MINUTES OF THE MEETING

of the

ASBESTOS STUDY COMMITTEE

Friday, June 1, 1973, at 9:30 A.M.
at the
Institute Office, E. 210 Route 4, Paramus, N.J.

MEMBERS PRESENT

I. E. Weaver, Chairman	Raybestos-Manhattan, Inc.
H. Wagner	Carlisle Corporation
E. E. Feierabend	Abex Corporation

OTHERS PRESENT

S. B. McGinnis (for J. C. Henning)	World Bestos Company
M. Jacko (for W. Spurgeon)	Bendix Corporation
D. E. Stone	Bendix Corporation
R. C. Wyatt	Maremont Corporation
W. H. Gustafson	Maremont Corporation
E. W. Drislane	Friction Materials Standards Institute, Inc.

MEMBERS NOT PRESENT

J. C. Henning	World Bestos Company
T. Bell	E. K. Porter Co.
W. Spurgeon	Bendix Corporation

The meeting was called to order by Mr. Weaver, Chairman, at 9:30 A.M.

MINUTES OF PREVIOUS MEETING

The Secretary read a summary of the Minutes of the Meeting held February 16, 1973. These minutes had been released and a motion for their acceptance had been obtained.

Upon motion duly made, seconded and unanimously passed, it was

RESOLVED: To accept the minutes of the February 16, 1973 meeting as distributed.

LABELING

At the February 16, 1973 meeting, the Secretary was directed to distribute information on typical CAUTION labels now in use. The purpose of this distribution was so that the Committee Members could review what is available and would be in a position to propose label specifications to meet the OSHA requirements.

One member suggested that the size of the labeling used should be of sufficient size to be noticeable on a large carton and should be commensurably smaller but

still noticeable on a smaller package. One member was using an insert with a CAUTION label stuffed into the package. The use of imprinted CAUTION labels on the carton is desirable because it is essentially a one-time tooling cost. The use of separate labels is a continuing added direct expense. Most members now using separate gummed labels will eventually go over to imprinting the carton when ordered.

In what must be a response or a reaction by others, many customers are now asking Members about how much percentage of asbestos is in the brake linings. This could be a reaction on the customer's part as to whether they would have to put control practices in their factories because of the asbestos that is contained in brake lining. To meet the true spirit of the OSHA regulations, manufacturers doing subsequent drilling, grinding or cutting of asbestos containing brake linings should use the care that OSHA suggests.

One member felt that where he was shipping drilled ground lining sets that he would not have to imprint these small cartons with the OSHA CAUTION label. Other members are simply putting the OSHA CAUTION labeling on everything. When it was suggested that the Committee should take a position on this labeling requirement, the members of the Committee were referred back to the Resolution that was made on February 16, 1973. This Resolution said, in effect, that OSHA labeling practices should be adhered to where asbestos containing materials do not have asbestos fiber completely locked in or where subsequent operations may be performed. The question concerning the drilled and ground set is: While it is unlikely that subsequent operations will be performed, is it possible that they may be performed?

After a lengthy discussion it was decided that no resolution concerning recommended CAUTION labels would be proposed. Rather, the Secretary is directed to send to the Membership copies of typical labels now in use.

It was called to the Secretary's attention that his yellow Bulletin of March 30, 1973 was in error. In that notice it stated, "Avoid breathing dust". The wording should have been, "Avoid creating dust". This error will be called to the attention of the Membership.

The Chairman brought up another point as regards labels. There is a sign that can be posted in the factory where there are restrictions concerning asbestos dust in the atmosphere. This is a standard sign for placing in the factory which says: "CAUTION - Asbestos dust hazard; avoid breathing dust; wear assigned protective equipment; do not remain in area unless your work requires it; breathing asbestos dust may be hazardous to your health". Information on the availability of these signs will be sent to the Membership.

