SUPERIOR COURT OF NEW JERSEY
LAW DIVISION: MIDDLESEX COUNTY
DOCKET NO. L-060148-87

JOHN PETERSON and SHIRLEY MAE PETERSON, his wife,

Plaintiffs,

vs.

DEPOSITION UPON ORAL EXAMINATION OF: SAMUEL S. EPSTEIN, M.D.

UNION CARBIDE CORPORATION,

Defendant

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TRANSCRIPT of the deposition notes of SAMUEL S. EPSTEIN, M.D., witness, called for oral examination in the above-entitled action, said deposition being conducted pursuant to the Rules Governing Civil Practice in the Superior Court of New Jersey, by and before CINDY L. NAGLE, a Notary Public and Certified Shorthand Reporter of the State of New Jersey, License No. XI01434 at the offices of Scadden, Arps, Slate, Meagher & Flom, Esquires, 919 Third Avenue, New York, New York 10022, on December 1, 1989, commencing at 10:25 a.m.

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A P P E A R A N C E S:

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S A M U E L S. E P S T E I N, Apartment 25M 860860 North Lake Shore Drive, Chicago, Illinois 60611, having been duly sworn by the Notary, testifies as follows:

DIRECT EXAMINATION BY MR. HOLLINGSHEAD:

Q. Doctor, good morning. Let me introduce myself for the record. I am Robert Hollingshead with the law firm of Pitney, Hardin, Kipp & Szuch. We represent the Defendant, Union Carbide Corporation in this action that was instituted by Mr. and Mrs. Peterson on whose behalf you have submitted an expert report.

As you know, and as I'm sure you are very familiar with this, this is a deposition of which is a time for me to inquire into that expert report and the various reports and factual information that are behind it.

Again, since I know you are familiar with depositions, I will give you a couple of cautionary notes.

One is, please allow me to finish my question before you start your answer and I will do you the courtesy of allowing you to finish your

answer before I ask the next question.

Secondly, please keep all of your responses verbal on behalf of the reporter who cannot interpret nods of the head or shakes of the head.

Thirdly, if you have any question whatsoever about my question, either it's not clear or perhaps has a misstatement of fact in it, please tell me so and I will do the best I can to rephrase it until you understand it.

Obviously, if Mr. Levinson has any objections to my questioning, you should allow him to put those objections on the record and then we will proceed with his instruction to you as to whether you may answer or not, or if the objection can be worked out between us, I will rephrase the question.

A. Sure.

Q. All right, do you have any questions before we begin?

A. No, not at all, thanks.

Q. Do you happen to have with you today a current or reasonably current curriculum vitae or resume?

A. I'm terribly sorry, I haven't, but I can ship it to you on Monday.

Q. Would you please do so?

A. Yes, let me make a note of it.

Q. Sure.

A. Okay.

Q. Without the benefit of that resume, let me just go through a few questions with regard to your background.

I should note for the record initially and as I think, Dr. Epstein, you recall I took your deposition approximately ten years ago in a different matter. I believe at that time we went through a fair amount of your background and so what I might wish to do is simply try to bring it current to a certain extent in the last ten years.

What is your current position, Dr. Epstein?

A. Same as it was then, professor of occupational and enviornmental medicine, School of Public Health University of Illinois Medical Center, Chicago.

Q. Has there been any change in that position that you hold at the University of Illinois Medical Center in the past ten years?

A. No.

Q. What degrees have you been awarded through your educational process and when were they?

A. None since 1980.

Q. Just refresh my recollection, this in regard as to what your degrees are that you hold?

A. I have a B.Sc., in the '46, MBBS, which is the university degree in medicine, in England. D Path, that's a degree in pathology, DTM&H degree in tropical medicine and hygiene and M.D., which is the most advanced degree in internal medicine, I think experimental medicine and sundry honorary degrees.

Q. Was the M.D. conveyed upon you in England?

A. Yes, all of these were in London University.

Q. When did you receive your M.D.?

A. 1958. The M.D. isn't the qualifying examination, it has no privilege equivalent to this. The equivalent to the American M.D. is the M.D. B.S. which I got in 1950.

Q. You say that's the equivalent of the United States version of the M.D.?

A. Correct.

Q. Have any degrees been conveyed upon you as a result of any education in the United States?

A. No.

Q. Are you a licensed physician of any state in the United States?

A. No.

Q. In your position as professor of occupational and environmental medicine at the University of Illinois Medical Center, do you have a practice wherein you diagnose, treat or see patients at all?

A. No.

Q. Do you have any board certifications in the United States?

A. No.

Q. When you provide me with your C.V., will it have a list of publications that you have authored over the course of time?

A. Yes.

Q. And up until the reasonably recent time frame?

A. Yes.

Q. Are any of those publications, to your recollection, on the subject of any of the chemicals that are in this case, and let me name them if I may as I understand them and as I read your report, polyvinyl chloride, vinyl chloride monomer, polystyrene, isopropilidene resin and various chemicals that are emanated by welding fumes which we will get to in more detail later?

A. Yes, I think I discussed several of these in a

book called the Particulars of Cancer published in 1978/79, the asbestos, the vinyl chloride, polyvinyl chloride.

It's very possible I have some 300 publications, likely that several of these chemicals have been discussed, but to be quite frank, offhand I can't recall.

Q. In my list of the chemicals a moment ago as you pointed out in your answer, I did forget to list asbestos, I did not mean to do so.

A. Oh, that's okay.

Q. Dr. Epstein, in the past ten years, have you appeared in any piece of litigation which PVC or polyvinyl chloride has been an issue?

A. Yes.

Q. Would you tell me, if possible, approximately how many?

A. Sure, I have these listed here.

Q. Did you prepare that list in response to the Notice of Deposition that I supplied to you?

A. Yes, I tried to anticipate all of the questions.

Q. Very good.

A. Prior VC/PVC Litigation, I will start in the beginning. You did say the last ten years?

Q. I did. Let's go back all the way, perhaps we can start with the first time?

A. The first case was Elias versus Parke Davis and Sprayon in 1979. I was deposed in that case but to the best of my recollection there was no trial.

Q. What was the issue, very briefly?

A. It was an infant that had surgery, and I think it was for a congenital Pyloricstenosis. And on a few occasions, two or three occasions, the incision scar was sprayed with a material called Sprayon, which when it hardens it forms a kind of an elastic bandage so you don't have to use sutures, to minimize the need for sutures and VC was the propellant, vinyl chloride, and the infant subsequently developed a malignant tumor known as hepato blastoma and I was the expert in that case and it was settled after that deposition.

The next was Maliko et al versus Union Carbide in 1979, which of course you're both familiar.

The third was Grasso versus B.F. Goodrich in 1981, in which Dr. Davidson referred to in his deposition. This is a man who lived about one and three quarter miles from a B.F. Goodrich Plant, didn't work in the plant, but he died from

angiosarcoma of the liver. I testified there, I was deposed there and also testified at trial.

The fourth case -- am I going too fast for you?

Q. No.

A. Fourth case, Mikyska versus Ford and Union Carbide in '81, in which I was deposed. This was workers at a Ford plant involved in PVC fabrication and exposed to VC as a result of that and developed a toxic hepatitis, and again I was deposed there and it was settled, to the best of my recollection, without trial. Those are the only four cases that I have any recollection or any record of.

Q. In the Grasso case, what was the exposure that Mr. Grasso sustained in his residence one and three quarter miles from the plant?

A. Well, vinyl chloride.

Q. Not fumes?

A. Yes, to the emissions from the plant and these were based on emission data of farmer plant material, balance studies, modeling and air dispersion data, we had no actual monitoring data.

Q. That was not a resin, it was a VC/PVC emission case?

A. It was VC/PVC plant but the exposure was to

VC.

Q. Do you keep copies of the expert reports that you render?

A. When I write a formal report I keep those, otherwise all I do for an old case is to simply keep a letter of the attorney and possibly one or two scraps of paper.

Q. Do you have a recollection as to whether you wrote a formal report in the Elias matter?

A. I don't think I did but I'm certainly willing to check.

Q. Would you please do that?

A. Sure. What you don't need is obviously the Maliko stuff, you have that already.

Q. No.

A. I will check to see if I have formal reports on any of those other cases.

Q. And also --

A. On three cases.

Q. Elias, Grasso and Mikyska.

A. VC prior litigation cases.

Q. Do you also retain deposition transcripts if they are provided to you?

A. Sometimes I do, sometimes I don't. It depends how interested I am in them. If they are of

particular interest to me I would retain them, absent to that I wouldn't.

Q. May I ask you to check for those too, and if you have them, advise Mr. Levinson?

A. If I do have that stuff, do you want my office to copy them and send them to you or do you want me to send you the originals?

Q. You can have your office copy them and bill me for the copying.

A. Fine.

Q. What files do you retain on other litigations in which you've appeared?

A. I generally keep virtually nothing, with the exception I have a letter from the attorney and if there's anything of a particular scientific interest I keep, otherwise once a case is over I discard the material.

I would keep any written report clearly and that's about it. Sometimes I would keep a summary, if, for instance, I had done a survey or a chart on that I would keep that because clearly there was work involved and I wouldn't want to throw that away.

Q. I would like to see any documentation that you have on any of these matters which might

include correspondence with the attorneys or identification of the attorneys for the defendants.

A. Sure. Also other paperwork, with pleasure, except for the Maliko.

Q. Excluding Maliko --

A. Okay, et al.

Q. -- Which I appeared ten years ago and I have those papers.

A. Yes, all right.

Q. Have you appeared in any cases prior to this litigation in which you have rendered an opinion on the chemical Polystyrene?

A. No.

Q. If memory serves, was Polystyrene involved in the Maliko case?

A. Yes.

Q. Would that be the only one then?

A. That's correct.

I'm sorry, my previous answer was incorrect. I had, in fact, it was in the Maliko case, I beg your pardon.

Q. I seem to recall that there were other chemicals in that case, too, but that was one of them?

A. Yes.

Q. But the main focus was PVC, if I recall?
A. Correct.
Q. Have you given expert reports in any other case on the chemical asbestos?
A. I've been involved in very little asbestos litigation, one or two, and I can't recall whether I've done any reports.
Again, I can check on that if you like.
Q. Would you please?
A. Three; check reports on asbestos litigation. Okay.
Q. And to round out the listing here, have you written any other expert reports on Isopropilidene Bisphenol Resins?
A. I've been involved in one case on Bisphenol A, and I will do the same also.
Q. All right.
Now, do you equate Bisphenol A with isopropilidene resins?
A. Yes.
Q. Let me remind you that Bisphenol A wasn't in the Maliko case.
A. Yes, when you ask me for any paperwork I'm excluding Maliko.
Q. But I do want your testimony to be

accurate and although you and I are assuming Maliko is not involved, the Bisphenol A report you rendered in Maliko was one of those?

A. Correct.

Q. But you don't need to send that to me.

We also have at issue in this case as I read your report basically emissions from welding fumes. Have you had occasion to write an expert report on that subject prior to Peterson?

A. No.

Q. Tell me very briefly what your current responsibilities are as professor of occupational medicine at the University of Illinois Medical Center?

A. Research, teaching and public service.

Q. Do you have a teaching course load at this present time?

A. Yes.

Q. Can you tell me what it is now?

A. I basically do one intensive course a year, generally the winter course in which I go in great depth into a series of current issues that have a scientific, strong scientific involvement in environmental or occupational matters and discuss these from a set of prospectives, including the

scientific, the decision making, the regulatory and regulative and this is basically more advanced students, postgraduate students of Ph.D. or M.D. who want to get further training in public health, so it's a fairly advanced course which I teach, I gave major assignments, I work with the students on this.

Q. Is that a course that you are teaching now?

A. Yes, I will be teaching in January.

Q. So for this fall you have not had a teaching load at all?

A. No.

Q. What is the research that you do presently at the University of Illinois?

A. I research a fairly wide range of issues; from problems with pesticides to on one hand problems of air pollution, problems of internal combustion, engine, gasoline, problems of hormones, the use of biotechnology and dairy farming, it's a wide range.

Q. When you use the word research in your answer, Dr. Epstein, what do you mean by that?

Are you talking about book research, if I may call it that?

A. At this stage it's largely book research and scientific publications based on book research, yes.

Q. Do you have graduate students assisting you in any of your research?

A. No.

Well, only insofar as if indeed in the course of my teaching a particular topic or issue pops up which one of the students wants to develop in great detail with me, I would then publish an example. For instance, in last year's course I had a gastroenterologist from University of Chicago who was one of the students, and based on the course we did a joint publication in environmental occupational causes of colorectal cancer. So only incidentally that way, but I don't work with students on my publications, it's only as an outcome of any course I happen to do.

Q. Do you mean to exclude from that any research you might do with regard to litigation in which you are appearing?

A. Oh, no.

Q. Let me go back then in terms of research that you do at the University of Illinois. Would it also include what I might refer to as ad hoc research in order to render an opinion in a particular case?

A. That wouldn't be part of my university --

Q. That would be part of your practice as a

consultant?

A. Precisely.

Q. Thank you for the distinction.

Let's stay with your work at the University of Illinois then. How much time on a weekly basis do you spend in your capacity as a professor with the University of Illinois?

A. I have a 90 percent appointment, so I'm part time with the University.

Q. I'm not too sure I know what that means?

A. Ninety percent; in other words, I'm not full time so I'm paid on a 90 percent basis not a 100 percent basis, which means that I, over and above whatever time, generally most people take about a day a week on private work, I have a little extra time because I'm only 90 percent as opposed to 100 percent so that's about an extra half day.

Q. Take the last six months or so, if you could, and take average, could you tell me how much time during the course of a week you might spend on university responsibilities and how much time on your consulting practice?

A. Well, my university work would be generally about four days a week.

Q. And generally would the fifth day of the

week, if we assume that we're talking about a five day work week, would that be devoted to your consulting work?

