SUMMARY AND EVALUATION

Subject: General discussion of asbestos air pollution in cities, with literature references and original findings in New York City. Of interest and importance.

Evaluation: Although the author had prepared and distributed a formal paper at the medical meeting, he did not actually read it. Instead he spoke extemporaneously from notes and slides, adding material which was not included in the formal version. The formal version has been evaluated. This is a transcript of the taped verbal presentation.

Both the formal and verbal versions consist mostly of material which has been previously published or presented. On page 7, the author mentions data "presented for the first time." The only new data may be the estimated figure "about 95 percent" of persons in New York City having uncoated fibers in their lungs. At previous meetings he had not given an estimated figure.

Principal departures from the formal text occur on pages 10-12 of the transcript wherein the author comments on various slides. These slides have also been shown at previous meetings. They include such subjects as open spraying of asbestos on girders, spray missing a girder, spray leaking out from enclosed areas, asbestos residue on passersby, traces of fibers in workmen's homes, etc. One set of slides showed two youngsters making a contour map with "excellent material -- Johns-Manville asbestos cement."

Author: Selikoff, I. J. (Mount Sinai School of Medicine, New York City).

Unpublished: Presented at the American Medical Association Air Pollution Conference, New Orleans, La., October 5, 1970. (Transcript of verbal presentation.)

Title: "Asbestos Air Pollution in Urban Areas."

Summary: As given above.

MARGINALIA REDACTED

SUMMARY AND EVALUATION

Subject: Discussion of the evidence indicating the presence of low level asbestos contamination of urban air. Of interest and importance.

Evaluation: While much of the material presented here may have been new to the audience, the paper must be considered primarily a review of material previously published or presented. One specific nugget of new information is included. Pages 7 & 8 give the first written information on the results of air sampling in New York City, reporting actual counts of chrysotile fibrils made at various points in the city. "While amounts ranging from approximately 10 to 100 x 10^{-9} grams per cubic meter of sampled air may appear to be exceedingly small quantities of asbestos, it is well to recall that chrysotile asbestos easily fragments into ultimate fibrils 300 to 400 angstroms in diameter and often 2000 angstroms or smaller in length. Thus, 10^{-9} grams of chrysotile asbestos could represent a million fibrils" (An angstrom = one ten thousandth of micron or one hundred millionth of a centimeter.) Tables IV-VI in the appendix, give readings from New York City and other locations.

On page 5 the author speculates on the significance of minute "fibrils" as distinct from "fibers" of asbestos, but presents no conclusions in terms of human health. On page 8 he points out that "it is hardly proper to speak of 'asbestos air pollution' in general terms. There are different sets of circumstances in which such pollution can occur, varying in intensity, intimacy and duration of exposure."

The overall effect of the paper, if followed closely, is that of an extremely complex and uncertain picture. Since the data are not organized with either summary or conclusions, the final sentence of the paper may sum up the intentions of the author: "Our experiences suggest that much education on appropriate methods for containing asbestos in commercial use will be necessary; here, the asbestos industry has both important responsibility and opportunity."

Authors: Selikoff, I. J., Nicholson, W. J. and Langer, A. M.

Unpublished: Presented at the American Medical Association Air Pollution Conference, New Orleans, La., October 5, 1970.

Title: "Asbestos Air Pollution in Urban Areas."

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Transcript

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Transcript of talk by Irving J. Selikoff, M.D., to the American Medical Association Air Pollution Medical Research Conference, October 5, 1970

Asbestos air pollution has long been known in urban areas. Until recently, however, this pollution was considered to be within the factory walls. Such containment was a rather comforting thought because the disease hazards within these walls of occupational pollution was considerable.

In 1924, Cooke described lung scarring following the inhalation of asbestos fibers, asbestosis. In 1935, Kenneth Lynch of South Carolina described the association of lung cancer and asbestosis in workmen so exposed. And during the 1950's, a previously very rare tumor, pleural mesothelioma began to be reported in random instances among asbestos workers. To make this current, this background of concern with asbestos air pollution under industrial circumstances, it might be useful to look at one set of data. May I have the first slide, please.

The asbestos insulation workmen in New York City began to be observed in 1943. And through September 15, of 1970, that is two weeks ago, three week ago, among the 632 men who were members of this union, on January 1, 1943, there now have been 406 deaths. As you can see, 78 of these 406 deaths have been due to lung cancer, 29 have been due to mesothelioma. To give you some idea as to the significance of this I might add that in the general population, as best as we can make out, there is generally one out of 10,000 deaths due to this cause. And among this group of workmen we've had 29 out of 406 deaths and 33 deaths of asbestosis.

At the present time, approximately one in four or one in five deaths is due to lung cancer, one in 10 is due to mesothelioma, one in 10 is due to gastrointestinal cancer and one in ten is due to asbestosis.

For this particular group of occupationally exposed men, we have really a disaster.

With such disease potential as a background, then, we can appreciate the dismay which greeted data which suggested that perhaps the hazards were not limited to the factory environment, that containment is imperfect, and that the problems of asbestos disease were being disseminated from the occupational area into the general environment.

Three general observations were responsible for this. The first was an observation concerning pleural calcification. It had long been known that this type of calcification of the pleura, which you see in this X-ray, occurred frequently among asbestos workers. For example, in the thousand, a little over a thousand, insulation workers examined, 150 cases of pleural calcification were found. Almost none in less than 20 years from onset of exposure, but commonly once this 20 year point had passed.

Knowing this, I think, you can appreciate the dismay that greeted Kiviluoto's report in 1960. He is a radiologist in Finland who did a tuberculosis survey in eastern Karelia there, and found 500 cases of this weird pleural calcification. None of these were in asbestos workers, however. He was doing a tuberculosis survey in this country. These were farmers and farmers wives and very perceptively he noted that this county had an asbestos mine and mill in it. In the next county without such a mine, he found none of these pleural calcifications.

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The next observation concerned mesothelioma. This is a characteristic pleural mesothelioma. As you can see it envelopes the lung from apex to base. When it hits the abdominal cavity it again has the propensity of going along the entire mesothelium lining the peritoneal cavity. This is at laparotomy you can see the tumor diffusely spread. Knowing this, the second observation, that of Wagner who at that time was in South Africa, evoked equal concern. We're very sensitive to this tumor at Mt. Sinai because the basic modern paper on the subject was that of Klemperer and Rabin in 1931 describing two cases of diffuse malignant pleural mesothelioma and we've been looking for it ever since. Dr. Otani, our surgical pathologist, was particularly interested, and from 1930 to 1960 we saw three cases.

This was all the more reason, then for looking at Wagner's data. He reported 47 cases of this otherwise very rare tumor in South Africa, all in one part of that country, the northwest portion of the Cape Province, a part of the country which happened to have a good many asbestos mines.

A pathologist turned epidemiologist, and with a good detective's nose, he interviewed the surviving relatives. And in 45 of the 47 cases was able to unearth previous asbestos contact, usually 25 or 30 or 40 years before. This contact was not occupational in most instances. It simply consisted of living in towns in which there had been an asbestos mill, or in one instance riding to town in a donkey cart with bags of asbestos alongside, etc.

In the Pneumoconiosis Research Unit of South Africa in Johannesburg in a five-year period there hadn't been a single case of mesothelioma in 10,000 silicosis autopsies.

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This raised a very serious question as to whether this tumor recognized to be highly associated with occupational asbestos exposure might also be associated with non-occupational exposure as in a family setting or by virtue of neighborhood exposure.

Molly Newhouse, whose name has already been mentioned today, an epidemiologist at the London School of Hygiene, looked into this question. She examined all 76 instances of mesothelioma in the pathology files of the London Hospital. One dated back to 1910, most were in 1960 to 1965, and she too visited the surviving relatives or in some cases the men who still survived.

As we expected, she found that 31 of the 76 cases had worked with asbestos. Well, this comes as no surprise any longer. But of the 45 who did not work with asbestos, 9 simply lived in a household with an asbestos worker, and 11 simply lived within a half mile of one of the asbestos plants in Barking in London.

Similar studies have been done in this country. Jan Lieben, when he was with the Pennsylvania State Department of Health looked at the 42 cases in their files and again, as Newhouse had found, demonstrated that 20 had industrial asbestos exposure. But again, of the 22 who did not, 8 lived with a half mile of an asbestos plant, and 3 had had family contact in the household of asbestos workers.

The next general observation depended upon the observation of asbestos bodies. All of us are familiar with these quite striking particles. Of considerable interest is that when you squeeze the surface of the lung that has been cut, as if you are making a malaria smear, squeeze out a drop of tissue fluid, you sometimes can see these asbestos bodies on the slide.

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John Thompson, who is professor of pathology at the University of Capetown, did this in 500 consecutive cases. And he found that the characteristic asbestos bodies present in very large numbers in the lungs of asbestos workers were easily to be found -- after all, he only looked at a drop of tissue fluid -- in the lungs of ordinary citizens of Capetown.

These findings have been confirmed many times over throughout the world.

He also made a prediction that mesothelioma or other neoplasms associated with asbestos exposure would become as common as cigaret lung cancer is at the present time by virtue of the inhalation in urban areas of fibers of asbestos. It should be noted right off that this prediction is a strong extrapolation and hardly supported at this time by evidence.

First of all, we know very little about the asbestos content of lungs, especially the comparable content of the asbestos in the lungs of an asbestos worker compared with those of let us say Molly Newhouse's neighborhood residents or family contacts or people in the general population who have asbestos bodies in their lungs.

In industrial circumstances it takes very little asbestos incidentally to be associated with extensive scarring or asbestosis. There have been a few reports, they vary from 0.6 percent of dried lung weight to .001 percent of dried lung weight. But we have no knowledge of lung content in other circumstances.

Secondly, he misled us, or rather we allowed ourselves to be misled, in that he pointed to asbestos bodies when he really meant to say the asbestos content of lungs.