EPA EMISSIONS STANDARDS FOR ASBESTOS

While the new EPA emssions standards appear to be reasonable, there is some difficulty in interpretation. For example, the standards are not simply "No visible emssions", but (1) if the control equipment does not meet the air cleaning requirements in the regulations, no visible emissions are permissible, or (2) one could even have visible emissions if they were using a collector with the specifications recommended by the EPA. In other words, if you have the EPA's recommended collector you could possibly have visible emissions and still be complying with the EPA requirements. It goes without saying, that interpretation of the requirements by individuals in the different EPA regions may vary quite a bit.

... standards that the wet collector is not as efficient

as the dry-bag collector. If an EPA Enforcement Officer sees a vapor from the stack where a wet collector is used, the source best be able to prove there is no asbestos being discharged. In other words, it can be inferred that if a source has wet collectors they may more likely be cited for visible emissions.

While it is apparent that the EPA's emissions standards promote the dry collection of asbestos in bags, many problems have been indicated with these collectors. One of the problems was repeated fires in the collection system. Another member stated that he too had this problem until cigarette smoking was banned in the factory. Since discontinuing smoking in the factory, he claims they have not had more than one or two fires in the last 25 years. Another member said that may be, but they have had a No Smoking rule for many years and they still have fires. This party blames the fires on the incentive program where the workers receive a bonus for exceeding certain work standards. This promotes the taking of heavier cuts with grinding wheels and creates sparks which apparently promote the fires in the system. The operation that has not had any fires for the past 25 years does not have an incentive system and does not permit smoking in the work place. Where the wet collectors are now in use, apparently the EPA is permitting their use as complying with the requirements.

At this point, the disposal of the materials picked up by the collectors was brought up. One member sent the dust to a pelletizing machine. In this process they add 5%-10% cement to the pelletizer. A volume reduction in the order of 3 to 1 was developed. The pellets are taken by truck and dumped as land fill. While the pellets could be broken down into a powder, if they receive reasonable handling they can be readily moved from the pelletizing machine to the land fill operation. It is this member's intention to install a vacuum system from the collecting areas to go to a central pelletizing machine. One member described his handling of dust from (1) a central collector, to (2) a screw conveyor, to (3) a truck, and to (4) the land fill. The workers in this case use respirators.

The pelletizing operation not only reduces the transportation cost by three times but eliminates the need for a watering truck and an individual to wet down the land fill. However, the costs of this pelletizing equipment are substantial. A manufacturer of pelletizing equipment is Ferro Tech Inc., 1231 Banksville Road, Pittsburgh, Pa. 15216.

Several members mentioned that in dealing with the EPA Regional Offices they were having difficulties deciding what was a "new source" and what was an "existing source". Also, where one manufacturer adds one machine to an existing collection system he may not be in compliance without getting a Waiver of Compliance. Apparently the EPA will not give a Waiver of Compliance that will take more than 12 months to complete. An applicant must give the steps to be taken and the schedule to be met. When each date arrives, the applicant must advise EPA concerning completion of that stage of the schedule.

One member felt that we should review the EPA source report form to get a better understanding of what they were calling for. Page one of the report would be used for each factory. As there would most likely be several points of emission, page 2 would be completed for each stack or collector that emits asbestos.

If a manufacturer wished to make an addition or modification in his plant with equipment that might put asbestos into the atmosphere, he must file with the EPA. On page 1 of the report he would cross off the words "Source Report" and type in either "Application to Construct a New Source", or "Application to Modify Existing Source". In reviewing page 2 of the report under "Process Description", some questions came up as to how to complete this section. One member who had worked on this report with the EPA said you should enter here the type of

machinery used without quantifying. Another member indicated that the EPA insisted that he list the type of equipment and the numbers of each piece of equipment. If the EPA specifically said to list the numbers and types of equipment in this section it was suggested that they would have said so on page 2 of the report. The question of putting down the numbers and types of equipment could become very cumbersome where a manufacturer wished to move a grinding machine from a location with one collector to another location where it would be hooked into another collector. The member who filed with the EPA worked on reports in 2 different jurisdictions: New York and Tennessee. He indicated that at neither location did he enter the number of pieces of equipment on this form. (Since the meeting he called to advise that the application filed in Tennessee without quantities was accepted by the EPA. His application in New York State had not been either accepted or rejected as of June 4, 1973.) It would appear that Regional Offices are not in agreement as regards quantification of the equipment under the "Process Description."