A. Yes.

Q. And is your consultant work primarily in the area of litigation?

A. I would say it's almost exclusively an area, yes.

Q. During the course of the past five years or so, have you consulted at all with any manufacturing or chemical companies on their bahalf?

A. I don't think any have invited me.

Q. Okay.

A. I'm always open.

Q. Has your work been pretty much exclusively on behalf of injured individuals in various capacities?

A. Correct.

Q. Would you say that over the course of the past five years your work load in terms of it being split between the university and consulting practice has been the same as you've indicated for me, that is four days a week for the university and one day a week for consulting practice?

A. Yes.

Q. In the course of your consulting work, have you ever appeared in a case or rendered a report in any litigated case where the injured person had a laryngeal cancer prior to Mr. Peterson's case?

A. Yes.

Q. Can you recall for me some of the details of that?

A. I think I can. I think it was a worker in a NASA plant.

Q. That's the National Aeronautics and Space Administration Plant?

A. Yes. Who was exposed to a fairly wide range of agents and developed laryngeal cancer. I forget the name of it, but I can try and find it for you and send you whatever paperwork I have.

Q. Thank you, I would appreciate that.

Is your recollection that that is the only prior situation wherein which you've appeared where there was a laryngeal cancer?

A. Well, minor correction, with due respect I didn't appear. In fact, I'm not even sure what -- I think there was a depo, yes. Well, okay, so I think there was a depo but no trial and excepting that and of course the Maliko, Wilkinson.

Q. One of the individuals?

A. Wilkinson.

Q. Was it Wilkinson?

A. Precisely.

Q. But other than those two instances you don't have a recollection of appearing in any other cases in which there was a laryngeal cancer?

A. No.

Q. Correct me if I'm wrong, Dr. Epstein, but you would not be qualified to sit to take any board certification examination in the United States?

A. No, I haven't. I don't have a medical qualification in this country.

Q. Which would be necessary to do that?

A. Yes.

Q. How did you become involved in this case, the Peterson case? When was your initial contact with Mr. Levinson?

A. Mr. Levinson wrote to me in April of '88 and mentioned the case and then both of us seemed to have let the matter drop for another six months or so, and about January of this year Mr. Levinson called me again and got me started on the case.

So really, I didn't formally get involved until the beginning of 1989.

Q. When Mr. Levinson called you in April of

19 --

A. Excuse me, he wrote to me.

Q. I'm sorry, you did say that. When he wrote to you in April of 1988, did he provide you with any information or materials with regard to the case?

A. No, just a letter in which he talked very briefly about the case, then I believe subsequent to that we had a brief discussion and I agreed in principle to get involved, but we didn't actually get started until about January of this year.

Q. In January of 1989, I think you said that he called you again and got you started on it?

A. Correct, yes.

Q. Was there further correspondence at that time advising you as to details of the case?

A. Yes.

Q. And did you receive materials from Mr. Levinson at that time?

A. Well, I don't know exactly at that time, but on an ongoing basis subsequent to that Mr. Levinson sent me records on the case.

Q. Have you done any work for Mr. Levinson or his law firm in your consulting practice in approximately the last ten years or since the Maliko

case?

A. Unfortunately we seem to have drifted apart.

Q. So this is the first litigation you've had with Mr. Levinson since the Maliko case?

A. Yes, correct.

Q. And that is also true of anybody else in this firm, you have not had any professional contact with them in the past ten years?

A. Correct.

Q. I supplied Mr. Levinson with, I believe he sent to you a Notice To Take Deposition And Produce Documents, and I believe you indicated earlier that you have some materials here with you?

A. Correct.

Q. Let me mark this as an exhibit, if I may, and we will see if we can figure out what you bought in response to it.

(Notice To Take Deposition and Produce Documents is received and marked Epstein 1 for identification.)

Q. Dr. Epstein, I have marked as Exhibit Epstein 1 the Notice To Take Deposition and Produce Documents. Let me so show it to you.

A. I have it here, thank you.

Q. Can we just run through this and see

what you might have in response to the six separate categories that I have asked for?

A. Either that or I can tell you what I have, whichever you prefer.

Q. I just need to know what might line up.

The first group of requests, which I will paraphrase, was asking for records of communications between yourself and Mr. Levinson or his office, including all documents that are in writing, obviously documents in writing that would include correspondence, memoranda, notes, et cetera.

Do you have anything that would be in particular response to that request?

A. Yes, I have his original letter to me of April '88, all subsequent correspondence or documents that he sent me I've incorporated in various sections of this notebook which we can go through at a later stage.

Q. Let me ask a broad question, Dr. Epstein. Is there anything in that notebook that either you or Mr. Levinson might consider to be confidential or is it all available?

MR. LEVINSON: I don't think so. I haven't looked at it yet, I might add, but I looked at my correspondence in the office, there's nothing

there particularly.

Q. Is the original letter also included in the notebook?

A. No.

Q. All right, why don't we put that there and I will come back to marking it.

So anything in response to item or request number one would be contained between the letter and note?

A. And the note, yes.

Q. Would that also be true for all materials supplied to you by Mr. Levinson's office?

A. Correct.

Q. And would the same be true for all documents reflecting communications?

A. Correct.

Q. This might require some interpretation. When this notice was written this was intended to indicate persons other than Mr. Levinson or his law firm.

A. Correct.

Q. Have you had any such conversations with anyone else regarding the subject matter of this litigation other than Mr. Levinson?

A. Yes.

Q. Who was that?
A. Dr. Velez.
Q. And is there anyone other than Dr. Velez that you have spoken with in this litigation?
A. No.
Q. Have you spoken at all with Dr. Davidson?
A. No.
Q. The communication with Dr. Velez, was that initiated by him or by you?
A. There were two phone calls from him in October of this year.
Q. Do you recall the subject of each of the phone calls?
A. Well, generally. The first one was on, I think he asked me for my report or he had seen the report and I had really forgotten -- let me see, yes, I remember now, the second one was what he asked me for the Tabershaw documents, he said he didn't have them and I sent him a copy of the Tabershaw documents.
Q. The Tabershaw?
A. I beg your pardon, there's three. There's two unpublished, one Tabershaw and Gaffey document, one Tabershaw document and unpublished Tabershaw/Cooper, and I will give you the references for those.

Q. Did Dr. Velez indicate in that conversation why he was asking for the Tabershaw documents?

A. He said he had seen in my report reference to them but he didn't have them and asked if I could send them to him.

Q. Did he tell you that approximately at that period of time his deposition was being taken in this case?

A. No.

Q. Are you --

A. Excuse me. No, that's not true, excuse me. He did tell me on the second conversation that he was going to be deposed and he would like to review the Tabershaw documents personally before that deposition.

Q. Do you have any indication in writing, perhaps in a diary or in any other writing as to when those two phone calls were made?

A. No. I can tell you the second one was prior to his deposition because I remember him saying to me he felt he should review both of those documents, all of those documents before his deposition.

Q. In the first conversation with him, is it your recollection that in general he was only

asking for a copy of your report?

A. Correct, yes.

Either that or he perceived it and had a question about it, I'm afraid I just can't recall.

Q. Did you make any notes regarding that conversation?

A. No, I didn't make notes on the conversations.

Q. Did you make any notes on the second conversation?

A. No.

Q. Did you discuss with him at all in substance any of the Tabershaw documents?

A. No, he just asked me if I had them. He said he was intrigued by them, could I send him copies. I said, sure.

Q. Did he ask you during that conversation, to your recollection, if you were aware of any particular studies that would relate to polyvinyl chloride with laryngeal cancer?

A. No, that discussion was just focused exclusively on the Tabershaw documents.

Q. Did he ask you any questions with regard to your view or opinion as to whether asbestos can cause laryngeal cancer?

A. No, there was no other subject of discussion

other than what I've stated.

Q. In your report, if I recall correctly there is a Tabershaw document referred to, I thought it was Tabershaw Cooper to my recollection?

A. I think there's more than one frankly.

Q. All right.

A. Let me just check.

Q. With regard to all of the documents that you refer to in your report, you include them in your reference, do you not, at the end of each section? I recall that there's a section of references.

A. Yes, you will find actually two references, actually there's one missing here -- No, no.

Q. You're looking at page 6 of your report?

A. Page 6, there's one missing there. There's a Tabershaw and Gaffey published study and then the supplementary Tabershaw/Cooper study, there was a prior unpublished Tabershaw report in 1974 which is basically very, very similar to the Tabershaw and Gaffey published paper.

Q. You've indicated here that the Tabershaw and Cooper report is an unpublished report in 1975?

A. Yes, but in addition to that --

Q. There's another unpublished one?

A. Another unpublished one, which is very, very

close to the Tabershaw and Gaffey published report in '74 and I gave all those three to Dr. Velez and I'm pretty sure from when I read his depo that he gave you copies of them.

Q. Is the Tabershaw and Gaffey report the one published report?

A. Yes. That's the 1974 one, yes.

Q. That's the only one that's been published?

A. Correct, to the best of my knowledge.

Q. When Velez called you, is your recollection that he asked you specifically for the Tabershaw papers?

A. Yes, in the second discussion.

Q. We'll go back to the report later.

A. Sure.

Q. All right, we were talking about item number three in the Notice To Take Deposition. Let's move to item number four which would be with regards to all materials which you have reviewed, consulted or read and considered in reviewing this litigation and the issue that was raised there and in rendering their opinions. It's a very broad category but I wonder if you have anything in the notebook that's responsive to that?

A. Yes, you will find as you go on a wide range of items which were responsive to that.

Q. Is it your testimony that the notebook would contain all of the items that you would have considered in rendering your report, including all studies, et cetera?

A. I'm not willing to state all, but I mean I have extensive background in these areas, but that certainly wouldn't preclude me from saying I haven't information on other matters.

Q. Are all the published materials that you cite in your expert report in this case contained within the notebook?

A. What do you mean all of them, the references do you mean?

Q. Sure.

A. Yes.

Q. Just pointing one out, Newhouse & Berry, the Lancet Volume II, I suppose that is Appendix 1 of your report. If I were to look in your notebook, would I find that?

A. No, what you would find is references to everything I've read, and in many instances tables or inferences drawn on those references but not the original articles.

Q. That's what I need to know. Thank you.

The report that I have been referring to, which we probably should mark, it is entitled Preliminary Report with a date of 9/13/89?

A. Correct.

Q. Is it not?

A. Yes.

MR. HOLLINGSHEAD: Let me ask the reporter to mark this Epstein 2.

(Preliminary Report is received and marked Epstein 2 for identification.)

Q. Dr. Epstein, do you have a copy of your preliminary report?

A. I do.

Q. My question was that the Preliminary Report has a date of 9/13/89?

A. Yes.

Q. Did you ever provide a subsequent report after this preliminary report in writing?

A. This morning I gave Mr. Levinson some additional material dealing with areas which I felt I hadn't adequately considered and which I felt needed further work and, in fact, when I wrote the original report I was under some time constraints, Mr. Levinson said he needed something pretty quickly and

I will be going over those materials with you today.

MR. HOLLINGSHEAD: Mr. Levinson, is it your intention to provide me with the subsequent report that you received this morning?

A. Excuse me, sir, I said I have these materials with me, I will be giving them to you today.

Q. My question is: Have you written a subsequent report?

A. Not a formal report.

Q. That's my question.

A. Let's go back. I haven't written a subsequent formal report, but what I have done is prepared some additional documents which will update that, but I haven't entitled them subsequent formal report.

Q. Are they contained within your notebook in a binder or some section of it?

A. Yes, all in this notebook.

Q. I'm a bit at a loss to determine where to go, perhaps you should indicate to me what those materials are.

A. Absolutely. There's basically three documents. One entitled, just for the sake of facilitating your further reference to these, there in the first section entitled Summaries, okay? The first is entitled, Factors Incriminating VC/PVC As

The Primary Cause Of Peterson's Laryngeal Cancer. It's a one page sheet.

This is a bit out of sequence but it doesn't matter, the second is, State Of The Art On VC/PVC Toxicology Prior To 1974.

The third is Illustrative Literature On The Toxic And Carcinogenic Effects Of VC/PVC In The Respiratory Tract Of Expermental Animals.

The final one is Illustrative Literature On The Toxic And Carcinogenic Effects Of VC/PVC In The Respiratory Tract Of Exposed Workers.

Q. What caused you to prepare those, as I heard you describe it, those four separate documents?

A. Basically, as I said, Mr. Levinson gave me very short notice for the first document. I think he called my office, I really don't recall exactly, about a week or so before, he said I've got to have something from you right away. I said I will do my best and for this reason I wrote preliminary, intending to go into further areas which I consider to be of critical relevance to this case That's basically what I did.

Q. Did you advise Mr. Levinson that you intended to go into other areas at a later point in time?

A. Absolutely. He said, why is your report preliminary? I said, look, you asked me at the last minute, I've done my best but I intend to mesh this thing out prior to my depo.

Q. Did you have a discussion with Mr. Levinson when you returned from Europe sometime in September, as I recall, do you recall that?

A. I would imagine so, I don't recall.

Q. Did you have a further discussion with Mr. Levinson on the subject of a subsequent report or series of documents as you've described?

A. At one stage shortly after I returned, I said, look, I intend to work up some further aspects of this case, and he said fine. He said, whatever you do, either send it to me or bring it to the depo.

Q. Turn back to the first page if you would, please.

A. Sure.

Q. I believe that first page is entitled something with regard to Factors Incriminating VC/PVC As The Primary Cause Of Peterson's Laryngeal Cancer?