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And everybody has been looking at asbestos bodies. This is not very healthy because it is very difficult to analyze the core of what looks like an asbestos body. That central core that you see here with the iron-rich gel-like material around it is very difficult to analyze. All you can say is that there is an inorganic fiber there. To analyze techniques such as electron defraction, electron microprobe analysis, etc.

And to do this you have to first micro-manipulate, generally under 100 times magnification, this particular asbestos body onto a suitable substrate. That means to analyze this core fiber, you have to use a light microscope. And anything you cannot see with a light microscope you cannot analyze.

Unfortunately, 95 percent of all of the asbestos used in this country is chrysotile, a variety that is unstable chemically in the lung -- magnesium is readily leached out -- and so the electron microprobe, microchemical analysis, does not give unchange chrysotile. And secondly, in a water solution like the lung tissue fluid it rapidly breaks down into its component fibrils.

That is not a fiber that you see, or rather it is a fiber but not a simple fiber. It's actually composed of probably dozens of fibrils, each of the unit diameter of around 350 angstrom units. Therefore, our trying to discover what the core of asbestos bodies really is kept us for several years from a much more direct approach to the question, namely, if we want to know if there is asbestos in the lungs of people living in the big cities, we ought to look for asbestos, and not particularly asbestos bodies.

In recent months, this has been done, both in London and in New York. And the answers are now in. Chrysotile is regularly found in measurable amounts in the lungs of residents of these two cities.

We in New York did it by taking a 25 micron by 1 cm. square section from each of seven sites in the lung , made a stack of them -- a Dagwood sandwich sort of thing -- ashed it in an electronically activated oxygen asher -- and then looked for the particles that could be seen under high magnification.

And this is what we have found. This data is now presented for the first time.

We took lungs of 3,000 consecutive autopsies in New York City, at Mt. Sinai, at Bronx VA Hospital, and at the City Hospital in Elmhurst.

Examining only 175 microns of tissue, we found asbestos bodies to be present in 50 percent. Obviously, it was more common in males than females, and with advancing age it also became more common.

But when we looked for uncoated fibers, we also found them. Indeed, thick fibers, those more than one micron in diameter, were seen in almost everybody. About 95 percent of the people had these. Thin fibers, that is between the limit of resolution of the optical microscope, around half micron and one micron, we still found very commonly these were much more likely to be Chrysotile asbestos. And these are the _____(?).

As you can see, where there were no asbestos bodies, there tended to be no thin inorganic fibers. On the other hand, where there were asbestos bodies, with many there tended to be thinner inorganic fibers.

In general, we can summarize that slide by saying that over 1,000 people had thin inorganic fibers consistent with chrysotile present in their lungs -- 1,000 out of 3,000. But that still doesn't prove that these thin fibers were really asbestos -- chrysotile asbestos. To do this we have to go to a level far beyond the optical microscope, namely electron microscopy.

Here we take advantage of the fact that Chrysotile asbestos has a unique morphology seen by electron microscopy. Here are some of the electron micrographs. The top shows the characteristic electron dense (form) with the characteristic capillary in the center. When this is kept under the electron beam for any length of time, the central core does become somewhat changed and the outer electron dense shell becomes somewhat amorphous.

When it is beaten in the industrial process this occurs more readily.

These are electron-micrographs of lungs of 28 consecutive people dying at Mt. Sinai and in 28 out of 28, the characteristic Chrysotile fibers -- fibrils, these are not fibers, these are the individual unit fibrils -- were found.

In these 28 cases, in 4 the results could have been due to background contamination. In 24 they were far beyond this. Incidentally, if you are going to do any studies in this, in all laboratory work involving the presence or absence of chrysotile fibers, please run controls. Because in this room, as in every laboratory, even perhaps under the clint (?) hood, there is some contamination and you may get false positives.

We predict in fact now that what has been found in New York and what has been found incidentally by Pooley and his colleagues in London will be the case in other urban areas as well.

There is no question now then that asbestos is commonly found in the lungs of city dwellers. Where does it come from? The simple answer, obviously, is that it comes from asbestos air pollution. The answer is really not that simple. Because there is no such thing as asbestos air pollution. There are many different kinds of asbestos air pollution.

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For example, there is the air pollution in the workplace -- occupational -- and there is also indirect occupational exposure which may be very important. For example, there are only about 50,000 asbestos insulation workers in the construction industry but they work in the midst of some 4,000,000 construction workmen. These are the Typhoid Marys of the construction industry.

And if we were to find asbestos in the lungs of an electrician, a steamfitter, etc., we might call this not the result of general asbestos air pollution but that of an indirect occupational exposure.

Similarly, family households, the men come home with the stuff on their shoes and clothes and so forth.

In the emanations from the factories, mines and mills in the neighborhood, in other words, neighborhood exposure.

And, of course, the general community which derives its insults from these various sources.

But also I would suggest probably some from natural sources. Serpentine outcropping gradually weather and I think a very small -- this has not been measured but I would anticipate that when measurements are made -- a very small but definite amount of asbestos may come from natural weathering of serpentine rocks.

Also the weathering of asbestos-containing materials such as shingles, board, etc., might add a small amount to the general community air pollution in addition to these particular sources.

Each of these has its own disease potential and the interweaving threads really should be unraveled before we say very much. It may be useful to scan some examples of these different sources.

For example, this is an asbestos worker. Our people are around so he's on his good behavior and he's wearing his mask, otherwise he's not.

He's mixing asbestos cement. But the two fellows in the back are steamfitters. And the asbestos fibers in that cement just don't know how to obey jurisdictional craft lines. Everybody else does in the construction industry, but not this.

Here, for example, when they get into their clothes changing shack the men not only change clothes but they also distribute to each other the contaminants each brings.

And when you go to a construction worker's household, as we did with this high volume sampler, the air contains these fibers -- the large ones are fibrous glass -- and the settled dust incidentally contains very much more. So this would be an instance, then, of family contact.

The general community does get some contribution perhaps from the use of products, for example, brake linings. The exact amount that is contributed frm this source is not known. Probably much of the brake does not end up as asbestos air pollution, being burnt under the high temperatures of stopping a car. We don't know exactly how much does. This is another problem that has to be solved.

Here's another family type. This is in California. These children have made this beautiful contour map. And what did they make it with. Excellent material, Johns-Manville asbestos cement.

And we had them actually make one for us and here is how they did it.

But I would suggest that the most important area to be looked at is really the construction industry. Because this is where the asbestos is used. As you can see from the bottom of that slide, the construction industry in 1965 used three-quarters of all the asbestos used in the United States. Therefore, this is where the money is. And a close look

For example, here is one particularly bad contribution. This is the spraying of steel girders in the fireproofing of buildings. This is on the corner of 55th Street and Third Avenue in New York, if any of you happen to be going down that way.

This is a passerby. As he passes by, what he had on his jacket. Many of you know Duncan Halladay who used his face as a sampler. And this is what Duncan had.

This is 54th Street. Take a good look at that. That's now going to be distributed through New York City.

This is the roof a quarter of a mile away of the High School of Art and Sciences and our future artists and scientists are breathing this.

The actual spraying goes like this. This is on the corner of Wall and Water Streets. And this has nothing to do with the current situation in the market. And this is when he hits the beam.

But this is when he misses the beam. And that is being distributed through that particular part of town.

This is on the Avenue of the Americas. This is the enclosure, so called, that is being used to contain these materials. About two minutes after this they started the spraying.

And this is what happened when they started. That's now distributed over Sixth Avenue.

I thought it appropriate to bring this slide because this is an AMA meeting. This you will recognize as Chicago and the next photograph is the building next to the Marina Towers. I think somebody tells me it's about five blocks from the AMA headquarters.

This is the spraying going on there. Not even any enclosure much less the flimsy ones we had until recently in New York. This is near the

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If you want to see the results of this type of spraying all you have to do is walk past any construction site. You've all seen this but nobody's paid any real attention to it. It's unbelievable.

And two other important sources, again from the construction industry. This is one, this is how the debris -- of course the whole problem of solid waste disposal is very much with us -- we've all walked past this.

And here, this is our Madison Avenue and 99th Street. We're tearing down buildings at Mt. Sinai to build some good laboratories for environmental research. (Laughter).

Well, to the present there has been almost no information on the existence or the magnitude of the pollution of urban air with asbestos. In part, this is because of the same technical factors that I mentioned before with regard to the lung content, especially the breakdown of chrysotile after it's been in air any length of time, to its constituent fibrils.

For example, this is the rock from which it comes. This is serpentine and those are the individual fibers. Each one of those fibers is composed of individual fibrils and even now you will recognize again the ultimate fibrils. When proper sampling is done, and we're at the beginning of this now, in the last several months this question has been looked at, and utilizing our technical improvements we do have now some initial results which I will present here for the first time.

These are air samples, these are clays that go with it, and we have yet to get an air sample taken with enough air that does not contain in urban areas chrysotile fibers.

Now the levels. We found that there is more in Manhattan than in the outlying boroughs of New York. To give you some idea, these are expressed in ten to the minus ninth grams per cubic meter.

Ten to the minus nine grams is somewhere around a million fibrils, if you can translate the mass to numbers. Much work still has to be done on a technical level with this but as you can see there is a distinct tendency for our more populous boroughs to contain more asbestos in the air.

Also, it makes a big difference whether you are upwind or downwind from specific sources. As you can see here, downwind very much more, up to 180 times ten to the minus ninth grams. So specific sources can be pinpointed. This bodes well for the future of control measures because we will be able to actually pinpoint and define specific sources.

We did some very limited work in outlying areas. Philadelphia had very much the same as New York in the few samples that we've done. Ridgewood, N.J., where I live, fairly suburban, had still asbestos in its samples but less. We've also done some sampling at Port Allegheny, Pa. Dr. Corn has helped us with this. There we did find chrysotile in the amounts that you see but that town also has an amosite factory. Different problem. We also found amosite fibers in the air of this community as well.

I think it might be useful now to summarize.

First of all, occupational asbestos air pollution is obviously associated with a significant health hazard.

Secondly, from such sources and others dissemination of asbestos fibers now occurs into community air. This has been identified and quantitation has been demenstrated to be possible.

