size of 5 micron, 95 per cent of the particles are eliminated. Hamlin[31] also stated that very few particles larger than 10 micron will reach the alveoli of the lung.

The literature indicates that the presence of aluminum will not give complete protection against silicosis nor against the silica particles in the dust, so any type of dust where contamination with silica is present should be considered potentially harmful.

METHODS

The methods used to investigate the effect of inhalation of airbrasive powder were as follows:

1. Intraperitoneal injection of 1 Gm. of sterile airbrasive powder in 2 rabbits. (All powder used was of the type used for the preparation of cavities. None of the powder was of the type used for prophylaxis.) Animals were sacrificed at 120 days.

2. One-half gram of sterile airbrasive powder was blown into the trachae of 2 rabbits by mouth pressure. The animals were sacrificed after 207 days.

3. Methods 1 and 2 were found to be unsatisfactory and were discontinued in favor of the following technic: Animals were placed in 112 L. (approximately 30 gal.) barrels having a tight fitting cover which had 3/8 inch holes in either side of the top. A 3/8 inch rubber hose protruded just through the top on one side. This was connected to a 2 L. suction flask which contained airbrasive powder. The top of the flask was fitted with a cork carrying a single glass tube 3/16 inch in diameter. This tube reached nearly to the bottom of the flask and was attached by a rubber tube to the compressed air line. The air pressure was adjusted to provide enough force to blow the powder into the barrel in a constant fine stream. The powder on entering the barrel was dispersed throughout the area as a uniform haze which easily permitted observation of the animals in the bottom of the barrel. Through the other opening in the top, a 3/8 inch rubber tube was passed to the bottom of the barrel. This was attached to the compressed air line having sufficient pressure to make the end of the hose describe a small circle. This re-agitated the dust and also provided an adequate supply of air for the animals, without which they would suffocate. Because of the development of static electricity, the barrels must be grounded (Fig. 1).

This method, called dusting, provided an atmosphere with an intense continuous supply of airbrasive powder. Rabbits, guinea pigs, and monkeys were dusted by this method as follows: 16 rabbits were exposed to new airbrasive powder 6 hours per day for intervals ranging from 13 to 22 days. The average exposure time was 200 days. Some animals were sacrificed immediately following dusting, while others were maintained without further exposure up to 500 days (Table I).

Twenty-five guinea pigs were dusted as follows: (a) Six guinea pigs were exposed to 6 hours per day for 15 days with new airbrasive powder. All animals died within 22 days of the initial exposure. (b) Twelve guinea pigs were exposed to new airbrasive powder 1 hour per day for periods ranging from 18 to

TABLE I
GROUP III, RABBITS (6 HOURS EXPOSURE PER DAY)

ANIMAL NO.	DAYS OF EXPOSURE	TOTAL HOURS	DAYS FROM FIRST TO LAST EXPOSURE	DAYS SINCE LAST EXPOSURE	TOTAL ELAPSED TIME
1	13	78	15	0	15
2	33	198	51	0	51
3	33	198	51	0	51
4	222	1,332	305	0	305
5	219	1,314	293	0	293
6	219	1,314	293	0	293
7	219	1,314	293	0	293
9	115	690	155	0	155
10	115	690	155	0	155
11	115	690	155	0	155
12	115	690	155	0	155
13	198	1,188	300	39	339
14	198	1,188	300	348	648
15	198	1,188	300	154	454
16	198	1,188	300	51	351
17	185	1,188	300	165	465

