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American Academy of Occupational Medicine

Asbestos as a Hazard to Health

Fact and Speculation

W. Clark Cooper, MD, Berkeley, Calif

A RESURGENCE of interest in the as-
bestos minerals as environmental hazards to
health has arisen because of increasing
world production of asbestos, successive in-
dications of associations between asbestos
and pulmonary fibrosis and various malign-
ancies, and, more recently, the demonstra-
tion of asbestos or asbestoid bodies in pre-
sumably nonexposed populations.

The major asbestos minerals of commerce
are chrysotile, crocidolite, amosite, and an-
thophyllite, 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 pro-
duction, practically all imported from Cana-
da 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 as-

bestosis. 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 hazard-
ous? The evidence is against this. Chrys-
otile 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, Rau-
nio² 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 com-
munes 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 hy-
giene. 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 Hy-
gienists (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, Wells³ 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 al⁴ 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 8, 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 counts⁵ 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. Balzer 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,⁶ 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 analysis⁷ of causes of death in certified cases of asbestos 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 Truan¹¹ appeared to have no excess risk. It is of interest that Jacob and Anspach¹⁵ 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-related malignancies

Place	Reported deaths
United Kingdom ⁷	Cases of asbestos-related malignancies
United Kingdom ⁸	Asbestos textile workers, 20 years
United Kingdom ⁹	Reported deaths
Quebec ¹¹	Chrysotile miners with over 5 years exposure
Pennsylvania ¹²	Workers in asbestos plant, aged 1938-39
New York & New Jersey ¹³	Insulating workers since joining
California ¹⁴	Insulating workers in trade, age
Dresden ¹⁵	All asbestos-related exposures
United States ¹⁶	Asbestos textile workers in 1948-51
United Kingdom ¹⁷	Reported deaths

* Includes cases reported by Merewether.

† Includes cases reported by Mitchell.

Within the past five years increasing evidence of an association between asbestos and diffuse mesothelioma. Since the demonstration of a major series by Wagner, Sleggs, and Macleod¹⁸ there have been over 25 reports. In many of them there have been associations with asbestos, occupational history or demonstration of asbestos bodies in lung tissue sections. Mesothelioma were regarded as rare tumors until recently, of even two or three in a series of workers becomes statistically significant. Separating fact and fancy is a bit more difficult. First, however, our diagnoses of diffuse mesothelioma. There is no question but that to exclude primary tumors necessary inasmuch as the histologic characteristics of undifferentiated mesothelioma of the pleura and peritoneum are always distinctive. Close series of diagnosed cases resulting in a high percentage of rigid criteria have been applied to cases studied by Hourihan¹⁹

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Table 1.—Epidemiologic Studies of Asbestos Exposures and Lung Cancer

Place	Population Studied	No.	Years Followed	No. With Lung Cancer	Comparison Groups
United Kingdom ⁷	Reported deaths from asbestos	235	1924-1947	31/235 (13.1%)	Silicotics (1.32%)
United Kingdom ⁸	Cases of asbestosis in 1247 autopsies with pneumoconiosis	121	...	17/121 (14.1%)	Silicotics (6.9%)
United Kingdom ⁹	Asbestos textile workers, dusty areas, 20 yrs or more exposure	113	1922-1953	11/39 deaths	0.8 expected
United Kingdom ¹⁰	Reported deaths from asbestosis	365	1924-1955	65/365 (17.8%)	...
Quebec ¹¹	Chrysotile miners & mill workers, with over 5 yrs employment	5,958	1950-1955	9/187 deaths	6+ expected
Pennsylvania ¹²	Workers in asbestos products plant, aged 25-64, employed as of 1938-39	1,495	1940-1960	19/186 deaths	5.61 expected
New York & New Jersey ¹³	Insulating workers, over 20 yrs since joining union	632	1932-1962	45/255 deaths	6.6 expected
California ¹⁴	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 ¹⁵	All asbestos trades mixed exposures	2,636	1924-1963	34/150 deaths	11.9 expected
United States ¹⁶	Asbestos textile workers, employed in 1948-51, age 15-64	2,833	1951-1963	24/286 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,¹⁸ 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 mesothelioma 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 Raunio² 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 Wagner²⁰ in Wales and in 1965 by Smith²¹ and others in New Jersey have led to production of mesothelial tumors in hamsters in which various types of asbestos were introduced intrapleurally. 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 Gough²² would prefer to call them, "mineral-fibre-bodies," or Gross, "ferruginous bodies." As far back as 1928, Stewart²⁴ 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 al²⁵ 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

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.

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, Cooke³¹ 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

quite warranted to speculate however. It is important positively these ferruginous bodies to quantitate them in known exposures to various asbestos bodies show increased risk of disease, more secure about those are scant in number.

It is also important to these fibrous minerals in lungs. The view that an indestructible is not a relative term only, and that the millions of tons that enter our physiology ever. Many are bound in products that do not release released are subject to acids, and other insults. Nevertheless, it is important of our asbestos balance.

Summary

In summary, with the indispensability of asbestos has come realization of standards that must be better controlled. Present standards do not appear adequate over a working life of industry aggregates problem have already orous standards. The asbestos minerals and lungs, pleura, and peritoneum increasingly convincing must be directed toward the importance of type importance of co-factors suggested that asbestos fit carcinogenic metals or vulnerable sites. The ferruginous bodies in from half of the lungs examined autopsies in a number of world points to an urgent identification of them are responsible. If they we do not know what response curve is required

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

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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 miner-
als. If those with small or moderate numbers
of asbestos bodies show no detectable in-
creased risk of disease, then one could feel
more secure about those in whom the bodies
are scant 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 ac-
tually enter our physical environment for-
ever. Many are bound indefinitely into prod-
ucts that do not release them; those that are
released are subject to the effects of heat,
acids, and other insults to minerals. Nev-
ertheless, 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 haz-
ards that must be better defined and con-
trolled. Present standards for dust control
do not appear adequate to prevent asbesto-
sis over a working lifetime, and many seg-
ments of industry aggressively attacking the
problem have already been using more rig-
orous 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 sug-
gested that asbestos fibers act as carriers of
carcinogenic metals or other substances to
vulnerable sites. The demonstration of fer-
ruginous 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 posi-
tive 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 conse-

quently 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 ex-
posure and the opportunities to study popu-
lations at risk. But when relationships are
actually suspected and then gradually estab-
lished, 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 knowl-
edge in other fields become menacing and
are misused and misunderstood. Polariza-
tion of views and severance of communica-
tion 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.

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FOR GOOD reason, dence, it is generally accepted that cigarette smoking is a manner with a risk of lung cancer. Allied to this is the relationship of the disease. How air pollution, or any other factor associated with urban living, cigarette smoking: Is the of lung cancer explained separate effects, or is it

To begin with, it can be argued that the pollution hypothesis is true that carcinogens in the air of cities, that lung cancer is greater in urban than in some extent, greater in small cities. But the relationship has been modest, with exception, and the characteristic measure of exposure to air pollution, area of residence, or rather of which may also be a measure of other things.

Granted, a similar argument against the cigarette habit, smokers may differ from

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