

1 NO. 03-CV-0801

2 RICK MONTALBANO, ET AL. * IN THE DISTRICT COURT OF
3 *
4 VS. * GALVESTON COUNTY, TEXAS
5 *
6 THE LINCOLN ELECTRIC COMPANY, *
7 ET AL. * 56TH JUDICIAL DISTRICT
8
9

10 ORAL DEPOSITION OF

11 DAVID H. GARABRANT, MD

12 OCTOBER 19, 2006

13
14
15 THE ORAL DEPOSITION OF DAVID H. GARABRANT, MD, duly sworn,
16 produced as a witness at the instance of the DEFENDANTS,
17 was taken in the above styled and numbered cause on the
18 19th of October, 2006, from 11:02 a.m. to 6:36 p.m., before
19 Suzi Gladney, CSR, RPR, Certified Shorthand Reporter in
20 and for the State of Texas, reported by machine shorthand,
21 at the offices of Strong, Pipkin, Bissell & Ledyard, LLP,
22 1301 McKinney, Suite 2100, Houston, Texas, pursuant to the
23 Texas Rules of Civil Procedure and the provisions stated on
24 the record.
25

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1 (Garabrant Exhibit No. 1 was marked)

2 DAVID H. GARABRANT, MD,

3 having been first duly sworn, testified as follows:

4 EXAMINATION

5 BY MR. LUBEL:

6 Q. Please state your full name.

7 A. David Hay Garabrant.

8 Q. What is your profession?

9 A. I'm a physician.

10 MR. BISSELL: Before we get to it, I just
11 want to say this was originally my notice of this
12 deposition pursuant to a motion that was heard before the
13 Court and discussions that were held before the Court. We
14 have agreed that you will go forward and take the
15 deposition that is intended to be a trial witness.

16 From my standpoint -- so, it's your
17 deposition -- I will agree with you on the record in spite
18 of all the prior off the record that because I noticed and
19 retained the reporter, I'll take the payment for it; but
20 it's your deposition.

21 Q. (BY MR. LUBEL) Did you finish your answer,
22 Doctor?

23 A. I am a physician and an epidemiologist.

24 Q. What state do you live in?

25 A. Michigan.

1 Q. What city?

2 A. Ann Arbor.

3 Q. How long have you lived there?

4 A. Almost 18 years.

5 Q. How long have you been a medical doctor?

6 A. I graduated medical school in 1976.

7 Q. Who hired you in this case?

8 A. I believe it was Ms. Haley McIntosh at
9 O'Melveny & Myers.

10 Q. And where is she located?

11 A. Los Angeles.

12 Q. And how did you know her?

13 A. I had worked with her on other welding cases.

14 Q. And when were you first consulted with regarding
15 any welding case?

16 A. I'm not sure I know exactly. I would estimate
17 2002.

18 Q. And have you pretty much on a regular basis
19 worked on various welding related matters or cases since
20 then?

21 A. I have at various times. I wouldn't say
22 regularly, but it comes and goes.

23 Q. And here's the point that I'm driving at and the
24 question: Would it be a fair statement that since you
25 started working on the welding related litigation four or

1 so years ago that your opinions, so to speak, have been
2 formed; and you're prepared to speak about them today?

3 A. Well, I had -- over that four-year period, I have
4 continued to review scientific literature and other
5 documents and my opinions have been reformulated in
6 response to that over the entire period but yes, I am
7 prepared to --

8 Q. As you sit here today, are you prepared to offer
9 us what your opinions are regarding welding?

10 A. Yes.

11 Q. And are you familiar with the literature
12 pertaining to manganese exposures?

13 A. Well, I'm familiar with the literature pertaining
14 to welding and --

15 Q. I take it you have not reviewed the literature
16 pertaining to manganese exposure?

17 A. I was trying to finish my previous answer.

18 I'm familiar with the literature regarding welding and
19 risks of neurological injury.

20 Q. That really wasn't my question, though, Doctor.
21 I asked you if you were familiar with the literature
22 pertaining to manganese exposure.

23 MR. BISSELL: Lance, he was trying to finish
24 up the answer in response to your question.

25 MR. LUBEL: I hear you.

1 A. I have some familiarity with some of the
2 literature regarding manganese exposure.

3 Q. (BY MR. LUBEL) And how did you familiarize
4 yourself with the literature regarding manganese exposures?

5 A. Well, I have read that literature at various
6 times throughout my career; and I sought to review some of
7 it over the past four or five years.

8 Q. And do you consider yourself an expert in that
9 general body of literature pertaining to manganese
10 exposures?

11 A. Well, you'll have to clarify for me what body of
12 literature you're talking about. Are you talking about the
13 industrial hygiene literature on manganese exposure, or are
14 you talking about the literature on the health effects
15 of manganese exposure?

16 Q. Health effects.

17 A. Oh. I have some familiarity with it.

18 Q. Do you consider yourself an expert on the
19 literature pertaining to the health effects of manganese
20 exposure?

21 A. I consider myself to be an expert in the area of
22 the epidemiology studies of the health effects of manganese
23 exposure. However, I have not prepared today to discuss
24 all of those things in detail.

25 Q. Why not?

1 A. Because I thought today's deposition was about
2 welding.

3 Q. Have you ever been, in your opinion, thoroughly
4 prepared to discuss the body of literature that pertains to
5 the health effects of manganese exposures?

6 A. I have not taken the effort to systematically
7 prepare opinions to discuss that nor have I ever been asked
8 to.

9 Q. And I take it that had you been asked to, you
10 would have performed this system -- what did you say --
11 systematic evaluation --

12 A. A systematic review, yes.

13 Q. Is that a fair statement that had you been asked
14 by the welding companies to perform this analysis, you
15 would have performed a systematic review or evaluation of
16 that body of literature?

17 A. It would be fair to say that had I been asked to
18 review all of the epidemiology of manganese exposure and
19 neurologic effects that I would have undertaken to do so;
20 but I have not been asked to do it nor has it come up in
21 deposition previously or in trial.

22 Q. Do you consider yourself an expert in the
23 neurological effects of exposure to manganese?

24 A. I consider myself an expert in the epidemiology
25 of neurologic effects of manganese. However, I have not

1 reviewed that literature systematically in preparation for
2 today's deposition.

3 Q. And I guess I'm kind of being more narrow in that
4 what I'm trying to find out -- because you're not a
5 neurologist, correct?

6 A. That's correct.

7 Q. And you've never held yourself out as a
8 neurologist, true?

9 A. That's correct.

10 Q. And you don't claim to have any special expertise
11 in the area of movement disorders, true?

12 A. Correct.

13 Q. And that's generally the body of medicine that
14 applies to neurologists and neurosurgeons, true?

15 A. I'm not sure I understand the question.

16 Q. Typically when somebody has a movement disorder,
17 aren't they referred to a neurologist?

18 A. I would say typically -- well, if they're
19 referred at all, that would be the appropriate place to
20 refer them.

21 Q. And if we're looking at the different branches of
22 medical doctors and if you were asked what branch would
23 fall under the movement disorder tree, if you will, it
24 would be neurologists, correct, or neurology?

25 A. I'm not sure I'd formulate it that way. I would

1 say that movement disorders tend to fall within the domain
2 of practice by neurologists.

3 Q. That's because they have expertise in that area,
4 I take it. Is that a true statement?

5 A. I believe it is one of the areas in which they
6 train.

7 Q. And given your training, what does a
8 neurologist -- how is a neurologist trained differently
9 from somebody like yourself with your background?

10 A. Well, I'm trained in internal medicine and in
11 occupational medicine and in epidemiology, which means that
12 I have familiarity with a broad range of adult diseases.
13 My training gives me more depth in many diseases that
14 neurologists don't have depth in. In contrast,
15 neurologists typically spend far more time dealing with
16 neurologic diseases than I do. So, my training is broader
17 and encompasses diseases that they have little contact
18 with. Their training is narrower and is intensive on the
19 neurologic diseases.

20 In addition to that, my training is in occupational
21 medicine, which gives me substantial familiarity with
22 workplaces, the tests that are done in various jobs, the
23 settings in which those jobs are done, how chemicals are
24 handled, how exposures are generated, and appropriate
25 strategies to minimize the risks from those exposures.

1 In addition to that, my training in epidemiology and
2 my experience conducting research studies in occupational
3 epidemiology equips me to understand the relationship
4 between workplace exposures and disease risk factors with
5 substantially more expertise than neurologists.

6 Q. What are the clinical manifestations of
7 Parkinsonism?

8 A. Parkinsonism refers to a group of disorders, all
9 of which share certain abnormalities of movement. Those
10 abnormalities of movement include bradykinesia, sometimes
11 cogwheel rigidity, sometimes diminution of facial
12 expressions, difficulties with balance, often resting
13 tremors, and other signs and symptoms in addition.

14 Q. Are those the most common?

15 A. I believe so.

16 Q. What's the difference between a sign and a
17 symptom?

18 A. Signs are things that healthcare providers
19 observe. Symptoms are things that patients report.

20 Q. And those can often be the same, but they can
21 also be different?

22 A. They are usually different. Only uncommonly are
23 we talking about the same things.

24 Q. So, when you gave us your list of the clinical
25 manifestations of this group of disorders that fall under

1 Parkinsonism -- for instance, you gave bradykinesia,
2 correct?

3 A. Yes.

4 Q. Is that a sign or a symptom?

5 A. That's a sign.

6 Q. It's something a doctor would observe?

7 A. Yes.

8 Q. What is it?

9 A. Slowness of movement.

10 Q. And how does the neurologist gauge whether it's
11 slow enough to be called bradykinesia?

12 A. Based upon experience.

13 Q. But what objective measure does the neurologist
14 have on, you know, whether you've crossed the threshold
15 from not being bradykinesia to being bradykinesia?

16 A. Well, the objective evidence is what the
17 neurologist observes.

18 Q. But I guess what I'm asking is: What do you
19 expect the neurologist to observe in order to call it
20 bradykinesia? Give us an idea of what that means other
21 than saying it's slowness of movement.

22 A. That when you ask a patient to do something that
23 requires fast movements, they cannot do it at a rate that
24 you believe represents a normal rate of speed.

25 Q. And what are the tests that are commonly

1 available to a neurologist to determine whether there is
2 bradykinesia?

3 A. Things like rapid, alternating hand movements.
4 That would be a good example that's commonly used. Finger
5 tapping, eye blinking, things like that.

6 Q. Well, I think you referenced as one of the
7 clinical manifestations of Parkinsonism -- I don't know if
8 you used this exact term -- but postural instability?

9 A. Yes.

10 Q. What is that?

11 A. Having difficulty maintaining one's posture or
12 balance.

13 Q. And how does the doctor objectively test for
14 that? And by "doctor" I mean the neurologist.

15 A. Or an internist, for example.

16 We typically test for that by the Romberg test or by
17 doing a pull test.

18 Q. What was the second one?

19 A. A pull test.

20 Q. What is that?

21 A. Have the patient stand and you pull them towards
22 you at the shoulders so that they are resisting being
23 pulled forward and then you let go suddenly and see what
24 they do.

25 Q. What's the Romberg test?

1 A. Standing together -- standing with feet together
2 with eyes open and observing sway and then sometimes having
3 the person stand feet together with eyes closed and
4 observing sway and then sometimes pushing the person a
5 little bit on the shoulder one way or other to see how they
6 respond to a stressor.

7 Q. Was gait change in your clinical manifestations
8 of Parkinsonism?

9 A. I don't believe I mentioned it, but it is often
10 part of Parkinsonism.

11 Q. Is it appropriate to include that within your
12 list?

13 A. Yes.

14 Q. What type of gait changes does one look for -- a
15 doctor -- in deciding whether he's consistent with
16 Parkinsonism?

17 A. One abnormality that's looked for is festinating
18 gait.

19 Q. What does that mean?

20 A. I have no idea what the word festinating actually
21 means.

22 Q. We won't hold it against you. What do you think
23 it means?

24 A. Well, I can describe the gait. Parkinson's
25 patients often have difficulty getting started from a stop.

1 And so, they will sort of march in place, first with very
2 small amplitude and then larger and larger amplitude. And
3 eventually they will lean forward and begin to move.

4 Q. You've just described my kid when I say, "Go
5 clean your room." Is it something like that?

6 A. I don't think so. I don't think so.

7 Q. What do you call that?

8 MR. LUBEL: Recalcitrance.

9 (Laughter)

10 A. And the second thing is that they halt. They
11 tend to get stuck. So, they'll halt for reasons that don't
12 stop the rest of us. They'll halt because there's a small
13 obstacle in their way like a leaf or crack in the sidewalk
14 or a bump in the sidewalk. They get stuck, and they have
15 trouble getting going again.

16 Q. (BY MR. LUBEL) Okay. Have you given us the
17 primary clinical manifestations of Parkinsonism?

18 A. I believe so.

19 Q. There's others, correct?

20 A. I believe so.

21 Q. What are the primary clinical manifestations of
22 Parkinson's disease, otherwise known as PD?

23 A. There's a great deal of overlap with Parkinsonism
24 because Parkinson's disease is a form of Parkinsonism. The
25 clearest differentiating factor between Parkinson's disease

1 and other forms of Parkinsonism is the patient's response
2 to a trial of L-dopa.