A. Yes.

Q. What caused you to write that page after your preliminary report which talks about a number of chemicals?

A. Well, basically, as I indicated before, the initial report was preliminary. Another factor was my reading of Davidson's depo which refreshed my memory as to the specifics of the problems with the PVC dust exposure, and after I read that I went back to the original files and got into all details of the extent and specifics of the exposure and it became very clear to me that this exposure to PVC dust was very prolonged and sustained and heavy and at that stage I felt that I should dot the i's and cross the t's, and while I'm not willing to exclude contributory role of the wide range of his other exposures, that I wanted to dot the i's and cross the t's in relation to the specifics of VC/PVC exposure and laryngeal cancer.

Q. When did you receive Dr. Davidson's deposition?

A. I think I got it shortly after the depo, but exactly when I don't know. But I didn't really read it until after I got back from Europe, and at that stage I decided to go back to the original file and look up all the details on his exposure and these were factors which influenced me to prepare this additional material.

Q. When did you actually write the page

that we're talking about, the one entitled Factors Incriminating VC/PVC, et cetera?

A. Shortly after I got back I started going through these materials and making notes on all of this.

When I say all of this, I mean not just this page but these other four exhibits, and I made notes on an ongoing basis and I think that one of the -- the one on the epidemiology or clinical studies was finished about a week or so ago or two weeks ago, so these have all been completed in the course of over the last couple of weeks.

Q. At the time you prepared your preliminary report, what was your understanding as to Mr. Peterson's exposure to asbestos?

A. I knew that he had asbestos exposure, as indeed I indicated in the report.

Q. Where had you learned that?

From which document did you learn that?

A. I learned that from two pieces of information.

One, from his X-ray which made it very clear. He had an X-ray which made it clear he had calcification of his diaphragmatic pleural and other changes in the lungs which are pretty characteristic of asbestos exposure.

Q. And which could only come from asbestos exposure?

A. In my view, yes. I'm not prepared to exclude a contributory role in some of these from PVC dust, but I would refer to Dr. Velez in this area as his experience as the respiratory clinician, which I am not.

And the second is a little time ago, I'm afraid I can't say exactly when, I received a note from Mr. Levinson's office documenting the fact that he had asbestos exposure.

Q. Do you have that note somewhere in the notebook?

A. No, the information in the report incorporates the information that Mr. Levinson sent me on the asbestos exposure.

Q. When you say you received a note from Mr. Levinson or his office, was that in the form of a letter?

A. No, it was one or two pages of additional information on his occupational exposure. At one stage I had Mr. Levinson -- Mr. Levinson sent me a document or notes on his personal history and his occupational history and then at a later stage, which

starting at the very beginning you talk about having done a review of available documentation and records of Mr. Peterson.

May I assume, based on what was just gone through, that any of that documentation or record of Mr. Peterson is referred to in some fashion in the notebook?

A. Correct.

Q. Or at least the notebook contains your summary of information that you obtained from medical records supplied to you from Mr. Levinson?

A. Correct.

Q. You also indicate you reviewed the relevant scientific literature.

With regard to any scientific literature that you found to be relevant on the issue impacting on Mr. Peterson's case, would I be correct in assuming that you have referred to it in some fashion in your preliminary report?

A. Yes.

Q. So that if there is additional scientific literature that you have reviewed, but have not included, that would indicate that in your view it's not relevant to the Peterson matter?

A. I wouldn't say that. I'm not saying there may

not be other relative material which I haven't looked at.

Q. Not a question of what you have not looked at or not found, but if you had reviewed anything that you considered to not be relevant, you would not have included it, I presume, in the report; is that correct?

A. If it wasn't relevant?

Q. Yes, if it was not relevant.

A. You're quite correct.

Q. And for anything that you reviewed and was considered to you to be relevant, it would be referred to in your report?

A. Yes. In other words, there may well be, in fact, other references which are of importance which I just haven't discussed, because you'll notice that in the tables I talk about illustrative references, so it's not meant to be a totally exhaustive complete analysis of the total literature.

Q. When you get involved in a piece of litigation and have to determine what the relevant literature might be, how do you go about that? Do you use some computer service?

A. No.

Q. Do you do your own personal research?

A. I have my own files which I keep updated.

Q. Do you use Toxline or Medline or any of those computer services?

A. Once in awhile I will, but I go through journals on a regular basis and generally keep my files up-to-date.

If, indeed, my files on any particular subject are not complete, I might use Toxline or Medline but in this case I didn't.

Q. How do you keep your own files? Do you keep it by subject matter of chemical or substance or injury or just how?

A. By chemical and, in fact, I bought you a collection of my cards on VC/PVC.

Q. Is this a good example of how you keep your own files?

A. Yes, that's typical.

Q. Now, these are index cards, three by five index cards or thereabouts --

A. Yes.

Q. -- That you have collected over the course of time?

A. Yes.

Q. Would these three by five index cards have an indication of when you added the cards to

your collection?

A. No.

Q. It would simply have the name of the author of the study?

A. Precisely.

Q. And date of publication.

In summary fashion in this first paragraph on page one, Dr. Epstein, you've indicated that you have concluded that Mr. Peterson's occupational exposures at ATC was causally related to the laryngeal keratosis --

A. Correct.

Q. -- He developed in 1978 and the laryngeal cancer he subsequently developed in 1984. And the sentence goes on so, but that's the part that I'm interested in at the moment.

A. Yes.

Q. For my working definition, what is keratosis?

A. A keratosis is a premalignant condition involving hyperplasia, epithelial hyperplasia, and generally characterized by atypical cells in the mucosa.

Q. What is a hyperplasia?

A. It means an increase -- let me try to find the

easiest way of explaining it.

It's a multiplicative reaction increase in a number of cells, cell lining? Hyperplasia will be one of the factors in keratosis.

Q. Is there something in the medical records that indicates that he was diagnosed with such a condition in 1978?

A. Yes.

Q. Do you have a recollection as to where you obtained that information?

A. Medical records.

Q. Was it the hospital records?

A. Surely, yes.

Q. Is the laryngeal keratosis a procurance to laryngeal cancer?

A. Very often, yes.

Q. Based on what you have reviewed in this case, is it your view that the laryngeal keratosis in Mr. Peterson's situation was, in fact, a procurance to a laryngeal cancer?

A. Yes, correct.

Q. When you talk about his, I quote here, quote, "His occupational exposures at Amboy Terminaling Company", which exposures in particular are you referring to and did you mean to refer to at

the time you wrote the preliminary report that's been marked as the exhibit?

A. You'll find these listed under section number one.

Q. Section number one is the section that follows this paragraph?

A. It's the second paragraph, yes.

Q. It was your conclusion as expressed in the first paragraph that all of these occupational exposures combined in some fashion to be causally related to the laryngeal keratosis and laryngeal cancer?

A. I didn't make any attempt to rank them, I just said that his occupational exposures were causally related to his laryngeal cancer and then in the next paragraph I list what they are and then I also give evidence for the toxicity and carcinogenicity in the subsequent information, including their affect on the larynx and respiratory tract.

Q. Just to be specific, throughout your report you're talking about occupational exposures to polyvinyl chloride, vinyl chloride, monomer, asbestos polystyrene, Isopropilidene resin, which you also referred to earlier today as Bisphenol A, and also to the emissions from welding fumes?

A. You left out one or two but in principle that's okay.

Q. If I have --

A. Epichlorohydrin for instance.

Q. Epichlorohydrin, if I recall your report, you indicate the Epichlorohydrin came from the Isopropilidene resin?

A. Yes.

Q. So I meant to include that.

A. I'm sorry, I beg your pardon.

Q. Is there another that I left out?

A. Well, the welding fumes and the --

Q. I said that.

A. You did? I'm sorry, and the heat sealing fumes.

Q. The heat sealing fumes would be as I again read your report, the degradation of a PVC resin?

A. Degradations of any resins.

Q. Correct. Polystyrene?

A. Correct, yes.

Q. Would that then cover the gamit of all of the occupational exposures you were aware of at the time you wrote the report?

A. Yes.

Q. Since you wrote this report, are you aware of any other occupational exposures that Mr. Peterson had at ATC?

A. I don't think so, no.

Q. Is it your opinion that those occupational exposures, all of them that we have just listed, to be combined in some fashion to cause Mr. Peterson's laryngeal keratosis and his subsequent laryngeal cancer?

A. They all had a -- it's not possible to exclude a causal role or contributory role of any of these exposures.

In other words, all of these had carcinogenic affects on the respiratory tract and/or irritant affects on the respiratory tract, the importance of some of them more important than others, but it's not possible to exclude a contributory role of any of these individual exposures.

Q. What do you define as the respiratory tract, if you can tell me everything that would constitute a respiratory tract?

A. It starts from the nose, goes all the way down to the terminal alveoli.

Q. That's at the base of the lungs. I'm

not a physician so I may misstate it, but it's in the lung area, is it not?

A. Yes.

Q. What would it include between those two end points?

A. Includes the nose, nasopharynx, pharynx, larynx, trachea, bronchi, bronchials, alveoli and pleura.

Q. Are all of the individual exposures that you have listed, if you took each of them separately, is each of them a carcinogen?

A. Well, I have given you the specific of each of these.

Q. In your Appendix 2?

A. In the appendix. First of all, for each of the major categories in Appendix 1, there is a description of each of them and data on their carcinogenicity and that's under Appendix 1 in which it deals with each of these major exposures; and then there's a brief summary table in Appendix 2.

Q. Well, referring to either one of them or both of those appendices, if you wish, let me just ask a simple question again, as to whether or not in your opinion each of those occupational exposures that Mr. Peterson had was to a carcinogen?

A. Yes or to a group of carcinogens.

Q. Well, I'm breaking them down. If I took each individual exposure, each of them would be a carcinogen, I'm trying to stay away from a carcinogistic affect at the moment.

A. I understand, but when you come to something like the welding, there's groups of carcinogens there and the other point that I wanted to make was in some instances in addition to the carcinogenic affect of some of these exposures, there's a chronic toxic or chronic irritant affect which is discussed under each exposure under Appendix 1. Is that responsive?

Q. Sufficiently so.

A. Okay.

Q. What information do you have, Dr. Epstein, as to Mr. Peterson's specific exposure to PVC resins and the result of VCM fumes from that?

A. This is listed in great detail in the section marked OH, occupational history.

Q. Which I have not seen before today so I wouldn't know that.

A. Fine. So let me go over this and also the other. In addition, it's listed with even greater specificity under the heading ATC, okay? So as far as the PVC resins are concerned, there's three major

-- well, first of all, let me start further and talk about the PVC/VC resins at ATC.

The amounts of these resins that were received were the resins that arrived at ATC, from ETC in 50,000 pound van containers.

Q. Excuse me, what is ETC?

A. ATC, I beg your pardon. It arrived at ATC, Amboy Terminaling Company from UC, from Union Carbide in 50,000 pound van containers and also in smaller van boxes and the total shipments were about one and a half million pounds a day or four-hundred million pounds a year.

Q. Let me stop you, Dr. Epstein, at the risk of being slightly rude.

My question was, what specific exposures did Mr. Peterson have, not to the amounts of material shipped to ATC.

A. Okay, fine. I believe this is important background. I am going to come to the specifics of that because what I wanted to do is to give an overall indication as to the amount of resins handled at ATC by which Mr. Peterson's exposure would be a subset of that, so I was leading to that. If you just allow me to complete just 30 seconds more and I will get onto the specifics, if I may.

So in other words, the total shipments there were about one and a half million pounds a day or four-hundred million pounds a year at about 350 to 400 bags an hour were processed.

Now, as far as the PVC resins are concerned, we have to first of all discuss what resins he was exposed to.

There were three kinds of resins which were handled at ATC; the suspension; the bulk; and solution, and from '60 to '75, somewhere in the region of 30 percent of all the resins were suspension according to the records.

This is an interesting contrast to Dr. Wheeler's statement that '85 percent of all U.S. production was, in fact, suspension.

Be that as it is may, from before '75, according to Dr. Wheeler, the levels of the VC in the PVC were 860 ppm, although in his interrogatory he gives different figures. He says goes up to 2000 ppm, and there's other indications that they go up to 3,700 ppm. And after '75, the levels of the VC and the suspension resin were reduced and probably less than about 100 ppm.

The bulk resins which had lower VC levels and solution levels, solution resins.

As far as the PVC dust levels are concerned, all I can say is ATC and UC failed to monitor the air area so there's no available data on the levels of PVC.

Q. Let me hold you right there. Have you seen the Gollob laboratory reports indicating the levels of work space contamination?

A. Levels of what? Which are we talking about?

Q. VCM.

A. I wasn't discussing the VCM, I was discussing PVC.

Q. Are you aware of the Gollob results?

A. Yes.

Q. Were the Gollob laboratory reports provided to you?

A. Yes.

Q. Do you have them back in your office?

A. I think I do, yes. I think I kept the Gollob report, I don't recall any data on PVC dust levels.

Q. Do you recall what the Gollob laboratory reports did show?

A. Yes, VC, but I wasn't discussing VC, I was discussing PVC. I will come to the VC if you allow me to complete.

Q. Yes.

A. As far as the PVC dust levels, it's my understanding there were no data on PVC dust levels in the ATC Plant.

Q. You're talking about airborne?

A. Correct.

Q. Pardon me if extremely long narrative requires me to do that.

A. Please do. Please interrupt.

Q. You are talking about airborne dust?

A. Yes. Airborne dust levels, yes.

We do, however, have some, according to Wagoner, the levels of PVC dust are highest in the bagging areas. We also have some data from NIOSH.

Q. Excuse me, let me back up.

What information do you have from Dr. Wagoner?

A. That's a Wagoner publication which I refer to, Wagoner, et al 1980.

Q. It deals specifically with ATC levels in the bagging area?

A. I already said there is no data. I've already said there's no data on PVC dust levels in the ATC plant, so the Wagoner report, in general, the levels are highest in bagging areas, not specifically ATC. And so that's basically all our data on PVC dust

levels which is non-existent as far as ATC is concerned.

