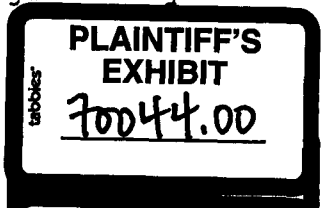
LIVELY WILSON'S DISCUSSION ON

STATE OF THE ART

As things are picking up and we're beginning to get into some of the development of the medical testimony and state of the art testimony, it might be helpful if we could talk about what has been done and what has happened in some of the other cases that have been tried, without any assurance that what happened last time might work the next time, but it might help if we all had the benefit of some of the experiences we've had. In this general region in the southeast, we've tried eight cases and the defendants have won six of them. So, at least at some point in time, somebody did something right.

I'd like to talk with you generally about the State of the Medical Art testimony and how it has been developed and the impact it has made, and generally what the State of the Art is. I would tell you that this is the State of the Art according to Wilson, it's not the State of the Art according to any particular witness because it cannot be attributed to any one person; but we've been able to develop it through the witnesses generally and the way it is outlined.



One of the witnesses that has testified for the plaintiff is a fellow named Albert Angrist. Dr. Angrist is a forensic pathologist from New York and back in the 40's and 50's, one of his residents wrote a couple of case reports that involved bronchogenic cancer and asbestos. He testified that he knew from that point on, in 1942, that asbestos was related to bronchogenic cancer. But the trouble is, in his article that he wrote, he said we really don't know, and so, the fact that he testifies that he knew in 1942, he also testifies, that maybe, he was unique, and that the general knowledge was not as general as his knowledge. So, the point is, the fact that one man may have suspected it, or knew it, doesn't necessarily mean that the State of the Art is not a viable defense.

Another generality is how medical knowledge develops; this is something about which Paul Kotin and some of the other witnesses the defendants have used have testified. Kotin makes the observation that there is a difference between a case report and a coincidental occurrence between asbestos and asbestos cancer. This does nothing to prove the cause and effect relationship because some of these case reports are subsequently wrong.

In this particular area, there were some case reports h[illegible] undertook to relate the increased incidence of asbestos exposure with tuberculosis. The suggestion early on was that asbestos exposure did have an effect on TB. But, we know now, and have known for many years, that this is not so; but, it indicates that one is not justified in putting any confidence in case reports in order to establish a causal relationship. The medical literature and the medical art generally proceed from a suggestion, an observation, to an epidemiological study that confirms a causal relationship.

In this particular area, Ford McClever, a fine and distinguished pathologist, did a tremendous amount of animal research on asbestos and cancer. He did it for years, in the 50's and never produced cancer in an animal. Medical knowledge doesn't just spring forth full blown, it develops in tortuous ways, and it is difficult oftentimes to find a finite time, when we can say, as of this point, it was known. Insofar as insulation workers were concerned, we're talking not about 1964, June 1, or what have you, but 1963, '64, '65.

William Weiss, Professor of Pulmonary Medicine at Honoman Medical College in Philadelphia has put together

some testimony with respect to long developing occupational diseases concerning the requirements necessary to establish a causal relationship. The criteria that he used were set out in the Surgeon General's report on cigarette smoking in 1964. He says that before one can say with any degree of medical certainty that a disease is caused by a particular entity, seven things are needed:

(1) Consistency - several studies, approached from different angles, but reaching the same result.

(2) Strength of Association - the frequency with which a disease is found in the presence of a particular suspected causative factor. (In cigarette smoking, for instance, the strength of association is very strong, you very seldom find bronchogenic cancer in the absence of cigarette smoking.)

(3) Specificity - The ability to predict the presence of the suspected causative factor from the existence of disease. (Considering the association between lung cancer and cigarette smoking, very rarely does it occur in the absence, so there is a high degree of specificity.)

4. Time Relationship - The exposure must precede the disease.

(5) Coherence - Does this disease, and this suspected cause, tie in with what is known generally about diseases of this type and how they manifest themselves?

(6) Animal Confirmation

(7) Most importantly, is there a decrease in the disease when the exposure is decreased?

