STATE OF NEW JERSEY)
) SS:
COUNTY OF MIDDLESEX)

IN THE SUPERIOR COURT OF NEW JERSEY
LAW DIVISION: MIDDLESEX COUNTY

JOHN PETERSON and SHIRLEY MAE PETERSON, his wife, Plaintiffs,	)	
vs.	)	No. L 060148-87
UNION CARBIDE CORPORATION, Defendant.	)	Civil Action

The continued discovery deposition of SAMUEL EPSTEIN, M.D., taken in the above-entitled cause before Sandra A. Kaspar, a notary public within and for the County of Du Page and State of Illinois, and a Certified Shorthand Reporter of said state, at 303 West Madison Street, Suite 1400, Chicago, Illinois, on the 16th day of December 1989 at 10:30 o'clock a.m.

APPEARANCES:

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On behalf of the plaintiffs;

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On behalf of the defendant.

I N D E X

EXHIBITS

Number	Marked For ID
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MR. HOLLINGSHEAD: Dr. Epstein, good morning.

THE WITNESS: Good morning.

MR. HOLLINGSHEAD: As you know this is a continued day for your first deposition and you're still under oath from the first day in New York.

THE WITNESS: Yes, of course.

SAMUEL EPSTEIN, M.D.,

the witness on the stand at the time of adjournment, having been previously sworn, resumed the stand and testified further as follows:

EXAMINATION (Continued)

BY MR. HOLLINGSHEAD:

Q. Doctor, I believe at the last deposition there were some items you were going to find for me, and I understand you brought some documents with you today.

Would you identify what you brought today?

A. Yes. The first is a deposition of Strader, S-t-r-a-d-e-r, versus Franklin Electric. It's my deposition on a case of meat wrappers asthma. The second is a deposition of myself in a case of Starling versus McDonnell Douglas, and this is a case of nasal sinus cancer following multiple

exposures including asbestos.

The third is a batch of documents relating to cases you asked me to dig out. The first is Grasso versus B. F. Goodrich. If you recall that was a VC case which we talked about last time. It's a report of mine which I kept, a report of January '81, and a letter from the attorneys so you can get all the information from the attorneys. Grasso - had some info at one time but dont recall case

The next is another vinyl chloride case, Ellias, E-l-l-i-a-s, versus Parke Davis and Spray-on, one word, with a letter from the attorney and an affidavit from me. I was deposed in that case but I haven't got the deposition. Baby got hepatic b dextroma from PVC spray bandage

The next is a letter -- the next is another case, a VC/PVC case, called Mikyska, M-i-k-y-s-k-a. I think it's Mikyska versus B. F. Goodrich and possibly Ford -- yes, it is. I think so -- and it's a letter of 1981. It was -- I don't have any other documents in this case. This was -- I believe I was deposed. I don't think there was a trial but I don't have any records. Mikyska was PVC film laminator Victim of mal practice No cancer

The next is a VC case of Ferrara, F-e-r-r-a-r-a, versus Tenneco, T-e-n-n-e-c-o;

letter from the attorney and a preliminary report by myself. I don't believe I was deposed. There certainly was no trial.

The next one is a meat wrappers asthma case -- well, actually one, two, three, four meat wrappers asthma cases. The first is Earle, E-a-r-l-e, versus Cryovac, C-r-y-o-v-a-c. The second is Wall versus Hobart, H-o-b-a-r-t. The third is Strader versus Franklin -- that's a bit silly of me.

The very first document I gave you was the deposition so this is just a letter with the attorney's name, et cetera.

The next is Sutkaitis versus Goodyear, S-u-t-k-a-i-t-i-s, which there is a report from me, a letter from the attorney. No deposition to the best of my recollection, no trial. The next is -- that's -- those are all the meat wrappers asthma cases.

The next is a letter from the attorney on the asbestos case -- on another asbestos case. This was a coworker of Starling called Ski, and it's Ski -- and that was a nasal sinus cancer in which asbestos was involved, S-k-i.

And the final one is a letter from the attorney in relation to the Starling case, and you've got the deposition of the Starling case here. That's the asbestos.

(Discussion off the record.)

THE WITNESS: Now in addition to that I've got some other tables and some corrected tables from my notebook. The first is a document called State of the Art on Residual VC in PVC Prior to 1975. I now have that in my notebook. It wasn't in the copy I sent you.

The next is -- there was a table if you recall in the book I sent you called Illustrative Literature on the Toxic and Carcinogenic Effects of VC/PVC in the Respiratory Tract of Exposed Workers. I have added quite a bit to that so this is a new table for that.

The final one is just a table from a paper by Infante et al. 1981 on respiratory tract cancers.

MR. HOLLINGSHEAD: Before we mark the documents you provided me with, let me refer to an index that the court reporter gave to me from the last deposition. I had asked her if she would just

give me a list of items that had been requested. .
BY MR. HOLLINGSHEAD:
Q. I had requested a curriculum vitae and you sent that to me in the mail?
A. Yes.
Q. The next item was a list of publications that you have authored over the course of time, and that's also included in the C.V., is it not?
A. Sure.
Q. Next were formal reports written in the Ellias, Grasso, and Mikyska cases, and you have given those to me today, correct?
A. Yes.
Q. The next was to provide deposition transcripts of any deposition of yours in those three cases, and I believe you've given us the deposition transcript from the Strader case.
Were there depositions taken in the Ellias, Grasso, or Mikyska cases?
A. Yes. There's been a deposition taken in the Grasso -- the Grasso case went to trial. So there was a deposition and a transcript of that, neither of which I have.
Q. And you don't have the transcripts from

the Ellias or the Mikyska cases?

A. No, I don't have them. The Ellias case didn't go to trial. I was deposed on that.

Q. Do you have that transcript?

A. No, and the Mikyska -- I can't recall whether I was deposed. I wrote a brief report on it but I can't -- it didn't go to trial.

Q. And you don't have a transcript of any of your testimony in that case, deposition or otherwise?

A. I'm not sure that I was deposed in the Mikyska case.

Q. The next item I had requested was documentation which might include correspondence with the attorneys on those three cases, and I believe you have provided us with some of that?

A. Yes.

Q. The next request was for any expert reports in any of the cases on asbestos.

A. Yes.

Q. And you've provided us with what you have on that?

A. Yes.

Q. And that's all you have with regard to

that item?

A. Yes.

Q. The next was for any other expert reports on isopropylidene bisphenol resins.

A. The only place where that came in is in the meat wrappers asthma. That was one of the exposures in the meat wrappers asthma cases.

Q. And you have given us what you have on that?

A. Yes.

Q. The next was any documentation regarding your involvement in any of the bisphenol cases. Same thing?

A. Yes.

Q. The next was any report rendered with respect to the NASA, N-A-S-A, plant worker who apparently had laryngeal cancer.

A. I mislead you on that. I was under the impression that one of these was a laryngeal cancer. In fact both are nasal sinus cancers.

I still am under the impression that I had an involvement in the laryngeal cancer case due to asbestos, but I searched and spent a lot of time looking in the basement and storage areas. I can't

lay my hands on it.

And I don't know whether it's faulty memory or I was -- as I said before I was under the impression that one of the NASA cases was a laryngeal cancer because I know I did a search of the literature.

Q. What did you find during your search that made you conclude they were not laryngeal cancer cases?

A. The documentation is very clear. They're both nasal sinus cancers.

Q. Now which were the NASA cases?

A. Both the Ski and the Starling. They were both coworkers in a NASA plant in Jacksonville, Florida.

Q. The next request was for any reports that you had written independent of the Peterson case relating to the toxic properties of any of the chemicals that may have caused his illness.

That would of course be the chemicals in this case and have we covered that?

A. Yes.

Q. Did you find any reference -- and I had requested this, but did you find any reference to

information supplied by Mr. Levinson's office regarding Mr. Peterson's exposure to asbestos at the time of his employment?

A. No. As I explained last time I had a two-page note from his office specifying the details of this. I incorporated that in the occupational history and then tossed it out. It was an unsigned -- I remember it was a two-page, unsigned piece of paper I got from the office.

Q. So I'll follow up on that with Mr. Levinson's office. Very good.

Let me ask you --

A. Can I see the recent curriculum?

MR. HOLLINGSHEAD: I have shown Dr. Epstein the C.V. that he provided to me in the mail the past week or so, and I'd like to mark this as Epstein 5.

(Whereupon, Deposition Exhibits 5-17, Witness Epstein, was marked for identification.)

MR. HOLLINGSHEAD: Dr. Epstein, we have marked the documents that you provided me with today and also the C.V. which you provided me with during the

week.

The C.V. is Epstein 5. The transcript in the Strader case is Epstein 6. The deposition in the Starling case is Epstein 7. We have marked as Epstein 8 a letter from an attorney by the name of Ellis E. Neder, N-e-d-e-r, Jr., dated May 14, 1982.

We have marked as Epstein 9 a letter and attachment from the law firm of Brown, Connery et al. dated February 9, 1981. The attachment is a report on John Grasso prepared by Dr. Epstein on January 21, 1981.

We have marked as Epstein 10 a letter of January 16, 1987, from the law firm of Brown Tyrell (phonetic) et al. to Dr. Epstein, and attached is a one-page report on Andrew Ski, S-k-i.

We have marked as Epstein 11 a letter from the law firm of Philo, P-h-i-l-o, Atkinson et al. to Dr. Epstein on the Earle case, E-a-r-l-e. The letter is dated October 26, 1984.

We have marked as Epstein 12 a letter from the same law firm dated February 3, 1984, to Dr. Epstein on the case of Wall versus Hobart, H-o-b-a-r-t.

We have marked as Epstein 13 a letter

from the law firm of Watkins, Boulware, B-o-u-l-w-a-r-e et al. dated December 9, 1983, to Dr. Epstein on the case of Strader versus Franklin.

We have marked as Epstein 14 a letter of December 1, 1986, from the Philo law firm on the case of Sutkaitis, S-u-t-k-a-i-t-i-s, versus Goodyear; and attached to that is a two-page report by Dr. Epstein regarding Mrs. N. Sutkaitis.

We have marked as Epstein 15 a letter of January 16, 1985, from the law firm of Smith & Goldstein on the case of the estate of Lucy Ferrara versus Tenneco Chemicals; and attached to that is a one-page report dated November 30, 1984, by Dr. Epstein entitled Preliminary Report on Mrs. Ferrara.

We have marked as Epstein 16 a letter of July 9, 1981, from the law firm of Bogus & Bogus, B-o-g-o-s, directed to Dr. Epstein on the Mikyska case and attached to that are -- excuse me. It is a three-page letter. There is no attachment.

Lastly as Epstein 17 we have marked a letter from the law office of Stanley M. Rosenblatt dated April 6, 1979, to Dr. Epstein on the Ellias case; and attached to that is an affidavit by

Dr. Epstein of four pages in that same case.

Let me put those aside and I will take them home with me and copy them and, Dr. Epstein, I will send back to you as you have requested the deposition transcripts in the Strader and Starling cases.

THE WITNESS: Thank you.

MR. HOLLINGSHEAD: Now if we can I'd like to turn to the tables that you have provided me with as you've indicated would constitute a change in what appears and what we have previously marked as Epstein 4.

I'm looking now at the document entitled Illustrative Literature on the Toxic and Carcinogenic Effects of VC/PVC in the Respiratory Tract of Exposed Workers.

BY MR. HOLLINGSHEAD:

Q. Can you tell me where the additions appear in that document or the changes?

A. We'd have to go over this one --

MR. HOLLINGSHEAD: Why don't we mark that first as Epstein 18.