In coming to the atmospheric levels of vinyl chloride, let us discuss them under the following headings. First of all, the sources of vinyl chloride, then the data for prior to '75, the air level data prior to '75, and then the data in '76.

Q. Dr. Epstein, I'm going to stop you there because this has gone on at some length now. I don't think it's responsive to my initial question, which is the specific levels of exposure that Mr. Peterson had to either PVC or VCM, the cause of PVC.

Let me see if I can shortcut this a bit.

For the record, I will indicate that you've been referring to the sheet marked ATC that appears in the notebook, and therefore, I'm sure that all of this information can be found there?

A. Sure.

Q. Do you have any information that would allow you to conclude on a parts per million basis how much exposure Mr. Peterson had, let's take on an average workday at ATC to either PVC or VCM?

A. Well, first of all, we don't have any

monitoring specifically for Peterson, so all the information has to be inferential and this information is derived from two sources.

First of all, description of the work conditions, which I will go through with you first; and then I will go through with you, if you so wish, the information on the monitoring levels in the area he worked in from which one can withdraw inferences on his exposure.

Q. Well, let's work backwards, if we can.

Why don't you give me the bottom line in your opinion as to what the exposure level was for Mr. Peterson and then we can work back as to how you determined that.

A. As far as the PVC dust levels are concerned, and these were very high dust levels indeed, that's based on the description of his work conditions and and the statements, his description of work conditions, but also what I know from the Maliko case and what we also know from the Davidson report. And, in fact, you will find this specified in detail in the section in the notes marked Factors Incriminating VC/PVC, which I state he was continually exposed to very high levels of PVC dust with high concentrations of residual VC from 1967 to '73. That was the first

point.

Then he was continually exposed to -- shall we stay with PVC or?

Q. I don't mind going back and forth provided we have an indication of when we talk about which.

Hold that statement for a moment, if you would?

A. Sure.

Q. You indicated from that, the dust levels from PVC I think you said were high, very high. When you use the phrase very high, if you would place it in terms of parts per million, what number would you put it at?

A. Well, you don't express dust levels in terms of parts per million. I don't know, you generally express them in milligram cubic meter.

Q. All right, let's use that.

A. I would say they're certainly in the high milligram cubic meter over 20, perhaps in the 20 milligram per cubic meter range, in excess of that the fact is that his hair, his clothing was covered in white dust and he used to gag, so clearly they were very high levels of PVC in the dust in the area where he was working.

It's difficult, if not impossible, to give you a precise figure on this or even a general figure on this because of the failure of Union Carbide and ATC to do any monitoring of the dust even in spite of the very substantial literature going back to the forties on the toxicity of PVC dust, so I can't give you an answer to that unless indeed there's unpublished ATC/UC literature on this.

Q. Is it your understanding that the dust that Mr. Peterson was exposed to, the PVC dust was in the respirable range?

A. Some was respirable and some was not.

Q. Do you know what percent of each was respirable and was not?

A. I would say in general and, in fact, I discussed this in some detail in here, as far as the levels of concern, there is a conflict of data on this.

Dr. Wheeler maintains levels of the dust was 50 to 150 micron; however, Dr. Davidson did some analytic work and found that the levels go down to five micron, which is the bulk were in the range of 50 to 60 micron, but they go down to five micron. But I would also emphasize that the impression of respirability is relevance to lung pathology but not

relevance in relation to laryngeal pathology.

Q. Why is that?

A. Because large particles will reach the larynx, it's only the large particles which will be filtered out or stopped before reaching into the small bronchi and bronchi elements, the question of respirability of less relevance, if indeed any relevance in relation to laryngeal cancer.

Q. Is that because in your view it's important to note the amount of PVC dust that came to actual physical contact with the larynx?

A. Well, obviously every breath he took he would breathe in material that was in the air and this would be progressively filtered out as it goes, as it reached down into the lower respiratory tract and upper large particles would impact and particles over ten microns would certainly impact, but as you go down into the depth of the lung you would have to reach, you would only have to consider the respirable size particles, that's a less than ten micron, if you're considering pathology in the terminal lung.

Q. Doctor, you're not being responsive to my question. Is it because what you're concerned with determined how much of the PVC dust was in contact with the physical larynx, is that what you're

looking for?

A. I would say everything he would breathe in in the air would reach the larynx, yes.

Q. And some of it would be passed on depending on its size?

A. Sure, other smaller particles would certainly go down to the lung, particles under ten microns and certainly the five micron.

Q. You're not suggesting that the remainder would remain in contact with the larynx?

A. I'm not suggesting; stating the large particles would impact the larynx and laryngeal-pharyngeal area.

Q. What would happen to those particles that would contact the larynx?

A. The VC, vinyl chloride in the PVC dust would be deluted and produce loal (phonetic) affect on the larynx in the larynx-pharyngeal area.

Q. As you say you allotted those VCM fumes, what would happen to the resin particle, it would pass through the system?

A. The particle could remain there for sometime, it could be coughed up and disposed of in that way.

Q. Would that be the only way that the body has to dispose of that particle?

A. No, not at all. In addition to being coughed up, I think some of them could be swallowed and pass through the digestive system.

Q. Now, you've given me an approximate number in terms of the milligrams per, what is it cubic meter?

A. Cubic meter,, all I can say is the air was very dusty, the conditions were described as terrible and the language that he used is self-explanatory, terrible and very dusty, hair on skin and clothing was covered with white dust. I cannot tell you what levels were, but I would say they were in the high milligram cubic meter range.

Q. Which you earlier have stated to me would be somewhere in the 20 milligram per cubic meter area?

A. I would say minimally, because we do know in a PVC fabricating plant they would reach 20 milligram per cubic meter, I would not be surprised if they were in the 100 cubic area, but I have no idea because I'm unaware of any monitoring data.

Q. In fact, you also have no idea whether or not they were 20 milligrams per cubic meter from any data?

A. That's not correct. Indeed PVC fabricating

plant you would have levels going up to 90 milligrams per cubic meter in an area where PVC dust was bagged, one could say minimally it would be in excess of that.

Q. So it's your testimony now that Mr. Peterson was exposed to PVC dust in the range of approximately 20 milligrams per cubic meter or is it your testimony that it was higher?

A. My testimony is that we cannot give any estimate on this because of ATC and Union Carbide's failure to monitor, in spite of long standing knowledge of the state of the art and toxicology going back to the forties. That's number one.

Number two, if PVC fabricating plant levels could reach 20 milligrams per cubic meter in this plant where it was bagging, where conditions were quote "terrible and very dusty" where his skin and clothing were bad, minimally it would be 20 milligrams. I don't know, it could be 100 milligrams, it could be more, we don't know.

Q. I will move to have the first portion of the witness' answer stricken as unresponsive to the question.

If we look at the exposure to vinyl chloride monomer, VCM?

A. Yes.

Q. Do you have any ability to determine with some specificity as to what Mr. Peterson's exposure was to VCM on an average workday at Amboy Terminaling Company?

We may have to divide it into time periods.

A. Sure.

Q. Let's take perhaps from 1967 to 1974; is that a good time?

A. Or '75.

Q. All right, before '74/'75?

A. Okay, I would like to, without trying to be totally responsive to your question, before I answer that I have to specify what the particular source is of VC because you have to consider a wide range of different sources of exposure.

Q. I'm not sure I understand. Why don't you tell me what it is? How do you want to answer it? I will see if that's going to be a problem.

A. Fine.

We have to consider a wide range of different ways and sources of vinyl chloride exposure and attempt, as far as possible, to see what data there are relating to each of these. There may be

data, there are data relating to some of these sources of exposure, but no data relating to other sources of exposure; therefore, I'm not prepared to give you an overall figure.

What I will do is I will tell you what were the particular ways in which he was exposed to VC and for some of these I will attempt -- I will summarize the existing information on what these levels are.

Q. Let's start with that approach.

A. Okay.

The other point I would also make is that it is my view, and as I have stated in the documentation, that the level, that the PVC dust levels are of much greater importance in relation to his laryngeal cancer than the vinyl chloride levels.

Q. Why is that?

A. The evidence for that you will find summarized in the section marked Illustrative Literature On The Toxic And Carcinogenic Effects of VC/PVC --

Q. That's a document I have not seen before today.

A. -- Where you will find several studies dealing with PVC dust in which it's emphasized that the PVC dust is a particularly important simulation to

respiratory tract cancers and let me just now point you in the direction of two or three of them, then I will get back to the original question.

First of all, you will find a whole series of studies on PVC dust demonstrating respiratory tract pathology including cancer. Then in the paper by Wagoner & Infante, in their review they conclude that PVC dust showed a significant association with the respiratory cancers.

Waxweiler also says, "'PVC dust appeared to be the most likely etiologic agent -- No relationship (exists) between VC (air levels) and lung cancer', probably due to its rapid absorption into blood. And they emphasized the role slow elution VC from trapped PVC particles in the lung."

Wagoner in '83 says the same thing, "Emphasized carcinogenic role slow elution VC from trapped dust particulates in lung."

So in other words, the literature makes it very clear in that when you're dealing with lung cancer in relation to PVC/VC, it's the PVC dust levels in the PVC dust which is of much greater importance than the VC, but let me now be more responsive to your question as to the VC levels, having already stated that it's the role of the PVC

dust which is the far greater importance than the VC, nevertheless --

Q. Pause one second.

A. Then we're going to discuss three things, sources of the vinyl chloride, then vinyl chloride levels prior to '75, and vinyl chloride levels after '75.

Before getting into this I should refer you also to the Union Carbide Monitoring Report of 1974 which talks about high VC air levels from PVC resins, okay?

Q. Which monitoring report is that?

A. The one which I referred to?

Q. The paper that talked about monitoring?

A. The Union Carbide 1974 report on Monitoring The Concentration Of Vinyl Chloride.

Q. Fine.

A. Okay, now the sources of VC are as follows: First of all, VC absorbed on the resin itself, the surface of the resin; secondly, the VC, the high concentrations in storage areas, bins, bags, hopper cars which is stated in the UC Monitoring Report of 1974, and in the Gollob report of 1974, there's references to 1400 ppm in the van.

Q. In the van?

A. In the van, yes.

Q. Excuse me, do you have any indication that Mr. Peterson was ever in the van?

A. We're not discussing that testimony, what I said --

Q. Well, one of the nice things about being a lawyer is I get to ask the questions.

A. Excuse me, sir, you asked me the sources, we were discussing sources. I'm going to come on to --

Q. Yes, sir, and as I said earlier, it sometimes becomes very difficult if you allow an answer to go on at the length it is without asking a question at the appropriate time, so since I referred to the van, may I simply ask --

A. No, I have no information that he was ever in the van.

Q. Thank you.

A. I'm giving you, what I'm now listing are the sources of the VC without any reference to the specifics of his exposure, but we will come back to that after I've listed all the sources of the VC for you.

Q. Go ahead.

A. The third is degassing of VC from pellets and powder and also degassing of VC from PVC dust on his

clothes, on his skin and in his body in either the larynx or in the lung.

The next is displacement of air in the 50 pound bags as PVC was pushed into these bags air would be displaced and air contaminated with VC is displaced.

The next, fixed point emission sources.

Q. I don't understand that. Would you tell me what that is?

A. In other words, at each of the area points where the bags were filled, these are what you call a fixed point emission source where the dust was, where the PVC pellets were coming down the dust was coming down from each of these areas, and finally, from pyrolysis or thermal degradation from two sources, from the heat sealer and from welding.

Okay, now, those are all the sources of vinyl chloride to which exposure is possible, although, as you correctly point out Peterson would not have been exposed to some of these such as the ones in the bag.

Now, let us review briefly the data, what available data there are on VC levels prior to '75.

The most important point to be made is

there were no sampling data prior to 1974. In spite of the overwhelming literature on the toxicology and carcinogenesis of VC, both UC and ATC failed to sample the air prior to 1974, so therefore we have no information on air levels of VC prior to '74.

Q. Let me interrupt you there. What was the date of the Gollob laboratory air sampling that was done?

A. '74.

Q. In '74?

A. Correct.

Q. Are you aware of any differences in the ATC handling or processing of the resins that would account for a higher VC level prior to '74 or the date of the Gollob sampling tests?

A. First of all, I haven't addressed myself to that, but secondly, we don't have data on -- we do know that some of the earlier resins had higher VC levels than the later resins, so it may well be that there were higher VC levels in PVC prior to '74, I just don't know.

We do have other data showing that. For instance, there's a Morgan and Tacquard reference showing that VC levels extend up to 3,700 ppm, so I can't answer you on that.

Q. Well, just so the record is clear, that's a study that has nothing to do with ATC, it's a general study.

A. It has everything to do with ATC because in indeed the industry was manufacturing PVC resins with much higher VC levels, it's unlikely that Union Carbide would be any exception to that.

Q. Are you aware of whether Union Carbide was or was not an exception to that?

A. I don't know.

Q. Do you know the manufacturing process that Union Carbide used that --

A. I can't answer that. All I know is that there was no sampling data prior to '74, number one; number two, that resins prior to '74 had much higher levels.

I'm not talking about actually at the time when the process design change was made, but I'm talking about the earlier production that productions of PVC could well have had higher VC levels, I don't know the answer to that.

Q. In general, what is your understanding as to what changed in 1974 to account for lower levels of VC?

A. Well, belatedly Union Carbide introduced a

vacuum stripping technology to reduce the levels of residual VC in the PVC, Union Carbide, as I indicated before, had extensive knowledge of high levels of VC in PVC prior to that, but delayed doing so until after the B.F. Goodrich announcement of the angiosarcoma deaths and also the OSHA emergency temporary standard, so this was subsequent to those events. So moving on from the statement that there was no data prior to '74, we have to see what available data there are subsequent to '74.

