SUBJECT: Handling, installing, removing and/or disposing of material containing asbestos.

1.0 PURPOSE:

1.1 To provide a safe procedure for the handling of material containing asbestos. Asbestos is defined to include Chrysotile, Amosite, Crocidolite, Tremolite, Anthophyllite and Actinolite.

2.0 INTRODUCTION:

2.1 Excessive exposure to asbestos fiber is harmful to your health. These fibers have been associated with cancer of the lungs, throat, stomach, colon and rectum. The maximum permissible exposure is two (2) fibers, longer than five (5) micrometers, per cubic centimeter of air when averaged over 8 hours. At no point in time may an employee be exposed to greater than 10 fibers per cubic centimeter of air.

2.2 Asbestos fiber is found in insulation used on piping, equipment, etc., that was installed earlier than about 1973 or 1974. This was when the Sistersville Plant deleted asbestos containing insulation from Stores and went to an "asbestos free" insulation.

2.3 Asbestos fibers are found in some brake linings used on automobiles, trucks and our work vehicles.

2.4 Asbestos fibers can also be found in the wall coverings, insulation, etc., used in the construction industry on older buildings.

3.0 RESPONSIBILITY:

3.1 The appropriate first line supervisor and craftspersons are responsible for controlling the safe removal and disposal of asbestos-containing material.

3.2 Based on size, complexity and location, the Safety and Health Department shall be notified prior to the start of the job. At that time a decision will be made as to the need for and extent of any monitoring. The Supervisor of the personnel handling the asbestos-containing material and the actual individual shall request the monitoring.

3.3 Proper Housekeeping is the responsibility of each individual in performance of the work assignment. Appropriate equipment for prevention of dust release and clean-up after the job has been provided. Please use this equipment but never use a broom, brush or blow with air when handling materials containing asbestos.

Responsible for review and update of this procedure - Maintenance Department

A 1647

PLAINTIFF'S EXHIBIT
UC-1618

3.4 The Environmental Protection Control Staff shall notify the EPA of the annual amount of asbestos activity in the plant.

3.4.1 The EPA must also be notified prior to large scale jobs. These are defined as stripping of more than 260 linear feet of pipe insulation or more than 160 square feet. The Insulator Supervisor shall notify the EPA Staff prior to such jobs.

(R) 3.5 The responsible supervisor shall ensure that employees using asbestos-containing material have received proper training on the hazards of asbestos. This will be done every six months by the Insulator Supervisor or a designated training supervisor.

3.6 If an employee must wear respiratory protection, the supervisor shall assure that the employee is medically approved and has received proper training on the use of respiratory protection.

(R) 3.7 The individual craftsperson is responsible for wearing respiratory equipment when required. The first line supervisor in the operating dept. shall ensure that the operators know and follow the guidelines outlined in this procedure.

4.0 PROCEDURE:

4.1 All asbestos-containing material will be handled with extreme care and our goal is to take appropriate measures to eliminate all airborne asbestos fibers.

4.2 When asbestos is involved, barricade tape will be used to isolate the area and caution signs will be posted at all access routes to the work area. These signs will be located at a distance which allows an employee to take the necessary protective steps before entering the area. This sign will comply with OSHA specifications and shall read:

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

4.3 Asbestos-containing material will be handled, mixed, applied, removed, cut, or otherwise worked on in a wet state sufficient to prevent the emission of airborne asbestos fibers in excess of exposure limits.

4.4 Asbestos-containing cement, mortar, coatings, plaster, wrappings, or other materials will not be removed from bags, boxes, or other containers, in which it was shipped, without first wetting, enclosing or ventilating.

4.5 Employees engaged in the handling or removal of asbestos-containing material will wear approved respiratory protection. The individuals will normally be insulators, insulator helpers, and vehicle mechanics. This respiratory protection is mandatory at this location for all exposures to asbestos-containing material. Two types of protection are possible.

4.5.1 Normal Exposure: This is where the concentration of asbestos fibers is reasonably expected to be less than 10 times the permissible exposure limit. In this case, use the Comfo II respirator with the Type H Ultra Filter.

4.5.2 Heavy Exposure: This occurs when the concentration of asbestos is reasonably expected to exceed 100 times the permissible exposure limit. In this situation, a type "C" supplied air respirator in the positive-pressure mode shall be used along with a full facepiece mask.

4.6 Wetting of asbestos-containing material will be done in such a manner as not to wash the fibers into the ground, drainage trenches or sewer facilities. Any fibers contacting the ground will be gathered up and disposed of in approved containers.

4.7 Special wetting agents for asbestos-containing material are available in the Sistersville Plant Store Room. This material is designed to bind the asbestos fibers and reduce or eliminate the potential of dust generation. This wetting material will be used where practical.

4.8 A special Vacuum Cleaner is available, with a special filter designed for 99.97% efficiency. This Vacuum Cleaner will be used where wetting is not appropriate, such as brake lining work, inside of structures, etc., and for removal of any asbestos-containing material on one's clothing.

4.9 Disposable Protective Clothing designed for exposure to asbestos-containing material will be used on all jobs involving the handling or removal of material containing asbestos.

4.9.1 Disposable Protective Clothing will be worn prior to contact with asbestos-containing material.

4.9.2 Disposable Protective Clothing will be removed at the work site and placed in approved containers.

4.9.3 Protective Clothing will be removed prior to coming in contact with other personnel, eating, smoking or using facilities used by other personnel.

4.9.4 New Protective Clothing will be worn for each job or continuation of the job if the previously used Protective Clothing was removed.

4.10 Asbestos-containing material, including used disposable protective clothing, will be collected in impermeable, sealed bags or containers that are labelled according to Paragraph 4.12.

what about: Jansen gaskets brake linings ? etc.

4.10.1 Within the shift that the job is completed, the craftsman or whoever collected the waste material will deliver the bags or containers to the asbestos waste area.

4.10.2 The appropriate supervisor will then notify the Contractor (Charlie Brown) within the same shift that asbestos waste has been delivered for burial.

4.10.3 The Contractor will bury the waste in compliance with federal regulations within 24 hours of being notified.

4.11 Contaminated clothing will be collected and transported in sealed impermeable bags or closed containers, properly labelled as described in Paragraph 4.12. This clothing will be sent to a commercial laundry which has facilities for handling asbestos contained clothing.

4.12 Disposal Containers or Laundry Bags shall read:

- Caution -

Contains Asbestos Fibers Avoid Breathing Dust Breathing Asbestos Dust May Cause Serious Bodily Harm

4.13 When an employee has clothing (other than disposable type) contaminated or suspected to be contaminated with asbestos, the employee will:

4.13.1 Enter the "dirty room" facility provided for insulators and remove clothing.

4.13.2 Contaminated clothing will be placed in sealed containers and sent to laundry.

4.13.3 Enter the shower facility to wash off all asbestos fibers.

4.13.4 The employee will next enter the "clean room" facility and put on clean/uncontaminated clothing.

(R) 5.0 AUDIT:

5.1 The Dept. Head of Maintenance/Materials Management is responsible to audit performance as per "The Removal and Handling of Asbestos Materials" procedure using checklist audit form attached.

A-0400

Audit Checklist for the Removal and Handling of Asbestos Materials

1. Has operating area been notified that work will be performed in their area? Yes/No

2. Have operators been informed of procedures to follow? Yes/No

3. Is area adequately flagged off and signs placed in areas visible to foot traffic? Yes/No

4. Is the craftsperson wearing the proper equipment required for job? Yes/No

5. Has plastic been laid on ground to prevent contamination (where practical)? Yes/No

6. Has asbestos been wetted down prior to removal? Yes/No

7. Is material being placed in bags and sealed as it is being removed? Yes/No

8. Is contaminated clothing being removed and placed in bag prior to leaving job site? Yes/No

9. Has "dirty room" and shower procedure been followed? Yes/No

10. Has area been cleaned up and all flags and signs been removed? Yes/No

11. Has Charlie Brown been notified to bury the material? Yes/No

1. Inspect job site to determine if insulation is asbestos.

2. Describe the proposed work area, i.e. enclosed area, pipe trail, office, hallway, tower, etc.

3. Determine the total amount of insulation to be removed.

4. Determine what method will be used to complete the job.
   a. Glove bag.
   b. Seal (encapsulate) and remove whole object.
   c. Short duration emergency job.
   d. Negative pressure enclosure.