(Whereupon, Deposition Exhibit No. 18, Witness Epstein, was marked for identifiction.)

BY MR. HOLLINGSHEAD:

Q. Just so the record reflects, Dr. Epstein, I understand that you have taken this page from the exhibit --

A. Not this page. These several pages.

Q. -- these several pages from the exhibit that had previously been marked as Epstein 4. You updated them and then unfortunately you discarded the earlier pages; is that correct?

A. Yes, having sent both you and Mr. Levinson copies of the original document.

Q. Yes, I understand that. I would ask from this point on that any exhibits that are left in your possession remain as they are when they were marked, all right?

A. Sure.

MR. HOLLINGSHEAD: Mr. Levinson, I do have copies of this table so we will replace it into that report.

MR. LEVINSON: All right.

BY MR. HOLLINGSHEAD:

Q. Let's look at this exhibit we have now marked as Epstein 18 and compare it to my copy of what was previously part of Epstein 4, and I would ask you if you could point out the changes for us.

A. Thanks, and I will put a tick on these.

Q. You don't need to do that. If you can just describe what the changes are.

A. Nothing on the first page.

Q. On the exhibit we just marked as Epstein 18, you put a check mark on the second page to a study by Byren, B-y-r-e-n, and that's a new addition; is that right?

A. Yes. There's a new one, Bufler, et al., B-u-f-l-e-r, and a new one, International Agency for Research on Cancer, '79. Then Infante, '81.

Q. And that's on page 4.

A. Then International Agency for Research on Cancer, page 5 -- that's 1987. And then Doll, '88, page 5. All that's all the additions I have ticked on Epstein 18.

Q. Now you have also got a document entitled State of the Art on Residual CV in PVC Prior to 1975.

Is that a new document or is that an update of something that was in Epstein 4?

A. Well, I think these references were -- these people were probably cited in paperwork in Epstein 4, but I just put them in the table to systematize this information.

Q. Did you take anything else out of Epstein 4?

A. No.

MR. HOLLINGSHEAD: Can we mark this then as Epstein 19.

(Whereupon, Deposition Exhibit No. 19, Witness Epstein, was marked for identification.)

MR. HOLLINGSHEAD: Lastly you provided me with a one-page document entitled at the top Infante et al., 1981.

BY MR. HOLLINGSHEAD:

Q. Can you tell me what that shows?

A. It's just a table from his paper on respiratory cancer in workers exposed to VC.

MR. HOLLINGSHEAD: Mark that as Epstein 20, please.

(Whereupon, Deposition Exhibit No. 20, Witness Epstein, was marked for identification.)

BY MR. HOLLINGSHEAD:

Q. Dr. Epstein, is there another page within Epstein 4 that you think you may have altered in some fashion?

A. Yes, I think I made an addition on State of the Art on VC/PVC Toxicology Prior to 1974. I'd like to check on that if I may.

Q. I will provide you with the copy that I have.

A. One minor thing under the animal data for Viola. I have handwritten in my copy at the end of the handwritten material in your copy in parens: "At 250 ppm admitted to NIOSH, 1973."

Q. I also note that what you did was to retype this page?

A. That's right.

Q. What appears in handwriting on the copy that I have, which was marked two weeks ago, has now been turned into print.

A. Yes, the Viola '70, the Tribukh '49, and

the NIOSH '73.

MR. HOLLINGSHEAD: Just so we can distinguish one from the other, I want to mark this as a separate exhibit.

THE WITNESS: Let me have a copy of it, please.

MR. HOLLINGSHEAD: Yes, we will. Let's mark this as Epstein 21.

(Whereupon, Deposition Exhibits 21 and 22, Witness Epstein, were marked for identification.)

MR. HOLLINGSHEAD: For the record let me describe what we have done.

We have managed to put back in its entirety the document that was marked as Epstein 4 at the last deposition by taking copies of some of my pages and reinserting them in Dr. Epstein's original copy, and we have taken out of that volume the two new versions of the pages that appear in Epstein 4.

And we have marked as Epstein 21 the new two-page document entitled State of the Art on VC/PVC Toxicology Prior to 1974, and we have marked

as Epstein 22 the now five-page document entitled Illustrative Literature on the Toxic and Carcinogenic Effects of VC/PVC in the Respiratory Tract of Exposed Animals (sic).

MR. LEVINSON: Let the record reflect that all the documents that have been offered for identification at this particular deposition of Dr. Epstein both today and on the prior day shall be considered for all purposes as amendments to my answers to interrogatories propounded by Mr. Hollingshead.

MR. HOLLINGSHEAD: I note Mr. Levinson's statement. I have some concern, however, that at the last deposition I was provided with new material. I am now being provided with new material today. I don't think it presents any insurmountable problem, but I don't wish to get into a prolonged discussion about it now.

MR. LEVINSON: If you want anything that you haven't been provided with, just let me know and I will try to get it for you.

THE WITNESS: I should merely comment that I have been working on these the last few days.

MR. LEVINSON: I don't follow what particular

items that you would like that we haven't provided you with.

MR. HOLLINGSHEAD: It's not a question. I have a letter from you, Alfred, of some time ago that says that Dr. Epstein would be relying upon his preliminary report and I should consider it to be final.

I then came to his deposition on December 1 and we both were provided with new material by Dr. Epstein which became a supplemental report --

MR. LEVINSON: Oh, I see.

MR. HOLLINGSHEAD: -- which I have now reviewed in preparation for today, and admittedly what he has given us today as anything supplemental is relatively minor but nonetheless it is new again --

MR. LEVINSON: I see.

MR. HOLLINGSHEAD: -- when I thought we were already done with --

MR. LEVINSON: I see your point. I'm sorry.

MR. HOLLINGSHEAD: In terms of discovery, et cetera, there's nothing outstanding that I'm aware of that you owe me.

MR. LEVINSON: Okay, but let me say this. I will cooperate with you entirely. If after today you want any additional information by written interrogatories, I'll be happy to give them to you.

MR. HOLLINGSHEAD: I understand that. I appreciate it. I think we can work through this.

Dr. Epstein, let's -- let me hold these for now.

At the last deposition, Dr. Epstein, you provided me with a letter from Mr. Levinson to you dated April 18, 1988, which we marked as Epstein 3, and I want to clarify something.

That letter contains some information with regard to Mr. Peterson's work history and exposure, but I recall that there was other information supplied to you at some point which I believe you said you either discarded or returned to Mr. Levinson, one or the other.

BY MR. HOLLINGSHEAD:

Q. Am I correct in that recollection?

A. I think you will recall that we discussed this in great detail and I said that the material I received from Mr. Levinson I incorporated in my notes on the occupational history and then

discarded them.

MR. HOLLINGSHEAD: Okay, and you discarded whatever you received from Mr. Levinson.

Therefore, Mr. Levinson, you and I when we go back to New Jersey I'd like to follow up on this to try to obtain from you a copy of whatever other information you sent to Dr. Epstein which provided information on Mr. Peterson's work history and exposure.

And in particular, Dr. Epstein, if I recall correctly there was information in asbestos exposure in that submission to you by Mr. Levinson; is that right?

A. Not in that submission. In a later submission.

MR. LEVINSON: Didn't I give you a set of the -- or was that included in Dr. Epstein's?

MR. HOLLINGSHEAD: It's in neither place. It's not in 4 and he doesn't have it.

MR. LEVINSON: I found it in the office, and the reason I didn't provide you with it was I just assumed it was in this document.

MR. HOLLINGSHEAD: No, what is in that document is his handwritten summary in effect --

MR. LEVINSON: I'll be happy to give it to you. I've got it right in my file.

MR. HOLLINGSHEAD: Dr. Epstein, if you would turn to Epstein 4 and in particular the summary sheet that you labeled O.H., which I understand stands for occupational history, I've had the opportunity now to look over this material. I only have a couple of questions with regard to this document.

THE WITNESS: Sure.

BY MR. HOLLINGSHEAD:

Q. When Mr. Peterson was at Leam (phonetic) Manufacturing Company from 1962 to 1967, you listed that he had no asbestos or --

A. Silica.

Q. Thank you -- silica exposure and then the next item is no, I believe --

A. No maintenance or cleaning kiln.

Q. No maintenance or cleaning kiln, k-i-l-n, and that's underlined.

Is there a reason why you made a note of that particular lack of activity on Mr. Peterson's part?

A. Sure, because had he done any maintenance

or cleaning of the kiln, there would have been a possibility of asbestos or silica exposure.

Q. You were looking for that as part of his work history?

A. Certainly.

Q. What would the concern be for either asbestos or silica exposure if you had found it when he was at Leam Manufacturing Company?

A. I would have noted it accordingly and I would taken it into account in the assessment of his subsequent medical events.

Q. Is there literature, scientific literature or medical literature, to your knowledge relating silica exposure to laryngeal cancer?

A. No.

Q. But there is with asbestos if I recall your testimony correctly?

A. Yes.

Q. Have you been able to determine from any source as to not exactly but approximately how much exposure Mr. Peterson had to asbestos while he worked at the ATC plant?

A. I described it to you last time in qualitative terms. That's the sum total of the

information that I have.

Q. Turning now to the document that you provided to me at the last deposition which, if you go along in Exhibit Epstein 4, this is a document entitled Factors Incriminating VC/PVC as the Primary Cause of Peterson's Laryngeal Cancer.

What caused you to write this particular page which came about after the submission of your preliminary report in this case?

A. Well, as I mentioned the preliminary report was a hurried response to a rapid deadline imposed on me by Mr. Levinson and I continued work subsequent to that, and the object of this was to summarize the data with particular reference to VC/PVC, which I felt to be the major factor.

Q. What caused you to conclude that the exposure to VC/PVC was the major or as you say on this page the primary causal factor of Mr. Peterson's laryngeal cancer?

A. For the seven reasons so stated on this page.

Q. Without getting into that at the moment, it is true, is it not, that Mr. Peterson had an exposure to asbestos in at least the first several

years of his employment at ATC?

A. That's not what I said last time. What I said last time was the exposure was primarily in the first six months. Subsequent to the first six months his exposure was extremely minimal; and changing insulation rope that once or so a year on the doors of the asbestos boiler and, in fact, even in the first six months the repairing of the brake lining was not a frequent job and as I said only in the first six months.

Q. But it is true that there is literature support for the proposition that exposure to asbestos can result in laryngeal cancer, is there not?

A. Yes, correct.

Q. Is it also true that other than the Tabershaw study that you have indicated that you talked about the last time, there's no other literature support for the proposition that laryngeal cancer has been related to exposure to PVC?

A. Well, first of all it's not the Tabershaw. It's three documents. There's Tabershaw, there's Tabershaw and Gaffey, and --

unpublished reports, and there's a Tabershaw and Cooper published paper.

Q. Including all of the Tabershaw papers, that's the only support that you have put forth for -- literature support for the proposition that there has been a finding of a connection between laryngeal cancer, that specific cancer, and exposure to PVC?

A. No, not at all. There is a wide range of reports on respiratory tract cancers --

Q. My question was the specific cancer of laryngeal cancer.

A. Well, you interrupted me.

There's a substantial number of reports on respiratory system or respiratory tract cancers which will include laryngeal cancers of which there's no breakdown, so we don't know how many of those are laryngeal and how many of these are lung cancers.

Q. But the only study that in fact -- the only studies that in fact talk in specifics about laryngeal cancer would be the Tabershaw studies as you have indicated?

A. With that one qualification I have just

so indicated and with also the qualification there was a coworker of Mr. Peterson, maybe Mr. Wilkinson, who also developed laryngeal cancer.