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Analysis of lungs of urban dwellers of New York, London, elsewhere, indicates that they commonly contain measurable amounts of asbestos. And it is likely that this is going to be found in other cities as well.

The amounts of asbestos in the lungs of workmen is likely much greater than in the lungs of people environmentally exposed, although this is not yet known.

And extrapolation of the occupational hazard to the general community is unwarranted. Much information is needed to define the dose-disease response relationship.

Since no effective threshold limit has been identified, it would seem prudent to identify sources of asbestos air pollution. All varieties, indirect occupational, family, neighborhood, general community, etc., so that appropriate controls can be developed and applied.

I am confident that this can and will be done and that this will be an area fruitful for cooperation among industry, labor, government and science.

END

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Q. (Do other states or cities have asbestos regulations similar to New York City?)

A. New York City, regarding the spraying of asbestos, requires rather stringent controls, including total enclosure, vacuuming after the use of the material, proper waste disposal, etc. There so far has been less than adequate compliance and if this continues it is my guess, I would hazard a guess, that the spraying of asbestos would be outlawed in New York City completely. Other cities have taken this ordinance -- that would include Philadelphia, San Francisco. I understand Chicago is now looking at this, Dr. Carnow tells me -- and some areas in Canada, etc. How they will enforce this I do not know.

Q. (Has the increase in construction in New York City led to an increase in mesothelioma?)

A. The spraying of asbestos began in New York City in 1959. One New York Plaza was the, I'm sorry, I forget the name of the building. The first building to be sprayed fireproof with asbestos was in 1959. Our experience is ahead of us. Incidentally, this long lapse period necessary between onset of exposure and evidence of clinical disease makes us a little bit uneasy. For example, shipyards have been identified in Great Britain and Holland as a very worrisome workplace. Peter Harries at the Royal Navy Yard in Devon reported in 1968 that in one yard in the previous year he had seen 14 cases of mesothelioma. Not one in an asbestos worker. All in shipfitters, electricians, welders, etc. I saw him two weeks ago. He tells me there are now 35 cases of mesothelioma in this one yard. Translating this to the American experience -- in other words what he is looking at is what has happened as a result of the work in that yard in 1935, '36, '37 because they began their shipbuilding before we did: we began ours in the Second World War

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in 1939 and '40 -- translating this concern, we are looking carefully at the shipyard population -- not today's work force but the work force in 1940, '41, '42, '43 and '44 -- when as many as 1,750,000 people were working in our shipyards in any one month. Incidentally, around 15-20 percent were women during the war. So I would urge all of you, on a clinical level, when there are obscure, diffuse situations in either the chest or abdomen, to carefully take an occupational history -- not the current or the last job of the person -- but what he did or she did during the war.

Q. (Question about other chest diseases.)

A. Oh, yes. I overemphasized mesothelioma because this is a little unusual and striking and exciting. Carcinoma of the lung is much more common among asbestos workers than is mesothelioma. For example, our age, year and sex specific expected rates for carcinoma of the lung among the 632 men we've been looking at in New York is that approximately 12 or 13 should have developed lung cancer. Actually, 78 have. This is much more worrisome for us than mesothelioma but I thought perhaps less interesting and less exciting to you. So far as the men are concerned, lung cancer is much more important than mesothelioma. They're just as dead.

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Distributed Paper

ASBESTOS AIR POLLUTION IN URBAN AREAS

I.J. Selikoff, M.D., W.J. Nicholson, Ph.D. and A.M. Langer, Ph.D.

INTRODUCTION

Early in the 1960s, the problem of asbestos disease was disseminated from the occupational area into the general environment. Three germinal observations were responsible for this.

First, Kiviluoto reported finding 499 cases of parietal pleural calcification among 6,312 residents of a rural county in Finland.[1] This came very much as a surprise, since the type of calcification observed had previously been described primarily in asbestos workers,[2] usually more than 20 or 30 years after starting work;[3] yet the cases were not among asbestos workers, but among farmers and farmers' wives. The asbestos link was, however, there - the county did have an asbestos mine. The natural supposition was that asbestos air pollution from the mine was responsible, a presumption that was strengthened when the specific type of asbestos produced by the mine (anthpohyllite) was found in the air up to 50 Km away,[4] and asbestos bodies were demonstrated in the lungs of cattle grazing in the nearby fields.[1]

The same year (1960) saw a second worrisome communication. Wagner[5] reported 47 cases of pleural mesothelioma in a part of South Africa important for asbestos mining. He unearthed potential asbestos contact for most of the patients two or more decades before, in many instances merely the result of living in the general area or by chance contact in a family setting. While pleural mesothelioma had previously been attributed to asbestos exposure,[6] the strength of this association had not been appreciated. More pertinent, was the demonstration that it could result from other than occupational exposure.

Amplification was soon added. Newhouse[7] studied all mesotheliomas at the London Hospital. She confirmed the close association with asbestos, 31 of the 76 cases having had occupational exposure. She also confirmed the importance of non-occupational contact: of the 45 who had not worked with the material, 9 had lived in the households of asbestos workers and 11 had lived decades before, within one-half mile of an asbestos plant! Lieben and Pistawka reported similar findings in Pennsylvania.[8]

What appeared to be a strong link in the chain of evidence for environmental asbestos disease was the report in 1963 by Thomson[9] that asbestos bodies were commonly to be found in the lungs of people in the general population of Capetown. He expressed a drop of lung tissue fluid onto a slide, as if preparing a smear for malaria examination, examined it by optical microscopy, and in one-quarter of 500 consecutive autopsies, found structures apparently identical with those seen in asbestos workers' lungs. But these were not asbestos workers - they had been ordinary citizens of this large city. Thomson concluded that they had inhaled

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Presented by I.J. Selikoff, M.D., at the AMA's Air Pollution Medical Research Conference on October 6, 1970, in New Orleans. Doctor Selikoff is Professor of Medicine and Professor of Community Medicine, Mount Sinai School of Medicine of the City University of New York and Director of Environmental Sciences Laboratory, Mount Sinai School of Medicine.

asbestos in the course of their urban living, from the many asbestos products about them. With knowledge of what exposure to these fibers could do under industrial circumstances as a background, and with Wagner's observations as an example, he suggested that we were now faced with a "modern urban hazard" and predicted that asbestos-associated neoplasms would rival cigarette-induced lung cancer in the future.

It should be noted right off that Thomson's prediction is an extrapolation. It is a wide step from occupational exposure, with large numbers of asbestos bodies, to community contamination, with as a rule far fewer bodies, particularly with little knowledge of a dose-disease response relationship. Too, the particles described had the appearance of those seen in asbestos workers, but some uncertainty existed that these necessarily had an asbestos core,[10] especially since it had been known for 30 years that such bodies could be found after exposure to other fibers as well.[11]

Despite these caveats, the three reports, taken together, posed a problem that is now very much with us. Occupational asbestos exposure may be associated with serious risk; for example, among asbestos insulation workers in the New York metropolitan area at this time, approximately one in 5 deaths is due to lung cancer, one in ten is due to mesothilioma, one in ten to gastro-intestinal cancer and one in ten to asbestosis and cor pulmonale.[12-14] These men have been exposed to amounts of asbestos surely greater than those in the community generally, and we would not expect their risk to be duplicated in the general population. But is part of their risk disseminated, with the dusts from their work? What sort of dose-response curve are we dealing with? Is there a threshold which once crossed leads to serious neoplasia hazard? An hypothesis could be formulated that such a threshold includes very little asbestos (since small amounts may still reflect billions of fibers and/or fibrils), that asbestos workers pass it early in their careers.

Such an hypothesis, or variations on it, would fit clinical observations made in the last several years. Neoplasms - lung cancer as well as pleural and peritoneal mesothelioma - occur in excess even among asbestos workers with little or no radiological evidence of asbestosis. It is now apparent that exposures insufficient to cause asbestosis may still produce neoplasia. The spread of this separation is not established, but is recognized by recent appreciation that while lower dust levels in industry may prevent much asbestosis, such levels will not necessarily prevent cancer.[15] It is not now known how low a threshold must be to prevent asbestos-associated neoplasms, if such a threshold indeed exists.

ASBESTOS IN LUNGS

It thus became important to know whether asbestos truly was a common contaminant of urban dwellers' lung. The demonstration that "asbestos bodies" were to be regularly found at autopsy in many cities of the world[16] confirmed Thomson's finding, but did not settle the question. There remained the nagging doubt that the cores were necessarily asbestos, an insecurity perpetuated by the technical difficulties involved in analyzing such cores.[17]

In recent months, the impasse has been resolved by avoiding the detour of asbestos bodies, with the direct search for asbestos fibers and fibrils.[18] Investigation of 3,000 consecutive autopsies in New York had shown that asbestos bodies were common; optical microscopic examination of 175u x 1cm^2 of lung tissue

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in each of these cases showed asbestos bodies in 1,449 (48.3%) (Table I). It is likely that, had a greater volume of tissue been submitted for study, asbestos bodies would have been found in all the cases, except perhaps the infants and very young children.

The same examination showed that, in addition to coated particles ("asbestos bodies"), uncoated inorganic fibers were also readily seen. Fibers thicker than 1.0u were almost universally to be found; most of these are as yet still unidentified although some were diatom fragments, glass fibers or phytoliths. We were more concerned, however, with thinner fibers - less than 1.0u in diameter - since these would be more consistent with chrysotile, the asbestos variety making up 95% of the asbestos used in the United States. Such thin fibers were also commonly present, being found in 1,038 of the 3,000 cases, and tending to vary with the number of asbestos bodies (Table 2).

The critical information was obtained by examining, in 28 of the 3,000 cases a very small portion of lung (conservatively estimated at 10^{-6}) by a technique which allowed qualitative analysis,[17] with the appreciation that the unique morphology of chrysotile allows its specific identification by high magnification electron microscopy.