3 Q. Is that also called levodopa?

4 A. Yes.

5 Q. Why do they sometimes refer it as L hyphen dopa
6 and then other times levodopa?

7 A. "L" stands for levo.

8 Q. It does?

9 A. Yes, sir.

10 Q. And what is levodopa?

11 A. It's a neurotransmitter that is synthesized in
12 the substantia nigra pars compacta.

13 THE REPORTER: Say that last part again for
14 me, please.

15 THE WITNESS: Pars compacta, P-A-R-S
16 C-O-M-P-A-C-T-A.

17 Q. (BY MR. LUBEL) Is there any other difference
18 between Parkinsonism and PD other than this response to
19 levodopa?

20 A. Well, there are many other types of Parkinsonism
21 besides Parkinson's disease; and yes, they have
22 differentiating features that allow a neurologist to
23 distinguish among them.

24 Q. What else is there other than -- you've given us
25 this response to levodopa.

1 A. I don't understand the question.

2 Q. What other features are you referring to other
3 than you've told us about one of them being a response to
4 levodopa?

5 A. I'm still not understanding. Features of what?

6 Q. The differences between PD and Parkinsonism.

7 A. Well, that's the clearest one. Other forms of
8 Parkinsonism have different signs and symptoms that overlap
9 with Parkinson's disease, but a good neurologist can
10 differentiate them.

11 Q. How so?

12 A. By the history and physical exam as to what the
13 findings -- what the pattern of findings and symptoms and
14 signs are.

15 Q. And what would one expect to see in the history
16 and physical exam that would be different between a PD and
17 a Parkinsonism?

18 A. I can't answer that the way you've asked it. PD
19 is a form of Parkinsonism.

20 Q. Well, I thought you told us that a good physician
21 can pick up the difference based upon a physical exam and
22 history. Did I hear you wrong?

23 A. Well, you've got -- no, you heard me right; but
24 that's only half of my answer.

25 Q. What was the rest of it?

1 A. I don't recall exactly. The question I think you
2 were asking is what are the characteristics of Parkinson's
3 disease and how do you differentiate that from other forms
4 of Parkinsonism, and I answered that.

5 Q. Let me do it this way: You've gone through and
6 given us the basic list of clinical manifestations or signs
7 that you would expect to see in a patient with
8 Parkinsonism, correct? We've done that.

9 A. I've given you -- yeah.

10 Q. Now, what I'd like you to do is to tell us what
11 the signs and the clinical manifestations that one would
12 find in Parkinson's disease, PD.

13 A. Well, we've done that. It's many of the same
14 symptoms and signs and the -- well, there are other things
15 as well. Parkinson's disease often has elements of
16 micrographia, fixed facies. The resting tremor is
17 prominent, often begins unilaterally. Cogwheel rigidity is
18 prominent particularly in Parkinson's disease.

19 Q. But those are also found in plain-Jane
20 Parkinsonism, correct?

21 A. Well, the way you keep asking the question makes
22 it difficult to answer. Parkinson's disease is a form of
23 Parkinsonism. And so, you've been asking me to
24 differentiate Parkinson's disease from the others; but
25 there is overlap.

1 Q. Let me tell you why: I hear you saying there's
2 overlap. But in my short experience in this field of
3 neurology, just from studying for this case, there seems to
4 be a difference between the diagnosis of Parkinsonism and
5 Parkinson's disease; is that true? Is there a difference
6 in those two, in diagnosing?

7 MR. BISSELL: But those two aren't two.
8 Those two are a whole bunch. I think that's the problem
9 with the question.

10 A. I'll try to answer it. There are other forms of
11 Parkinsonism. And so, Parkinsonism is an umbrella term
12 that includes Parkinson's disease, manganism, Shy-Drager
13 syndrome, progressive supranuclear palsy, and others. And
14 so, you keep saying, well, compare Parkinson's disease to
15 Parkinsonism; and I can't because Parkinson's disease is a
16 form of Parkinsonism.

17 Q. (BY MR. LUBEL) Let's do it this way: Have you
18 heard of neurologists diagnosing a patient with PD?

19 A. I have heard of that.

20 Q. Have you also heard of neurologists diagnosing
21 patients with Parkinsonism?

22 A. Yes.

23 Q. Are they the same, or are they different?

24 A. Well, they can be the same. They can be
25 different. In other words, Parkinson's disease is a type

1 of Parkinsonism; and I'm not sure that the usage within the
2 neurologic specialty is entirely uniform. And so, you may
3 get some neurologists who say this patient has Parkinson's
4 disease. You may get others who say they have Parkinsonism
5 even though they fulfill all the diagnostic criteria for
6 Parkinson's disease.

7 Q. Maybe that's the way I need to ask it. What is
8 the diagnostic criteria for Parkinson's disease?

9 A. I would have to get them out. I don't have them
10 memorized.

11 Q. Grab them if you don't mind.

12 A. Sure.

13 Q. Thanks.

14 (Recess from 11:28 a.m. to 11:30 a.m.)

15 A. I have them in front of me.

16 Q. (BY MR. LUBEL) Okay. Can I just look at what
17 you're looking at? Are they two different ones? I mean, I
18 know there are two different ones but is there a preferred
19 one or do you need them both?

20 A. I don't know whether one is preferred. Both are
21 commonly used.

22 Q. Okay. I'm going to mark these.

23 (Discussion was held off the record)

24 (Garabrant Exhibit Nos. 2 and 3 were marked)

25 Q. (BY MR. LUBEL) Doctor, you've been kind enough

1 to pull out of your materials what I've marked as
2 Exhibits 2 and 3, which I believe you've told us constitute
3 the references to the diagnostic criteria for PD?

4 A. Yes.

5 Q. Do you have anything similar for the diagnostic
6 criteria for Parkinsonism?

7 A. No.

8 Q. All right. Let me get those back real quick.
9 What is the unified Parkinson disease rating scale?

10 A. It's the document you've marked as Exhibit 2.

11 Q. Have you seen in the literature references to
12 using this unified Parkinson disease rating scale in
13 diagnosing Parkinsonism?

14 A. I don't recall any.

15 Q. Would it surprise you if there is references to
16 that?

17 A. I'm not easily surprised.

18 Q. You see anything wrong with it?

19 A. You'd have to show me how it's used before I
20 could answer that.

21 Q. Well, do you see anything wrong with a doctor
22 using this unified Parkinson's disease rating scale to
23 diagnose Parkinsonism?

24 A. Again, I'd have to see how it was used before I
25 could answer that.

1 Q. I'm not asking you about the particular
2 reference. I'm just saying in general, in your practice,
3 do you ever see any problem with it?

4 A. I don't have an answer in general. It depends on
5 how it's used.

6 Q. Can you think of any circumstances in which it
7 would be appropriate to use it?

8 A. Well, the UPDRS provides a rating system for
9 rating movement disorders. It reflects a way of codifying
10 the results of a physical examination. That has utility
11 simply because it tries to standardize what signs are noted
12 and how severe they are. It can be used for that purpose
13 without using it to make an inference as to whether a
14 patient does or does not have Parkinson's disease. It's
15 most commonly used to rate the severity of Parkinson's
16 disease in someone who has that constellation of findings
17 and in whom a diagnosis of Parkinson's disease has been
18 made.

19 Q. But I take it by the first part of your answer
20 that it's appropriate to use the UPDRS, that scale, to rate
21 movement disorders including Parkinsonism, correct?

22 A. I said it can be used for that if it -- when used
23 in that way would not establish that a patient has
24 Parkinson's disease, but it would allow one to rate the
25 various aspects of a movement disorder and to try to

1 quantify them.

2 Q. And when we look at Exhibit No. 3 -- which has
3 multiple pages, correct?

4 A. Yes.

5 Q. -- it provides for -- are these signs or symptoms
6 or both of Parkinson's disease?

7 A. I think it includes both. Let me see it.

8 It includes signs and symptoms.

9 Q. Now, if you use that same Exhibit No. 3,
10 Dr. Garabrant, does it include the same signs or symptoms
11 that can be diagnostic of Parkinsonism?

12 A. Well, some of the signs and symptoms on this
13 document are present in Parkinsonism. I don't believe that
14 rating a person according to this document would allow one
15 to reach a diagnosis of Parkinsonism.

16 Q. But from Exhibit No. 3 where it lists the signs
17 or symptoms, which ones are not appropriate for
18 Parkinsonism?

19 MR. BISSELL: You know, I'm going to object
20 to the form of the question. It's already been established
21 that PD is a Parkinsonism; and the minute you don't give
22 him anything more than that, the form of the question is
23 totally defective because he can't possibly answer. And
24 he's told you that.

25 MR. LUBEL: That will be denied.

1 MR. BISSELL: I don't think so. I often
2 find my objections overruled. I often overrule them
3 myself. That one in particular, I think, is good.

4 THE WITNESS: Could you repeat the question?

5 Q. (BY MR. LUBEL) Sure. Are there any signs and
6 symptoms on Exhibit 3 that are not applicable to
7 Parkinsonism? Or if applicable is not a word you're
8 comfortable with, attributable to.

9 A. Well, I can't answer it the way you've asked it.
10 Parkinson's disease is a Parkinsonism; and if these are
11 signs and symptoms for Parkinson's disease, they are
12 applicable to that form of Parkinsonism.

13 Q. Okay. Can overexposure to manganese cause a form
14 of Parkinsonism?

15 A. Yes.

16 Q. What do you call that form?

17 A. Manganism.

18 Q. Are there any signs and symptoms on Exhibit 3
19 that you believe are not attributable to the form of
20 Parkinsonism called manganism?

21 A. Well, there's some I don't know about. There are
22 other things that are typical of manganism that are not on
23 this list; and there's some here that are clearly more
24 associated with Parkinson's disease than they are with
25 Parkinsonism or manganism. So, I don't know how to answer

1 the question as asked.

2 Q. Let's do it this way.

3 A. Well, let me finish.

4 Q. Go ahead.

5 A. So, there's overlap; but this would not be the
6 way one would rate manganism. One would want to come up
7 with a different set of criteria.

8 Q. What signs or symptoms on Exhibit 3 are you
9 unsure of as to whether they also show up in manganism?

10 A. Well, for example, Parkinson's disease often
11 begins unilaterally. Manganism is typically bilateral.
12 Not to say it can't be the other way in both diseases, but
13 typically manganism is bilateral. Often, not always,
14 Parkinson's disease begins unilaterally. I don't recall
15 drooling as being part of manganism. That's all I can
16 recall.

17 Q. All right. You're looking at Exhibit 3, which
18 you've already told us constitutes a list of signs and
19 symptoms that are diagnostic of Parkinson's disease,
20 correct?

21 A. These are signs and symptoms that are typically
22 seen in Parkinson's disease. This alone is not adequate to
23 reach a diagnosis of Parkinson's disease.

24 Q. What is a diagnostic criteria for Parkinson's
25 disease? What must you have to diagnose it?

1 A. I'm not sure there's any clear delineation of
2 what you must have. It's a clinical diagnosis where you
3 must have typical signs and symptoms, and neurologists
4 often use a therapeutic trial of L-dopa as diagnostic
5 confirmation. And so, sometimes neurologists do not feel
6 comfortable diagnosing Parkinsonism based on just the
7 symptoms and signs. They want to see how the patient
8 responds to L-dopa. Sometimes the disease is so classical
9 in its presentation that a neurologist will reach that
10 diagnosis without a diagnostic or therapeutic trial of
11 L-dopa.

12 I should also mention part of reaching a diagnosis of
13 Parkinson's disease often includes ruling out other
14 possible explanations for movement disorders such as
15 cerebrovascular disease.

16 Q. Trauma?

17 A. Trauma, there are a number of other etiologies.
18 And so, simply filling out the Hoehn & Yahr staging is not
19 reaching a diagnosis. One has to evaluate other possible
20 causes and rule them out. One has to see a typical set of
21 signs and symptoms, and one sometimes seeks confirmation
22 with a trial of L-dopa.

23 Q. Well, let's see if we can kind of put that all
24 together. In your opinion in order for a physician to
25 diagnose PD, the patient will need to have signs and

1 symptoms consistent with it, there needs to be a ruling out
2 of more plausible or possible causes; and in a lot of
3 instances, there needs to be a sustained response to
4 L-dopa; is that true?

5 A. To the extent of my knowledge of neurology, that
6 is a reasonable characterization.

7 Q. Now, how does one measure what a sustained
8 response to levodopa is?

9 A. I would have to say I'm not adequately familiar
10 with that to answer that question today. I didn't review
11 that topic.

12 Q. But you've seen, Dr. Garabrant, in the literature
13 references to levodopa having partial success, efficacious,
14 partially efficacious, terms like that; have you not?

15 A. I don't recall those terms. I have not reviewed
16 this entire topic for today's deposition.

17 Q. Well, what I'm trying to find out is if you can
18 give us any indication of how the neurologist judges -- as
19 you sit here today -- whether or not the response to
20 levodopa is indicative of PD or not indicative of PD.

21 A. I can't answer that as I sit here today.

22 Q. One of other areas of the diagnosis you mentioned
23 was that the neurologist should perform a differential
24 diagnosis in an attempt to rule out other possible causes,
25 true?

1 A. Yes.

2 Q. Do you know what that list is, what that looks
3 like?

4 A. I don't understand the question.

5 Q. What the other possible causes are.

6 A. Well, I've listed two of them. That's all I can
7 think of from memory.

8 Q. And that was trauma and cerebrovascular disease?

9 A. Yes.

10 Q. Commonly known as stroke?

11 A. Could be a stroke, could be vascular
12 insufficiency. I suppose it could also be vasculitis. And
13 so, there -- I do not recall from memory what the
14 differential diagnosis of causes of Parkinsonism is. So --
15 but there would be a list of things to rule out.

16 Q. From your experience do you know whether or not
17 there's any type of scan that can be done that will
18 objectively tell a neurologist whether or not the patient
19 has PD? And by that I mean like MRIs, CTs, PET scans,
20 SPECT scans, anything like that.