5. Glove Bag

   The surrounding area will be posted with asbestos hazard warning signs. All personal in the area will be informed of the stripping location and the procedure to be used. The Safety and Health Dept will be notified to monitor personel in the area. Once it has been statistically shown by IH monitoring that exposures are below the TEL, monitoring will be discontinued on a regular basis. Periodic monitoring will then be implemented. Operations may continue in the area where stripping is being performed, with operators wearing their Comfo II masks with the MSA filter cartridge type "H". (This will only be needed if operators are working directly below or standing next to the stripping.) The designated glove bag stripping procedure for training will be followed during stripping.

   All glove bags, full protection body coveralls, and any other waste materials will be put in properly marked asbestos bags and delivered to the asbestos landfill.

6. Seal/Encapsulate

   To remove objects with insulation intact the insulation must be wrapped in plastic with both ends and the seam taped. Duct tape or equal is required. (Note - masking tape is not permitted.)

7. Short Duration Emergency Jobs

   Standard stripping procedure for short duration jobs. (This reserved to describe short duration emergency work.)

8. Negative Pressure Enclosure

   A. When using an enclosure the following procedure must be followed:

      1. Remove all objects from the area to be enclosed, if there is detectable or suspected fibers, a Comfo II mask and protective clothing should be put on, and wipe down equipment with a wet cloth or towel. Anything that is not able to be removed should be covered with

plastic and taped to the floor, wall, or ceiling, to prevent contamination.

2. Shut down the HVAC system, to avoid inadvertent activation of the HVAC system while removal operations are in progress, the control panel should be tagged and locked out. During final cleanup, the HVAC filters should be changed and disposed of as contiminated material.

3. After deciding the location of the enlcosure, a single entrance to be used for access and egress to the work area should be selected, all other doors and windows should be sealed with 6 mill plastic sheeting and securely taped.

4. All four walls should be covered with plastic from the ceiling to the floor and taped top and bottom, and sides.

5. Next a layer of plastic should be taped to the top of the wall, the entire inside surfaces of all wall areas are covered in this manner, and the sheeting over the walls is extended to the center of the floor area, where it is taped to form a single layer of material incasing the entire room, except for the ceiling. A final layer of plastic sheeting is then laid across the plastic covered floor area and up the walls to a level of about 2 feet. When covering the ceiling, overlap the walls about 24 inches.

6. Overlapping sheets of plastic shall be hung over the opening for access and egress.

7. After stripping has started fresh air respirators are required unless continuous monitoring justifies a lesser requirement.

B. The negative air machine will be set up so it will have a direct pull from the area being stripped to the negative air machine. The exhaust of the negative air machine will be exhausted through the enclosure wall to the outside and away from the enlcosure as far as possible. Check the area to make sure the exhaust hose is not aimed toward other bad asbestos insulation that can be blown into the outside work area.

1. Next install a negative air monitor inside of the enclosure.

2. A portable clean room/shower room/decontamination room will be attached to this enclosure if feasable. If not then a decontamination room will be provided in the egress of this enclosure. The decontamination room

will house the resources (vacuum, wet wipes, clean coverals, etc.) for individual decontamination.

3. If a portable shower is used it will be set up directly outside the enclosure, or as close to the enclosure as possible.

4. Contact the Safety and Health Dept to monitor the stripping of the asbestos.

5. Before starting to strip, check the filters in the negative air machine and change them if necessary.

6. An HWP is required before this machine may be started up. The HWP must be signed by the insulator supervisor. This is to insure inspection of the enclosure.

7. At the end of the regular shift or at the end of the overtime shift the negative air machine will be turned off and the HWP returned to the production foreman. When working in offices or equal the machine will be left on until decontamination is complete.

8. Check the enclosure to make sure everything is sealed after the negative pressure machine has been started, if there are any tears or loose ends that need taped, now is the time to do it. Check the monitors to make sure they are working properly before starting to strip.

C. Cleanup is very important, a wet-dry HEPA vacuum should be used to pick up any loose particles of insulation from the plastic and any other coverings. All pipes should be wet wiped to get any loose particles removed. Wip down plastic walls before removing the first layer of plastic on the floor which will be rolled towards the center. After the first layer of plastic has been removed, wipe down must be done again including the negative air machine. At this time the final monitoring can be done to insure everythig is clean. If tests come back the final plastic can be removed and the negative air machine shut down. This only applies to indoor jobs.

John K. Kyle

KBP/slm

A 10404

THE ASBESTOS INSTITUTE

Montreal, June 19, 1986

PLAINTIFF'S EXHIBIT 707.4

DOCUMENTS FROM AI ARCHIVES

Dear Sirs:

For your information we take pleasure in sending from our library, current year's fourth shipment of documents dealing with asbestos and health, as follows:

AI Nos.:

6550	6600	1513
6564	6601	588
6570	6602	592
6579	6723	593
6598	6769	602
6599	1503	618
		627

Yours truly,

Premysl V. Pelnar

A 16423

Premysl V. Pelnar, M.D.

PVP/tb

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Sherbrooke, Quebec
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Telephone: (819) 821-7633
Telex 058-36149 (BIBUNIV SHB)

DATE	INDEXED BY	EDITED BY	PAGE OF	DOCUMENT NO.
				3095

DOCUMENT TITLE

* Savers, I.C.:

LOCATION

KEY CODE	KEY WORD OR CHARACTERISTIC	SCOPE NOTES
56	Asbestos as a Health Hazard in the	
6	United Kingdom.	
80	Union Carbide U.K. Ltd. 1968.	
82	CONFIDENTIAL - not circulated.	

INDEX/TRACING CARD

A 16430

DOCUMENT NUMBER

DATE	INDEXED BY	EDITED BY	PAGE	OF	DOCUMENT NO.
					3070

DOCUMENT TITLE: Gross, Paul & deTreville, Robert T.P.:

LOCATION

KEY CODE	KEY WORD OR CHARACTERISTIC	SCOPE NOTES
43	The Pulmonary Response to Coalinga	Not to circulate.
40	Asbestos Dust: A Preliminary	Limited to committee
53	Investigation.	only.
72	Ind. Hy. Foundation, July 1968	
52	FINAL REPORT - NOT PUBLISHED	
80 20		

Evaluation: This study was undertaken to determine the relative pathogenicity of Coalinga chrysotile from California with Canadian chrysotile and other non-asbestos dusts when injected into the trachea of rats. It was found that Coalinga dust produces effects similar to Canadian chrysotile. "The lesions observed in the lungs of these rats suggest that the pathogenic potential of Coalinga dust resembles that of chrysotile dust. Whether this potential is less or greater than that of chrysotile dust cannot be determined from the available data."

INDEX TRACING CARD

DOCUMENT(S)

INTENTIONALLY

REMOVED

AMERICAN THORACIC SOCIETY
1740 Broadway
New York, N.Y. 10019-4374
(212) 315-8700
Medical Section of the American Lung Association

Gerard M. Turino, M.D., *President*
Joseph H. Bates, M.D., *President-elect*
A. Sonia Buist, M.D., *Vice President*
Herbert Y. Reynolds, M.D., *Secretary-Treasurer*

RECEIVED
1987
H. C. LEWINSOHN, M.D.

August 18, 1987

TO : Ad-Hoc Committee to develop ATS Statement "Diagnosis of Non-Malignant Asbestos-Related Disease"

FROM : Marilyn Hansen
Acting Executive Director

SUBJECT: Attached

The attached is an article from the San Francisco Banner Daily, a law journal, concerning this paper. I am sending to you for information only - no action is requested. In May ATS received a subpoena to produce all documents related to the development of this paper. The material provided by ATS through ALA legal counsel was the summary of the two Board of Directors meetings where the paper was reviewed and the Assembly review comments written by Sonia Buist and William Rom (each in role of Chairman).

Since May, Raymond Murphy has also received a subpoena requesting materials he has. To date, ATS has received no additional requests.

I thought you should be aware of this. If you have questions, please call the ATS office.

/ng
Attachment

Committee Members
Margaret R. Becklake, MD
Stuart M. Brooks, MD
Edward A. Gaensler, MD
Bernard L. Gee, MD
Allan M. Goldman, MD
Jerome I. Kleinerman, MD
Hilton C. Lewinsohn, MD
Roger S. Mitchell, MD
Mark J. Utell, MD
Hans Weill, MD

Diagnostic Report Latest Legal Fight In Asbestosis War

New Standards Used By Defense Counsel Are Under Challenge

48,000 Plaintiffs

By DEE ZIEGLER

The disputed credibility of a medical association report on diagnosing asbestosis has caused the latest battle in the ongoing war between defense and plaintiffs' attorneys over awards for asbestos-related diseases.