Chrysotile fibers and/or fibrils were found in every specimen. (Figures 1,2). In 4 of the 28, background contamination could conceivably have been responsible for the findings. In 24 of the 28, the number found was greater than background counts could explain (Table 3). The morphological appearance and other characteristics of these fibers and fibrils and recorded elsewhere.[19]

It is evident that chrysotile asbestos is a common contaminant of the lungs of New York City residents at this time. Similar electron microscopic observations have been recorded in London,[20] where not only was chrysotile asbestos found in almost 80 percent of cases, but it was noted to be the most common and most abundant of all fibers detected.

The question inherent in Thomson's observations in 1963 "Is chrysotile asbestos commonly found in the lungs of urban dwellers at this time?" has now been answered, "Yes, unequivocally."

RELATION OF ASBESTOS LUNG BURDEN TO ENVIRONMENTAL ASBESTOS DISEASE

There are few data at this time that would allow judgement of the significance of the asbestos we have found in lung tissue of urban residents. Whether or not the amounts present as the result of other than occupational exposure are associated with frequent risk of disease is not known. In part, this is a reflection of the paucity of data concerning the asbestos content of lungs in general, including those of asbestos workers. Such data as are available suggest that the amount in lungs of the latter is quite small, ranging from 0.6 to 0.001 percent of lung weight.[21,22] It would be expected to be even lower in those not occupationally exposed.

No information is at hand concerning the asbestos content of the lungs among Wagner's cases, or Newhouse's or those of other cases with environmental disease.[23,24] Nor is there quantitative information concerning the asbestos content of the lung in individuals in the general population without asbestos stigmata. This is rather urgently needed, with age, sex, residence and occupational exposure taken into account.

Studies now in progress in our Laboratory and elsewhere[25] indicate that suitable quantitative techniques for estimating asbestos lung content will be feasible and that fairly accurate estimates - approximating an order of magnitude - are to be anticipated in the future.

ASBESTOS AIR POLLUTION

It is probably an entirely justified concept that the asbestos found in urban dwellers' lung is derived from the inhalation of air contaminated with these fibers. Very little is known, however, of the conditions of such contamination and facile assumptions should be avoided at this time.

Previous Epidemiologic Observations

An example of where an "obvious" explanation might also be inaccurate may be found in the assumptions adopted to explain Kiviluoto's observations. It seemed natural to expect that the demonstrable anthophyllite asbestos air pollution from the mine and mill was etiologically related to the equally demonstrable asbestotic pleural calcification in the population living about that point source. It turns out, however, that this may not be the entire explanation, and that intimate contact with local asbestos bearing rocks including those used in building houses, saunas, barns and the like might also play a role.[26]

Indeed, the latter association better explains almost identical epidemiological findings in Bulgaria, where, again, pleural calcification was found in a rural population. In this district, too, an asbestos mine was being worked but it was an underground mine and had opened only in 1943, too recently for its discharges to confidently be expected to have had the effect noted (the lapsed period between initial exposure and evidence of pleural calcification is 20, 30, 40 or more years).[27] As in Finland, the local field rocks have a high asbestos content and are used for various structures by the farming population, which then may have intimate contact with what is shed from them. More important, perhaps, the soils tilled by these farmers can be demonstrated to have asbestos fiber (anthophyllite) content. The importance of this observation was emphasized by the discovery that those farmers working plots without asbestos soil contamination had little pleural calcification, whereas farmers working soil with anthophyllite readily showed the radiological changes.[28] It may be that similar explanations will become available for the finding of endemic pleural calcification in some rural areas of Czechoslovakia.[29,30]

Technical Factors

At first glance, it seems somewhat surprising that so few data are available concerning the asbestos content of ambient air, especially since so much is known regarding the asbestos content of air within the work place. Perhaps the best explanation is that once the factory gates are passed, a whole new set of technical problems is encountered and sampling procedures, analytical approaches and measuring methods useful for industrial controls are no longer applicable.

Fiber Identification

Under industrial circumstances, there is usually no problem in knowing exactly what is being measured, since the materials being used are either well characterized or can be readily analyzed. Thus, whatever fibers are seen can be confidently labelled as "chrysotile," "crocidolite", "amosite", "fibrous glass", etc.

If these same fibers were to be seen in a random sample, especially if they are small, our confidence disappears. All that can be said is that inorganic fibers are present. Even then, if these fibers were found in very large numbers, identification could be readily accomplished by such mass techniques as x-ray diffraction; but when they occur singly or randomly scattered in small numbers, these techniques are no longer applicable and readily available alternate approaches such as polarized light microscopy, hardly have the same definitive assurance. While it is true that analytical attack on single fibers is still possible, these approaches (including electron microprobe analyses, electron diffraction and electron microscopy) are time consuming and often restricted by the size of the particle available for analyses. (See below). Our own experience suggests that reticence is usually warranted when identifying single small fibers in ambient air samples, unless extended techniques are used.

Particles and Fibers

There has long been an anomaly in particle counting for asbestos threshold levels. In the United States, a threshold level was proposed in 1938 for occupational exposure to asbestos, based upon experiences necessarily limited to that point.[31] The recommendation reflected the instrumental restrictions of the times and were based upon counting of "particles" by optical microscopy. It was recognized that such particles could either be fibrous or non-fibrous and that the proportion could vary widely according to the materials used, process studied, etc. Since the presumption is that only the fibers are responsible for the biological effect, such analytical dilution is hardly acceptable at this time and current approaches to industrial threshold limit values are based entirely on fiber count. But even here, the matter is not so easily disposed of, since the admixture of particles and fibers often includes surface interactions among them. Figures 3 and 4 show particles of clay and a diatom collected near a construction site during sampling by our Laboratory. By light microscopy these would be categorized as "particles." Yet by electron microscopy it was found that numerous fibers (fibrils) were attached to the surface of the particles (opposite surface charges!). In such circumstances, any biological effect of the fibers could be incorrectly attributed to the particles. The question of size and magnification are critical.

Fiber-fibril

Each asbestos fiber variety is quite different chemically, physically, structurally and morphologically.[32] Chrysotile seems unique in its tendency to physical instability under a number of circumstances. The chrysotile "fiber" is not a unit whole but is rather composed of a large number of individual fibrils, each from 300Å-400Å (Figure 5). These unit fibrils cannot be seen with the optical microscope. The intact chrysotile fiber can be seen. Under industrial circumstances, this dichotomy is understood. It is recognized that when a population of fibers is counted, "invisible" fibrils are also present,[33] but that the optically visible fibers reflect, in varying proportions under different circumstances, the total chrysotile population, even though there be but one fiber for a very large number of fibrils. Also, with proximity to the industrial source, many fibers are still present, not having been subjected to influences which could result in their separation into fibrils.

In the ambient air, however, very little is known about the proportion of fibers to fibrils. The matter is of some importance, since not only must number be categorized as fiber and fibril, but the surface area and potential biological effect might be quite different with the same total chrysotile mass, with different percentage fibrillation. Once again, we return to the problem of ultramicroscopic

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size, since the proportion of fibers to fibrils cannot be determined without the electron microscope.

Size

It may be seen from the foregoing that a critical factor in studying asbestos air pollution is the utilization of techniques which will measure very small particles. It is unlikely that approaches which do not include the electron microscope will be effective. Indeed, one might add that high magnification electron microscopy will be needed, including magnifications of at least 20,000X (direct) and probably over 30,000X. While optical microscopy may be suitable as a guide for occupational asbestos exposure, it has insurmountable inadequacies in studying asbestos pollution of the ambient air.

In addition to the fine diameter of the fibrils, it has been our experience that many of them are quite short, as well. We have observed chrysotile fibrils with lengths less than 1000Å in many instances; when these are enmeshed in sampling debris, not only is high magnification electron microscopy necessary, but visual scanning may be inadequate, and inferior to photographic recording.[17]

It may be worth noting that such small particles are at present subject to identification by their morphological characteristics only; structural and microchemical analysis by electron microprobe or electron diffraction study has many difficulties. It may be hoped that instrumental advances will remedy this situation (greatly improved microprobe definition can be anticipated, for example). Turkevich's "World of Fine Particles"[34] is surely with us!

Quantitation

Under industrial circumstances, again, there is little difficulty in estimating the quantity of asbestos in a given sample of air. From this, the surface area of the fibers can be estimated or directly measured by nitrogen absorption or other techniques. It is even possible to conceive of gravimetric methods, rather than the more laborious counting of fibers. Quantitation is much more difficult in ambient air samples and, in our experience, only approximations can presently be obtained.

Current Approaches to Asbestos Air Sampling

With the foregoing factors in mind the absence of published information on asbestos levels in urban ambient air may be appreciated. Few air monitoring agencies have had available the technical equipment for the examination of ultramicroscopic asbestos fibrils. Moreover, even those fibers seen by optical microscopy in air samples required elaborate techniques (such as electron diffraction or microprobe analysis) for positive identification.

Present approaches to quantitating asbestos levels in ambient air, using ultramicroscopic techniques, have sought two objectives. First, to obtain a measure of the mass of asbestos per unit air volume at various locations in urban centers and later, for comparison purposes, in more rural areas. This would provide a stable estimate, since fiber size distributions change as sampling is undertaken at different distances from asbestos emission sources. In such circumstances, equal numbers of fibers can represent significantly different amounts,

by weight, of asbestos. Moreover, it is not currently known how strongly biological effect is dependent on fiber size. This being so, a measure by mass or weight may be more conservative for the establishment of air quality criteria and standards, as the biological effect of a sample dominated by large fibers may be overestimated.

Second, fiber size distribution is important. At the moment, to obtain such a distribution at each sampled site is time consuming, and the presence of other material and existing transfer and sampling methods may distort the observed fiber distribution. However, experiences in current studies suggest that these problems can be overcome and that complete fiber size distributions will be obtained at selected sites in the near future.

Sample Preparation

In our studies, air samples were collected on membrane filters having an effective pore size of either 0.8 or 1.2 microns. (Millipore AA or RA filters were used). While this pore size is larger than the largest dimension of some asbestos fibrils, it has been found that the surface charge properties of the filter and the asbestos, as well as the circuitous path through the filter, allow virtually complete collection of all asbestos material.