The question came up concerning interpretation of question 3, the "Amount of Pollutant." In many factories a set of dry mix brake blocks could emit into a collection system at the mixer, at the briquette press, at the cut-off wheels, at grinding, at drilling, and at inspection and boxing. The problem is that this is the same original asbestos which entered the process and might be counted 6-8 times. So, in effect, a factory taking in one million pounds of asbestos might list one million pounds of asbestos going into 8 different collection systems. This, in turn, would make it appear that eight million pounds of asbestos is going into the operation. From the wording of the form, it would appear that this is exactly what the EPA wants. However, another member was told that this in not what the EPA wants. He suggests that if a factory takes in one million pounds of asbestos into the process that it should not report in total more than one million pounds of asbestos. If he had 10 different emission points, he would divide the one million pounds of asbestos by 10 to give the "amount of pollutant." Again, their apparently has been a difference in interpretation from different Regional Offices of the EPA.

On page 3 of the report, under "Waiver of Compliance," it was stated that Sections 2a and 2b did not have to be completed unless EPA specifically requests this information.

INSTITUTE SEMINAR ON SAFETY AND HEALTH

At the February 16, 1973 meeting, suggestions were made that the Institute consider the sponsoring of a seminar for members associated with plant operations. The Institute indicated it would be willing to sponsor such a seminar if sufficient interest developed.

A question was raised as to whether this seminar would apply only to asbestos. The Secretary indicated that such a seminar would apply to any field of interest but it should be related to problems that can be tied into State and Federal regulations. Among the topics suggested for a seminar were the following:

Air sampling and asbestos concentration determination.
The pulmonary function test and X-Ray.
Possible extension to include noise and heat stress.
Cooperation between management and workers in meeting the regulations.
Asbestos bag opening machinery.

The Secretary was directed to make up a list of subjects which might interest the Membership and to canvass the members as regards their interest. In addition to the agenda items to suggest to the Membership, it was suggested that the canvassing letter ask if an individual from that member company would attend, where the meeting should be held, and when the meeting should be held. It was indicated that a meeting in the late fall would be desirable and such locations as Chicago, Detroit, Pittsburgh and Paramus were suggested.

When the Secretary has prepared a questionnaire it will be submitted to Mr. Feierabend for his review prior to distribution to the Membership. The actual agenda will be drafted after the members have indicated their preference. The question was raised as to whether outside speakers would be involved and it was suggested that we were not interested in a commercial pitch at the meeting. Johns-Manville had indicated an interest in approaching such a seminar with the idea of promoting their HEAF (High Energy Air Filter) pollution control equipment. It was suggested that perhaps it might be worthwhile to have outsiders make presentations concerning asbestos bag opening equipment, pelletizing, collection, etc. This will have to be worked out at a future Committee meeting.

Brake and Clutch Emissions Generated During Vehicle Operation

This particular study was run by Bendix Research Laboratories under sponsorship of the EPA. A paper was presented to the S.A.E. by Dr. M. Jacko and Mr. R. DuCharme of Bendix, and Mr. J. Somers of the EPA. The actual report to the EPA is a massive document explaining every test procedure and every method of collection used in the study. A technical paper was presented by these 3 gentlemen at the SAE Meeting in Detroit in May, 1973. The study essentially centers on how much asbestos is being put into the atmosphere from brake linings and clutch facings. As Dr. Jacko was in charge of this investigation he discussed the paper at our meeting. He advised that a condensed version appears in the magazine AUTOMOTIVE ENGINEERING. Among the points that Dr. Jacko made was that there were problems where a brake on one side was enclosed and the other brake was open to the regular atmosphere. Modifications had to be made involving cooling of the outside of the shroud so that there would not be too great a temperature difference from the left side to the right side. This was more of a problem with the disc brakes on the fronts. Actually with the necessary cooling, there was hardly any difference between the drum brake rears side to side.