21 A. Well, there are scans that will allow a
22 neurologist to get an image of the basal ganglia and to
23 evaluate the size and radiodensity of the various ganglia
24 which assists in defining whether, for example, the ganglia
25 are the right size or they have adequate blood supply,

1 whether their metabolic activity is normal or abnormal; and
2 those scans assist in reaching a diagnosis. The scans are
3 not diagnostic to my knowledge.

4 Q. In and of themselves?

5 A. In and of themselves.

6 Q. So, for instance, in an individual patient --
7 let's just assume that they have the signs and symptoms
8 indicative or reflective of PD, the good physician has
9 ruled out the possible or plausible other potential causes
10 that you've referenced, and then they have a scan. Okay?
11 What would you expect to see in a scan that would tell you
12 one way or the other?

13 A. I have not reviewed that topic, and I couldn't
14 answer it from memory.

15 Q. Generally speaking, Dr. Garabrant, isn't it
16 believed that a patient with PD, the reason they have PD as
17 opposed to something else is because there's been some
18 reduction of dopamine neurons in the brain?

19 A. Well, it's far more specific than that. It is
20 believed they have Parkinson's disease because the
21 dopaminergic, D-O-P-A-M-I-N-E-R-G-I-C, neurons in the
22 substantia nigra pars compacta die and cease to produce
23 adequate amounts of dopamine.

24 Q. The neurons die?

25 A. Yes, in the substantia nigra pars compacta.

1 Q. Then do you have a complete loss of dopamine, or
2 do you have something less than a complete supply of
3 dopamine?

4 A. Depends how many neurons die.

5 Q. But how many neurons need to be dead in a
6 particular individual for it to be PD?

7 A. I don't know.

8 Q. Does anybody?

9 A. I don't know.

10 Q. Does there need to be a particular level of
11 dopamine reduction in order for the neurologist to call it
12 PD as opposed to something else?

13 A. I haven't reviewed that literature. I don't
14 know.

15 Q. Do we know from the literature how the neurons
16 die or what the potential causes of the neurons dying are?

17 A. I think that's the great mystery. We don't
18 largely.

19 Q. Well, if we take like Muhammad Ali. Did he have
20 PD, or do you know? I know you didn't examine him, but I'm
21 just talking about kind of what you've read.

22 A. He's still alive, I think. Let's put him in the
23 present.

24 Q. Right.

25 A. I don't know whether he actually does or does not

1 because I haven't seen his medical record. I haven't
2 examined him. He clearly has a movement disorder. The
3 movement disorder he has, it is my understanding, is not
4 uncommon in boxers as they get older; and it is believed
5 that it comes from repeated head trauma. I don't know
6 whether it is -- whether a qualified neurologist would
7 classify that as Parkinson's disease or not.

8 Q. Okay. So, you believe in Parkinson's disease
9 that brain damage is in a particular sector of the brain,
10 and I think you called it the substantia nigra pars
11 compacta. Is that true?

12 A. Well, I believe that is the source of the
13 problem.

14 Q. Is there any other area of the brain that is a
15 source or contributing source of the problem for PD?

16 A. Not to my knowledge.

17 Q. Let's talk about Parkinsonism caused by
18 overexposure to manganese. What are the areas of the brain
19 that are affected by it?

20 A. The globus pallidus.

21 Q. Are there any others?

22 A. I'm not aware that there is reliable evidence
23 that manganism results from damage to any other area of the
24 brain besides the globus pallidus.

25 Q. Can you pull for me in your boxes of materials

1 what you're relying upon for that opinion? Take your time.

2 MR. LUBEL: We can go off the record.

3 (Recess from 11:52 a.m. to 11:58 a.m.)

4 (Garabrant Exhibit No. 4 was marked)

5 Q. (BY MR. LUBEL) You've pulled out as an example
6 of support for your opinion the exhibit that I've marked as
7 Exhibit No. 4 with the proviso that you're not saying that
8 this is the only one in your materials, correct?

9 A. That's correct.

10 Q. All right. Let me ask you this: There clearly
11 is support in the literature that the basal ganglia is
12 adversely affected from overexposure to manganese. Is that
13 a true statement?

14 A. Well, it's too vague to answer. The globus
15 pallidus is one of the basal ganglia. The globus pallidus
16 is affected, but I don't think it's fair to that say the
17 rest of the basal ganglia are all affected or are affected
18 in the same way.

19 Q. But would you acknowledge that I could probably
20 pull out a number of literature references that would refer
21 to the damage done by manganese exposure as affecting the
22 basal ganglia? That wouldn't -- you've seen that?

23 A. I don't know whether you could pull those out or
24 not.

25 Q. Okay. Would that surprise you if I could?

1 A. The question is too vague to answer.

2 Q. Let me ask it better.

3 A. You can find manganese in other basal ganglia
4 besides the globus pallidus, and there's plenty of
5 literature that will support that. But as to whether it's
6 actually doing damage in those other structures or is
7 simply present is a much more subtle question.

8 Q. All right.

9 A. And we'd have to look at that literature before I
10 could answer that.

11 Q. Are you prepared to answer that today?

12 A. I did not prepare on that question today. I
13 prepared on the epidemiology.

14 Q. All right. So, you're not prepared to discuss it
15 today, correct?

16 A. That's correct.

17 MR. BISSELL: Other than what he's told you.

18 MR. LUBEL: Yeah, I can understand what
19 you're saying.

20 Q. (BY MR. LUBEL) But you would agree that there is
21 a substantial amount of evidence that manganese can
22 accumulate in other areas of the brain than the globus
23 pallidus, correct?

24 A. I would agree that manganese can be found. I'm
25 not sure that it's fair to characterize it as accumulating.

1 It can be found in other areas of the brain; and, in fact,
2 manganese is an essential micronutrient that is required in
3 small amounts in every cell.

4 Q. And has from your review of the literature -- or
5 at least your recollection of the review of the
6 literature -- have you seen references to manganese being
7 found in the substantia nigra?

8 A. I do not recall those references. I believe
9 there are some. But as I just said, manganese is an
10 essential cofactor for some enzymes in all cells. It is
11 present in all cells in normal health. So, yes, it can be
12 found everywhere if one has an adequately sensitive method
13 of looking for it.

14 Q. And have you seen the literature that refers to
15 MRI studies of where the high signals are found in these
16 manganese exposed people?

17 A. I have seen some literature on MRI signals among
18 manganese exposed workers.

19 Q. In what areas of the brain do those studies
20 reflect manganese can be found?

21 A. Well, I can't answer the question as you've asked
22 it. Typically those studies look for abnormal
23 hyperintensity of certain signals rather than, as you've
24 characterized it, areas in which some manganese can be
25 found.

1 Q. And what areas of the brain would those abnormal
2 signal intensities be found?

3 A. Typically the globus pallidus.

4 Q. Any other areas?

5 A. I can't say from memory; but in people who have
6 been overexposed to manganese, typically one finds signal
7 hyperintensity in the globus pallidus.

8 Q. Now, how does the person that is overexposed to
9 manganese, how does the manganese get from the air into the
10 brain?

11 A. Well, I don't believe there is any direct
12 evidence of how that occurs. I don't think anyone's ever
13 done tracer studies that would allow us to trace a
14 manganese exposure from air into the brain of a human.

15 Q. Do any of the studies make reference to how
16 manganese gets from the air into the brain?

17 A. Well, I think some of the studies make
18 assumptions about that.

19 Q. What do they assume?

20 A. We'd have to look at them. I'm not sure they all
21 assume the same thing.

22 Q. Does a person that's overexposed to manganese,
23 for instance in a welding environment, do you assume that
24 the manganese enters their lung; or do you know?

25 MR. BISSELL: Object to form.

1 A. The way you've characterized welding presumes
2 someone is overexposed to manganese, and I'm not aware that
3 that's a fair characterization.

4 Q. (BY MR. LUBEL) I'm asking you to assume that a
5 welder is overexposed to manganese. What I'm trying to
6 find out is: Does the welder inhale it, and is that how
7 the manganese makes its way to the brain? Or does it
8 happen some other way?

9 A. I don't know what you mean by "overexposed to
10 manganese."

11 Q. Exposed at all. Just assume with me that the
12 welder has been exposed enough for which manganese is found
13 in the brain. Okay?

14 A. Manganese can be found in the brain in people who
15 don't weld. So, I'm not sure I understand the question
16 yet.

17 Q. Okay. Do you know whether or not a welder who's
18 overexposed to manganese can inhale manganese that makes
19 its way to his or her brain?

20 A. Could you tell me what you mean by "overexposed
21 to manganese"?

22 Q. An exposure sufficient to make its way from their
23 body up to their brain.

24 A. Well, I don't believe we've ever done any tracer
25 studies that establish how that occurs. I believe that --

1 and let me try and restate your question because there are
2 elements to it that are -- that make it such that I cannot
3 answer as asked. If your question is does the inhalation
4 of welding fume that contains manganese result in manganese
5 getting into the blood, I can answer that. The answer is
6 it can.

7 Q. How does it get into the blood?

8 A. It is believed that the fume is deposited in the
9 respiratory region of the lung and that some of it is
10 absorbed from the alveoli into the blood. It is not clear
11 to me exactly how that occurs, as to whether that
12 represents simple diffusion, meaning that there has been --
13 that the manganese that is present in the fume dissolves in
14 part; or whether that is mediated by macrophages and
15 enzymatic systems that break down the welding fume and
16 solubilize the manganese.

17 So, I don't know the mechanisms by which that occurs
18 though it is believed that manganese in welding fume can
19 get from the alveolar air into the blood by some series of
20 mechanisms, in part, not one for one; and it has to do with
21 many factors that your question hasn't specified and that
22 would need to be specified in order to get a meaningful
23 answer.

24 Q. You said the alveolar space?

25 A. Yes.

1 Q. What is that?

2 A. The air in the alveoli.

3 Q. What is the alveoli?

4 A. It's the basic unit of the gas transfer region of
5 the lung in which the transfer of oxygen from the air into
6 the blood and carbon dioxide from the blood back into the
7 air occurs.

8 Q. So, it's believed that it's that particular area
9 in the lung to where this manganese transport takes place?

10 A. Well, it's in the respiratory region of the lung
11 which includes all of the structures downstream from the
12 terminal bronchioles.

13 Q. Okay.

14 A. Including the alveoli.

15 Q. All right. So, the actual transport of manganese
16 from the lung into the bloodstream occurs at numerous areas
17 including the alveoli?

18 MR. BISSELL: Objection; form.

19 Are you including diffusion? I mean, he
20 said it could be diffusion or transport. You're not -- you
21 don't mean transport necessarily by any kind of mediation?

22 MR. LUBEL: Correct. Just transport.

23 A. Well, let's try and break the question down. Is
24 your question directed at understanding where in the lung
25 anatomically this occurs; or is it directed at

1 understanding how it occurs in the region in which it
2 occurs?

3 Q. (BY MR. LUBEL) Well, first I'm trying to find
4 out the region of the lung that this transfer of manganese
5 from the lung to the bloodstream takes place.

6 A. Again, I'll have to restate in all these
7 questions to the best of my knowledge no one has ever done
8 tracer studies that establish exactly how this occurs. The
9 answers I'm giving are based on fundamental concepts in
10 respiratory physiology which are believed to apply to
11 welding fume but which -- the extent to which they have
12 been tested, I'm not sure.

13 Q. Okay.

14 A. We think of the lung functionally as two
15 different regions. The airways are typically thought of as
16 the conducting region of lung, and they simply serve to
17 move air back and forth from the ambient atmosphere to the
18 gas transfer region of lung. The gas transfer region is
19 that region in which small molecules in air diffuse across
20 the alveolar surface through the interstitial space across
21 the endothelial surface and into the blood or back from the
22 blood to the alveoli.

23 And so, functionally the lung is broken into an area
24 where the gas just moves back and forth through the airways
25 and the region in which gas transfer occurs. It is

1 believed that there is no gas transfer in the conducting
2 airways. So, whatever reaches the conducting airways has
3 no relationship to things that are transferred from the air
4 into the blood. It has to get down to the gas transfer
5 region of the lung. That region of the lung includes a
6 number of structures.

7 It would probably be easier to draw it than to try to
8 describe it in words. I could draw it easily. It wouldn't
9 be hard to do --

10 Q. That's fine. You can draw it.

11 A. -- to draw it and then name the structures.

12 (Recess from 12:13 p.m. to 12:18 p.m.)

13 (Garabrant Exhibit No. 5 was marked)

14 Q. (BY MR. LUBEL) We've marked as Exhibit 5 your
15 drawing, correct, Doctor?

16 A. Yes.

17 Q. And could you describe for me where in your
18 drawing you believe that this manganese transport takes
19 place?

20 A. I didn't characterize it as manganese transport.

21 Q. Well, I'm just asking where in the drawing you
22 think it takes place?

23 MR. BISSELL: Objection; form.

24 A. In the respiratory region of the lung, which is
25 the region to the right of my vertical dotted line.

1 Q. (BY MR. LUBEL) Can you put an "X" or something
2 over there to designate that area?

3 A. (Witness complies)

4 MR. BISSELL: He's got it labeled.

5 A. Well, the respiratory region is the label on it
6 right here (indicating).

7 Q. (BY MR. LUBEL) So, you put a box around it?

8 A. Yes.

9 MR. BISSELL: Right there where it says
10 "respiratory region."

11 Q. (BY MR. LUBEL) Okay. So, what else have you
12 attempted to draw on Exhibit 5 for us?

13 A. Well, I tried to draw the conducting region
14 starting with the trachea, which splits to form the left
15 main stem bronchus and the right main stem bronchus and the
16 main stem bronchus which split to form the lobar bronchi
17 which split to form the segmental bronchi and so on and so
18 forth until you get down to about 16 divisions.