MR. HOLLINGSHEAD: Some of this I appreciate we may have gone over to some extent the last time, but because the interim provided me with the time to read what was new to me the last time I want to go back over a couple of items.

In your paragraph 2 of this same document which you produced at the last deposition, Factors Incriminating VC/PVC, et cetera, you talk about Mr. Peterson having been continually exposed to VC from several sources. One of them is high concentrations from degassing of PVC pellets and dust in hoppers, cars, bags, and storage areas.

BY MR. HOLLINGSHEAD:

Q. Were you ever able to determine the actual concentrations at which he was exposed?

A. Well, no. In spite of Union Carbide's recommendation in '74 that the atmosphere should have been monitored to detect levels of VC and prevent injury to workers, Union Carbide failed to do any monitoring whatsoever prior to '74. We have some post '74 monitoring data which I discussed

with you last time.

Q. Do you have information that was provided to you from any source that would indicate that Mr. Peterson was in fact working in the hoppers or cars as you state in this paragraph?

A. No, other than the fact that he was a maintenance worker and therefore could have been exposed in any area he went into.

Q. All right, but on a regular occurring basis it's your understanding that his exposure to PVC occurred largely in the bagging area; is that correct?

A. Correct.

Q. Now is the bagging area different --

A. Excuse me. That's not the case. In the bagging area but also even after the bagging area from degassing of dust on his clothes --

Q. No, I asked you where he worked.

A. Oh, I see. Okay.

Q. Do you have information that he worked in the storage area of the ATC facility?

A. You have already asked me about the hoppers, cars, bags, and storage areas. I have responded by saying that as a maintenance worker he

would periodically be -- go into all areas in the plant requiring maintenance work.

Q. I don't want to quibble with you but my earlier question dealt only with hoppers and cars.

A. Fine, okay.

Q. You mentioned also in the same paragraph that there were high concentrations from thermal degradation of PVC pellets and dust during heat sealing and welding. I believe we went over this last time in a more general context, but since this document is new again I just want to come back to that.

The thermal degradation of PVC results in what type of fumes?

A. It depends entirely what you're dealing with. If you're dealing with a PVC film, the major degradation products would be HCl and a variety of other materials ranging from benzene, carbon monoxide, phosgene, et cetera.

Q. HCl is --

A. Hydrochloric acid or hydrogen chloride. However, when you're dealing with pellets and dust, particularly with the pre '75 pellets and dust, the major source of -- a very major source of exposure

would be vinyl chloride.

Q. To what percentage would that be a major source?

A. It's very difficult to say except I indicated that this will depend on a wide range of factors which were discussed with you in extenso by Dr. Davidson.

And in the State of the Art on Residual VC in PVC, the actual residual VC levels in the PVC would be an important factor.. The thickness of the originating material, the PVC; the size of it, the presence of catalyst, a whole range of physico-chemical factors would also influence the release of VC from PVC.

One can say that minimally the VC levels would be the levels which you find from degassing absent thermal degradation, but with thermal degradation there would be virtually an instantaneous release of these very high levels.

Q. When you talk about, as you just have -- when you talk about the major components of thermal degradation of PVC pellets resulting in an emission of VC fumes, on what are you relying for that statement?

A. Well, there's a -- first of all you'll find a note from Boettner in the State of the Art on Residual VC on the emissions of residual VC from PVC and that these are maximal at temperatures of 280 to 350.

There's a variety of papers. Simmonds et al., '49, talks about as early as 1949 Union Carbide knew that residual VC was released from VC (sic) during processing, calendering, moldering, and extrusion at temperatures from 100 to 200. So there's a big literature prior to '75 on release of VC from PVC by heat treatment. This was discussed among other things by Dr. Davidson.

And as far as the actual levels are concerned, we don't -- I can't provide you with specific levels at ATC because of Union Carbide's failure monitor contrary to its own recommendations in 1974, and I quote: "Resin users must monitor the work space air VC concentrations so that they could take appropriate action to protect their employees".

MR. HOLLINGSHEAD: I move to strike every part of that answer. That was not responsive to my question.

My question deals with the source of your information that on thermal degradation of PVC the major component that is given off is VCM. I believe you have referred to various references that are in your -- that are contained within Epstein 4 on the page entitled State of the Art on Residual VC in PVC Resin to 1975.

THE WITNESS: 1975, yes.

MR. HOLLINGSHEAD: Now --

THE WITNESS: And the additional point I made was that Dr. Davidson discussed this in detail in his deposition and also in his document.

MR. HOLLINGSHEAD: I agree that he did.

BY MR. HOLLINGSHEAD:

Q. Is it your testimony that upon thermal degradation of PVC pellets as they were at the ATC plant, the major ingredient that is given off is not hydrogen chloride?

A. I didn't say that. I said a major ingredient.

Q. I'm asking you what size is the percentage if you know of the ingredient of VCM that is given off upon thermal degradation of these PVC pellets?

A. I suggest you refer that question to Dr. Davidson. That's more his area of expertise. I'm merely aware that high levels are given off upon thermal degradation. The specifics of this you should discuss with an industrial hygienist.

Q. You would rely upon Dr. Davidson's testimony in that regard?

A. Among other things. Among that and also the literature.

Q. But if Dr. Davidson were to give a percentage of the amount of hydrogen chloride as compared to VC that is given off upon thermal degradation, you would accept that as authoritative, wouldn't you?

A. This is a straw man. You asked me about major emissions. I said a major emission is VC. I'm certainly not precluding hydrochloric acid. I would think it's very likely that hydrochloric acid is also a major component.

We do know that after the calendering, after the welding, Peterson had to leave the room because of the choking and it's clear that hydrochloric acid was given off. I don't think there's any question that hydrochloric acid would

be given off.

You clearly have misunderstood the point that I made: That when we're dealing with PVC film, VC emission is not a major component. HCl is the predominant major component when it comes to PVC pellets. In addition to HCl and all the other factors which are given off from PVC film, there will be very high emission levels of vinyl chloride.

Q. Why is there a difference between PVC film and PVC pellets to your understanding?

A. Well, there's an overwhelming difference for very obvious reasons. There's high levels of residual VC in PVC resins and dust particularly those prior to 1975.

And there's very, very low minimal levels in VC film -- in PVC film because of the PVC -- the VC will constantly evaporate from the film and the film is so very, very thin that the measured levels of VC are minuscular.

Q. Is this also true with regard to thermal degradation of PVC upon welding, which is also contained within your paragraph?

A. Correct.

Q. So your testimony with regard to what is given off and to what extent it's given off remains the same whether it's from the heat-sealing process or the welding process?

A. That's what the report states.

Q. That's all I'm trying to find out.

In Paragraph 4 on that same page where you talk about VC being a multipotent carcinogen in roads and exposed workers, et cetera, is this a summary of what is contained in your table that appears several pages later or tables with regard to the illustrative literature on the effects of VC/PVC in both animals and workers?

A. Sure --

(Discussion off the record.)

THE WITNESS: The answer is sure with respect to the respiratory tract tumors. It's no with respect to the multipotent carcinogenicity.

The tables that follow deal specifically with respiratory tract effects. The multipotent carcinogenicity is referred to incidentally, but the tables don't deal with the multipotency per se as opposed to respiratory tract per se.

BY MR. HOLLINGSHEAD:

Q. What studies do you rely upon for the multipotency effect that's cited in paragraph 4 of this page?

A. These are legion. They're not dealt with in the tables that follow but you'll find them and with references -- some early references under the report on Harry Wilkinson and the attachments, the appendix attachments.

Q. So they are contained within the document we have marked as Epstein 4?

A. Yes, and also in the two tables there's periodic incidental reference to multipotency.

Q. In paragraph 5 of this -- on this page you talked about adenocarcinomas and large cell and differentiated carcinomas in exposed workers.

Just so I understand how this relates to the rest of the document, what studies or what particular exposed workers are you talking about in that paragraph 5?

A. In workers exposed to PVC dust.

Q. And which studies are you referring to?

A. There's about four or five studies in the table.

Q. Contained in the table, right?

A. Sure.

Q. Then you also refer to laryngeal cancers in VC/PVC-exposed workers, and I'm assuming that in general, without regard to the ATC bagger, in general you're referring to those who were mentioned or studied in the Tabershaw studies?

A. Correct.

Q. And then the ATC bagger that is mentioned in this paragraph 5 refers to Mr. Wilkinson; is that right?

A. Yes.

Q. Now Mr. Wilkinson was both a bagger and a bag room utility worker, so that reference is just to him; is that --

A. Correct.

Q. With regard to the chart that is entitled Illustrative Literature on the Toxic and Carcinogenic Effects of VC/PVC in the Respiratory Tract of Experimental Animals, first let me ask whether or not you've made any changes in this chart. I understand the answer to that should be no, but --

A. I don't think I have, no.

Q. Do you want to check it with my copy?

A. Might as well take a quick look at it. I don't believe I have.

Lucky we did.

Q. Is there a change?

A. Yes.

MR. LEVINSON: What is this the doctor is reviewing?

MR. HOLLINGSHEAD: He's looking at my copy of what is contained in the exhibit we marked as Epstein 4, and I have asked if he would compare it with the current copy of Epstein 4 just to be sure he did not change that, and I believe he perhaps has found that he did.

THE WITNESS: Yes, there is one addition: Cornish, C-o-r-n-i-s-h, and Abar, A-b-a-r, 1969, dealing with rats' inhalation of fumes from heated PVC producing interstitial edema and focal hemorrhages in the lungs.

MR. HOLLINGSHEAD: Why don't we pull that out and replace it with a copy and we'll mark this as a new exhibit.

I'm going to ask the reporter to mark this revised table on experimental animals as the

next exhibit, and we'll make a copy of the old page and place it back into the book.

(Whereupon, Deposition Exhibit No. 23, Witness Epstein, was marked for identification.)

BY MR. HOLLINGSHEAD:

Q. Adenocarcinomas occur in the lung, don't they?

A. Yes. Well, they occur many places.

Q. On this chart when it refers to an adenocarcinoma with the designation of AC, is that supposed to indicate any particular location for that cancer?

A. Yes, the heading for that as you see is pulmonary effects. That's effects in the lung.

Q. What is the disease of fibrosis very briefly if you will?

A. It's basically just scarring.

Q. And the disease of granuloma?

A. It's chronic inflammatory reaction generally characterized by formation of nodules and multinucleate giant cells associated with fibrosis.

Q. And what is adenoma?

A. Adenoma is a premalignant -- or it's a tumor of the lung which hasn't progressed to malignancy yet.

Q. Can you describe pneumoconiosis for me?

A. Yes, pneumoconiosis is a chronic inflammatory condition of the lung generally associated with fibrosis and granular nodulation and granuloma formation.

Q. Pneumoconiosis is normally associated with dust in some form, isn't it?

A. Correct, yes.

Q. Can any form of dust cause -- potentially cause pneumoconiosis?

A. That's my understanding.

Q. Would something even not chemically related, for instance, something such as road dust or dirt cause pneumoconiosis or could it?

A. At high concentrations it could, but generally there has to be a -- there's generally an inflammatory role, element to pneumoconiosis. But to all intents and purposes dusts can also cause pneumoconiosis at high concentrations.

Q. With regard to the reference to Frongia in 1974, in the comments section there's a comment

that says, quote, "Exposed with workers bagging PVC (Wagoner, '77)."

Can you explain that comment?

A. Certainly. This study, the Frongia et al. study, took rats and guinea pigs and exposed them in the area where workers were bagging PVC. So these animals like the workers both developed fibrosis nodulation granulomas.