Among the items discussed in the paper were how much asbestos is used in friction materials. It is indicated that there are about 103 million pounds of asbestos in the friction materials which are used in the United States each year. There apparently are some differences of opinion as regards how much asbestos is involved but it generally falls in the 90-120 million pound range. Actually, the amount subject to wear is about 66-2/3% of the actual lining that gets on to the brake (after grinding). When asbestos is being used in brake linings it is discarded in one of three ways: It gets deposited on the surfaces of the brake, such as on the caliper, and around the wheel cylinders. (This is surface debris). Additional material is collected on the lining surfaces, in the rivet holes, and on the brake drum. (This is called sump debris) Additional material becomes airborne and is collected on membrane filters. (This is called airborne debris). It is this latter airborne debris that the researchers are seeking to quantify.

Based on the samples that were collected, the conclusions were that more than 99.7% of the asbestos in the brake lining is converted to other products. By extrapolating the data that they were able to develop on a passenger car the researchers indicate that a total of 5060 pounds of asbestos is put into the

atmosphere. This airborne asbestos emission is 3.2% of the total asbestos emitted from all automotive brake linings and clutch facings in the U.S.

A question arose as to what happens to the asbestos debris that drops out. Does it eventually get into the atmosphere? It was indicated that based on the study of other materials that apparently there have been build-ups such as lead along the sides of turnpikes. This material apparently goes into the earth's surface and whether it is picked up again is dependent on other factors such as the proximity to streams, etc.

A gentleman from Ford Motor Company was also to present a paper to the SAE meeting concerning asbestos particulate emissions into the atmosphere. No paper was available at this time. There were some questions concerning procedures and a source of data on the Ford paper, but in any event the paper indicated a lower total asbestos emission than the Bendix paper. These two papers should serve as source information when others are attempting to quantify the asbestos emitted into the atmosphere from brake linings and clutch facings.

OTHER ITEMS

The topic of OSHA inspections and enforcement was brought up briefly and the members indicated that no new actions had been taken by OSHA as regards enforcement concerning the asbestos standards.

The Asbestos Information Association (AIA) is to put out a Compliance Manual concerning control practice. This is still preliminary and there is no advance copy available at this time.

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There being no further business brought to the attention of the Committee, upon motion duly made and unanimously passed, it was

RESOLVED: to adjourn.

Adjourned: at 2:30 P.M.

E. W. Drislane
Secretary

TABLE 18—*Summary of Injection Experiments by Intraperitoneal Technic*

Dosage: Each animal* received a single intraperitoneal injection of 3 cc. of a 10 per cent dust suspension. Total amount of dust injected was 0.3 Gm.