19 At about that point in the lung, then the little,
20 teeny airways begin to develop little outpouchings. Okay.
21 The last generation of airways before the outpouchings
22 occur is called the terminal bronchiole, and then there is
23 a transition in the function of the lung that it is no
24 longer conducting air back and forth. There is transfer of
25 gases from the alveoli into the blood and back.

1 Q. Is that where the transfer of manganese takes
2 place?

3 A. Hold on. Let me finish explaining.

4 And then I've drawn a blowup of this area of the
5 respiratory region showing the respiratory bronchiole,
6 which is a little airway that has little outpouchings; and
7 there is gas transfer in those outpouchings. And then
8 there is a gradual transition from what looks like an
9 airway into what looks like alveoli or just little --
10 almost like little clusters of grapes or little balloons.
11 This is the region in which gas transfer occurs. And so,
12 that's the respiratory region of the lungs.

13 Q. So, when we're looking at Exhibit 5, the bottom
14 part of this blowup where you've got a picture of the
15 alveoli -- you see that?

16 A. Yes.

17 Q. -- that's the respiratory region?

18 A. No, this whole thing is the respiratory region.

19 Q. Okay. I'll tell you what I'm going to do.

20 A. Right here (indicating).

21 Q. Can I borrow your pen? I'm going to put a --
22 actually I'm going to use this red pen. I'm going to put a
23 red circle. That includes the respiratory region, correct?

24 A. That is the respiratory region.

25 Q. Where the red circle is?

1 A. Yes.

2 Q. Is this where it's believed that the manganese
3 transport takes place between the lungs and the blood, I
4 think you called it?

5 MR. BISSELL: Objection; form.

6 A. That is the region where it is believed that
7 manganese that is inhaled would by some mechanism be
8 transported from the air space, which I've marked with a
9 red "X," into the blood.

10 Q. (BY MR. LUBEL) Now, how does it get from the
11 blood to the brain?

12 A. Well, I don't think that can be answered.
13 Clearly once it's dissolved in the blood, the blood
14 circulates throughout the body; but manganese is typically
15 bound to proteins that very effectively regulate its
16 concentration in blood. And I do not know how manganese
17 gets from the blood until it's bound to proteins into the
18 brain. It circulates through the blood supply and through
19 the capillaries, but I don't know how it actually gets out
20 of the capillaries and gets into the brain.

21 Q. Do the capillaries that have this blood in them
22 go to the brain?

23 A. No, the capillaries do not move. The blood
24 moves.

25 Q. But does the blood move into the brain is what

1 I'm asking.

2 A. Blood moves to every tissue in the body.

3 Q. Including the brain?

4 A. Yes.

5 Q. So, what you're saying you don't know is how it
6 moves from the blood inside the brain to the various
7 tissues in the brain, correct?

8 A. I don't know how it gets out of the blood and
9 into the brain.

10 Q. Has anybody studied that?

11 A. I am not familiar with that area of the
12 literature.

13 Q. What do you call that area?

14 A. An unfamiliar area.

15 Q. No, but I mean is it called toxicology; or what
16 do you call that area?

17 A. I don't know. It could be toxicology. It could
18 be toxicokinetics. It could be -- I'm not sure all the
19 areas that would include that type of research
20 investigation. It could well be toxicology.

21 Q. Okay. So, somehow the manganese makes its way
22 into the tissues of the brain, certain tissues; but nobody
23 knows how at this point in time?

24 A. First off, I think the way you've asked the
25 question presumes some things that aren't known to be

1 right. The manganese may or may not make it into the
2 brain. It doesn't always get into the brain just because
3 it's in the blood, and I do not know a mechanism by which
4 it moves into the brain when it does.

5 Q. But is it generally accepted that the manganese
6 can make its way from the blood to the brain?

7 A. Well, there's no general answer on that. There
8 are circumstances in which it may; and there are industries
9 in which people have been exposed to manganese such as in
10 manganese mining and/or refining, the ferro manganese
11 industry, dry cell battery manufacturing, in which they
12 have been found to have elevated levels of manganese in the
13 brain.

14 Q. How did it get --

15 A. It just got in there. We don't know. And then
16 there are other situations in which people have exposure to
17 manganese in which there was no evidence that they have any
18 increase in manganese in their brain. So, it's not -- you
19 can't assume it gets there. It may, or it may not.

20 Q. Can a welder from welding with
21 manganese-containing products have exposures to manganese
22 that go to the brain?

23 A. Well, when you are asking if it can, I suppose
24 it's theoretically possible. There are many sectors in
25 which people weld in which there is no evidence that they

1 have any increase of manganese in their brain.

2 Q. Under what circumstances can a welder's exposures
3 lead to manganese reaching the brain?

4 A. I don't know how to answer that. I am aware of
5 only two case reports in which it is believed that that
6 occurred.

7 Q. I'm just asking you if it can occur?

8 A. I am aware of two case reports in which it is
9 believed that that occurred. That's all -- that's all I
10 have seen.

11 Q. Do you agree --

12 A. In the world's literature.

13 Q. Do you agree?

14 A. Do I agree with what?

15 Q. That a welder exposed to manganese in the welding
16 process can have -- can be inhaled, enter the bloodstream,
17 and make its way to the brain?

18 A. It has only been documented in two instances that
19 I am aware of in which welders suffered from manganism.
20 Those case reports do not show or comment upon how the
21 manganese got into the welders' brains, but those two
22 individuals were believed to be suffering from manganism.

23 Q. I'm just asking you if you have an opinion as to
24 whether it can occur or not?

25 A. I believe those two case reports suggest that

1 there -- that it is possible that under some circumstances
2 manganese from welding can get into the brain, but they
3 don't say how.

4 Q. Do you believe it?

5 A. I believe what I just said: They suggest that it
6 is possible under some circumstances that manganese from
7 welding can get into the brain.

8 Q. So, you agree it can happen?

9 A. Under some circumstances I believe it is possible
10 it can happen.

11 Q. Under what circumstances?

12 A. Well, those two cases, to the best of my
13 knowledge, welded using high manganese content welding rods
14 in a confined space in which there was very little or no
15 ventilation; and it is believed that their exposures were
16 to welding fume -- to welding fume containing a high
17 manganese content -- were extraordinarily high.

18 Q. What were the levels?

19 A. I do not believe the levels were measured, or I'm
20 not aware that the levels were measured.

21 Q. What was the manganese content of the rods they
22 were using?

23 A. I do not know from memory. My recollection was
24 20 percent, but I'm not sure that's correct.

25 Q. What was the size of the confined space they were

1 working in?

2 A. I do not remember the dimensions. It was inside
3 a rock crusher. It was pretty small.

4 Q. And those two case reports the only reports that
5 you're aware of that discuss welders having overexposure to
6 manganese causing Parkinsonism?

7 A. The only ones from the United States that I've
8 ever seen.

9 Q. Are there any from other countries?

10 A. There are reports, case reports from other
11 countries that I cannot judge the reliability of.

12 Q. How is that? Why not?

13 A. Because I -- they do not give adequate
14 descriptions to know whether what they are reporting is a
15 reliable report or not.

16 Q. What were they missing that the U.S. case reports
17 had?

18 A. We would have to look at them one by one.

19 Q. Well, just from your recollection, what type of
20 information were they missing?

21 A. They varied. To the best of my recollection,
22 descriptions of exposure circumstances; descriptions of
23 tasks; descriptions of the type of welding materials, the
24 base metal, the welding rod; what the purpose of the
25 welding was; what type of welding it was; many factors that

1 would be -- well, let me keep going: Adequate descriptions
2 of what had happened to the people who were supposedly
3 affected, whether they really had neurologic disease or
4 whether they really had manganism or some other disease.

5 So, inadequate diagnostic information, inadequate
6 exposure information. They simply didn't give enough
7 details for me to judge that they were reliable reports.

8 Q. So, in essence you would have liked to have seen
9 more information on the exposure levels and the diagnostic
10 criteria used, correct?

11 A. More than that.

12 Q. What else?

13 A. Descriptions of the types of materials that were
14 being used in welding; what the manganese content of the
15 materials were; what type of welding; what the
16 circumstances of welding were, whether it was open-air
17 welding or confined-space welding; factors that would allow
18 me to make a better sense or to form a better sense for the
19 validity of what they were reporting.

20 Q. But those things you just referenced, whether it
21 was open air or not or what the content of the manganese
22 rods were, that information points you to an exposure
23 level, correct? That's what you want it for?

24 A. You want it for that plus more.

25 Q. What else do you want it for?

1 A. It's not possible to make any conclusions about
2 exposure levels simply by knowing some of the determinants
3 of exposure levels. There are many things that determine
4 exposure levels including the space in which welding is
5 being done; the ventilation in the space; the type of rod;
6 the type of base metal; the current flow; the type of
7 welding, whether it's TIG or MIG or stick welding; or
8 whether someone's using a cutting tool. So, there are lots
9 and lots of factors that are important to know about.

10 Q. Can't those be measured?

11 A. Yes, they can be measured.

12 Q. Aren't there devices that can measure the
13 exposure level, take in account all your factors and
14 actually give you what the welder was exposed to?

15 A. Well, there's a "yes" part of the answer; and
16 that's a "no" part of the answer. It's my understanding
17 that not all welding fumes are identical. And so, while
18 you might measure the total mass of welding fume per cubic
19 meter of air, you could end up with a very different
20 meaning from two different samples that represented the
21 same concentration in the air.

22 In other words, what's not clear is the extent to
23 which the metals in the welding fume are biologically
24 available, whether they're soluble or insoluble and how
25 rapidly they are cleared versus how rapidly they're

1 solubilized.

2 Q. Have the companies not tested that that make
3 these welding rods?

4 A. I do not know. That's not an area I've prepared
5 for today.

6 Q. Have you asked any of these lawyers that hired
7 you to provide you with that information?

8 A. That's not an area of my expertise.

9 Q. So, it wouldn't make any difference if you knew
10 it, correct?

11 A. Well, I prepared to talk about the epidemiology
12 today. You're asking me questions about welding exposures
13 and the physiology of manganese absorption and transport,
14 which are not areas that I prepared on. So, you're going
15 off into areas I didn't prepare on.

16 Q. In other words, you don't hold yourself out as an
17 expert in those areas, correct?

18 A. That is correct.

19 Q. All right. What's the diagnostic criteria for
20 manganism?

21 A. I'm not aware of any written diagnostic criteria.
22 There is a clinical syndrome that is often seen and which
23 is required in order to reach that diagnosis.

24 Q. What are the features of that syndrome?

25 A. Let's look at the exhibit, this one (indicating).

1 Q. Which number is that, Doctor?

2 A. No. 4.

3 "An akinetic-rigid Parkinsonian syndrome predominates,
4 although many individuals with chronic manganese
5 intoxication" have a program of psychiatric symptoms
6 "before they develop their movement disorder."

7 Q. Can you go slower? What was the first one you
8 mentioned, akinetic?

9 A. Akinetic-rigid Parkinsonian syndrome.

10 Q. Okay. Go ahead.

11 A. The psychiatric symptoms may include
12 "irritability, emotional lability, illusions, and
13 hallucinations. These symptoms have been documented mainly
14 in manganese miners." In other words, they haven't been
15 seen in welders.

16 "The movement disorder is dominated by bradykinesia
17 and rigidity with little resting tremor. Axial and
18 extremity dystonia are present often." Postural tremor may
19 be present although resting tremor is absent. The
20 characteristic gait, which is called the "cock-walk," is
21 sometimes seen. Dementia may be present.

22 I think that describes it.

23 Q. How many of those must a person have for their
24 Parkinsonism to be called manganism?

25 A. I don't think that there's any checklist that

1 would allow one to say you can simply count them up and
2 satisfy it. It's a clinical diagnosis, and in many ways
3 it's -- the diagnosis is reached in a manner similar to
4 reaching a diagnosis of Parkinson's disease where one looks
5 for a typical clinical presentation. One does diagnostic
6 testing to rule out other etiologies, and often one looks
7 for evidence of a hyperintense signal from the globus
8 pallidus on imaging studies that he believes to reflect an
9 accumulation of manganese in the globus pallidus.

10 Q. All right. Let's do it this way: Can a person
11 have manganism without the characteristic feature of the
12 cock-walk?

13 A. I believe they can.

14 Q. Can a person have manganism without dystonia?

15 A. I don't think so.

16 Q. Okay. How about can a person have manganism with
17 a rest tremor?

18 A. My belief is that would be quite unusual, and
19 that would argue against it.

20 Q. So, if somebody has a rest tremor, it's your
21 opinion that that would point toward something other than
22 manganism?

23 A. I believe so.

24 Q. But you would acknowledge that a person could
25 have a rest tremor and still have manganism, correct?

1 A. Well, people can have two disorders on top of
2 each other. That happens, too; but the resting tremor is
3 not characteristic.

4 Q. Is any tremor characteristic of manganism?

5 A. Postural tremor.

6 Q. What is that?

7 A. Postural tremor is the tremor that you get when
8 you try to hold a specific posture. Neurologists typically
9 think of three types of tremor: Movement tremor, postural
10 tremor. And resting tremor.