Defense attorneys are increasingly pointing to the report by the American Thoracic Society in an attempt to sway juries away from large asbestosis awards. And, at least one plaintiffs' attorney is readying a legal challenge to the use of the report.

A successful challenge may help win jury verdicts for some 48,000 plaintiffs whose claims against asbestos manufacturers are pending in courts throughout the nation, said the plaintiffs' attorney, Ronald Motley, a litigator from South Carolina representing 15,000 clients with asbestos related diseases.

The report, issued slightly more than a year ago by the American Thoracic Society, sets a more stringent standard than ones used previously for determining that a person has asbestosis — a combination of scarred lung tissue and thickened lining around the lungs that impairs breathing.

Attorneys representing asbestos manufacturers have wasted no time introducing the ATS standards in their defense, said Motley, vice president of the Association of Trial Lawyers of America. "The asbestos industry raises this report in every case involving asbestosis nationwide," he said.

Defense Using Report

In San Francisco, plaintiffs' attorney Harry Wartnick said the defense has been using the ATS report one way or another in every case his firm has tried so far this year. If Motley's efforts are successful, "then people who have asbestosis will be far less likely to be thrown out of court with no recovery," said Wartnick, partner in the San Francisco firm Cartwright, Slobodin, Bokelman, Borowitz, Wartnick, Moore & Harris.

The firm has several hundred clients with asbestos-related diseases, Wartnick said. Most of these, 70 percent, have asbestosis and the remainder have lung cancer. Of the four asbestosis cases it tried this year, the Cartwright firm has won two jury awards in excess of $150,000, lost one, and one resulted in a hung jury. In the losing case, the client didn't meet the ATS criteria.

The firm has 50 more asbestosis cases set to go to trial before the end of the year "and I suspect that the ATS standards will play a role in most of them," Wartnick said.

Madelyn Chaber, an associate in the Cartwright firm who tried the hung jury case, said defense attorneys in the Bay Area are using the ATS guidelines for diagnosing asbestosis as the basis for expanding the burden of proof that a person has lung disease.

The ATS lists four criteria as having "recognized value" in diagnosing nonmalignant asbestos-related diseases: a certain degree of scarring, below normal lung capacity, below normal ability to exchange oxygen for carbon monoxide, and detection in the lung via stethoscope of a sound like Velcro ripping apart.

The report states that scarring is the best indicator and that while it is possible for disease to be present with none of the criteria met, the diagnosis of asbestosis should not be made.

In the past, medical experts have relied on the standards set by the International Labor Organization to rate the amount of scarring and determine if a person is afflicted with a so-called "dust disease" like asbestosis or black lung disease. These standards rate a normal person 0/0 and a person near death from lung disease 3/3.

Raising the Standards

A person exposed to asbestos showing an abnormal X-ray is rated 1/0, but the ATS report raises the standard to 1/1. It is a subtle difference, Motley said, but it is sufficient to prompt the defense bar to argue that a large number of plaintiffs are not suffering from asbestosis.

Chaber said defense attorneys are disregarding the qualifying phrase "recognized value" used in the report and are telling juries all four criteria must be met for the plaintiff to have a successful case.

"People who have asbestosis don't necessarily get all the symptoms and sometimes they arise at different times and aren't detected on testing," said Chaber.

To increase the impact of the ATS report in a recent asbestosis trial, the defense attorneys enlarged part of the report to make it as big as a desk, said Chabers, but the judge wouldn't let them use the display in court.

Nonetheless, the defense attorneys from Brobeck, Phleger & Harrison and Morganstein & Jubelirer relied on the ATS report heavily throughout the trial, said Chabers.

"It certainly helped the defense," she said. Chabers said her medical experts made a strong case that there was sufficient lung

)iagnostic Report Latest Legal Battle in Asbestosis Litigation

Continued from Page 1

plaintiff fell just below the ATS standard. The report is an official statement from of the foremost societies," said Chaber. e jurors say to themselves, 'What do I w? This is what the doctors are telling ' " and they are impressed, Chaber said. fill Valley plaintiffs' attorney Jack Clap- , who has 360 active asbestosis cases ding in Bay Area courts, said he learned ut the ATS report recently when "the endants trotted out the new standard." he defense used the report to cross-exam- doctors appearing for the plaintiff and to ation whether the plaintiff had met the S criteria, Clapper said. "It doesn't help." Clapper has lost five out of six cases during past year, not always because the ATS iteria were not met. His one victory ought a $140,000 award.

"Whether the problem you have meets me doctor's definition of a disease seems most irrelevant. What matters is, was your fficulty in breathing caused by asbestos," apper said. "My client may meet one doctor's definition of asbestosis, not meet another's and exceed a third's."

Oakland plaintiffs' attorney Steven Kazan said the ATS report is "important to the defense in minimizing smaller cases where it is not clear that a person is seriously disabled from asbestos exposure." Kazan's firm represents about 1,000 asbestos victims, but 60 percent of them have lung cancer.

Defense attorneys at Brobeck, Phleger downplay the importance of the ATS report. Said partner James Miller: "In any case that's being tried to a jury, the important thing is never what the books say about various subjects, but rather what the various expert witnesses relying on those books have to say. Sometimes the standards are a factor, and sometimes they are not."

Miller, whose firm represents a group of manufacturers organized as the Asbestos Claims Facility, said the ATS report comes into play in "borderline cases" and in most cases the diagnosis is very clear.

Partner Steve Snyder said the ATS report clearly states that someone can have asbestosis without meeting all the criteria listed. He said Chaber "terribly misstates the situation" when she says the defense is telling the jury all of the criteria must be met.

South Carolina litigator Motley has embarked on a campaign to discredit the ATS report by charging it was adopted by a biased board largely composed of pysicians who work for the asbestos industry.

Motley contends that six of the 10 ATS board members are "asbestos industry consultants who have testified in court for the asbestos manufacturers on a regular basis."

He has already subpoenaed copies of the board's records and plans to begin deposing the board, beginning with the board chairman, Dr. Raymond L. Murphy of Boston, on Wednesday.

By the end of fall, Motley said he hopes to have taken depositions from the entire board and will plan his strategy for introducing the evidence in his asbestos cases.

The president of the ATS rebuffed Motley's charges.

The report "was published in medical journals with worldwide circulation and it has been well-received by pulmonary physicians and sceintists," ATS President Gerard Torino said through a spokeswoman.

"To our knowledge, no medical specialists have filed dissenting opinions in medical journals. ATS has been advised that the contents of the statement have become an issue in pending litigation. ATS realizes that parties to litigation have positions to assert and may support or attack the paper depending on those positions. However, the society believes that the statement is a sound, state-of-the-art review of the topic."

The report was developed by a joint committee of the American College of Chest Physicians and ATS, composed of 9,000 physicians and scientists specializing in pulmonary medicine, the spokeswoman said. The joint committee included "a broad spectrum of opinion and expertise," she said, and during the development of the paper, drafts were reviewed by the ATS Scientific Assembly on Environmental and Occupational Health.

neath the plaque in continuity with the surrounding normal parietal pleural connective tissue, suggesting that plaques are extrapleural and develop between the latter and its covering layer of mesothelial cells (23). On microscopic examination, calcium deposition is present in a large proportion of plaques, and occurs as deposits along the course of the collagen fibers, ceasing abruptly where the connective tissue changes into normal pleural tissue (23). Well-differentiated cuboidal mesothelial cells are present on the surface and the edges of the plaque (23); metaplastic changes are uncommon (24, 25).

Although coated asbestos fibers have not been reported in relation to pleural plaques in the extensive series of 172 sections examined by Meurman (23) using polarized light, the presence of many uncoated fibers may be noted when ashed tissue is studied (26, 27). Recent studies of the digestate of a small number of plaques has demonstrated the presence of submicroscopic fibers of both chrysotile and amosite fiber in these structures. It is of interest that these are more concentrated in the calcified zones than in the fibrous zones (28).

The usual effect of asbestos on the visceral pleura is a focal or diffuse thickening. This varies from a thin, milky white discoloration, detectable only on gross visualization to a thick peel encasing the lung and easily seen on chest roentgenogram. In contrast to plaques, which are frequently diagnosed roentgenologically in asymptomatic persons, pleural fibrosis may cause symptoms and impair pulmonary function.

Pleural effusion may be caused by inhalation of asbestos; this is an early manifestation and is usually an exudate. On rare occasions it may persist for months or years. It may recur on the same or the opposite side after several years of exposure. In unusual circumstances it may be bilateral. Macroscopically, the fluid may be blood stained with variable numbers of erythrocytes, macrophages, lymphocytes, and mesothelial cells (29).