Both high volume samplers capable of drawing 40 cubic feet per minute through an 8" x 10" filter and small battery operated personnel monitoring samplers with a capactiy of 2 liters per minute through 8 cm^2, were found effective in sample collection. Portions of each collected sample were ashed in an activated oxygen asher which oxidized the membrane filter and all organic or carbonaceous material in the sample.[35] The residue, consisting mostly of fly ash and mineral matter, was dispersed on a microscope slide by grinding in a solution of 1% nitrocellulose in amyl acetate for 2 to 5 minutes. Upon evaporation of the amyl acetate, the dispersal was scanned for uniformity by optical microscopy and a representative area chosen for transfer to an electron microscope grid for scanning.

During this procedure, those chrysotile fiber bundles present are broken into their elementary fibril form. The grid so prepared of the dispersed chrysotile is scanned at 42,000X in an electron microscope. Typically, six grids are prepared of each sample and three 100 by 100 micron squares of each grid scanned. The mass of asbestos is obtained by measuring the volume of asbestos per grid square and multiplying by the appropriate density. Figure 6 shows several isolated fibrils in an ambient air sample as seen by electron microscopy. Usually only single fibrils, if any, are observed in the microscopic field.

However, as one samples near sources of asbestos, more large fiber bundles are present in the initial sample. As these are dispersed during sample preparation, occasionally some groups of fibrils remain intact. Such a group is shown in Figure 7. The presence of these bundles, or even localized fibril clumps, gives rise to a variation between samples greater than one would expect from statistical considerations alone. However, the scanning of many grid squares serves to average their effect.

Results of Initial Investigations

Table 4 gives the ranges of values for ambient air levels measured at various sites in New York City. These samples were taken at selected locations of the sampling network of New York City's Department of Air Resources. The sites were all located on public buildings distant from any known significant source of

asbestos. While preliminary in nature, the samples from Manhattan tended to be higher than those from other boroughs. Lowest values were usually from sites most distant from densely populated business areas (Staten Island).

While amounts ranging from approximately 10 to 100 x 10^{-9} grams per cubic meter of sampled air may appear to be exceedingly small quantities of asbestos, it is well to recall that chrysotile asbestos easily fragments into ultimate fibrils 300Å to 400Å in diameter and often 2000Å or smaller in length. Thus, 10^{-9} grams of chrysotile asbestos could represent a million fibrils.

That Manhattan has higher levels of asbestos that other boroughs is to be expected, as greater use of asbestos in building construction takes place in that borough. During the past 10 years it has been common practice to spray fireproofing material containing from 10 to 30% asbestos, onto girders, spandrels and decking of high-rise office buildings. Often inadequate precautions were taken by contractors to contain the spray material and extensive "snowfalls" of asbestos containing material took place over wide spread areas of Manhattan. This practice was of such obvious concern that the New York City Department of Air Resources instituted stringent regulations controlling the procedure in May, 1970.[36]

Table 5 records findings on several days at various sites in lower Manhattan near buildings under construction. Data were obtained prior to the implementation of New York City's regulations. During the two days on which data were obtained sites 1 and 4 were downwind from a spray source, 3 and 5 were upwind from any source, and 2 was 45° from one source and upwind from others.

The data in New York are being supplemented by measurements in other urban and more rural areas. Initial data are shown in Table 6. The sampling locations in Philadelphia were near sites where spraying was taking place but after the issuance of control regulations.

These data are preliminary. They serve to establish that, at least in the areas sampled, there is a background of chrysotile contamination of the ambient air and that this is considerably higher about construction sites in urban areas. Much more information will be required, however, before reliable estimates can be made concerning quantitative levels of such contamination.

EPIDEMIOLOGICAL PERSPECTIVES

Environmental Disease

The occurrence of asbestos pollution of urban air is now established. What has not been defined, however, are the dimensions of disease which may be associated with this pollution. Indeed, it is hardly proper to speak of "asbestos air pollution" in general terms. There are different sets of circumstances in which such pollution can occur, varying in intensity, intimacy and duration of exposure. For example, the pollution which might exist about a shipyard or an asbestos mill can hardly be equated with that in the ambient air of an urban community. It would be well, at least until much more is known of the dose-disease response relationship, to separate each type of asbestos air pollution, in the study of associated disease.

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Lapsed Period

A problem common to all types of asbestos air pollution is the long lapsed period between onset of exposure and appearance of disease. In general, this is 20, 30, 40 or more years insofar as neoplasia is concerned. There are variations, of course. It may be that intensity of exposure is one such variable; others could include fiber variety, fiber size, competitive risk of asbestosis[37] co-factors as cigarette smoking,[13] trace elements, and perhaps other concomitant air pollutants.

Defined Populations

It should be recognized that the different kinds of asbestos air pollution are not limited to well separated compartments. Exposure to general community asbestos air pollution may be overwhelmed by indirect occupational exposure, in the case of a construction workman. Similarly, the asbestos inhaled by virtue of family contact in the household of an insulation worker could hardly be attributed to the scant asbestos fibrils in the air of a rural community in which that employee happened to live.

Such permutations are common and may be misleading unless identified. When considering neighborhood air pollution about an asbestos plant (30 years ago; since it would be these people with whose fate we would now be concerned) it is well to remember that, at least in the 1930s and 1940s, people who worked in a plant tended to live near it. Thus, the population about a plant being studied would have to be well characterized, to identify those with direct occupational exposure, before the effects of neighborhood contamination could be evaluated. Other examples could be given. The construction industry, for instance, employs over 4,000,000 men in the United States. They live in all areas, in all types of communities. Asbestos disease among them may not be entirely the result of asbestos air pollution in those communities. Special circumstances have also occurred. During World War II, shipyard employment rose rapidly in the United States, with close to 1,750,000 people engaged in ship building and repair at peak periods. These people, or at least those still alive, are now reaching 30 years from onset of shipyard employment and such asbestos disease as they might have as a consequence of asbestos exposure during their shipyard working days, will now be seen. Yet few of them are still employed as shipyard workers, so that unless this previous employment history is extracted, current disease could be improperly attributed to asbestos air pollution of various other sorts.

Stigmata of Asbestos-Associated Disease

The matter is further complicated by consideration of what is meant by disease caused by asbestos air pollution. Most of our current concepts are derived from the experiences of occupationally exposed workmen. While the same kind of disease can be seen in people not occupationally exposed, its attribution is much more difficult. It is complicated, moreover, by the necessity for separating those effects of asbestos which may be present but which do not actually constitute ill health, from those effects which should be categorized as disease.

Pleural plaques and pleural calcification, for example, are often associated with no disability, even when impressively extensive on roentgenograms.[1,3] The same could be said for the mere demonstration of asbestos fibers and asbestos bodies in lung and for scattered areas of minimal fibrosis associated with the asbestos fibers in the lung.

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Neoplasia is a different matter. Here, there is considerable concern. Those neoplasms which have been found associated with occupational asbestos exposure have included bronchogenic carcinoma, pleural mesothelioma, peritoneal mesothelioma, gastrointestinal cancer, oro-pharyngeal neoplasms[14] and other neoplasms are also under investigation, but more data are needed before their association can be considered firmly established.

There has been the problem of identifying neoplasms associated with environmental asbestos exposure against the background of random neoplasms of the same kind, not due to asbestos exposure. Lung cancer, for example, is highly associated with cigarette smoking and the added influence of environmental asbestos exposure is not easy to define. There is an advantage in studying mesothelioma, since evidence suggests that, apart from asbestos exposure, it has been a fairly uncommon disease. Yet even here more data are needed. It would be good to know, for example, exactly what the incidence of mesothelioma has been in the past 70 years, during decades in which the neoplasm was often not considered by pathologists. Too, it would be useful to have information on the percentage of mesothelioma which occurs other than with asbestos exposure. With such refinements, we will be able to use mesothelioma as an accurate index of asbestos disease under various environmental conditions.

Sources and Control

Sources for asbestos air pollution can be looked at in two ways. First, they can be identified and measured without reference to exposed populations. Epidemiological attention may then be attracted to these "contamination sources in search of disease." Alternatively, sources for asbestos air pollution can be studied in relation to their potential for exposure of human populations.

Both approaches are hampered by inadequate information at this time of the relative significance of peak exposures compared to constant background contamination. There are clinical experiences which indicate that heavy exposure for brief periods (days, weeks, months), with retention of the inhaled fibers for the rest of the individual's lifetime, may carry serious disease potential.[38] Therefore, intermittent high peak exposures may carry an unusual risk, especially when added to the cumulative retention associated with background pollution over long periods of time.

Adequate information is needed concerning the natural history of asbestos air pollution, including persistence, variations with meteorological conditions and ultimate fate. Asbestos fibers, being mineral, may persist in the environment for long periods. It is not known, however, if this is so, or to what extent attrition occurs by a variety of physical processes. We have found amosite asbestos fibers in the settled dust and in the household air, within homes which had been occupied 15 years before by workmen of an amosite factory. Similarly, both settled dust and ambient air in a construction workman's home contained chrysotile fibers at levels beyond those usually observed as background. Neighborhood contamination from factory sources may also be associated with long persistence of the mineral fibers. In preliminary studies, we have found this to be true of both superficial soil contamination and settled dust on attic rafters, in such neighborhoods. It is apparent that information concerning persistence and fate of asbestos air pollution would be important, if only as a background to the evaluation of air levels from current emission sources.

Natural Sources of Asbestos Air Pollution

It is likely that some air contamination occurs from natural sources. Serpentine rock outcroppings occur in many parts of the United States and other countries. Studies in our laboratories suggest that, on an ultramicroscopic level, serpentine very frequently contains some fibrous mineral components, which are properly classified as chrysotile. In addition, some outcroppings contain frank chrysotile veins. Such chrysotile-containing rocks are widely distributed, although not necessarily in commercial concentrations. It may be of interest to recollect that the original sources for chrysotile asbestos used by the company which later became the Johns-Manville Corporation, were in small deposits on Staten Island, in New York City.