Now, Wheeler's simulation -- I beg your pardon, Dr. Wheeler's simulation in modeling started of claims that there were levels 0.98 ppm during bagging. This is in contrast to Dr. Davidson's estimate of up to one order of magnitude higher; in other words, up to about ten --

Q. Approximately double?

A. No, order of magnitude is ten fold of about ten parts per million.

The Gollob analitycal data are as follows: -- We're going to briefly review Gollob analitycal data.

First of all, there are the irrelevant data as Mr. Hollingshead has pointed out of the 1400 parts per million levels in a van. Then there's

Gollob's data on three to four parts per million in the breathing zone for the bagging, three to four parts per million in the breathing zone in the bag room, and then there's the irrelevant data on 27 parts per million between the pallets in the warehouse where he didn't work, but these data ignore the following.

They ignore the role, first of all, of PVC dust, which we've already discussed in some detail, which has maximal relevance to the respiratory tract cancers.

It ignores the levels near the heat sealer where periodically during heat sealing you can have very high levels of exposure, and it also ignores levels from welding where during welding you could have sudden high levels.

It also ignores, of course, the role of a wide range of co-pollutants, the styrene and ozone, et cetera, which could have an interactive, or as you previously recognized, synergistic affect with the PVC with the VC. After '75 and '76, Gollob analytical data still finds more than one half of part per million in the bag room, but this again excludes the following: It excludes PVC dust, it excludes VC from welding and from sealing and from

the sealer exposures which according to Davidson were very high. That is the brief summary of the VC levels.

Q. And I take it that brings us to the end of your preliminary comments as you are leading up to my original question which was, what were the VC levels of exposure that Mr. Peterson had?

A. That's correct.

Q. Good, that sounds like a good time for a lunch break.

A. Great.

(whereupon, a lunch recess is taken. Time noted 1:00 p.m.)

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

(Time noted: 1:30 p.m.)

Q. Dr. Epstein, we're now back after the break and we will pick up with my question as to what were the VC levels of exposure that Mr. Peterson had while he was employed at ATC?

A. Again, I have still one more general thing, namely the source, the degassing of the VC from PVC, and to be specific in the answer, I have to go through in the same way as I've just gone through the data on atmospheric VC levels. I have to tell you what my position is on the VC exposures as a result

of degassing from PVC and the major types.

The first is VC will degas from PVC in the following ways: The residual or the sources of VC, the residual VC from the suspension resins, we've talked about the Wheeler data of 860 parts per million VC residual in suspension resins at Union Carbide, and about a week later about 565 parts per million.

Davidson emphasizes that the degassing is non-linear, so it's very rapid then flatens off subsequently. That's the first thing.

Now, the degassing of VC from PVC dust would occur from PVC dust in the air and on his clothing and on his skin. Gollob points out that degassing is continual and the exposure is continuing. Additionally there's likely to be very high loal concentrations of vinyl chloride in the body from very rapid elution in the body.

Q. When you say the body, what do you mean by the body?

A. Either lungs or larynx.

In other words, if you have a PVC particle or agglomerate, the VC would be eluted from it rapidly producing very high loal concentrations of vinyl chloride.

Additionally, you will have very high levels of VC reaching the hundreds part per million from thermal degradation. So when you ask me what are the levels of VC exposure, I can say it's impossible to give you an overall figure because of these very wide range of sources each without characteristic types of exposure.

One can talk about what the levels in the bagging area, the breathing into the bagging area are, but this will give you no indication whatsoever as to the VC levels from degassing air on his clothing, from degassing from elution in the body of PVC particles and also the massively high levels, loal levels from heating, from thermal degradation in relation to the sealer and the welder.

So the answer is a very wide range of levels of VC going up to the hundreds of parts per million and going down to levels which are found to have been recorded in the bagging room, the bagging area of the bagging room which I should point out is some 50 foot away and the sealers are some fifteen feet away from the baggers.

Q. What is your understanding of how Gollob went about performing their tests in 1974?

A. That data were largely on breathing zones of

the baggers.

Q. How do they accomplish that?

A. Personal sampling.

Q. Personal sampling by way of a personal monitoring?

A. Yes. My understanding is they also did some area monitoring, too.

Q. Stay with the personal monitoring for a moment.

That would be a personal monitor placed upon the bagging operator?

A. Yes, correct.

Q. For how long a period did they have it on the operator, do you know?

A. I forget. You can get those details from Davidson or refreshing your memory in the report.

Q. Did you take into account the Gollob personal monitoring results in the rendering of your report?

A. It's one additional piece of information which is of some help, clearly it's not directly relevant to Peterson because he wasn't a bagger and therefore it wouldn't be directly relevant, but in view of a failure of UC and ATC to do area sampling and area monitoring and monitoring of Peterson himself, one

has to take a look at all available information, some of which is more relevant, some which is less relevant than others.

Q. What is your understanding of what Mr. Peterson's position was at ATC?

A. Well, I thought we had discussed that, but I can briefly go over it again.

Q. I want an answer to the question in terms of what you understood his position to have been.

A. From what period of time are you talking about?

Q. Prior to the 1974 period.

A. He was a maintenance and repair mechanic.

Q. What was his position after '74?

A. Maintenance supervisor.

Q. Based on what you know about the ATC operation, due to your review of a considerable amount of information, were the concentration levels of VCM in the atmosphere likely to have been the highest on an average basis in the bagging operation or elsewhere?

A. Well, you have to define, with due respect, your question more clearly because the levels in the bagging area one can discuss or one can look at the

data on personal monitoring in the bagging area of VC. This will give you no indication as to levels inside the body from elution of vinyl chloride from PVC dust particles and agglomerates.

Second, it will give you no indication whatsoever as to VC levels resulting from thermal degradation due to pyrolysis and welding.

Additionally, it will give you no indication whatsoever as to the gradual and sustained long term degassing, continuing degassing, as Gollob pointed out, from clothing and skin and this kind of thing, so the bagging, the personal monitoring in the bagging area will merely give you one aspect of a fairly complex problem.

Q. But isn't the purpose of the personal monitoring that is done by an outfit such as Gollob intended to show what the level of a particular chemical is in the atmosphere in the breathing zone of the individual?

A. Precisely, but it won't tell you what is occurring, what is inside the body.

Q. Well --

A. Hang on one second. And it wouldn't tell you also what would happen a few feet away where there's sealing and where there's welding.

Q. In your attempting to determine what is in the breathing zone of the individual in the bagging room, would not the personal monitoring that's done particularly over an eight hour basis give you an accurate indicator of what the individual has breathed in, no matter where he is in that room during the course of the day?

A. Not at all.

First of all, you said eight hours, I don't know that it was eight hours, it may have been for a shorter period of time.

Q. My question was hypothetical.

A. I beg your pardon, I thought you were asserting it was eight hours.

Q. I am not. If we had gone over the course of eight hours, would that give an accurate indication of what that person breathed in in terms of that chemical during those eight hours?

A. That person, yes. But a person working a few feet away the answer is no, and even as far as that person is concerned, it would give you no indication as to what levels of VC his larynx or his lungs were exposed to because of the additional very rapid high level exposures from elution.

Q. Are you talking about the fact that the

individual may have digested or swallowed some of the resins?

A. Not an individual may have, any individual in the bagging area would inhale substantial amounts of PVC particulates and therefore there would be continuing elution of VC from the PVC in his body, in his or her body.

Q. Now, all of this I assume is preliminary to your conclusion that it's basically impossible for you to determine at this point in time what the exposure levels were that Mr. Peterson had during the course of his employment at ATC; is that fair?

A. What I would say is he was exposed on a continuing basis that particularly in that early period to very high levels of PVC dust and also to variable levels of vinyl chloride which on occasion, which on substantial occasions would have been very high from these, considering these multiple and separate types of exposure.

Q. Now, over the course of the last half hour we were talking about PVC then VCM, let's go to asbestos for a moment.

A. Sure.

Q. Do you have any indication from any of the information available to you as to what Mr.

Peterson's level of exposure was to asbestos?

A. Well, again, the answer is no. And the reason for this is the failure of UC to supervise work practices at ATC and the failure of ATC to have undertaken any monitoring.

Now, having said that one can say that he clearly was exposed to asbestos, the evidence of this is coming from two sources.

One, the radiological changes in his lungs; and two, the description of the work practices.

Q. What description of the work practices are you referring to that gives you the indication that he was exposed to asbestos?

A. Well, I'm told by information perceived from Mr. Levinson's office that, and also the Davidson depo that he repaired brake linings, but this was quote "not a frequent job".

Q. Now, whose quote is that?

A. That was Davidson's quote, "Not a frequent job".

Q. Let me rephrase my question.

Do you have any information that comes from Mr. Peterson himself as to what his history of exposure to asbestos at ATC was?

A. Yes.

Q. What?

A. That he changed brake shoes in large carriers.

Q. Where do you get that information from?

A. From Mr. Levinson's office.

Q. What document or other piece of information provided you with such information?

A. You may recall you asked me this morning, and I said that in addition to the original documents, he sent me a personal list on occupational history. He subsequently sent me a one or two-page statement dealing with his exposures to asbestos.

Q. Do you recall when you received that?

A. I think sometime in the late summer but certainly before I wrote my preliminary report.

Q. If I recall what you said earlier, you don't have a copy of that document at this time, correct?

A. Correct. As I've mentioned on several occasions, all the information I have got I have abstracted and summarized and this information is contained in my files.

MR. HOLLINGSHEAD: Mr. Levinson, in light of the fact that Dr. Epstein did not apparently retain at least a sizeable amount of the information

provided by your office, I would ask you if you could tell me exactly what it is that he received.

You may or may not be able to, but I am specifically concerned with this document from your office that would indicate that Mr. Peterson had exposure to asbestos at the time of his employment at ATC.

MR. LEVINSON: Sure.

MR. HOLLINGSHEAD: Do you recall that document?

MR. LEVINSON: Yes, the first document to which reference has been made is the narrative, the history given to me by Peterson in great detail.

MR. HOLLINGSHEAD: Which you supplied to Dr. Epstein?

MR. LEVINSON: Right. In that history he did tell us that the first six months he was employed by OTD his job was maintenance mechanics, and in that job he would put brake linings on these large carriers and picked up these 20 ton containers of PVC and would, in turn, dump them into the hopper which went into the bagging room. That's where that came from. But I will provide you with it.

MR. HOLLINGSHEAD: I would ask for a copy of that because Dr. Epstein clearly has referred

to it in his report.

MR. LEVINSON: I have a copy of that at the office, I will make a note of that.

MR. HOLLINGSHEAD: Thank you.

Q. Dr. Epstein, did you read the deposition of Mr. Peterson?

A. With difficulty. The copy I got was extraordinarily illegible. I asked Alfred if he had a better copy and I did go through it.

Q. Based on what you could read, do you recall his testimony at all with regard to the issue of asbestos?

A. To be quite frank, I can't recall.

Q. Your memory is correct because I can tell you that there is no reference in his deposition to asbestos exposure.

A. The thing I say that triggered me on this was the radiological report.

Q. That's the one by Dr. Velez?

A. No, before that.

Q. Oh, I'm sorry.

A. No. In April '85, the calcified right diaphram, that's atypical asbestos pathology.

Q. So your information on this issue, that is the exposure asbestos came from the narrative

supplied by Mr. Levinson's office, as well as your review of that radiological report and additionally something in Dr. Davidson's deposition I think you said?

A. Davidson, two other things Davidson talks about, that is repairing the brake linings wasn't a frequent job and Velez also refers to the asbestos, Dr. Velez.

Q. Those are the areas or the sources for your information on asbestos?

A. I haven't quite finished his asbestos exposure.

Q. Go ahead.

A. There were two types of asbestos exposure; one was the changing in the brake shoes, which wasn't a frequent job in the first six months, but clearly was enough to produce the radiological changes; and secondly, there was a very, very minor additional exposure, he assisted the boiler workers about once a year or so in replacing the insulation of the doors with asbestos rope and that was once a year, '63 to '73.

Q. Is it your opinion that the asbestos exposure, as you understand it to be, could have caused his laryngeal keratosis and subsequent

laryngeal cancer in the absence of any other chemical to which he might have been exposed?

A. Yes.

Q. Is there literature support for that opinion, and I would refer you now to your appendix on asbestos, which is Appendix 1, and I realize you put some of the articles and studies in here, so I would refer you to that when I ask you this question, and I will repeat it; if you could point me to the literature support that would specifically conclude that?

A. It is summarized in that particular appendix, the studies on asbestos in relation to laryngeal cancer.

Q. All right. Before we get into that, allow me to ask whether or not there are different forms of asbestos, to your knowledge?

A. Yes, I don't know what form of asbestos he was exposed to.

Q. You anticipated my next question.

A. I have no idea at all.

Q. All right, would it make a difference in your opinion and in your review of the literature as to which form of asbestos he was exposed to?

A. All asbestos is carcinogenic.

Q. Have you had any experience with asbestos in litigation before this?

A. Yes. With due respect, I think you asked me this.

Q. I may have. I also may have forgotten.

A. There was one case, laryngeal cancer which I mentioned to you earlier this morning.

In addition to that I think I had one in relation, one case. I've done very little asbestos litigation but there was one I believe on an atypical mesothelioma. I think I've only had one or two, maybe three.

Q. Based on the information available to you, do you have a recollection or a reaction as to when Mr. Peterson was last exposed to asbestos in his employment at ATC?

A. I would say the last substantial or the last significant exposure would have been in '67 to '68.

Q. And is that because at that point he changed that particular job?

A. No, because according to the information that I got, the changing of the brake shoes, which was not a frequent job, was in the first six months '67 to '68.

Q. Is there a latency period for laryngeal

cancer, an average latency period from exposure to the carcinogenic agent until the actual manifestation of the cancer?