Mineral	Size of Dust Particles	Guinea Pigs Used	Maximum Survival after Injection, Mo.	Results
Chrysotile (Thetford)	5 microns and less (ball milled 210 hr.)	15	36	No fibrosis or asbestosis bodies. Dust particles ingested by phagocytes, chiefly multinucleated variety. Six mo. after injection fibrous elements of dust appear to have dissolved leaving only the insoluble magnetite, a contaminant. No reaction in surrounding fat or areolar tissue. No transporting of dust to regional lymph nodes. Observations made at intervals from 1 to 36 mo. after injection.
Chrysotile (Thetford)	Long fiber (through 100 mesh)	4	8	Definite fibrous reaction produced, delicate and nonhyaline. Atypical asbestosis bodies developed, but all were unusually small. No evidence of extra long fibers seen. Observations only at 8 mo.
Amosite	3 microns and less (ground in agate mortar)	9	12	Infection interfered with interpretation. Dust reaction appeared to be of inert type and limited to phagocytosis with a moderate tendency to lymphocytic infiltration. Observations at 1, 4, 8 and 12 mo.
Crocidolite	3 microns and less; also some long spicules (ground in agate mortar)	17	12	Dust foci consisted only of large mononuclear and giant phagocytes surrounded by a minimum amount of cellular connective tissue. The injected dust contained not only fine material that in grinding had been mashed into irregular [illegible] but also many long spicules 10 microns or more in length. No asbestosis bodies seen, although the longer spicules appeared slightly swollen and granular. Observations at 1, 4, 8 and 12 mo.
Anthophyllite	3 microns and less (ball milled [illegible] hr.)	6	24	Essentially inert foreign body reaction. In early animals (1, 4 and 8 mo.) focus of monocytes and small giant cells and a little central necrosis. In the 4 mo. animal there was also slight peripheral fibrosis. At 12 and 24 mo., nonprogressive mass of monocytes and giant cells; no fibrosis.
Anthophyllite (originally labeled talc)	Mostly 3 microns and less, some fibers 5 microns or more long (ground in agate mortar)	6	12	Reaction, which consisted of very large giant cells surrounded by a variable number of lymphocytes, was much heavier to these considerably long fibers than to the fine dust in the experiment above. There was more or less proliferation of fibroblasts producing cellular connective tissue visible in areas where the quantity of foreign particles was not so great that it obscured the reaction. Observations at 1, 4, 8 and 12 mo.
Tremolite (soda free)	5 microns and less (ball milled 15 hr.)	6	12	Inert type of response never progressing beyond the stage of very slight lymphocytic reaction about masses of dust filled phagocytes. No fibrosis. Observations at 1, 4, 8 and 12 mo.
Tremolite (soda)	3 microns and less (ball milled 48 hr.)	6	18	Inert nonprogressive foreign body type of reaction. No fibrosis. Observations at 1, 4, 8, 12 and 18 mo.
Anthophyllite	100 microns and less	4	12	Distinct early fibrosis produced by anthophyllite and fibrous pyrophyllite with subsequent regression. Crystalline pyrophyllite inert throughout. At 1 mo., giant cells about long thick spicules, at 4 mo., definite fibrosis replacing giant cells of anthophyllite and fibrous pyrophyllite reaction; at 8 mo., fibrosis which started at 4 mo. had decreased, especially with fibrous pyrophyllite. At 12 mo., reaction to all three dusts consisted of foreign body giant cells with lymphocytes but without necrosis or fibrosis. No asbestosis bodies.
Pyrophyllite (fibrous)	100 microns and less	4	12	
Pyrophyllite (crystalline)	100 microns and less	4	12	

* Each animal receiving long fiber chrysotile was given an injection of 2 cc. of a 0.6 per cent dust suspension.

Protective Action of Aluminum Compounds

When colloidal aluminum hydroxide had been added to a suspension of long fiber chrysotile prior to injecting this suspension intratracheally into rats, the aluminum compound did not prevent the irritation of tissue due to chrysotile. If anything, the acute inflammatory response evoked by the injected fibrous mineral was accelerated. One month after the last injection of the dust suspension the bronchiolitis was becoming fibrous. King and his associates also found that aluminum failed to protect pulmonary tissue from the irritation caused by asbestos fibers [11]; in their experiments metallic aluminum was used instead of the hydroxide.

Formation of Asbestosis Bodies

The iron in the coating of the asbestosis body appears to be derived from blood or tissue elements and not, as has been suggested, from the mineral fiber. After two kinds of chrysotile were injected subcutaneously into the groin of a guinea pig—one kind containing 2 per cent and the other 0.2 per cent ferric oxide—the asbestosis bodies were equally numerous at both sites of injection and showed no difference in their reaction to prussian blue, the reagent which stains iron. This finding is in agreement with that of Giroux.[12]