11 Q. So, give me an example of postural tremor.

12 A. (Indicating)

13 Q. Your hands can shake?

14 A. No, I'm trying to hold a posture.

15 Q. Oh.

16 A. I'm holding a posture. I shake while I'm
17 holding. That's different than a resting tremor where the
18 muscles are at rest and I'm shaking.

19 Q. Okay. What is an intention tremor?

20 A. (Indicating) When I'm trying to put my finger on
21 my nose and back to my finger, and that's my intention.
22 And as I try to complete that, I shake. So, we think of
23 three different kinds: Resting, postural, and intention.

24 Q. Is an intention tremor consistent with manganism?

25 A. I've never seen that. I believe manganism is

1 characterized by postural tremor.

2 Q. All right. What's rigidity in layman's terms?

3 A. I'm not sure there is a layman's term for
4 rigidity other than rigidity.

5 Q. How does a doctor recognize rigidity?

6 A. By trying to flex and move the patient's
7 extremities.

8 Q. Are they tight?

9 A. They resist.

10 Q. That's a judgment call the doctor makes, right?

11 A. Yes, based on experience.

12 Q. So, a person with rigidity, that particular
13 trait, would be consistent with manganism?

14 A. I believe so.

15 Q. But not necessarily required?

16 A. Well, not diagnostic of.

17 Q. No, but I mean a person can have manganism
18 without rigidity, true?

19 A. No, I believe manganism is dominated by
20 bradykinesia and rigidity with little resting tremors.

21 Q. What is bradykinesia?

22 A. Slow movement.

23 Q. And you've given us a list of psychiatric
24 symptoms. Are those necessary for a diagnosis of
25 manganism?

1 A. No.

2 Q. Are they consistent with a diagnosis of
3 manganism?

4 A. I believe they've only been seen in one setting,
5 and that is in manganese miners. So, they've been seen.
6 It's been described. It's my belief that in other
7 settings, they have not been seen.

8 Q. Do you know whether depression is characteristic
9 of manganism?

10 A. I don't believe it's characteristic of manganism.

11 Q. Can it be caused by manganism?

12 A. I believe it is sometimes seen in the setting of
13 manganism.

14 Q. Akinetic-rigid Parkinsonian syndrome, is that
15 different than bradykinesia and rigidity?

16 A. It's quite similar.

17 Q. How would you characterize it or define it?

18 A. Akinesia is absence of movement. Bradykinesia is
19 slowed movement.

20 Q. Can you have both at the same time?

21 A. Well, I think they reflect just different degrees
22 of the same phenomenon.

23 Q. You were looking at Exhibit 4 to give us the
24 signs and symptoms, correct?

25 A. Yes.

1 Q. Does it mention anything in there about a mask
2 like face?

3 A. I don't see that it does.

4 Q. Do you know whether that can be kind of a unique
5 feature of manganism?

6 A. I'm not aware that it is.

7 Q. All right. Now, have you seen references in the
8 literature to the term chronic manganese poisoning?

9 A. I don't recall whether I've seen those exact
10 words used.

11 Q. Do you know if chronic manganese poisoning is the
12 same thing as manganism or if it's different?

13 A. Again, you'd have to look at who's using those
14 terms to see whether that was their intention.

15 Q. Have you seen in the literature any reference to
16 the term manganese induced Parkinsonism?

17 A. I have seen that.

18 Q. Is that the same thing as manganism or different?

19 A. I believe that would be synonymous with
20 manganism.

21 Q. Have you seen something similar to manganese
22 induced Parkinsonian syndrome? Have you seen that general
23 characterization?

24 A. I don't recall that general characterization.
25 We'd have to look at those references if there is such a

1 thing.

2 Q. If there has been such a thing, do you have any
3 idea as to whether that would be synonymous with manganism?

4 A. You mean if it exists, would I know what it was?

5 Q. Right.

6 A. I'd have to see it. I mean, it's an unusual
7 usage. I don't know what the authors who hypothetically
8 might have written that hypothetically might have meant.

9 Q. Can Parkinsonism be synonymous with manganism?

10 A. I don't believe so.

11 Q. What's the difference between the two?

12 A. Well, Parkinsonism is a broad term that includes
13 many different movement disorders that are due to many
14 different causes. Manganism is a specific Parkinsonism
15 that is due to manganese intoxication.

16 Q. But if the treating doctor is not aware of
17 manganese intoxication, would you expect that doctor to
18 diagnose that patient with Parkinsonism as opposed to
19 manganism?

20 A. It would depend on that doctor's clinical skill.

21 Q. Well, if the doctor's not familiar with manganese
22 causing manganism -- you with me so far? You understand
23 what --

24 A. In other words, this is a doctor who's never
25 heard of manganism and knows nothing about it? That's

1 your --

2 Q. Correct.

3 A. -- question?

4 Q. Yes, sir.

5 -- and the patient exhibits signs and symptoms that
6 are consistent with Parkinsonism and manganism, what do you
7 expect that doctor is going to diagnose it as?

8 A. It depends on that doctor's diagnostic ability.
9 It's a common witticism that you can only diagnose diseases
10 that you're familiar with. And so, if you don't know about
11 a disease, you'll never diagnose it; and you will
12 misdiagnose the person as having something else.

13 Q. All right.

14 A. So, as to answer your question as to what a
15 hypothetical doctor might misdiagnose manganism as, it
16 depends on what diseases that doctor is familiar with and
17 what ones he's never seen or she's never seen. Could
18 misdiagnose it as lots of things, and that happens commonly
19 in medicine.

20 Q. Well, given the overlap in signs and symptoms
21 between Parkinsonism and manganism, wouldn't you think that
22 that doctor that's not familiar with manganism would
23 diagnose that patient with Parkinsonism?

24 A. It depends on that doctor's skill and experience.
25 If that doctor is a gynecologist, it is very unlikely that

1 that doctor would reach a correct diagnosis because it's a
2 realm of disease that is way out of that doctor's area. If
3 that doctor is a neurologist, I would expect a qualified
4 neurologist to be aware of manganism and to be able to
5 correctly diagnose it. Diagnosis of manganism requires
6 overexposure to manganese, and I would expect a good
7 neurologist to elicit a history of manganese exposure.

8 Q. You think that's what neurologists do?

9 A. A good neurologist seeing a movement disorder
10 that is potentially manganism should ask about a history of
11 manganese exposure.

12 Q. "Should have, could have, would have." I'm
13 asking you if you think that's what routinely happens in
14 neurology clinics around the United States. They ask about
15 the exposure to the substances they work around?

16 A. I can't answer what happens routinely in
17 neurology clinics around the United States.

18 Q. Would it fall below the standard of care for a
19 physician not to ask about the patient's occupational
20 exposure?

21 A. What standard of care are you referring to?

22 Q. Standard of care applicable to physicians.

23 A. It would depend on the circumstances.

24 Q. Under what circumstances would it fall below the
25 standard of care?

1 MR. BISSELL: Object to form.

2 A. Could you rephrase the question so I'm sure I
3 understand what it is?

4 Q. (BY MR. LUBEL) If a medical doctor is confronted
5 with a patient that has signs and symptoms that you and I
6 know could be consistent with Parkinsonism or manganism and
7 that doctor does not inquire or ask questions about the
8 patient's occupational exposure history, would that doctor
9 be falling below the standard of care?

10 A. Depends what the standard of care is in that
11 doctor's specialty.

12 Q. Let's talk about neurology.

13 A. I don't know the standard of care in neurology.
14 I'm not a neurologist.

15 Q. How about internal medicine?

16 A. In internal medicine I think the standard of care
17 would be to recognize that there was a movement disorder
18 and to make a referral to an appropriate movement disorder
19 specialist for a proper evaluation.

20 Q. So, you wouldn't expect the internal medicine
21 doctor to ask questions about occupational exposure
22 history, correct?

23 A. An internist might well ask them. I don't
24 believe that that would be a requirement under the standard
25 of proper care in the field of internal medicine. I think

1 standard of care would be to recognize the presence of a
2 movement disorder and to make an appropriate referral.

3 Q. But you would agree that the standard of care
4 that would be applicable to a neurologist would include
5 going into the patient's occupational medical history,
6 correct?

7 A. I have no knowledge of the standard of care in
8 the field of neurology. We're really far afield from my
9 area of expertise here.

10 Q. You're familiar with the standard of care in your
11 area of internal medicine, though, correct?

12 A. To some extent.

13 MR. LUBEL: Take a short break?

14 MR. BISSELL: Yeah.

15 (Discussion was held off the record)

16 (Recess from 12:56 p.m. to 2:05 p.m.)

17 Q. (BY MR. LUBEL) Doctor, are there ways in which
18 blood is transported to all parts of the human brain?

19 A. Yes.

20 Q. What do you call that system?

21 A. Circulation.

22 Q. The circulatory system?

23 A. Yeah.

24 Q. And I think you said earlier, if I heard you
25 correctly, that the blood reaches all tissues in the body

1 or something to that effect?

2 A. Yes.

3 Q. What happens if blood doesn't reach the tissues?

4 A. It dies.

5 Q. Now, in a patient that has manganese induced
6 Parkinsonism, is there cell death somewhere in the brain?

7 A. I believe there is.

8 Q. Do we know where it is?

9 A. In the globus pallidus.

10 Q. And do we know how much cell death is necessary
11 to turn it from a patient that's doing okay to somebody
12 that's got manganese induced Parkinsonism?

13 A. Well, we know it has to be enough that the globus
14 pallidus does not take up adequate amounts of dopamine to
15 do its function; and that's reflected in clinical illness.

16 Q. But has there been any characterization in the
17 literature as to, for instance, how many cells must be
18 destroyed or die in order for it not to be able to take up
19 the dopamine necessary for it to function properly?

20 A. I don't know whether there have been such
21 studies.

22 Q. Do you have an opinion as to how one would
23 characterize the number of cells that would have to be
24 adversely affected or killed in order for there to be
25 manganese induced Parkinsonism?

1 A. That's not an area that I've studied, and I don't
2 have an opinion on that.

3 Q. Do you know from the literature what level of
4 exposures are necessary for a person to have a manganese
5 induced Parkinsonism?

6 A. That's been characterized in some industries, and
7 we could look at that. There is some literature on that
8 point, but it's important to recognize that there may be
9 substantial differences in those levels across industries
10 depending on the characteristics of the type of manganese
11 compound at issue.

12 Q. For instance, what are the industries in which
13 the literature reports any connection to manganese causing
14 these types of adverse health effects, Parkinson's disease?

15 A. I don't know of any connection between
16 Parkinson's disease and --

17 Q. Parkinsonian's syndrome. Sorry. Parkinsonian's
18 syndrome.

19 A. Parkinsonism?

20 Q. For instance.

21 A. Battery industry based on manganese oxide in
22 batteries, dry cell batteries, in Taiwan. There's a good
23 characterization there.

24 Q. Anything else?

25 A. I believe there's been characterization in the

1 manganese mining industry.

2 Q. Any other industries?

3 A. I don't recall. There may be. I don't know.

4 Q. How about the steel industry?

5 A. I don't recall -- well, first off, in the steel
6 industry I'm not aware that manganism has ever been
7 reported. If you're talking about the ferro manganese
8 steel industry, it's a special type of steel. There has
9 been manganism reported in that industry. I don't recall
10 if they have characterized the exposure levels adequate to
11 cause manganism.

12 Q. How about foundries?

13 A. I don't -- I'm not aware of manganism being an
14 issue in foundries in general. Are you talking about some
15 particular types of foundries?

16 Q. Have you seen in it any foundries?

17 A. I'd have to look at the literature. That's not
18 the way I'd characterize the studies I've seen.

19 Q. Okay. So, at least as you sit here, you can
20 recall manganese exposures causing a particular form of
21 Parkinsonism called manganism, right?

22 A. In two industries we've identified, the manganese
23 mining and in dry cell battery manufacture.

24 Q. Not the ferro manganese steel industry? I
25 thought you mentioned that, too.

1 A. Right. Yes, I did mention that. I didn't recall
2 whether there were specific exposure levels mentioned in
3 those studies.

4 Q. All right. Now, what was the form of manganese
5 in the mining studies?

6 A. I'd have to go back and look. It was manganese
7 ore, and I believe the studies also involved refining of
8 the ore. So, I'd have to go back and look at them.

9 Q. Do you have those studies with you?

10 A. I think they're in the books I have, yes.

11 Q. Great. We need to find them.

12 (Recess from 2:14 p.m. to 2:38 p.m.)

13 Q. (BY MR. LUBEL) You can proceed with your answer.

14 A. What was the question?

15 (Requested question was read back)

16 A. In the Moroccan mines it was pyrolusite ore that
17 was rich in manganese.

18 Q. (BY MR. LUBEL) That was rich in manganese?

19 A. (Nods head affirmatively)

20 Q. Is that a "yes"?

21 A. Yes.

22 Q. If we looked at the body of mining literature on
23 manganese intoxication, is it going to be the same form of
24 manganese; or do you know?

25 A. I don't know offhand. We'd have to look at the

1 body of literature.

2 Q. I thought that was what you were doing earlier.

3 A. Well, I don't have the whole body of literature
4 on that. I don't have every study there is.

5 Q. Well, let me ask it this way: Do you know if
6 there is a difference between the manganese in the miners
7 that causes the intoxication versus the manganese in the
8 battery industry that causes intoxication?

9 A. It's likely there is because in the battery
10 industry I believe it's manganese dioxide.

11 Q. And you think in the mining industry it's not
12 manganese dioxide?

13 A. That's correct.

14 Q. Okay. How about in the ferro manganese steel
15 industry? What form of manganese?

16 A. Ferro manganese steel is a manganese/iron alloy.
17 I believe in that instance it is the manganese fume from
18 the steelmaking.

19 Q. And what's the form of manganese in the fume from
20 steelmaking?

21 A. I'm not sure. It could be pure manganese.

22 Q. But you're not sure?

23 A. I'd have to look at those studies.

24 Q. Have you done any analysis of all the literature
25 involving these different industries to assess whether or

1 not the manganese is in a different form?

2 A. No.

3 Q. Is that typically left with somebody with
4 expertise in industrial hygiene?

5 A. There are people in industrial hygiene who would
6 be qualified to do that.

7 Q. I mean, typically in cases where that's an issue,
8 do you rely upon an industrial hygienist to get you an
9 answer to whether the, you know, form of whatever the
10 product is is different in different studies?

11 A. It would depend on the specifics of the
12 literature. In some instances I could do that myself, and
13 in some instances I would need the expertise of an
14 industrial hygienist. In other instances I would need the
15 expertise of a metals chemist who could sort out what the
16 chemical form of the exposure was.

17 Q. And in this instance, these cases, the welding
18 cases, you've done none of those, correct?

19 A. I'm not sure I understand the question. Which
20 instance are you talking about?

21 Q. In the welding lawsuits that you're involved in,
22 you've not sought any -- the industrial hygienist, the
23 chemist with expertise in metal, you've not sought their
24 help in analyzing the form of manganese?

25 A. I haven't needed to. What I've looked for is

1 evidence of whether welding as it is commonly done is
2 associated with Parkinsonism and isn't regardless of the
3 form. So, if there were studies suggesting that
4 Parkinsonism or Parkinson's disease was associated with
5 welding, then it would be important to go back and figure
6 out under what form it was and how did that happen; but we
7 don't see it in any of the studies.

8 And moreover, there is some information on the
9 manganese content of welding fume from typical welding rods
10 that are used in mild steel welding showing that it's in
11 the range of 4 to 6 percent. So, even without knowing the
12 form, we know that it is a low content; or it's a low
13 percentage of what's present in welding fume.

14 Q. Of the total fume?

15 A. Of what?

16 Q. Of the total fume?

17 A. No, of the respirable fume.

18 Q. What do you mean by respirable?

19 A. That fraction of the particulates that have an
20 aerodynamic diameter of less than ten microns.

21 Q. What does less than ten microns look like? Can
22 we see that with the naked eye?

23 A. No.

24 Q. You have to use like a microscope for that?

25 A. Yes.

1 Q. We're talking about something that's invisible to
2 the naked eye, correct?

3 A. You can't see the individual particles. You
4 could see evidence of fuming in the air. I don't
5 understand the question the way you've asked it.

6 Q. The particle that you're saying is respirable,
7 you can't see with the naked eye; can you?

8 A. You can't see an individual particle, no; but you
9 can see evidence of fume in the air when the fume is made
10 up of particles that are respirable.

11 Q. You can?

12 A. Yes, you can.

13 Q. Have you seen it before?

14 A. Yes.

15 Q. When?

16 A. I've seen welding done.

17 Q. Have you welded?

18 A. I have.

19 Q. Did you see the fume?

20 A. I did.

21 Q. Could you smell it when you welded?

22 A. I don't recall smelling it. I have been in
23 welding environments where I could smell it.

24 Q. Were you exposed to it when you smelled it?

25 A. Why, yes.

1 Q. Have you ever tasted it before?

2 A. I can't say as I've ever tasted it, no.

3 Q. So, you don't know what it tastes like?

4 A. Well, I've been in many settings in which welding
5 was being done right in front of me. I've never tasted it.
6 So, I'd say yeah, it has no taste.

7 Q. So, when you're looking at this fume -- you say
8 you can see if you're welding or if you've seen welding
9 done -- how much of that visible fume is made up of these
10 microscopic respirable particles that are invisible to the
11 naked eye?

12 A. Let's clarify the question a little bit. I don't
13 understand it the way you've asked the question. I don't
14 understand what you mean by fume made up of microscopic
15 particles that are invisible to the naked eye.

16 Q. You want me to ask it a different way?

17 A. Well, I'm not sure what you're actually asking.
18 The fume is visible even though the individual particles in
19 the fume are too small to be seen individually.

20 Q. And I'm asking you what percentage of the fume is
21 made up of the invisible particles, the less than ten
22 micron sized particles?

23 A. The majority of them, a high percentage.

24 Q. What does that mean, 50 percent, 70 percent?

25 A. Oh, for welding fume I don't know -- certainly

1 more than half, could be as high as 90 percent; and to some
2 extent it would vary depending on the circumstances, the
3 welding, the welding process; presence of contaminants.
4 Much of the fume is respirable; but welding can also --
5 depending on what you're doing can also generate some
6 larger sized particulates in addition.

7 Q. Where does these microscopic particles go when
8 you're welding? They just float off in the air?

9 A. Yes.

10 Q. And where do they go?

11 A. They stay suspended in the air and eventually
12 settle out.

13 Q. Settle on the ground?

14 A. Settle out on whatever horizontal surface there
15 is.

16 Q. And then is there anything that can cause those
17 microscopic particles that are settled on a horizontal
18 surface to get back in the air and float around?

19 A. Well, if you did something to stir up whatever
20 dust was on the horizontal surfaces, you could re-entrain
21 them into the air.

22 Q. And wouldn't that possibly expose the workers in
23 that room again?

24 A. Well, it's a hypothetical. I can't answer that
25 unless you come up with some concrete scenario.

1 Theoretically if you stir up dust, you re-entrain it in the
2 air. It's back in the air. If you breathe the air, you
3 could be breathing the air in your lungs.

4 Q. Now, is this --

5 A. That's a theoretical. That could happen, but the
6 amount of that is going to be dependent upon what you're
7 doing.

8 Q. And you call it re-entrainment?

9 A. Yes.

10 Q. That means stirring it up, in layman's terms?

11 A. Re-entrainment means putting it back into the
12 air.

13 Q. Now, when it's put back into the air, is there
14 going to be a visible fume of these microscopic particles;
15 or is it going to be invisible?

16 A. I need you to clarify something: Are you talking
17 about the individual particles being too small to be seen
18 by the naked eye when you mean invisible, or are you
19 talking about the fume not being visible?

20 Q. I'm talking about the individual particles being
21 invisible to the naked eye, not invisible like they don't
22 exist but like you have to have a microscope to see them,
23 those particles.

24 A. To see the individual particles?

25 Q. Right.

1 A. Okay. And what was the question? Are they still
2 invisible when they're re-entrained?

3 Q. Yeah, when they settle along like a workbench,
4 like a horizontal surface -- you understand that to be like
5 on a workbench?

6 A. Yes.

7 Q. -- and then you come back the next day and for
8 whatever reason they get stirred up, you expect the
9 worker's going to see some fume that these particles
10 consist of?

11 A. Well, it's a complex answer. A number of things
12 could happen. First off, when you create fresh welding
13 fume, the majority of the particles are respirable, meaning
14 less than ten microns in size. Many of the particles are
15 charged, and they tend to aggregate real quickly. And so,
16 they aggregate into larger particles, which settle out
17 faster.

18 So, fresh fume is different than old fume in that it
19 has undergone some changes. Once it has settled on a
20 surface, it may lose its charge. It may absorb water -- it
21 may absorb water vapor in the air if you're welding in a
22 humid climate. There may be some chemical reactions on the
23 surface, and there is this aggregation of small particles
24 into larger particles. So, what you have settled on a
25 surface once it's aged is not the same as fresh fume that's

1 in the air.

2 Q. It's safe, is what you're saying?

3 A. No, I didn't say that. I said it's not the same.

4 Q. Okay. Let's go through a few things. I'm going
5 to mark some exhibits.

6 (Garabrant Exhibit No. 6 was marked)

7 Q. (BY MR. LUBEL) Exhibit 6 is your 1979 version of
8 the effects of welding on health, and then you've got one
9 page marked; is that true? I'm not suggesting you don't
10 have highlights on other pages, but you've got one page
11 that's flagged.

12 A. I have a tag on Page 29.

13 Q. Okay.

14 A. And that tag's a section called manganese
15 intoxication, which is on Pages 29 and 30.

16 Q. Did you use anything else in that book other than
17 those two or three pages you referenced? I've given you a
18 question that takes you to another page. I know that can
19 happen. I'm just saying as you sit here today, do those
20 appear to be the pages that you're relying upon for some
21 opinion in this case?

22 A. Well, there are other pages as well. I tagged
23 those pages because they gave a summary of manganese
24 intoxication, but there are other pages in addition to that
25 that are important to me.

1 Q. Is that American Welding Society publication
2 authoritative in your opinion?

3 A. Well, it contains some things that I think are
4 accurate. I don't vouch for everything in it.

5 Q. So, is it authoritative or not?

6 A. Well --

7 MR. BISSELL: Object to form.

8 A. I don't know what you mean by "authoritative."

9 Q. (BY MR. LUBEL) Is it something you relied upon
10 for your opinions?

11 MR. BISSELL: Object to form.

12 A. It --

13 Q. (BY MR. LUBEL) I mean, you brought it. I didn't
14 bring it.

15 MR. BISSELL: Object to argument.

16 MR. LUBEL: Oh, okay.

17 Q. (BY MR. LUBEL) This is yours, isn't it, Doctor?

18 A. It is mine and I brought it and it is a document
19 that was published approximately 27 years ago and it gives
20 a concise summary of what was known about manganese
21 intoxication 27 years ago. It does not provide much
22 information on the issue of welding and Parkinson's disease
23 or welding and Parkinsonism.

24 Q. It doesn't?

25 A. It doesn't.

1 Q. Well, this was put out by the American Welding
2 Society; wasn't it?

3 A. Yes, 27 years ago.

4 Q. Okay. So, what did they do between 1979 and, for
5 instance, the year 2000 in the way of studies that would
6 illuminate this issue of welding and Parkinsonism?

7 A. There are many studies that have been published
8 since then.

9 Q. By them?

10 A. Who is "them"?

11 Q. The American Welding Society.

12 A. I don't know that the American Welding Society
13 has published any of them. The body of scientists around
14 the world have published independently in most instances of
15 the welding industry and the American Welding Society.

16 Q. What studies did the American Welding Society
17 commission since 1979 and between the years 1979 to 2000 on
18 the potential neurological effects associated with welding
19 fumes?

20 A. I don't know.

21 Q. You haven't seen that in your review of all the
22 literature?

23 A. I haven't seen what?

24 Q. Anything that the welding society did?

25 A. I haven't looked for such literature nor have I

1 kept it catalogued in that manner.

2 Q. When they hired you -- I take it you understand
3 you're not here just on behalf of one company? You're here
4 for a consortium of companies?

5 MR. BISSELL: Objection; form. That's
6 absolutely not correct. The only consortium I know of is a
7 different group, not a consortium.

8 Q. (BY MR. LUBEL) Who are you here for?

9 A. I thought I was here on behalf of Lincoln
10 Electric.

11 Q. Okay. So, you're not here as an expert for
12 Hobart or Miller Electric or any of those other welding
13 companies?

14 MR. BISSELL: Yes, you are.

15 A. For Hobart, I think so. I don't know about
16 Miller.

17 Q. (BY MR. LUBEL) In general were you hired by
18 lawyers that you understood to represent this group of
19 welding defendants in a lawsuit?

20 A. I don't know what you are referring to when you
21 say "this group of welding defendants."

22 Q. Well, what companies are you working for other
23 than Lincoln Electric in the lawsuits, not just this
24 lawsuit but just the lawsuits?

25 A. I'm not sure I know all of them. I think I have

1 worked on behalf of Lincoln Electric and Hobart. I think
2 in one instance perhaps General Electric.

3 Q. ESAB?

4 A. I don't know.

5 MR. BISSELL: Yes.

6 A. Yes.

7 Q. (BY MR. LUBEL) Union Carbide Corporation?

8 MR. BISSELL: Yes.

9 A. Yes.

10 Q. (BY MR. LUBEL) Miller Electric Manufacturing
11 Company. You don't know?

12 A. I don't know.

13 Q. ITW?

14 A. I'm not aware of it.

15 Q. Illinois Tool Works?

16 A. I'm not aware of it.

17 MR. BISSELL: They're a parent of Hobart.
18 They're affiliated with Hobart.

19 He is working for in this case the ones that
20 I represent.

21 Q. (BY MR. LUBEL) All right. When you got first
22 hired in the lawsuits, did you ask anybody "Who are the
23 companies that I'm going to be working for"?

24 A. I think I was made aware of that in the first
25 case.

1 Q. And there's been a bunch of companies, correct?

2 A. I believe I have worked on behalf of a number of
3 companies that manufacture welding consumables.

4 Q. And when you got hired by these people, did you
5 ever ask them or their lawyers, "Hey, have y'all got any
6 studies on the potential neurologic effects from welding"?

7 A. I think I have discussed with the lawyers on
8 numerous occasions all of the scientific literature
9 regardless of who wrote it; and to the best of my
10 knowledge, I have received whatever studies those attorneys
11 were aware of.

12 Q. Okay. So, what I'm asking you is: For
13 approximately 20 years after this 1979 version of effects
14 on welding health, what did these welding companies do to
15 try and come up with answers to questions posed by welding
16 and potential neurological deficits?

17 A. Well, I think it would be fair to say that there
18 was no evidence or reason for concern about Parkinsonism
19 related to welding in the scientific literature adequate to
20 have warranted that any studies be done. Every existing
21 study had failed to show evidence of any problems, any
22 neurologic problems whatsoever related to welding.

23 Q. So, the answer is they didn't do any studies in
24 that 20 years, correct?

25 MR. BISSELL: Objection; form.

1 A. I think I answered the question as you asked it.
2 There was no reason to think that any study ought to be
3 done. There was no suspicion of the problem.

4 Q. (BY MR. LUBEL) So, you hadn't seen any studies
5 in that 20-year period; have you?

6 A. I've seen many studies.

7 Q. From the welding companies?

8 A. No, many studies in the scientific literature.

9 Q. No, man. I'm talking about the welding company
10 studies. Have you seen any of those between 1979 and 1999?

11 A. There was no reason to have done any. There was
12 no suggestion of an issue that needed to be studied.

13 Q. I understand that's what you say. I'm just
14 asking if you know of any. I understand that you say they
15 didn't need to do them. I'm just saying did they do any?

16 A. You're asking me did they need to do something --

17 Q. No.

18 A. -- did they need to do it?

19 Q. No, I said, "Did they do any?" I didn't ask you
20 if they needed to. Did they do any?

21 A. Well, I think the issue is did they need to; and
22 the answer is no, they didn't.

23 Q. No. You don't get to decide the issues. My
24 question is --

25 A. I mean --

1 Q. -- they --

2 A. -- as a scientist --

3 THE REPORTER: Wait, wait --

4 A. -- I do have opinions. That's what I spend my
5 career doing.

6 Q. (BY MR. LUBEL) You've said it. My question is:
7 Did they do any?

8 A. I'm not aware that they did any, which I regard
9 as an appropriate response to the lack of evidence of a
10 health concern.

11 Q. Did they ever do any?

12 A. Well, I know that they did studies to
13 characterize the manganese content of welding fume and
14 found that it was low, from which it was a reasonable
15 conclusion that welders who worked with mild steel would
16 not have been overexposed to manganese.

17 Q. Is that the only study they ever took part in?

18 A. It's the only one I recall as I sit here. To
19 answer your question, there may have been others.

20 Q. How about Fryzek? How about Fryzek? Didn't they
21 participate in some study, some guy you know from Michigan?

22 A. You asked me about studies prior to 2000 --

23 Q. And then I said open it up to ever.

24 A. Well, then I misunderstood your question. I'm
25 sorry.

1 Q. Then I'm asking you: Did they ever commission
2 any studies?

3 A. Okay. Could you restate the question? Is it did
4 "they" ever commission any studies --

5 Q. Right.

6 A. -- who is "they" and any studies -- any domain
7 for studies or just any studies of anything? It's a vague
8 question.

9 Q. Welding defendants for Parkinson's like disease.

10 A. I'm having difficulty understanding your
11 questions because they are so vague.

12 Q. Let me ask --

13 A. Did they do any studies? You're going to have to
14 tell who they are and what realm of science we're talking
15 about because I clearly misunderstood your previous
16 question. I thought we were talking prior to 2000, and
17 suddenly it's changed.

18 Q. Let me ask it this way: Are you aware of any
19 welding company funding in whole or in part any study to
20 answer questions posed by any neurological deficits that
21 may or may not be related to welding?

22 A. I do not understand the question.

23 Q. All right. Are you aware of any welding company
24 or the American Welding Society or other industry group
25 assisting in any studies that have been published on

1 welding and neurological issues ever?

2 A. Yes. Yes.

3 Q. What were the studies called?

4 A. What were they called?

5 Q. Right. How would you identify them?

6 A. Well, I can think of three off the top of my
7 head: A study conducted by Dr. John Fryzek; a study
8 conducted by Dr. Michael Fored, a study conducted by
9 Dr. Gary Marsh.

10 Q. Any others?

11 A. I'd have to look through my references. There
12 may well be.

13 Q. See if you can find them, please. Take your
14 time.

15 A. May I ask for clarification on what you mean by
16 welding companies, trade associations, or other industry
17 groups in your question? It's a little vague.

18 Q. You don't know who the welding company is?
19 You're working for them. Is that the part you don't
20 understand?

21 A. No. It's the trade organization or other
22 industry groups that's a little hard for me to understand
23 what you're getting at.

24 Q. Well, the American Welding Society would be an
25 example of an industry group or trade organization, just to

1 give you an example.

2 MR. BISSELL: Object to form. I'll accept
3 your identification if that is what you're asking him
4 about --

5 MR. LUBEL: He --

6 MR. BISSELL: That's not what that
7 organization is.

8 MR. LUBEL: Okay. Anyway --

9 MR. BISSELL: But you've used an example.
10 That's fine. I don't have a problem with it.

11 A. Would your question include, for example, trade
12 organizations in the steel industry or the mining industry
13 or the ferro alloy industry or the dry cell battery
14 industry?

15 Q. (BY MR. LUBEL) We're talking about welding
16 industries right now. So, if in the battery industry you
17 see a reference to welding, the answer is yes. If in the
18 ferro manganese steel industry there's references to
19 welding, yes.

20 A. Okay. Well, I just found another one, the study
21 by Clewell in 2003.

22 Q. How do you spell Clewell?

23 A. C-L-E-W-E-L-L.

24 Q. Okay. Any others?

25 A. Well, I got to keep looking.

1 Q. Yes, sir.

2 A. It's going to take awhile.

3 (Recess from 3:08 p.m. to 3:44 p.m.)

4 (Garabrant Exhibit No. 7 was marked)

5 Q. (BY MR. LUBEL) Exhibit 7 is the list of studies
6 that you could find with your proviso that you're not sure
7 that this constitutes every study that fits the question I
8 asked?

9 A. Let me try to answer. Exhibit 7 is a list of
10 studies that address Parkinsonism and Parkinson's disease
11 related to manganese exposure and/or welding that are
12 funded by industries or industry groups.

13 Q. Okay. That you could locate?

14 A. That I could locate within about a 15-minute
15 period.

16 Q. All right.

17 A. And there may be --

18 MR. BISSELL: Among the materials that you
19 have with you.

20 A. Among the materials that I have with me today.

21 Q. (BY MR. LUBEL) You're not saying that the
22 studies referenced on Exhibit 7 references every study
23 funded by industry or a company related to manganese, true?

24 A. Oh, my goodness, no.

25 Q. All right. Fair enough. Okay. One of your

1 binders is labeled critical documents or something to that
2 effect. Do you have that handy?

3 A. Yes.

4 Q. Is that something you put together?

5 A. Yes.

6 Q. You call it "manganese critical studies"?

7 A. Yes.

8 Q. Just in general can you give me a brief
9 description of what makes these critical studies?

10 A. Yes. These are the studies that have looked
11 specifically at risks of Parkinson's disease or
12 Parkinsonism among welders.

13 Q. I'll mark that as Exhibit 8. Okay?

14 A. Sure.

15 (Garabrant Exhibit No. 8 was marked)

16 Q. (BY MR. LUBEL) Let me talk to you for a minute
17 about studies. When you use the term "studies," do you
18 mean epidemiological or toxicological studies; or do you
19 mean any study?

20 A. Well, it depends on the context in which I use
21 the word "studies."

22 Q. In the context of this critical study manual that
23 I've marked as Exhibit 8.

24 A. Epidemiological studies.

25 Q. So, that doesn't include all the review articles

1 or toxicological studies and whatever else there could be
2 in the universe, true?

3 A. Correct.

4 Q. Exhibit 8 is epi studies, right?

5 A. Yes.

6 Q. All right. I'm going to talk to you for a minute
7 about epidemiology studies. Are you familiar with the term
8 called power calculation?

9 A. Yes.

10 Q. What is it?

11 A. The power is one minus the beta error, and a
12 power calculation is a calculation of one minus the beta
13 error.

14 Q. And what is it designed to tell somebody?

15 A. It's designed to tell somebody the probability
16 that a research study would have missed finding an
17 association to be statistically significant when it, in
18 fact, existed.

19 Q. If it existed, right?

20 A. If it, in fact, existed.

21 Q. Now, you yourself have done epidemiological
22 studies in an original research context, correct?

23 A. Yes.

24 Q. In your studies did you perform power
25 calculations?

1 A. We routinely perform power calculations in the
2 planning phase of epidemiology studies.

3 Q. Is that generally accepted in the field of
4 epidemiology?

5 A. Yes.

6 Q. Now, is it generally accepted within the field of
7 epidemiology to report the power calculation in the actual
8 study or the published material?

9 A. Well, the epidemiology community largely agrees
10 that post hoc power calculations, meaning after the study
11 has been conducted, are relatively uninformative.

12 Q. I'm asking you if it's generally found in the
13 epidemiological community that power calculations are
14 reported by the authors of the study in the actual study or
15 the published materials?

16 A. Well, I'd have to ask you do you mean power
17 calculations that were done in the planning phase or power
18 calculations that are done after the data has been
19 collected and analyzed?

20 Q. Well, let's start with the planning phase.

21 A. No.

22 Q. How about after the data has been analyzed?

23 A. Some journals like to see that. Most do not.

24 Q. So, as a general rule the authors are not
25 publishing the power calculation in the study, true?

1 A. That is far more common than the converse.

2 Q. And now, have you historically performed a power
3 calculation in the planning stage of the studies that you
4 worked on?

5 A. Yes.

6 Q. Can you think of any occasions where you did not
7 perform that power calculation at that stage of the game?

8 A. There may well have been in the distant past. I
9 can't think of any specifically. The typical setting where
10 we don't perform power calculations is where the data set
11 already exists and we have no opportunity to gather
12 additional data or to enlarge the study in which case one
13 is stuck with whatever the power the data set has, and
14 performing a power calculation doesn't help you. It simply
15 tells you that you have low power or high power and doesn't
16 change what you do.

17 So, the answer is when we're planning a study where
18 we're actually going to go out and design the study and
19 have opportunities to create the study, we always perform
20 power calculations before we do the study.

21 Q. Does the power calculation add any interpretive
22 value to an epidemiologist such as yourself?

23 A. You're talking now about the power calculation
24 done in the planning phase or in the post hoc phase?

25 Q. Start with the planning phase.

1 A. Yes. It helps you tremendously in interpreting
2 what you ought to be doing and altering your study design
3 to improve it.

4 Q. Now let's talk about the -- what did you call it,
5 the post hoc study?

6 A. Yes.

7 Q. What is your answer for that?

8 A. No, I don't think it helps at all.

9 Q. Doesn't help you interpret the study?

10 A. Not if the data had been properly reported out
11 with confidence intervals, no.

12 Q. Does the confidence interval give you the power
13 calculation?

14 A. No.

15 Q. Do they mean the same thing?

16 A. No.

17 Q. So, how can you glean anything from the
18 confidence interval about the power calculation?

19 A. You don't need the power calculation once you've
20 got the results with the confidence interval. The power
21 calculation doesn't tell you anything.

22 Q. Why?

23 A. Because the confidence interval tells you what
24 the estimated association is and gives you the range of
25 associations that are compatible with the data you found,

1 and you could easily see from that whether the study is
2 reasonably compatible with the null hypothesis or whether
3 it is not reasonably compatible with the null hypothesis.
4 Power calculation doesn't assist you beyond that. You've
5 got the answer. It is what it is.

6 Q. Okay. Well, let me give you an example.

7 (Garabrant Exhibit No. 9 was marked)

8 Q. (BY MR. LUBEL) I'm going to mark as Exhibit 9
9 what I've written out as 95 percent confidence interval,
10 .9 to 8.0. What does that tell us generally?

11 A. It tells us that whatever it is you've
12 measured -- and I have no idea what it is -- well, actually
13 let's back up. What did you measure here? The confidence
14 interval, without knowing what it is you measured and
15 without knowing what the measure of association was,
16 doesn't tell me anything.

17 Q. Let's say it's a benzene study on AMLs.

18 A. And what is it that you measured?

19 Q. I measured the relative risk of AMLs in a
20 refinery setting versus in a control group.

21 A. Now I can interpret it.

22 Q. Talk to me.

23 MR. BISSELL: Can he mark on your exhibit?

24 MR. LUBEL: Sure, as long as he clearly
25 delineates what he writes versus what I write.

1 A. I'm trying to make an estimate of what the
2 relative risk is. It's probably 2.8 or thereabouts, which
3 would have aided me in interpreting the confidence level.
4 What this says -- assuming that the relative risk is about
5 2.8, what this says is that the most likely value for the
6 relative risk is 2.8 and values between .9 and 8.0 are
7 reasonably compatible with that point estimate.

8 Q. (BY MR. LUBEL) Okay. Let me ask you this now:
9 If I were to say that I performed a power calculation and
10 the study was not designed to show a statistically
11 significant relative risk of 2 or less, can you look at
12 that confidence interval and tell me whether I'm full of it
13 or not?

14 A. I can't answer this question the way you've asked
15 it. It doesn't make sense.

16 Q. Okay. I'm sure that doesn't surprise you.
17 Do you understand what I'm trying to ask? Can you ask
18 the question for me?

19 A. If you'll answer it.

20 Q. I can't. I'm not the scientist. You are.
21 You know what I'm trying to ask?

22 A. I think I sense what you're trying to ask.

23 Q. Let me see if I can ask it better.

24 A. Well, let me just try and answer -- let me try
25 and answer the issue. What this confidence interval says

1 is that even though our best estimate of the relative risk
2 is about 2.8 -- and that's a guess -- it could be 2.7. I
3 don't know.

4 Q. You're just giving us an example, right?

5 A. Yes.

6 -- even though our best estimate is that the relative
7 risk is 2.8, in fact we have little sense for -- or I
8 should say, in fact, the data would reasonably well support
9 that it could be 1.0, meaning there's no association, and
10 reasonably well support that it could be 7 or 8, meaning
11 there's a very strong association. So, what the confidence
12 interval is telling us is that we have very little
13 information about the relative risk because it could be
14 anything from no association to a very strong association.
15 Now, what that tells me is this was probably a small study
16 that had low power.