A. Well, it's really impossible to answer that because latency will depend on the intensity of the exposure, the duration of the exposure, the age of the first exposure on a very, very wide range of cancers, very wide range of factors and also particularly individual carcinogens tend to have different patterns, so it's really not possible to answer that.

The only real way one can -- the only kind of relevant information in this is in relation to data on cessation of exposure, analysis of risk factors in relation to cessation of exposure, and we have reasonable data on that when it comes to smoking because that is an example of a carcinogenic exposure which at relatively early stages for respiratory tract cancers can be reversible, so when it comes to smoking, one can discuss this in relation to specifics of latency.

It's really not directly relevant but it's as close as I can come to answering your question.

Q. You mention in your Appendix 1, is it

Vigliani et al?

A. Yes.

Q. Studies in 1965 with regard to asbestos workers with respiratory tract cancers?

A. Yes.

Q. Do I understand you correctly that of the 19 asbestos workers one of them had laryngeal cancer, but all of them had general respiratory tract cancers, and I'm referring you to the top of Appendix 1.

A. They had different kinds of respiratory tract cancer.

Q. All of them?

A. Yes. To the best of my recollection some of them were bronchial carcinomas and others were mesotheliomas, but I can't recall the relative distribution of these.

One of these additionally, besides his respiratory tract cancer, also had a laryngeal cancer, too.

Q. If you know, what is the general rate of laryngeal cancer in the population at large?

A. In males it's about 8.5 per 100,000.

Q. Would that be considered a rare cancer or a frequent cancer or something in the midst?

A. I consider that an uncommon cancer, but incidentally, this figure of eight and a half per 100,000, you're bringing up a very interesting point because the age distribution, because that's an overall in the total population. Of course the -- laryngeal cancer is relatively uncommon under the age of 60.

Q. So the 8.5 out of 100,000 would take into account both those persons under 60 as well as over?

A. It's an overall figure.

Q. So the suggestion is at above the age of 60 it is not quite uncommon?

A. It's not a suggestion, it's a fact. It's an uncommon cancer, laryngeal cancer, but it's much more common after the age of 60 than under the age of 60.

Q. Is there any significance to be drawn from the fact that one out of 19 asbestos workers with respiratory tract cancers have laryngeal cancer? Put differently, is that consistent with the general population or is that considerably higher than the general population figure?

A. It could have been a fluke, it's difficult to answer that. On the other hand, it's a pointer, that's the reason why I mentioned it there because it

was an interesting pointer of literature that subsequently was confirmatory, had I just seen that by itself and had that been all the information there was, one may not necessarily have ascribed a high degree of relevance to that.

Q. Was the Vigliani study what is known as an epidemiological study?

A. Well, it's an epidemiology study in the sense there were a series of case reports.

In other words, it was an analysis of asbestos workers with respiratory tract cancer and among them there was one case of laryngeal cancer.

Q. Does the author draw a conclusion at all with regards to the relationship between asbestos and laryngeal cancer in that report?

A. To be quite frank, I've forgotten.

Q. Do you have it as part of your notebook?

A. If I have it here I will show you. I don't think I have.

No, I haven't.

Q. Is there any indication in the report to your recollection as to the frequency deration or extent of exposure to asbestos of these workers?

A. I don't recall.

Q. With regard to the Stell & McGill study

which is referred to in Appendix 1, what do you recall about that particular study?

A. This was an analysis of cases of laryngeal cancer. It was a fairly brief report and it was noted that of the cases of laryngeal cancer, in about 17 of them there was a history of asbestos exposure.

Q. Specifically there were 59 cases of laryngeal cancer that were studied by the authors?

A. Correct, and this was statistically signified.

Q. Was this also a series of case reports?

A. Actually this was just an analysis of 59 cases of laryngeal cancer and in each of these they looked into occupational history and just reported this. It wasn't a formal epidemiological on a cases total study, but it was just a brief report, almost like a preliminary report.

Q. To your recollection, did the authors draw any conclusion as to a connection between asbestos and laryngeal cancer with the study?

A. I'm pretty sure they did. I may have it here.

Yes, I do have it here.

Q. Are you referring now to the back of your notebook?

A. Yes, the Stell & McGill study. It was exposure to asbestos is most likely to be of

importance in laryngeal cancer in a more deep study, and a more detailed study of this group is being undertaken.

Q. Does that result of the Stell & McGill b study that is reported in your appendix?

A. Correct.

Q. I take it they followed up at a subsequent point and came up with another study, that's what that study indicates?

A. That was the 1973 b study and what they found there, a highly significant association between occupational exposure and to asbestos and laryngeal cancer. There was one problem about this study, they noted that there were more smokers among the patients with laryngeal cancer than in the controls, so that makes this study somewhat difficult to interpret because if you're smoking there's an additional factor, but nevertheless the study was in general consistent. I would regard it as consistent with other studies in demonstrating cause and associations between laryngeal cancer and asbestos exposure.

Q. Can you tell from the article or copy of the study in your notebook as to what the frequency duration or extent of exposure was, or is it not noted?

A. I'm pretty sure it was not noted.

Q. Was it your understanding that these were workers? By that I mean the people who studied in the Stell & McGill studies, these were workers in the asbestos industry?

A. Yes.

Q. In the manufacturing end of the asbestos industry, do you know?

A. I can't recall.

Q. Does the study indicate what exactly the higher percentage of non-smokers was among the controls or put in the reverse, what the percentage of smokers within the group of workers with laryngeal cancer was?

A. I can't recall, I'm sorry.

Q. Did the study itself indicate that?

A. It may give data, I can't recall whether it does, but the study criticized itself or rather recognized the limitations while stating that the results showed cause and association, appear to show cause and association, it recognized that as a limitation, the fact that there were more non-smokers in the controls than there were in the test group.

Q. My question, if it wasn't clear though, was just asking whether or not there is a number

given for the greater percentage of smokers?

A. I'm sorry, I don't recall.

Q. Well, the study is right here, does it have it?

A. I don't have that here.

Q. You're referring to the b study which you don't have a copy of?

A. Well, I have -- all that I have for the b study is just a very, very brief summary. For instance, I say the patients' smoking habits were similar with respect to the exposure to asbestos, but I don't have the exact figures. In fact, I don't have any figures at all for that.

Q. Does the study, in any way, in your view support the proposition that laryngeal cancer can also be related to smoking cigarettes?

A. There's no question.

Q. That can be related?

A. Of course, there's a clear literature on smoking and laryngeal cancer.

Q. Can you cite that literature anywhere in your preliminary report?

A. I recognize this, sure, I'm certain I do. In the preliminary report look at page two. Excuse me, do you have page two of the preliminary report?

Q. Yes.
A. Under item number six?
Q. Yes, but there's no studies listed there. The U.S. Surgeon General report?
A. That's right. You'll find in the section of larynx cancer several studies on the relationship between smoking and laryngeal cancer.
Q. While we're on the subject, why don't we turn to that and you can point out the studies for me.
A. Well, I will lead you.
Q. I'm sorry, you're referring to the preliminary report?
A. Sure.
Q. Let's turn to the preliminary report and if you would go to any section in here that talks about smoking which cites particular studies?
A. Any section in where?
Q. In the preliminary report.
A. There isn't, just a statement that they're recognized risk factors.
Q. What page are you on?
A. Page two, "While alcohol and smoking, particularly in combination and at high exposure levels are recognized risk factors" -- in other

words, this is well recognized in the literature.

Q. Can you give me an indication in the literature where I can find that if I wish to go read that?

A. Sure, you will find, first of all, at the beginning of the section marked "Larynx Cancer", there's a heading marked "Lifestyle".

Q. You're now referring to the notebook, not the preliminary report?

A. That's correct. And then it says tobacco and alcohol, it gives a few references and then as you go along through this, you will find about half a dozen papers on larynx and smoking.

Q. So there are references within your notebook that I can look at?

A. Sure.

Q. Can you tell me, I think it's apical?

A. It means apex.

Q. Pulmonary fibrosis is?

A. It means at the apex of the lungs.

Q. You've indicated on Appendix 1 under the asbestos section, at the end of first paragraph that, "It should be further noted that apical pulmonary fibrosis, considered to be unrelated to the asbestos exposure, was noted in 5/31 laryngeal cancers;

pulmonary asbestosis was noted in conjunction with one of these cancers."

Can you tell me what the significance of that statement is in the context of the Peterson case?

A. I don't really think there is any necessary association. It just happened they found in five of the laryngeal cancers there was apical pulmonary fibrosis and it was considered to have nothing to do with the asbestos exposure.

The only reason why I mention it, it was just an association idea of concepts because Peterson prior to his coming to work at ATC was noted to have minimal apical pulmonary fibrosis, but the authors of this, the Stell & McGill people say they thought it had absolutely nothing to do with it, with his laryngeal cancer; however, one of the laryngeal cancers, one of these have pulmonary asbestosis.

Q. Does Mr. Peterson show that he has pulmonary asbestosis?

A. Yes.

Q. Do you distinguish between pulmonary asbestosis and chronic obstructive lung disease?

A. Well, pulmonary asbestosis is generally more restrictive but you can have obstructive lung disease

in it.

Q. When you talk about chronic obstructive lung disease in your preliminary report?

A. Yes.

Q. Which you do specifically in paragraph one on page one.

A. Yes.

Q. Do you intend to mean that that is different from pulmonary asbestosis which you use as a different term in different locations in the report?

A. Well, with pulmonary asbestosis you don't necessarily have chronic lung disease.

Q. You mean you don't necessarily have them at the same time?

A. Yes.

Q. One does not necessitate the other?

A. Yes. The Velez report makes it very clear that he had chronic obstructive pulmonary disease.

Q. Perhaps you just threw me again. If he has chronic obstructive lung disease, would that be a separate illness from either chronic obstructive pulmonary disease or pulmonary asbestosis?

A. I must apologize, chronic obstructive lung disease is the same as chronic pulmonary disease.

Pulmonary asbestosis is from the point of view of lung function tests, which Dr. Velez is the expert and I'm not, is more of a restrictive lung disease.

So he had, in fact, two types of lung problems; one, the chronic obstructive lung pulmonary disease or lung disease and also restrictive disease characteristic of pulmonary asbestosis.

Q. Is it your opinion chronic obstructive lung disease can be caused by exposure to asbestos without regard to any other chemical exposures?

A. Well, it's more in terms of the restrictive, but you can't have chronic obstructive lung disease --

Q. Would you necessarily have chronic obstructive lung disease as a precursor to pulmonary asbestosis?

A. No.

Q. You can't have pulmonary asbestosis without having chronic obstructive lung disease?

A. That's my understanding, but I would refer to Dr. Velez.

Q. But it would be your understanding and your opinion that pulmonary asbestosis is caused by asbestos exposure?

A. By definition.

Q. One would think?

A. Yes.

Q. That we can agree on?

A. I thought we've agreed on virtually everything.

Q. Probably.

Going back to a question or series of questions I posed to you just a little while ago about the cigarette smoking, looking at your Appendix 1, I note and I want to correct the record if that's necessary, that you do have a reference to cigarette smoking being associated with laryngeal cancer, it's the Shettigara and Morgan report of 1975, am I reading that correctly?

A. Sure, but that was part and parcel, that really wasn't a study on cigarette smoking per se, it was a study on asbestos exposure in which smoking was considered, so this isn't a definitive reference on smoking.

Q. There there was no reference in the preliminary report?

A. Yes, there was no reference to study of smoking, this was purely experimental on the study of asbestos.

Q. Is it your opinion that to a reasonable degree of medical probability that Mr. Peterson's exposure to asbestos during his employment at OTD and ATC has placed him at excess risk of future disease and cancers?

A. It's one of the factors, sure.

Q. When you say with regard to the relationship between asbestos and an increased risk or excess risk of future disease and cancers, are there any particular future diseases that you have in mind or particular future cancers that you have in mind?

A. With relation to what, asbestos?

Q. Yes.

A. Yes, bearing in mind that I say asbestos is only one and not necessarily the most important one, but restricting your response just to asbestos, the cancers which one would have to consider would be the following: Would be bronchial cancer or lung cancer, firstly; secondly, would be mesothelioma; and thirdly, would be gastrointestinal cancer, particularly a colorectal cancer, there are some other rarer cancers associated with asbestos, but those are the ones which I would have particular concern for only with relation to the asbestos.

Q. Now, the listing you just gave me, they're all cancers, if I heard you correctly?

A. Correct.

Q. Are there any other diseases other than cancer that he has in excess risk of contracting?

A. From specifically the asbestos exposure?

Q. Yes.

A. Sure. From any -- when you have pulmonary fibrosis and asbestosis, you can have, first of all, an increased susceptibility to infection and therefore pneumonia, and also you can have cardiovascular strains which can result in addition, that's called cor, C-o-r, pulmonale P-u-l-m-o-n-a-l-e, which is an essentially right heart failure, as the lung, as you have fibrosis and scarring in the lung it becomes more difficult to pump blood through the lungs from the pulmonary artery, so the right heart can gradually enlarge and you can get right heart failure?

MR. HOLLINGSHEAD: I have to take a very short break.

(Recess is taken.)

Q. With regard to the cancers that you indicated, the mesothelioma is a cancer that is linked specifically with asbestos, is it not?

A. Well, actually there's two kinds of mesothelioma, there's a plueral of mesothelioma, that's a lining of the lung and peritoneal lining of the abdomen, both of them to all intents and purposes are exclusively related to asbestos exposure.

Q. So regardless of any other exposure that Mr. Peterson had, the future risk for these cancers could only be associated with asbestos exposure, am I correct?

A. With relation to these mesotheliomas?

Q. Yes, the mesotheliomas could only be related to asbestos exposure?

A. Right.

Q. I may have read your report incorrectly in this regard, but I thought you also suggested that the colon or rectal cancers would also primarily be a result or a possible result of under the exposure to asbestos?