Tissue Reaction to Asbestosis Bodies

Asbestosis bodies recovered from human lung tissue and injected intratracheally into guinea pigs failed to produce a fibrous reaction. The material for injection was obtained by digesting with sodium hypochlorite solution the lung tissue removed at autopsy from an asbestos worker. The asbestosis bodies could be seen in the guinea pigs for at least a year after injection. This experiment shows that the asbestosis body has a rather resistant coating which is not destroyed by moderate hypochlorite treatment, which may be maintained in vivo for a year or longer and which renders the fiber incapable of producing fibrosis. It thus appears that the coating is a protective mechanism. This thought was expressed by Beintker as early as 1934.[13]

THEORY OF IRRITANT ACTION

Two hypotheses have been proposed to explain the tissue irritation and reaction caused by asbestos fibers: the chemical and the mechanical. In the chemical theory, which is based on experience with quartz, it is assumed that the asbestos minerals dissolve in the body fluids and that in this process their bases are leached away to leave silica in a form capable of irritating tissues. According to this hypothesis asbestosis is merely an indirect silicosis. Several facts make the chemical theory untenable: Intratracheal injection of brucite fibers, which had a silica

12. Giroux, M.: Amiantose experimentale: valeur pathognomonique du "corps d'amiante," Laval med. 8:239, 1943.

13. Beintker, E.: Über die Asbestosiskörperchen: Bemerkungen zu der Arbeit von Beger, Virchows Arch. f. path. Anat. 293:537, 1934.

content of only 0.90 per cent, caused typical fibrosis like that produced by the asbestos minerals; free silica particles increase in potency as the particle size becomes less, but asbestos fibers shorter than about 10 to 20 microns are relatively innocuous; aluminum hydroxide neutralizes the irritating effect of quartz but not of asbestos; serpentine has the same chemical composition as long fiber chrysotile, but it produced only an inert type of tissue reaction; there is a wide range in the chemical composition of the minerals which do cause asbestosis (table 19). In view of this evidence it seems more likely that asbestosis is caused by an unusual mechanical irritation due to long asbestos fibers, this irritation being related to the peculiar filamented structure of the fiber and the associated flexibility, which are possessed by no other foreign body studied. Thus, ignition of chrysotile fibers changed their structure and made them inert, although the same fibers, before being heated, would have produced fibrosis (table 14). Further support for the theory of mechanical irritation is that asbestosis occurs in an organ of high mobility —the lung—and that a fibrous reaction can be produced by injecting

TABLE 19.—*Analyses of Fibrous Minerals*

Fibrous Minerals	SiO_2 %	Fe_2O_3 %	FeO %	Al_2O_3 %	CaO %	MgO %	Na_2O %	K_2O %	Ignition < 105° C. %	Loss > 105° C. %	Total %
Amosite	46.23	4.08	22.48	1.09	2.01	4.23	0.23	0.20	0.66	5.23	99.+
Amphibole	55.04	3.08	.....	1.69	12.22	23.29	0.43	0.12	0.22	3.80	99.+
Anthophyllite	57.69	0.27	.....	0.22	0.44	28.37	0.32	0.15	0.27	4.06	99.+
Brucite	0.40	4.76	0.28	0.45	0.04	51.99	1.91	0.16	0.53	22.05	99.+
Chrysotile	38.55	2.22	.....	0.34	0.01	40.56	0.21	0.09	4.21	14.99	99.+
Crocidolite	51.49	16.27	4.79	1.01	0.49	12.93	4.92	0.37	0.05	2.26	99.+
Tremolite	56.51	1.21	.....	0.56	0.44	24.22	0.24	0.09	0.24	2.79	99.+

asbestos fibers into the peritoneum, where there is also a degree of mobility, but not by injecting them into other extrapulmonary organs such as the liver, the spleen and subcutaneous tissue.

COMPLICATIONS

The experimental investigations with asbestos minerals were concerned primarily with the effect of the dust on normal tissue, but some attention was given to other phases, such as susceptibility to infection. The only experiment in which the effect of asbestos dust on a pulmonary infection was studied was the first inhalation experiment, carried on with King's floats dust. It is unfortunate that, owing to the lack of adequate facilities at that time, infection studies could not be made in the other inhalation experiments also.