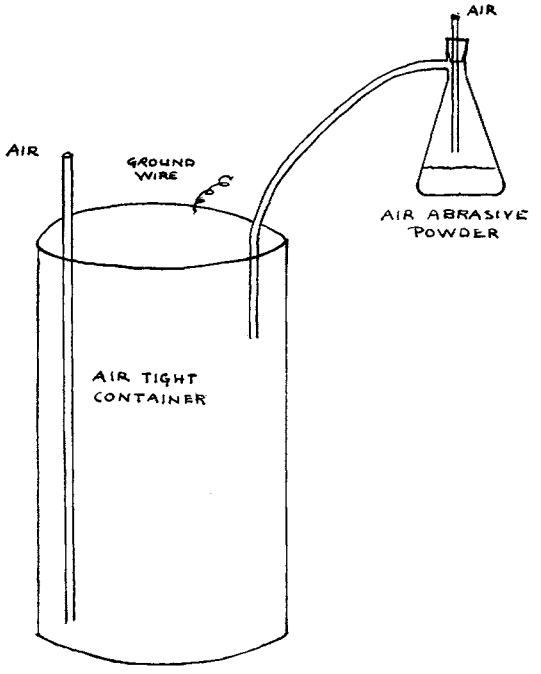

Fig. 1.—Airtight 30 gal. paper barrel (with metal top) which was used as dusting chamber. Air is passed through the block to supply fresh powder. It is also directed through a second inlet to the bottom of the barrel to provide air for animals and to reagitate powder.

205 days. One animal was alive at the end of 475 days. The remainder died at intervals ranging from 22 days to 352 days. (c) Seven guinea pigs were exposed to used airbrasive powder 1 hour per day for 33 days. All animals died in a period of 77 to 85 days (Table II).

Three Rhesus monkeys were exposed in a dust chamber as follows: (a) One was exposed 1 hour per day for 60 days to used airbrasive powder. Time elapsed before sacrifice 94 days. (b) One was exposed 2 hours per day for 60 days to

TABLE II
GROUP IV, GUINEA PIGS

ANIMAL NO.	DAYS OF EXPOSURE	TYPE POWDER	DAYS FROM FIRST TO LAST EXPOSURE	DAYS SINCE LAST EXPOSURE	TOTAL HOURS	TOTAL ELAPSED TIME	
1	15	New	17	0	90	17	
2	14	New	16	0	84	16	
3	15	New	17	3	90	20	6 Hrs. Exp. Per Day
4	15	New	17	3	90	20	
5	15	New	17	5	90	22	
6	15	New	17	3	90	20	
7	18	New	20	0	18	20	
8	23	New	25	5	23	30	
9	25	New	33	0	25	33	
10	19	New	21	0	19	21	
11	20	New	22	0	20	22	
12	27	New	35	0	27	35	
13	27	New	35	0	27	35	
14	205	New	275	77	205	352	
15	205	New	275	35	205	310	
16	195	New	275	1	195	258	
17	205	New	275	35	205	310	
18	205	New	275	348	205	623	
19A	33	Used	44	36	33	80	1 Hr. Exp. Per Day
19B	33	Used	44	41	33	85	
20	33	Used	44	36	33	80	
21	33	Used	44	41	33	85	
22	33	Used	44	33	33	77	
23	33	Used	44	33	33	77	
25	33	Used	44	35	33	79	

used airbrasive powder. Time elapsed before sacrifice 186 days. (c) One was exposed 3 hours per day for 60 days to new airbrasive powder. This animal died at 261 days (Table III).

TABLE III
GROUP V, MONKEYS

ANIMAL NO.	DAYS OF EXPOSURE	TYPE POWDER	HOURS OF EXPOSURE	DAYS FROM FIRST TO LAST EXPOSURE	DAYS SINCE LAST EXPOSURE	TOTAL HOURS	TOTAL ELAPSED TIME
3801	60	Used	1	93	93	60	186
3805	60	Used	2	93	1	120	94
3804	60	New	3	93	168	180	261

At the time of death or sacrifice all animals were autopsied and examined and tissues selected from trachae, lung, heart, liver, kidney, spleen, adrenals, and intestine for microscopic examination. The lungs were cut in cross section from apex to base by means of 6 cuts. Tissue was also selected from any other than the above-mentioned areas if it appeared abnormal. All tissues were prepared by paraffin technic and stained with hematoxylin and eosin.

OBSERVATIONS

Animals, especially guinea pigs, exposed to dust for 6 hours per day developed severe conjunctivitis, hemoptysis, and weight loss. Those exposed for shorter periods appeared to tolerate exposure well. No symptoms were evident and the animals grew and gained weight equal to the control animals.