A. I didn't say that at all. What I said was they are risk factors to colorectal cancer.

Q. Well, you've indicated on page one of your report at the bottom, and I'm not quoting the entire section, but you say that there is a risk of pulmonary mesothelioma and colon cancer from asbestos?

A. Correct.

Q. Now, that is from there that I had the impression that you were saying the potential future risk of colon cancer was a result of only asbestos exposure, have I misread it?

A. Not an unreasonable interpretation, but it's not actually correct because I go on to say, also cancers at a wide range of other sites, and the reason for that being is because the VC/PVC, vinylchloride produces a very, very wide range of which gastrointestinal tract happens to be one, I don't specify all of them, though, I just say a very wide range of sites.

Q. Do you have an opinion as to the particular likelihood that Mr. Peterson will contract a mesothelioma in the future from asbestos exposure?

A. I would defer to Dr. Velez on this, he's much more experienced in the asbestos area than I am.

I believe it's generally a five-fold risk, something of that kind, but this isn't my area I wouldn't profess to on a high level of expertise.

Q. I understand that and I accept it. I want to ask you what you mean by five fold?

A. In other words, his risk of lung cancer is increased five fold that of the general population.

Q. Five fold, do you mean five times as great?

A. Yes, five times greater than that, again, I defer to Dr. Velez on this.

Q. Just speaking of this issue in general, what is the general risk that any of us run that we will contract cancer in our lifetime?

A. Basically on an overall basis one in three chances, and of contracting and diagnosis, one in four.

Q. If I understand you correctly, it's one out of three in terms of the risk of contracting cancer and one out of four of contracting and diagnosing?

A. Correct.

Q. Now, that would be with regard to all varieties of cancer?

A. That's the total U.S. population, that's an overall figure for cancer incidents, excluding skin cancer of incidents and mortality.

Q. Why does it exclude skin cancer?

A. Just the quirk of the way it's reported, because there's a pretty wide range of skin cancers, the reporting for skin cancer is much less good.

In other words, somebody could have a

little bit of nodule on the skin which can be excised and not be reported. Generally, when you talk about overall incidents and overall mortality, you generally relate this to cancers and you say it's almost like a religious caveat. When it comes to mortality, of course, that would be a different matter, but when it comes to incidents, one would be screwing up the data by including skin cancer.

Q. Is it generally considered by doctors that skin cancer is more prevalent than other forms of cancer, as well?

A. It depends exactly where you are, because in the south you can have, particularly where there's a lot of sun, premalignant changes, keratosis of the skin, skin cancer is much more common, so as I say the overall figure is one in three and one in four, I'm excluding skin.

Q. Now, when you said a moment ago, recognizing that you defer to Dr. Velez, the pulmonary and asbestos expert in the case, when you said a moment ago that you would anticipate that Mr. Peterson's excess risk of contracting mesothelioma is perhaps five fold, are you talking five fold that normal risk of anyone contracting cancer?

A. No, excuse me --

Q. One in three?

A. I may have misspoken. I think what really I should have said would be his risks of contracting malignant disease of the lung, somebody with asbestosis has about a five fold increase the risk of getting a lung malignancy which includes a bronchial carcinoma and mesothelioma.

Q. Well, I'm not sure you misspoke, because my question was specifically geared to mesothelioma, I did not open it up to --

A. I beg your pardon, essentially what I was talking about, what I should have been talking about was malignant lung disease. In other words, if you have asbestosis your chances of getting malignant lung disease is somewhere, what, in the region of five fold.

The reason one has to put it this way is because the general population has no reason at all in getting mesothelioma, so it would be a meaningless statement when it comes to mesothelioma. What I was talking about was malignant lung disease, I'm sorry. I really expressed very clumsily.

Q. Well, if we used that understanding that you're talking about malignant lung disease, when you say that someone might have a five-fold risk factor,

are you saying that that is a risk factor above the standard one in three risk factor that the general population has?

A. No, the one in three is an overall for all cancers.

Q. Yes, I'm just trying to understand what you're saying.

A. Sure. If you look at the general population, what are the chances of the general population getting lung cancer, and then the chances if you have asbestosis, it is a five-fold risk. But again, as I emphasize, I defer -- what I'm saying is, if you have asbestosis there is a substantial increased risk of getting malignant disease of the lung. More than that I would wish to defer the specifics to Dr. Velez.

Q. I won't press too hard, but of the two people involved in this deposition testimony, you have a little more information than I do, so let me probe one second longer.

Do you have a percentage figure in mind for the risk that anyone runs of contracting lung cancer in his or her lifetime?

A. Well, it depends whether they're smokers, of course, and you have to break it down to smokers or a

smoker can have a ten percent, it's very difficult to say this because all you can relate is that the amount of smoking and deration of smoking, but say you have all of these factors, you can say roughly how many people get lung cancer every year, let's say it's 100,000 lung cancer deaths and 400,000 or say 450,000 overall deaths, so one can say a little more than a quarter of all cancer deaths are due to smoking -- more than a quarter of all cancer deaths are due to lung cancer.

Q. Is this another area that you would feel more comfortable deferring to Dr. Velez?

A. Which area?

Q. The discussion of increased risk on lung cancer?

A. In smoking?

Q. In smoking or --

A. No, I'm fine as far as smoking is concerned, it's in relation to the asbestos that I would defer to him.

Q. Let's go to the appendix on vinyl chloride and PVC which I believe is 2.

A. Yes.

Q. Do you have the Harris report with you in your notebook?

A. Which was that?

Q. Harris, 1953, referring to VC levels ranging up to 8,000 ppm?

A. No.

Q. Do you have a recollection as to which particular form of PVC Harris was referring to?

A. I don't recall. My guess is it must have been suspension.

Q. Why is that?

A. Because that's the highest. In general those are the highest levels. I mean that's the resin for the highest levels.

Q. Do you know how much residual vinyl chloride is contained in the various manufacturing methods that were used in the resin that was shipped to ATC?

A. How much?

Q. At the time of manufacture?

A. I thought we discussed this. According to Wheeler it's 860 ppm.

Q. For which?

A. For the suspension. Although, I've indicated that according to the literature much higher levels of VC are recorded than of course in the bulk and in the solutions there are lower levels.

Q. Do you have a number in mind to the bulk?

A. Yes, about five to 15.

Q. Five to 15?

A. Five to 15 ppm.

Q. And for the solution?

A. About one ppm.

Q. That's at the time of manufacture, correct?

A. Correct, although not necessarily being the case from '67 to '73.

Q. I'm sorry, why is that again?

A. Because I have no data on these as produced at individual years.

Q. Doesn't Dr. Wheeler give both prior and post '74?

A. Yes, but I don't recall whether he gives them for each of these years independently.

The other one point I should mention is that there's an inconsistency in Wheeler's statement. He states that the levels were 860 ppm in the Wheeler versus Maliko, but he changes his mind when it comes to the interrogatories where it says they go up to 2,000 ppm.

Q. This is for the suspension?

A. So you pay your money and you take your choice.

Yes, suspension.

Q. For each of the three systems of manufacturing, that is the solution process, the bulk process and suspension process?

A. Not in the solution.

Q. I said solution, bulk.

A. I beg your pardon.

Q. For each of those three processes, are you aware as to whether or not after manufacture, the residual vinyl chloride monomer, what's the word I want, dissipates in some fashion from the resin?

A. Sure.

Q. Do you know the rate at which it dissipates for each of the resins?

A. Well, you recall you had an extensive discussion with Dr. Davidson.

Q. I do entirely but this is your deposition.

A. I thought it might be helpful to refresh your memory.

Q. My memory is quite refreshed.

A. Okay, it's fairly rapid degassing.

Q. For all three?

A. Well, for everything it's fairly rapid, it depends on a wide range of considerations. It depends on the initial concentration. It depends on the initial concentration, blastosizes (phonetic), the size of the resin particle, and a whole series of factors of this kind. But in general, one can say there's a non-linear degassing, in other words, at high, particularly at high concentrations, degassing is very rapid initially then tapers off. And Wheeler, for instance, said that there was 860 initially at manufacture, by the time it reaches the plant, reached ATC, it was somewhere down to 580 or whatever it was, something like that.

Q. In rendering your preliminary report, did you have any particular number in mind for any of the three varieties of PVC resin for the amount of residual vinyl chloride monomer that was in existence at the time that Mr. Peterson was exposed to the resin?

A. Well, I just knew that there were high levels of residual PVC in suspension.

Q. You said PVC?

A. VC in the suspension resin and much lower in the others. Then, of course, when it comes to his VC exposures, I indicated to you this is a reflection

not only of the VC levels in the air but also VC levels from inhaled or ingested PVC, plus other sources like thermal degradation over and over and above that, as I indicated before the PVC itself considered to be greater importance than the VC.

Q. Well, let me break the three systems, the three processes down for purposes of the question.

With regard to the solution process, PVC which you indicated a few moments ago had a RVCM content of approximately one ppm at the time of manufacture?

A. According to Dr. Wheeler, yes.

Q. I assume you're accepting that testimony, are you not?

A. I really have no way of knowing whether this was the case for all the years, but let us assume that for a moment.

Q. Do you have any information from anyone, Dr. Davidson or anyone else, that would suggest that that number one ppm as RVCM number is wrong?

A. He may well be correct, but I have to have a certain degree of caution because as I've indicated to you before, Dr. Wheeler has seemed to have changed his mind over a ten-year period as to what the levels

of VC were in the suspension PVC, and furthermore, there are other references on high levels, much higher levels of residual VC and PVC than Dr. Wheeler has suggested at any stage, so I really just have to keep an open mind on this.

Q. Have you not done any independent review of literature on PVC manufacturer in the preparation of the report in this case?

A. No.

Q. Did you do such an independent review of literature prior to the rendering of your report in the Malika case ten years ago?

A. No, I just refer to the fact in the literature, there are reports of much higher levels of residual VC than Dr. Wheeler has stated.

Q. In the solution?

A. Excuse me, but what I was going to say, I leave the details of this, I rely on the details of this on Dr. Davidson.

Q. Well, before I leave the area, is it your testimony that you are aware that in the general literature that higher numbers than one ppm are listed for the RVCM contents of the solution process resin?

A. No, my discussion in relation to high levels

related to suspension.

Q. Good, that's where we started. Let's go back to solution process resin, you're accepting Dr. Wheeler's analysis that there is 1 ppm RVCM in the solution process for the purpose of rendering your report in this case, are you not?

A. No, I'm not. I have a degree of caution in accepting that in view of the disparity of the levels, in the different levels he has reported for suspension on two occasions, and it's fact that the literature has reported much higher levels for suspension. Now, because of these disparities I have to have an open mind on solution, but I have not investigated the matter and it's not my particular area of expertise and I really can't go any further than that.

Q. In particular, you don't have any information as you sit here today that would specifically tell you that Dr. Wheeler's conclusion with regard to the RVCM content in the solution resin is wrong, do you?

A. But I have information in relation to other resins, therefore I have to approach all of his statements on VC residual levels with caution.

Q. But you do not have any other

information with regard to this solution?

A. Correct.

Q. Assume that the RVCM content of the solution process resin at the time of the manufacture is 1 ppm, have you utilized any particular assumption as to what the RVCM level for solution resin was at the time Mr. Peterson was exposed to it?

A. I would say it would be pretty similar.

Q. Around one?

A. Around about.

Q. Is there a particular reason for that? Does that follow the explanation you gave earlier that tends to leave the resin in a linear fashion and then after a period of time it takes longer for that last element to leave?

A. Correct, it's an expression, non-linear degassing. Yes, you're absolutely right.

Q. Would that also account for why the suspension RVCM level, whatever it may be, would decrease rather rapidly in the beginning after manufacture and then not continue to decrease at that same rate later on?

A. Right. Rapid initial degassing, then tapering off.

Q. Would the same basic principle be true

for the bulk process which has, according to Dr. Wheeler has a RVCM content of between five and 15 ppm?

A. That would be closer to the solution kind because the initial levels were relatively low compared to suspension, but in principle, yes.

Q. Did you have a particular RVCM level in mind when you prepared the report as to Mr. Peterson's exposure to the bulk process resin?

A. No.

Q. What do you mean in your report about the middle of the first paragraph under polyvinyl chloride on page three, quote, "The unreacted monomer can be eluted out of PVC by body fluids" -- this is where you made the change, isn't it?

A. That's okay, I don't make any change in relation to that.

Q. Well, let me stop there in the quote because what I wanted to ask you was with regard to the body fluids?

A. Sure.

Q. I assume you are talking about after the PVC has been in some way ingested by the body?

A. Ingested or inhaled.

Q. Or inhaled?

A. Sure.

Q. And it would be then the human body's fluids that is causing some of the unreacted monomer to be taken out of the PVC resin?

A. Yes, it would dissolve it out.

Q. How would that happen?

A. The vinyl chloride would partition itself between the level in the PVC particle and the level in the plasma or extra cellular fluid and you would have a very rapid diffusion out or what's called elution and there's very substantial repeated references to this in the literature.

Q. The beginning of the following paragraph reads as follows: I will read for the record, "VC is a potent carcinogen which induces a wide range of malignant neoplasms in a wide range of expermental animals, rats, mice and hamsters, following chronic inhalation, oral or parenteral exposure." With regard to those, the reference to malignant neoplasms and in particular looking at the rest of the paragraph there, are there any studies that have indicated in animals that laryngeal cancer has been found to be caused by exposure to VC?

A. Not that I'm aware of.

Q. Are you aware of studies that have been

done whereby animals have been exposed to PVC resin or PVC dusts?

A. Yes, these are listed for you in one of the more recent documents which I've included.

Q. Is there any indication in those studies that there is a connection between the exposure to PVC resin or dust and laryngeal cancer?