Susceptibility to Tuberculous Infection

The development of a tuberculous process initiated at the beginning of exposure to dust, and also of a tuberculous infection superimposed on an established asbestosis, was described in preceding sections of this paper. It may be stated that asbestos when classified according to the effect of a dust on tuberculous infection would be placed below an active

dust like quartz but above an inert dust such as iron oxide. In animals injected with attenuated tubercle bacilli, quartz causes the infectious process to progress until the animal dies of tuberculosis. Inert dusts have no effect on the infection, and the lesions usually heal and the disease disappears. Asbestos dust is in a different category. In the experimental investigation, when the fibrous dust was being inhaled during the evolution of the infection, there was spreading of the tuberculous process for a time, but usually the stimulus for continued proliferation of the tubercle bacilli was not sustained, the progression was arrested and healing followed. In guinea pigs injected with attenuated tubercle bacilli after being exposed to asbestos dust for slightly more than two years, progressive disease did not develop. The only modification of the infection was one of localization, a few bacilli being retained in the fibrous terminal bronchioles and forming tubercles there, in addition to the usual foci beneath the pleura. Such tubercles healed in a few months.

Susceptibility to Nontuberculous Infection

There was no specific experiment concerning the effect of inhaled asbestos dust on nontuberculous infection. Intercurrent pneumonia was rather common among animals exposed to asbestos dust, the frequency in guinea pigs exposed in the four inhalation experiments ranging from 16 to 39 per cent. This incidental evidence suggests the possibility of an effect of asbestos dust on nontuberculous infection. Nevertheless, since such epidemics are not uncommon in inhalation experiments with other dusts and even in the colony of normal animals, it is felt that the inhalation of asbestos dust does not exert a significant effect on the susceptibility to nontuberculous pulmonary infection.

COMMENT AND SUMMARY

Owing to the vast amount of data included in this investigation, it seems most convenient to summarize and to state as concisely as possible the various observations which emerged from the experiments and to follow each with a brief résumé of the evidence.

A. Various species of animals, including the guinea pig, the rat and the rabbit, but not the mouse and the dog, develop peribronchiolar fibrosis of the lung similar to human asbestosis after being exposed by inhalation or intratracheal injection to long chrysotile asbestos fibers.

Both inhalation and injection experiments provide ample support for this statement. Figure 8 *A* reveals the cellular fibrosis that occurs in guinea pigs following inhalation of long fiber asbestos; figure 9 shows the fibrosis caused in the cat by inhalation of long fiber asbestos dust. Similar but less extensive fibrosis occurred also in rats and rabbits (table 1). Mice and dogs failed to respond. This variation in response of different species to identical dust exposures is still to be accounted for.

B. Long asbestos fibers are essential in the production of the peribronchiolar fibrosis; short fibers are incapable of producing this reaction.

Inhalation experiments with asbestos dust suggest, and intratracheal injection experiments confirm, that peribronchiolar fibrosis is produced by asbestos fibers between 20 and 50 microns in length but not by particles shorter than 20 microns (tables 16 and 18). This indicates that the minimum length of fiber possessing the capacity to produce the typical peribronchiolar fibrosis in animals is somewhere between 20 and 50 microns. Pointed studies have not been carried out to determine the upper limit of effective fiber length. It appears, however, that that limit will be determined by the inhalability of the fiber.

C. The mode of action of the long asbestos fiber in the production of asbestosis is primarily mechanical rather than chemical in nature.

The evidence for this conclusion has been reviewed in a preceding section, page 39. The flexible filamented structure of asbestos fibers plays an essential part in the irritating action, since the solid, inflexible fibers of glass wool do not produce fibrosis (fig. 13 *B*).