A third factor which needs to be considered is the general environment at the time being considered. The first suggestion of asbestos as a cause of fibrosis occurred about 1927 in England. Two years later, we went into the greatest economic depression that our society has known. The concern of everybody was not that whether or not the work place was safe, but whether or not there was work to be done. The general attitude of society, or working people generally, was one of scratching and clawing to find something to do. There was simply no concern about whether or not it was safe to work in a particular place. Thus, from an economic standpoint, the consumer protective approach that is common today, was totally foreign to anybody's thinking. We moved from that time in the late 30's and early 40's into World War II and again, it would have been absolutely unpatriotic for anybody in the shipyard to complain that conditions were

not what they would like. Our focus was someplace else. Overwhelmingly so. It is in this kind of environment, then, that we're talking about how medical or scientific knowledge develops. There wasn't any emphasis on it, and even some of the plaintiffs' doctors, particularly Dr. Carnow has written and has testified, that it wasn't until the early 50's that people began to be aware of occupational and industrial hazards to the point that they were expressing concern. Now, there were times, and there were different situations and there were individual cases that came to light and some people expressed concern, but as a general rule, society didn't concern itself with those things until the 1950's.

Another factor which is relevant and that the medical witnesses support, is that there really is only a finite amount of medical expertise available. In the 30's and 40's, medical science was concerned with bacterial diseases, such as dyptheria, typhoid, and whooping cough. They just weren't focused in on diseases that occurred in the work place, these were far more serious, far more dramatic. In addition, the big scourge of medical science from a chest standpoint, from a pulmonary standpoint in the 30's and 40's was tuberculosis. There were literally millions of people at risk to tuberculosis. On the other hand, the literature

on asbestos indicates that in the 1930's, there were about 4,500 to 5,000 people at risk to asbestos-related disease. Well, medical science couldn't be concerned with a potential risk to 5,000 people when there were literally hundreds of thousands of people dying with tuberculosis. Medical science was directed away from this kind of thing. In addition, they didn't recognize that at that point in time, they were dealing with a disease that didn't manifest itself for a period of 20 to 25 years. If they found a cancer it wasn't in their thinking that that cancer may have been the result of some exposure to a noxious substance 20 or 30 years ago. Nobody made the connection. So, from a medical standpoint, all of these factors directed the attention of the community and the medical world away from any consideration of occupational and environmental diseases.

It wasn't until the late 1970's that the Federal Government began to set up some occupational disease control centers in various parts of the country. Paul Kotin has testified at some length about the fact that medical schools simply paid no attention to occupational disease. In a very recent report of the Association of Medical Schools, they point out that even as recently as the 1970's, medical schools were not turning out enough occupational physicians to staff more than 10 or 15 of the country's

major corporations. Occupational disease as a medical specialty has not had the emphasis that society now wants it to have. But that's another aspect of the medical art, a development that we need to keep in mind as we talk about it. It's awfully difficult to try to get a jury to think in 1939 and 1940 terms in 1979.

Another problem is the fact that now days, medical knowledge is disseminated much more rapidly than it ever has been in the past. A couple of years ago, a doctor in Louisville happened to observe three cases of angiocarcinoma at the B. F. Goodrich Plant that made vinyl-chloride monomer. They were nothing more than case reports, but they stimulated his thinking to such an extent, that with the aid of the Federal Government, they were able to mount an epidemiological study and determine within a couple of weeks that there was in fact a cause and effect relationship. This was disseminated to the medical community and steps were taken to protect it. In this situation, there are two interesting parallels. Cigarette smoking began its upswing in the middle teens and case reports of cigarette smoking and lung cancer began to appear in the literature in the 1920's. It was not until 1965, after an extensive study, that the Federal

Government put a warning on cigarette packages. If we're talking about cause and effect relationship, we're talking about studies that take a good deal of time, a good deal of money, and a bit of time to get in print and circulated. The obvious caveat of all this about medical art, is that if a company knew of the risk, all of the State of the Art defense in the world isn't worth a damn. I think this is best illustrated by the fact that at least J-M, in 1965, accepted the fact that asbestos was a carcinogen. But there were articles in the literature that vigorously denied the fact, even as late as 1970. Dr. Weiss doesn't think the majority of the medical community was convinced that asbestos was carcinogenic until about 1970.

In the asbestos situation, the first case report of a person, a textile worker dying of asbestosis, was in 1906. When Dr. Cooke wrote his article in 1924, he was not aware of that earlier case report at all. Apparently as a result of his writing his case report in 1924, someone called his attention to the earlier report, because in 1927, he mentioned that the Montague Murray case was the first one, his was the second one. So here is a span of 18 years before the medical community reports two cases. It gives you some idea, at that point in time anyhow, how

long it took for medical science to get the word around. In this country, the first epidemiological study that was undertaken was the Lanza study that was begun in 1928, but wasn't published until 1935. The Cooke articles "triggered" the study by Dr. Merewether in 1930, which confirmed, at least in the textile mills in England, that asbestosis was a problem. In 1935, Dr. Lanza, in this country, made his study, the first one in this country, and the concern was whether or not we had the same problem that England had in their textile mills. Sure enough we did have. It's significant that the Merewether study points out the very few people that he thought were to be at risk to asbestos related disease. He made the comment that if we could keep the dust concentration below that which this particular group was exposed we probably wouldn't see any disease. Lanza pretty much said the same thing in the sense that he said that the disability from asbestosis didn't appear to be a very severe one; not as bad as silicosis. He reached the conclusion that one could not state at this point in time what the safe level of exposure was. All of the literature at this time recognized the problem, but the implications were that it really wasn't a serious problem. As a matter of fact, the articles that immediately followed the Cooke articles, and particularly the Merewether article in 1930, said that since we've discovered the problem, and British industry

has adopted some controls, we won't expect to see this disease in the future. I think that's a significant point because following the Merewether study, the inspector of the factories adopted some regulations for British industry that specifically did not include insulation workers. There were no regulations adopted in the U. S. The regulations were significant, and the medical articles commented on the fact, that, since the adoption of the regulations, we probably won't see as much disease. And later on, in the 40's and 50's, journal articles would report that just about everybody with the disease had a substantial exposure prior to the time of industry regulations in 1930. They noted a decline in the disease after the adoption of the regulations. The point is, medical science and the industrial community, at least at that time, thought that they had identified and addressed the problem, and thought the solution would be to control the dust, which they thought they had done.

Two articles in particular, one by Dr. Doll in 1955, and one by Dr. Smith in '52 or '53, state that since the adoption of the regulations, there may have been a risk, but there doesn't seem to be any anymore.

The next study of consequence was the Dressen study in 1938, significant in the fact that it did set the standards. American industry did not so much adopt the regulations as they did set standards and say you have to comply with those standards. The Dressen study pointed out that there was no substantial disease for people who were exposed to less than five million particles/cubic foot of air, and, therefore, it suggested this as a tentative safe level. It's interesting that Dressen's study noted the threshhold concentration of dust should be the highest dust concentration that would not produce pneumoconiosis in originally healthy workmen during their entire working life. Based on that, he came up with the idea that this safe concentration to which one could be exposed eight hours a day, 40 hours a week, for his entire working life, was five million particles or less on a time-weighted average.

Now, this was reflected as a safe standard in the literature. It was adopted by the American Conference of Government Industrial Hygienists, and remained the standard until about 1969 when they began to think in terms of fibers as opposed to dust particles. Interestingly enough, it was not adopted as the standard in England until 1967.

It really was the only standard from 1938 until 1969 when they began to change it. The only other standard that was ever adopted was the OSHA standard.

The medical community had the opinion that if seven million cubic particles of dust were in the air, and if four million of those were asbestos and three million were something else then that was an acceptable working condition, eight hours a day, 40 hours a week.

There's some evolution in people's thinking about the standard stemming from the fact that in the middle and late 40's, there began to be a realization that it really wasn't the concentration of dust that was important as it was the fiber, that maybe the mechanical action of the fiber was pathogenic. They began to turn their attention not to the "total dust" but to the fibrous content of the dust. It was then that they began to understand the difference. In the Fleischer - Drinker report they measured both total dust and asbestos dust. They came to recognize a distinction and pointed out that in practically every instance the exposure to asbestos dust was below the threshhold limit and the exposure to total dust far exceeded the five million particles; but despite

that they reached the conclusion that the work that those men engaged in was not a hazardous occupation. There again is confirmation of the fact that what medical science and industry believed was that they were dealing with the asbestos content. These numbers would be the standard for a person who was working in this environment without any kind of protective equipment eight hours a day, five days a week, for his working life. A fairly recent study that Hans Weill conducted in New Orleans in a cement factory confirmed that someone could work without any protection at all while being exposed to five million particles per cubic foot of air. J-M began to develop some appreciation of the fact that it was fibers rather than ~~coal~~ total dust and it was recommended that J-M go to a fiber count in their plants. By that time the membrane filter had developed to the point that it could be used economically and efficiently to make the distinction between fibers and particles. J-M adopted a one million fibers/cubic foot of air standard for their factory. Now, even the plaintiffs' witnesses say that you cannot correlate one million fibers and five million particles. J-M did not abandon the five million particle standard but generally used both of them in those plants where it made dust measurements. So, while it sounds like a lesser

standard, it really is not, it's simply a different standard. Now, this is to me the crux of the situation. Everybody accepts the fact that asbestos related disease is a dose related disease. The more exposure, the more risk of disease. A corollary of that is that there is a level of exposure below which it would have no biological effect on a human body. This is not to say that is you breathe fiber and retain them that you don't have some biological reaction within your lungs. If we define disease as the manifestation of symptoms, then there is clearly a level below which asbestos exposure won't cause disease. It was thought that five million particles was the safe level and this exposure is that of people who were working in asbestos manufacturing plants and asbestos textile mills plants. It was thought that if one could control that exposure, the problem was solved. There simply was no comprehension of the fact that people who were using the product in the field would be exposed to the same concentration of asbestos as people in the factories. As a matter of fact, they are not and were not. What Selikoff determined was not that insulation workers were exposed to more than five million particles, but that five million particles was too high. It really didn't take that much to cause the disease and subsequent dust studies that were carried out by Balzer and Cooper in the 1968 time frame vertified the fact that generally

their exposure was more on the order of two million particles. But, it is the fact that asbestos conforms to recoqnized principles of dose response that enables us to say that the exposure to factory workers would not cause us to believe insulation workers would also be subject to disease. There are a lot of folks who have testified that there is no safe level for something that is carcinogenic. DNA research in the last ten years and other areas of research that he referred to have simply established the fact that the body is able to accept certain carcinogenic material and dispose of them without risk. One of the documents that we have used is a publication from the Department of HEW that lists every suspected carcinogen. With the exception of vinyl chloride monomer which which has a zero level, every single one of them has a safe threshold level. It is hard to believe that the government, as protective as they are, would undertake to establish safe levels for exposure to carcinogenic material if there were no safe levels.

Arguments have been made that we should have taken the hint from early British literature because that's where most disease occurred. There are three articles in particular -- one by Lanza in '36, one by Selikoff in '70

and one by Dr. Scheppers in 1964 that make the point that you simply cannot extrapolate from one industry or one environment to another. Lanza says specifically that apparently our industry is less dusty than England because the severity of disease is not as great. Scheppers makes the point that you cannot study the textile industry and extrapolate from that to the insulation industry or the cement industry. You've got to study each industry before you can say whether or not there is a degree of risk here. Selikoff says the same thing. The fact that others may have found disease in a particular industry doesn't necessarily mean that those criteria would be applicable in a different industry. A perfect illustration of this is that Dr. Doll in 1955 studied a manufacturing plant in England and determined that there was an increased incidence of cancer and that it was related to asbestosis. This provided the stimulus for a study of the Canadian mining industry by Drs. Brown and Truan in 1958 where they found no increasedincidence of cancer at all. Brown and Truan did a lousy study but the significant factor is that they were dealing with two different kinds of asbestos. There were dealing with chrysotile in Canada and amosite in England. Those factors keep one from saying that because there was a particular disease identified and

risk identified in England that doesn't necessarily mean that it can be applied in this country and it's recognized.

When we talk about insulation workers, we're talking about two medical reports, Fleischer-Drinker in 1946 and Selikoff. Drinker was an eminent industrial hygenist. He and the others determined that insulation workers were not at risk. For its time it was an elegantly done study. The study in retrospect can be faulted in many ways. There were only three people that had worked in the shipyard for more than 20 years and all three of them had asbestosis. They simply didn't appreciate the fact that you needed 20 years of that kind of exposure to develop disease. It received no criticism in the medical literature; as a matter of fact, the only recognition that it did receive was by Selikoff in '64 and by a couple of others in '66 that recognized the study as one of insulation workers and what it revealed. Between 1946 and 1964 other cases began to appear enough to justify Selikoff doing his study and to pay for that study. Selikoff indicated not so much that the insulation workers were exposed to more than what was thought to be a safe level, but that really the "safe" level was too high.

One study appeared in the Danish Medical Bulletin in 1956 -- study of 31 insulators in Denmark. Nobody knew

who Drs. Frost and Gorg were, but it was published as an abstract in the Industrial Hygeine Bulletin and circulated throughout the country.

Raybestos-Manhattan Papers are a series of correspondence beginning in the early 1930's between Vandiver Brown, who was a lawyer and corporate secretary for J-M, and Sumner Simpson. The Lanza study was a study that was paid for by Raybestos and J-M. In keeping with what is an accepted standard, they told Dr. Lanza that they wanted to see what he was going to write before he printed it. So Dr. Lanza sent Vandiver Brown and Sumner Simpson "galley proofs" of his article. He had sent them a prelim study a couple of years earlier, and this was the published study that he was getting ready to put in the U. S. Public Health Report. They wrote back to Dr. Lanza and said that they would like for him to replace a couple of comments that appeared in an earlier study that were favorable to the industry. Or they would like for Lanza to omit a comment because it would not be favorable to the industry. Dr. Lanza accepted some of their suggestions, and did not accept others.

There is another series of correspondence between Sumner Simpson and the editor of the magazine called

"Asbestos", a trade association magazine for the asbestos companies, in which he objected to "Asbestos" magazine running articles about some of the English literature that indicated that there was a disease potential. He noted that it hadn't been established yet, and didn't think it would be good for the industry to have articles printed about the English experience that may not be applicable here. The plaintiffs have seized on that correspondence as some attempt by J-M and R-M to conceal the State of the Art, to conceal the fact that they knew that asbestos was pathogenic, and to implead the flow of information about this situation.

The answer to that, is that if they were trying to keep from the medical community or the industrial community the fact that asbestos would cause disease in textile mills and in the workers, they did a poor job of it; Lanza's article was published in 1935, which included all the essential findings. Secondly, we're talking about factory workers and we don't have any factory worker cases here, and so, whether or not we were trying to conceal that information back in the '30's, really doesn't have any significance where we're talking about somebody in an entirely different industry.

Kotin Deposition

Paul's deposition in Denver was an absolute disaster. He gave monosyllabic answers on cross-examination that absolutely ruined us. It was just awful. I suppose that he was not feeling well that day, we had only had an afternoon with him before, although we had asked for more time. I suppose that after having had your deposition taken ten times, you lose your spontaneity, to say the least. Motley was much better prepared. The deposition was lousy.

Somewhere between October and November 30, we put him back on re-direct and he redeemed himself. We would ask if he responded in a particular way and he would qualify his statement. I think Paul has had his interest piqued in this area again, and has indicated a willingness to lend a hand. As we get into our more serious bronchgenic cases, I think he'll be available to us.

Treating Physicians

Most treating physicians don't get into the subject of the State of the Art. All they know is that they treat what they see, they know asbestosis when they see it and that's it, and that's generally been the way they've come on.

Asbestos and Cancer

There are three medical articles that are significant in this area. One is the article by Lynch and Smith in 1935 which is the first report of the coincidental appearance of lung cancer and asbestosis.

The second article is the article by Dr. Doll in 1955 which is an epidemiological study of the factory workers in England; it was the first study which determined that there was an increased incidence of cancer and a causal relationship between asbestos exposure and bronchogenic cancer. Between 1935 and 1955, there are probably between 50 or 60 case reports where people have reported that they saw lung cancer and the guy either had asbestosis or he had a substantial exposure to asbestos. But no competent scientist could say that the two were causally related until the Doll study made that connection. Interestingly enough, until about 1953, nobody ever raised the question of what/the smoking habits of these people were. Dr. Doll's study does not mention the smoking habits of his cohort.

Selikoff's article in 1965 determined that insulation workers were at risk.

Perhaps there is a fourth article that we ought to add to the list and that is Selikoff's 1972 article which asserts

that in the absence of cigarette smoking there is not an appreciable increase in the risk of lung cancer. An awareness of the relationship between cigarette smoking and lung cancer is as important as any knowledge of the carcinogen asbestos. Hans Weill has written and testified that in a bronchogenic cancer case, one simply cannot ascribe a cause because of the overwhelming impact of cigarette smoking. I don't think we ought to be reluctant to try any case involving bronchogenic cancer where the plaintiff has smoked, because the chances are 99 - 1 that emphysema, chronic obstructive disease and other smoking diseases will be present in connection with the cancer.

Weiss, in his papers in 1970 and 1975 has pointed out not only that it is a causal relationship insofar as cancer is concerned, but that cigarette smoking so compromises the protective mechanism of the pulmonary system that it also enhances the risk of asbestosis -- it doesn't cause asbestosis - but it compromises the mechanism that clears the lung, permits the asbestos to get into the lungs and to stay there and has that effect on it.

Mesothelioma

Mesothelioma is a third related disease. The first

suggestion of the connection was in 1960, when Dr. Wagner did his survey in South Africa. It is interesting to note that he was doing a study of people working around the mines, in two different provinces, and he found a lot of meso in one area and he didn't find any in the other. He suspected that it might be related; but it is interesting that we find it in this group of people who are exposed and we don't find it in this group with the same type of asbestos. His study made such an impact that it provoked a world of research and a half dozen articles to be written between 1960 and 1964 examining mesos in various parts of England. He concluded his own study in 1965, stating that there is an increased incidence of meso in people exposed to asbestos.

Let me caution you about one thing -- we're not suggesting that an epidemiological study of this kind proves that there is a causal relationship between asbestos and this disease. It is not a clinical study, we can't implant it in a person, and watch it and get this result. What these studies are saying is that there is an increased incidence of disease in this given population who have been exposed to asbestos.

Two things we know, and that is that mesos appear in the general population among people who have had no asbestos exposure whatsoever. We don't know what causes it, the only thing that has been identified is asbestos, but just as sure, there is something else in the environment which can cause meso. It's been stimulated by other elements in some animal studies. The point is, that just to say someone has meso that was exposed to asbestos, does not necessarily mean that the two were related. It could have been caused by the asbestos, but it also could have been caused by something else. One study done at the Mayo Clinic found a majority of cases had no asbestos exposure at all.

The second thing is that meso, like just about every other carcinogen, is a multi-factorial disease. Two things have to come into play in order to produce the problem. Mesos ought to be the easiest to defend from a State of the Art question because there was no appreciation of the disease until 1965. They are easy to defend, one because we don't know what else causes them, and, two, we don't have the smoking factor that we have in the bronchogenic cancer and asbestosis cases.

Now, there are a couple of things that we do have in them. One is the fact that a meso is an extremely rare cancer. We're talking about one in a hundred thousand cancer deaths in the general population. So it is an extremely rare disease. And the average pulmonary physician wouldn't know it if it slapped him in the face.

It seems as though the plaintiffs' position in this case is premised on the fact that the manufacturers had a duty to warn. If we did not know or should have known that asbestos was a factor, or was dangerous, then what's there to warn against? It goes to the question as to whether or not we have a duty to warn. I think it applied in every case with the possible exception of plant worker cases. State of the Art as a defense to strict liability can be asserted on the theory that a failure to warn is the defect. By definition, the product is not defective if there was a warning on the product. And it wasn't defective if there was no knowledge to put a warning on the product.

We've also had it argued that we had a duty to test. They have asked why we didn't conduct dust counts in the field to determine whether or not these people were exposed to pathogenic quantities of the material. If we had indeed done that, we would have found that insulators were exposed to less than what was generally thought as a safe level.

Owens-Illinois Documents

Let's talk just a minute about the Owens-Illinois documents. We talked about Raybestos-Manhattan, and since all of us are affected by these things, I'll give you my thoughts about the OI documents.

In 1943, Owens-Illinois developed a product called Kaylo. It is a hydrous calcium silicate with about 15-20% asbestos in it. The industry being what it is, it wasn't long before everybody else had a hydrous calcium silicate laced material on the market, too. OI sent samples of the material and of the dust created when it was manufactured to Seranac Laboratories and said that it was a new insulation material they were manufacturing and they wanted Seranac to test it and see whether it was dangerous for people in the work place or for those who would be using it in the field. First thing, Seranac did some preliminary tests on it. They thought OI had a substitute for asbestos, but they tested it anyway.

Again OI asked if it posed a hazard to people who would use it. Seranac proposed to expose a lot of animals to Kaylo dust and see whether or not it caused any disease.