A. You've asked me as to whether on laryngeal cancer the PVC -- the answer is no.

Q. Is it Keplinger study? Is that how you pronounce it?

A. Yes.

Q. Have the Keplinger and Viola studies been fully accepted by the community, scientific community, to your knowledge?

Are there any flaws in those studies that would cause them not to be readily accepted by the scientific community?

A. Well, there were questions, to the best of my recollection, some of the MCA has challenged, I know, the lung tumors as to being secondary to metastasis rather than primary.

Q. Was the Keplinger work actually completed or was that a preliminary study that was not completed?

A. That's odd.

Q. Drawing a blank?

A. No, it is interesting, I report Captain Joe but don't give a reference to him. I'm sure it was reported to the New York Academy of Sciences, you'll find no reference in Keplinger. I apologize, Captain Joe worked for a non-distinguished or reprehensible company, IDT, that you're very well aware of, but I think the report was New York Academy of Sciences.

Q. Were there any criticisms at Viola other than indicated from Maltoni with regard to the concentration level of VC that you exposed to scientific animals?

A. Yes, there are a wide range of criticisms against it. First of all, he gave the impression of the report that you only got tumors at 30,000 ppm, which is untrue.

MR. LEVINSON: Who are you discussing now?

Q. Viola, your other expert.

A. He's your boy.

That was the first thing. In fact, there were tumors down to 500 ppm, and at one of the MCA meetings there was reference to the fact that he got tumors down to 250 ppm, I'm pretty sure that was

right for Viola.

Anyway, there were tumors down to 500 ppm, then my major criticism, I think that was the criticism, and also the influence that these are very, very rare and unusual tumors that don't really have much relevance to humans, which is one point, but in general Viola's paper was important an statement and the first clear cut statement on the carcinogenicity of compound which had been in use by Union Carbide since 1930 which had never been tested before for carcinogenicies, had never been tested for over a period of four decades.

In other words, it's the first indication of carcinogenicity or compound that had been wildly used in commerce and in spite of very substantial evidence on toxicity, in spite of also the findings of the unreported findings of liver cancers or angiosarcomas in workers prior to 1970.

Q. Well, since my question dealt with whether or not there were criticism levels to the Viola for the way he conducted the study, I will move to strike anything after that portion of the answer.

A. Of course, another criticism was these were unrealistically high concentrations and I don't think that's a bout of criticism.

Q. Why not?

A. First of all, there were tumors going down to 500 ppm and later pointed out levels below that.

Q. On that paper?

Was there a subsequent paper by Viola?

A. I think it was MCA meeting, I can try and find it for you.

Let me just go back, I have misspoken. The 500 ppm that were in his data, in Viola's data, there were tumors recognized at 500 ppm, also in his paper ear tumors were noted at unspecified concentrations below 500 ppm, they were unspecified.

Q. To your knowledge, and in your review of the literature, when is the first time that there is a report or study linking vinyl chloride with cancer?

A. In animals?

Q. Take whichever came first. If you wish to take animals, I assume it's animals?

A. We will take what you like. The Viola data with the first data in animals.

Q. What year was that again?

A. 1970, some four decades after the vinyl chloride was manufactured.

Then as far as humans are concerned, we have the following; we have a Worker's Compensation

case, Mr. Parks, I think you recall that was a B.F. Goodrich case that was a Worker's Compensation case in about '65, which I believe was, he had angiosarcoma, and then prior to 1970 there were several angiosarcomas reported.

In fact, from '65 to '71 there were eight cases so-called liver cancer reported in VC workers, and many of these were rediagnosed as angiosarcoma and that was including the Parks' case, I referred to Park or Parkers.

Then in 1969 Maltoni found malignant type cells in the sputum of VC workers, although this wasn't reported until 1975, and then -- I'm sorry, what was your date, 1977?

Q. No, I asked you when was the first report in the literature with regards to a link between vinyl chloride and cancer, you said Viola on the animal studies.

A. Viola on the animals, then a series of cases and then the Maltoni findings reported in '75, 1969 findings reported in '75.

Q. At the top of page four of your appendix regarding PVC and VCM, you say, and I will exclude the reference to the studies, you say, quote, "Numerous reports have drawn attention to the

development of chronic pulmonary disease in VC/PVC workers. The factor characterizing most of these reports is chronic exposure to PVC dust."

A. Yes.

Q. Is there a reference in those studies to the particular form of PVC dust that you can recall?

A. Not that I recall, I don't think so.

Q. I asked you if it refreshed your recollection, didn't they all refer to the dispersion method of manufacturing this particular dust?

A. I don't recall.

Q. At the end of that paragraph you say, quote, "The view was expressed that the PVC dust was likely to concentrate in and damage the small airways, besides also acting as the carrier for carcinogenic VC monomer."

Whose view was being expressed in that statement?

A. In particular Lilis and Miller, although, in fact, subsequent studies much more clear cut statement.

Q. So it's intended to refer to the study in the preceding sentence?

A. Yes, at this stage with that reference subsequent more detailed statements to that effect.

Q. Towards the end of that page there's a reference to the Infante studies of 1981. With regard to that, my question is whether or not Infante noted any laryngeal cancers in his review?

A. No.

Q. Is the follow from that therefore that Infante did not conclude that the larynx was one of the carcinogenicities in humans?

A. No, not at all.

Q. Why not?

A. Because it says he discussed a wide range of sites, he said it was a multi-potent to which you can get tumors in a very wide range of sites.

Q. Okay, there may be a question of semantics, however, within the wide range of sites he does not specifically refer to the larynx; is that correct?

A. No, just occur in a very wide range of the sites in the body. He just happened to review the data on these particular sites and emphasize those particular sites which are much more common than larynx.

Q. I'm going to ask it again for a different reason. I understand what you said but I asked you in a negative and you said no, which makes

it sound like a positive.

A. I'm sorry.

Q. So it is correct that he does not site the larynx in particular?

A. As far as I recall he didn't. He discussed respiratory tract cancers which could include larynx.

Q. With regard to the Tabershaw & Gaffey study that's cited at the bottom of page 4 --

A. Yes.

Q. -- Where there is a discussion apparently on cancers of the larynx, pharynx and digestive tracts, what was the incidents reported in that study? In other words, how many cancers of the larynx, pharynx and digestive tracts were reported out of how many individual studies, if you know?

A. Well, you'll find it's detailed for you in the table, there were about eight and a half thousand in the initial '74 study, there were about eight and a half thousand workers with at least one year exposure.

One of the problems in the study was that about 15 percent of the workers of the cohort who had the highest exposure levels were missing and the authors recognized, in fact, that whatever they found would underestimate risk and then they found an

excessive respiratory tract cancers in workers particularly with over five years employment and particularly with high exposures, and for respiratory tract cancers there was an elevated standardized mortality ratio, and among these respiratory tract cancers was a one extrinsic laryngeal cancer. And in addition to that the authors also recognized the multiple site cancers in experimental animals, so the Tabershaw & Gaffey study was of interest that they recognized multiple site cancers induced in workers and experimental animals that was in the additional study. In the subsequent study --

Q. Can I hold you there one second?

A. Sure.

Q. You say that's summarized. Just for the record, would you say where that summary is?

A. In the more recent document entitled Illustrative Literature On The Toxic And Carcinogenic Effects Of VC/PVC In The Respiratory Tract Of Exposed Workers.

Q. I think I heard you say Tabershaw & Gaffey were looking at approximately 8500 workers?

A. Correct, I didn't say they were looking at them in a sense, and in a sense they weren't because 1500, the data were missing on 1500.

Q. I'm sorry.
A. The total study was eight and a half thousand, yes.
Q. And they were missing another 1500 of the more heavily exposed?
A. Yes.
Q. But the study itself was 8500?
A. Yes.
Q. Out of that, how many laryngeal cancers did they have?
A. One was noted there.
Q. What was the subsequent studies?
A. The subsequent was basically an extension of the earlier one that's in 1975, an additional 700 workers were included and again they found an excess of respiratory tract cancers and here they reported an additional two laryngeal cancers and pharyngeal cancer, so we have at least three laryngeal cancers among this group.

So in other words, of in the first Tabershaw & Gaffey study of 12 workers with respiratory tract cancers there was one extrinsic laryngeal cancer, in the subsequent study of a total of -- in the total cohort of 37 respiratory tract cancers there were two laryngeal cancers.

Q. What is the incidents of laryngeal cancer in the general population?

A. You asked me this before.

Q. I'm sorry. If I did, I forget.

A. 08.5 per 100,000.

Q. I'm sorry, you did say that, I even made a note of it.

A. Yes. I'm sorry, I misled you now. That's, I think, for white males, or is it for white males? Perhaps it's for males. Yes, I misled you a second time, it's for all males.

Q. Stop misleading me and give me the right answer.

A. I'm trying. It's for all males, I'm sorry. 08.5 per 100,000 for all males.

Q. In the Tabershaw & Gaffey studies, were these workers in the fabricating part of the industry?

A. Well, to the best of my recollection, these were VC/PVC industries, these were PVC industries, they were based on about nine different companies, so they were VC/PVC, to the best of my recollection.

Q. Which would mean, in your mind, they were the fabricating end of the industry or can you not tell?

A. No, I think VC and PVC, in other words, it was a mix of 19 different industries.

Q. Is there any indication in the studies as to what the exposure levels were for the various workers?

A. Similarly in the same way as Union Carbide and ATC failed to monitor their work environment prior to 1974, the Tabershaw & Gaffey was based on industries that similarly had failed to monitor and the exposure was ranked on the basis of work practice and on exposure deration as in terms of three levels, three exposures, index one exposure, index two and exposure index three, and exposure index three being the highest exposure levels and the longest and excess respiratory tract system cancers, which as you know include laryngeal cancers, were found and those were the highest exposure index.

Q. Are you aware of any study that's been published that shows a connection between VC and lung cancer?

A. Oh, yes.

Q. Can you tell me which?

A. You will find a nice little table on this.

Q. A new section which I have not seen?

A. Which you will be able to take home and show

it to your experts and have some distinction in the trial for me on December 15th.

Q. Are they summarized there, Dr. Epstein?

A. Beautifully.

Q. A simple yes would suffice.

A. Essentially it states whether VC or PVC and there's three pages for you.

MR. LEVINSON: You're referring to what?

Q. These all refer to the lung or respiratory tract?

A. Respiratory tract and there's a similar kind of table, of course, for experimental animals.

MR. LEVINSON: Those are all parts of your record?

THE WITNESS: Oh, yes.

Q. Now, if I heard you correctly earlier in your testimony, the larynx is considered by doctors to be part of the respiratory tract?

A. Sure.

Q. Do any of the studies that are listed in your beautifully prepared summary --

A. Well, thank you.

Q. -- Indicate or show a cancer of the larynx in those studies?

A. Yes.

Q. Can you point that out to me?

A. We've just been discussing Tabershaw/Cooper?

Q. Tabershaw/Gaffey?

A. Yes.

Q. Is that the only one?

A. Those are three studies, yes. I am happy to say that they're basically one study reported in three different ways.

Q. Three studies by Tabershaw/Gaffey?

A. Tabershaw and Cooper and Tabershaw and Gaffey.

Q. Okay. In preparing the reference portion of your report on VC and PVC?

A. Yes.

Q. And perhaps better stated, what I mean is in preparing your actual report on this subject, did you have occasion to review the paper by Richard Doll?

A. Which one?

Q. That's the one in the Scandinavian Journal Of Work And Environmental Health in 1988?

A. On what subject?

Q. A review of the epidemiologic study relating to health effects of vinyl chloride exposure?

A. No, I'm not aware of that. Why don't you give me the reference.

Q. I think I just did, I don't know the exact --

A. Doll 1988?

Q. Scandinavian Journal Of Work And Environmental Health.

A. And that's on VC and --

Q. It's VC and it's a review of epidemiologic studies relating to the health effects of vinyl chloride exposure.

A. Sure, I will take a look at it and chat to you about it in December.

Q. Very good, I look forward to it.

MR. LEVINSON: Off the record.

(Discussion off the record.)

Q. Stay on that for a moment, if we can.

A. Sure.

Q. You indicated to me earlier that with regard to your accumulation of literature on different chemicals including PVC, it comes to your attention and if it's on the subject you make a point of it -- I'm sorry, you acknowledge it and perhaps put it on the index cards?

A. As you've already made it clear, that's an

imperfect process as Doll has been missed.

Q. My question really was, is it your system and your procedure to make a note of any study that comes out on the particular chemical, would you only make a listing and a note of those studies that you read and intend to at a later point in time rely upon?

A. In general I would like to try to keep abreast of most of the literature on toxic chemicals, but I tend to exercise my interest somewhat selectively and I tend to keep more details, bibliographies on certain chemicals more so than others.

Q. Would you say you have an extensive catalog with regard to VC and PVC?

A. I would say it's reasonable, but obviously not complete.

MR. HOLLINGSHEAD: Off the record.

(Whereupon the above deposition concluded at 3:15 p.m.)

CERTIFICATE

I, CINDY L. NAGLE, a Notary Public and Certified Shorthand Reporter of the State of New Jersey, License No. XI01434 do hereby certify that prior to the commencement of the examination DR. SAMUEL EPSTEIN was duly sworn by me to testify the truth, the whole truth, and nothing but the truth.

I DO FURTHER CERTIFY that the foregoing is a true and accurate transcript of the testimony as taken stenographically by and before me at the time, place, and on the date hereinbefore set forth.

I DO FURTHER CERTIFY that I am neither a relative nor employee nor attorney nor counsel of any of the parties to this action, and that I am neither a relative nor employee of such attorney or counsel, and that I am not financially interested in the action.

Cindy L Nagle

Notary Public of the State of New Jersey
My Commission expires March 29, 1994

I N D E X

E X H I B I T S

EPSTEIN EXHIBITS: