

IN THE SUPERIOR COURT OF THE STATE OF DELAWARE
IN AND FOR NEW CASTLE COUNTY

IN RE: ASBESTOS LITIGATION) C.A. No. 77C-ASB-2

BEFORE: HONORABLE JOSEPH R. SLIGHTS, III, J.

APPEARANCES:

JOHN J. SPILLANE, ESQ.
BARON & BUDD, P.C.
for the Plaintiff

CAMERON R. WADDELL, ESQ.
LeBLANC & WADDELL, LLP
for the Plaintiff

JOSEPH BIDEN, III, ESQ.
BIFFERATO, GENTILOTTI, BIDEN & BALICK
for the Plaintiff

THOMAS C. CRUMPLAR, ESQ.
JACOBS & CRUMPLAR
for the Plaintiff

DAUBERT HEARING TRANSCRIPT
TUESDAY, OCTOBER 18, 2005 - A.M. SESSION

LYNNE BELL COALE, RDR, CRR
SUPERIOR COURT OFFICIAL REPORTERS
500 N. King Street, Suite 2609, 2nd Floor
Wilmington, Delaware 19801-3725
(302) 255-0562

APPEARANCES CONTINUED:

SAMUEL L. TARRY, JR., ESQ.
 McGUIREWOODS LLP
 for the Defendant DaimlerChrysler

JAMES M. KRON, ESQ.
 SOMERS S. PRICE, JR., ESQ.
 POTTER, ANDERSON & CORROON LLP
 for the Defendant DaimlerChrysler

STEVEN T. JOHNSON, ESQ.
 PEPPLER, JOHNSON, CANTU & SCHMIDT, PLLC
 for the Defendant OI, Inc.

ALSO PRESENT:

David deBruim	Joseph A. Gabay
Erin Farris	Christian J. Singewald
William A. Kohlburg	C. Scott Reese
Kathleen D. Hadley	Miranda D. Clifton
Michael Angelides	Gary Kaplan
Melissa Crowe	Mark Reardon
Perry J. Browder	Kai Seelaus
Amy Garrett	J. Michael Johnson
Christopher Lyon	Neal Glenn
Bernard Kuery	Christine Boyd
Francis J. Gribbin	Eric Henry
Margaret England	

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1 TUESDAY, OCTOBER 18, 2005
2 Courtroom No. 8B
3 9:03 a.m.

4 PRESENT:

5 As noted.

6 - - - - -

7 THE COURT: Good morning.

8 ALL COUNSEL: Good morning, your Honor.

9 MR. WADDELL: Plaintiffs will call Dr. Sam
10 Hammar to the stand.

11 SAMUEL HAMMAR, M.D.

12 having first duly affirmed, was examined and testified
13 as follows:

14 DIRECT EXAMINATION

15 BY MR. WADDELL:

16 Q. Dr. Hammar, are you a trained physician in the
17 diagnosis and cause of asbestos disease in humans?

18 A. Yes.

19 Q. And I want us to go through with his Honor your
20 background and explain to him about that, how you became
21 a trained physician in the diagnosis and cause of
22 asbestos disease in humans. Okay?

23 A. All right.

1 Q. What type of doctor are you?

2 A. Pathologist.

3 Q. All right. And what areas of pathology are you
4 board certified in?

5 A. I'm board certified in anatomic and clinical
6 pathology.

7 Q. What's the difference?

8 A. The difference is, anatomic pathology deals
9 with the diagnosis and diseases by looking at cells,
10 tissues and organs. Primarily, we receive biopsy
11 specimens from clinical doctors, and we are asked to
12 evaluate them, to determine if that biopsy specimen
13 shows some type of a disease process, such as a cancer,
14 an inflammatory process, a degenerative process,
15 etcetera. We write reports indicating what we see.
16 That information is then sent back to the clinical
17 doctor, who asks us to review the specimen and they act
18 upon what information we give them.

19 Clinical pathology deals with running a
20 laboratory. At this point in time, our main job is to
21 make sure that all of the machines that perform tests on
22 things like blood are accurately calibrated and that we
23 have very good quality control in the lab, to make sure

1 the results that are generated in the clinical
2 laboratory are correct.

3 We will sometimes help the clinical doctors
4 interpret some of the laboratory findings and try to
5 help make a specific diagnosis in a patient.

6 Q. Okay. Dr. Hammar, your board certifications
7 were in 1975; correct?

8 A. That is correct.

9 Q. Now, when did you first become involved in
10 seeing or participating in the diagnosis of asbestos
11 disease in humans?

12 A. Very early on in my training. As a resident in
13 pathology, I saw cases of asbestos-related lung disease.
14 That would probably be the first time. That would be in
15 the early 1970s.

16 Q. Okay. And where do you practice, Dr. Hammar?

17 A. I practice in a small town, a small city called
18 Bremerton, Washington, which is about 15 miles across
19 Puget Sound from Seattle.

20 Q. Is there something unique about where you
21 practice that got you heavily involved in asbestos?

22 A. Yes.

23 Q. Could you explain that to the Court.

1 A. The Puget Sound Naval Shipyard is located in
2 Bremerton, Washington, and has been in existence since
3 about 1909. As a result of that, about a fifth
4 generation of individuals living in the Bremerton area,
5 which is in Kitsap, K-I-T-S-A-P, County, live in that
6 area. As a result of the shipyard and those individuals
7 working in the shipyard, a significant number of those
8 people have been exposed to asbestos. And as a result
9 of that, we see a very high incidence of asbestos
10 related disease in Bremerton. We see ten to 20 new
11 cases of mesothelioma per year, about a similar number
12 of cases of lung cancer related to asbestos per year,
13 and probably 40 cases or so of asbestos-induced plural
14 disease in Bremerton every year.

15 Right now, the highest incidence of
16 mesothelioma in the United States is in Bremerton. We
17 have a population in Kitsap County of about 3,000 --
18 excuse me, 300,000 people, and we see, again, ten to 20
19 new cases of mesothelioma per year, which is an
20 incredible incidence, if you compare it to the
21 background, which is basically zero.

22 Q. Is even seeing one single case of mesothelioma
23 significant, in your opinion?

1 A. Yes.

2 Q. And why is that?

3 A. It's because that is such a rare tumor in
4 general, that seeing even one case is significant.

5 Q. When did you begin seeing mesothelioma cases
6 specifically in your regular practice as a pathologist
7 in Bremerton?

8 A. I started actually seeing them when I worked in
9 Seattle. Before I went to Bremerton, I worked at an
10 institution in Seattle called the Virginia Mason Medical
11 Center, and we saw a significant number of cases there.
12 And in fact, in early 1980s, we put on an exhibit for
13 the International Academy of Pathology on mesothelioma,
14 in which we described a number of cases and showed their
15 various pathologic features.

16 Q. Right now, currently, could you describe for
17 the Court the types of cases you see in your practice,
18 or outside of your medical practice, so to speak?

19 A. Sure. I see cases from physicians who take
20 care of patients with asbestos-related disease in
21 Bremerton. These are primarily the pulmonologists and
22 surgeons who take care of these individuals. Like I
23 said, we see about ten to 20 new cases of meso per year.

1 I also receive a significant number of cases
2 that are sent to me in consultation from other
3 pathologists, sometimes in the northwest. But I also
4 see cases from all over the United States. And I also
5 see a few cases that are sent to me from pathologists
6 and physicians outside of the United States.

7 I also see a significant number of cases of
8 mesothelioma and other asbestos-related lung diseases
9 that are sent to me by attorneys like yourself.

10 Q. Okay. And you've testified in courts before in
11 litigation involving asbestos; correct?

12 A. Yes.

13 Q. And, initially, you testified for defendants in
14 litigation; right?

15 A. That is correct.

16 Q. And now, it's currently primarily at the
17 request of plaintiffs' lawyers like myself?

18 A. Yes.

19 Q. Are you a researcher or have been a researcher
20 regarding asbestos disease in humans?

21 A. Yes.

22 Q. Could you explain that to his Honor.

23 A. Well, I've been very interested in

1 mesothelioma. And we have, myself and some other
2 individuals, have published quite a few papers that deal
3 with various aspects of mesothelioma.

4 In 1986, myself and two other pathologists, one
5 by the name of John Bolen, B-O-L-E-N, another by the
6 name of Michael McNutt, published a paper on what we
7 termed the multipotential subserosal cell, which was a
8 paper that dealt with injury to the pleural surface and
9 how the pleural surface responded to injury and how one
10 could relate that response to injury to the morphology,
11 which is the features of mesothelioma that a pathologist
12 saw through the microscope.

13 We started doing a significant amount of
14 evaluation of cases of mesothelioma through the electron
15 microscope, and it turns out that a subtype of
16 mesothelioma, called epithelial mesotheliomas, have very
17 unique ultrastructural characteristics which allow for
18 their diagnosis. I would say, for epithelial
19 mesothelioma, that probably is the single best
20 diagnostic modality there is.

21 And then in the mid 1990s, Dr. Dodson and I
22 started working together on a variety of different
23 projects. In 1997, we wrote an article on fiber

1 analysis in lung tissue with 55 people with
2 mesothelioma. In the year 2000, we wrote an article
3 published in a journal called *Chest* that dealt with
4 translocation of asbestos from the lung to
5 extrapulmonary sites -- specifically, the omentum and
6 mesentery tissue, which is the fat tissue in the
7 abdominal cavity. In that article, we demonstrated that
8 asbestos does translocate to other parts of the body,
9 and specifically other parts of the body where
10 mesotheliomas develop.

11 Right now, Dr. Dodson and I are doing a
12 research project, in which we're looking at the
13 concentration of asbestos in various tissues;
14 specifically, the normal visceral pleura, the normal
15 parietal pleura, hyaline pleural plaques, mesothelial
16 tumor tissue, and all of the lymph nodes that are
17 present around where the vessels and air tubes enter the
18 lung, and also the lymph nodes that are present in the
19 central part of the chest, which is called the
20 mediastinum. So, that's the research projects we have
21 going right now.

22 Q. And your research has made it into the
23 scientific and medical literature?

1 A. It has, yes.

2 Q. And, in fact, under publications on your
3 curriculum vitae, there are 114?

4 A. Yes. The latest -- actually, there's one more.
5 I'm part of the US-Canadian Mesothelioma Panel,
6 and we just wrote an article on a rare type of
7 mesothelioma called a localized malignant mesothelioma.

8 Q. So, there will be 115 then?

9 A. That's correct.

10 Q. Peer reviewed?

11 A. Yes.

12 Q. And Dr. Hammar, I need to ask this question:

13 When you're doing research and you're doing
14 your writings, are you applying generally accepted
15 scientific and medical principles and methodology when
16 doing so?

17 A. Yes.

18 Q. How about -- we talked about publications and
19 things. Textbooks, have you done that?

20 A. Yes, I have.

21 Q. Okay. Could you explain to his Honor textbooks
22 that you've written, chapters that you've written that
23 have made it in other textbooks regarding asbestos.

1 A. Yes. That book there is a second edition of a
2 book called *Pulmonary Pathology*. The first edition was
3 published in 1987. Myself and David H. Dail are the
4 editors in that book there. And in the first edition, I
5 wrote five of the chapters. And in that book there,
6 Dr. Dodson and I wrote the chapter titled "Asbestos,"
7 which is Chapter 28. I wrote Chapter 34 which is titled
8 "Pleural Diseases," of which about 90 percent deals with
9 the disease mesothelioma.

10 Myself, Dr. Dail, and Dr. Thomas Colby, who's a
11 pathologist at Mayo Clinic, wrote another textbook
12 called *Pulmonary Pathology Tumors* that deals with the
13 neoplasms that occur in the lung and chest cavity.

14 Dr. Dodson and myself just wrote a book that
15 was just published called *Asbestos: Risk Assessment,*
16 *Epidemiology and Health Effects*, in which we discussed
17 some of the technical aspects about asbestos and
18 asbestos measurements, and also epidemiology, molecular
19 biology, and the pathologic changes that one sees in
20 asbestos-related lung disease.

21 And I've written about 40 chapter in textbooks
22 for other individuals, several of which have dealt with
23 mesothelioma.

1 Q. Because of your experience, your training, and
2 your work, has your expertise risen to the level in
3 skill where your peers have placed you in positions of
4 leadership?

5 A. Yes.

6 Q. Could you explain that to his Honor.

7 A. Well, I guess, in leadership, is that I guess
8 my peers think that I know enough about mesothelioma to
9 be selected to the US and Canadian Mesothelioma Panel,
10 which is a panel of 12 pathologists, ten in the
11 United States, one in Canada, and one in Wales, Great
12 Britain. And what we do is review cases for other
13 pathologists or other physicians who are sent to us
14 where there's a question of whether a disease is or is
15 not mesothelioma. And we do this free of charge, and we
16 indicate on a form whether we think the case is or is
17 not mesothelioma. And the information is tabulated and
18 then sent back to the person who sent us the case.

19 I'm also a member of the International
20 Mesothelioma Pathology Group which is again a relatively
21 small group of about 25 pathologists throughout the
22 world. And we just finished writing a book for the
23 French government because of their epidemic of

1 mesothelioma in that country, and the title of that book
2 is *Pathology of Mesothelioma*.

3 I have been asked to lecture on mesothelioma
4 fairly extensively. The last talk I gave was in San
5 Antonio, Texas, in February of 2005. And that had to do
6 with the macroscopic, histologic, histochemical
7 immunohistochemical, and ultrastructural features of
8 mesothelioma.

9 Dr. Dodson and a few other people are also
10 involved in doing, again, some additional research
11 projects that we are involved with that are, hopefully,
12 going to be published.

13 And I've also been a member of the World Health
14 Organization. We published a book in 1999, which was
15 the *Current Classification of Lung Cancers and*
16 *Mesothelioma*, and another book in 2004 which was the
17 *Pathology and Genetics of Lung Cancer*, including
18 mesothelioma as well as tumors of the thymus and tumors
19 of the heart.

20 Q. And even in groups outside of your specialty, I
21 guess, area in asbestos, have you also risen to the top
22 in societies that you are a member of in pathology?

23 A. I have. I've been very fortunate. I was the

1 second president of the International Pulmonary
2 Pathology Group. I've been the past president of the
3 Society of Ultrastructural Pathology. I've been on --
4 the program chairman of both of those societies for many
5 years. I still am very active in both of those
6 societies and have been involved in organizing programs
7 for the Pulmonary Pathology Society. And this coming,
8 see -- it will be next August, I'm going to chair a
9 session on diseases of the pleural -- pleura for the
10 International Academy of Pathology, which is going to be
11 held in Montreal Canada.

12 Q. Having discussed briefly your training and
13 experience and skill as a pathologist and in asbestos
14 disease in humans, have you had the opportunity to
15 become, I guess for lack of a better word, very familiar
16 with other areas of medicine, about asbestos disease
17 other than pathology?

18 A. I would say so, yes.

19 Q. Okay.

20 A. And one thing that I get to see when I see the
21 legal cases that are sent to me by attorneys and also
22 because of where I work, I get to see the entire
23 histories of these individuals and how they presented,

1 how they were treated, what happened to them, and know a
2 lot about the radiology of asbestos-induced lung
3 disease. I've seen over 4000 cases of mesothelioma.
4 I've done over -- about 475 autopsies on patients with
5 mesothelioma. And I've seen over a thousand cases of
6 autopsy tissue from patients with mesothelioma and lung
7 cancer that have been sent to me, primarily by
8 attorneys, sometimes by the pathologists. So, I've been
9 able to see a lot of that.

10 I sometimes am asked by the thoracic surgeons
11 who do surgery, to come into the operating room and help
12 them determine where the best place is to biopsy. I've
13 presented a chest conference in Bremerton, where I work,
14 every month, where we discuss cases of lung disease,
15 including asbestos-related lung disease, where all of
16 the various aspects of the disease are presented, such
17 as a clinical history, the radiographic findings, the
18 pathologic findings and the treatment of those
19 individuals.

20 So, I have been in a very somewhat unique
21 position of being able to see not only the pathologic
22 features of asbestos-related disease, but also the
23 clinical features of that disease.

1 Q. And not just in the context of cases that
2 appear in courtrooms like this; correct?

3 A. That's right. I happen to be the chairman of
4 our Cancer Committee at the hospital in Bremerton, which
5 is Harrison Hospital. And we discuss cases like this
6 and try to determine what the best treatment is for
7 these individuals.

8 Q. Do you also have an occasion -- because most
9 people think pathologists are stuck in some room in a
10 hospital, that they never get to see their patients. Do
11 you get a chance to see and visit with your patients as
12 well?

13 A. I do. Pathologists usually don't see patients,
14 but there are times when I have been fortunate enough to
15 have contact with the individual patients, primarily
16 individual patients who have mesothelioma, and have
17 gotten to know these people extremely well.

18 Q. You told me about one this morning, I think,
19 that came from Italy; right?

20 A. Yes. I told you about a Mr. Neltson (PH.),
21 Douglas Neltson, young man, relatively young, 58 years
22 old, was diagnosed in Bremerton with mesothelioma; had a
23 very unique course, in that he was treated primarily

1 with chemotherapy, initially with some Carboplatin and
2 Taxol. And, then, he received some Gemcitabine and
3 subsequently received Pemetrexed and Cisplatin, and had
4 a very good response and actually survived about eight
5 years with the tumor. And then, unfortunately, he died
6 from Gemcitabine interstitial fibrosis as a consequence
7 or as a bad side effect of the chemotherapy that
8 actually was keeping his tumor at bay.

9 Q. We're going to need a few minutes after . . .

10 You mentioned histories of individuals that
11 have, unfortunately, been diagnosed with mesothelioma.
12 Do you also have the opportunity of seeing those in the
13 context of just your normal practice outside the
14 courtroom setting?

15 A. Sure. Again, I see that on a -- basically, a
16 weekly basis or monthly basis in Bremerton, where I
17 work.

18 Q. Are the principals of medicine and science that
19 you use in the courtroom any different than those that
20 you use outside the courtroom?

21 A. Not really. As far as my involvement in the
22 asbestos-related legal arenas, I do the same thing there
23 as I would do as a practicing pathologist. The only

1 difference would be that, often, with the legal cases, I
2 see more of the actual clinical history on the patient
3 than I do on the cases that I am treating pathologist.

4 Q. In a minute, we're going to go through a few
5 more things in detail.

6 But when it comes to causation, is there just
7 one thing you're going to look at as a pathologist and
8 say, "Ah hah, that is it, and I don't need to look in
9 anything else in the fields of medicine and science"?

10 A. No, I think you always have to keep your mind
11 open to a variety of causes of any disease.
12 Pathologists do what clinical doctors do in some
13 respect, in that we have what are called pathologic
14 differential diagnoses, in that you could have a tumor,
15 for example, that had a certain morphology and you would
16 have to determine whether that was a mesothelioma or was
17 it a lung cancer that looked like a mesothelioma.

18 Q. And in diagnosing, you apply a lot of the same
19 principles as you would to causation; right?

20 A. Well, the diagnosis is certainly, in some
21 respects, the same but also is different. In diagnosis,
22 you're basically trying to say what a tumor is from a
23 pathology point of view.

1 As far as the causation, obviously, that can be
2 extremely important. It can be important, obviously, in
3 asbestos-related disease because, if one person has an
4 asbestos-related disease, there's a chance that that
5 individual is going to get another asbestos-related
6 disease that the individual should know about that, to
7 potentially be screened or somehow have some type of a
8 followup, so if he does develop another asbestos-related
9 disease, that, hopefully, that disease can be detected
10 at a stage or time where he might, or she might, have
11 some type of therapeutic options.

12 Q. And I think I asked a bad question in that
13 regard. I guess one of the things I wanted to convey to
14 his Honor, do you look at the whole picture?

15 A. Of course.

16 Q. Do you even -- do you even look at things, even
17 in the diagnosis and look to exclude certain things?

18 A. Well, you have to always make sure that you're
19 not going to exclude things and that you make the
20 correct diagnosis because what treatment is based on is
21 the diagnosis, and the diagnosis has to be correct.

22 Q. I apologize. And in regards to looking at
23 causation, is there fields of science that you just want

1 to totally disregard --

2 A. No.

3 Q. To the exclusions of others?

4 A. You never disregard, you -- to try to exclude
5 something else. You look at the whole picture.

6 Q. When someone comes in and you get involved in a
7 case, either in a legal setting or in a hospital
8 setting, and you're asked to look at the potential cause
9 or causes of the injury to the victim, do you start off
10 with the checklist and go, "Let me just make sure that
11 that passes epidemiological muster before I consider
12 anything"?

13 A. I don't think you do it that way, no.

14 Q. Do you try to fit all the pieces of the puzzle
15 together to get the best picture of what happened?

16 A. Sure.

17 Q. Now, I want to go through just a few things
18 real quick because, as a doctor, it is a little bit
19 different than a cell biologist or someone. But
20 focusing on the anatomy of the human lung, okay, and
21 inside the lung and outside the lung and what happens
22 when humans breathe asbestos.

23 A. All right.

1 Q. My first question to you --

2 MR. WADDELL: And this is my drawing, so I
3 apologize, your Honor.

4 THE COURT: It's pretty good.

5 BY MR. WADDELL:

6 Q. Is a brake mechanic's lung any different than
7 your lung or my lung?

8 A. No.

9 Q. Okay. When a brake mechanic breathes asbestos,
10 does anything magical happen with the fibers outside his
11 body?

12 A. No.

13 Q. Does anything magic that's different than me or
14 you, does anything magical happen once those fibers are
15 inhaled inside the lung?

16 A. No.

17 Q. Does the reaction that the body has in
18 ultimately producing disease, is it any different in a
19 brake mechanic as it is with anyone else?

20 A. No.

21 Q. There are commercially used in the
22 United States what types of fibers?

23 A. Primarily, it's been chrysotile, amosite, and

1 crocidolite and, to a lesser extent, tremolite and
2 fosterite.

3 Q. And a few of the things that I want to briefly
4 cover with his Honor today is the diseases that asbestos
5 fibers, when inhaled into a human cause inside the lung
6 and outside the lung?

7 A. Yes.

8 Q. And I've written scarring enhancer. If you
9 could walk his Honor?

10 MR. WADDELL: Your Honor, may he come down?

11 THE COURT: Absolutely.

12 BY MR. WADDELL:

13 Q. If I could ask you, Dr. Hammar, if you could
14 explain how fibers are inhaled and how they cause injury
15 in humans, including brake mechanics.

16 A. Well, the way that asbestos fibers happen to be
17 inhaled are usually in the situation where an individual
18 is in an area where some type of asbestos-containing
19 product is being altered that results in that product
20 releasing asbestos fibers into the air and that
21 individual would then breathe the air that contained the
22 asbestos fibers.

23 There are other situations that potentially

1 could result in exposure to asbestos. Let's just say,
2 for example, if you were in a certain part of the world
3 where asbestos naturally occurred, that you could
4 potentially inhale fibers in that situation -- like, for
5 example, in Quebec, Canada, South Africa, Bolivia, even
6 in certain parts of the United States, where you could
7 potentially inhale asbestos fibers as a result of
8 environmental release of fibers into the air. But that
9 is very uncommon compared to a person inhaling asbestos
10 fibers as a result of inhalation of fibers that came
11 from a product.

12 So, when you breathe the air, the air comes
13 into your nose or mouth, goes down the windpipe, which
14 is called the trachea, and goes through the main stem
15 bronchi, which are these large tubes that carry the air
16 to the right and the left lung. And the asbestos
17 fibers, many of them are actually trapped up in our nose
18 and mouth. Some of them are cleared by what's called
19 the mucociliary apparatus, which is a type of
20 specialized epithelium that lines the surface of these
21 air tubes. But a certain percentage of these fibers do
22 get into the outer part of the lung, and they are first
23 deposited in the region of what's called the respiratory

1 bronchial and the alveolar ducts. And these are really
2 tiny air tubes, and they are lodged in that area. And
3 once they are lodged in that area, then they can cause
4 injury.

5 Q. And when a -- in the process we're describing
6 as fibers, are you limiting it to any specific kinds?

7 A. No, it's all the same. They are all deposited
8 in that location. Whether they get inhaled or get into
9 the lungs probably depends a little bit on their
10 diameter. For example, the larger their diameter, the
11 less chance they will have of reaching the lung. And,
12 also, it's dependent on how good a person's clearance is
13 or how good their defense system is of trying to inhibit
14 the fibers from getting into the lung.

15 Q. Okay. Do short fibers of asbestos cause
16 disease?

17 A. Sure.

18 Q. Do long fibers in -- Dr. Dodson spoke about
19 this yesterday -- cause disease in humans?

20 A. Yes.

21 Q. Okay. And it's whether they can become
22 respirable and get down to a specific target, so to
23 speak, where they can cause injury; right?

1 A. That is correct.

2 Q. All right. The inside of the lung, the
3 scarring that's caused there, is called what?

4 A. It's called asbestosis. And that is a type of
5 disease in which it usually occurred first in the lower
6 parts of the lung, although, interestingly, it probably
7 occurs just as much in the upper lobes, and that
8 asbestos concentration has been shown by Dr. Dodson and
9 Dr. Churg that you have basically as much asbestos in
10 the lower part of the lung as do you in the upper parts.
11 But, usually, what you see clinically is that the
12 scarring first occurs in the region of the respiratory
13 bronchial and alveolar duct. And that is sometimes
14 referred to as small airways disease or, according to
15 the CAP-NIOSH criteria published in 1982, would be focal
16 grade 1 asbestosis. And that's the earliest stage of
17 the scarring process that occurs.

18 Q. And you mentioned clinically occurring. Is
19 there a -- there is a difference between clinical and
20 pathological asbestosis, for instance?

21 A. There is.

22 Q. Okay. And the difference would be what?

23 A. The difference would usually be the degree of

1 severity because a person, in fact, in the study that
2 Dr. Dodson and I wrote in 1997, where I went back and
3 looked pathologically at the 55 cases of mesothelioma,
4 29 of those had actual pathologic asbestosis; but of
5 that 29, there are only three that actually had clinical
6 asbestosis. So, if a person sees features of clinical
7 asbestosis, which would be evidence of scarring in the
8 lungs identified radiographically, or abnormal pulmonary
9 function tests, or abnormal physical examination
10 findings, that would generally indicate, in most
11 instances, at least grade 3 CAP-NIOSH asbestosis.

12 Q. When does injury begin to occur on a cellular
13 level in a human that inhales asbestos fibers?

14 A. Almost immediately after they are inhaled,
15 that's when the actual injury starts.

16 Q. Okay. And my question is geared towards that.
17 If the injury actually starts shortly after inhalation,
18 whether it be the scarring or ultimately the cancer, is
19 one just suddenly going to see it?

20 A. No, that's not the way it happens.

21 Q. Okay. Well, then, walk us through that, that
22 process of how a clinical asbestosis finally appears.

23 A. Okay. Well, first of all, not everybody that

1 inhales asbestos into their lung are going to develop
2 clinical or pathologic asbestosis. And that is what I
3 like to refer to as individual susceptibility or genetic
4 variability in response to an agent.

5 But in the people that get it, here's how it
6 works. Basically, the body sees asbestos as a foreign
7 material because it's not normally there. So, it
8 initially incites an inflammatory process that, if you
9 look at the experimental studies, is an acute process
10 where you involve cells called neutrophils and
11 eosinophils, which are the acute inflammatory cells in
12 inflammation. And that, over a period of time, gives
13 rise or changes to a chronic inflammatory process, in
14 which you have cells called lymphocytes, plasma cells,
15 and macrophages that are involved in that inflammatory
16 process. And the lymphocytes and macrophages then can
17 start releasing things that are called cytokines and
18 chemokines. And these are mediators of inflammation and
19 mediators of other events.

20 For example, the lymphocytes can produce a
21 fibroblast growth factor, and that is a factor that
22 causes the interstitial cells of the lung, called
23 fibroblasts, to start producing more of the things that

1 they normally produce, which would be collagen and
2 elastin. So, the way it would happen in a person who
3 did develop, say, grade 3 or grade 4 asbestosis would be
4 that they initially had this inflammatory response in
5 the region of the alveolar duct and respiratory
6 bronchial. Over a period of time, that response would
7 expand and would involve adjacent areas of the lung with
8 fibrosis around the bronchi -- I mean around the small
9 bronchials. You then start developing fibrosis between
10 the respiratory bronchial units, and that is referred to
11 as interstitial fibrosis. And then as the progress or
12 the process progressed, it would start causing more and
13 more fibrosis until a certain amount of your lung was
14 destroyed and replaced with scar tissue.

15 And that's how it would happen. And that might
16 take as long as maybe even 40 years for that whole
17 process to happen, and not everybody who actually
18 inhales the asbestos are going to develop that.

19 Q. Is clinical asbestosis the accumulation of the
20 changes that you've just described in an individual?

21 A. Yes.

22 Q. Okay. And in that accumulation of changes that
23 occur on a cellular level, are you able to separate out

1 the accumulation of those changes and say which fibers
2 caused it and which once didn't?

3 A. No, you can't do that.

4 Q. Okay. And the term cumulative exposures, is
5 that applying that principle that all of the exposures
6 contribute to the end result?

7 A. That's the idea, yes.

8 Q. Okay. Is chrysotile unique in that it doesn't
9 cause those changes you just described?

10 A. No.

11 Q. All right. Is chrysotile and friction products
12 that a human inhales unique and does it cause those
13 changes?

14 A. No.

15 Q. The -- we talked about the accumulation of
16 those pathological changes to produce clinical
17 asbestosis or scarring inside the lung. Does scarring
18 happen outside the lung as well?

19 A. It does. There's a variety of ways that
20 scarring occurs or locations that scarring occurs in the
21 lung. Probably, the most common area of scarring would
22 involve the visceral pleura, which is the pleura that
23 covers the lung, and also, not shown here, would be the

1 parietal pleura, which is the pleura that covers the
2 chest cavity. And what happens is that asbestos is
3 translocated from where it is initially deposited in the
4 region of the respiratory bronchial and alveolar duct to
5 the pleura, where it can cause an inflammatory and
6 fibrotic response and end up with scarring. I have
7 several pictures in that book, for example, of visceral
8 pleural scarring.

9 And, then, in the parietal pleura, the scarring
10 is somewhat unique, in that it tends to be localized
11 more on the diaphragmatic surface of the parietal pleura
12 and in the lower part of the chest cavity involving the
13 parietal pleura, usually in the distribution of the
14 ribs. And that scarring is called hyaline pleural
15 plaque.

16 There's a subtype of visceral pleural scarring
17 in which the pleura gets so scarred, that it starts to
18 wrinkle or invaginate in, and that process is referred
19 to as round atelectasis.

20 Q. The scarring or fibrosis on the pleura, whether
21 it be the visceral or the parietal pleura, are
22 chrysotile fibers unique in that they cause those
23 changes?

1 A. No, all types of asbestos can cause that. It
2 turns out that, if you were to analyze the parietal
3 pleura, hyaline pleural plaques by energy dispersive
4 x-ray analysis and x-ray diffraction after digesting
5 that tissue, you would find the dominant fiber to be
6 chrysotile.

7 Q. Is chrysotile fibers that are liberated during
8 the use of friction products unique in that they can't
9 cause those changes?

10 A. No.

11 Q. We talked about the scarring inside and outside
12 the lung. And if we could discuss cancer that occurs
13 inside and outside the lung, what -- no one knows,
14 correct, Dr. Hammar, exactly how cancer happens; right?

15 A. Well, we don't know exactly how it happens, but
16 we do know some things.

17 For example, in every solid tumor that has ever
18 been looked at has shown at least two mutations in what
19 are called protooncogenes and tumor suppressor jeans.
20 There's also a lot of changes in the way the cells cycle
21 works. There's changes in the receptors on the cell
22 membrane of cancer cells. And there are a lot of
23 changes that go on.

1 And these have been described a lot in various
2 publications. In our book, we have a good chapter on
3 the molecular biology of asbestos-related disease. A
4 report by Kemp and Whitesman in 1999, in July, in the
5 *Journal Of Thorax* is an excellent overview of the
6 molecular biology. And there have been several others.

7 I guess the bottom line is that it seems to be
8 a very complex process involving a lot of different
9 systems, and we don't know exactly how it happens. We
10 don't know exactly the time course that it happens, but
11 we kind of know the general stages that it goes through
12 pathologically. And we know that all the solid cancers
13 originate from just a single cancer cell that is formed
14 from the effect of a carcinogen like asbestos that then
15 undergoes this clonal proliferation to form a tumor
16 mass, be it a tumor mass that's in the lung parenchyma
17 or a tumor that arises from the pleura, which would be
18 primarily mesothelioma.

19 Q. Has there ever been any discussions that you're
20 aware of that brake mechanics somehow get cancer
21 differently or don't get cancer differently than other
22 human beings?

23 A. No.

1 Q. And one of the problems I guess in knowing the
2 exact mechanism of how cancer develops -- for instance,
3 in asbestos-exposed individuals -- would be that there's
4 not the technology to have fiber cams when fibers get
5 inhaled; correct?

6 A. You can't see what's going on in there all the
7 time. And we really don't know. We know that there are
8 cases, for example, of mesothelioma and lung cancer that
9 have a very short latency, maybe as short as five to ten
10 years; and we also know there are cases of lung cancer
11 and mesothelioma that have latent periods over 60 or
12 70 years, even. So, we don't know what's going on, and
13 we don't know exactly why there is a difference in those
14 latency periods, for example.

15 Q. And you mentioned -- I'll let you go back up
16 there -- latency. And we've talked about disease
17 process -- I've kind of got it to the side -- the
18 scarring versus the cancer that we're talking about.
19 You mentioned that all the fiber types cause the
20 scarring and the cancer both in and outside the lung;
21 right?

22 A. Yes.

23 Q. Okay. Latency for the disease process of

1 scarring inside the lung and outside the lung versus
2 cancer, is there a difference that has been observed by
3 doctors over time?

4 A. There may be a little bit of difference, but
5 there would be a wide overlap.

6 Q. Okay. And what would the -- I guess there's
7 averages, obviously --

8 A. Sure.

9 Q. -- as to what to expect. And those averages in
10 an individual would depend maybe on a lot of factors;
11 right?

12 A. They could be, yes.

13 Q. Okay. Is one of those factors the dose that an
14 individual is getting of asbestos, the amount of fibers
15 he's breathing?

16 A. That usually is, yes.

17 Q. Okay. And why is that important in looking at
18 that, in an individual case, for latency?

19 A. Well, at least in mesothelioma -- and I'm not
20 sure this is true in every asbestos-related disease, but
21 it may be -- is that usually the higher the dose, the
22 shorter the latency. But that's not a hundred percent.

23 Q. Okay.

1 A. There are exceptions to that rule.

2 Q. And that factors in something again I'm writing
3 down, is individual susceptibility.

4 A. Right.

5 Q. All right. And for whatever reason we don't
6 know, some individuals can get cancer, for instance,
7 from smoking cigarettes, and some don't.

8 A. That is correct.

9 Q. But the general rule would be regarding dose
10 and response is what?

11 A. Is that there's a definite dose response
12 relationship to every asbestos-related disease, which
13 means that the more you're exposed to, the higher your
14 risk would be of developing any of these diseases.

15 Q. Okay. We talked about accumulation of the
16 injuries and separating out which exposures can be a
17 contributing cause and not be. Are you able to separate
18 out if an individual has been exposed to mixed fiber
19 types, amphiboles and serpentine fibers, the chrysotile
20 versus the amosite and crocidolite, which ones
21 contributed and which ones didn't?

22 A. No.

23 Q. Okay. Do you have to also factor, I guess in

1 that kind of discussion, this, latency?

2 A. Sure.

3 Q. Okay. And why is that? I mean, if somebody's
4 exposed to a product that contained asbestos last week,
5 is that going to factor into a physician that's trained
6 in causation assessment?

7 A. Usually not, no, because if you look at
8 cancers, there's probably a very good chance that most
9 of the lung cancer and mesothelioma probably have been
10 in existence on a microscopic scale probably at least
11 ten years before a person is diagnosed clinically to
12 have the disease.

13 Q. In the causation question, because I want to
14 get -- move along to this question specifically,
15 Chrysler has filed a motion with his Honor, asking him
16 to exclude any and all evidence asbestos-containing
17 friction products cannot be a cause or increase one's
18 risk, an individual's risk, of developing an asbestos
19 disease. Okay?

20 A. Okay.

21 Q. Do you agree with that?

22 A. No.

23 Q. Why do you, as a trained physician in the

1 diagnosis and cause of asbestos disease in humans,
2 disagree with that?

3 A. Well, I disagree for several reasons.

4 Q. Okay.

5 A. First is that I think there's overwhelming
6 evidence that chrysotile asbestos can cause injury and
7 cause disease, specifically scarring disease, and
8 neoplastic disease. I think that chrysotile has the
9 ability to do that.

10 With respect to the -- the brake chrysotile
11 issue, I think that there have been a number of case
12 reports which have shown the development of mesothelioma
13 and other diseases in individuals who have been exposed,
14 at least according to the best information we have, only
15 to chrysotile asbestos from friction products.

16 Third would be the studies -- for example,
17 there have been a couple of -- one of the earliest
18 studies in the case reports was a case reported by Dr.
19 Elliott McCaughey, who was the previous chairman of the
20 US-Canadian Mesothelioma Panel, and Dr. Arthur Langer,
21 who is a well-known scientist involved in
22 asbestos-related disease where, in 1983, they -- they
23 reported a 55-year-old man who developed mesothelioma,

1 whose only occupation had been as a brake mechanic,
2 brake repair. And they found only chrysotile asbestos
3 in his lung tissue, and it turned out that ten percent
4 of that chrysotile asbestos had a fiber length of
5 greater than ten micrometers long.

6 Another report about the same time was by
7 Dr. Huncharek, H-U-N-C-H-A-R-E-K, of the mesothelioma
8 that developed in another person who it appeared that
9 only the exposure -- the only exposure they had to
10 asbestos was from chrysotile. And, then, Dr. Lemen, in
11 the *American Journal of Industrial Medicine* in 2004
12 wrote a review article in which he stated that there was
13 evidence of 165 cases of mesothelioma in individuals who
14 had friction product exposure to chrysotile asbestos.
15 So, that's -- that's that information.

16 Then, the other information, actually, that I
17 think is important actually has to do with something
18 that came out of Australia.

19 Q. Well, let me stop you before we get into that
20 because I want give his Honor just a brief overview, and
21 then we'll go into a little bit more specifics.

22 A. Okay.

23 Q. I wrote down "Others have seen it," that

1 exposure to asbestos-containing friction products are
2 causing disease in an individual. Have you seen it?

3 A. I've seen it about ten times, yes.

4 Q. Okay. And those ten times that you've seen it,
5 was that in the context of litigation or outside of
6 litigation?

7 A. Well, I think probably most of those cases did
8 end up in litigation. But there were at least three
9 cases that I saw initially as a treating pathologist.

10 Q. Okay. And I guess one thing to clarify is that
11 you can see a case outside of litigation when it's
12 asbestos caused and it can end up in litigation?

13 A. That usually is what happens, yes.

14 Q. Okay.

15 A. At least with mesothelioma.

16 Q. And you've said ten cases. Now, all of those
17 ten cases, I take it you wrote a report and had it
18 published in the medical literature?

19 A. I didn't report any of those. I did write a
20 report about those cases, but they were not published.

21 Q. So, you're seeing cases in the real world that
22 aren't being published in the literature?

23 A. That is correct, yes.

1 Q. Okay. Are you aware of other trained
2 physicians like yourself that are seeing cases that are
3 similar to that that aren't getting published in the
4 medical literature?

5 A. I think that's probably true of almost any rare
6 diseases: That you see a certain number of cases that
7 you see and you know about these cases and you know some
8 of the interesting information about these cases, but
9 you basically just don't have the time to publish them,
10 or you're just not inclined to publish them.

11 Q. Okay. And maybe one of the reasons is there's
12 just enough cases that have been out there that people
13 know this?

14 A. Well, that's right. For example, I'm very
15 interested in a rare type of mesothelioma called
16 deciduoid mesothelioma. And there now have been about
17 30 cases reported of that. I have about 120 cases. And
18 it comes to a point of whether or not does it add
19 anything to what has already been published. And
20 sometimes it's hard to say.

21 Q. Okay. And sorry to have interrupted you, but
22 the third reason is that you mentioned Australian
23 literature.

1 A. Yeah. There's -- Australia has the highest
2 incidence of mesothelioma in the world, and that's been
3 the case for some time. And, actually, even though they
4 had a number of asbestos mines in Australia, the most
5 notable of which is the Wittenoom, W-I-T-T-E-N-O-O-M,
6 Mine, even though they had these mines, they actually
7 imported more asbestos than they actually produced. So,
8 they actually, at this point in time, have the highest
9 incidence of mesothelioma than any other country in the
10 world.

11 And what they did for a significant period of
12 time was have a mesothelioma registry, in which,
13 initially, they actually had cases or individuals that
14 were interviewed by usually nurses about their
15 occupational history, and all of the cases were reviewed
16 by the Australian Mesothelioma Panel. Later, that
17 changed to the point where they just sent out
18 questionnaires, and the cases were looked over to see if
19 it made sense that they were the correct diagnosis. And
20 what they did was have a list of various occupations
21 that these mesotheliomas occurred in. And that was
22 published in an article written by Dr. James Leigh,
23 L-E-I-G-H, in 2004, where he talked about mesothelioma

1 in Australia and actually had a list -- I think it's on
2 the second to the last page -- of all of the various
3 occupations in which one saw mesotheliomas in. And one
4 of the occupations listed there were either people that
5 repaired brakes or people that manufactured brakes.

6 And, then, in some -- in a report by Dr.
7 Henderson, Douglas W. Henderson, who is the chief of
8 Pathology in the University of Adelaide in South
9 Australia, he wrote part of the WTO document that was a
10 response that was requested by the French government to
11 try to determine whether or not they should allow the
12 importation of chrysotile into the country. And in that
13 document that was written by Dr. Henderson, he talked
14 about the Australia Mesothelioma Registry, he talked
15 about the release of asbestos from friction products, he
16 talked about the concentrations that were generated by
17 various activities. He also mentioned the
18 epidemiological studies that have been published to
19 date. And, then, he did an analysis of what his
20 conclusion was of how many cases of mesothelioma they
21 had in Australia that were directly related to brake
22 repair, which was the dominant thing, or brake
23 manufacturing, which was a lesser number of patients,

1 and came up with a significantly elevated number
2 compared to what they considered to be a background
3 rate, which was one to two cases of mesothelioma per
4 million person years.

5 Q. And we'll get back to those specifically.

6 The Australian experience -- and just, is there
7 a mesothelioma registry here in the United States?

8 A. There is no mesothelioma registry in the
9 United States. There is a thing called the SEER,
10 S-E-E-R, data, and that stand for Surveillance,
11 Epidemiology, and End Points Review. And where I work
12 in Bremerton, for example, we're part of the 13 percent
13 of the hospitals in the United States that provide data
14 to the SEER organization, which keeps track of the
15 incidents of various types of cancers in the
16 United States. And that would be about the only thing.

17 And you have to realize -- for example, like in
18 Bremerton -- we have basically the highest incidence of
19 mesothelioma in the United States, and there would be
20 other places in the United States, probably -- I don't
21 know, maybe the mid west -- where there would be a
22 significant lesser number of cases, so that it's always
23 possible that the SEER data, which only represents,

1 again, about 13 percent of all hospitals in the
2 United States, may be somewhat skewed.

3 Q. I have three major categories. Anything else
4 that you would like to add?

5 A. I don't think so. I think that's basically
6 about it.

7 Q. Okay. And I want to go back in just a little
8 bit of detail because you've referenced some reports.

9 The first one was the McCaughey. Who was Dr.
10 Elliott McCaughey?

11 A. Elliott McCaughey was the -- he worked in a
12 hospital in Ottawa. He was the head of Pathology there.
13 He was the first chairman of the US and Canadian
14 Mesothelioma Panel. On the latest issue of the -- of
15 the AFIP fascicles, he was a co-author with Dr. Hector
16 Battifora on tumors of the serosal membranes, which is
17 basically mesotheliomas and some things that look like
18 mesotheliomas. He was an extremely well-versed
19 individual in the disease mesothelioma and had seen a
20 significant number of cases that occurred in Canada.

21 Q. And going back in time, he reported
22 mesotheliomas fairly early; right?

23 A. Yeah, he was actually second. It was Paul

1 Cartier in 1952 that reported two cases of mesothelioma
2 in the Quebec miners. And, then, Dr. McCaughey, in
3 1958, I think he reported either eight or 12 cases of
4 mesothelioma in the Quebec miners/millers.

5 Q. Now, if a trained physician, in diagnosing
6 asbestos disease and the causes of asbestos diseases,
7 had waited until epidemiology said you can make that
8 connection, would doctors that were seeing, like
9 Dr. Wagner in 1960, would they have been able then, if
10 this is the criteria, to connect asbestos to
11 mesothelioma back in 1960?

12 A. Obviously not. Dr. Wagner's report was a from
13 part of South Africa where they had a crocidolite mining
14 region that he reported in the *British Journal of*
15 *Industrial Medicine*, with Dr. Steggs and Dr. Marchand,
16 33 cases of mesothelioma, and he related 32 of those
17 cases to crocidolite asbestos exposure, even though
18 there was never a control group for that study. It was
19 not an epidemiologic study. It was a multiple case
20 report study, but it was that study that probably more
21 alerted the world's medical attention to the disease
22 mesothelioma than anything else, even though as early as
23 about 1955, the South Africans were seeing cases of

1 mesothelioma in their country.

2 And the unique thing about the Wagner thing was
3 also the fact that where they were located, which was in
4 Kimberly, South Africa, that was actually a TB hospital,
5 and a lot of the patients that were initially
6 hospitalized there, they thought that they had TB. And
7 instead of usually getting better like most of the TB
8 patients did, these people died. It was actually those
9 people that had the mesotheliomas.

10 Q. Okay. And did Wagner, Dr. Wagner, or Wagner,
11 also discuss chrysotile in his 1960 paper?

12 A. He did. And he referred to the cases by
13 Cartier and by McCaughey in the discussion part of that
14 paper. And I think the reason he did that was because,
15 obviously, those were cases in which people were exposed
16 to asbestos, but they had not drawn the link between
17 asbestos and mesothelioma, which Dr. Wagner did.

18 Q. And from your training and reviewing the
19 medical literature after Dr. Wagner's case reports came
20 out, was there a discussion within the medical community
21 of disclaimers, that, "We don't have that epi studies
22 now, so, therefore, let's don't be going around saying,
23 'Asbestos causes meso'?"

1 A. No, there were not those studies. But that
2 information probably had started to initiate those
3 studies.

4 Q. And what happened in the American experience
5 with Selikoff?

6 A. Well, the American consequence is that
7 Dr. Selikoff and his group in Mount Sinai in New York
8 City, they started evaluating a case, a unique --
9 somewhat unique group of individuals, the members of the
10 Heat and Frost Insulation Unions, in the United States
11 and Canada. And it turned out that there was 17,800 of
12 those, and some of those individuals went back as early
13 as the 1920s and 1930s when they did the insulating
14 work. What Dr. Selikoff and these individuals did was
15 look at the incidents of mesothelioma and other
16 asbestos-related diseases in that group of people. And
17 the -- they didn't obviously have everybody die at the
18 same time, so they looked at these at various time
19 periods. For example, the first major reports that came
20 out were in about 1964, 1965, where they showed that
21 about ten percent of the insulators developed
22 mesothelioma, about 21 percent lung cancer, and about
23 16 percent asbestosis. And, then, they kept following

1 this cohort over a period of time, updating the
2 incidents of the diseases that occurred in them over the
3 period of time.

4 Q. And I'm asking you a little bit about this
5 experience that we had in America on this. Did
6 Dr. Selikoff, when he was reporting, insulators are
7 getting sick from asbestos, also report mixed exposures
8 that they're having?

9 A. Sure. He recognized that early on. He
10 initially thought there were individuals who developed
11 mesothelioma from chrysotile exposure only because of
12 the information they had on what types of asbestos were
13 imported into the United States. In 1964, in the *New*
14 *England Journal of Medicine*, he made an observation
15 that, of the types of asbestos that were imported into
16 the United States, crocidolite seemed to be the least
17 imported. And that turned out to be probably very
18 important because, as late as 1991, Dr. Wagner was
19 stating that the only cause of mesothelioma in the
20 United States and other parts of the world was
21 crocidolite asbestos, which was proven to be incorrect.

22 Q. And scientists and doctors can argue about
23 things?

1 A. I suppose so.

2 Q. Okay. Well, and I guess when Selikoff was
3 reporting the American experience with insulators, did
4 he tell the medical and scientific community the
5 insulators had something really funky with their lungs
6 and the reason why they were getting asbestos disease as
7 opposed to other trades?

8 A. No. In fact, he said the exact opposite. He
9 said that the only thing that was important was being
10 exposed to asbestos.

11 Q. And did he say, I think it was, asbestos fibers
12 do not respect job classifications?

13 A. That's right. And for example, in the
14 shipyards that he studied, he showed a number of
15 different occupations in the shipyard and showed that
16 there was a dose response relationship between the
17 asbestos-related disease and how much the individual
18 group of workers were exposed to asbestos.

19 Q. Okay. Dr. McCaughey, real quick, and
20 Dr. Langer, can you read with me, Dr. Hammar, because
21 we've gone through this, just to try to shorten this as
22 much as we could. But you pointed out with McCaughey
23 and Langer that it was important, in your opinion, what

1 they were discussing in this article.

2 A. Right.

3 Q. And could you just review with his Honor
4 what you feel is of significance.

5 A. Well, they started out that by talking about
6 what Merewether's speculation was about asbestosis that
7 would include manufacturers of asbestos, brake linings,
8 where it was then unknown whether or not that happened,
9 that that did happen; and, also, asbestos-related
10 pleural disease happened in brake workers. And, then,
11 they reported a case of mesothelioma in a 55-year-old
12 man whose only exposure to asbestos was from friction
13 products as primarily a brake mechanic; and when they
14 did the autopsy on that individual, they did a fiber
15 analysis on that individual's lung and found only
16 chrysotile asbestos. They did not find any amosite or
17 crocidolite. And they also subsequently published that
18 ten percent of the fibers were ten micrometers or
19 greater in length. And was that kind of an example or
20 probably the first well-documented example of a
21 mesothelioma occurring in a person exposed to friction
22 products.

23 Q. And this is what you're talking about, that no

1 amphibole fibers were found; correct?

2 A. That's correct, yes.

3 Q. And I'm trying to find the ten percent.

4 A. I don't think that's in there. That was
5 actually in a later --

6 Q. Okay.

7 A. -- publication. In fact, that's referenced in
8 Dr. Lemen's chapter in our book.

9 Q. Okay. And you mentioned something interesting,
10 is that Dr. Merewether -- and this hasn't come up yet.
11 I know Dr. Lemen is going to discuss this, or maybe
12 discuss this.

13 Who was Dr. Merewether, just real quick? And I
14 wrote the date down, 1930.

15 A. Well, he was the chief inspector of British
16 factories. And in the 1930s and 1940s, he was looking
17 at the incidence of various diseases, including the
18 diseases that occurred in individuals who worked in
19 these factories that had to do with asbestos. And what
20 he found -- and this was actually published in JAMA, the
21 *Journal of the American Medical Association*, in 1948,
22 that there was an increased incidence of lung cancer and
23 tumors that he referred to, either tumors of the lining

1 of the lung or pleural tumors that certainly one would
2 think probably were mesotheliomas. I guess one might
3 speculate maybe they were pseudo mesos, possibly.

4 But he also compared the incidence that he
5 found in the -- in the factories to the people who did
6 not work in the factories and found an increased
7 incidence of lung cancers and tumors in the lining of
8 the lung in those individuals.

9 Q. Okay. And that was a little bit later than
10 what I put down, 1930?

11 A. That's when he started, though.

12 Q. Okay. And in 1949, he reported increased
13 incidence of those workers getting lung cancers and
14 pleural tumors?

15 A. That is correct, yes.

16 Q. And I wrote down "end product users" because it
17 was discussed in McCaughey and Langer's article,
18 *Mesothelioma and Brake Repair Worker*, and it says
19 Merewether's speculation in 1933 that asbestosis would
20 manifest itself in industries other than what he was
21 looking at. And I wrote "end product users" down.

22 A. Right.

23 Q. In that Merewether study, did he also, I guess,

1 caution that others can be affected than just the
2 population he was studying?

3 A. He did, yes.

4 Q. Including brake linings?

5 A. Yes.

6 Q. And that's because -- or is it because,
7 asbestos fibers do not respect job classifications?

8 A. That is correct.

9 Q. The fibers get in, they can cause disease?

10 A. Yes.

11 Q. Now, the next report which you mentioned and
12 the significance -- you may have covered this -- I think
13 you did real quickly -- but was the pleural mesothelioma
14 in a brake mechanic.

15 A. Right. That's just another case. This is by
16 Dr. Huncharek, who's written about the subject.

17 Q. Okay. And I had highlighted "47-year-old." If
18 we can just briefly in this, what's the significance
19 again of this?

20 A. Again, he thought the only exposure he had was
21 from friction products.

22 Q. Okay and that was just one of the reports in
23 the literature. And one of the discussions -- and we've

1 briefly talked about this -- is epidemiological
2 research. And just within those studies itself, has it
3 also been reported that individuals are getting sick
4 from asbestos friction products?

5 A. It is, yes. They have reported that, that
6 there have been cases where they have looked at people
7 for epidemiologic evaluation, where they have identified
8 mesotheliomas in those individuals.

9 Q. And in looking as a trained physician in
10 asbestos disease and the causes in a specific
11 individual, epidemiological studies are an important
12 thing; right?

13 A. Sure, they are.

14 Q. Okay. And they're an important piece of the
15 puzzle, along with all of the other things that you
16 consider; right?

17 A. I think they are, yes.

18 Q. Okay. The Australian experience -- and we're
19 talking about studies of groups of population,
20 population groups now. Are Aussies' lungs different
21 than American lungs?

22 A. No.

23 Q. Okay. And this is malignant mesothelioma in

1 Australia. This is Dr. Leigh?

2 A. L-E-I-G-H. Leigh, I think, is the correct
3 pronunciation.

4 Q. And in this report or article published in the
5 *International Journal of Occupational Environmental*
6 *Health* -- I'm going to skip all the way to the
7 next-to-the-last page first, there's a chart.

8 A. Right.

9 Q. And it's "Asbestos Exposures as Documented in
10 the Australian Mesothelioma Register from January 1986
11 to the end of December 2001."

12 A. Yes.

13 Q. And segregated are job classifications.

14 A. Right.

15 Q. And one of those that's segregated is brake
16 linings made slash repaired.

17 A. Correct.

18 Q. And we've highlighted this. And what's
19 significant?

20 A. Because this is one of the major categories of
21 individuals who developed mesothelioma in Australia.

22 Q. Okay. And, for instance, right above it,
23 there's a boilermaker --

1 A. Right.

2 Q. -- a category for them.

3 Contained in that report -- and it's a few
4 pages long -- is a sentence that says, "The risk in
5 brake mechanics is also elevated consistent with
6 chrysotile-only causation." And in that one sentence,
7 is there additional information that you'd like to
8 discuss about those findings?

9 A. Well, I think there's information that is the
10 basis for that statement, yes.

11 Q. Okay. Not only contained in the back, right,
12 that we just showed?

13 A. Right.

14 Q. And I have the letter that you referenced on
15 the information quality guideline staff directed to them
16 as well as the World Trade Organization document. Which
17 would be best to put it in context?

18 A. I'd say the WTO document first.

19 Q. Okay.

20 A. That was written in 2001 by Douglas W.
21 Henderson, again, chief of Pathology at the University
22 of Adelaide, South Australia --

23 Q. Okay.

1 A. -- where he was one of the individuals hired by
2 the WTO to evaluate that.

3 Q. And you mentioned quite a few names. For
4 instance, you mentioned Dr. McCaughey. You knew him,
5 didn't you?

6 A. Sure.

7 Q. In fact, you all worked together?

8 A. He was a good friend of mine.

9 Q. Okay. And Dr. Henderson, you know him?

10 A. He and I do a lot of stuff together right now.

11 Q. And I take it, from what I can appreciate, the
12 pathologists that have this experience and have risen to
13 the levels that you are and Dr. McCaughey and others
14 are, you all kind of, I guess, hang out together at
15 seminars and do things like that worldwide; right?

16 A. I guess you might say that, yes.

17 Q. Okay. And has anybody ever stopped you,
18 Dr. Hammar, and said, "You know, you're nuts. You're
19 espousing some opinions here that are just so
20 unscientific and invalid"?

21 A. No.

22 Q. Okay. Are the opinions that you're sharing
23 with his Honor and for those who may see in the record

1 of this case generally accepted within the medical
2 community?

3 A. Yes.

4 Q. Are they based upon reasonable and reliable
5 principles of science and medicine?

6 A. Yes.

7 Q. Are they based upon reasonable and scientific
8 principles and methodology of medicine?

9 A. Yes.

10 Q. In applying that, those fields -- and those
11 fields are quite big -- in an individual case like you
12 do?

13 A. That is correct.

14 Q. The Australian experience -- and you mentioned
15 Dr. Henderson in this article. If we could briefly
16 cover the importance of what Dr. Henderson is telling
17 us. And I had "Automotive" starting with "Friction
18 Products," Page 300. And we can either -- if you can
19 summarize or you can go through what we --

20 A. I don't think we have to go through all of it.

21 Q. Okay.

22 A. I think maybe we can start at that 5.244.

23 Q. Okay. Right here.

1 A. It says, "Literature contains anecdotal reports
2 of malignant mesothelioma among automotive and brake
3 mechanics. However, the question that arises is whether
4 these anecdotal reports are explicable as a chance
5 occurrence of spontaneous or background mesotheliomas
6 among a large population of mechanics, or whether this
7 group of workers has sustained other significant
8 exposures to asbestos, including one or more amphiboles.
9 In other words, the question is whether there is a
10 general increase in the incidence of mesothelioma among
11 automotive and brake mechanics with no other exposures
12 to asbestos."

13 Q. That question sounds familiar.

14 A. Well, I think that's the crux of the whole
15 question, I guess.

16 Q. Okay.

17 A. And, then, I think you could probably -- he
18 then goes through really and talks about the various
19 concentrations that -- of asbestos that are released by
20 various types of activity on brakes. He, then, also
21 goes in and cites several epidemiological studies that
22 have not shown any increased incidence of mesothelioma
23 in the brake mechanics. And, then, he finally does an

1 analysis of the data that he had from Australia on what
2 the Australian evidence was, of whether or not there was
3 or was not an increased incidence of mesothelioma in
4 people exposed to chrysotile asbestos from friction
5 products.

6 And I should have also said that, from the
7 beginning, he states that, as far as he knows, the only
8 type of asbestos that was used in friction products in
9 Australia was chrysotile. And I've asked him this many
10 times, because chrysotile (sic) was a country that did
11 mine asbestos, and primarily crocidolite asbestos, as
12 well as other types, and did use it but not to the same
13 extent that they actually imported it. So, it's my
14 understanding from Dr. Leigh and Dr. Henderson that they
15 only used chrysotile asbestos in the friction products.

16 Q. Okay.

17 A. And, then, in the last part of this, he goes
18 through this analysis of what the mesothelioma registry
19 showed and what the number of mechanics was in
20 Australia. And it's probably worth reading that, 5.253.

21 Q. And let's do that, 5.253. Dr. Henderson's what
22 kind of doctor?

23 A. He's a pathologist, like myself.

1 Q. Pathologist?

2 A. Yes.

3 Q. Trained physician in the diagnosis and cause of
4 asbestos disease; correct?

5 A. He's maybe one of those pathologists that
6 probably is at the real top of the ladder, so to speak,
7 in that, in his 2004 article on lung cancer that was
8 published in a journal in -- in Australia called
9 *Pathology*, which probably is more epidemiology in that
10 article than there is anything else. So, he's a pretty
11 knowledgeable gentleman.

12 Q. Okay. And he discusses what's out there, and
13 you've summarized what he's discussing in his paper, the
14 epidemiology studies and the case reports; right?

15 A. Right.

16 Q. And after reviewing all of that material, this
17 physician, who's at the top, discusses what as far as
18 the opinion, the Australian experience?

19 A. He says -- he states, "Nonetheless, the 1990
20 report for the Australian Mesothelioma Register (AMR 99)
21 records 58 mesotheliomas among brake mechanics with no
22 other exposure to asbestos, during the almost 13-year
23 period between 01 January 1986 and 31 October 1999

1 (total cases with a stated history of asbestos exposure
2 = 2585). Mechanics who frequently or consistently work
3 on brake linings and brake locks represent only a
4 subfraction of the total workforce of mechanics in
5 Australia. If one takes the 1996 census figure of
6 82,827 for male mechanic, this amounts to 58
7 mesotheliomas in 1,062,946 person years (equals 54.6
8 mesothelioma per million person years). If one rounds
9 off the workforce to 100,000 male mechanics, the figure
10 becomes 45 mesotheliomas per million person years. If
11 one then doubles the workforce population to take into
12 account retirees and other workers who moved on to other
13 occupations (although a figure of 200,000 is almost
14 certainly an overestimate because it would include all
15 mechanics, where brake mechanics constitute a smaller
16 sub-class), the mesothelioma rate becomes 22.6 per
17 million person years, well under the rate of 337
18 mesotheliomas per million person years for the Quebec
19 chrysotile miners and millers, but still substantially
20 above the upper limit of the estimated background rate
21 of 1-2 mesothelioma per million person years (about
22 10-fold). One might suspect that mesotheliomas in brake
23 mechanics were clustered in those involved in the

1 grinding, bevelling, and other operations on new brake
2 blocks and brake linings (i.e., brake materials
3 unaltered by heat)."

4 Q. The key, if I'm -- correct me if I'm wrong --
5 is that?

6 A. 22.6 per million person years compared to a
7 background rate of one to two cases per million person
8 years.

9 Q. Okay. And he goes on to say that that rate is
10 less than expected of miners and millers; right?

11 A. Yes.

12 Q. Is that surprising when discussing dose
13 response?

14 A. No.

15 Q. Okay. And, yet, still significantly above the
16 background rate of one to two expected cases?

17 A. Yes.

18 Q. Is that significant, in your opinion?

19 A. Yes.

20 Q. Are the opinions you've given today based upon
21 reasonable medical probability?

22 A. Yes.

23 Q. Can, more probable than not, medically

1 asbestos-containing friction products cause disease in
2 individual cases?

3 A. Yes.

4 MR. WADDELL: Pass the witness, your Honor.

5 THE COURT: Why don't we take our morning
6 recess at this time. We'll break for ten minutes and
7 return back at 20 until 11:00. Our plan would to go
8 roughly until noon, maybe a little after, and then take
9 our luncheon recess.

10 All right. Thank you.

11 (Short recess.)

12 BY MR. TARRY:

13 Q. Good morning, Dr. Hammar.

14 A. Good morning.

15 Q. My name is Sam Tarry. I don't believe we've
16 met before. I appreciate you coming out and answering
17 questions for us today.

18 A. You're welcome.

19 Q. And I also appreciate your candor earlier with
20 respect to what is known and unknown about cancer and
21 especially mesothelioma. So I'm actually going to
22 shorten up some of the questions that I intended to ask
23 you on that front.

1 Without getting into too much detail about what
2 was known at the molecular level, will you agree with me
3 that we don't understand how a fiber starts the process
4 that results in mesothelioma; there are theories about
5 it; we're doing work on that, but we don't understand
6 the whole process?

7 A. I agree.

8 Q. And, in fact, the Kamp paper that you
9 referenced -- this is it. When they talk about what
10 scientists are uncovering, they frame it as just that,
11 as a hypothesis; correct? A radical hypothesis is the
12 heading of that chapter; correct?

13 A. Yes.

14 Q. And there -- this is a thorough paper. There
15 are I think about 193 references. We're not going to
16 talk about all those.

17 But do you know how many, if any, of these
18 reference experiments relied upon by Kamp involved
19 experiments using Grade 7 chrysotile asbestos?

20 A. No, I don't know the answer to that.

21 Q. So then we also don't know how many, if any, of
22 those experiments were involving chrysotile with the
23 same surface chemistry, chrysotile that would had been

1 heated, bound, and resins and actually gone through the
2 process that chrysotile fibers or friction products go
3 through; correct?

4 A. I agree.

5 THE COURT: Mr. Tarry, it just dawned on me
6 that I did not, at least for the record -- I think we
7 decided that the order of presentation, or at least
8 questioning, was going to allowed by Mr. Johnson to step
9 in if he wished.

10 MR. JOHNSON: I can solve the problem. I have
11 no questions at this point based on plaintiffs'
12 examination.

13 THE COURT: I apologize. I just didn't clarify
14 that on the record.

15 MR. TARRY: I apologize, too. That was
16 presumptive. I thought he went after me yesterday.

17 MR. JOHNSON: I think I did. I'm generally not
18 going to have a lot to say given the kind of alignment
19 that --

20 THE COURT: That's fine. I think it made some
21 sense in our initial discussions that you would go
22 second in the line with plaintiffs' witnesses so that
23 the cross-examination could address all those issues.

1 MR. JOHNSON: Okay. That's fine, Your Honor.

2 THE COURT: Appreciate it.

3 BY MR. TARRY:

4 Q. Dr. Hammar, when science doesn't fully
5 comprehend the mechanism of disease, what we talk about
6 is a concept called risk; correct?

7 A. I think that's correct, yes.

8 Q. And to oversimplify it, we identify certain
9 factors that might be risk factors in contributing to
10 the development of a disease; right?

11 A. Fair enough.

12 Q. What epidemiologists -- I know you're not an
13 epidemiologist, but just a couple quick questions on
14 this. What epidemiologists do is to try to test
15 hypotheses about risk factors; correct?

16 A. I agree.

17 Q. Now, there was a discussion about different
18 disciplines. I'm sorry. Could I get this?

19 THE COURT: Sure.

20 BY MR. TARRY:

21 Q. I wanted to find the listing where you talked
22 about the whole picture and the evidence that you
23 looked.

1 MR. TARRY: Chip, can you find that? It had
2 one, two and three.

3 (Pause.)

4 BY MR. TARRY:

5 Q. I'm sorry if this is a stupid question. We
6 have never met. I've never taken your deposition. But
7 do you prescribe medicine in your practice?

8 A. Hardly ever, no. I would never prescribe it to
9 a patient. I might prescribe something for my wife or
10 my son or daughter, but I mean hardly ever.

11 Q. Do you belong to the American Medical
12 Association?

13 A. I don't belong to that association, no.

14 Q. Do you know of something called the
15 evidence-based medicine movement generally within the
16 medical field?

17 A. Yeah, I do know something about that.

18 Q. Okay. Tell us what that is.

19 A. Well, what it is is kind of a catchie phrase
20 for what's been done for basically ions of time. I
21 mean, when I went to medical school, we didn't call it
22 evidence-based medicine. But basically all medicine is
23 evidence based.

1 For example, if you have a patient whose got a
2 pleural effusion and you're trying to figure out what
3 the pleural effusion is caused by, you do certain things
4 to gain evidence of what that pleural effusion was
5 codified. To me that's evidence-based medicine just
6 like it is in a person who comes in with a chest pain
7 and you want to determine if that person has a
8 myocardial infarct. You'll do certain tests, such as a
9 component, EKG, a CPK, things like that to get evidence
10 to determine what disease a person has.

11 So to me this new thing, evidence-based
12 medicine is something that has been going on forever.

13 Q. Right. In fact, it's not really new. Good
14 doctors have been practicing evidence-based medicine for
15 a long, long time?

16 A. Yeah. I don't know. I was just told by a
17 friend of mine who is a pulmonologist that this
18 apparently is something big now no medical schools. And
19 somebody that he interviewed asked him if he practiced
20 evidence-based medicine. He started laughing at him.

21 Q. The reason it seems new is because the AMA
22 actually put together multi-disciplinary panel and they
23 generated a consensus report on evidence-based medicine

1 and a need to just try to and make sure that there was
2 consistency across the country in how we deal with
3 issues of both causation and treatment; right?

4 A. I didn't know that. I guess I know that now.

5 Q. Are you aware of the final publication in the
6 Journal of the American Medical Association?

7 A. I don't look at that -- I don't receive that
8 are journal. And I don't belong to the American Medical
9 Association.

10 Q. Would you be surprised if they had reported
11 that, while you ought to consider everything, you don't
12 disregard any type of evidence; that there are -- there
13 is a hierarchy of certain types of evidence depending on
14 what you're looking at that may be more probative or
15 more relevant for your inquiry?

16 A. I think that's totally true. For example, the
17 American Rheumatologic Society for years has published
18 major and minor criteria for diagnosing collagen
19 vascular diseases. I don't think that's really
20 something new. It doesn't surprise me that they did
21 that. In some respects maybe that's a good thing. I
22 don't know.

23 Q. And it's really not altogether different if

1 we're talking about treatment or identification of
2 etiology of a disease, is it?

3 A. I guess it depends on how you -- what you're
4 trying to do, whether you're trying to determine the
5 etiology of the disease or whether you're trying to
6 determine what the disease is or how to treat it.

7 For example, there's a lot of diseases that we
8 don't know what the etiology is that we do have good
9 treatments for.

10 Q. Let's go back to the concept of risk factor.
11 Okay? A drug really is a risk factor for a cure.
12 There's a good chance hopefully that a good drug is
13 going to have a biological effect which actually cures
14 you or makes you better; is that correct?

15 A. That's true.

16 Q. We call that positive effect or a protective
17 effect?

18 A. Okay.

19 Q. For instance, if I came to you as a doctor and
20 said, what's going to happen to me if I increase the
21 fruit intake in my diet, you would probably tell me
22 that's a good thing; I'd encourage you to do that;
23 because there do appear to be some protective effects of

1 eating fruit; correct?

2 A. That is correct, yes.

3 Q. So that's protective. And then on one side
4 there are also risk factors that can be harmful, that
5 can contribute in a meaningful manner to the development
6 of disease. Those are the bad things that we can
7 identify throughout evidence-based medicine; correct?

8 A. I would agree, sure.

9 Q. And that hierarchy that we're talking about
10 starts with randomized clinical trials. You wouldn't
11 dispute that that's probably, all things being equal,
12 where a doctor ought to start; right? In other words --

13 A. I think, yeah. Randomized clinical trials is
14 probably the thing that has been done today, at least in
15 testing new drugs, for example, or testing new
16 therapeutic devices, etc., etc.

17 I think in the earlier years in medicine it was
18 probably kind of a -- kind of chance and observe or do
19 something and observe to see what happens. But I think
20 randomized trials now are obviously the best way to
21 determine if something is or is not effective.

22 Q. And so that we're not confusing anything on the
23 record, just so the Judge understands exactly what we're

1 talking about with randomized clinical trials, that's
2 actually monitoring in a prospective manner some dose of
3 something in human beings and following them for a
4 certain period of time and watching the result; correct?

5 A. Sure. Like chemotherapy, that would be
6 probably the big thing right now.

7 Q. Now, that is absolutely unavailable in this
8 inquiry we're involved in today because there's no way
9 we're ever going to give people a dose of asbestos to
10 see how they respond; correct?

11 A. Well, you're not going to do that knowingly.
12 What people have tried to do retrospectively is tried to
13 see what dose is necessary to cause a certain disease.
14 For example, in the case of asbestos, I think it's
15 fairly well accepted that it takes less asbestos to
16 cause mesothelioma than it does to cause, say, lung
17 cancer or asbestosis or Grade 4 asbestosis.

18 Q. And that's why observational studies, which
19 tend to call epidemiology, observational studies where
20 you're not controlling the dose, you're just watching
21 people either prospectively or retrospectively,
22 observational studies fall below the trials on the
23 hierarchy of evidence-based medicine; correct?

1 A. I would think that would probably be correct,
2 yes.

3 Q. And specifically what we know is that the best
4 way to look at that is through a systematic review of
5 observational studies. In other words, you don't just
6 want to cherry pick. We don't want doctors answering
7 the question about fruit, for instance, by just picking
8 up one article and ignoring three others; you need to do
9 a systematic review; correct?

10 A. I agree.

11 Q. And below observational studies, we get down to
12 the observation of an individual clinician; right, the
13 descriptive observational techniques such as case
14 reports; right?

15 A. Fair enough, yes.

16 Q. So it's not a matter of asking anybody to
17 exclude anything. We want you to review all of the
18 evidence. But it's not fair to say that in the whole
19 picture everything is of equal weight, is it?

20 A. No.

21 (Pause.)

22 BY MR. TARRY:

23 Q. Are you affiliated with the University of

1 Washington still?

2 A. Yes.

3 Q. And you know Dr. Checkoway, Harvey Checkoway,

4 A. No.

5 Q. You don't know him?

6 A. No.

7 Q. I'll represent to you that Dr. Checkoway is a
8 full-time faculty member there at the School of Public
9 Health and a professional epidemiologist. I think if
10 you reviewed the epidemiology on brake mechanics or on
11 broad exposures, you may be familiar with him as one of
12 the Teschke paper?

13 A. Okay. I know the Teschke paper at least by the
14 first name, but I don't know all the other authors on
15 that.

16 Q. Are you aware of the textbook that he published
17 or he and Dr. Pearce and Dr. Kriebel published entitled
18 *Occupational Epidemiology Research Methods*?

19 A. I'm not familiar with that. That's something
20 that I don't study a lot.

21 Q. He makes a statement that I'd like to get your
22 reaction to.

23 A. Okay.

1 Q. I think this is maybe germane to the question
2 of different types of evidence.

3 And it's not going to let me show this, is it?
4 Let's see.

5 I apologize for the glare. Can you read that
6 at all?

7 A. Yeah. Right there is fine.

8 Q. "It is sometimes helpful to distinguish dose
9 from the concept of biologically active dose, which
10 arises when only some fraction of the dose can produce
11 an effect. Although it is possible to measure tissue
12 levels of some toxic substances at the sites of
13 biological activity, it is generally impossible to
14 determine the amount that is or has been biologically
15 active."

16 Let me stop there. So far, do you agree with
17 the science behind that statement?

18 A. I do.

19 Q. "For example, asbestos fibers can be quantified
20 in lung tissue and related to the severity of pulmonary
21 fibrosis."

22 Do you agree with that?

23 A. I do.

1 Q. "These measurements are actually measures of
2 organ burdens." -- is that legible up there?

3 A. It is.

4 Q. "Rather than biologically active doses because
5 of uncertainties about the time course of burden and the
6 fiber characteristics that initiate or promote disease
7 process."

8 I think that's consistent with your earlier
9 testimony. I just want to make sure.

10 A. It is, yes.

11 Q. All right. And I mentioned that Dr. Checkoway
12 was one of the authors of the Teschke study entitled
13 "Mesothelioma Surveillance to Locate Sources of Asbestos
14 Exposure." Do you recognize that paper?

15 A. I do. I have it right here.

16 Q. I think we'll stipulate on the record, you
17 don't understand that anybody is making a claim that
18 there's anything magical about the lungs of any
19 occupational brake work, do you?

20 A. No.

21 Q. What Dr. Checkoway and others were
22 investigating in this were the different health outcomes
23 of different circumstances of exposure to different

1 types of asbestos in different atmospheres at different
2 doses; correct?

3 A. Yes.

4 Q. And they concluded that that three -- can you
5 read that or do I need to zoom in? I'm on page 164.

6 A. No. I can see that.

7 Q. They found three occupational groups with odd
8 ratios of 5.0 or greater in the initial analysis, sheet
9 metal workers, plumbers, pipefitters, and ship building
10 workers. And ship building we talked about a little
11 bit. That comes as no surprise to you because you know
12 that they have a very high increase of risk of
13 mesothelioma; is that correct?

14 A. They do, yes.

15 Q. They also investigated, because there was
16 suspicion about the release of asbestos fibers from the
17 resin in brake linings, they investigated vehicle
18 mechanics?

19 A. Right.

20 Q. I don't know whether you can see the heading.
21 This is small. But they're actually included in a
22 priori suspect occupational groups; correct?

23 A. I see that.

1 Q. Not only was it not over five, it was .8, which
2 is technically -- that would be protective, but nobody
3 is ever going to make the argument that you ought to
4 have dust inhalation to prevent mesothelioma; correct?

5 A. I think that's correct.

6 Q. So effectively what that is a finding of no
7 increased risk by working on vehicles for this disease?

8 A. That's what they said.

9 Q. And, in fact, it's a little bit lower than the
10 number they were reported for people who are sales
11 clerks, who were in the other occupational groups not
12 necessarily expected to be exposed to asbestos; right?

13 A. Well, that would be true. But I think even
14 though if you look at the confidence intervals in both
15 of those there is an upper end to that. Again, I'm not
16 an epidemiologist. I'll let Dr. Lemen go on there. It
17 seems to me there's a variation there. And it's always
18 going to depend on exactly on what the person did.

19 Q. But although you don't know Dr. Checkoway, you
20 know the University of Washington, and you wouldn't
21 question his scientific credibility here today, would
22 you?

23 A. No. I wouldn't have any reason to.

1 Q. In fact, I think you can see -- and we'll get
2 to Australia in just a moment, but up until the time of
3 Australia, the evidence you were aware of indicated no
4 increased risk of mesothelioma by working on cars or
5 working in in a garage?

6 A. No. And I said that before. That based on
7 those studies that have been done, I don't see any.
8 There have been some criticisms of those studies that I
9 am not in the position to discuss one way or the other.

10 Q. Dr. Roggli is someone you know?

11 A. I do.

12 Q. Is he also on the U. S./Canadian mesothelioma
13 panel?

14 A. He is, yes.

15 Q. And he's someone who you respect?

16 A. Sure.

17 Q. And he comes to a different conclusion on the
18 question of before the Court today about the risk of
19 malignant mesothelioma and garage workers and brake
20 mechanics, do you agree?

21 A. He does.

22 Q. That was a bad question. Do you agree that he
23 comes to a different conclusion?

1 A. He has, yes.

2 Q. Let's talk about Australia.

3 A. Okay.

4 Q. It's true, isn't it, that the impact on your
5 thinking of these claims made by Dr. Henderson and
6 Dr. Leigh from Australia is really even more important
7 than you testified to today? In other words, the
8 Australian data is extremely important to you, isn't it?

9 A. I think it is. At least to me that would be a
10 epidemiologic evidence that there was an increased
11 incidence of mesothelioma in the people that were
12 dealing with brake products, primary in brake mechanic
13 repair and less likely as actual manufacturer of the
14 brake shoes.

15 Q. And, in fact, in the process of weighing all of
16 the evidence, which you've always done -- you didn't
17 just start doing that today, did you?

18 A. No.

19 Q. In the process of weighing all of the evidence
20 on this question of whether doing this work with this
21 type of product under those circumstances one would
22 expect increase rate of mesothelioma, until Australia
23 you're opinion was that there is no evidence of an

1 increased risk?

2 A. Well, that was correct based on what had been
3 published by those people and based on their opinions,
4 yes.

5 Q. Just so I don't misstate it, let me show you
6 testimony you have gave in Texas. There was a
7 hypothetical by Mr. Lopez.

8 MR. WADDELL: Your Honor, I know this is kind
9 of a freelance hearing, so to speak. I object to the
10 use as improper on cross-examination, impeaching his
11 testimony. He's done nothing but agree with what he
12 said.

13 THE COURT: Overruled.

14 BY MR. TARRY:

15 Q. Just to be clear, I don't want to be unfair. I
16 understand the answer. I just want to make sure the
17 record is clear about the prior testimony about the
18 importance of the Australian data. All right?

19 A. Okay.

20 Q. Mr. Lopez asked you: "For example, if you've
21 been hired, say, in 2001 in this case by a defendant
22 representing a brake or clutch manufacturer, your
23 opinion would have been that Mr. McDowell's exposure to

1 friction or clutch products was not a causative factor
2 in the development of his mesothelioma; correct?"

3 And you said: "No, I think it would be exactly
4 the opposite."

5 He says: "I'm talking about over two years
6 ago."

7 "ANSWER: Oh, two years ago.

8 "QUESTION: Yes. I said let's just say roughly
9 2000."

10 Your clarifying question was: "You mean before
11 the Henderson document came out?"

12 "Yes, the year 2000."

13 And you responded: "Okay. Before the
14 Henderson document came out, I would say that I could
15 not prove that that was a cause of mesothelioma."

16 Is that still your opinion?

17 A. That's correct, yes.

18 Q. So it's difficult to overstate how important
19 then in the weighing of all the evidence this Australian
20 information is to you when you testify to this Court
21 today?

22 A. It is. And I think there it kind of suggests
23 that something is wrong, either that the Australian

1 information is wrong or maybe that the information
2 that's been published by these various individuals -- I
3 think there's been six individual reports and three META
4 analyses that something is wrong with their data. I
5 guess that's something that the epidemiologists have to
6 decide.

7 I still think, though, that if you look at it
8 from just a pure point of view of the development of
9 mesothelioma and exposure to asbestos that it would be
10 very hard to not conclude that, say, the case of
11 McCaughey and Langer reported, that that wasn't an
12 example of an asbestos-induced mesothelioma, just like
13 the one Huncharek reported.

14 So despite the epidemiology, it would be very
15 hard for me to conclude from those case reports that
16 asbestos were not the cause. I believe in the
17 epidemiology. I think it's very important. And I think
18 that somehow that there seems to be a big significant
19 discrepancy here with respect to what both Henderson and
20 Leigh have published versus what has been published in
21 this country primarily and Canada also.

22 Q. Thank you for clarifying that, because that is
23 something that this Court needs to understand. There is

1 a huge discrepancy between the risks reported by
2 Dr. Henderson and Leigh out of Australian and all of the
3 other global research that was available to you prior to
4 the time that came out. They're not even close?

5 A. They're not even close, that's correct.

6 Q. Either everybody in the world who looked at
7 this issue was wrong and there is a risk, and not only
8 is it a risk, it's a really, really high risk of
9 mesothelioma, or there's something wrong with Henderson
10 and Leigh's claims?

11 A. It's a really, really high risk of
12 mesothelioma. Again, you look at the total number of
13 cases, though. It's not a real big risk that way.
14 Because still, of the number of cases of mesothelioma
15 reported in these brake individuals is relatively few
16 compared to, say, Puget Sound Naval Shipyard. But it's
17 still a significant number because it's a horrible
18 disease.

19 Q. I'm not trying to beat a dead horse. So the
20 Judge is clear, those case reports that you find
21 important, Huncharek or anybody else's, they were not
22 important enough to you, using evidenced-based medicine
23 or whatever rubric you employ prior to the information

1 from Australian coming out, would have led you back then
2 to giving the opinions that you're prepared to give
3 today that it increases the risk, working with brakes
4 increases the risks?

5 A. That is correct.

6 On the other hand, though, the reason that I
7 would say that I couldn't prove it is because I would
8 not have any epidemiologic evidence. But if you ask me,
9 though, how do -- what would be my opinion with respect
10 to the case that McCaughey and Langer reported and the
11 case that Huncharek reported with respect to what would
12 cause that individual's mesothelioma was, I would say
13 chrysotile asbestos. I wouldn't have any doubt about
14 that. I'm just saying that sometimes you can't always
15 prove things from an epidemiologic studies. I can give
16 you another example that's even better.

17 Right now the only known cause of mesothelioma
18 in the United States is asbestos. That's the only known
19 cause. Myself and a number of other individuals
20 happened to believe very strongly that there is another
21 cause of mesothelioma in the United States. And what
22 that is therapeutic radiation that is given to other
23 cancers. And there have been epidemiologic studies to

1 see if there's any evidence of an increase incidence of
2 mesothelioma in people receiving therapeutic radiation.

3 And one of the biggest groups that they've
4 actually studied are women who have breast cancer who
5 then received therapeutic radiation for breast cancer.
6 There is no epidemiologic evidence that therapeutic
7 radiation causes mesothelioma. But I have seen at least
8 five cases of lymphomas who have been radiated where
9 there's no history of exposure to asbestos who developed
10 mesothelioma.

11 I saw one very sad case of a young child who
12 developed a tumor in the pineal gland who then had
13 neuraxis radiation who developed mesothelioma. I report
14 in that book, for example, a case of a child who had
15 Wilms tumor who received therapeutic radiation who
16 developed mesothelioma.

17 But here's another example where epidemiologic
18 does not show that there's an increased risk of this
19 disease where it's clearly evident to myself and a lot
20 of other people that therapeutic radiation is a cause.
21 I would say there's nothing different between this and
22 potentially, say, chrysotile asbestos in friction
23 product exposure. Maybe the risk is small enough that

1 the epidemiologic studies cannot evaluate this. And
2 that's something that I'm not an expert on.

3 Q. Thank you for clarifying that. And we'll deal
4 with the technical epidemiologic questions with a
5 professional epidemiologist.

6 But for your testimony to the Court today, I
7 want to be clear. The reliability, the scientific
8 reliability and integrity of both the data and the
9 methodologies employed by Dr. Leigh and Dr. Henderson in
10 reporting from what they reported from Australian
11 directly impacts the reliability and the scientific
12 credibility of your testimony to this court today on the
13 question?

14 A. That's true.

15 Q. You testified about latency. It's important,
16 is it not, when considering mesotheliomas and whether
17 particular exposure may have played a role in developing
18 mesothelioma to look at the latency factor?

19 A. It is, yes.

20 Q. If I showed up at your office today with
21 mesothelioma and I told you that I changed my brakes
22 yesterday but I had some other exposures ten or 20 years
23 ago, the ten or 20 years ago exposure would be the one

1 that you would focus on; you would not be concerned
2 about what I did yesterday?

3 A. That is correct.

4 Q. And the reason is because there was not
5 sufficient latency between the exposure and the
6 development of the disease, the progression of the tumor
7 time?

8 A. That is correct.

9 Q. And you use -- what is the general latency --

10 A. Well, the Helsinki consensus criteria, which I
11 have a copy if anybody wants it -- they give the
12 shortest latency as ten years. The shortest latency that
13 I know for asbestos that has been published in the
14 medical literature is five and a half years. The
15 shortest latency for therapeutic radiation,
16 interestingly, is only a year. The longest latency that
17 I have seen myself is 72 years. And in the article that
18 Dr. Dodson and I reported in 1997, it ranges between 25
19 on the short end, 72 on the long end. 90 percent, 95
20 percent were between the 30 and 60 years. And the
21 average was 47 years.

22 Q. All right. In any event, if Dr. Leigh and
23 Dr. Henderson are making claims about cases of

1 mesothelioma where the suspected cause or the only
2 identified exposure is from professional brake work or
3 from the manufacturing of brake linings, they ought to
4 take latency into effect, shouldn't they? Shouldn't
5 they record the time when the people were exposed?

6 A. Well, I suspect that they probably should have.
7 They might even have that information, although it might
8 not be published. I don't know.

9 (Pause.)

10 BY MR. TARRY:

11 Q. You're aware, Dr. Hammar, that -- can we switch
12 this back to my computer?

13 THE COURT: I can give it a shot.

14 (Pause.)

15 THE COURT: Is it working now?

16 MR. TARRY: Our screen is still blank.

17 (Pause.)

18 MR. PRICE: Mr. Gribben is an IT person from
19 our office. If I could ask him to join in the
20 assistance?

21 THE COURT: Sure.

22 BY MR. TARRY:

23 Q. Are you aware that Dr. Leigh gave sworn

1 testimony in American litigation about the process used
2 in making these claims of increased incidence from
3 Australian?

4 A. I heard that hearsay. I heard that hearsay
5 that two U. S. attorneys deposed him at a time that I'm
6 not sure exactly it was. And the only thing I heard was
7 that some of the cases -- and I don't know how many --
8 that he had initially stated were exposed only to
9 friction product asbestos chrysotile was not correct,
10 that they had some other exposures. As far as how many
11 cases, I never heard that number. And exactly when this
12 happened, I don't know that either.

13 Q. It would be important to you, as you just
14 stated, though, to know whether or not they considered
15 latency in reporting these numbers?

16 A. Well, I think with respect to the other
17 exposures, yeah, that would be important, yes.

18 Q. Well, wait a minute.

19 A. It would be important to know if what the
20 latency was for any exposure they had to asbestos.

21 Q. Let me let you listen to what Dr. Leigh said
22 about latency in these numbers.

23 A. Okay.

1 (Whereupon the following video clip was
2 played.)

3 "QUESTION: What I want to ask you about this
4 particular case is you'd agree that the work exposure,
5 possible exposure to brakes in work in the garage over
6 the last ten years would be -- have sufficient latency
7 to be included as a --

8 "ANSWER: Well, it's a very short latency, very
9 short latency, but it's possible. It can occur in less
10 than ten years. The shortest latency we had was four
11 years.

12 "QUESTION: So you haven't -- you included any
13 exposure regardless of latency in your 43 --

14 "ANSWER: Yes.

15 "QUESTION: You've got to let me finish --
16 vehicle mechanics without other exposure?

17 "ANSWER: Yes.

18 "QUESTION: And you would agree with that, that
19 exposure actually occurred, since it's ten years and he
20 was diagnosed in 1992, sometime between 1982 and '92;
21 right?

22 "ANSWER: Yes.

23 "QUESTION: Okay."

1 BY MR. TARRY:

2 Q. They just pulled together all the reports they
3 could regardless of the time of exposure. That's what
4 he said; right?

5 A. I don't know. Could you ask that again. I'm
6 not sure.

7 Q. They reported this number 58, then later 43 --

8 A. Okay.

9 Q. Dr. Leigh.

10 A. Right.

11 Q. They reported that without regard to when the
12 exposure occurred. Is that what he just said?

13 A. Did he say that for all the cases or just the
14 one case?

15 Q. That was one example of somebody with a latency
16 under ten years that was included. But we can watch it
17 again. The question was: Did you consider latency?
18 Would it be helpful to watch that again? I know I was
19 flipping back and forth.

20 A. Yeah.

21 Q. Let's watch that again.

22 (Whereupon the following video clip was
23 played.)

1 "QUESTION: What I want to ask you about this
2 particular case is you'd agree that the work exposure,
3 possible exposure to brakes in work in the garage over
4 the last ten years would be -- have sufficient latency
5 to be included as a --

6 "ANSWER: Well, it's a very short latency, very
7 short latency, but it's possible. It can occur in less
8 than ten years. The shortest latency we had was four
9 years.

10 MR. TARRY: The next question.

11 (Whereupon the video clip continued.)

12 "QUESTION: So you haven't -- you included any
13 exposure regardless of latency in your 43 --

14 "ANSWER: Yes.

15 "QUESTION: You've got to let me finish --
16 vehicle mechanics without other exposure?

17 "ANSWER: Yes.

18 "QUESTION: And you would agree with that, that
19 exposure actually occurred, since it's ten years and he
20 was diagnosed in 1992, sometime between 1982 and '92;
21 right?

22 "ANSWER: Yes."

23 BY MR. TARRY:

1 Q. They didn't consider latency?

2 A. Seems to me that he's saying two things there.
3 He's talking about an individual case where he said that
4 the latency between not that individual case, that the
5 '82 to '92 was a ten-year latency. And he said the
6 shortest they had it and latencies -- and I don't know
7 whether this was in general or for friction products --
8 was four years. And then they -- asked about the
9 43 cases. But he didn't say what the latency was. He
10 didn't say which ones had a short latency and which ones
11 had a long latency. He didn't say whether he really had
12 the data for that.

13 Q. That's exactly the point, isn't it? He didn't
14 offer any information on latency because that's not what
15 they did when they were reporting these numbers?

16 A. Well, it wasn't like you said, though. It's
17 not that he didn't report it, though. What he was
18 talking about was a single case. Now, what the
19 latencies were on the other 43 cases, I have no idea.
20 Unless you know those numbers, I don't know what those
21 numbers are.

22 Q. We may talk about that depending on how much
23 time we have or we can talk about it with Dr. Lemen.

1 But is it your testimony that he considered
2 latency or he didn't consider latency when selecting
3 cases to claim were mechanics whose only exposure, only
4 cause of exposure could have been from brakes?

5 MR. WADDELL: I apologize for interrupting,
6 Mr. Tarry. It's getting argumentative as to what
7 actually was said and played. Is he interpreting -- I
8 apologize, Your Honor. It is what it is.

9 THE COURT: The witness' interpretation is I
10 think the subject of the question. And I'll allow that
11 in that context. How you interpret, if you're able to,
12 what Dr. Leigh was saying.

13 THE WITNESS: I can't interpret it.

14 Here's the deal about latency, though. It's
15 not as clearcut as people think it is. For example,
16 some people say that there's an inverse relationship
17 between latency and dose. But that's not always the
18 case.

19 For example, in the Dodson paper that he and I
20 wrote, I can show you examples of people that had short
21 latencies who had short or minimal exposures. And what
22 Dr. Leigh was saying in his, if I understood it, that he
23 has short latencies as earlier as four years. And there

1 had been, by Dr. Roggli's reports some people who had
2 had sarcomatoid mesotheliomas who have had very short
3 latencies also. And if you look at the only data
4 published on doubling times of sarcomatoid
5 mesotheliomas, there can be some incredibly short
6 latencies. So it seems to me that there's more to this
7 issue than just purely the number of years.

8 And the other issue that also comes up that's
9 been published in that Kamp and Weitzman article and
10 that has to do with the fact that asbestos is not only
11 an initiator of cancer, it's also a promoter. So even
12 if the person had a latency, say, of ten years, that it
13 is still possible that continued exposure to asbestos
14 would contribute be to the development of that tumor.
15 And that's in the Kamp and Weitzman article.

16 THE COURT: Just for clarification, though, as
17 you listen to the testimony that was just played, were
18 you able to determine from that whether or not the Leigh
19 or Henderson study considered latency or not?

20 THE WITNESS: I can't tell what that said.
21 Seems to me there was one case that they reported where
22 there was a suggestion of ten-year latency and that the
23 shortest they had was four. But I can't tell what they

1 meant by that other than -- maybe I'm not getting it,
2 but I don't know what they meant.

3 MR. TARRY: We're going to stipulate to put the
4 transcript in and let the Court decide instead of
5 beating this to death.

6 THE COURT: All right. I suspect we might
7 revisit this with some other folks.

8 MR. TARRY: Good likelihood.

9 BY MR. TARRY:

10 Q. Dr. Henderson first made these claims --
11 actually, I don't need to put it on the overhead. Don't
12 worry about it, Your Honor.

13 THE COURT: It's available to me.

14 MR. TARRY: Okay.

15 BY MR. TARRY:

16 Q. He first made these claims in March 2001 before
17 the Word Trade Organization in what was a trade dispute
18 involving the importation of chrysotile asbestos; is
19 that correct?

20 A. That is correct.

21 Q. And Dr. Henderson believes, like you do, I
22 believe, that chrysotile, even short fiber chrysotile
23 may be carcinogenic and we shouldn't assume that it's

1 safe in the abstract; correct?

2 A. That is right.

3 Q. He was advocating that position in a political
4 forum; right? And these were numbers generated for the
5 first time ever in public to support his hypothesis;
6 right?

7 A. As far as I know, that is correct, as far as
8 published numbers. I had not seen that before. I had
9 talked to Doug Henderson before about chrysotile
10 asbestos and mesothelioma causation. And he said his
11 opinion, like I have for many years, that chrysotile
12 caused this mesothelioma in general.

13 Q. And the number 58 -- we need to make this clear
14 on the record. The number 58, Henderson was not saying
15 these are profession brake mechanics. He was saying
16 these are professional brake mechanics with no exposure
17 that I know of based on information plus some people who
18 worked in mills where they fabricated from raw asbestos
19 the actual product; right?

20 A. That is correct.

21 Q. And, in fact, the category -- the Leigh paper
22 illustrates that the category they were referring to was
23 called "brake linings, made, repaired; right?

1 A. That's correct.

2 Q. Is that a category that you have ever heard of
3 under any of the standardized occupational codes? Do
4 you know what --

5 A. Yeah, I understand what you're saying. I don't
6 think I've ever seen that before.

7 Q. It's not, is it?

8 A. No.

9 Q. There's no, either in Europe or the United
10 States, there is no standardized occupational code for
11 brake linings, made and repaired?

12 A. That's true. But it turns out that Dr. Leigh
13 did actually in a letter to the EPA indicate that group,
14 exactly how many were involved in the manufacturing
15 versus the repairs. So he did know that information.

16 Q. Dr. Leigh and Dr. Henderson were the ones
17 responsible for the creation of that category in the
18 mid-1990s in the Australian registry, weren't they?

19 A. I have no idea if they were.

20 Q. If we went and looked at the data prior and, in
21 fact, subsequent to the time of these reports, there is
22 no such category reported; they've gone back to the
23 occupational codes that they used prior to this; right?

1 A. First of all, I don't know if Dr. Leigh and
2 Dr. Henderson did that or not. I have no way of knowing
3 that.

4 But with respect to doing that I don't think
5 it's all that -- it's not that surprising that they
6 would combine categories for individuals who were
7 exposed primarily to chrysotile asbestos in this type of
8 work. I mean, they were either brake mechanics that
9 they were exposed or they actually were producing the
10 product themselves. I mean, in a way I think you could
11 have exposure to chrysotile asbestos in either one of
12 those situations and that perhaps would not be
13 surprising.

14 Q. All right. Would it be concern you if the
15 timing of this creation of the special category happened
16 to coincide with the time that Dr. Henderson and
17 Dr. Leigh started doing litigation consulting in
18 Australia?

19 A. You know, that's not fair. Dr. Henderson is
20 probably one of the smartest and honest guys in the
21 world I know. And Dr. Leigh is the same way. I don't
22 know him as well as I do Dr. Henderson.

23 But, you know, that is -- if that was the case,

1 I would be very, very disappointed. And I just don't
2 think -- I've never known Doug Henderson to do anything
3 that's dishonest. And I know him as well as anybody as
4 a pathology.

5 Q. No accusation of dishonesty --

6 A. That's what it sounded like, though.

7 Q. Perhaps opportunism in the advancement of a
8 political agenda, so just to be clear.

9 A. But that's not fair in science. If you're a
10 good scientist and you're going to start lying and start
11 doing things that really distort the truth, then you
12 better get out fast. Because that's exactly what that
13 is all about. I mean, if they're doing that -- and I
14 can't see how it would be any gain to them from a
15 financial point of view unless you say, well, they get
16 more cases that they're involved in legally. I just
17 can't believe those two guys would do that, especially
18 Dr. Henderson.

19 Q. Well --

20 A. I hate -- you know, it really bothers me when
21 somebody accuses or makes an accusation like that
22 because I don't know -- Dr. Henderson is just not that
23 type of person in my.

1 Q. And I don't intend to make an accusation. And
2 I will withdraw the implication.

3 Let me ask you the question. Are you aware of
4 that category?

5 A. I was not aware of that category. I had always
6 seen it as that category in the articles that I had
7 read.

8 Q. Here's the question. The Court can draw
9 whatever inference it decides to. Subsequent to the
10 deposition Dr. Leigh gave -- and we're going to look at
11 some more of these clips -- where he was forced to
12 withdraw a number of these, are you aware of a change
13 being made in this category if they report in the
14 register?

15 A. No. What I heard about that was is that when
16 Dr. Leigh looked over some of the additional
17 occupational information that he was given, that he
18 would take those people out of one of those categories
19 because they had been exposed to asbestos in another
20 setting. That's the only thing that I had ever heard.

21 Q. The integrity of the day is important. And you
22 would not advocate the publication of inaccurate or
23 unreliable data, would you?

1 A. No. In fact, Dr. Leigh in his article -- I
2 mean his letter to the EPA actually talked about some
3 possibilities based on some of the variations that could
4 exist.

5 Q. This is the book we talked about earlier that
6 your name is on the front of. You edited it.

7 A. It is.

8 Q. What did you undertake to insure the accuracy
9 and reliability of the statements and data presented in
10 this book?

11 A. Basically the references that were given.

12 Q. Page 241. And the chapter "Epidemiology of
13 Asbestos Tolerated Diseases," can you read this?

14 A. I can.

15 Q. "Henderson reports that 58 mesotheliomas were
16 reported among Australian brake mechanics, mechanics,
17 having no other exposures to asbestos?

18 Is that accurate in light of what we just
19 talked about, that number 58 had already been disowned
20 by Leigh and it was actually 43?

21 Let me ask it a different way. Did
22 Dr. Henderson ever represent that the 58 were brake
23 mechanics?

1 A. Well, the way he said it, he said it exactly --
2 he says...

3 Let me go back.

4 (Pause.)

5 THE WITNESS: He said this: He said,
6 Nonetheless, the 1999 report, the Australian
7 mesothelioma register, he references that, records 58
8 mesotheliomas among brake mechanics with no other
9 exposures to asbestos during the almost 13-year period
10 between '01 January 1986 and 31 October 1999. That's
11 what he said.

12 BY MR. TARRY:

13 Q. What we know now is that it's not 58 brake
14 mechanics, at most it was 43, and the rest were people
15 who worked in the fabrication plants; right?

16 A. Let me see if I can -- I don't know if that's
17 where the rest of them worked there or not. Let me see
18 if Dr. Leigh's letter to the EPA states that.

19 (Pause.)

20 THE WITNESS: Dr. Leigh says in his letter
21 addressed to the EPA, 1 October 2003, states in the
22 fifth paragraph states that: In the cases 1986-2001,
23 78 cases, and then in parentheses, out of three -- 3,956

1 responding to the occupational history questionnaire or
2 providing a history gave a history of asbestos exposure
3 in brake lining repair or manufacture, 73 were brake
4 lining repair, five manufacture. In 43 of these brake
5 lining repair was the only source of asbestos exposure.

6 BY MR. TARRY:

7 Q. So by 2003 when Dr. Leigh is writing a letter
8 -- by the way, this is also a letter advocating a
9 political position; correct? He was writing to the EPA
10 encouraging them not to withdraw a document called the
11 Gold Book. That's the reason he wrote this letter;
12 right?

13 A. Yeah. But I think, again, though, I would have
14 an opinion that he had a different reason for writing
15 the letter. My reason would be that he disagrees with
16 the six reports and three META analyses that were
17 published in the United States.

18 Q. We don't need to disagree. I'm not reading
19 anything nefarious into this. He could be advocating
20 public health to the best of his ability.

21 The point is is that it was -- he wasn't
22 submitting this for peer review. He was submitting it
23 for political reasons?

1 A. What reason would it be? Maybe he was sending
2 to it to try to tell the EPA that there actually had
3 been a mistake and that in Australia they actually did
4 have an excess number mesotheliomas due to these people
5 who worked with brake linings or brake repairs. Isn't
6 that just a logical of a reason that he did it?

7 Q. May be fair enough. Back to our original
8 question. We're down to 43 now.

9 A. Okay.

10 Q. All right. So the 58, which you report in your
11 textbook --

12 A. Well, that's what was taken out of the WTO.
13 Maybe Dr. Lemen, who wrote this chapter, had not seen
14 that letter. I hadn't seen that until just recently.
15 And that was published in the actual Australian register
16 itself. So there would be no reason to believe that
17 that be was not correct.

18 Q. The data was published without the discussion?

19 A. Yes.

20 Q. Now, Dr. Leigh in his article "Malignant
21 Mesothelioma in Australia" was published in the
22 *International Journal of Occupational and Environmental*
23 *Health* --

1 A. Okay.

2 Q. -- made some comments that you discussed -- or
3 he provided the data again. I think we saw that chart.

4 A. Yes.

5 Q. Is this a real peer review journal? Are you
6 familiar with that journal?

7 A. I'm not familiar with that journal. Let me
8 see. I don't know. I would assume that it's peer
9 review.

10 Q. It's an online publication, isn't it?

11 A. I don't know that.

12 Q. You don't know who the owner, who the two
13 owners of the journal are?

14 A. I don't, no.

15 Q. If this article wasn't subject to rigorous peer
16 review, it would impact your ability to rely on it or
17 your willingness to rely on it, wouldn't it?

18 A. I guess what really would effect my ability to
19 rely on it, I guess, is basically what the correct
20 information is. And I guess if this is not correct,
21 yeah, it could potentially have an effect. But I think
22 Dr. Leigh in that letter to the EPA made some statements
23 that...

1 It states the following: "It should be noted
2 that this is a different analysis to the case reference
3 studies included in the META analysis of Wong." And
4 they have a reference in 2000. "The comparison in these
5 studies estimates odds ratio (relative risk
6 approximation) forever employed as auto mechanic
7 compared to never employed as auto mechanic and
8 effectively compares risk as auto mechanic to risk
9 average over all other occupations, which is not the
10 same as risk compared to background environmental
11 exposures."

12 Then he says: For comparison for such studies,
13 the cohort equivalent would be approximately 73. And
14 then it has the number 49,000 ten times 16 times 70,
15 average lifetime risk for Australia. Then he puts in
16 parentheses all occupations but equal to 0.6 percent,
17 slash, 0.22 percent equals 2.96, which is still clearly
18 greater than unity.

19 And then he states: If restricted to auto
20 mechanic work where this was the only asbestos exposure
21 the corresponding figure would be -- I'll just give you
22 the number -- 1.7, which is greater than unity.

23 BY MR. TARRY:

1 Q. And you would hope that those statements were
2 subjected to credible peer review?

3 A. Yes. Well, again, maybe I believe in basic
4 honesty more. I can't believe that people are going to
5 really write something that's not true. That's like,
6 you know, basically hanging your neck out -- or whatever
7 that cliché -- is to dry. If you're not going to be
8 honesty in what you publish, the consequences are
9 potentially disastrous.

10 Q. Can I get an agreement from you right now that
11 during this whole lining of questioning I'd be more
12 comfortable using the word "accurate" instead of
13 "honest"? I'm not impugning anyone's honesty. I'm
14 talking about the accuracy of data. Is that fair?

15 A. That's fair.

16 Q. Can we use that term instead of "honesty"? I'm
17 not accusing anybody of lying, and I don't want to be.

18 A. Yes. But you're saying that they did that for
19 political reasons. If that hasn't to do with honesty --
20 I mean, we know enough about politicians nowadays to
21 know why they do things one way or the other. So it's
22 not about facts sometimes, it's about what's going to
23 get them the most, I guess, whatever.

1 THE COURT: Let me just say that I don't know
2 that an attorney or a witness should really be
3 commenting on the motives of any other person unless
4 those are clearly identified in some meaningful way. So
5 trying to go figure out why he said what he said when he
6 was testifying or why he wrote what he wrote when he was
7 submitting papers to the EPA, we'll leave that to the
8 folks who wrote it.

9 MR. TARRY: Thank you, Your Honor.

10 THE COURT: And also, I don't think anyone is
11 attempting to attack the honesty of folks.

12 MR. TARRY: Thank you.

13 BY MR. TARRY:

14 Q. The point is, Dr. Hammar, is that there's a
15 reason why we have something called peer review process;
16 correct?

17 A. Sure, there is. Absolutely.

18 Q. And it's important because even the best
19 researchers are human beings and they can make mistakes.
20 They can have data that's inaccurate or conclusions that
21 haven't been well thought out that the peer reviewers
22 can catch and there can be a correction; right?

23 A. Yeah. But just take, for example, okay, say

1 that this wasn't a peer review, and they were going to
2 peer review it. Say they were just going to send it to
3 Jane Tata, and send it to your friend at the University
4 of Washington.

5 Now, you're going to tell me that they don't
6 have a bias? You're going to tell me that they have a
7 way that they can tell what Dr. Henderson and Dr. Leigh
8 is saying is honest -- drop that word -- is accurate or
9 not accurate? Basically, the only way they can do it is
10 -- from a peer review is to determine whether the
11 statistics are correct or not. If they think that the
12 statistics are correct or I guess they would request,
13 say, that they don't believe what Dr. Henderson or
14 Dr. Leigh said was accurate, and they want a list of all
15 of the cases, and they want a list of when the person
16 was first exposed, when he or she was last exposed, the
17 time that they first developed symptoms of mesothelioma
18 or the time they were diagnosed, I think that would be
19 probably a reasonable request.

20 And maybe then in contrast -- or not in
21 contrast. That's not the best word. Somebody should
22 have also asked for the same data for those six articles
23 that were written by Tata, etc. And they want the exact

1 same list. They get the same list. They want to know
2 exactly the dates of the information; how many people
3 have mesothelioma, were there any excluded because of a
4 different time period or they didn't look at the
5 records. Everybody gets to look at the same thing. And
6 then I would say that, once you had that, then maybe
7 they could figure it out. Because it can go both ways.

8 Q. Is it important to you in testimony you give
9 today that this article was subjected to credible,
10 meaningful peer review by somebody who was qualified?

11 A. I would say that that is always important.
12 Peer review is very important.

13 MR. TARRY: Your Honor, can we toggle back to
14 the computer?

15 I'd like you to see what Dr. Leigh says about
16 this journal and this issue.

17 THE WITNESS: Okay.

18 (Whereupon the following video clip was
19 played.)

20 "QUESTION: All right. And I'm going to ask
21 you now about the estimates of lifetime risk that you
22 made for vehicle mechanics in your article with
23 Driscoll, Exhibit No. 14, "Malignant Mesothelioma in

1 Australia".

2 Now, this appeared in the International Journal
3 of Occupational Environmental Health; is that correct?

4 "ANSWER: Yeah.

5 "QUESTION: Okay. Is that a peer review
6 publication?

7 "ANSWER: Yes.

8 " QUESTION: Who reviewed your paper, do you
9 know?

10 "ANSWER: No.

11 "QUESTION: Do you know who the referees were?

12 "ANSWER: No.

13 "QUESTION: Does the -- does the journal have
14 any particular reputation:

15 "ANSWER: No. I think -- I think -- I think
16 it's a -- it's an international journal. And there is a
17 certain, you know, it does tend to sort of publish
18 provocative papers at times.

19 "QUESTION: Okay.

20 "ANSWER: It's not afraid to publish
21 provocative papers.

22 "QUESTION: It appeared in Volume 9, No. 3, the
23 July/September 2003 edition.

1 "ANSWER: Yes.

2 " QUESTION: And it's a special issue of this
3 journal; right?

4 "ANSWER: Yes.

5 "QUESTION: And the issue is titled "The
6 Asbestos War."

7 "ANSWER: Yes.

8 "QUESTION: The guest editor is Laurie
9 Kazan-Allen.

10 "ANSWER: Yes.

11 "QUESTION: Do you know a Laurie Kazan-Allen?

12 "ANSWER: Yes.

13 "QUESTION: Who is she?

14 "ANSWER: She's a -- a journalist and runs the
15 British asbestos newsletter. And she's a bit of an
16 international activist.

17 "QUESTION: But she's not a scientist; right?

18 "ANSWER: I don't -- I don't think she's got a
19 science degree.

20 "QUESTION: What is our exhibit? What are we
21 at? No. 18? Can we mark that, please.

22 (Pause.)

23 "QUESTION: I've got a copy of the journal in

1 which your article appeared. It's now marked Exhibit
2 No. 18, International Journal of Occupational
3 Environmental Health, Volume 9, No. 3,
4 July/September 2003.

5 As I open the journal up to the table of
6 contents, the listing of the articles in the table of
7 contents are -- "The Asbestos War" is the first one by
8 Laurie Kazan-Allen. The next one is "Scientific
9 Controversy and Asbestos." The next one is by you,
10 "Malignant Mesothelioma in Australia," by you
11 Dr. Driscoll. The next one is "Cape Place South African
12 Mine Works, Quest For Justice." The next one is
13 "Asbestos Mining in Southern Africa." The next one is
14 "Asbestos, Sorrowful Legacy: A Photo Essay." The next
15 one is a cross-country comparative overview of the
16 asbestos situation in ten Asian countries. And it goes
17 on.

18 Doctor, do you agree that the purpose of this
19 special interest publication, in which your article
20 appears, is to give the public forum for asbestos
21 victims and -- and victim support groups?

22 "ANSWER: It's partly that. I think it's
23 obviously a -- an issue that's drawing -- drawing

1 attention to the global asbestos problem.

2 "QUESTION: Indeed, that's what the editor of
3 the article says that its purpose is; right?

4 "ANSWER: Yeah, not just -- not just in the aid
5 of victim groups. It's also, you know, directed at
6 governments, industry, etc.

7 "QUESTION: And you'd agree with me that
8 there's not a single case control study or cohort study
9 in this publication; right?

10 "ANSWER: In this issue?

11 "QUESTION: In that issue.

12 "ANSWER: Yes. It's not primarily an issue
13 published on epidemiologic papers. It's more, you know,
14 national overview sort of papers, asbestos problems in
15 various countries.

16 "QUESTION: Okay. With respect to the
17 calculation of risk that -- the lifetime risk that you
18 calculated in this article that appears at Table 5, for
19 vehicle mechanic, you've stated previously that you
20 used -- you actually used 78 cases for that?

21 "ANSWER: Yes.

22 "QUESTION: Is that right?

23 "ANSWER: I did -- I did the vehicle mechanic

1 *plus, plus or minus for exposures for that particular*
2 *one.*

3 "QUESTION: Okay. So that really shouldn't be
4 *stated as a risk, a lifetime risk for vehicle mechanics.*
5 *Would you agree?*

6 "ANSWER: Not really. It's -- it's vehicle
7 *mechanics -- not solely vehicle mechanics, ever being a*
8 *vehicle mechanic. It's a lifetime risk for vehicles --*
9 *it's different to the ones in the EPA. That's why I*
10 *didn't report that in the EPA letter. So that's*
11 *different. All the lifetime risks there are based on*
12 *the -- they're sort of giving the high end, if you*
13 *like."*

14 BY MR. TARRY:

15 Q. *In this journal there was not a single case*
16 *control cohort study including Dr. Leigh's. That's what*
17 *he's admitted to; correct?*

18 A. Yes.

19 Q. *In other words, there's no analytical*
20 *epidemiology, the sort of the information that would*
21 *have been at the stage above case reports?*

22 A. Well, in that journal. But what about the one
23 *before it? What about the one after it? Just because*

1 there's isn't any analytic epidemiologic studies in
2 there, doesn't mean that's it's not accurate and not
3 correct and doesn't reflect the people -- reflect the
4 opinion of the people who wrote those articles.

5 It's nothing different that happens all the
6 time in the United States every day. In fact, about,
7 what, five years ago the USA Today had a three-day
8 period where on the front page was the problem about
9 mesothelioma in Eastern European countries where people
10 are dying from mesothelioma in their thirties.

11 And, you know, something is happening in this
12 world where people are concerned about that. And just
13 because we are -- people are concerned about that
14 doesn't mean that when they publish those articles that
15 it's not necessarily accurate. But I do agree with what
16 you said.

17 Q. And everybody ought to be concerned and try to
18 reduce the amount of disease; correct?

19 A. Absolutely.

20 Q. Part of that is accurately identifying the
21 actual risk factors for a disease so we don't waste
22 resources where there are false risk factors; correct?

23 A. I would agree with that.

1 Q. All right. And it's important when you are
2 conducting scientific research on a topic like this to
3 be as careful as possible; correct?

4 A. I agree.

5 Q. And to be as thorough as possible so that you
6 can assure accuracy of the data you report and the
7 conclusions to be drawn from those; correct?

8 A. I agree.

9 Q. And for you to assume -- well, for you to give
10 the testimony you have today, you have to assume, don't
11 you, that Dr. Leigh and Dr. Henderson were careful and
12 thorough in both selecting the data and drawing
13 conclusions from the data?

14 A. Well, that is correct. I mean, I have the
15 highest regard for both individuals. Dr. Leigh was the
16 head of EPA in Australia. Dr. Henderson is probably one
17 of the world's best known individuals on
18 asbestos-related lung disease.

19 Q. If I wasn't clear before, let me state it
20 again. I am not suggesting anything about their
21 reputations or about other work they've done. The
22 inquiry before the Court today is how careful and
23 thorough they were on this data.

1 A. Right.

2 Q. Is that all right?

3 A. That's fine.

4 Q. Okay. Let's look a little bit of Dr. Leigh's
5 testimony about how careful they might have been in
6 reporting these cases.

7 (Whereupon the following video clip was
8 played.)

9 "QUESTION: And I understand that you may not
10 be able to say with confidence whether the case reported
11 in the notebook No. 11 is the same as the person
12 identified as 89090 on your Exhibit 6?

13 "ANSWER: No, I can't say that.

14 "QUESTION: Okay. Is that fair?

15 But just using the information on Exhibit 6,
16 there is certainly no information suggesting that this
17 person was an auto mechanic -- a vehicle mechanic; is
18 that right?

19 "ANSWER: I counted him though.

20 "QUESTION: You did count him?

21 "ANSWER: I did count him.

22 "QUESTION: Do you agree that there's not any
23 information that would allow --

1 "ANSWER: I agree that that could be -- that
2 could be validly, you know, not counted.

3 "QUESTION: Okay.

4 "ANSWER: I mean, he could have been a vehicle
5 mechanic. He could have been a home amateur, you know,
6 as you say. But I made the decision to count that case
7 for the -- "

8 BY MR. TARRY:

9 Q. Do you still believe that Dr. Leigh and Dr.
10 Henderson were careful in reporting data from this
11 register?

12 A. Depends how you evaluate that. I said he could
13 have been. Okay. What if he worked as a shade tree
14 mechanic for 30 years and did, say, a thousand brake
15 jobs? How do you know that that's not correct?

16 What you're implying is that what he said was
17 that the guy never did that. That's not what Dr. Leigh
18 said.

19 (Whereupon the following video clip was
20 played.)

21 "ANSWER: He worked with flaking asbestos roof
22 and worked with brake line.

23 "QUESTION: Would you agree that this person

1 *should have been classified as having other exposure?*

2 *"ANSWER: Possibly could have been.*

3 *"QUESTION: When you say possibly could have*
4 *been, this guy worked with -- this guy worked with --*
5 *under flaking asbestos roof, which I think is one of the*
6 *actual circumstances you actually described earlier*
7 *talking about when insulation is breaking apart; right?*

8 *"ANSWER: Yes.*

9 *"QUESTION: That would definitely be*
10 *categorized under one of these other circumstances of*
11 *exposure; right?*

12 *"ANSWER: Okay.*

13 *"QUESTION: You agree?*

14 *"ANSWER: Yep.*

15 *"QUESTION: Okay."*

16 *BY MR. TARRY:*

17 *Q. Based on that example, do you believe that*
18 *Dr. Leigh and Dr. Henderson were careful and accurate in*
19 *reporting their claims of increased incidence from*
20 *mesothelioma among brake workers with no other exposure*
21 *in Australia?*

22 *A. We've seen three cases now where you've given*
23 *some information that they may have had other exposure.*

1 I would say that they didn't seem to be particularly
2 careful in those. But maybe they thought that the brake
3 was still the dominant exposure. But I would agree with
4 you that they should have put in any information that
5 they had of any other exposure.

6 Q. In the interest of time, I've got a lot of
7 these. I think based on that last statement, we can
8 agree that the number that is reported in your textbook
9 is inaccurate. Is that fair?

10 A. I would say it's inaccurate with respect to
11 what you've given the information today, yes.

12 Q. Who is the target audience of your technical
13 book?

14 A. Pardon me?

15 Q. Who is the target audience of your technical
16 book?

17 A. I don't even think text books have target
18 audiences. I would say this is for anybody who wants to
19 know information about asbestos-related lung disease.

20 Q. It includes the chapter on clinicians in the
21 courtroom; correct?

22 A. It does, yes.

23 Q. And what's the purpose of that chapter?

1 A. I guess to make somebody related be aware of
2 what usually happens in the courtroom.

3 MR. TARRY: Dr. Hammar, thank you for your
4 time.

5 THE WITNESS: You're welcome. Thank you.

6 MR. JOHNSON: I think I'm after you. I think
7 we've got that correct.

8 Am I right about that, Your Honor?

9 THE COURT: No. The clear going forward, I
10 think the order for plaintiffs' witness should be
11 plaintiffs' counsel, Mr. Johnson, and then
12 cross-examination. So right now we've got
13 cross-examination. I think you would be next,
14 Mr. Johnson, if you have questions.

15 MR. JOHNSON: I may have. Let me make sure I
16 have it straight. I'm sorry to belabor this, Your
17 Honor.

18 THE COURT: We're out of order for this
19 witness, so we're going to jump back in. I think we
20 were in order yesterday but perhaps we --

21 MR. JOHNSON: Let me see if I understand. You
22 wanted the direct followed by the cross?

23 THE COURT: No. Because you're --

1 MR. JOHNSON: Then I would be part of the
2 direct, in essence, but I would follow plaintiffs on
3 direct.

4 THE COURT: I think that's how we discussed it
5 during our conference.

6 Mr. Crumplar, is that accurate?

7 MR. CRUMPLAR: Yes. I think last time he
8 waived his direct.

9 MR. JOHNSON: That's my understanding, that
10 they did direct. I had no further direct. We then went
11 to cross. And then my understanding would be that we're
12 back to redirect followed by me followed by cross.

13 THE COURT: All right. Fine.

14 MR. CRUMPLAR: That was the confusion. I don't
15 think he had any direct at this time, and he didn't last
16 time also.

17 THE COURT: I follow.

18 MR. JOHNSON: That's what I'm trying to alert
19 the Court to. I may have some additional direct, but
20 it's probably going to be nominal.

21 THE COURT: Redirect. Fine.

22 MR. WADDELL: I want to I apologize to the
23 Court, Your Honor, for not standing up.

1 THE COURT: That's all right.

2 BY MR. WADDELL:

3 Q. What's the No. 1 risk factor for a person to
4 develop an asbestos disease?

5 A. Asbestos.

6 Q. Breathing it?

7 A. Yeah. Exposure.

8 Q. All right. And as a trained physician in
9 diagnosing asbestos disease and the cause or causes of
10 asbestos disease, you consider individual circumstances;
11 right?

12 A. Sure.

13 Q. Because would it be fair if you saw a case
14 where this is the information, Dr. Hammar, an individual
15 develops mesothelioma, he is an insulator, is that
16 mesothelioma caused by asbestos?

17 A. Well, you certainly would think that's a good
18 possibility, yes.

19 Q. Because the job classification tells you some
20 information, doesn't it?

21 A. It does.

22 Q. But it also doesn't tell you the individualized
23 circumstances of that person; right?

1 A. That's correct.

2 Q. Because if I further told you that individual
3 was an insulator who only worked with cold insulation,
4 that gives you some more information; right?

5 A. It does.

6 Q. And if the -- if he only worked with cork
7 insulation and nonasbestos-containing information now
8 that you had that, that classification doesn't apply to
9 that individual, does it?

10 A. It does not. It would distort it.

11 Q. And, in fact, cases get reported in peer review
12 literature where there is no epidemiology associated --
13 to support something; right?

14 A. Right.

15 Q. Because I want to talk about a case that's
16 surprising came from Delaware that you reported. And
17 it's on 106 of your CV. I'll just read it into the
18 record to speed it along.

19 Rom, R-O-M, was the author and you. And the
20 name of this peer review publication was "Malignant
21 Mesothelioma From Neighborhood Exposure to Anthophyllite
22 Asbestos."

23 Now, do you recall that?

1 A. Sure.

2 Q. Okay. And just a little bit of a background on
3 that case. Could you tell His Honor about the
4 circumstances of how you got involved -- how it made it
5 into this peer review publication and additionally into
6 the medical and scientific literature?

7 A. It made it in was a case Mr. Crumplar sent to
8 me and where the individual had mesothelioma. And we
9 did some investigation. And I can't remember all the
10 details about it. But it turned out that this patient
11 lived fairly close to a plant that made these very large
12 plastic containers.

13 And as part of the plastic containers and
14 making these, they added anthophyllite asbestos into
15 these the containers. And then to additional
16 information it was found that this plant, in producing
17 these large plastic containers actually released
18 asbestos fibers of an anthophyllite into the area. And
19 this individual had what is referred to as neighborhood
20 exposure, which was described by a new house many, many
21 years ago as a type of way individuals can be exposed to
22 asbestos.

23 So we obtained lung tissue on this be

1 individual I think after he died. And that was sent to
2 Dr. Dodson, who did asbestos fiber analysis, who found
3 elevated numbers of anthophyllite asbestos fibers in
4 that individual's lung tissue.

5 Q. Now, when you submitted this for peer review
6 and eventually publication, did they, when you submitted
7 the article, they said, wait a minute; we don't have
8 "epi" studies on women in Delaware living near a
9 plastics plant; you can't put this in the literature?

10 A. Well, they must not have thought that. Because
11 when a lot of other articles published by -- about
12 asbestos that were published has not been peer reviewed
13 literature. And I think the reason that they published
14 it was not because it wasn't peer review literature was
15 because that there was other examples of neighborhood
16 exposure that resulted in the development of
17 asbestos-related disease like mesothelioma. And that
18 was an acceptable way that an individual could be
19 exposed.

20 Q. Generally accepted in at least the publications
21 that made it out?

22 A. Right. It wasn't in re any different than what
23 Cartier and McCaughey did with chrysotile asbestos in

1 Quebec. They reported those cases. It wasn't any
2 different than what Wagner, Sluggs, and Marshan did in
3 South Africa 1960 in the British Journal of Industrial
4 Medicine. That wasn't a epidemiologic-type study.

5 There have been all kinds of case report
6 studies in the United States and throughout the world
7 that have drawn attention to disease and exposure.
8 Certainly, angiosarcoma in the liver and vinyl chloride
9 was based on five cases of exposure.

10 Like I said, therapeutic radiation, which I
11 think the majority of people who study mesothelioma
12 believe can cause mesothelioma. There's no
13 epidemiologic literature that suggests that that is the
14 cause of mesothelioma. There are some things that you
15 observe and you -- based on basic scientific premises
16 that asbestos is a carcinogen, that asbestos is known to
17 cause mesothelioma. And you find a case of it in an
18 unusual setting or something that's unique is often
19 important to be published because then people can look
20 for more of those cases. And they can potentially try
21 to do something about that exposure.

22 Q. Well -- and the reason why I ask those
23 questions, as a trained physician in diagnosing asbestos

1 disease in humans and the cause of that, is it a
2 scientifically sound and reasonable approach -- to have
3 a blanket approach to go, uh-uh, it doesn't occur in
4 individuals because we looked at groups that don't have
5 risk, for instance, anthophyllite exposure in Delaware
6 women? Would that be a fair and reasonable approach
7 scientifically?

8 A. No, it wouldn't be reasonable.

9 Q. 'Cause when you're looking at individuals, you
10 have to look at the individual circumstances; right?

11 A. That is correct.

12 Q. And in what may or may not have been a cause;
13 correct?

14 A. That is correct.

15 Q. A blanket approach by product elimination or by
16 job classification would be unreasonable and
17 unscientific; right?

18 A. Well, if you did it in a blanket approach, yes.

19 Q. When you're looking at individuals?

20 A. Right.

21 Q. And that's why they're wrong?

22 A. Well, that's why I think they are wrong with
23 respect to stating that chrysotile doesn't cause

1 mesothelioma in a setting where people are exposed from
2 brake products. Because we know that chrysotile in
3 generally does cause mesothelioma. It causes it in most
4 settings. It could can cause it in different types of
5 situations. There wouldn't had been any reason to
6 exclude the possibility that chrysotile could not cause
7 mesothelioma in an individual who was exposed in
8 basically any way, whether brakes, whether it was a
9 clock, whether it's whatever.

10 Q. Would it be fair to say that an individual who
11 has developed an asbestos-related disease has
12 100 percent risk of getting asbestos disease?

13 A. No.

14 Q. He's got it already; right?

15 A. He's got it already, yes.

16 Q. You have to go back and look at the
17 circumstances of how; right?

18 A. That's correct.

19 Q. And there's been some figures thrown out. Is
20 58 individuals in looking at a job classification or a
21 product use significant in your opinion, Dr. Hammar?

22 A. Well, it's extremely significant when you look
23 at what the background issue is. That's something we

1 didn't talk a whole lot about.

2 But the background incidence of mesothelioma,
3 according to this data that was first published by
4 Selikoff in his group in the Annals of the New York
5 Academy of Science in 1964 -- I think that was the
6 year -- '65 -- excuse me -- the background incidence
7 that they gave for mesothelioma was zero. And they
8 basically couldn't calculate a relative risk, because
9 with a zero denominator, you can't do it. It's
10 basically infinity.

11 And if you look at the most recent publication
12 that I know on about low level exposure and exposure
13 from nonoccupational exposure, that's by Gunner
14 Hillerdaul published in 1999 in a peer review journal --
15 I have that article with me -- published in Occupational
16 Environmental Medicine in 1999, Volume 56, pages 505 and
17 513, basically states in there that he thinks the
18 background level for mesothelioma is way below the one
19 case per million people per year. And that's why
20 basically any case of mesothelioma is highly
21 significant.

22 Q. And whether that number is 48 -- how about 43?

23 A. Again, if that was the accurate data, that

1 would be very significant.

2 Q. How about 40?

3 A. Also, very significant.

4 Q. And using those figures going back into what
5 was the articles we discussed is still statistically
6 significant?

7 A. Yes.

8 Q. Okay. Talking about, I guess, background or
9 unknown idiopathic spontaneous cases of mesothelioma,
10 that lady from Delaware who unfortunately got
11 mesothelioma, did you at first think or did you know
12 what the exposures were and all of that --

13 A. I didn't know initially what the exposures
14 were. I think start looking -- any time a person has
15 mesothelioma such as a signal tumor, you start looking
16 for potential ways that individuals develop mesothelioma
17 and where they were exposed to asbestos. That's the
18 first thing you would look for.

19 The second thing I would look for usually would
20 be if there was any history of therapeutic radiation.

21 Q. Okay. And possible exposure, latency, dose,
22 those other things?

23 A. Sure. You always look for that. That's

1 important. Latency. And that's talked about in the
2 Helsinki consensus report.

3 Q. I guess to put it simply, this unfortunate lady
4 could have been a spontaneous meso but upon further
5 review she had asbestos exposure; right?

6 A. Yes, elevated concentrations of anthophyllite
7 in the lungs.

8 Q. Let's simplify this. As a doctor, okay, simple
9 doctor's opinion, if a person came into a clinician's
10 office, got a broken arm, hit by a white Ford Taurus,
11 should a doctor under those circumstances in accessing
12 causation in an individual basis go, uh-uh? What kind
13 of car did you get hit by? A white Ford Taurus. We
14 don't have the data on a white Ford Taurus causing
15 injuries that way, should causation be excluded?

16 A. Of course not.

17 Q. Hypothesis. Wasn't it once a hypothesis that
18 asbestos could cause disease in humans?

19 A. Sure.

20 Q. Has that hypothesis been played out over and
21 over and proven correct?

22 A. Yes.

23 Q. Has it been played out and proven correctly in

1 *individuals that others have seen and that you have seen*
2 *(indicating), that the world has seen when it comes to*
3 *asbestos-containing friction products?*

4 A. *I believe it has, yes.*

5 MR. WADDELL: *Thank you, Your Honor.*

6 MR. JOHNSON: *I have a few minutes worth of*
7 *questions, Your Honor. Thank you.*

8 THE COURT: *All right.*

9 BY MR. JOHNSON:

10 Q. *Good afternoon, Dr. Hammar.*

11 A. *Good afternoon.*

12 Q. *So the Court knows, we're both from Seattle.*
13 *We've known each other for a long, long time.*

14 A. *We have, yes.*

15 Q. *We generally see each other -- like*
16 *99.999 percent of the time, you're testifying for the*
17 *other side?*

18 A. *That is correct.*

19 Q. *We're good friends?*

20 A. *We're good friends.*

21 Q. *Okay. I'd like to follow up a little bit very*
22 *briefly on the background incidence. Can you tell the*
23 *Court what the largest cohort ever studied of asbestos*

1 exposed people is?

2 A. The asbestos insulators, members of the heat
3 and frost insulators union.

4 Q. And how many people have been in that cohort?

5 A. 17,800.

6 Q. What's the expected incidence of mesothelioma
7 going against the general public in a cohort of 17,800
8 people?

9 A. Basically, the experience there is that they
10 have 10 percent of the people have died from
11 mesothelioma. The expected rate of death in
12 mesothelioma in generally is basically zero.

13 Q. So if it was -- the expected rate would be zero
14 in a cohort of 17,800 people?

15 A. That is correct.

16 Q. What would the expected rate be in 178,000
17 people?

18 A. Zero.

19 Q. Okay. What would the expected rate be in
20 500,000 people?

21 A. Zero.

22 Q. Does this, in your opinion, in terms of your
23 review of epidemiology, this fact that we're dealing

1 with a disease that is so rare that the background rate
2 approaches zero, does that have an impact on the use of
3 epidemiology in the evaluation of that disease?

4 A. Sure, it does.

5 Q. Okay. And not to belabor the obvious, but
6 could you explain briefly why that is.

7 A. Because if you think that mesothelioma has a
8 certain rate of existence as a spontaneous tumor in the
9 background population, that's going to really change
10 your data with respect to if there was an increased
11 number of cases versus a lack of increased number of
12 cases in a population of people that are being studied,
13 especially in case -- control type studies where there
14 are a small number of people.

15 I'm not an epidemiologist, but that's going to
16 markedly skew that data. Because if you say you have a
17 number of three cases per million people as a
18 background, and that's how many cases you find in a
19 certain number of people that you calculate that, then
20 there's going to be no increase in incidence. To me
21 that doesn't mean, based on what I think is correct,
22 that those people with mesothelioma was not caused by
23 asbestos.

1 Q. In terms of scientific information available to
2 you in formulating the opinions that you express here
3 and that you express when you testify, is the data
4 regarding the effect of asbestos and its impact when
5 exposed to tissue in the laboratory in experimental
6 animals and in human tissue relevant to you?

7 A. Well, it's relevant to me in that I think that
8 there has to be certain types of mechanisms that
9 asbestos uses to cause disease or cause injury. And in
10 that respect it's relevant. It's relevant I think also
11 in animals in that, if you can produce the disease in
12 mammals, which are what humans are, then there's
13 evidence again that that would be important as a
14 potential cause of a disease in humans.

15 But it's not totally the same. And you've got
16 to understand that there's certain limitations between
17 what you can extrapolate from animal studies and in
18 vitro scientific studies. And you've got to be very
19 careful about that. Like the lifespan of a rat, for
20 example, I think is about, what, one and a half to two
21 years. And lifespan of a human is, say, 75 to 80 years.
22 You can't necessarily totally relate one to the other,
23 but you can relate some of the mechanisms of that.

1 The same way in vitro studies. Just because,
2 for example, you can produce mesothelioma cancer cell
3 lines that have doubling times as short as about
4 15 days, that doesn't mean that doubling time of an
5 epithelial mesothelioma in a human being is going to be
6 15 days. Because if was, it would kill the person
7 probably in about three or four months.

8 Q. Without going through chapter and verse of
9 everything, outside of epidemiology in formulating your
10 opinions that you've expressed here today, are you
11 looking at things like the scientific data and
12 information on asbestos effects on tissue, on
13 translocation of asbestos fibers to get them out to the
14 site where cancers are caused, on the propensity of
15 chrysotile asbestos to cause genetic changes on the
16 ability of friction products to release fibers that are
17 respirable? Are you looking at all that kind of
18 scientific data?

19 A. Absolutely. In fact, in Dr. Roggli's paper
20 where he talked -- and I reviewed that paper. And
21 Dr. Roggli's one of my best friends. I'd never say
22 anything bad about him. But one thing when I reviewed
23 that paper that was published in Ultrastructured

1 Pathology, I think it was 2003, I said that it would be
2 very interesting to know what the concentrations of
3 asbestos were at the target sites, say, the pleura
4 versus the lung tissue if you're going to make a
5 conclusion that asbestos did not contribute to a
6 mesothelioma in a person who had multiple exposures.

7 And that's just basically simple knowledge. Or
8 I think it's simple knowledge, in that if you believe
9 you have to have the carcinogen at the site of exposure,
10 wouldn't in a way you would want to look at where the
11 exposure was and what the concentration was there versus
12 what the concentration was in the lung and say that
13 based on the concentration in the lung, I'm going to say
14 that this mesothelioma is cause by amphiboles; and it
15 doesn't matter that he was exposed to chrysotile because
16 I can't find any chrysotile in the lung. And even
17 though the Helsinki consensus report states that it's a
18 lousy way to determine chrysotile exposure by looking at
19 concentrations of chrysotile in the lung because it's
20 clear.

21 So, you know, there's some things that happen
22 or people make conclusions that I think other people can
23 make conclusions that are not necessarily the same and

1 potentially maybe both can be right.

2 Maybe what Dr. Roggli should have said, and
3 that's what I think he maybe should have said, and maybe
4 in some respects he is saying that. And that is that if
5 you find amphiboles in the lung at a higher
6 concentration than you'd expect, then it's likely that
7 they contributed to a person's mesothelioma. But that
8 doesn't necessarily take away that the chrysotile didn't
9 also contribute to the person's mesothelioma just
10 because you can't find it in the lung.

11 Q. I'm not sure whether you're aware of this, but
12 I don't think it's being contested in this particular
13 hearing that chrysotile is an agent that causes
14 mesothelioma and asbestos disease in human beings.

15 Let me clarify one other thing. And that is,
16 prior to the publication of the Henderson and Leigh data
17 from Australia, if you had been given a case such as a
18 case report that you described and without the benefit
19 of epidemiological proof, you had in that case a history
20 of chrysotile exposure due solely to brake or friction
21 product work and you had tissue findings showing a high
22 concentration of chrysotile fibers in the body, in a
23 case of a mesothelioma under those circumstances, what

1 would your conclusion have been as to the cause of that
2 mesothelioma?

3 A. My conclusion would have been that that
4 person's mesothelioma was caused by chrysotile asbestos.

5 Q. Notwithstanding the absence of epidemiological
6 proof as to the generic question?

7 A. That is correct.

8 Q. Finally, and I think this was touched on, but
9 just to make sure it's clear, the Wagner study in 1960,
10 was that an epidemiological study?

11 A. No.

12 Q. To the best of your knowledge, how many cases
13 of mesothelioma did he report?

14 A. There were 33 cases on the northwestern cape
15 province of South Africa of which he thought 32 were
16 directly related to crocidolite asbestos exposure.

17 Q. To the best of your knowledge, was there any
18 doubt in the mind of any medical authority anywhere that
19 that study established that asbestos had caused those
20 33 cases of mesothelioma?

21 A. No.

22 Q. Are there any epidemiological studies to your
23 knowledge that show whether calcium silicate pipe

1 *covering taken alone, i.e., a person exposed only to*
2 *calcium silicate pipe covering in the field causes*
3 *mesothelioma? Has that ever been studied*
4 *epidemiologically to your knowledge?*

5 A. No.

6 Q. Okay. Does the absence of an epidemiological
7 *study on calcium silicate pipe covering cause you to*
8 *doubt whether that product is capable of causing*
9 *mesothelioma?*

10 A. No.

11 MR. JOHNSON: I have no further questions.
12 *Thank you.*

13 MR. TARRY: Your Honor, I think I just have a
14 *couple questions, if in the interest of time the witness*
15 *will concede the more technical questions on*
16 *epidemiology to the two witnesses who are going to*
17 *testify later about that?*

18 THE WITNESS: I will.

19 BY MR. TARRY:

20 Q. Okay. Then I just have a couple of questions I
21 *think.*

22 In the case of a broken leg caused by a car, do
23 *we understand the mechanism by which pressure applies*

1 and creates in a break?

2 A. I suppose we do.

3 Q. We don't, don't we?

4 A. Yes.

5 Q. We understand how pressure can create breaks in
6 bones?

7 A. I suppose, yes.

8 MR. TARRY: That's it. Thank you.

9 THE COURT: Doctor, I just have a couple
10 questions. There was some testimony that you offered
11 earlier about the fact that it's really impossible to
12 identify an asbestos disease being caused by a single
13 fiber or even group of fibers as you do some fiber
14 studies later and identify asbestos fibers in the lung
15 or in other areas. So it's really a cumulative exposure
16 that causes you to reach the diagnosis for the causation
17 opinion.

18 THE WITNESS: Right. I mean, it's all the
19 exposures that a person would have had to asbestos that
20 would have caused that.

21 The issue, though, that does come up sometimes
22 would be -- and that's what's talked about in that 1982
23 versus that 1992 thing -- the issue we don't know really

1 too much about is exactly when the first cancer cell
2 develops, and that anything after that -- say, a person
3 was diagnosed with mesothelioma in 2000, and say they
4 were exposed to asbestos, say, up to 1995, say, if it
5 was an epithelial mesothelioma, I think the evidence
6 would suggest, based on what we do know, which is not
7 very much, that probably the exposures only before say
8 1990 would have been causative of the mesothelioma,
9 unless you accept the idea that asbestos is a tumor
10 promoter. I don't know if that answers your question or
11 not.

12 THE COURT: Actually, I was just making sure I
13 was clear on your testimony before I asked my question.

14 And my question is, and just to be clear, so it
15 is this sort of cumulative exposure that allows you to
16 make a diagnosis; it's not so much that you can say
17 exposure at this moment in time or even this period in
18 time in the big picture or in the continuum of exposure
19 is what actually has caused the disease process.

20 THE WITNESS: No. I would say this. If you
21 look at the mechanism of carcinogenesis, which is very
22 complicated, is that what is thought to happen is
23 basically you get mutations in protooncogenes, which

1 control cell growth; mutations in tumor suppressor genes
2 that control cell death; changes in the cell cycle
3 system which basically organizes the cell -- what time
4 to make the DNA, what time to divide, changes in the
5 receptor mechanism of the cell which determine how fast
6 a cell can grow, all of these changes that occur over a
7 period of time by the cumulative exposures that cause
8 the cancer.

9 And you can't really separate these out at this
10 point in time. We know quite a bit about what those are
11 from a biochemical point of view. But we don't know
12 much about the timing effect of it exactly; how fast it
13 happens, say, for a mutation in a tumor suppresser gene
14 to occur or how long it takes for, say, a change in the
15 cell cycle to happen. But we do think that it's entire
16 exposure that contributed to this.

17 THE COURT: Can you identify other areas in
18 science or medicine where that is a dilemma that
19 clinicians like yourself or that scientists face,
20 meaning you can't pinpoint a single individual causative
21 agent because of the nature of the disease process or
22 the nature of the carcinogen involved or whatever the
23 dynamic might be that precludes one from doing that; but

1 nevertheless that is what is done in order to make a
2 diagnosis or to link an agent to a disease?

3 THE WITNESS: I think the answer to that
4 question is yes.

5 I think the best example would be cigarette
6 smoking and lung cancer. Say, for example, like in
7 Bremerton where I work, we have an epidemic of lung
8 cancer there because kids start smoking at ten, 12 years
9 old. And they say they got lung cancer at age 50, which
10 is not uncommon. And they you went back and tried to
11 say, well, what cigarettes was it that they smoked that
12 caused the lung cancer. What you would have to say --
13 and there we have more information on doubling times --
14 that probably all of the exposure they had from the
15 carcinogens in cigarette smoke from the time that they
16 first started up until about ten years prior to the
17 diagnosis except for one type of lung cancer called
18 small cell lung cancer that grows faster was responsible
19 for causing that cancer. You couldn't separate out --

20 THE COURT: Can I stop you for just a second,
21 though, just to make sure that I've expressed all of the
22 variables involved here.

23 Is cigarette smoking not different from

1 asbestos exposure in the sense that cigarette smoking is
2 a tobacco product with probably not a whole lot of
3 variation in terms of one product to the next that one
4 might be inhaling; whereas with asbestos, as I gather,
5 you've got multiple types of asbestos to which one might
6 be exposed in a lifetime; and, therefore, does that not
7 make a cumulative exposure to asbestos in a diagnosis or
8 a causation opinion related to that different than, say,
9 a cigarette smoking situation?

10 THE WITNESS: It may be. But even there
11 there's some potential problems like in cigarette smoke,
12 for example, the low tar cigarettes contribute as much
13 as the high tar cigarettes, for example. The answer is
14 probably no because people smoke cigarettes different.

15 With respect to the asbestos, it does get into
16 some issues that potentially there might be some
17 differences. For example, there's a suggestion or
18 there's some evidence -- and this is, again, an area of
19 area high controversy -- that there's relative potency
20 of the various types of asbestos in causing
21 mesothelioma.

22 For example, it's basically stated that the
23 amphiboles are more tumorigenic in causing mesothelioma

1 than chrysotile. And I think that's true. I think that
2 chrysotile isn't as tumorigenic on a fiber per fiber
3 basis. Yet when Wagner and other people were trying to
4 prove that in experimental animals, they couldn't prove
5 it at all. In fact, the latest study that was done in
6 1992 by direct injection where they directly injected a
7 certain milligram dose of asbestos into the peritoneal
8 cavity of rats, it turned out that the one that produced
9 the most mesotheliomas was UICC chrysotile B, which is
10 in not amosite and not crocidolite.

11 And then the other issue that comes up is this,
12 is that if you were to assume that what Hodson and
13 Darden published in, I think it was 2000, on the
14 relevant potencies of asbestos fiber types with respect
15 to causing mesothelioma where they said that crocidolite
16 was 500 times more potent than chrysotile and that
17 amosite was a hundred times more potent than chrysotile,
18 what would happen if it turned out that you had, say,
19 10,000 times more chrysotile than you had amosite or
20 crocidolite? How would you that work?

21 Because that's often the situation, especially
22 not necessarily in brakes, of course, but, say, in
23 insulation where the dominant fiber actually is

1 chrysotile. And it's things like that that cause
2 really -- it's really hard, I think, to separate out.

3 And the other reason --

4 THE COURT: Let me just ask, to move along
5 because we do have some time pressures, would that
6 variable injected into the causation equation or even
7 the diagnosis equation, are there areas in science that
8 you would identify as being similar to that that
9 confront clinicians like yourself where you're dealing
10 with these multiple barriers -- variables and yet you
11 are called upon ultimately to make the diagnosis in
12 terms of causation?

13 THE WITNESS: Yeah. I think the diagnosis by
14 itself stands by itself. So that's not an issue of
15 causation. I can diagnose a mesothelioma without ever
16 knowing what happened to the person based on pathology.

17 THE COURT: Sure.

18 THE WITNESS: Then with respect to the
19 causation, another example for that might be the case --
20 I think there are other carcinogens where you might be
21 exposed to, say, three or four carcinogens that
22 potentially could cause, we'll just say lung cancer,
23 we'll take, for example, beryllium, chromium, arsenic,

1 and lead. Each of those have the ability to cause lung
2 cancer. And they might be different abilities based on
3 their concentration.

4 And I think there that if you have a situation
5 like that, I think doctors would say that, well, this
6 person was exposed to a number of inorganic agents that
7 caused lung cancer, and therefore, we're going to say
8 that this lung cancer was caused by that. I think it
9 would be very difficult to separate that out and say
10 that 20 percent of his lung cancer was caused by
11 arsenic, 30 percent by lead, etc.

12 THE COURT: So you're looking at the individual
13 patient, the individual exposures; if you identify
14 multiple carcinogens, then you're not going to attempt
15 to separate those out in terms of ultimately saying this
16 is what caused the problem.

17 THE WITNESS: That's my take on it.

18 And the biggest example of that in asbestos is
19 lung cancer and cigarette smoke and asbestos. And that
20 is that most of the studies have shown that there's a
21 multiplicative synergistic increase in lung cancer that
22 people who are exposed to asbestos and cigarette smoke.
23 And that ranges as high as, say, 90 times relative risk.

1 And in most smokers exposed to asbestos it would be
2 about 50. But the rate that is generally given for lung
3 cancer causation to asbestos would be about five times
4 that of a nonasbestos exposure. And to a smoker would
5 be about 11. But when you put them together is 55.

6 And as far as I know, nobody has ever worked it
7 out to say that we're going to apportion this amount to
8 cigarette smoke and this amount to asbestos. Because
9 from a molecular biology point of view, you really don't
10 have any idea what exactly is going on especially if you
11 contrast asbestos and cigarette smoke carcinogens can
12 cause some of the same mutations in the protooncogenes
13 and the tumor suppressor genes.

14 THE COURT: I want to move on to case reports.
15 I just want to get some practical understanding of what
16 you do when a new peer review case report comes across
17 your desk that is of interest to you.

18 How do you incorporate that, if at all, into
19 your practice; to what degree do you rely upon it; how
20 is it utilized by you as a clinician in the big picture?

21 THE WITNESS: I think as a pathologist it's
22 very important. Because a lot of things that are
23 published in pathology are case reports. And it might

1 be, for example, a different variant of a mesothelioma.

2 For example, in 1985 there was a case of a
3 tumor described in the peritoneal cavity of a 13-year
4 old girl that was initially described as a gynecologic
5 neoplasm but later was diagnosed as a type of
6 mesothelioma called deciduoid mesothelioma, where they
7 stated that this was not a type of mesothelioma caused
8 by asbestos.

9 And then, like a lot of things, I ripped that
10 out of the journal -- I'm a ripper. I rip this stuff
11 out that I think is important and save them, file them.
12 So I knew about that article. And then the first case I
13 saw was actually probably about 15 years later, maybe
14 ten years later in a old woman. Actually, she was
15 89 years old, who had been exposed to asbestos. I said
16 I've seen this type of tumor before. I said this is a
17 deciduoid mesothelioma. But this was a pleural
18 mesothelioma rather than a peritoneal mesothelioma. And
19 we actually did asbestos analysis on her lung tissue.
20 And she had elevated concentrations of asbestos.

21 So these case reports are very important to me
22 just because I think you can recognize things. And if
23 you see it again, you can diagnosis it.

1 Sometimes it's not so important with respect to
2 causation from how what happens to the person and how
3 they're treated, though. That's the way it is.

4 THE COURT:

5 (Pause.)

6 THE COURT: You gave some examples of instances
7 where epidemiology has not kept pace necessarily with
8 the ebb and flow of the medical practice, for example,
9 and what you're seeing in presentation of your patients
10 and decisions that you're having to make. And I assume
11 that you were making those comments in the context of a
12 clinician who is being asked to give advice or give
13 treatment to a particular patient who is there in your
14 office or whose case has been submitted to you for
15 review.

16 What about in the scientific world when you're
17 being called upon to offer guidance to the scientific
18 community in terms of cause and effect? How does the
19 lack of a epidemiological study or guidance from that
20 field affect your ability to offer that guidance to your
21 colleagues in the form of either published papers or
22 presentation that you're making knowing that folks are
23 going to be probably taking what you had to say and

1 maybe making very important decisions based on that?

2 THE WITNESS: Well, that's a very good
3 question.

4 And I think in a disease where a cause has not
5 been demonstrated, then you have to be very, very
6 careful. And I think if you have a situation like that,
7 say, you saw some type of a rare lung cancer or, say,
8 you saw a gastrointestinal stomal tumor and there was
9 somebody that thought that this was related to something
10 that had been reported, I think that the prudent thing
11 for a clinician or pathologist to do would be to say
12 that here is the information we know. We know this is a
13 rare tumor. We know that X, Y, and Z have reported that
14 this was associated with this. But at this point in
15 time there are too few cases to make any absolute
16 conclusions about it.

17 And I think that to make a statement that just
18 because a case report or two or three case reports had
19 stated, well, it's likely this was caused by that, and
20 you still have to be really careful about that, very
21 careful about that. Because if you don't, you can do
22 just as much harm by making false statements that way as
23 you can the opposite way.

1 THE COURT: All right. Did that prompt any
2 additional questions from counsel?

3 MR. JOHNSON: Just one, possibly two. Try to
4 make it one.

5 BY MR. JOHNSON:

6 Q. Dr. Hammar, in the case of chrysotile asbestos,
7 whether it comes from brake linings, whether it comes
8 from some other source, is that a situation in your
9 opinion where you're dealing with a disease where the
10 cause of the disease has not been identified?

11 A. No. I think that there is overwhelming
12 epidemiologic proof that chrysotile causes mesothelioma.
13 I don't think it's even a question there.

14 I guess in the brake mechanic issue is that
15 based on the epidemiologic studies there seems to be a
16 question as to whether chrysotile exposure causes an
17 increased incidence of mesothelioma based on what has
18 been published. And that if you -- and here -- and I'm
19 not an epidemiologist. And here is where my problem
20 comes with that is just what I've said, is that so here
21 I see a case of a person who we have good evidence that
22 was the only exposure they had. They have elevated
23 concentrations of asbestos in their lung. They have

1 elevated concentrations of asbestos in their pleura.
2 You don't find anything else. How can you not say that
3 that was caused by asbestos?

4 Q. In terms of the situation you were just
5 describing for the Court in response to the Court's
6 question, was that a situation in which you have a rare
7 form of disease and as of today there's only theories;
8 medical science doesn't really have any idea what is
9 causing it?

10 A. Yeah. And maybe the best example was is that
11 deciduoid mesothelioma. Because part of the publication
12 on that was that here's an example of mesothelioma
13 that's not caused by asbestos. Ten cases later there
14 were several case reports of deciduoid mesothelioma
15 being caused by asbestos or finding elevated
16 concentrations of asbestos in the lungs of those
17 individuals.

18 So, again, it's a thing one has to be -- a
19 situation I think -- and this is both ways -- you have
20 to be very, very careful about stating that it was not
21 caused by something or caused by something. Because you
22 can be wrong both ways, especially when there's
23 barely -- you know, there's not very much information

1 about it.

2 Q. Finally, just to make sure it's clear in
3 response to something else the Court was asking -- and
4 I'm going to try and shorthand a subject you and I have
5 discussed a million times over a period of years.

6 Neither you nor -- in a case where somebody is
7 exposed to multiple products, neither you nor anybody
8 else can say which fibers from which products were the
9 ones that actually caused the changes to the cell that
10 led to the cancer; correct?

11 A. That's absolutely correct.

12 MR. JOHNSON: Okay. Thank you.

13 BY MR. TARRY:

14 Q. Dr. Hammar, short fiber chrysotile, the
15 shortest available commercial grade or even shorter than
16 that -- I think Dr. Dodson testified yesterday about
17 short amphiboles, also. Those short fibers are the
18 types of fibers that all of us just in the general
19 population, to the extent we're exposed, are exposed to,
20 the shorter fibers; correct?

21 A. Well, I think that would be the most likely you
22 would get from nonoccupational exposure, yes.

23 Q. You said mesothelioma is a rare disease. The

1 vast majority of us who are carrying around these short
2 chrysotile fibers in our lungs right now -- when I say
3 short chrysotile fibers, I mean counting everything else
4 as equal; not talking about things like changes in
5 surface, charged or surface chemistry; it's been milled
6 or anything like that, just short fiber chrysotile --
7 the vast majority of us walking around with that stuff
8 in our lungs, in our pleura never develop mesothelioma;
9 correct?

10 A. I think that would be correct. But it's my
11 understanding -- maybe Dr. Dodson said something
12 differently -- but if you look at his article published
13 in 2000, I think, in the American Journal of Industrial
14 Medicine on asbestos concentrations on people who are
15 non-occupationally exposed, it's my understanding that
16 he didn't find any asbestos in the people that were
17 under 25 years old, short fibers, long fibers,
18 amphiboles, chrysotile, etc. And that's really terrific
19 news in a way.

20 You're a young guy. I'm an old guy. I don't
21 think that everybody, all the young guys in here have
22 chrysotile asbestos in their lungs. I don't think they
23 have anything in their lungs nowadays. I think those

1 people are free. I know where I was exposed to
2 asbestos. I did a lot of work when I was young kid on a
3 lot different things. But I don't think the young
4 people really are at risk.

5 Q. Let me make the three categories. Whether the
6 people who never get exposed to a single fiber or some
7 who get exposed just environmentally because they live
8 in the urban area perhaps, and that's been demonstrated,
9 or they have occupational exposure, the vast majority of
10 all of those never develop mesothelioma?

11 A. That is true. You could say that about
12 asbestos, cigarette smoke. The vast amount of people
13 who smoke cigarettes are not going to get lung cancer.
14 But the primary cause of lung cancer is cigarettes smoke
15 carcinogens. And the primary cause of mesothelioma is
16 asbestos.

17 Q. And we're able through the use of control
18 groups, right, to identify types of exposures and
19 certain circumstances of exposure where those risks
20 might weigh out?

21 A. That is true.

22 MR. WADDELL: Your Honor, just real quick on
23 that vein, too.

1 BY MR. WADDELL:

2 Q. If someone was to throw out really in regards
3 to smoking and lung cancer that there's no
4 epidemiological studies on Marlboro cigarettes, is that
5 accepted in the medical and scientific literature, well,
6 you know, that's right; Marlboros don't cause lung
7 cancer; is that reasonable?

8 A. No. Of course that's not reasonable.

9 Q. How about if they were Marlboros or Marlboro
10 Lights?

11 A. That would be a little more complicated, but
12 probably not any different.

13 Q. Filtered versus unfiltered?

14 A. No difference because of the way people smoke.

15 Q. Modified versus unmodified?

16 A. No different.

17 Q. How about because it all goes to an individual
18 circumstance, the dose; right?

19 A. Right.

20 Q. Okay. Any safe level dose of asbestos where
21 one's not at risk of developing mesothelioma?

22 A. Not with respect to an occupational or
23 bystandards exposure.

1 Q. And, Dr. Hammar, in applying, as a medical
2 doctor, causation principles, you have to look at the
3 individual circumstances; correct?

4 A. Yes.

5 Q. Blanket approaches on doses by classification
6 or products can be maybe helpful as a clinician, as a
7 physician, but not always the rule of thumb; right?

8 A. Sure. I think that's true, sure.

9 Q. It depends on the circumstances again,
10 individually?

11 A. It does.

12 MR. WADDELL: Thank you, Dr. Hammar.

13 THE COURT: All right. Doctor, I think that's
14 it. You may step down.

15 THE WITNESS: Thank you, Judge.

16 THE COURT: Folks, we're going to take our
17 luncheon recess. We'll recess for an hour, at least
18 that's when I'm going to ask you to get back. I'm going
19 to tell you, if I'm at the DMV at ten till two and they
20 have not yet taken care of me, then I'll leave and do
21 that on another day if I need to. But I'm going to try
22 to get it done today because it needs to get done. So
23 if I'm a little late, I guess what I'm saying is, please

1 *excuse me. We'll stand in recess.*

2 *(Whereupon a lunch recess was taken at 12:49*
3 *p.m.)*

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CERTIFICATE OF COURT REPORTER

We, Lynne B. Coale, Registered Diplomate Reporter and Certified Realtime Reporter, and Domenic Verechia, Registered Professional Reporter, Official Court Reporters of the Superior Court, State of Delaware, do hereby certify that the foregoing is an accurate transcript of the proceedings had, as reported by us, in the Superior Court of the State of Delaware, in and for New Castle County, in the case herein stated, as the same remains of record in the Office of the Prothonotary at Wilmington, Delaware.

WITNESS our hands this 18th day of October, 2005.

Lynne Bell Coale, RDR, CRR,
Cert. # 165-PS
Official Court Reporter

Domenic Verechia, RPR, Cert. # 162-PS
Official Court Reporter

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