Pulmonary Asbestosis

Definition

The term asbestosis should be reserved for the interstitial fibrosis of the pulmonary parenchyma in which asbestos bodies or fibers may be demonstrated. While pleural abnormalities are commonly associated with parenchymal disease, they should be separately classified as there are differences between pleural and parenchymal fibrosis in epidemiology, clinical features, and prognosis.

Pathologic Features

In lungs with minimal or moderate fibrosis, the changes may be subtle and difficult to demonstrate. On examination of gross specimens, they appear as gray opaque areas devoid of air spaces in an otherwise brown lung. The microscopic changes in pulmonary asbestosis vary from small areas of basal fibrosis to a diffuse, fine fibrosis of both lungs. In general, the more extensive the process the smaller the volume of the lungs. The cut surface of the lung has a dark brown color with streaks of a fine, gray-colored fibrosis that generally appears to affect subpleural areas first and, frequently, in multiple and separate areas. This fibrosis may accentuate lobar and lobular septa and extends projections into the lung parenchyma. The parenchymal fibrosis, which has a linear and reticular appearance by X-ray, affects the lower lobes first, and extends upward with prolonged or heavy exposure (10, 30, 31). The fibrosis may take one of three forms. One is a diffuse fibrosis without air space enlargement; a second form is called honeycombing. This form may affect the lower lobes and subpleural regions (31). The third form is a combination of both diffuse fibrosis and honeycombing. This latter form is the one most frequently observed. The honeycombing is characterized by enlarged thick-walled air spaces ranging in size from 1 to 15 mm (32). The pleural surface adjacent to the fibrosis is invariably involved in the fibrotic process, either mildly with the appearance of a milky covering to the fibrosis, or with widespread fibrosis and symphysis (30, 31). The hilar lymph nodes are only slightly enlarged and soft unless other disease coexists. While emphysema has been reported in the past, it is unusual, and may have been incorrectly labelled as honeycombing (10, 33).

Microscopic Appearances

There is little information on the early pathophysiology of asbestosis in humans. Current opinion is largely based on inferences from animal studies. Some evidence exists that release of lysosomal enzymes may result from the partial ingestion of the asbestos fibers and the incomplete fusions of the phagosome membrane, so allowing the release of enzymes into the medium from lysosomes which have fused with the phagosome. The cytotoxic activity of asbestos exhibited by the release of lysosomal enzymes may result from the fibers penetrating intracellular structures, such as the nucleus and lysosomes, by preventing the movement of organelles within the cytoplasm, or by disrupting the cytoplasmic organization provided by microfilaments and microtubules (34). In any case, the initial reaction to asbestos fiber introduced into the lung is the immediate exudation of neutrophils and macrophages into the alveolar spaces in the locus of asbestos. This exudate varies with the age of the lesion. In the initial stages the exudate may be predominantly neutrophilic. Macrophages are the most common cells in the infiltrate. The inflammatory infiltrate soon is associated with varying degrees of organization with fibrosis (35, 36). The process is believed to be concentrated initially in peribronchiolar regions (10, 30). The initial lesion after exposure by inhalation is in the region of the respiratory or terminal bronchioles. There is a macrophage exudate in the lumen associated with asbestos fibers and bodies. Metaplasia of the cuboidal epithelium to squamous type may occur. In the early stages, fibrosis may be minimal but, when present, is in the respiratory and terminal bronchiolar regions and in the alveoli arising from the most proximal alveolar ducts. Alternatively, there may be coboidalization of the epithelial cells. Characteristically, in early stages only an occasional pulmonary subunit is involved. More advanced cases show a diffuse fibrosis, involving the interstitium, frequently associated with areas of extensive fibrosis with obliteration of air spaces and condensation of the bronchovascular structures. Areas adjacent to the fibrosis may show accumulation of alveolar macrophages, some of which have ingested asbestos fibers or other fragments. Alveolar epithelial hyperplasia may also occur. When moderate or severe degrees of fibrosis are present, the small pulmonary arteries and arterioles are thickened and sclerotic (30). (The presence of uncoated asbestos fibers and asbestos bodies in the presence of interstitial fibrosis is mandatory for the pathologic diagnosis of asbestosis.)

Before a pathologic diagnosis of asbestosis can be made we must consider a number of problems, including the following:

1. There are numerous other causes of interstitial fibrosis.
2. The distribution of interstitial fibrosis in asbestosis may be irregular, and therefore, adequate sampling of the lung must be done. The lingula and the right middle lobe are particularly prone to nonspecific fibrosis and sampling must take this into consideration.
3. While advanced asbestosis characteristically shows numerous asbestos bodies, they may not always be found because many fibers are cleared from the lungs and some, particularly chrysotile, may undergo dissolution and fragmentation with time (32). Thus, in unusual cases it may be difficult to demonstrate fibers or asbestos bodies in the histologic preparation. When that is the case, 5 to 10 additional sections from the same block, and 5 to 10 additional new blocks from areas with fibrosis, should be prepared, stained, and surveyed for asbestos bodies. If they are not found, the diagnosis of asbestosis is unlikely.
4. Even in the absence of a history of asbestos exposure, the presence of several or more asbestos bodies in areas of extensive interstitial fibrosis is sufficient evidence for a morphologic diagnosis of asbestosis.
5. Not everyone who inhales an asbestos fiber, or even a few fibers, develops even microscopic asbestosis. Normal lung defense mechanisms remove fibers via several well-described mechanisms.

The Pneumoconiosis Committee of the American College of Pathologists and the National Institute for Occupational Safety and Health dealt with these considerations when formulating the following statement with which we concur:

> "The criteria that permit the pathologist to establish the diagnosis of asbestosis have evolved during a review of many cases of the disease. Presently, the minimal features that permit the

diagnosis are the demonstration of discrete foci of fibrosis in the walls of respiratory bronchioles associated with accumulations of asbestos bodies. These morphologic findings, although adequate to establish the diagnosis of asbestosis in an early evolutionary stage, have not been shown to result in functional and radiologic alterations. The demonstration of asbestos bodies in the absence of fibrosis is insufficient evidence to justify the diagnosis of asbestosis. Conversely, a definite diagnosis of asbestosis cannot be made by the pathologist in cases that show characteristic fibrosis in the absence of asbestos bodies or other evidence of fibers. Because asbestos bodies are unevenly distributed in tissue, an adequate number of samples should be examined thoroughly." (32)

They further state that although the demonstration of asbestos fibers by the electron microscopic study of tissue digestates provides evidence of exposure, ultrastructural technique alone cannot be used to establish definitively the etiologic role of asbestos in disease (32).

This Committee has published guidelines for methods of assessing lung fiber concentration and pathologic grading of asbestosis. The certainty of the cause and effect relationship of asbestos to the fibrotic process increases with increasing numbers of such particles and fibers visualized by light microscopy. Electron microscopy of digested lung preparation from documented cases of asbestosis shows very large numbers of uncoated fiber fragments (37).

Since asbestos bodies and fibers appear in lungs without evidence of asbestos-related disease, the question arises as to how many such bodies are necessary to infer a cause and effect relationship between asbestos particles and fibrosis. No precise answer exists, but efforts to quantify the numbers of asbestos particles in known cases indicate that it is high. In the cases of asbestosis studied by Whitwell, the lungs nearly always showed three million light visible fibers per gram; control lungs generally show less than 20,000 fibers per gram (38).

Electron microscopy is a more sensitive index of asbestos exposure than light microscopy. It will detect 10 to 100 times more fibers than seen by light microscopy. Fibers seen only by light or electron microscopy, in the absence of parenchymal fibrosis, indicates only that exposure to asbestos has occurred. Additional studies are required to define the number of fibers in the lungs of persons with a variety of occupational exposures, and with varying periods of exposure, as well as in nonoccupational populations.

Investigations have demonstrated that as many as a million fibers per gram of dry tissue of chrysotile may be present in the lungs from nonoccupational exposures in the general population. By contrast, lungs containing a million fibers of amosite or crocidolite per gram are considered to reflect substantial occupational exposure to asbestos dust (39). An electron microscopic field of necessity represents a small sample of the lung and analysis of multiple fields is required to reflect the true asbestos lung burden. In our opinion, additional studies on the numbers and types of asbestos fibers in the lungs of control and exposed persons must be performed to provide the informational bases for interpretation of quantitative data concerning asbestos fibers in the lung and their relation to the presence of asbestosis or other asbestos related diseases.

