American Academy of Occupational Medicine

Asbestos as a Hazard to Health

Fact and Speculation

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A RESURGENCE of interest in the asbestos minerals as environmental hazards to health has arisen because of increasing world production of asbestos, successive indications of associations between asbestos and pulmonary fibrosis and various malignancies, and, more recently, the demonstration of asbestos or asbestoid bodies in presumably nonexposed populations.

The major asbestos minerals of commerce are chrysotile, crocidolite, amosite, and anthophyllite, with tremolite and actinolite being of considerably less importance. The properties of these various fibrous minerals have led to an extraordinary variety of uses, so that world production has increased from a few thousand tons in 1900, to 1.3 million tons in 1950, to over 3.5 million tons in 1965. Over 90% of this is chrysotile. The United States uses about one fourth of world production, practically all imported from Canada or Africa.[1]

Asbestosis

The first facts to be discussed will be those relating to asbestos pneumoconiosis, the disease that has come to be known as asbestosis. No one disputes a cause-and-effect relationship here; but we are still deficient in our knowledge of pathogenesis.

Are all forms of asbestos equally hazardous? The evidence is against this. Chrysotile disappears more readily from the lungs than other forms and in animals seems to be less fibrogenic. The prominence of pleural calcification varies from area to area and type to type. For example, in Finland, Rannio[2] has recently reported a review of 600,000 chest x-ray films which show that in communes where anthophyllite mines are located, up to 9% of films showed pleural calcifications, whereas in other parts of East Finland not bordering on producing communes the prevalence was only 0.5% per thousand. Nothing comparable to this has been reported in other asbestos-mining areas.

A second question regarding asbestosis relates to its prevention by industrial hygiene. Is it true that all that is needed is the application of currently recommended standards for dust control? Most people who have looked into the basis for the present threshold limit value of 5 million particles per cubic foot for asbestos, which has been recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) since 1946, realize that it rests on shakier evidence than most. Midget impinger sampling used in determining dust concentrations include all dusts; and the asbestos-containing dust that is counted is mainly grains, although, rarely, some fibers are included. A large proportion of asbestos

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fibers have diameters below the resolving power of the light microscope and are not counted at all. Nevertheless, most industrial hygienists have felt that the impinger count is a good indirect measure of dust control and that dust counts averaging 5 million particles per cubic foot (mppcf) should control asbestosis. Evidence is beginning to develop that this is not true. In 1965, Wells[7] reported briefly on his observations over 30 years in asbestos textiles. His view was that multiplying average counts by years of exposure provided a good rough guide and that after 50- to 60-mppcf years workers began to show evidence of asbestosis. At the threshold limit value (TVL), this would be reached in 10 to 12 years. In 1966, Ferris examined insulating workers in a New England shipyard that had been surveyed by Fleischer *et al*[4] 20 years earlier. His preliminary results indicate that conditions and counts now appear comparable to those of 20 years ago and average near 5 mppcf (B.G. Ferris, oral communication, Dec 5, 1966). Nevertheless, a large proportion of workers had radiographic asbestosis, which had begun to appear as early as 10 to 12 years before. Marr's counts[8] in shipyards, in 1964, did not indicate weighted averages above 5 mppcf; and current studies of insulating workers in San Francisco bear this out (J.L. Balser and W.C. Cooper, unpublished data, 1967). Nevertheless, asbestosis appears, which suggests that a time-weighted average of 5 mppcf may be too high. This is consistent with actual practices in the better controlled asbestos industries. Mitchell,[9] reporting in 1961 on improved conditions in North Carolina textile mills, cited counts ranging from 0.4 to 4.6 mppcf. English industries under Asbestos Industry Regulations have never operated with a numerical standard, instead depending upon enclosure, ventilation, and masks to keep exposures as near zero as possible.

There are numerous other questions that one can ask relative to asbestos pneumoconiosis. Have we been too complacent in assuming that the expectoration of large numbers of asbestos bodies is the expected response of an asbestos worker, or is this in the new employee really an indicator that we are tolerating too much exposure? I do not think we know the answer. There are also fundamental questions on pathogenesis, on the nature of the pulmonary vascular impairment, and the factors determining susceptibility.

Malignancies

Asbestos would not be in the headlines if asbestosis, affecting a few hundred miners, millers, and insulating workers, were the only problem. It is the more recently demonstrated association between asbestos and malignancies that has created an entirely new series of facts, fantasies, fallacies, and fancies. Let us first look at the evidence.

That there is an increased incidence of lung cancer in many groups of workers exposed to asbestos is a fact. The cumulative evidence from epidemiologic studies (Table 1) that began with Merewether's analysis[1] of causes of death in certified cases of asbestosis in the United Kingdom from 1924 to 1947 is now overwhelming.

The evidence is not convincing as to the relative importance of different types of asbestos. Chrysotile seems less certainly implicated; and one looks forward with interest to an up-dating of the experience of Quebec chrysotile miners who, several years ago, when analyzed by Braun and Truan[11] appeared to have no excess risk. It is of interest that Jacob and Anspach[16] who a few years ago could demonstrate no increased risk in Dresden asbestos workers now find twice the expected number of cases of lung cancer in men and ten times the expected number in women. Their exposures were mixed, and we do not have the answer to whether the association is truly less for chrysotile than it is for crocidolite and amosite.

The next question, and this will apply to other malignancies to be mentioned later, is whether or not asbestos minerals per se are carcinogenic. A promising area of current research is aimed at determining the role of the asbestos fibers and fibrils as carriers of trace metals or of carcinogenic chemicals, such as the polycyclic hydrocarbons, to vulnerable sites. The idea of a fibril that can migrate and then become a semipermanent implant is an intriguing one. This falls into the area of speculation, or fancy if you wish to call it that, but one that may prove productive.

Table 1.—*Epidemiologic Studies of Asbestos Exposure and Lung Cancer*

Place	Population Studied	No.	Years Followed	No. With Lung Cancer	Comparison Groups
United Kingdom[2]	Reported deaths from asbestosis	239	1924-1947	31/239 (13.1%)	Silicosis (1.32%)
United Kingdom[3]	Cases of asbestosis in 1247 autopsies with pneumoconiosis	121	...	17/121 (14.1%)	Silicosis (6.9%)
United Kingdom[4]	Asbestos textile workers, dusty areas, 20 yrs or more exposure	113	1922-1953	11/39 deaths	0.8 expected
United Kingdom[5]	Reported deaths from asbestosis	365	1924-1955	65/365* (17.8%)	...
Quebec[6]	Chrysotile miners & mill workers, with over 5 yrs employment	5,958	1950-1956	9/187 deaths	6+ expected
Pennsylvania[7]	Workers in asbestos products plant, aged 25-64, employed a of 1938-39	1,498	1940-1960	19/188 deaths	5.61 expected
New York & New Jersey[8]	Insulating workers, over 20 yrs since joining union	632	1932-1962	45/255 deaths	6.8 expected
California[10]	Insulating workers mixed, 15 yrs in trade, age 35-64	529	1954-1957 to 1962 av. 7.1 yrs	10/41 deaths	2.8 expected
Dresden[9]	All asbestos trades mixed exposures	2,636	1924-1963	34/190 deaths	11.9 expected
United States[6]	Asbestos textile workers, employed in 1948-51, age 15-64	2,833	1951-1963	24/285 deaths	11.9 expected
United Kingdom[17]	Reported deaths from asbestosis	584	1924-1963	146/584† (25%)	...

* Includes cases reported by Merewether 1949.
† Includes cases reported by Merewether 1949 and 1955.