Such surface deposits have long been known to geologists and are sometimes used as a guide in their explorations. The tale is told of fortuitous forest fires initiated by lightning in the 1860's in Quebec, resulting in clearing of brush overgrowth. This uncovered surface chrysotile-bearing serpentine formations, leading to the discovery of the valuable Quebec chrysotile fields.

Abrasion and weathering of such surface formations may be accompanied by release of chrysotile fibers into the surrounding air. It is expected that this has occurred. It is not known, however, whether this adds a measurable burden to the ambient air, but it should be considered as one source in the evaluation of ambient air levels. It is apparent that quantitative studies in this regard would be helpful; we are investigating the chrysotile content of ice core samples, accurately dated for many years past, in one study. Other approaches would be useful, and can be designed.

Soil contamination has already been mentioned (see above) and epidemiological data have been collected indicating that human exposure from this source can produce pleural changes.[27,28] Wider dissemination from this source is also possible, and should be investigated. Parenthetically, ground waters coursing through asbestos-bearing rock formations may be expected to become contaminated by random fibers, and water from such sources could contain occasional fibers, without invoking the necessity for asbestos air pollution.

Industrial and Commercial Sources

Although natural sources for asbestos air pollution should be considered, it is likely that they add but an infinitesimal amount to the asbestos air burden in urban areas. Most is derived from commercial and industrial sources. Here, emission-source inventories can be prepared and would include transport and storage of raw fiber supplies, manufacture of the many useful asbestos-containing products, transport and end-use of these products, and their weathering and ultimate disposal as waste. The relative contribution would vary with the type of product. In general, the potential for pollution varies with the degree of fixation of the fiber in the product. Under circumstances in which asbestos is loose or the material friable (asbestos cements, insulation materials, etc.) the likelihood of fibers becoming airborne is greater; conversely, where the fibers are more or less bound in the product's matrix, the likelihood is considerably decreased (asbestos cement materials, asbestos containing plastics, etc.). The spectrum of this variation requires detailed study, since control efforts will be guided by the findings.

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Such analyses might well be focused, at the outset, in the construction industry. Approximately two-thirds of the asbestos used in the United States is used in construction products. Ship building and repair, and waste disposal of asbestos products are important areas for study. Spraying of asbestos-containing mineral fiber insulation has already been mentioned. Factory emissions are an obvious - and controllable - difficulty. Housekeeping in all asbestos using facilities may turn out to be a knotty problem, and ultimately associated with much asbestos air pollution. Our experiences suggest that much education on appropriate methods for containing asbestos in commercial use will be necessary; here, the asbestos industry has both important responsibility and opportunity.

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Illustrations

Figure 1 Chrysotile fibrils separated from human lung tissue.

Figure 2 Examples (A-G) of the association of chrysotile fibrils and incompletely digested lung tissue.

Figure 3 A clay particle with adsorbed chrysotile fibrils, in an air sample about a construction site.

Figure 4 Adsorption of chrysotile fibrils onto surface of a diatom. The fibrils would not be seen by optical microscopy.

Figure 5 A chrysotile "fiber," considered a single unit by optical microscopy, is seen composed of individual fibrils (300Å-400Å diameter) by electron microscopy.

Figure 6 Single, short fibrils collected 3/16 of a mile downwind of a chrysotile source. Arrowhead indicates position of a clay particle.

Figure 7 Group of chrysotile fibrils in air sample near an asbestos source. Such bundles or clumps are less common in samples at greater distances from emission area.

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Table I

Asbestos Bodies in 3,000 consecutive autopsies, N.Y.C. 1966-1968

Age	Male	Female	Total
<1	2/73 (2.8%)	2/53 (3.8%)	4/126 (3.2%)
1-19	0/7 (0.0%)	4/25 (16.0%)	4/32 (12.5%)
20-39	34/102 (33.3%)	19/58 (32.8%)	53/160 (33.1%)
40-59	316/606 (52.1%)	108/247 (43.7%)	424/853 (49.7%)
60-79	555/997 (55.7%)	220/491 (44.8%)	775/1488(52.1%)
80+	106/186 (57.0%)	83/155 (53.5%)	189/341 (55.4%)
	1013/1971(51.4%)	436/1029(42.4%)	1449/3000(48.3%)

A. Analysis by age.

	Asbestos Bodies				
	0	1-4	5-14	15+	TOTAL
<1	122	2	2	0	126
1-19	28	4	0	0	32
20-39	107	53	0	0	160
40-59	429	359	45	20	853
60-79	713	630	105	40	1488
80+	152	156	40	3	341

B. Analysis by sex.

	Asbestos Bodies				
	0	1-4	5-14	15+	TOTAL
Male	958 (48.6)	802 (40.7)	152 (7.7)	59 (3.0)	1,971 (100.0)
Female	593 (57.6)	392 (38.1)	40 (3.9)	4 (0.4)	1,029 (100.0)
	1,551	1,194	192	63	3,000

Table II

Inorganic fibers (optical microscopy) in lungs of 3000 consecutive autopsies, NYC, 1966-1968. Correlation with asbestos bodies.

A.

Asbestos Bodies	Thin inorganic fibers			
	0	1-4	5-14	15+
0	1,168 (59.5%)	332 (38.3%)	35 (28.0%)	16 (33.3%)
1-4	705 (35.9%)	424 (49.0%)	52 (41.6%)	13 (27.1%)
5-14	76 (3.8%)	89 (10.3%)	21 (16.8%)	6 (12.5%)
15+	13 (0.66%)	20 (2.3%)	17 (13.6%)	13 (27.1%)
	1,962 (65.4)	865 (28.8)	125 (4.1)	48 (1.6)

B.

Asbestos Bodies	Thin inorganic fibers				
	0	1-4	5-14	15+	TOTAL
0	1,168 (75.3%)	332 (21.4%)	35 (2.3%)	16 (1.0%)	1,551 (51.7)
1-4	705 (59.0%)	424 (35.5%)	52 (4.3%)	13 (1.1%)	1,194 (39.8)
5-14	76 (39.6%)	89 (46.3%)	21(10.9%)	6 (3.1%)	192 (6.4)
15+	13 (20.6%)	20 (31.7%)	17(27.0%)	13(20.6%)	63 (2.1)

Table III

Chrysotile in 28 Cases Studied

Group	Numbers of Chrysotile Fibers + Fibrils	Cases	Male	Female
1	= or < 9	4/28	3	1
2	10-50	11/28	5	6
3	51-99	6/28	1	5
4	100-200	4/28	4	0
5	= or > 201	3/28	3	0
"Blank Grids"	= or < 9	---	---	---

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Table IV

Chrysotile Content of Ambient Air in N.Y.C.
Preliminary Results

Sampling Locations	Asbestos air level in 10^{-9} grams/m^3
Manhattan	25-60
Bronx	25-28
Brooklyn	19-22
Queens	18-29
Staten Island	11-21

Table V

Chrysotile content of N.Y.C. air in vicinity of spray fireproofing with asbestos-containing materials

Site	Asbestos level in 10^{-9} grams/m^3
1- Downwind from source	45-180
2- 45° from source	15-30
3- Upwind from source	20
4- Downwind from source	45
5- Upwind from source	20

Table VI

Chrysotile content of air in three selected locations

Location	Asbestos level in 10^{-9} grams/m^3
Philadelphia, Pa.	45-100
Ridgewood, N.J. (suburban)	20
Port Allegany, Pa.*	10-30

*We have also found amosite fibers in the air of this community; a factory using this material is present.

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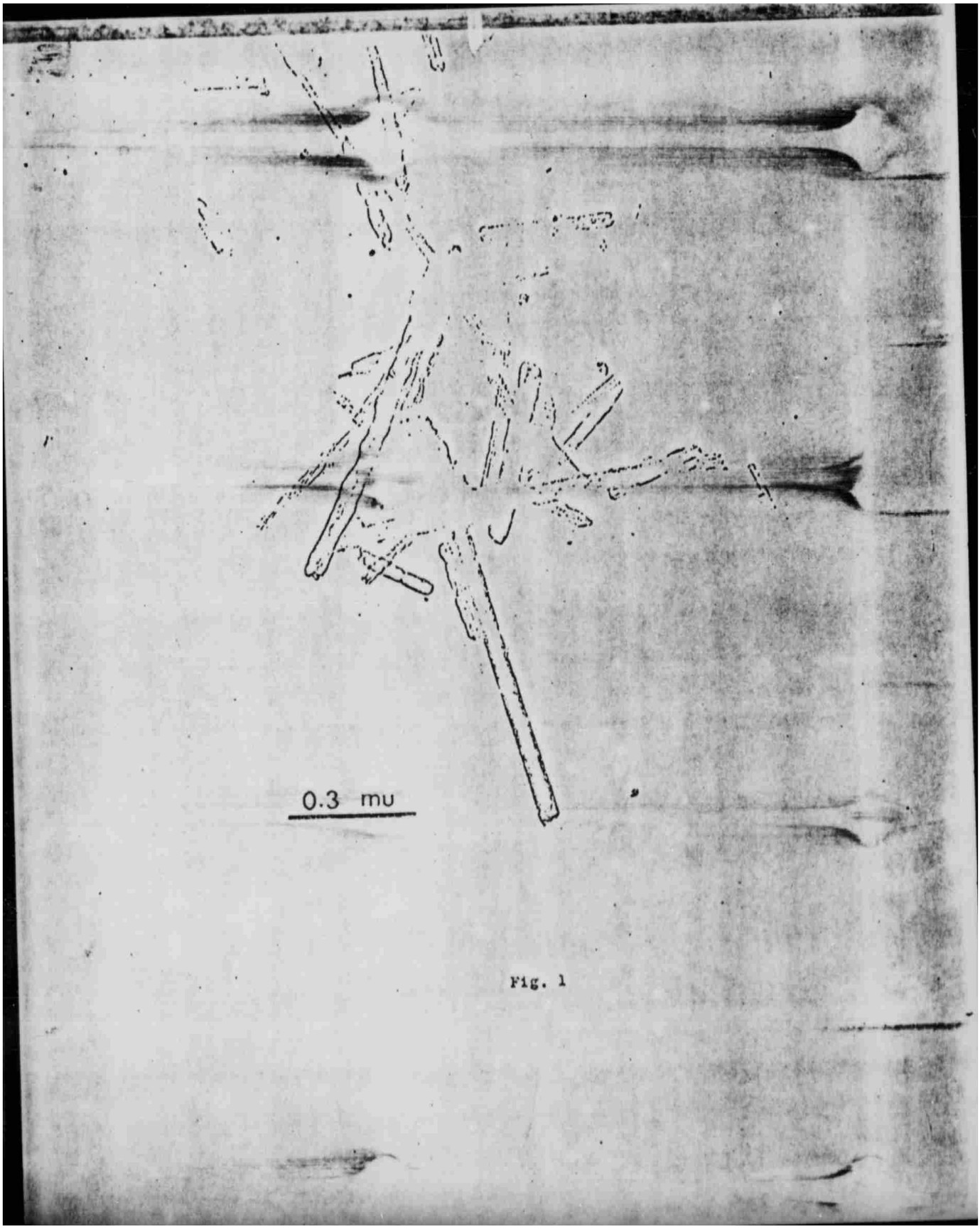