Exposure History

Numerous studies have shown that asbestosis has a relatively close association with both the magnitude and the duration of exposure to inhaled asbestos; the more intense and longer the exposure, the greater the numbers of affected workers and the greater the severity of their illness. There is no evidence that casual or indirect exposure, such as occurs in the general population, causes asbestosis. The major problem facing the clinician is to assess whether an exposure has been sufficient to cause disease. Although dust levels have been measured in many industries for many years, they are not usually available to or easily interpreted by clinicians. Nevertheless, some general statements can be made. A careful sequential history of all exposures to all potentially harmful substances is obviously important. Particular attention should be paid to occupations in which direct contact with asbestos has occurred. Consultation with physicians trained in occupational medicine or with industrial hygienists may be helpful in unclear cases.

Evidence of asbestosis has been found many years after relatively brief but extremely heavy exposure. Such exposure often occurred in the asbestos textile industry over 50 years ago and has occurred more recently in workers who have not used respiratory protection while spraying dry asbestos on steel beams. Fortunately, such exposure is rare at this time. With levels of exposure common in the past few decades, the latent period between the state of the exposure and the discovery of the manifestations of asbestosis is likely to be a minimum of 15 years, and more often considerably longer. With exposures below the current recommended permissible exposure limit value, asbestosis is not likely to be found during the course of a working career. With proper engineering controls, work practice, and where necessary, personal respiratory protective devices, asbestosis should not occur.

Clinical Diagnosis

In the usual clinical setting, the diagnosis of asbestosis has to be made in the absence of histologic examination of lung tissue. Open lung biopsy is rarely indicated in the assessment of workers for compensation purposes. The benefit of the doubt should be given whenever the clinical features and occupational exposure data are compatible with the diagnosis. In most instances, the clinician and epidemiologist must still rely on indirect methods of diagnosing asbestosis. These principles of diagnosis are based on observations from pathologically proven cases. When biopsy is done, careful attention must be paid to the sampling considerations mentioned above and the surgical technique employed (40). Assessment of lung dust burden in such biopsy material is desirable. Diagnosis of asbestosis does not mean that measurable impairment of lung function or physical disability is necessarily present.

Clinical Features

In advanced stages, asbestosis is a restrictive lung disease associated with dyspnea, clubbing of the fingers, basilar crackles and widespread irregular opacifications on roentgenograms. The latter are usually more prominent at the lung bases. Pleural thickening and calcification may also be present as noted above. The vital capacity is usually reduced with preservation of the FEV_1/FVC ratio and gas exchange impaired. Cor pulmonale may occur in advanced disease. When many or all of these features are present the diagnosis is made without difficulty. However, in the absence of the opportunity to examine lung tissue microscopically, the diagnosis is always inferential. The certainty increases with increasing numbers and severity of typical clinical abnormalities.

The chest roentgenogram appears to be the most valuable examination in diagnosing asbestosis. A diffuse irregular interstitial pattern coupled with evidence of pleural disease, e.g., plaques or extensive pleural thickening in a person with known exposure, presents little diagnostic difficulty. The difficulty with the use of the chest roentgenogram relates to the detection of lesser degrees of interstitial fibrosis. Efforts have been made to standardize the interpretation of roentgenograms in the pneumoconioses. The most widely accepted and extensively studied method for assessing the degree of roentgenologic involvement in the pneumoconioses was developed by the International Labour Office and is currently called the ILO-1980 Classification (41). This scheme evolved from studies of miners and focused initially on the detection of silicosis. The X-ray appearance of silicosis is characterized initially by small rounded opacifications. The classification was later broadened to describe abnormalities which occur in asbestosis and do not have a rounded appearance. These are fine, medium and coarse, small irregular opacifications, and they are called s, t, and u, respectively. The classification, originally developed for describing radiologic changes in epidemiologic studies, has also been used in the clinical context for case detection and/or diagnosis. In the latter instance, the information given in the chest radiograph is added to all other information about the individual in order to arrive at a diagnosis.

The number of these abnormalities in a given area of the chest film, whether rounded or irregular, is called their profusion. The profusion was initially graded as 0 for none, 1 for slight, 2 for moderate, and 3 for severe.

It became apparent, however, that even experienced readers had difficulty in grading opacifications into these categories in a reproducible fashion. However, if observers were asked to give two classifications, i.e., the one category they thought was most likely and another which they thought might also be considered, the observer reliability (i.e., in terms of reproducibility) was considerably improved. This method of giving the observer two options (the one he thought most likely and next most likely) was called the expanded classification. It formed a 12-point scale that has proven to be very useful epidemiologically.

It is likely that an individual who develops asbestosis moves more or less uniformly from the normal roentgenologic appearances (-/0, 0/0, 0/1) to the abnormal (1/2, 2/1, 2/2, etc.). The problem is that the interpretation of the lesser degrees of abnormality on this scale is subjective and that numerous causes of such roentgenologic shadowing other than asbestosis exist. In the presence of marked diffuse pleural thickening, it is difficult to diagnose or grade the severity of interstitial fibrosis. Accordingly, criteria other than roentgenographic ones have been sought.

Dyspnea

Asbestosis has been described as a monosymptomatic disease, dyspnea being the major complaint of the affected individual (42). There is no doubt that shortness of breath is common and troublesome in individuals with clinically significant interstitial fibrosis. Dyspnea, however, is a nonspecific symptom, common in many other cardiopulmonary disorders, and it is particularly subject to emotional factors likely to be relevant in instances of suspected industrially-related disease. Accordingly, it is not adequate to use dyspnea as the only evidence on which to base a clinical diagnosis of asbestosis in an individual at risk.

Clubbing

Clubbing of the fingers occurs more commonly in asbestos-exposed workers than in controls (43, 44, 45). The diagnostic usefulness of clubbing is limited, however, by two important considerations. There are many other causes of clubbing and clubbing, when present, is a late finding in pulmonary asbestosis (46). Since the majority of persons with significant asbestosis do not have clubbing, and asbestos workers with clubbing may have it for reasons other than pulmonary fibrosis, the diagnostic usefulness of clubbing is limited.

Basilar Crackles

Crackles have been recognized as a feature of asbestosis for over 50 years and are believed by many to be an early finding (47, 48, 49). They have been described as characteristic in their sound ("fine," "cellophane," "velcro," "close to the ear") and in their bilateral, basilar distribution (50). They differ in quality and timing from the crackles of bronchitis which tend to be fewer in number and earlier in timing. Bronchitic crackling begins with the beginning of inspiration and usually discontinues prior to mid or late inspiration. Characteristically, the crackles of interstitial fibrosis are pan inspiratory or have an end inspiratory accentuation. They appear first at the bases in the mid-axillary lines and tend to spread toward the posterior bases. As the disease advances, the crackles become distributed at progressively high levels up from the bases (50). They are often difficult to distinguish from the crackles of congestive heart failure. Reported rates vary, but about half of the persons considered to have asbestosis on clinical grounds have crackles (47, 51, 52, 53); prevalences in exposed populations range from about 10–20%. Such prevalences depend on duration of exposure, the age of the population, and prevalence of other diseases causing fine crackles. Observer variability exists in chest auscultation, but this can be reduced by training and waveform analysis (54, 55, 56). In summary, under carefully controlled circumstances crackles can be useful in diagnosing interstitial fibrosis. However, they are not also specific for the interstitial fibrosis related to asbestos.

Pulmonary Function

The characteristic features of pulmonary asbestosis are those of a restrictive lung disease, i.e., a reduction in lung volumes, with inspiratory capacity and vital capacity being primarily affected, functional residual capacity being less affected, and residual volume even less. These changes are consistent with a decrease in pulmonary compliance. Hypoxemia may be present at rest or develop with exercise. Diffusing capacity is also usually impaired, depending on the extent of the disease. By contrast, large airway function as reflected in the FEV_1/FVC ratio is generally well preserved. Review of the prediction formulas for pulmonary function tests reveals there is no one set applicable to all laboratories and patient populations. Predicted normal values used in pulmonary function laboratories should be based on regression equations from studies whose testing equipment, methodologies and control populations most clearly resemble the patients under study. Numerous studies have shown that the effects of asbestos on lung function are dose related (57, 58, 59).

There is convincing evidence that an asbestos related pulmonary abnormality can occur in the absence of definite radiologic change. These pathologic changes of early asbestosis have been demonstrated in biopsy material from asbestos-exposed individuals with minimal or no radiologic abnormality (60). Likewise, exposure response relationships for certain pulmonary function abnormalities (including reduced lung compliance and impaired flow at low lung volumes) have been demonstrated in asbestos-exposed subjects without radiologic abnormalities or reduction in vital capacity (58), and their occurrence subsequently confirmed in large animal models with biopsy confirmation of the associated pathologic changes. The impairment associated with such abnormality is usually modest.