Within the past five years, there has been increasing evidence of an association between asbestos and diffuse pleural and peritoneal mesotheliomata. Since the first demonstration of a major series of cases, that of Wagner, Sleggs, and Marchand,[16] there have been over 25 reports in the literature. In many of them there have been suggested associations with asbestos, either by occupational history or demonstration of asbestos bodies in lung tissue sections. Inasmuch as mesotheliomata were regarded as excessively rare tumors until recently, the occurrence of even two or three in a series of asbestos workers becomes statistically significant. Separating fact and fancy here becomes a bit more difficult. First, how accurate are our diagnoses of diffuse mesotheliomata? There is no question but that rigid criteria to exclude primary tumors elsewhere are necessary inasmuch as the histologic characteristics of undifferentiated spreading tumors of the pleura and peritoneum are not always distinctive. Close scrutiny of most series of diagnosed cases results in the exclusion of many of them. Nevertheless, where rigid criteria have been applied, as in the cases studied by Hourihane,[20] in 1964, in London Hospital, the association with asbestos has been strengthened, not weakened. A second question regarding these tumors is whether or not their incidence in the population as a whole is increasing. The impression one gets is that they are. Third, if there is an association, is it more evident with one form of asbestos than another? Here, there is no question but that crocidolite, and especially Cape blue crocidolite, is the type where evidence is strongest. In this country, reported series have involved mixed exposures, usually including amosite. Claims have been made that because amosite was not used in the United States to any extent before 1935 and since mesotheliomas characteristically have very long latent periods from first exposure to diagnosis, we are either seeing responses to chrysotile or are seeing only the first cases in an epidemic of effects from amosite. That anthophyllite is not a problem in this respect is strongly suggested by a recent report by Raunio[3] in which the very high prevalences of pleural calcification in anthophyllite mining areas of Finland was not associated with any increased frequency of cancer of the lung or of

malignant pleural or peritoneal tumors. We are left with a strong body of evidence that individuals with light, moderate, and heavy exposures to crocidolite, amosite, or mixtures of amosite and chrysotile show tumors of a type that is much less common in the general population.

Studies in animals in 1962 by Wagner[20] in Wales and in 1965 by Smith[21] and others in New Jersey have led to production of mesothelial tumors in hamsters in which various types of asbestos were introduced intropleurally. The difficulty in preventing cross contamination in laboratories has complicated some of these animal studies, so that differences in response to different types of asbestos and co-factors remain unclear. Nevertheless, the carcinogenic potential of asbestos minerals appears to have been confirmed, although the role of co-factors has not been settled.

Ferruginous Bodies

The final question is, is it true that from a fourth to a half of the general population harbors asbestos fibers in its lungs? The basis for such a statement is the fact that a number of studies of routine consecutive autopsy specimens in recent years have demonstrated "asbestos bodies," or as Gough[22] would prefer to call them, "mineral-fibre-bodies," or Gross, "ferruginous bodies." As far back as 1928, Stewart[24] had shown that smears of the cut surface of the fresh lung at autopsy would reveal large numbers of asbestos bodies in the asbestotic. If anyone tried this in a supposedly unexposed population before studies done in South Africa in the 1960's, it was not published. In 1963, Thomson et al[25] showed 26.4% in 500 consecutive autopsies in Cape Town and later, in 1966,[26] 27.2% in 400 autopsies in Miami. These and other studies are summarized in Table 2. The facts that have emerged from these are as follows:

1. Morphologically and in staining properties, these meet all criteria for asbestos bodies.

2. In no studies, so far published, have there been supporting data to indicate whether all, most, or some of the bodies were asbestos.

3. In most series, about 80% to 90% of the positive reports were on the basis of relatively few asbestos bodies per case.

4. In those with many bodies, there were often but not invariably occupational or residential clues as to a source of asbestos and they were most commonly in males.

5. In none of the series, all small, where correlations were attempted with diagnosis was there any apparent association with malignancies or other specific causes of death.

6. There is ample evidence in the literature that so-called asbestos bodies can be found in workers with exposures other than asbestos, eg, graphite workers, soft coal miners, diatomaceous earth workers, etc.

7. Ferruginous bodies can be produced experimentally in guinea pigs with other dusts. Presumably, they represent a nonspecific response to any relatively insoluble fibrous material in the lungs.

Speaking of this, Cooke[21] said, ". . . there is no reason why any fine spicule of mineral should not have colloidal matter deposited around it and become moulded into a curious body. But as no other mineral dust is fibrous, this occurrence must be so rare as to be negligible from a diagnostic point of view."

The question now, is Cooke's statement of 40 years ago true today? Or, are we adding fibers of many kinds to our environment, giving a confusing picture of our human samplers?