A summary of the findings for each group of animals follows:

Group I.—Rabbits having intraperitoneal injection of airbrasive powder: At necropsy numerous smooth gray plaques of variable size were present on the surface of the gut and the abdominal wall at the site of injection. These were slender peritoneal adhesions. Microscopic examination of these plaques revealed large quantities of airbrasive powder having an intense foreign body reaction

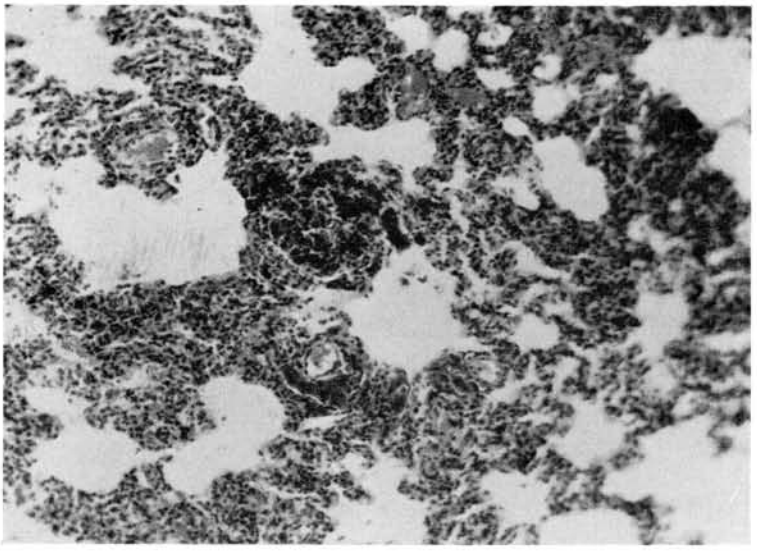

Fig. 2.—Monkey 3805. Focal distribution of airbrasive powder. Thickening of alveolar septa. (H & E stain; ×238.)

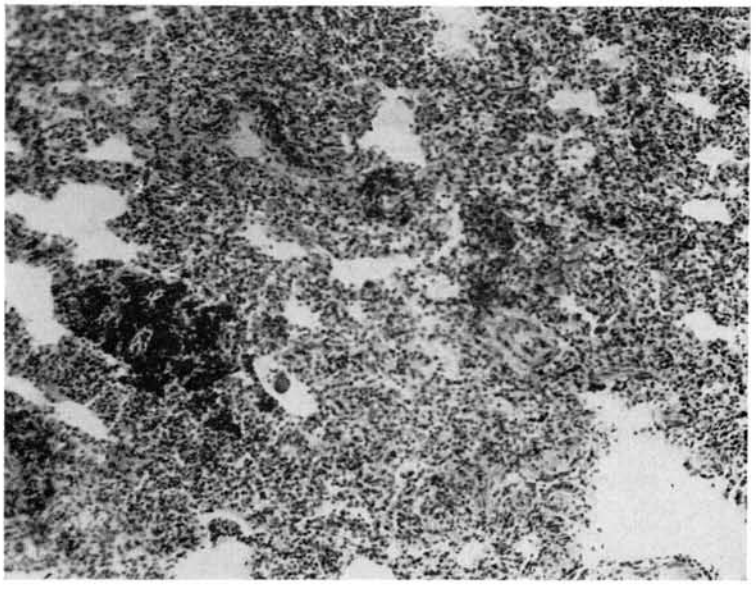

Fig. 3.—Monkey 3805. Consolidation of lung. Active pneumonitis. Focal deposition of airbrasive material. (H & E stain; ×238.)

about the powder particles. Some of the particles were dissociated and appeared as fine black granules. There was minimal evidence of fibrosis and no evidence of necrosis in the surrounding area. The exudative response was minimal.