D. Typical experimental asbestosis was produced by the inhalation of an atmospheric suspension containing an average of 1.35 million asbestos particles per cubic foot of air by light field count, of which less than 1 per cent consisted of fibers longer than 10 microns.

In the inhalation experiment with 100 per cent ball-milled asbestos dust containing 0.6 per cent of fibers longer than 10 microns (table 11) typical fibrosis was obtained (table 9). The evidence presented shows at least that an atmospheric concentration of asbestos dust containing less than 1 million (0.6 per cent × 1.35 million) fibers longer than 10 microns per cubic foot of air is capable of producing experimental asbestosis in guinea pigs. The actual lower limit of concentration of long fibers necessary to produce asbestosis in animals cannot be established from these studies.

E. The duration of exposure required to develop the pulmonary reaction to inhaled asbestos dust is inversely proportional to the concentration of long fibers in the atmosphere; as the concentration is increased, the reaction develops in shorter time.

The basis for this statement appears in the data of the inhalation experiment with long fiber asbestos. For that experiment the average concentration of the atmospheric dust was about 40 million particles per cubic foot of air, and size-frequency determinations disclosed that 6.7 per cent of the air-suspended material consisted of fibers longer than 10 microns (table 11). Thus, by calculation, it is estimated that the concentration of the longer fibers was 2.7 million (6.7 per cent × 40 million). The lungs of animals exposed to the long fiber asbestos dust revealed that the pulmonary reaction developed in approximately one-half the exposure time required for its development in animals inhaling the ball-milled product, for which the concentration of the longer fibers was only 0.8 million (0.6 per cent × 1.35 million).

F. Established experimental asbestosis ceases to progress on discontinuance of dust exposure.

The experimental investigation shows, in fact, that on discontinuance of exposure there was an appreciable clearing of the mature pulmonary

lesions, due to contraction of the fibrous tissue. In contrast, an immature tissue response, evidenced primarily by cells with little or no fibrosis, continued to progress. It is assumed that, following attainment of fibrotic maturity, the same process of contraction would ensue as was noted for the mature lesion.

G. The formation of asbestosis bodies represents a coating of the fibers by blood and tissue elements, which results in loss of ability of the fiber to produce fibrosis.

Intratracheal injection of asbestosis bodies failed to produce the typical asbestotic tissue reaction in experimental animals. The cessation of progressive reaction observed soon after exposure terminates may be due to the formation of asbestosis bodies.

H. Aluminum hydroxide failed to neutralize the fibrosing action of the long fiber asbestos.

Aluminum hydroxide added to the suspension of chrysotile asbestos prior to intratracheal injection did not retard or prevent the development of asbestosis in rats.

I. Inhalation of asbestos dust did not alter significantly the final outcome of experimental tuberculosis in two series of guinea pigs exposed to the dust.

The apparently mild influence of asbestos dust is in distinct contrast to the stimulating effect exerted by inhaled quartz on a tuberculous process in the lung. The interpretation must remain tentative, however, since it is based on an investigation limited to two series of guinea pigs exposed to only one kind of asbestos, namely, King's Beats. Table 4 shows that when the infection was coincidental with the onset of dust exposure, there was temporary progression of the tuberculosis [illegible] with subsequent healing; when infection was initiated after 26 months of dust exposure, the course of the tuberculosis was not appreciably altered. The latter finding is quite different from our usual experience with quartz dust or with mixed dusts containing quartz, wherein the adverse influence of quartz on a tuberculous infection is manifested more strikingly when infection is initiated after a period of dust exposure, viz., superimposed on a background of established silicosis. As indicated above, this more sensitive test, when applied to asbestos dust, failed to demonstrate that the latter had an adverse influence on a tuberculous infection. The inability of asbestos dust in that experiment to affect unfavorably the tuberculous process furnishes strong support for the interpretation that inhaled asbestos dust has no more than a mildly unfavorable effect on pulmonary tuberculosis.

This investigation was made possible by the generous financial support of a group of companies of the asbestos industry.