Fig. 1

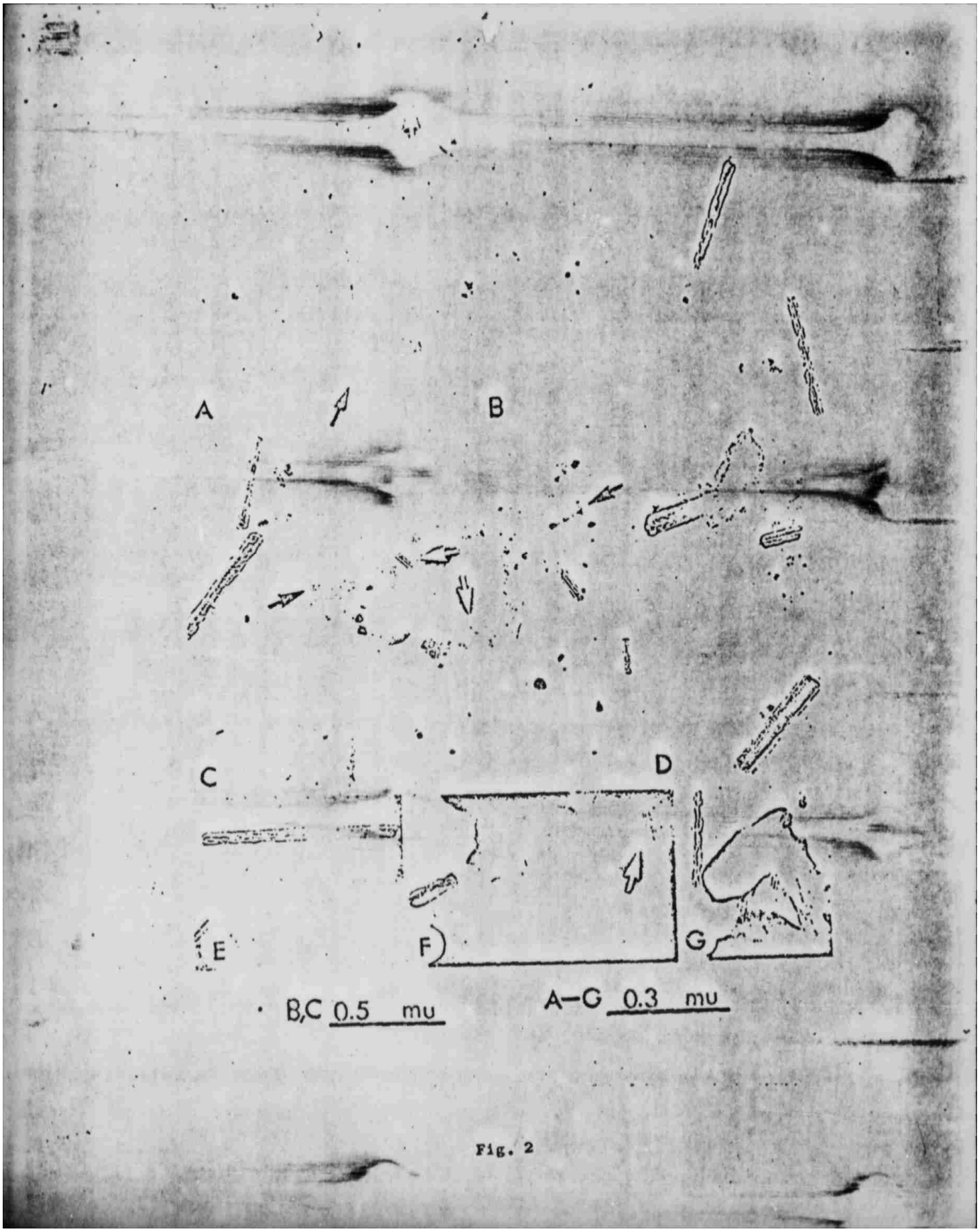


Fig. 2

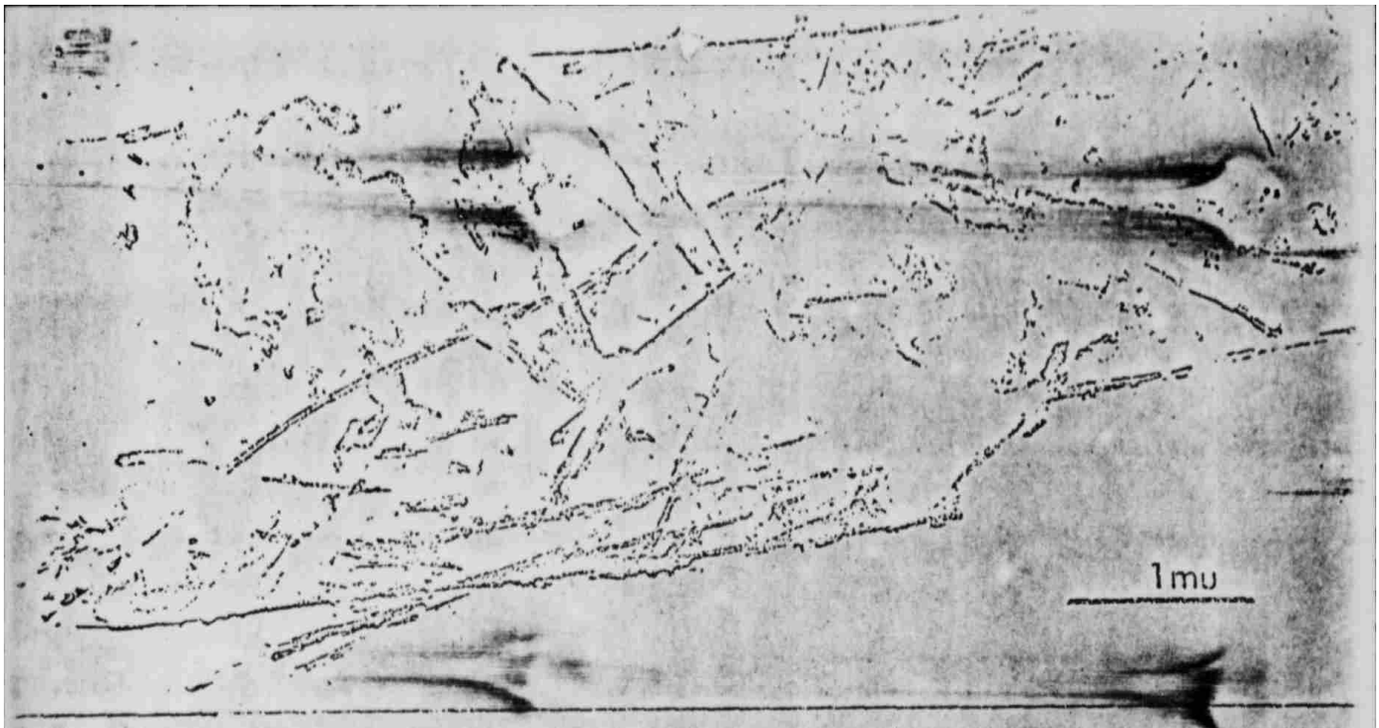


Fig. 3

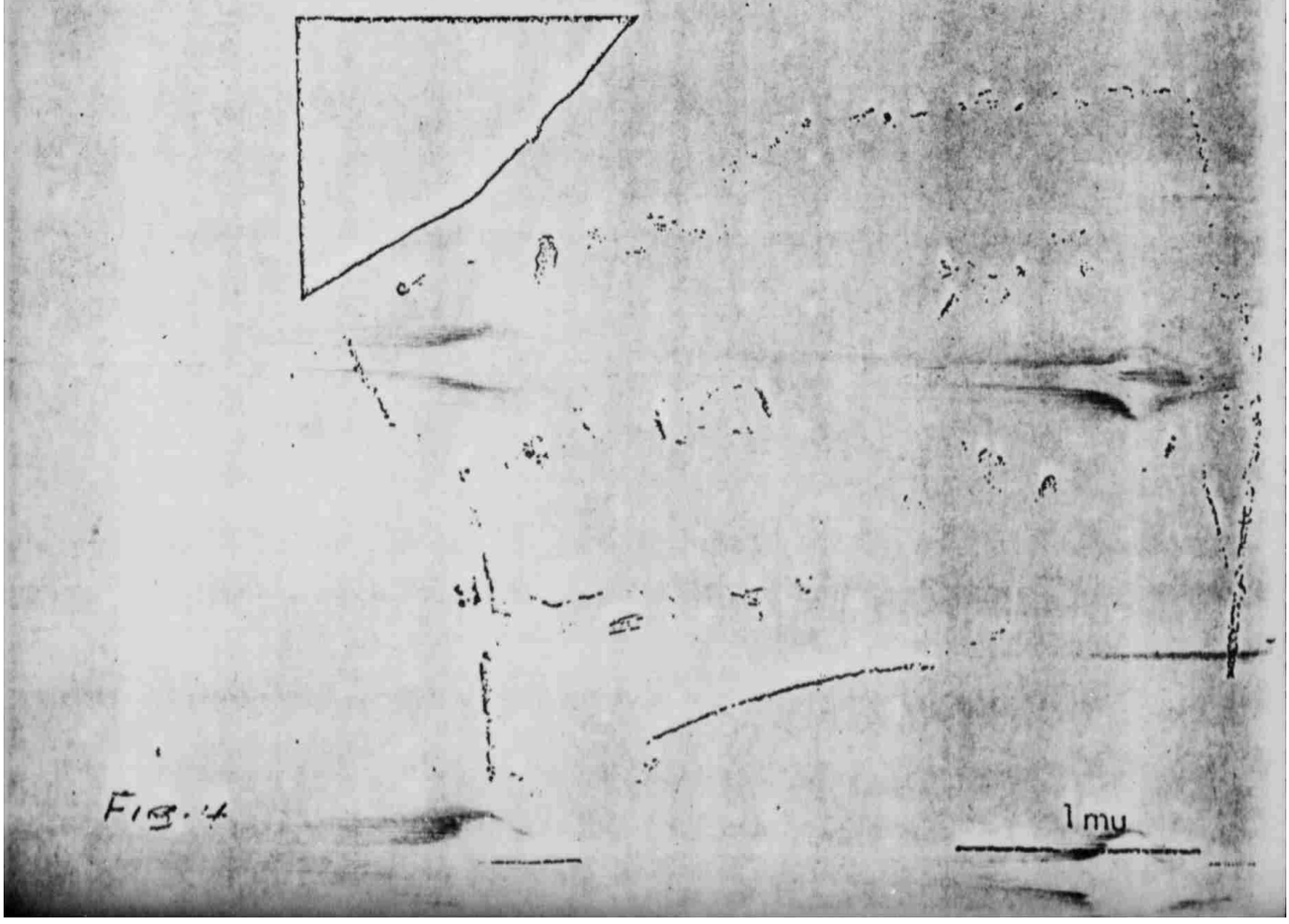


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