Group II.—Airbrasive powder blown into trachae: At necropsy the gross changes were minimal except for moderate congestion. When the lungs were

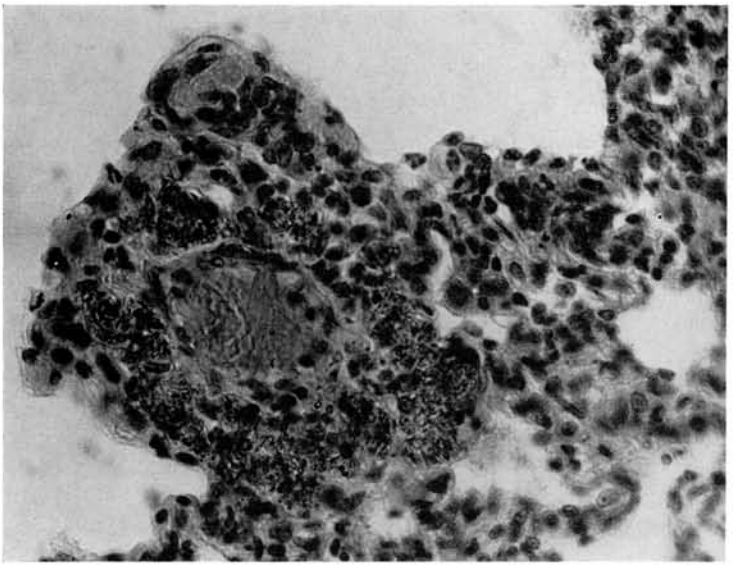

Fig. 4.—Monkey 3805. Perivascular deposition of airbrasive powder. Phagocytized particles and crystals in alveolar septa. (H & E stain; ×840.)

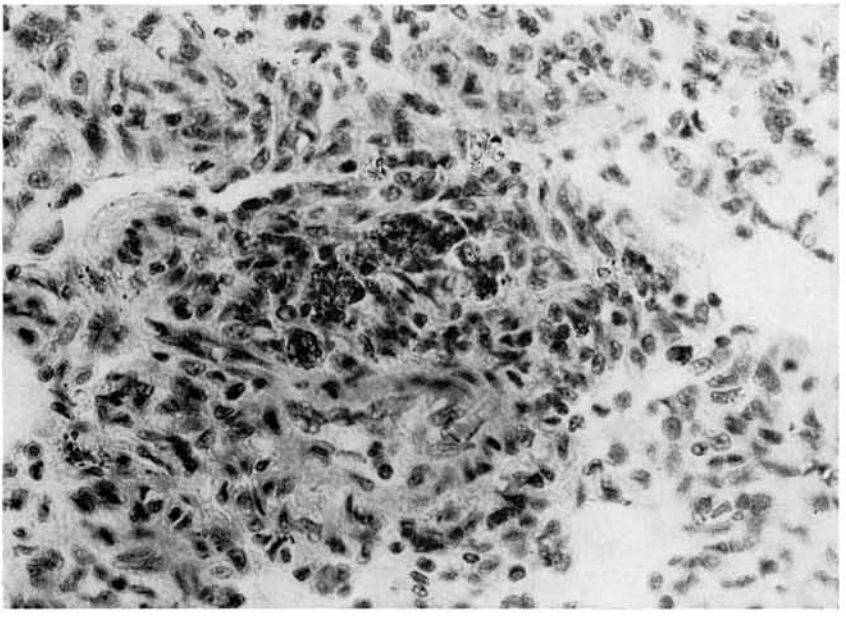

Fig. 5.—Monkey 3805. Airbrasive material with associated fibrosis. (H & E stain; ×840.)

examined microscopically only a small quantity of airbrasive powder was found in the alveolar walls, and this incited only a minimal inflammatory response.

Group III.—Animals exposed in dusting chambers 6 hours per day for varying periods of time: Rabbits. Six of the animals died from suffocation due

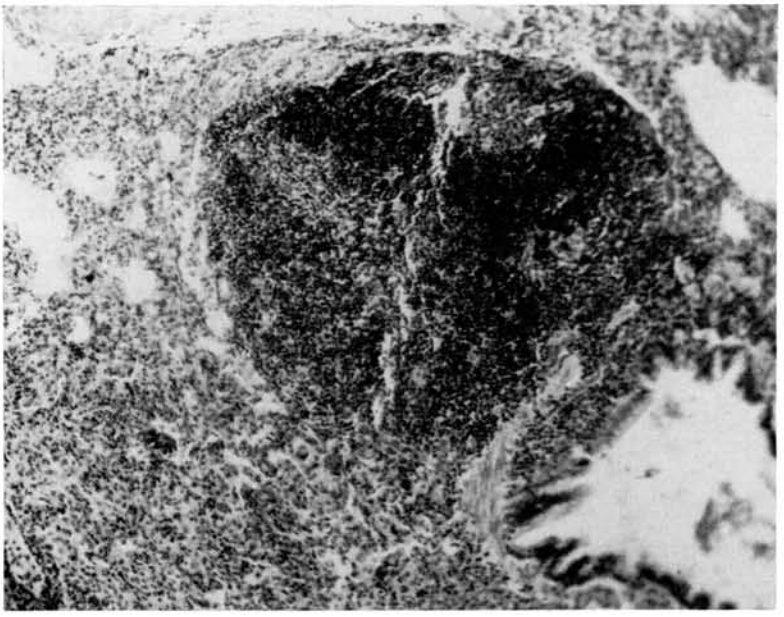

Fig. 6.—Rabbit 4. Hyperplastic lymphoid follicle containing fine airbrasive powder. (H & E stain; ×238.)

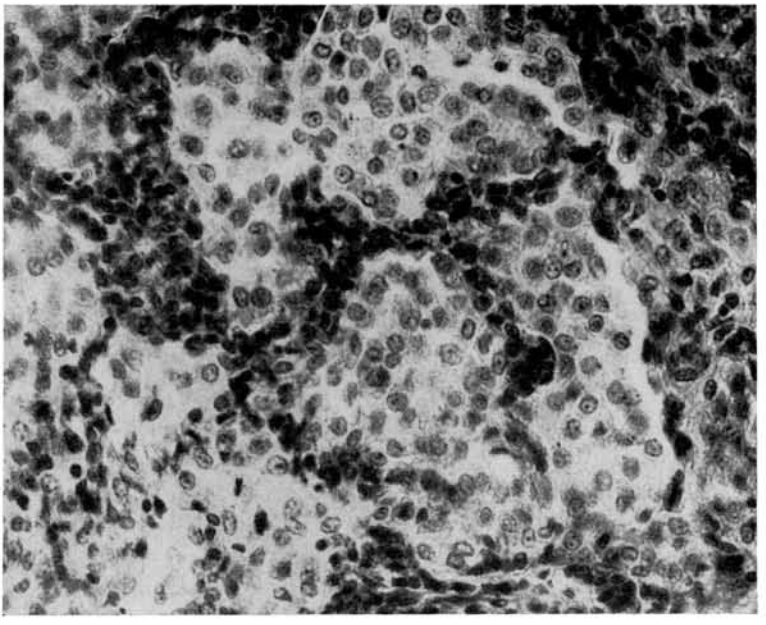

Fig. 7.—Rabbit 4. Granulomatous reaction producing consolidation of lung. Alveoli filled with epitheloid cells. (H & E stain; ×840.)

to the air being shut off, and the others were sacrificed. At necropsy the suffocated animals exhibited only changes of acute oxygen deficiency. The other animals showed acute passive congestion and slight emphysema. Microscopic findings in all animals were intense acute passive congestion, patchy atelectasis and emphysema, pulmonary edema, peribronchial lymphocytic infiltrations with some eosinophils present. Phagocytized dust particles and occasional small crystals of airbrasive material were present (see Fig. 6). The quantity of dust in the lungs was minimal. In the animals exposed for the longer intervals, there was slight fibrosis and slightly more dust present in the lungs.

Group IV.—Guinea pigs exposed in dust chambers: (a) Six animals exposed 6 hrs. per day for 15 days, (b) 7 animals exposed 1 hr. per day for 18 to 27 days, (c) 7 animals exposed 1 hr. per day for 33 days, and (d) 5 animals exposed 1 hr. per day for 205 to 275 days. At necropsy the gross findings were those of congestion, patchy atelectasis, and emphysema. Microscopically, there was evidence of congestion, patchy atelectasis, and emphysema; hyperplasia of rudimentary lymphoid follicles, focal patchy fibrosis, and slight hyaline thickening of basement membrane of bronchioles. There were minimal quantities of phagocytized airbrasive dust. The animals with the 6-hour exposure had a nonpurulent lobular pneumonia and occasional dust crystals in the alveoli. The changes were more severe in the animals receiving the longer exposures.