Q. Were any -- strike that.

Obviously some of these tests were inhalation studies, correct?

A. The overwhelming majority. There were only two that were not in that table.

Q. Which two were not and what type of exposure were they?

A. The Agarwal, A-g-a-r-w-a-l, et al., '78. This was intratracheal injection. And the Feron et al., '81, was either gavage -- that means putting a tube down into the stomach -- or incorporation of the PVC in the diet.

Q. In the chart on the illustrative literature with regard to exposed workers, on the Tabershaw studies on the first page there is a reference in the column entitled Respiratory Tract

Effects to a #3354.

Could you just explain that for me?

A. That's the code number in the Tabershaw paper.

Q. For the individual who was found to have an extrinsic laryngeal cancer?

A. Correct.

Q. And on the Comments side the note at the end of that lists "respiratory system cancers, ICD equals 160 - 164."

What does that mean?

A. That's an international code for -- tumors internationally are coded with a coding number. That's -- 160 to 164 is respiratory tract or respiratory system.

Q. Following that same answer, laryngeal cancer must be Item 161 in the code; is that --

A. Correct, yes.

Q. Would you remind me again, please, as to what workers were exposed in the Tabershaw studies? What was their occupation?

A. These were basically VC workers to the best of my recollection.

Q. At the manufacturing end as compared to

the bagging or storage end one might say do you recall?

A. To be quite frank this is a whole series of 19 industries -- based on 19 industries and I believe they were -- they included PVC. It was a VC/PVC set of studies.

Q. Before we leave this particular document, which is the illustrative literature regarding exposed workers, let me ask you about the Doll study which is now one of the items on this five-page document.

I take it that after we had our first day of deposition you went and located the Doll study because I believe you were not familiar with it at that time.

A. Correct.

Q. What is it that you found with regard to the Doll study?

A. Well, it is summarized in my table if you'd like to give me a copy of my table.

Doll reviewed the literature and among other things confirmed the fact that there was an excess of respiratory tract cancers in the exposed workers.

I should also point out that the Doll -- with regard to respiratory tract systems, there was a wide range of omissions in the references, critical omissions in the references cited by Doll. He failed to refer to the Waxweiler '76 study, the Wagoner et al. '80 study, the Waxweiler et al. '81, the Wagoner '83, and the Infante '81 studies.

As far as respiratory tract cancer, I would categorize it as once over lightly with omission of the critical documentation. In spite of that, he recognized higher risks for lung cancer in heavily exposed workers.

Q. What conclusions or findings did he come to other than that?

A. Well, he -- it was a review of the whole area of the epidemiology of VC/PVC and I quote here:

> "No positive evidence of a hazard of nonmalignant disease or any type of cancer other than angiosarcoma of the liver has been found except possibly for a small hazard of lung cancer when exposure was heavy.
>
> "There are, however, consistently

higher risks in the subgroups of men in the U.S. and U.K. series in which occupational hazards would be more likely. That is, men employed for more than ten years exposed to higher than average concentrations or observed more than twenty years after first exposures.

"The mortality from lung cancer has been rising throughout the period of observation."

I should also note that Doll's conclusions on the lack of evidence of other diseases besides angiosarcoma of the liver and lung cancer and nonmalignant disease are in striking contrast to a wide range of governmental studies in the United States, in striking contrast to the conclusions of innumerable international experts representing academia, industry, labor, and government and a branch of the WHO dealing with cancer; namely, the International Agency for Research on Cancer.

So Doll is way out on left base in this area, and some of the additional references that I cite in the amended table -- for instance, the IARC

'87 and the IARC '79 -- indicates how isolated Doll is.

I also note that the Doll paper is also characterized by an absence of reference to any source of research support. This is particularly interesting as Green College where Doll works has been partially financed by industry including Monsanto and this is to me of particular interest. It is customary when you write scientific articles based on research of one kind or another to cite source of research support.

So not only is he way out on left base -- field in reference to some of the references I cited, not only does he fail to cite critical references such as Waxweiler et al., he also recognizes the lung cancer but also he doesn't reflect his source of research support.

Q. He found -- if I recall the paper right, he found that there was a specific hazard of angiosarcoma of the liver to men -- for men occupationally exposed to vinyl chloride, correct, and he also --

A. That was found twenty, thirty years before --

Q. Well, he's not finding anything original. He's doing a review of the literature, isn't he?

A. He's doing a partial -- a highly selective review of the literature.

Q. By that I only wanted to indicate for the record that he was not conducting original research as I recall the paper.

A. Correct.

Q. Is there a listing in the Doll report as to -- or the Doll paper as to other occupational hazards that men exposed to vinyl chloride may have had?

A. He says there's no positive evidence of nonmalignant disease.

Q. And you take issue with that statement based upon the studies that you have supplied to me in the course of two days of your deposition?

A. It's a statement that is in total contrast to a vast body of documentation.

Q. Can I see the table again.

A. Sure.

MR. HOLLINGSHEAD: Now the other new papers that were included on here -- if we can just take a moment to find them. Off the record.

(Discussion off the record.)

MR. HOLLINGSHEAD: For the record in looking at what we have marked this morning, there's some confusion. Apparently I have marked the same exhibit twice. Both Epstein 18 and Epstein 22 are the same with the exception that Epstein 18 has the check marks that Dr. Epstein put on the new references for this table, which is what I am turning my attention to now.

THE WITNESS: Turn to page 2 --

MR. HOLLINGSHEAD: This is the first new reference and this is --

THE WITNESS: Byren et al., '76. That's merely an excess of lung caner.

BY MR. HOLLINGSHEAD:

Q. Were you not aware of Byren prior to the deposition two weeks ago?

A. Yes, sure, I was aware of that.

Q. Is there a reason why it was not cited at that time?

A. No particular reason. The table wasn't claimed to be totally comprehensive.

Q. What can you tell me about the Byren study?

A. It was nearly one of many studies confirming the excess of lung cancer in VC/PVC workers.

Q. Do you recall -- this document appears to indicate it was an SMR study?

A. Yes.

Q. Do you recall what the expected proportion would have been as compared to the actual proportion --

A. You mean the observed to the expected?

Q. Yes.

A. No, I don't recall.

Q. Was it significant do you know, statistically significant?

A. I'm not sure. I doubt it. It's biologically significant but probably not statistically significant.

Q. What's the next one?

A. Bufler et al., '79. This was a statistically significant excess of respiratory tract cancers in VC workers and particularly in more heavily exposed workers, and this was a particularly important paper because the excess of respiratory tract cancers persisted after

adjustment for smokers.

Q. That means that once somebody balanced the study to take into account smokers, there was still a statistically significant excess of lung cancers -- I'm sorry, respiratory cancers?

A. Yes, correct.

The next is International Agency for Research on Cancer, '79. This is a review of the literature prior to 1978 on VC/PVC in animals and humans. The report was prepared by a working group of twenty international experts representing academia, industry, labor, and government together with the Secretariat of the IARC, which as I mentioned before is a branch of the World Health Organization dealing specifically with cancer.

First of all they stated vinyl chloride is a human carcinogen. Its target organs are the liver, brain, lungs, and haemo-lymphopoietic system. This incidentally is in striking contrast to the contrary and isolated statements of Doll.

IARC goes on to say there's no evidence there's an exposure level below which there's no increased risk of cancer in humans. IARC also goes on to -- there's a statement that IARC reviewed the

literature demonstrating fibrotic lung changes, pneumoconiosis, and reduced lung function in workers exposed to PVC dust.

Q. Dr. Epstein, when you see the phrase target organs or you use it yourself, how do you employ that phrase?

A. When you talk about multipotency, that means that a carcinogen can induce tumors in a variety of organs. Then generally one lists what the predominant organs are and you refer to them as target organs.

Q. And the IARC report in '79 lists liver, brain, lung, and haemo-lymphopoietic?

A. Yes.

Q. Did the report conclude whether or not the larynx was a target organ for vinyl chloride?

A. There was no discussion of larynx, no reference to larynx.

Q. What is the next new one that you have listed?

A. In this connection you may recall that the only publication by Tabershaw would have been the Tabershaw and Gaffey paper on general occupational medicine which to the best of my

recollection only referred to one laryngeal cancer. The other industry studies were never published and IARC only deals with published studies. The next it Infante '81 --

Q. Could we hold that for a second?

A. Sure.

Q. If an agency such as IARC was aware of the existence of an unpublished study, would they include it in your experience?

A. In general the answer is no, they would not.

Q. Is there a reason for that?

A. Because they only cite published data from the literature which are available to scientists, the scientific community.

Q. Published studies would go through some form of peer review, would they not?

A. Not necessarily.

Q. No?

A. Some do, some don't. Depends what journal it is. All you can say is unpublished studies certainly don't have any peer review.

Q. And therefore are they considered less authoritative by members of the medical or

scientific community?

A. Not necessarily. They can vary in quality. Some may be highly reliable. Others may be highly manipulated.

Q. What is the next new study?

A. Infante '81. This is a review and seven of eight studies which he reviewed showed -- quote, "showed excess risks of lung cancers ranging from 7 to 200 percent." Data from two of the studies suggested a qualitative dose-response relationship between exposure and lung cancer risk.

In the one remaining study, which was negative, the short duration of followup limited the validity of any inferences that could be developed.

The epidemiological evidence -- Infante summarizes by saying the epidemiological evidence demonstrates that the carcinogenic effects of VC in humans extend beyond the liver. The brain and the lungs should also be considered target organs.

And workers exposed to VC demonstrated a significant excess of mortality for lung cancer, and in fact he also stressed the sensitivity of the epidemiological studies reflecting small cohorts

and inappropriate methodologies, et cetera.

Q. With regard to a study such as this one, which is a review I will note, or with regard to a paper such as this one that talks about an excess of lung cancers, does that have particular significance in your mind with regard to the Peterson litigation and the fact that Mr. Peterson contracted laryngeal cancer?

A. Well, it only has it insofar as that respiratory tract or respiratory system cancers include laryngeal. If a study specifically refers to lung cancer as opposed to respiratory tract cancers, its relevance would only be an expression of the fact that the respiratory tract per se including the lung is sensitive to these carcinogenic effects.

The other point of course that should be made is that lung cancer is very, very much more common than laryngeal cancers and one would not expect -- and if indeed these studies are insensitive to even common cancers such as lung cancer, the sensitivity to relatively rare cancers would be of a very low order.

Q. Laryngeal cancer is one of those that are

relatively rare?

A. Yes.

Q. I think you gave a figure of 8.5 for hundred thousand?

A. Superb.

Q. What's the incidence for lung cancer?

A. More than ten times that.

Q. The next new item on the table?

A. The next new one is IARC 1987 and this updated the IARC '79.

And this is a report reported by a working group of 35 international experts including the Secretariat of IARC and basically stresses again the multipotency of vinyl chloride; stresses again, and several studies confirm, that exposure causes lung tumors. Exposure to PVC dust was associated with an increased incidence of lung tumors too.

Q. In your --

A. Excuse me. Something else was handwritten on this one which is not on that --

Q. Tell me what it is.

A. Under Doll -- you have it. I beg your pardon.

Q. So the two exhibits seem to be exactly the same?

A. Yes.

Q. In the exhibit we marked as Epstein 4 you have a number of literature references actually in their entirety in the back of the volume.

Are these new items that we've been talking about contained in the report as it exists now?

A. The references in the back relate to larynx cancer, not necessarily to these. You may recall last time I brought with me batches of cards on my VC/PVC references.

MR. HOLLINGSHEAD: If you would, staying with Epstein Exhibit 4 --

MR. HOLLINGSHEAD: Do you mind if I break for a second?

(Discussion off the record.)

BY MR. HOLLINGSHEAD:

Q. Turning to that portion of Epstein 4 in which is contained Dr. Loomis's report of November 2, 1989, could you turn to that.

A. Yes.

Q. You have a certain amount of underlining

and a couple of what I would consider to be exclamation points that you made in the margin.

Could we turn to the underlining and the exclamation points.

A. Yes.

Q. Did the exclamation points mean anything other than the fact that it's something to be emphasized? For instance, do you disagree with the statement that's made at that point in the report?

A. It could range from astonishment to incredulity.

Q. Why don't we turn to page 2 where a couple of them appear. There are three sections that are underlined on page 2. The first seems to be underlining and a notation on your part.

Is there anything of significance to that underlining?

A. No, that was simply confirmation of my under -- that underlining that -- it said that prior to his employment at ATC he had no specific occupational exposure to any industrial chemical. This is consistent with my understanding and also with Davidson's understanding.

Q. The next underlining talks about him

working most of the time in the vinyl packaging room.

A. Again consistent with my understanding and Davidson's understanding.

Q. The next underlining is a section which contains the exclamation point, so tell me the significance of that.

A. I would characterize that as a rubbish -- statement as total scientific rubbish. He says PVC is toxicologically a very safe compound but by, according to literature, industrial exposure produces only minimal nonspecific primary effects.

Q. He then goes on to talk about unpolymerized vinyl chloride coming out of the -- from which the PVC -- sorry.

It then goes on to talk about unpolymerized vinyl chloride.

Does that straighten out any of the concerns you have with regard to the earlier statement on PVC?

A. No. The statement that PVC is toxicologically very safe and produces only minimal nonspecific pulmonary effects I characterized as scientific rubbish. Whatever he may say afterwards

is another matter, but that statement is rubbish.

Q. The next page contains an underlined section which also has exclamation points. Can you talk about that for a minute?

A. That again is scientific nonsense. There's general knowledge about the possible tumorigenic properties of VC as well as the federal regulatory act that didn't come about until almost seven years after Peterson was initially employed at ATC in 1974. There's substantial information on this going back to minimally 1970 and possibly also as early as 1965.

Q. And support for your statement can be found throughout your report and the various studies you have referred me to?

A. I would think these are discussed in various places, yes.

Q. I'm only seeking now to go back over things we've done before.

In the next section you've made some handwritten notes in the left-hand margin. Let's go through that for a moment.

A. Yes. Davidson estimates that up to 10 ppm vinyl chloride before 1975.

Q. Was there a statement by Dr. Loomis that was inconsistent with that?

A. I didn't have an exclamation mark there.

Q. I'm just trying to figure out your code. What's the reason for the comment there? Just to remind yourself as to what Dr. Davidson had said?

A. Because the Loomis document is misleading. The -- first off, when he refers to samples that were obtained showed less than .1 to maximum .45, this is untrue. Three to 4 ppm were demonstrated in the breathing zone of the bag room.

And even in 1975 his statement that weighted average concentrations were .68 and .76 is untrue. Levels over 1 ppm were found. There's also no recognition of the high potency of vinyl chloride including carcinogenicity at the lowest level tested; namely, 1 ppm.

Q. Was that the Maltoni?

A. Correct. There was also one other study, an ovarian study, which I refer to in mice and rats with carcinogenic effects of 1 ppm and there's no consideration of PVC dust. There's a substantial literature on the carcinogenicity of PVC dust such

as the Wagoner, Waxweiler, IARC studies. We are on page 4 now.

Q. On page 4 there's one underlined section with a couple of --

A. Yes. Laryngeal cancer has never been documented to have been causally related to exposure to VC or PVC is nonsense. He hasn't obviously read the industry studies.

Q. The only one that specifically links laryngeal cancer are the Tabershaw studies, correct?

A. Tabershaw studies, correct. Lastly it's only in Velez's report that exposure to asbestos is suggested. This makes it clear that he has no knowledge at all on pathology and radiology because just by looking at the X-ray report on Mr. Peterson it's clear that he had asbestosis.

Q. Do you know that he had it?

A. I have no idea. I presume that you and Union Carbide furnished him with all the necessary documentation and medical reports.

Q. Wouldn't you also have to assume that I had it as well?

A. You had the medical reports.

Q. Yes, I had the medical reports.

A. And if you had the medical reports you had the X-ray report. The medical reports clearly state that there was calcification. There was -- you don't have to look at the X ray. The medical reports state clearly there's calcification.

Page 5: "I am unable to find a single report in the literature on laryngeal cancer." We discussed that already. "In humans VC is believed to be associated with accounts only of the rare tumor angiosarcoma of the liver." I characterize that again as scientific rubbish and hopeless ignorance of the field in which presumably he's acting as an expert.

Q. Going back to the first statement, the only published article on laryngeal cancer would be the one Tabershaw study. The other two are unpublished, correct?

A. That's right, but he doesn't say -- he doesn't talk about the published literature. He says "in the literature," which includes unpublished data.

Q. How does one get unpublished data?

A. By acting as an expert to industry, as a

consultant to industry, presumably industry files will become available to you. Furthermore, these unpublished data have been widely circulated.

The unpublished reports of Tabershaw have been widely circulated and have been referenced in various places in the published literature particularly by government in government reports.

Q. You have a chart in here on meat wrappers asthma as I recall?

A. A couple of tables, yes.

Q. In particular there is one entitled Chronic Meat Wrappers Asthma, Illustrative Quotations on Irreversibility and another on Meat Wrappers Asthma, Illustrative Statements on Prevention and then there's another one looks like a continuation of the statement on prevention.

Would you just indicate for me, Dr. Epstein, the significance of the meat wrappers asthma literature for the laryngeal cancer case that Mr. Peterson presents.

A. I would say none. The only reason I put it in was because you expressed an interest in any cases before which I had been involved on PVC film. I was responsive to your interests.

Q. Oh, I see.

Could you turn to the section on the literature, the Austin 1982 document.

A. Sure.

Q. What is this compilation? How would one refer to this? Is this a review of laryngeal cancer?

A. Well, this is in the book by -- multi-authored book edited by Schottenfeld and Fraumeni, S-c-h-o-t-t-e-n-f-e-l-d, and Fraumeni, F-r-a-u-m-e-n-i. There's a chapter on laryngeal cancer and this chapter was authored by Austin and this is just extracts of material from that chapter.

Q. Do you consider it to be authoritative?

A. I would say yes on the whole. It's a once over lightly but it's a pretty good basic source.

Q. Was this published in '82?

A. Yes, it's a textbook -- it's a reference book rather than a textbook.

Q. What was the year of the Tabershaw study that indicated an existence of a laryngeal cancer and a relationship with PVC?

A. About '74, '75. In the published one I believe there was only reference to one cancer.

Q. To the laryngeal?

A. Yeah. The other two unpublished studies contained reference to the two others.

Q. Unless I've missed it, when I read the Austin paper there was no mention of the Tabershaw study.

A. You're right. As I stated before in that one published paper there was only one laryngeal cancer. You wouldn't expect it to be referred to.

Q. When were the unpublished studies of Tabershaw?

A. '74 and '75.

Q. And presumably Austin would have had access to the unpublished literature?

A. I doubt it.

Q. Why is that?

A. Because he doesn't work specifically in the VC/PVC field. Had he worked specifically in the VC/PVC field, he would have known about it.

Q. Had he known about it would you have expected him to include Tabershaw in this review?

A. He might well have done so.

Q. Why would he not have -- in your opinion why would he not have included the reference to the one Tabershaw study that was published?

Was it not statistically significant because it was only the one finding?

A. First of all, it's obviously not statistically significant. The question is whether it's biologically significant and one wouldn't in fact ascribe biological significance to one cancer.

But when you're dealing with three and four laryngeal cancers, which we are now, then it becomes more biologically significant and also taking into account the fact that in several of the VC/PVC studies the results are reported as respiratory system cancers or respiratory tract cancers, which would include laryngeal cancer. So this particular chapter wouldn't reflect the specific information on laryngeal cancer and VC.

Q. And that would account for the fact that the entire paper is devoid of any comment about PVC or VC, correct?

A. Correct.

Q. Austin lists asbestos as a risk factor at

on occupational factors the major risk factors as being tobacco, alcohol, asbestos, nickel, and mustard gas exposures. Would you --

A. Yes, I have underlined those for you.

Q. Thank you. It helped me to find them but I even read those sections that were not underlined.

A. Oh, bravo.

Q. Would you agree with his conclusion that those are the major risk factors?

A. They are major risk factors. He doesn't say "the" major risk factors. He said they are major risk factors.

Q. If there were other major risk factors would you have expected him to include it in his paper?

A. He does. He discusses them in increased mortality of organization, with air pollution, in a variety of occupations in which no particular breakdown of chemical exposures has been identified. Additionally even in meat cutters he notes an elevated PMR was found. I should have underlined that for you.

Q. I saw that too.

A. Sorry, I didn't underline it.

MR. HOLLINGSHEAD: I see you're also marking the exhibit. Let the record reflect that Dr. Epstein has underlined that portion of the Exhibit Epstein 4 under Occupational Factors talking about meat cutters. Off the record.

(Discussion off the record.)

MR. HOLLINGSHEAD: Dr. Epstein, let me ask you to look at your report in this case which was entitled Preliminary Report 9/13/89 and which we marked as Exhibit Epstein 2 on the last occasion we were together. Starting on page 7 there's a discussion with regard to polystyrene. I would ask you to turn to that.

BY MR. HOLLINGSHEAD:

Q. What form of polystyrene is it that was present at ATC if you know? By that I mean whether it pellet or powder.

A. To the best of my recollection they were pellets. Let me see if I have a notation on that.

I don't have a notation on that.

Q. For the record I think I can represent to you that they were pellets to the best of my knowledge. That's also your recollection?

A. That's my impression but I don't have a notation, however, on that.

Q. At the end of the first paragraph on polystyrene you talk about exposure of animals to PS, meaning polystyrene, dust induces pneumoconiosis.

Do you know if that was with -- strike that.

Do you know what study that was that demonstrated that?

A. To be frank I don't and I would think this is unfortunate that I didn't reference that.

Q. Would it be one of those references at the bottom of the page?

A. I would imagine so, yes.

Q. Are you aware whether or not there's any study that indicates that polystyrene pellets give off dust and thereby cause pneumoconiosis?

A. Well, if you have pellets you'll certainly get dust from the pellets.

Q. How do you get dust from the pellets?

A. From fragmentation. From handling of pellets you'll generate dust.

Q. So your view would be whether the study

is with regard to powder or pellets of polystyrene you would still get dust and therefore would likely get pneumoconiosis?

A. It depends.

Q. Depends on the exposure and the size of the dust fragments?

A. Yes, certainly.

Q. What micron size of dust would you need in order to induce pneumoconiosis?

A. Well, dust would have to be respirable and the particles of about 5 microns are 20 to 30 percent respirable, 10 microns is nonrespirable, and 2 microns are about 100 percent respirable. So in that range.

Q. Is there any study you are aware of that with regard to polystyrene that shows a direct causal relationship between exposure to polystyrene and laryngeal cancer?

A. No. There's a study on styrene which shows association with laryngeal cancer, Hobson and Jones.

Q. What was the exposure do you recall in Hobson and Jones?

A. To styrene.

Q. Were these animals?

A. No, that's an epidemiological study.

Q. What were the levels of exposure do you recall?

A. I don't recall.

Q. Do you recall who, in terms of the subject of the study, who was being studied?

A. Yes, workers, the exposed workers.

Q. Do you know what level they were being exposed?

A. You just asked me that. I said I don't know.

Q. Do you know in what end of the manufacturing --

A. I don't recall.

Q. -- business they were in?

A. The reference will give you all that information.

Q. Yes, thank you.

How does IARC list their -- come up with a list of carcinogens?

You've indicated here that you have -- at the bottom you say styrene is ranked as a group II-B carcinogen.

Is that a listing of the probable human carcinogens?

A. Well, it's a ranking of carcinogenicity in which human and animal data are taken into account.

Q. What are the rankings that IARC puts on carcinogenicity?

A. Group I: when you have clear-cut epidemiological data. II-A and II-B: II-A is where you have reasonable epidemiological data.

Q. In humans?

A. Epidemiological is humans. II-B is -- and you can have a II-A where you have excellent animal data. II-B is animal data without -- is very good animal data. III is animal data with no epidemiological data. That's basically -- and IV is basically noncarcinogenic or questionable validity of animal data.

Q. Group I again is known carcinogenicity?

A. One again is unequivocal epidemiological data in humans, and IARC frequently states that when you have valid animal data this creates a strong presumption or basis for presumption of human cancer risk.

Q. Do these rankings line up with the EPA rankings?

A. Put it the other way around. EPA rankings in general reflect the IARC although not necessarily, and sometimes EPA refers to the IARC rankings.

Q. Does styrene monomer pass from the pellet to the air and if so can you tell me how and under what conditions?

A. Sure. It's degassed in the same way as VC is degassed from PVC and, additionally, when you heat-treat polystyrene you will liberate styrene. This is discussed in the first paragraph of page 7.

Q. Is there a residual level of styrene contained within the pellet similar to PVC?

A. Yes, I refer you to sentence 2, paragraph 1, and sentence 3.

Q. Sentence 2 indicates that polystyrene contains up to 1 percent of the residual -- of the volatile residual styrene monomer.

Is it your understanding that that is the maximum that is contained within the pellet?

A. I haven't frankly looked at the early literature to find whether there were higher levels

but these are references in '79 and '83. Whether the levels at the time Peterson were (sic) exposed were higher, I don't know.

Also we do know that to have high concentrations of unreacted styrene released from fresh polystyrene, the high concentrations are released. That was known as early as 1968.

Q. Mr. Peterson was not exposed to relatively fresh polystyrene, was he, to your knowledge?

A. Polystyrene comes from UC.

Q. Right, but it was shipped from Texas, wasn't it? Isn't that part of your information?

A. Yes. So what?

Q. When you talk about fresh polystyrene --

A. As opposed to aged polystyrene. You and I are aged. When we were born we were fresh.

Q. When polystyrene is first manufactured it's fresh, correct?

A. Fresher than two weeks later. But in addition to that I should point out to you that when you degas a monomer from the polymer, the monomer particularly in closed conditions such as in a vat, et cetera, will adsorb itself on the

polymer.

So it really doesn't make much difference. You'll have the adsorbed monomer on the actual polymer as opposed to contained within the polymer as when you have it under closed stored conditions.

Q. So what you're saying is when it's degassed out of the pellet, it then clings to the outside of the pellet and that's what you referred to as adsorbed?

A. No, that's not what I said. I said when it is degassed under closed conditions such as in a car or a vat or a box. When you degas in the air you will have a progressive reduction in levels of the monomer in the polymer.

But when you degas in a closed system, essentially what's happening is that there's a migration from within the polymer to the closed dead air space and with the secondary adsorption on the surface of the polymer.

Q. With regard to the Hobson and Jones study which according to your paper demonstrated a major excess of laryngeal cancer, I take it by the reference to 3/0.5 that your expected rate of

cancer was .5 and the actual occurred cancer was at rate 3?

A. Yes, in other words a six-fold increase in workers exposed to polystyrene in the total cohort; and when it comes to the subcohort of men under the age of 45, the ratio then becomes twenty-fold excess of laryngeal cancers.

Q. Do you know the amount of styrene or -- well, the amount of polystyrene that Mr. Peterson was exposed to?

A. No.

Q. Do you know the levels of the exposure?

A. Union Carbide failed to monitor --

Q. The answer is no --

A. I haven't finished -- and Golub (phonetic) wasn't requested by ATC or Union Carbide to monitor. Therefore, we have no information.

Q. Then I take it the answer to my question is no?

A. Yes, the answer was no.

Q. Do you have information as to what percent of Mr. Peterson's time was spent with regard to either bagging polystyrene or in any other way being around polystyrene as compared to

the amount of time he spent with the PVC?

A. Well, he didn't spend time specifically with -- he worked in the bagging room.

If I may rephrase your question, it could be posed as what percentage of the total polymers entering into the bagging room were polystyrene as opposed to polyvinyl chloride. I don't know --

Q. I'll accept that question and you can answer that one.

A. The answer is I don't know, but it's a good question once rephrased.

Q. But the germ of it was good too.

A. Dr. Davidson may have information on that.

Q. I only wish to establish what information you have at the moment. I appreciate he may have other information.

With regard to that same form of question, do you have any understanding as to what percentage of Mr. Peterson's time was spent -- off the record.

(Discussion off the record.)

MR. HOLLINGSHEAD: Dr. Epstein, let me withdraw that and rephrase the question.

BY MR. HOLLINGSHEAD:

Q. Do you have any information that would tell you what percentage of time Mr. Peterson spent on bagging any of the other resins in the ATC plant as compared to PVC?

A. With due respect, as you know perfectly well Mr. Peterson never bagged or was employed as a bagger so I can't answer that.

Q. I will rephrase the question.

What percentage of time did he spend in the bagging room when isopropylidene resins were being bagged do you know?

A. I don't know. I don't know whether they were being bagged at the same time as PVC, whether they were being bagged in separate batches. I just don't know.

Q. You have acted on the assumption in this case that the majority of Mr. Peterson's time was spent in the presence of PVC, have you not?

A. That is an assumption but I regret to say that I haven't substantiated that assumption -- well, I know that the vast amounts of VC/PVC were bagged. I went into the amounts in terms of bags per day and pounds per day. I don't have the

equivalent information for polystyrene.

Q. Mr. Levinson's original letter to you in April of 1988 that we have marked as Epstein 3 only talks about PVC pellets if I read it correctly; am I right?

A. Correct.

Q. And that's one of the reasons why you've assumed throughout your efforts in this case that PVC was the main chemical to which Mr. Peterson was exposed; is that --

A. I believe this to be the case, but I will prior to trial attempt to find out the percentage of polystyrene and the other resins which were used and the amounts used. That's provided this information is available. If it isn't perhaps you will pass this on to Mr. Levinson.

Q. With regard to the isopropylidene bisphenol resins discussion which begins on page 8, you indicate that bisphenol is a potent primary irritant and sensitizer, and you go on to talk about other things from the Shell (phonetic) report, 1982.

Is there a particular ingredient in the isopropylidene bisphenol resins that makes it a

primary irritant and sensitizer?

A. Well, there's various components. Epichlorohydrin -- epichlorohydrin is certainly an irritant and a sensitizer. Over and above that there's a variety of other volatile components particularly phenolics and the glycidol ethers which are sensitizers too.

Q. I think I asked you the last time but let's put it in context for today's deposition.

You have referred to isopropylidene bisphenol resins and I think in particular to 4.4 isopropylidene --

A. I'm sorry. I beg your pardon. What was that?

Q. You have referred to isopropylidene bisphenol resins and particularly 4.4 isopropylidene diphenyl, have you not?

A. Yes.

Q. Do you understand those materials to be the same as bisphenol A resins?

A. Yes.

Q. And is it your understanding that epichlorohydrin is present in the bisphenol A that was in existence at the ATC plant?

A. That's my understanding.

Q. Is it the epichlorohydrin that in fact is the primary ingredient in making it a potent irritant and sensitizer?

A. Well, that's the result -- the literature on epiclorohydrin and also glycidol ethers as irritants and sensitizers particularly epichlorohydrin.

Q. Are you aware of any literature that shows a causal relationship between bisphenol A resins and laryngeal cancer?

A. I am unaware if there is any literature on this. I know there's literature on respiratory tract cancers including lung cancer and nasal cancer.

Q. And that's referred to in the references down at the bottom?

A. Yes.

Q. Are you aware of any literature showing a causal relationship between epichlorohydrin and laryngeal cancer?

A. No. As I said before, there's literature on respiratory tract cancers and the respiratory tract includes the larynx. There are references on

nasal cancer and lung cancer but I haven't seen any on laryngeal cancer.

Q. What are the conclusions in the Interline reports that are the first two references at the bottom of the page?

A. There are a couple of reports, two or three reports, by Interline and the very first report demonstrates an excess of lung cancer, respiratory tract cancer, and one of the subsequent reports limited the statistical validity of the earlier reports.

But again the interest of these agencies with regard to laryngeal cancer is any agent that is a respiratory tract irritant will certainly increase the susceptibility of the larynx to agents that can induce laryngeal cancer, and there's a possibility of synergistic and interactive effects.

When you're dealing with a whole complex of chemicals of this kind, the effects on any particular target organ can differ very substantially from the effects of exposure to a single chemical.

Therefore, the data on studies based on experimental or occupational studies based on a

single exposure would understate risks to particular target organs which would be seen -- which would develop when you expose workers to a wide range of these different carcinogens, toxic, and irritant agents.

Q. With regard to the welding emissions, which is a discussion commencing on page 9 of your report, what is your understanding as to how much exposure Mr. Peterson had to welding fumes or welding emissions while he was at ATC?

A. We know he welded. The amount of time he spent on welding on a percentage basis every day I don't know. The statements to the best of my recollection are during welding he would sometimes cough and choke and have to leave the room, but I can't tell you the percentage of time he spent on welding as opposed to other activities.

I'm not sure these data are available although you might check with Union Carbide or ATC on this to see whether they kept such records, which I doubt.

Q. What makes up welding fumes?

A. First paragraph will -- gives you a fairly comprehensive -- or attempts to give you a

fairly comprehensive statement of what can be generated.

Of course you have to include in this thermal degradation of any polymers which are on contaminated surfaces such as polyvinyl chloride or polystyrene which would result in release of the residual monomer or the adsorbed monomer such as vinyl chloride or styrene.

Q. Without regard to the thermal degradation of PVC or polystyrene -- and by that I'm asking you to assume that a particular welding episode or series of welding episodes took place where there was no thermal degradation because those materials were not present on the metal that was being welded -- is it your opinion that the welding fumes that are given off during the welding process contributed in some fashion to Mr. Peterson's laryngeal cancer?

A. I would say that it's likely they played a contributory role.

MR. HOLLINGSHEAD: Why don't we stop there.

(Whereupon, a luncheon recess was taken until 1:15 p.m. this day.)

A F T E R N O O N S E S S I O N

(Whereupon, the deposition in the above-entitled cause was resumed at 1:15 o'clock p.m. this day.)

MR. HOLLINGSHEAD: Dr. Epstein, directing your attention to page 1 of your report which has been marked as DC-2 as I recall, you talk on the first page particularly in numbered paragraph 3 that Mr. Peterson is at future risk of developing progressive chronic obstructive lung disease and attendant complications.

SAMUEL EPSTEIN, M.D.,

the witness on the stand at the time of recess, resumed the stand and testified further as follows:

EXAMINATION (Continued)

BY MR. HOLLINGSHEAD:

Q. Can I ask you what you mean by attendant complications?

A. Sure. First of all for progressive chronic obstructive lung disease there's a variety of complications that can occur from this. One is just death from progressive respiratory failure.

And when you have obstructive lung

disease particularly in association with restrictive disease you can get right heart failure; and because the -- from the right ventricle you have a pulmonary artery going to the lung, as you get increased resistance in the lung because of fibrosis you can get gradual -- what's called corpulmonale, c-o-r-p-u-l-m-o-n-a-l-e, with progressive right heart failure. Then of course increased incidence of respiratory infections, pneumonia, et cetera.

Q. Can you quantify for me the increased risk that Mr. Peterson does have to the development of progressive chronic obstructive lung disease and attendant complications?

A. I think that I should defer on this to Dr. Velez. This is more his background. I can only talk in general terms about this.

Q. Would you also defer on the first sentence of paragraph 4 which talks about Mr. Peterson being at future risk of developing other cancers from his past occupational exposures at ATC? Can you quantify that list?

A. As far as asbestos is concerned, I think Velez has already done this for asbestos. I think

he talks about a five-fold increase in risk of lung cancer. I can't quantify this in relation to the whole mix of chemical carcinogens. I would say he's at substantial excess risk for a variety of reasons.

First of all he has excess risk for respiratory tract cancers, particularly lung cancer now, for two reasons. He's been exposed to a wide range of pulmonary carcinogens, and these I've tabulated for you in a table in Appendix 2 which of them are carcinogenic to the larynx and which are carcinogenic to the lung.

Secondly we know that once you get a single primary in one site in the respiratory tract, your chances of getting another cancer in that same system are substantially increased and that the second cancer can occur at a long time subsequent to the diagnosis and treatment and cure of the first.

Then of course you have the total impossibility of developing any form of quantitative prediction because of the multiple interactions between these different carcinogens, and then you've got carcinogenic effects at other

sites.

So I would say that he is at substantially excess risk of future cancer with particular reference to lung cancer but also cancers at other sites.

Q. But when you use the phrase "substantially increased risk" or "substantially excess risk," you don't have a particular percentage in mind as to what that substantial risk -- increased risk is?

A. No, I can't quantitate it. But substantially increased risk is more than sufficient to justify the need for lifetime medical surveillance and also justifies, creates a valid basis of fear of the development of such risk and of such future cancers.

Q. In your opinion to a reasonable degree of medical probability can you say that Mr. Peterson will in fact contract some additional cancer in the future?

A. No, I can't say that. All I can say is he is at substantial excess risk both of which justify as I say two things. One, the need for medical surveillance and the fear of such risk of

such eventuality -- eventualities.

Q. When a certain -- when chemicals are put in combination, which is also referred to I suppose as synergism --

A. No, excuse me, they're not. The effects could be -- you could have interactive effects of various kinds. You can have theoretically a negative association or you could have a positive association.

The positive association could be additive, could be simply the sum of the individual carcinogenic effects. Or it could be a synergistic which could be a multiplicative. So the effect could be a multiplicative effect.

Q. In some combinations of chemicals would one chemical act -- or can one chemical act as an inhibitor upon other chemicals?

A. Theoretically, yes.

Q. You say theoretically. Have you not seen that circumstance?

A. There's some experimental evidence along these directions, but I don't know of any evidence as far as humans are concerned. All the human data point in the direction of either additive or

synergistic interactions.

Q. When you talk about Mr. Peterson having a substantially increased risk with regard to both progressive chronic obstructive lung disease and I think you also said the same thing with regard to developing other cancers, what are you comparing that risk to?

In other words if you have a substantially increased risk, you have to be using some baseline risk to begin with I would imagine.

So what are you comparing the risk to?

A. Well, I'm frankly -- as far as the obstructive lung disease I'm not comparing it to the general population because the general population doesn't have in general a risk of chronic obstructive lung disease.

What I'm saying is compared to his present status. In other words there's a substantial probability that there will be deterioration in his medical status. That's what I'm saying with relation to the chronic obstructive lung disease.

With relation to cancer, as far as cancer's concerned, however, I would say as

compared with the general population.

Q. So if I recall your testimony correctly from the first day, you're using a risk -- a general risk of 25 percent to 33 percent for the population at large and you're talking about a substantial increase on that risk?

A. Yes. There's a 1 in 3 risk of getting cancer, which is as you say 33 percent, and 1 in 4 risk of dying from cancer, which is the general population.

Q. I read the Austin paper which is included in your report --

A. Abstract.

Q. Whatever it is that appears in Epstein 4, when I read that there was a considerable discussion with regard to the effects of smoking and alcohol upon a laryngeal cancer.

You have stated on page 2 of your report -- and if I'm interpreting it correctly you are suggesting that the fact that Mr. Peterson has not smoked since 1968, which was twelve years before he developed laryngeal keratosis and sixteen years before he developed laryngeal cancer, is a factor that you have taken into consideration.

Can you tell me why in your mind the fact that he had not smoked in that period of time should not be considered more strongly than apparently you have in this report?

A. Yes, I can give the U.S. Surgeon General as one of several references showing the effects of the risks associated with light smoking firstly and secondly the cessation of a prolonged duration of smoking cessation.

These are two factors which make the role of tobacco to say the least minimal on the relatively small amount he smoked, but far more importantly is the prolonged duration of smoking cessation.

Q. He was smoking, however, at the rate of one pack per day --

A. Yes.

Q. -- when he first started employment with ATC; is that not correct?

A. Yes, from the age of about 17 to 39.

Q. I'm sorry, say that again?

A. From the age of about 17 to 39 he smoked about one pack a day, and you may recall that he started work at ATC actually about the age of 38 so

it was just about the first year.

Q. You just looked at your list. When did he first start at ATC?

A. I have a note that he started at the age of 38; 38 to 44.

Q. Do you have a reference as to what year that was?

A. Yes, 1967. So '67 -- if he was born in '29, that's about 38. Am I right or am I off a year?

He was born in April 1929 and he went to work at ATC in 1967, in June '67. So what's April '29 to June '67? That's two months and -- it's more like 37. I won't rub it out and change it.

Q. Are there studies that talk about a synergistic effect of cigarette smoking and exposure to asbestos?

A. Oh, yes, sure.

Q. Is there a synergistic effect when those two exposures are present?

A. Yes -- well, there's an element of controversy on this. Selikoff has published extensively on this. Wagoner has claimed that the evidence for this is less persuasive. I don't have

a position on this.

Q. You do not?

A. No. My general understanding would veer more towards the Selikoff position but I haven't researched this in detail.

Q. Is this an area you would defer to Dr. Velez on?

A. Yes, I think so. I think Velez has looked at asbestos more closely than I have.

Q. Are there any studies to your knowledge that talk about smoking -- cigarette smoking and its impact on laryngeal cancer that also discuss the concept or the effect of the cessation of smoking?

A. Yes, sure. The U.S. Surgeon General's report summarizes various studies on cessation of smoking. You'll find that quoted in extracts on that under the section laryngeal -- Larynx Cancer.

Q. Would you tell me what your recollection is of the Surgeon General's report?

A. Sure. Basically there's a progressive reduction in risk following cessation of smoking. By the time you reach about ten or eleven years or so, the risks are virtually normal.

Q. Is that with regard to virtually all possible effects of smoking?

A. No, not at all.

Q. Perhaps I'm confusing you.

In terms of your answer are you talking about an elimination of virtually all risk of contracting cancers as a result of smoking?

A. I was referring specifically to larynx cancer. Similarly it relates to lung cancer. The situation as far as cardiovasoular disease and cancers at other sites in which smoking plays a role I don't know.

I think as far as cardiovascular disease there is such an effect although the specifics of the timing I don't know. As far as other sites at which smoking is a risk factor, although much less of a risk factor than for lung or larynx, I don't know. I haven't looked up those data.

Q. Are you aware of any studies that show a causal relationship between cigarette smoking and laryngeal cancer?

A. Oh, yes.

Q. Are you aware of any studies that talk about the latency period for the onset of the

laryngeal cancer as a result of smoking?

A. May well be in the -- I should think the U.S. Surgeon General's report deals with it. I don't offhand recall.

Q. You don't offhand recall what the latency period might be or --

A. No, except that in general we do know that Peterson got his laryngeal cancer at a much earlier age than is known in the general population; and as we know that smoking as -- that one of the major risk factors for laryngeal cancer in the general population is smoking, the fact that Peterson got his lung cancer at a much earlier age would also imply that the latency for smoking and laryngeal cancer is a much longer one.

For instance if you turn to Table 2 of the Austin, you see that by far the most -- the highest incidence of laryngeal cancer is above the age of 60. Now Peterson got his laryngeal cancer in the 50 to 54 age bracket and his laryngeal keratosis he got even before that.

So one would say that Peterson got his laryngeal disease at a very, very much earlier age than one would have anticipated from factors other

than occupational.

Q. I'm sorry, I'm not following what implication you draw from the fact that he had contracted his cancer at an earlier age than the general population.

A. I'm saying that in the general population one of the major risk factors is smoking, and in the general population the maximal incidence of laryngeal cancer is the age of 60-plus.

And if you'll recall Peterson got his laryngeal cancer at a very much earlier age. In fact he got it at the age of 54. But in fact even at the age of 49, which was five years before that, he had developed laryngeal keratosis.

Q. And what inference do you draw from that?

A. I would say this is just one of many factors all of which tend to minimize the role of -- one of many factors which tend to minimize the role of smoking and to maximize the role of occupational factors.

Q. Wouldn't this table set forth by Austin include smokers both active and former --

A. Sure.

Q. -- in the general population?

A. Sure. It would reflect the general population, yes, but the fact is that as we know that smoking is a very strong risk factor, particularly smoking in relation to alcohol, I would say that the greatest impact of smoking would appear to be in the age of 60-plus. It's difficult to take it very much further than that.

Q. Even for someone who began smoking at the age of 17?

A. Sure. He smoked about a pack a day which would make him a relatively light smoker. If you turn to the Surgeon General's report, you'll find the relationship between smoking and laryngeal cancer is strongly dependent on the numbers.

For instance, if you turn to Figure 21 you'll see that for males who smoked cigarettes of under a pack a day, it's relatively small compared to people who smoke four packs a day. And again if you turn to Table 20 you'll find this laid out again. For people who smoke under a pack a day, it's lower than a half of people who smoke four packs a day.

Q. Does the Surgeon General list a light smoker as someone who smokes one pack a day?

A. I don't know the Surgeon General's definition, but under one pack a day is certainly light compared to four packs a day and the data here are listed for four packs a day.

But you have two factors: One, the amount he smoked and, two, the duration of cessation which is given in figure 23; and you can see by eleven years the risks are the same as the general population.

Q. Somewhere in your report you indicate -- page 4 of your report you talk about a latency period of nineteen years.

A. That's for angiosarcoma of the liver. There have been various figures from 19 to about 21 seems in most of the literature to be the average latency for the angiosarcoma of the liver.

Now the angiosarcomas of the liver are associated with relatively high exposures, so when you're dealing with much lower exposures you would expect a much longer latency.

Q. Just so the record is clear, the latency period is for the effect of angiosarcoma of the liver but --

A. Excuse me. That's from the time of

initial exposure to the time of diagnosis.

Q. I understand that. I want to make the record clear.

It's the latency period from the first exposure to cigarette smoking, correct?

A. You've got me -- I don't understand what you're talking about.

Q. Let me find the reference --

A. Just say that -- oh, do you mean duration of cessation of smoking?

Oh, page 2 discusses that.

Q. I'm confused. I was referring to page 4. There is a reference to nineteen years, but it's the average latency period from first exposure to diagnosis --

A. Of angiosarcoma, yeah, sure.

Q. -- of angiosarcoma. So there is nothing in your report that talks about latency period of contracting any cancer as a result of exposure to cigarette smoking except as referred to if at all in the Surgeon General's report?

A. Well, the Surgeon General only -- parts that I quoted here only deal with laryngeal cancers. As far as lung cancers are concerned I

don't have any material here.

There's a dose response relationship with very high levels of smoking and particularly when you smoke starting from the age of ten or so, latency is shorter.

But one of the points about the respiratory system cancers and respiratory tract cancers are that the longer you follow these people -- the VC/PVC workers -- up, the higher is the rate of lung cancer.

This is stated in several of the references in the table; that the incidence of respiratory tract cancer is higher in those, A, that have been more heavily exposed and, B, that have been followed up for longer periods.

Q. Do you believe that you can have a no observable effect level for a carcinogen?

A. What? All one can say is there is an overwhelming body of information in the literature that we don't know any way of setting safe levels for chemical carcinogens.

Q. Why is that?

A. This is an analysis of the literature; and as far as vinyl chloride, for instance, is

concerned, even at the lowest levels to which it's been tested -- which vinyl chloride has been tested, namely 1 ppm, carcinogenic effects have been found.

Carcinogenic effects have been found also with exposure to vinyl chloride for one hour, one hour.

Q. What is the concern? Is --

A. Can I move on? I want to say --

Q. I'm sorry.

A. In addition to the experimental evidence, we have evidence from human beings showing that very low levels of exposure are associated with major increased risks. For instance, people living in the vicinity of VC/PVC plants have an excess risk of angiosarcoma of the liver.

In fact, calculations were developed to the effect that even at the part per billion -- 20 part per billion level, the risks of angiosarcoma are significantly increased. These are parts per billion.

So -- but that again is consistent with the experimental data that we have and the human data that we have that any exposure confers finite

risk. The higher the level of the exposure, the greater is the risk.

Q. What is the reason for that concern? Is it that a single molecule of a carcinogenic material can cause cell damage and as a result of that lead to a tumor?

A. Well, that with due respect is a reductio ad absurdum because a part per billion level of vinyl chloride is equivalent to billions of trillions of molecules, billions of trillions; for instance, maybe some region of 10 to the power of 15. That's quadrillions of billions. So we're not really dealing with a one molecule.

All one can say is at the lowest level tested in animals there's still carcinogenic effects; and even at the lowest levels of possible exposure to humans, such as an accountant working in a plant without ever going to the workplace still gets a carcinoma of the lung, people living a few miles away from a VC/PVC plant still get angiosarcoma of the liver.

From a theoretical standpoint one could say that theoretically one hit, one molecule on one receptor site, can create genetic damage which can

theoretically be associated with carcinogenic effects. But these levels -- even the very low level of a part per billion is associated with millions of trillions of molecules.

Q. Cigarette smoking has a carcinogenic effect, does it not?

A. Correct.

Q. Would there not be a similar concern for extremely low levels of exposure to cigarette smoking?

A. Absolutely right. A person who smokes one cigarette a day has more risks of getting lung cancer than a person who smokes no cigarettes a day, no question.

Q. Mr. Peterson was exposed to that carcinogenic chemical, if I can call it that --

A. Well, mix of chemicals.

Q. How so?

A. There's a vast mix of chemicals in tobacco smoke.

Q. I'm sorry --

A. From the age of 17 until the time he went to work at ATC, but as I say that he had stopped smoking for about eleven years or so before he

developed his laryngeal cancer; and based on the analysis of all available data by Winder (phonetic) and by the U.S. Surgeon General, the risk of his laryngeal cancer is the same as that of a nonsmoker.

Q. Assume for a moment that Mr. Peterson had been exposed to VC for approximately a twenty-year period and then had moved on to different employment and thereby had a cessation of exposure to VC.

Would you believe after -- for some time after the cessation of that -- strike that.

Would you believe that Mr. Peterson would be at risk for the contraction of cancer for the rest of his life from that exposure to VC?

A. Nice try. In fact his risks would progress and would become greater the longer he lived.

Q. Why is that?

A. I was hoping you'd ask that.

There's an enormous difference between what's called a late stage carcinogen such as tobacco where the carcinogenic effects of tobacco depend on the -- until you reach an irreversible

point, depend upon the continuing exposure.

In other words once you remove somebody from the exposure to tobacco, their risks decrease progressively. You see the similar effects with women with endometrial cancer from estrogens, from estrogen replacement therapy. The moment you remove them off from their estrogen replacement therapy, their risks of endometrial cancer shoot way down.

When it comes to the overwhelming majority of carcinogens, which we call early stage carcinogens, the longer -- very often with these people the longer you wait, the higher is their risk.

You see this -- as far as respiratory tract cancers are concerned, the highest rates of respiratory tract cancers are on people who have been followed up for a long period of time; and Infante and others, however, have pointed out the insensitivity of many of the epidemiological studies insofar as people haven't been followed up long enough.

Doll in fact also, even in spite of all the criticisms I've made about the Doll study, is

forced to recognize that the risks of lung cancer are greater in those who have had a longer period of observation.

Q. In essence you're telling me that all carcinogenic materials do not have the same impact on the body and therefore do not raise the same concerns?

A. Well, your language is your business. What I'm saying is that when it comes to tobacco there is a very marked decrease in risk proportional to the cessation. When -- the period of cessation from smoking.

There are a few other carcinogens that fall into that category which are called late stage carcinogens. For the overwhelming majority of carcinogens, however, you're faced with a lifetime risk once you've been exposed.

In fact, you can have just one exposure for one hour only with vinyl chloride and then get tumors later in life as we know from the Consumer Products Safety Commission studies on vinyl chloride with mice and rodents.

So once you have initiated a carcinogenic effect, these risks will last for a lifetime and

the longer you follow people up, the greater will be the number of cancers you will uncover.

To give you one example for the asbestos which we are discussing here, the asbestos risks, you can be exposed for five years -- a period of five years and then no more. Then the risks don't really become very serious until about a thirty-year period later.

Q. Is asbestos known as an early stage carcinogen or a late stage --.

A. An early stage carcinogen.

Q. And that would make it similar to --

A. Vinyl chloride and the other carcinogens he was exposed to, the other occupational carcinogens.

Q. So that an exposure to asbestos theoretically on one occasion carries with it a greater --

A. A greater risk than no exposure.

Q. -- risk than no exposure?

A. Well put, yes.

Q. But that risk with regard to asbestos continues for the rest --

A. For lifetime.

Q. -- of the person's life?

A. Correct. And the same with vinyl chloride and polyvinylchloride.

MR. HOLLINGSHEAD: I'll move to strike that response as not responsive to the question. To be more precise, I move to strike the last portion of that answer as not being responsive to the question.

Let's take a short break.

(Short recess.)

BY MR. HOLLINGSHEAD:

Q. Dr. Epstein, just going back to the latency period discussion for a moment, normally is the latency period given from first exposure to a carcinogenic material or from last exposure?

A. Latency is often defined in very -- neither.

Q. Why don't you tell me how you define latency period and from what point in time do you count the latency period?

A. Latency is defined in different ways by different authors. Let me just tell you what these are.

Generally latency is defined from time of

initial exposure to the time of the diagnosis of the particular adverse effect. That's one. Sometimes latency is defined, however, from the time of initial exposure to the time of death; and that is a little sloppy because the time -- when it comes to fatal cancers like lung cancer, it doesn't really make any difference. You simply add one or two years on.

But when it comes to other cancers of which you can have prolonged remissions or even cures like laryngeal cancer, latency defined from time of initial exposure to death for laryngeal cancer would be a ridiculous thing because you have a 50 to 60 percent cure rate for laryngeal cancers.

So those are two of the forms: from time of initial exposure to the time of diagnosis is one or to the time of death is a second. And sometimes latency is defined more sloppily from the midpoint of exposure.

But I would say that the best way of defining latency is from initial exposure to diagnosis bearing in mind that this isn't a particularly good definition because initial exposure may not reflect the period of maximal

exposure.

But when you're dealing with an individual when you can characterize different exposure levels, one could refine it. But when you're dealing with a population and an epidemiological study, the only real way to handle these data are from times of initial exposure to times of diagnosis and that's generally the way latency is defined.

Q. We may have discussed this over the course of the two days and, if so, I'm sorry to raise it again but --

A. Sure.

Q. -- do you have a latency period in your mind as to vinyl chloride?

A. Well, we talked about it in relation to angiosarcoma of the liver. When it comes to respiratory tract cancers, clearly the latency is longer in general.

This is stressed in the various references I have given in the table where it talks about the lung cancer rates are particularly high in those with longer periods of observation and longer duration of followup.

And also it's reflected in comments of Infante and others that most epidemiological studies are insensitive because it's not an adequate latent period -- followup. In other words people haven't been followed up for a longer period of time.

What one does seem to see with vinyl chloride is at relatively high exposure levels you get angiosarcoma and the average latency is about nineteen, twenty years or so. With lower exposure levels you get a shift in the target organ from angiosarcoma to other sites, and the latency is much greater than twenty years.

Q. Do you have a range in mind?

A. No, it's not possible to say this because most of the epidemiological studies have had an inadequate followup. But you see the longer you observe these people, the higher the lung cancer rates become -- the higher the respiratory tract cancer rates become.

Q. In any event, whether it's angiosarcoma of the liver or some other carcinogenic effect, you're looking at a latency period that is commenced with first exposure generally?

A. Yeah, that's the proper way of looking at it.

Q. And that's the way you look at it?

A. Yes, sure.

MR. HOLLINGSHEAD: That's all I have, Dr. Epstein. Thank you very much.

THE WITNESS: Fine. Thank you.

FURTHER DEPONENT SAITH NOT...

STATE OF ILLINOIS)
) SS:
COUNTY OF DU PAGE)

I, Sandra A. Kaspar, a notary public within and for the County of Du Page and State of Illinois, do hereby certify that heretofore, to-wit, on the 15th day of December 1989, SAMUEL EPSTEIN, M.D., personally appeared before me at 303 West Madison Street, Suite 1400, in the City of Chicago, in the County of Cook and State of Illinois, a witness in a certain cause now pending and undetermined in the Superior Court of Middlesex County, New Jersey, wherein John and Shirley Mae Peterson are plaintiff and Union Carbide Corporation is defendant.

I further certify that the said witness was first duly sworn to testify the truth, the whole truth, and nothing but the truth in the cause aforesaid; that the testimony then given by said witness was reported stenographically by me, in the presence of the said witness, and afterwards reduced to typewriting by computer-aided transcription, and the foregoing is a true and

correct transcript of the testimony so given by said witness as aforesaid.

I further certify that the signature of the witness to the foregoing deposition was waived by agreement of counsel for the respective parties.

I further certify that there were present at the taking of this deposition the attorneys as hereinbefore noted.

I further certify that I am not counsel for nor in any way related to the parties to this suit, nor am I in any way interested in the outcome thereof.

In testimony whereof I have hereunto set my hand and affixed my notarial seal this 22nd day of December 1989.

Sandra A. Kaspar, CSR
Notary Public, Du Page County, Ill.

OFFICIAL SEAL
SANDRA A. KASPAR
NOTARY PUBLIC STATE OF ILLINOIS
MY COMMISSION EXP. MAR 24,1992