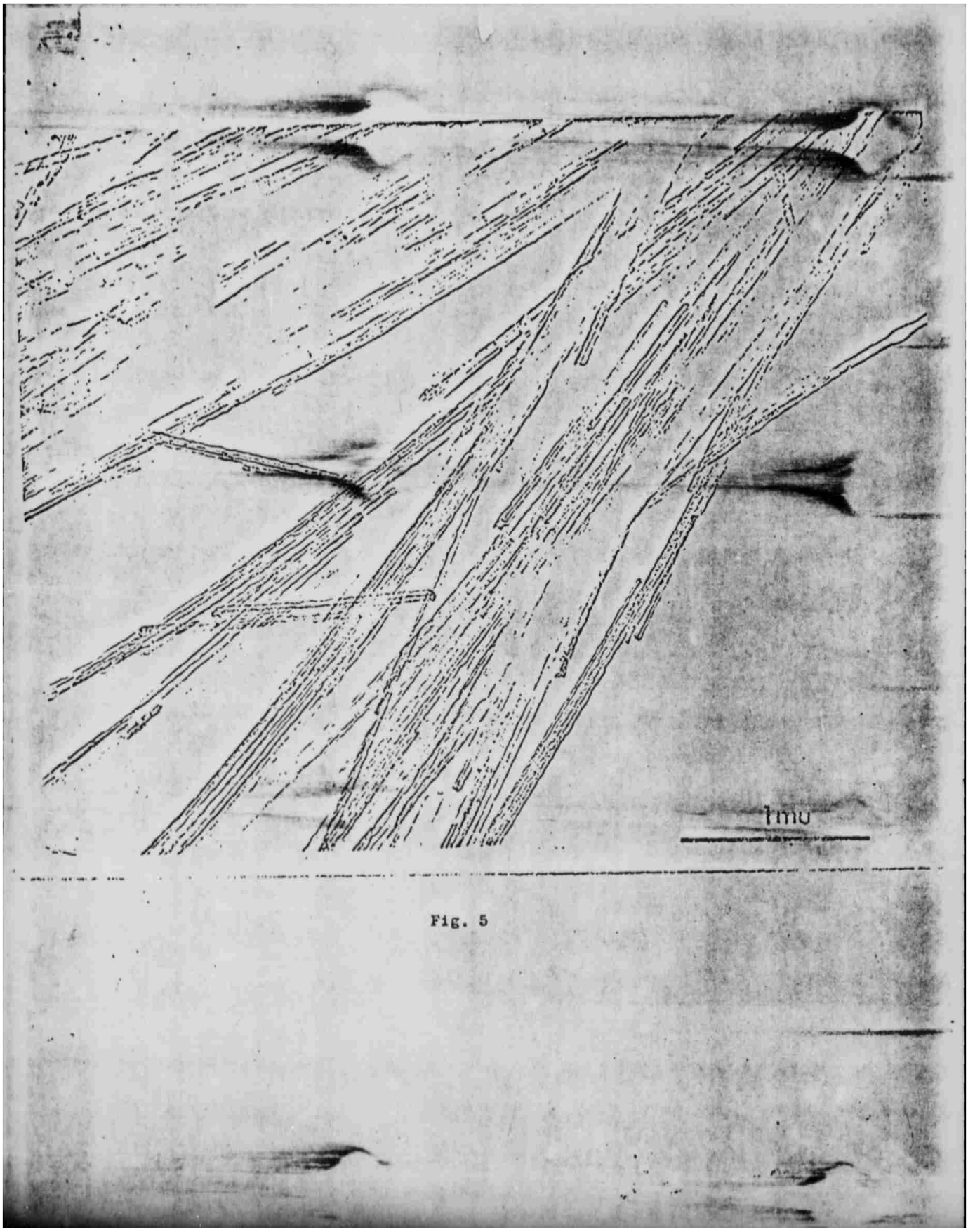


Fig. 5

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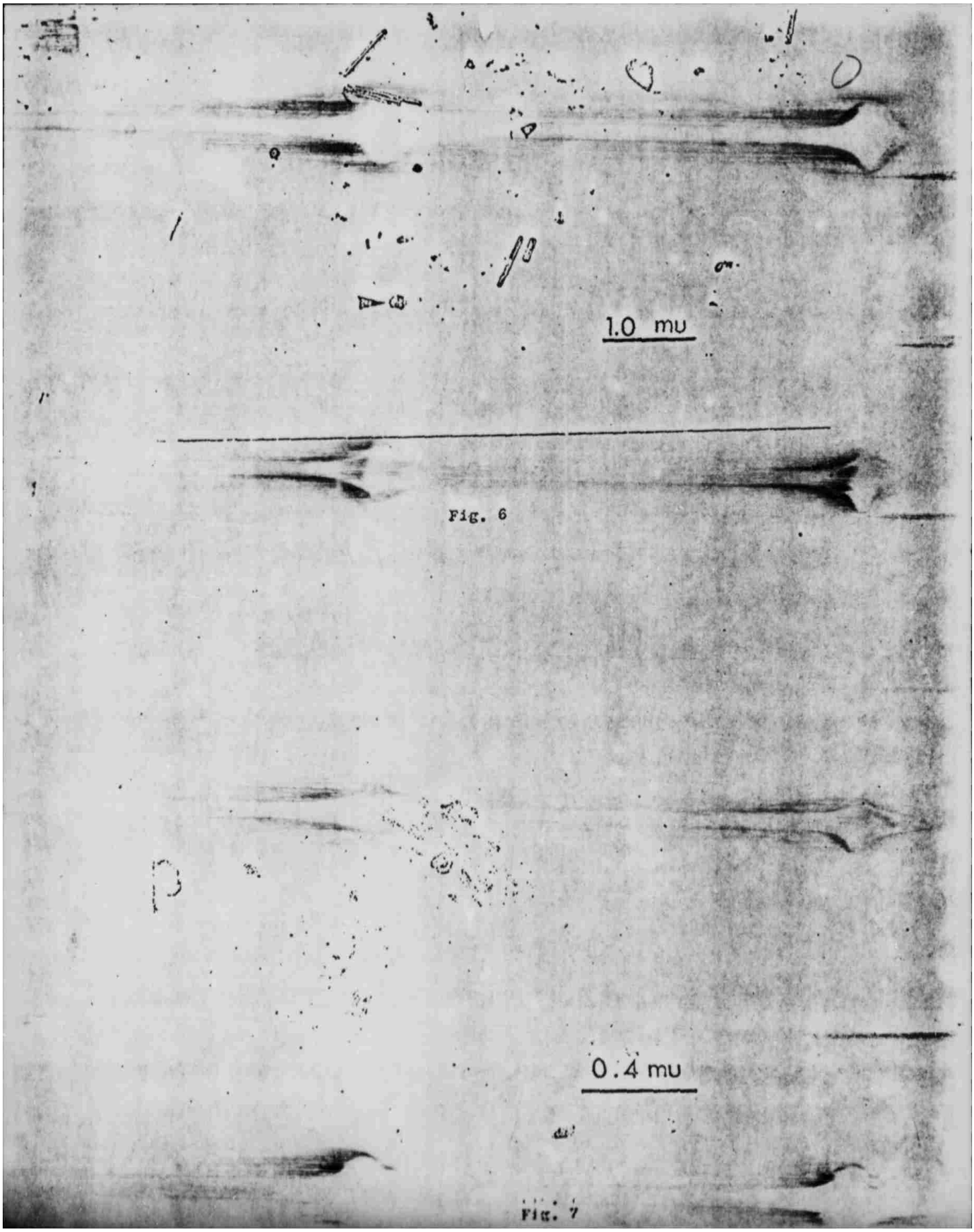


Fig. 6



Fig. 7