If, as is probable, many of the bodies that are being seen are really asbestos minerals, there is need to determine their significance. It is unwarranted to draw the conclusion from what we now know that they necessarily presage an epidemic of neoplasms. It is

Table 2.—*Ferruginous Bodies in Human Lungs in Autopsy Series**

Place	Year	Percent Positive
Cape Town[25]	1963	26.4
Miami[26]	1965	27.2
Pittsburgh[27]	1965	41
Johannesburg[28]	1965	39.2
Finland[29]	1966	57.6
Montreal[30]	1966	48
San Francisco†	1966	42

* Based on lung smears except series from Finland where thick sections were examined.

† W. C. Cooper and I. R. Tabershaw, unpublished data, 1966.

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quite warranted to speculate on the matter, however. It is important not only to identify positively these ferruginous bodies but also to quantitate them in populations with known exposures to various fibrous minerals. If those with small or moderate numbers of asbestos bodies show no detectable increased risk of disease, then one could feel more secure about those in whom the bodies are most in number.

It is also important to learn the sources of these fibrous minerals that are found in lungs. The view that asbestos minerals are indestructible is not quite true; this is a relative term only, and we cannot assume that the millions of tons used annually actually enter our physical environment forever. Many are bound indefinitely into products that do not release them; those that are released are subject to the effects of heat, acids, and other insults to minerals. Nevertheless, it is important to get a better idea of our asbestos balance, so to speak.

Summary

In summary, with the increasing use and indispensability of asbestos minerals, there has come realization of some very real hazards that must be better defined and controlled. Present standards for dust control do not appear adequate to prevent asbestosis over a working lifetime, and many segments of industry aggressively attacking the problem have already been using more rigorous standards. The associations between asbestos minerals and malignancies of the lungs, pleura, and peritoneum have become increasingly convincing. Current efforts must be directed toward better definition of the importance of type, size of fiber and the importance of co-factors. It has been suggested that asbestos fibers act as carriers of carcinogenic metals or other substances to vulnerable sites. The demonstration of ferruginous bodies in from one-quarter to one-half of the lungs examined in consecutive autopsies in a number of cities around the world points to an urgent need for the positive identification of the mineral fibers that are responsible. If they prove to be asbestos, we do not know what point on the dosage response curve is represented and consequently are not now in a position to estimate their significance in terms of human health.

Conclusions

I have one final set of conclusions. I think you will notice that I have not conformed to the usual pattern of talks with titles such as the one I was assigned, lining up on the one hand a list of the things we really know and then decrying unwarranted and mischievous extrapolations. Actually, that could have been done here, as I do think misleading statements have been made with respect to asbestos. The more fundamental problem is one that is a major affliction of occupational health. We often try to emphasize that part of the challenge of the field is that it is at the forefront of medicine and technology, where environmental hazards can first be detected, in view of the relative levels of exposure and the opportunities to study populations at risk. But when relationships are actually suspected and then gradually established, very real and practical consequences become apparent. So the speculations, call them fancies if you will, that are part of the normal process of developing new knowledge in other fields become menacing and are misused and misunderstood. Polarization of views and severance of communication is the rule, not the exception. I think that this has been the case history of much occupational disease research in the United States, and I cannot say I know how it could be eliminated. I do believe, however, that scientific progress requires both fact and fancy.

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PUBLIC MEDICAL PROBLEMS

We must stop this fragmentation in medicine as well as the fragmentation of our services to the large indigent segment of our population. Our four public assistance medical programs should be one. And here I include prenatal, delivery and postpartum care, general hospital care for medical and surgical problems and the Head Start Project and school health program. Only in this way can we insure continuing supervision and control of disease.

Consider the present situation in one of our larger cities. Families must visit, often simultaneously and also in another location, the health department for prenatal care, the county hospital for general medical services, the Head Start Project for their younger children, the school health program for those that are in school, the crippled children program if they have a crippled child, and the speech defect program for speech therapy. A typical pregnant indigent woman with four or five children must spend so much time and money for transportation to and from these nine separate so-called free services that it is no wonder she has no time or money to keep her family well, let alone happy.—Reis, R.A., MD: "Progress, Problems and Priorities," read before the AMA National Conference on Infant Mortality in San Francisco (Aug 12, 13) 1966.