Diffusing Capacity

Diffusing capacity or transfer factor has been the subject of numerous studies with somewhat conflicting results. In most studies of unexposed populations it is lower in asbestos exposed workers than in normal controls although not always at a statistically significant level (44, 45, 57, 62, 63). There is not always a clear relationship to dust exposure indices (58, 64). It has, however, been shown to correlate with the severity of the histologic lesion in interstitial fibrosis (64), and its reduction can precede roentgenologic abnormalities. At this time a reduction of the diffusing capacity in an asbestos worker, in the absence of other known causes for impaired gas exchange, would provide suggestive evidence for asbestosis, but further population studies are necessary to elucidate the precise role of this test.

Other Studies

Various other measurements have been employed for monitoring persons exposed to asbestos. A reduction in roentgenologic lung volume appears promising since it is applicable to serial studies of patients, but it requires careful control of inspiration (66). This approach has not yet been taken in a large number of subjects exposed to asbestos.

Inspiratory capacity was shown to be suitable for surveillance of workers but is not likely to add much more than vital capacity (VC) as the two tests are highly correlated (59). This finding suggests that tests for small airways disease might in the future be applicable to early detection (67). In one study, neither closing volume nor closing capacity correlated with the duration of exposure or with the asbestos dust index (68). Gallium scanning, bronchoalveolar lavage, and transbronchial biopsy need further evaluation with respect to their usefulness in diagnosing asbestosis. CT scanning is of particular value in detecting and quantitating pleural disease and aiding in the differentiation of pleural from parenchymal disease. The value of CT scanning in the detection of interstitial fibrosis also needs to be further evaluated. Thus, at this time, criteria other than crackles, restrictive lung functional abnormality, reduced diffusing capacity of the lung (D_L), and roentgenogram consistent with interstitial fibrosis of 1/1 or more are either impractical, of unproven value, or are not likely to yield additional information because of their high correlation with one of these four.

In our opinion, combinations of these abnormalities are more reliable in terms of specificity, relation to duration of exposure, consistency, and predictive value; however, little work has assessed combinations of abnormalities.

Combinations

Combinations of abnormal test results are not

likely to prove more effective in detecting the earliest changes in asbestosis. Intuitively, one test is likely to become abnormal first. It is likely that the first abnormality is not always the same one (e.g., one worker may have only an abnormal D_L [69], and another only crackles as the first manifestation). This has been demonstrated with respect to restrictive lung function pattern and reduced D_L. It is likely that observations will have to be made in large groups over long periods to delineate clearly the best single test for early diagnosis of asbestosis if, indeed, a single initial abnormality exists.

Differential Diagnosis

Streaky densities on chest films consistent with a parenchymal disease have many causes. All alternative diagnoses must be considered before accepting the presumptive diagnosis of asbestosis.

Occasionally, asbestosis is coexistent with chronic obstructive pulmonary disease. Evidence is accumulating that obstruction may also be related to an individual's occupational exposure (70). The relative importance of cigarette smoking and asbestos in the development of the combined problem of restrictive and obstructive disease may be difficult or even impossible to assess.

Since a not uncommon feature of asbestos exposure is bilateral pleural thickening, the question arises as to the helpfulness of such thickening in indicating that a patient with pulmonary fibrosis has asbestosis. Indeed, asbestos appears to be a rather potent stimulus for the development of pleural abnormalities. Selikoff found pleural fibrosis in 65% of persons he studied 40 years from the onset of their initial exposure to asbestos. With patients with no known exposure to asbestos or other known hazardous materials (71), the question arises as to whether an indirect or occult exposure to asbestos might have caused the pleural thickening. Severe diffuse pleural thickening is not common even in asbestos exposure. It was present in only 2.5% of the asbestos workers studied by Selikoff. Since there were no controls in that study, it is difficult to be certain that asbestos was the cause of the pleural fibrosis in those subjects. Indeed, Gilson reported in 1969 that pleural thickening was found on 187 of 3,860 (0.05%) routine roentgenograms of the chest in Great Britain (72). He conducted one of the few objective studies of pleural thickening by comparing the asbestos exposure of 113 of the 187 subjects with that of 113 age- and sex-matched controls. He found "a slight but unimpressive excess of positive histories of exposure to asbestos among the cases." Thus, it is not necessary to assume an occult exposure to asbestos in every instance of pleural thickening; the presence of pleural thickening is not definitive evidence of asbestos exposure. In another study, bilateral pleural thickening was found in 52 of 824 consecutive patients admitted to the hospital. Only 13 of these 52 had definite asbestos exposure as compared to 2 of 32 age-matched controls. In contrast to the relatively nonspecific finding of pleural thickening, the demonstration of pleural plaques with or without calcification is better evidence of asbestos exposure. Unfortunately, the latter usually occurs only many years after the onset of the exposure, thus limiting usefulness of early diagnosis.

A major problem exists in the differential diagnosis when more than one disease is present, whether it is congestive heart failure, COPD, or other chronic lung disease. There are diseases unrelated to asbestos exposure but with similar symptoms, and these may occur in some persons with asbestos exposure. However, given a clear history of exposure to asbestos, a diffuse interstitial fibrosis can be presumed to be due to the asbestos as other forms of interstitial fibrosis are relatively uncommon. The prevalence of lesser degrees of interstitial fibrosis is not well known and considerable caution has to be exercised in attributing all such phenomena to asbestos exposure, either known or occult.

Summary

This document has focused on clinically detectable interstitial fibrosis due to asbestos exposure. While direct examination of lung tissue is the most reliable method of diagnosis, as stated above, this is rarely indicated in the assessment of workers for compensation purposes. Open lung biopsy is indicated in our opinion only when a clear health, rather than financial, benefit is likely to be provided. In the absence of pathologic examination of lung tissue, the diagnosis of asbestosis is a judgement based on a careful consideration of all relevant clinical findings. In our opinion, it is necessary that there be:

1. A reliable history of exposure.
2. An appropriate time interval between exposure and detection (see pages 9-10)

Furthermore, we regard the following clinical criteria to be of recognized value:

1. Chest roentgenographic evidence of type "s," "t," "u," small irregular opacifications of a profusion of 1/1 or greater
2. A restrictive pattern of lung impairment with a forced vital capacity below the lower limit of normal
3. A diffusing capacity below the lower limit of normal
4. Bilateral late or pan inspiratory crackles at the posterior lung bases not cleared by cough

Of these, the findings on the chest roentgenogram are the most important. When this criteria is not met, considerable caution is warranted. The specificity of the above criteria increases with increasing numbers of positive criteria. As in all clinical judgments, confounding variables, such as the presence of other clinical conditions that affect these criteria, should be evaluated.

It is possible that interstitial fibrosis may be present even though none of these criteria are satisfied, but, in our opinion, in these circumstances the clinical diagnosis cannot be made.

This statement was prepared by an Ad Hoc Committee of the Scientific Assembly on Environmental and Occupational Health in conjunction with the American College of Chest Physicians. The members of the Committee were:

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References

1. Hamilton A, Hardy H. Industrial toxicology. Ed. 3, Publishing Sciences Group Inc., Acton, MA, 1974; 421.
2. Gilson GC. Asbestos cancer: Past and future hazards. Proc R Soc Med 1973; 66: 395.
3. Zussman J. Asbestos: Nature and history. Rev. Ciba Geigy 1972; 2: 3.
4. Smither WJ. Asbestos and asbestosis. Ann Occup Hyg 1970; 13: 3.
5. Biological effects of asbestos. Selikoff IJ, Churg J. Co-chairmen, Proceedings of a conference held at the New York Academy of Sciences, Oct. 19-21, 1964. Ann N Y Acad Sci, 1965; 132:1-766.
6. Biological effects of asbestos. Anspach M, Chairman Deutsches Zentralinstitut fur Arbeitsmedizin: Gesellschaft fur Arbeitshygiene and Arbeitsschutz in der DDR. Dresden (April 22-25) 1968; 1-312.
7. Biological effects of asbestos. Bogovski P; Gilson JG; Timbrell V, Wagner JC, eds. Proceedings of a working conference at IARC, Lyon, October 2-6, 1972, IARC Scientific Publications No. 8, Lyon 1973; 1-341.
8. Proceedings of the Pneumoconiosis Conference. Johannesburg, South Africa, February 1959, Orenstein AJ, ed, J and A Churchill Ltd., London 1960; 1-629.
9. Pneumoconiosis. Shapiro HA, ed, Proceedings of the International Conference, Johannesburg, South Africa, 1969, Oxford University Press, Cape Town, 1970; 3-645.
10. Parkes WR. Asbestos-related disorders. Brit J Dis Chest 1973; 67:261.
11. Stell PM, McGill T. Asbestos and laryngeal carcinoma. Lancet 1973; 2: 416.
12. Newhouse ML, Berry G. Asbestos and laryngeal carcinoma. Lancet, 1973, 2, 615.
13. Libshitz HI, Wershba MS, Atkinson GW, Southard ME. Asbestos and carcinoma of the larynx. JAMA 1974; 228: 1571.
14. Guidotti TL, Abraham JL, DeNee PB. Asbestos exposure and cancer of the larynx. West J Med 1975; 122: 75.
15. Graham J, Graham R. Ovarian cancer and asbestos. Environ Res 1967; 1: 115.
16. Doniach I, Swettenham KV, Hathorn MKS. Prevalence of asbestos bodies in a necropsy series in East London: Association with disease, occupation, and domiciliary address. Br J Ind Med 1975; 32: 16.
17. Weiss W. Clinical epidemiology. Arch Environ. Health 1970; 20: 5.
18. Speil S, Leineweber JP. Asbestos minerals in

modern technology. Environ Res 1969; 2: 166.

19. Wright GW. Asbestos and health in 1969. Am Rev Respir Dis 1969; 100: 467.

20. Harries PG. Asbestos dust concentrations in ship repairing: A practical approach to improving asbestos hygiene in naval dockyards. Ann Occup Hyg 1971; 14: 241.

21. Respiratory Diseases: Task Force Report on Problems, Research Approaches, Needs. The Lung Program, National Heart and Lung Institute, October 1972, U. S. Department of Health, Education and Welfare.

22. Lee GL, Smith DJ. Steelwork insulated with sprayed crocidolite asbestos: Controlling a potential hazard. Ann Occup Hyg 1974; 17: 49.

23. Meurman L. Asbestos bodies in pleural plaques in a Finnish series of autopsy cases. Acta Pathol Microbiol Scand 1966; (Supplement) 181: 8.

24. Mattson SB, Ringqvist T. Pleural plaques and exposure to asbestos. Scand J Respir Dis 1970; (Supplement) 75: 4.

25. Lewinsohn HC. Early malignant changes in pleural plaques due to asbestos exposure: A case report. Brit J Dis Chest 1974; 68:121.

26. Roberts GH: The pathology of parietal pleural plaques. J Clin Path 1971; 24:348.

27. Hourihane DO, Lessof L, Richardson PC. Hyaline and calcified pleural plaques as an index of exposure to asbestos: A study of radiological and pathological features of 100 cases with a consideration of epidemiology. Brit Med J 1966; 1:1069.

28. Le Bouffant: Biological effects of asbestos. Bogovski P, Gilson JC, Timbrell V, Wagner JC, eds. Proceedings of a working conference at IARC, Lyon, October 2-6, IARC Scientific Publications, No. 8, Lyon, 1973; 249.

29. Hillerdal G: Non-malignant asbestos pleural disease. Thorax 1981; (Sept) 36(9):669–675.

30. Hourihane DO, McCaughey WTE. Pathological aspects of asbestosis. Postgrad Med J 1966; 42:613.

31. Heard BE, Williams R. The pathology of asbestosis with reference to lung function. Thorax 1961; 16:264.

32. The Pathology of Asbestos-Associated Disease of the Lungs and Pleural Cavities: Diagnostic criteria and proposed grading schema. Craighead JE, Chairman, Report of the Pneumoconiosis Committee of the College of American Pathologists and the National Institute for Occupational Safety and Health. Arch Pathol Lab Med 1982; 106:544.

33. Solomon A, Goldstein B, Webster I, Sluis-Cremer GK. Massive fibrosis in asbestosis. Environ Res 1971; 4:430.

34. Johnson NF, Davies R. An ultra structural study of the effects of asbestos fibers on cultured peritoneal macrophages. Brit J Exptl Pathology 1981; 62:559–70.

35. Gaensler EA, Kaplan AI. Asbestos pleural effusion. Ann Intern Med 1971; 74:178.

36. Gaensler EA, Carrington CB, Coutu RE, Tomasian A, Hoffman L, Smith AA. Pathological, physiological and radiological correlations in the pneumoconioses. Ann N Y Acad Sci 1972; 200:574.

37. Miller A, Langer AM, Tierstein AS, Selikoff IJ. "Non-specific" interstitial pulmonary fibrosis: Association with asbestos fibers detected by electron microscopy. N Engl J Med 1975; 292:91.

38. Whitwell F, Scott J, Grimshaw M. Relationships between occupations and asbestos fibre content of the lungs in patients with pleural mesothelioma, lung cancer and other diseases. Thorax 1977; 32:377–86.

39. Churg A. Fiber counting and analysis in the diagnosis of asbestos-related disease. Human Pathology 1982; (April 13) 4:382–92.

40. Gaensler EA, Carrington CB. Open biopsy for a chronic diffuse infiltrative lung disease: Clinical, roentgenographic and physiological correlations in 502 patients. Ann Thorac Surg 1980; 30:411–26.

41. International Labour Office. Guidelines for the Use of ILO International Classification of Radiographs of Pneumoconioses. Rev. Ed. 1980. Occupational Safety and Health Series. No. 22, International Labour Office, Geneva, 1980.

42. Selikoff IJ, Churg J, Hammond EC. The occurrence of asbestosis among insulation workers in the United States. Biological Effects of Asbestos. Ann N Y Acad Sci 1965; 132:139.

43. Kleinfeld M, Massite J, Kooyman O, et al: Effect of asbestos dust inhalation on lung function. Arch Environ Health 1966; 12:741.

44. Regan GM, Tagg B, Walford J, et al. The relative importance of clinical, radiological and pulmonary function variables in evaluating asbestosis and chronic obstructive airway disease in asbestos workers. Clin Sci 1971; 41:569.

45. Wallace WFM, Langlands JHM. Insulation workers in Belfast. Comparison of a random sample with a control population. Br J Ind Med 1971; 28:211.

46. El-Sewefy AZ, Awad S, Abdel-Salam MS. Chest symptomatology in an Egyptian cement-asbestos pipe factory (a field survey). J Egypt Med Assoc 1970; 53:84.

47. Epler GR, Gaensler EA, Carrington CB. Crackles (rales) in the interstitial pulmonary diseases. Chest 1978; 73:333–39.

48. Shirai F, Kudoh S, Shubuya A, Sada K, Mikami R. Crackles in asbestos workers: Auscultation and lung sound analysis. Br J Dis Chest 1981; 75:386–96.

49. Elmes PC. Health parameters other than mesothelioma. Proceedings of Asbestos Symposium, Johannesburg, South Africa, October 3-7, 1977. National Institute for Metalllurgy, Randburg 1978; 9–16.

50. Smither WJ. Secular changes in asbestosis in an asbestos factory. Biological Effects of Asbestosis. Ann N Y Acad Sci 1965; 132:166.

51. Rossiter CE, Berry G. The interaction of asbestos exposure and smoking on respiratory health. Bull Europ Physiopath Resp 1978; 14:197–204.

52. Huuskonen MS. Clinical features, mortality and survival of patients with asbestosis. Scand J Work Environ & Health 1978; 4:265–74.

53. Segarra F, Baselga MM, Lopes IP, Perez NJ. Asbestosis in a Barcelona fibrocement factory. Environ Research 1980; 23:292–300.

54. Workum P, Del Bono EA, Holford SK, Murphy RLH. Accuracy of a technician in chest auscultation for crackles. Submitted for publication to Chest 1985.

55. Murphy RLH, Gaensler EA, Holford SK, Del Bono EA, Epler GR. Crackles in the early detection of asbestosis. Am Rev Respir Dis 1984; 129: 375–79.

56. Murphy RLH, Holford SK, Knowler WC. Visual lung sound characterization by time-expanded wave-form analysis. N Engl J Med 1977; 296:968–71.

57. Murphy RLH, Gaensler EA, Ferris BG, Fitzgerald M, Sollidayl N, Morrisey W. Diagnosis of "Asbestosis." Observations from a longitudinal survey of shipyard pipe coverers. Am J Med 1978; 65:488–98.