Group V.—Monkeys exposed 60 days: No significant gross findings were noted at the time of necropsy. Microscopically, there was patchy atelectasis and emphysema; large quantities of phagocytized dust in perivascular and peribronchial areas (Figs. 2, 3, 4, 5); patchy fibrosis (Fig. 3); hyperplasia of peribronchial rudimentary lymphoid follicles (Fig. 6); some crystalline dust in alveoli and peribronchial area and patchy consolidation produced by granulomatous reaction (Fig. 7). Peribronchial lymph nodes contained dust.

STUDIES ON DUST CONCENTRATION IN DENTAL OFFICES

Dust studies were made in 2 dental offices under ordinary working conditions to determine the concentration of dust to which the dentist would be subjected. This study was made by Mr. George Hama of the Bureau of Industrial Hygiene of the city of Detroit.

LOCATION	LIGHT-FIELD COUNT MILLIONS OF PARTICLES PER CUBIC FOOT
1. Dr. D's office, in the breathing zone of dentist using Air Dent.	4
2. (Same location and conditions as sample 1.)	4
3. At discharge of dust collector in Air Dent cabinet. The collector uses an Air-Tec cyclone collector.	13
4. Office of Dr. L, breathing zone of dentist, using Air Dent.	4
5. In the office of Dr. L, general air sample, immediately after using Air Dent.	2

DISCUSSION

All animals showed some reaction to the inhalation of airbrasive material. Those exposed to large quantities developed pulmonary symptoms. A large number of animals died after the dusting period. Rabbits, guinea pigs, and one

monkey all died within a 48-hour period when the temperature was over 100° F. and the humidity high. They died of no apparent reason other than respiratory embarrassment, believed to be due to a reduction in vital capacity. This finding has been observed by other investigators.[8] All animals exhibited some dust in the lung. The amount present was dependent upon the length of exposure time and varied with the type of animal. The rodent type of animals appears to be more resistant to the accumulation of dust than are monkeys. Monkeys with shorter exposure time exhibited a greater quantity of dust in the lungs and a more severe reaction to the dust present. It is apparent from the findings in the lungs of the animals studied that airbrasive powder is toxic when inhaled in large quantities. However, the changes are slow to develop and are not of the severity of those produced by other dusts such as silica.

Because the respiratory system of monkeys is anatomically similar to that of man and because of the apparent natural susceptibility of monkeys to respiratory disease, it appears desirable to use them as experimental animals in studies of this type. More rapid and comparable results could be obtained if monkeys were substituted for the rodent which appears to have a high resistance to pneumoconiosis.

The dust concentration studies in dental offices indicate a low concentration of dust which is below the level considered to be effective in the production of pneumoconiosis, even for dusts containing silica. It therefore appears that the use of the airbrasive technic is not a hazard to the dentist. The airbrasive technic is not used widely and, when used, its application is limited in time. Therefore, exposure is periodic and limited, eliminating the possibility of it being a public hazard even though the dust is toxic.

SUMMARY AND CONCLUSIONS

1. Airbrasive powder can be introduced into the lungs of animals by the dusting technic.

2. The quantity of dust residual in the lung is different for each species and varies within the same species.

3. The rodent type of animals has a high dust tolerance while monkeys have a low dust tolerance.

4. Monkeys are the animals of choice for studies on the effect of dust inhalation.

5. Airbrasive dust in large quantities is capable of producing pulmonary changes which result in reduction of vital capacity.

6. The changes produced are not characterized by fibrosis. They are diffuse rather than nodular and are difficult to produce even with intensive exposure. This would indicate that the material has a low degree of toxicity, even though it can be demonstrated in the lungs in large quantities.

7. Due to the periodic slight exposure obtained by both dentist and patients to a very low concentration of dust, the airbrasive technic does not provide a health hazard.

8. All particles smaller than 3 to 5 microns should be removed from the powder.

9. Although the quantity of silicate is small, it would be desirable to have the airbrasive powder silica free.

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