Basically Seranac was and always has been an animal research laboratory. That's what they did best. What they did in this instance was to take the poor little mice and rats and guinea pigs and hampsters and everything else and expose them to this material in concentrations of up to 200 particles to a million particles per cubic foot of air for an extended period of time. Sure enough, they developed some fibrosis. Now, what they try to hang us with is the fact that OI asked all the right questions. 'Is this going to be a hazard to people working with it in the field?' They asked an eminently qualified laboratory to determine that for them. Seranac didn't respond to that question. The tests which they told OI they were going to run and which they did run were not calculated to determine whether or not a user was at risk. To do that, you've got to know how much, and there was never anything in any of the OI tests or the Seranac tests that was calculated to determine how much of the product would cause disease. With animal experiments, you can determine whether it will cause disease but you simply cannot extrapulate from an animal study to determine how much of it it takes to cause disease. In that way, we get back to dose response again.

The only explanation can be that Seranac, with its eminence in the field was aware of the then current TLVs. Some of the documents that have been discovered at Seranac make reference to the fact that there were TLVs on other material and they simply did not appreciate the fact that people who were using or would be using a product like Kaylo would be exposed to the concentrations that would be required to cause the disease. The reports laid dormant at Seranac, and OI kept inquiring as to when they were going to finish the thing and publish it. The man that did the experiments was a man named Worwald. When Schepers came in, he reviewed the experiments and published them in 1955. It is interesting that the article he wrote simply says something about the fibrous nature of commercial hydrous calcium silicate. If you looked at the title of the article you wouldn't think that it had anything in the world to do with asbestos. What they concluded from that study is simply that hydrous calcium silicate contains 15 to 50% asbestos and behaves just like any other asbestos material. So as far as adding to the sum total of knowledge at the time they didn't think it did. The danger, of course, the problem, from the jury's standpoint, is that OI had the foresight to ask and all we can do is speculate as to why Seranac didn't answer them. I can only assume that if there had been any question in their mind about it they

would have done something about it.

How is the OI request relevant to other manufacturers? I don't think it is. The published literature on the OI tests would not affect any of us.

One of the problems with the Raybestos and the OI literature is the manner in which it came to light.

The R-M papers came to light in a normal motion to produce. As far as the raybestos papers are concerned, JM still doesn't have the correspondence, we have a better records disposal program than R-M had. The OI people can make an awfully good story on their documents because included in the OI correspondence, the OI file, are a number of letters asking Seranac "When are you guys going to get this stuff out? We want to publish it." So the published report of the results of the OI Seranac studies is in the literature as of 1955.

There is probably not any relevance of the Raybestos letters to the other side. Wouldn't it be appropriate for motion to be remedied to prevent that evidence to be introduced as to other defendants? Yes, I think you could do it that way, I think you could go even farther and not just restrict its being introduced as to other defendants but to

be restricted, excluded all together, because the adverse impact on the other defendants would far exceed any beneficial impact on plaintiff's case. There is no way a jury could separate that.

Well, I've told you about all I know about the asbestos litigation. The plaintiffs have gotten a great deal stronger in the presentation over the last two or three years as you might expect; there are many things that are favorable to their position that I have not touched on. For example, there is a report of an industrial hygenist named Hemian, that did a study of some textile plants in the east back in the 40's that was critical of the threshold limit value of five million particles. He went to the Asbestos Textile Institute, and suggested that they do some tests and run some further studies to see if they couldn't get a better handle on what was actually a safe level and the ATI turned him down. Of course, they make a great hue and cry about that, but again we are talking about the asbestos textile mills and not talking about insulation workers, we are not talking about - and we need to recognize the fact that one could do all the tests in the world in the textile mills and it wouldn't have any relevance to this situation here.

There is an even stronger case for the shipyards than there is in the insulation field because while shipyards employ insulators not all the people at shipyards are or not all the plaintiffs are. Selikoff wrote an article in 1978 called "Asbestos-Associated Disease in United States Shipyards" that's probably the first which includes this catagorical statement - 'In 1968 the first warning that asbestos disease might be a serious problem in shipyards was sounded by Harries in Great Britain and Stumphious in the Netherlands when they reported instances of mesothelioma among shipyard workers.' If that was the first warning that shipyard workers generally were at risk to disease, then certainly all of us had labeled products and are in pretty good shape from that standpoint. Now here again, there are case reports of a shipyard worker who has mesothelioma or a shipyard worker who had asbestosis but this is a pretty good place to hang your hat as far as shipyard cases are concerned.