58. Weill H, Ziskind MM, Waggenspack C, et al. Lung function consequences of dust exposure in asbestos cement manufacturing plants. Arch Environ Health 1975; 30:88.

59. Becklake MR, Fournier-Massey G, Rossiter C, et al. Lung function in chrysotile asbestos mine and mill workers of Quebec. Arch Environ Health 1972; 24:401.

60. Epler GR, McLoud TC, Gaensler EA, Mikus JP, Carington CB. Normal chest roentgenograms in chronic diffuse infiltrate lung disease. N Engl J Med 1978; 298:934–39.

61. Jodoin G, Gibbs W, Macklem PT, McDonald JC, Becklake M. Early effects of asbestos exposure on lung function. Am Rev Respir Dis 1971; 104:525.

62. Murphy RLH, Ferris BG, Burgess WA, et al. Effects of low concentrations of asbestos. Clinical, environmental, radiologic and epidemiologic observations in shipyard pipe coverers and controls. N Engl J Med 1971; 285:1271.

63. Langland JAM, Wallace W, Simpson M. Industrial workers in Belfast. 2. Morbidity in men still at work. Brit J Ind Med 1971; 28:217–72.

64. Murphy RLH, Sorenson K. Chest auscultation in the diagnosis of pulmonary asbestosis. J Occ Med 1973; 15:272.

65. Chiappino G, Zedda S. Value and significance of some diagnostic parameters in workers exposed to asbestos. Securitas Anno (Italy) 1973; 58:841. 27:163.

66. Reger RB, Young A, Morgan WKC. An accurate and rapid method of determining total lung capacity. Thorax 1972; 27:163.

67. Jodoin G, Gibbs GW, Macklem PT, et al. Early effects of asbestos exposure on lung function. Am Rev Respir Dis 1971; 104:525.

68. Konietzko N, Gerke E, Schlene H, et al. Closing volume in workers exposed to asbestos (German). Prax Pneumol 1974; 28:829.

69. Thomson ML, McGrath MW, Smither WJ, et al. Some abnormalities in measurement of pulmonary diffusion in asbestosis with chronic bronchitis and emphysema. Clin Sci 1961; 21:1.

70. Becklake MR. Asbestos exposure and airway disease. In: Gee JB, Morgan WK, Brooks SF, eds. Occupational lung disease. New York: Raven Press, 1984.

71. Selikoff IJ. The occurrence of pleural calcification among asbestos insulation workers. Biological effects of asbestos. Ann N Y Acad Sci 1965; 132:351–67.

72. Gilson JC. Asbestos Health Hazards: Recent observations in the United Kingdom. Proceedings of the International Conference on Pneumoconiosis, Johannesburg, South Africa, London: Oxford University Press, 1969.

THE DIAGNOSIS OF NONMALIGNANT DISEASES RELATED TO ASBESTOS

THIS OFFICIAL STATEMENT OF THE AMERICAN THORACIC SOCIETY WAS ADOPTED BY THE ATS BOARD OF DIRECTORS, MARCH 1986.

Objective

The health effects of asbestos have become a cause of serious concern in recent years. It has been estimated that from 1940 to 1979, in the United States alone, 27,500,000 individuals were exposed to this mineral at work. Recognition of the diseases caused by asbestos exposure has led manufacturers to reduce exposure in a variety of ways, such as by using alternative materials and by instituting improved work practices. It has also led to widespread public concern over the presence of asbestos in the environment, and fear on the part of persons with minimal exposure. This and projections of future asbestos related illness have posed important public policy questions—whether to remove all asbestos in public buildings and what to do about the enormous estimated legal liability. In this context, physicians are commonly asked for advice. Furthermore, they are also consulted regarding the diagnosis of an asbestos-related respiratory condition in an exposed individual. While abundant literature exists on the health effects of asbestos, there is much that is conflicting. Accordingly, this report has been prepared by a group of experts to present an authoritative consensus view of the current state of knowledge while pointing out areas where additional information is necessary. An attempt is made to summarize our present knowledge on the diagnosis of nonmalignant asbestos-related pulmonary disease and provide the sources on which the opinion is based.

Reprints may be requested from your state or local Lung Association

Asbestos—the Mineral

The generic term "asbestos" is used to describe a group of minerals which, when crushed, break into fibers rather than dust. They are hydrated fibrous silicates which have great tensile strength, heat resistance, acid resistance, and some varieties are also flexible. This weavable rock has numerous important uses in an industrial society, and world production and use climbed steadily since its commercial introduction in the late 19th century. Geology, mineralogy, and uses have been well described elsewhere (1, 2). World production of asbestos has dropped markedly since the mid 1970's. The cumulative production of asbestos, however, continues to increase. There has been a good deal of debate about the mineralogic definitions of asbestos and asbestiform fibers. The most complete recent discussion on trends in asbestos production and now widely accepted mineralogic definitions are found in the NAS Report on asbestiform fibers (3).

Asbestos in Lung Tissue

Inhaled asbestos that is retained in the lung can become coated with a proteinaceous iron staining material. The resulting asbestos or ferruginous body is a most characteristic index of asbestos exposure. It is usually recognized by its beaded-necklace or drumstick appearance. The longer fibers are more likely to become coated (2-22). The core of asbestos, i.e., bodies found in human lungs, is more likely to be an amphibole fiber than to be chrysotile, perhaps due to the greater ability of the former to survive in lung tissue, while chrysotile tends to disappear over time. The majority of the lung burden of asbestos, however, is uncoated and consists of short fibers, i.e., less than 5 microns (2-21). These are poorly visible or invisible on light microscopy but have been demonstrated by phase microscopy and electron microscopy. Asbestos bodies are commonly found in small numbers in lungs of city dwellers at routine autopsies in the absence of occupational exposure to asbestos and asbestos related illness. These are usually few in number and unassociated with pathologic abnormality in the parenchyma. This observation emphasizes the importance of developing standardized techniques to quantify the number and type of asbestos fibers in lung tissue (to be discussed below).

Benign Pleural Abnormalities Associated with Asbestos

Asbestos causes pleural plaques, pleural thickening and pleural effusion. Pleural plaques are discrete, elevated, opaque, shiny, rounded lesions. They characteristically occur on the posterolateral aspect of the lower parietal pleura or diaphragm, but usually not on the visceral pleura, at the costophrenic angles, or at the apices. Thin plaques are smooth and grayish-white. Thicker ones are ivory-colored or gray and may have either a smooth surface or bosselated surface, or be coarsely nodular with the consistency of cartilage. Plaques are of two types, diffuse or nodular, and elevated. They can vary in size and shape. On inspection of gross specimens, calcification may be present but is not common. Microscopically, plaques are seen to be laminated collagenous connective tissue, acellular, with few inflammatory or fibrocytic nuclei; many are covered by a thin layer of regular and well-differentiated mesothelial cells. Capillaries are rare (23, 24). Elastic staining shows intact lamellae be-

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12 July, 1982

Dr H.C. Lewinsohn,
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Dear Hilton,

Thank you for your letter of January 27th together with the papers by Nicholson and Schneiderman. I am sorry that I have been unable to write about these before. I think Nicholson's paper is impressive although inevitably it depends on a number of uncertainties because of the lack of definite hard data on exposive levels, job histories, etc. even in exposed groups that have been studied extensively, let alone in the total group to which he extrapolates. But the results in figures 5, 6 and 7 do not surprise me greatly; they could be overestimates but not by large factor. These figures are mainly a prediction into the future based on what has already been observed. With an increasing number of mesothelianas being observed over the last 20 years it is reasonable to suppose that the increase will continue for sometime. The length of time before there is a downtown would depend on whether there was a change in the numbers exposed with time and on changes in dust levels. The main point is that the Bridbord et al estimate is shown to be far too high, and although most people in the trade never believed Bridbord's estimates it is good to have this confirmed by calculations based on reasonable assumptions, despite the uncertainties that must remain.

Incidentally Molly Newhouse and I are doing an update on the Cape group. The number of new mesothelianas since 1976 is less than we predicted in our 1976 paper. This used methods similar to Nicholson's applied just to our own group. At this stage we can't make too much of the reduced number but let us hope it continues.

I am afraid I find it difficult to get excited over Schneiderman's paper. It is so far removed from any data, and discussion of levels necessary for lifetime risks of 1 per million seems even further removed from any reasonable assessment of priorities in reducing mortality. I expect Schneiderman was working to a brief of questions and I think that WHO are keen on the 1 in a million criterion.

With best wishes.

Yours sincerely,

G. Berry
Associate Professor of Biostatistics