17 Q. All right. Let me ask --

18 A. Because the answer it gave is relatively
19 uninformative.

20 Q. And in your world of epidemiology, when you
21 calculate out 2.7 or 2 -- relative risk for what's
22 contained in Exhibit 9, do you frame that or represent that
23 to be an association but of no statistical significance?
24 What do you call that?

25 A. I would typically write that the relative risk

1 was 2.8 and the 95 percent confidence interval was .9 to
2 8.0. And now, if the journal in which I was publishing
3 that needed a further explanation -- and the epidemiology
4 journals do not -- I might add a sentence that says this
5 suggests an association, but it was not statistically
6 significant.

7 Q. And the reason you say it suggests an association
8 is because it has a positive relative risk; but the reason
9 you say that it was not statistically significant is
10 because the confidence interval included 1 or was below 1
11 and included 1, true?

12 A. Not quite true. The reason I would say it
13 suggests an association is because the relative risk is
14 substantially different than 1. I wouldn't say that if the
15 relative risk was 1.1. In other words, that's so close to
16 1 that even though it's positive, I wouldn't even say it
17 suggests an association.

18 Q. Okay. So, if it was 1.1, you'd say it does not
19 suggest an association?

20 A. I wouldn't say that it suggests one.

21 Q. Gotcha. All right.

22 A. And, again, one has to look at the confidence
23 interval as well. If the relative risk was 1.1 and the
24 confidence interval went from 1.05 to 1.15, I would say,
25 well, it suggests or it indicates there is a weak

1 association that is statistically significant. In other
2 words, that's a study that has very high power to find very
3 low risks and, in fact, found a very low risk to be
4 statistically significant.

5 Q. And in your world of epidemiology, don't y'all
6 prefer more narrow confidence intervals rather than wide
7 ones as a general rule?

8 A. Well, we prefer studies that have a great deal of
9 information in them that assist us in getting definitive
10 answers.

11 Q. I mean, help me put some teeth in this. If you
12 see a confidence interval, for example, that's 1.1 on the
13 low end to 105 on the high end, is that one -- everything
14 else being equal -- more meaningful than one that's 1.1 to
15 5?

16 A. No, I wouldn't say that's more meaningful. In
17 your first instance what that confidence interval says to
18 me is that the relative risk could be practically 1, no
19 association, or it could be 100. In other words, I have
20 very little sense for where it is. It could be anything
21 from no association to extraordinarily strong association.
22 What that says is I don't have a lot of ability to
23 interpret.

24 Whereas the second one where the range is 1.1 to 5, I
25 have more information; and it tells me the relative risk is

1 within a much narrower range. So, I have a much better
2 ability to say, you know, that's -- I mean, that one would
3 give about a 2.2-fold relative risk. And so, I say, you
4 know, it's 2.2-fold; and the confidence interval varies by,
5 you know, about two times that or one half that on either
6 side. I've got a much more precise sense for where the
7 truth is, and the issue is precision and narrow confidence
8 intervals give you a better sense -- give you more
9 precision than wide confidence intervals.

10 Q. And what you wrote -- I'm just going to draw a
11 line in between the first example and the second example
12 and that's -- if you'd write relative risk next to the 2.8.

13 A. (Witness complies)

14 Q. How do you calculate the relative risk from a
15 confidence interval?

16 A. You convert it to the log scale and find halfway
17 on the log scale.

18 Q. You know that doesn't mean anything to me.

19 A. I don't know.

20 Q. What is the log scale?

21 A. You take the --

22 Q. You're joking, aren't you?

23 A. No, I'm not. You take a logarithm of the two
24 points and find half the distance on the log scale and then
25 take the antilog.

1 Q. All right. So, in some of these studies where
2 you see a confidence interval, that they don't report the
3 relative risk?

4 A. I've never seen that. I've seen the relative
5 risk without the confidence interval. I've never seen the
6 confidence interval without the relative risk.

7 Q. Well, it sounds like the confidence interval is
8 more meaningful to you because you can backtrack from that
9 and determine the relative risk; and then you have both.
10 Is that a fair characterization? Whereas if you just have
11 the relative risk, you can't assess what the confidence
12 interval was.

13 A. Neither of those statements is correct.

14 Q. I'm sure that stops you. I really don't know the
15 answer. I'm trying to learn something --

16 A. Well, you know, typically we go through -- or I
17 go through the study and pull out of it the information I
18 can find that helps me to interpret it. If there's a
19 relative risk without a confidence interval, sometimes I
20 try to calculate the confidence interval from the data in
21 the study.

22 Q. But not from the relative risk by itself? You
23 have to look at the --

24 A. You can't do it from the relative risk. You need
25 the relative risk in the standard error, and you have to

1 figure out what the standard error is from the data in the
2 study. And then you can calculate the confidence interval,
3 and there are ways you can make approximations. If you
4 have the confidence interval, you can back calculate an
5 estimate of the relative risk because the confidence
6 interval typically is the relative risk plus or minus 1.96
7 standard deviations.

8 Q. Okay. Let me ask you this --

9 A. So, the difference between the lower limit and
10 the upper limit is about four standard deviations. Just
11 try and get the midpoint on the logarithm scale or a more
12 applicable scale.

13 Q. All right. Let me go back to when I asked you
14 the question that didn't make any sense. You tried to help
15 me by answering the question you thought I was asking, but
16 let me be more specific. Where Dr. Lewis says "I've done a
17 power calculation post hoc study," you've heard him say
18 that or seen that, right?

19 A. I don't recall that, but he may well have.

20 Q. Okay. Just assume with me he's done that; and
21 let's say he says something like on a particular study
22 "This study was not powered to detect a relative risk less
23 than 100." Okay? You with me so far, or is that not
24 making any sense in your world?

25 A. Well, as stated it's not technically correct. I

1 mean, you've left out a number of details that are
2 necessary to make that an interpretable statement.

3 Q. Assuming he's done the right workup to make that
4 statement. Okay?

5 A. Let's assume that he had a complete sentence that
6 contained the right details.

7 Q. What should it say?

8 MR. LUBEL: I'm going to go to the restroom
9 and let you --

10 A. Can you ask the question again?

11 Q. (BY MR. LUBEL) We can --

12 MR. LUBEL: I'm going to go to the rest
13 room.

14 MR. BISSELL: Take a short break?

15 MR. LUBEL: Yeah.

16 (Recess from 4:08 p.m. to 4:15 p.m.)

17 Q. (BY MR. LUBEL) Have you reviewed Dr. Lewis'
18 opinions or been told that some of his comments about
19 particular studies are designed to -- not designed -- that
20 they claim that the power calculation when performed post
21 hoc study reflected that they would not yield or find a
22 relative risk, for instance, below 100? Are you familiar
23 generally with those comments?

24 A. I have read some of Dr. Lewis' testimony. Let me
25 try to ask your question for you, if I may.

1 Q. Thank you. That would be helpful.

2 A. I think the issue you're getting at is some of
3 his concerns are that some studies have low power and that
4 they -- which means that they would be unlikely to find
5 even a strong association to be statistically significant.
6 So, in other words, to rephrase your question, some
7 hypothetical study to which you're referring had such low
8 power that it would only have found a relative risk of 100
9 or more to be statistically significant.

10 And so, one of the missing elements was the theme that
11 whether it would find the association to be statistically
12 significant. That's different than whether it would find
13 the association.

14 Q. Gotcha. In general are you troubled with that
15 kind of commentary?

16 A. No, I'm not. And here's why: Some of the
17 studies to which he refers, in fact, have low power, which
18 means that when the relative risks and confidence intervals
19 are calculated, the confidence intervals are quite wide.
20 Okay. But those studies show no association between
21 whatever it was they were studying, typically welding and
22 Parkinson's disease or welding and Parkinsonism.

23 So, the correct interpretation of those studies is not
24 based upon their power. It's based on what did they find.
25 They found no association. It's perfectly reasonable to

1 ask, well, could there have been a two-fold or a five-fold
2 association? Let me rephrase that. Perfectly reasonable
3 to ask if there had been a two-fold or five-fold
4 association, could a study of this size have found it to be
5 statistically significant? And the answer to that question
6 would have been, well, it would be unlikely.

7 Now, what's the more important question in which I
8 haven't seen Dr. Lewis ever address is we have perhaps a
9 dozen studies, none of which show a positive association.
10 It's absolutely inappropriate to sit and criticize each of
11 those studies independently as having low power and to
12 ignore the fact that if you put them all together in any
13 summary analysis or a more formal meta analysis, what you
14 actually have is a substantial body of evidence that has
15 failed to find an association and that in the aggregate has
16 good power to find an association.

17 And so, what you really want to do is summarize them
18 in a meta analysis, calculate a meta relative risk and a
19 meta confidence interval and then talk about what the body
20 of literature in the aggregate sense. When you do that, it
21 is quite clear that welding has no evidence of an
22 association with Parkinson's disease or Parkinsonism; and
23 it gives reasonably good assurance that that's either a
24 weakly negative association, no association, or perhaps a
25 weakly positive association; and that's it.

1 Now, it also needs to be said that the burden of proof
2 is on the scientist who claims the association exists. You
3 can't say, well, it's been looked at twelve times. Nobody
4 ever found it; but since it didn't have much power, I
5 concluded it's there. That is a nonscientific opinion.
6 That's simply groundless. All the evidence you have is
7 that there's no association. You can't say, well, it
8 doesn't disprove that there could be an association. We've
9 looked and looked and looked. We never found any evidence
10 of an association. That's what the evidence is.

11 Q. Has anybody done a meta analysis to your
12 knowledge?

13 A. Well, I've done everything except the final
14 calculations to calculate the meta relative risk and meta
15 confidence interval. I've summarized all the studies with
16 all the relative risks and all the confidence intervals. I
17 just haven't done the final calculation to sum them up.

18 Q. You don't have that here today, do you, Doctor,
19 the final calculation?

20 A. No, I haven't done it.

21 Q. Okay.

22 A. But the overall -- you know, if you put all the
23 relative risks and confidence intervals together, it's
24 pretty clear what they say. There's no association. I
25 mean, you don't really need to do the calculation to see

1 it. The calculation only tells you what you can already
2 see.

3 Q. All right. Let's take a mental break for a
4 minute, and I'm going to take some of your materials and
5 mark them and ask you to identify them. That's pretty
6 simple stuff, and then we'll jump back into the other
7 stuff.

8 I'm not going to mark, like, your books from your
9 library; but I am going to ask you to identify them and
10 tell me what you're using them for. Like, the "2006 TLVs
11 and BEIs" from the ACGIH Worldwide, are you using that in a
12 case?

13 A. Well, I only refer to this when someone asks me
14 what the TLV is for welding -- for manganese because I
15 don't memorize it. I look it up.

16 Q. How about the "Pocket Guide to Chemical Hazards"
17 from the CDC in 1997?

18 A. Well, it's actually from NIOSH; and I have the
19 newest one, which is the same publication from September,
20 2005. And I use it in a similar manner. If somebody asks
21 me what the OSHA PEL is or the NIOSH REL is for manganese
22 or welding fume, I look it up. That's all.

23 Q. Okay. And then you've got a "Washington State
24 1950 to 1979 Occupational Mortality" book from the
25 U.S. Department of Health and Human Services. What do you

1 use that for?

2 A. That just provides background for one of the
3 studies I rely upon, which has now gone to a Web based
4 format. And that's the study I refer to as "Milham"
5 because Sam Milham was the state epidemiologist for
6 Washington state for many, many years; and he created the
7 database that allows us to examine the portional (phonetic)
8 mortality among welders and flame cutters and specifically
9 look at the Parkinson's disease mortality in that
10 occupation. So, I don't actually use the book. I use just
11 the single page from the Web printout, but the book tells
12 more about the study.

13 Q. You're talking about the printout that's in the
14 manganese critical studies that we've marked as Exhibit 8?

15 A. Yes.

16 Q. All right. Thanks. Then I'm going to set these
17 aside and put them back over on your side of the room.

18 (Garabrant Exhibit No. 10 was marked)

19 Q. (BY MR. LUBEL) Okay. I'm going to mark as
20 Exhibit 10 this characterization of arc welding fume by the
21 American Welding Society, Incorporated. What do you use
22 that for?

23 A. Well, this characterizes arc welding fume; and of
24 greatest importance to me is the characterization of the
25 welding fume from E6010 rods and E7018 rods.

1 Q. Why?

2 A. Because it shows that the welding fume contains
3 about 4 to 5 percent manganese when those rods are used.

4 Q. The fume or the electrode?

5 A. Huh?

6 Q. The fume?

7 A. It's the fume.

8 Q. Have you seen any characterization of the 7018
9 MIL-SPEC?

10 A. I do not recall it.

11 Q. Do you know what the portion of the manganese in
12 the fume of that particular rod is?

13 A. I am not an expert on the differences among the
14 rods. If the 7018 MIL-SPEC is the same as the 7018, I
15 would assume they have similar manganese content; but I do
16 not know that there's a difference between the MIL-SPEC and
17 the non MIL-SPEC rods.

18 Q. Do you know whether a welder using either the
19 6010 or the 7018 can be overexposed in conditions such as a
20 confined space?

21 MR. BISSELL: To what?

22 MR. LUBEL: To welding fumes.

23 A. Well, it would depend on many factors.
24 Confined-space welding is an important issue for industrial
25 hygienists to ensure that welders are safe when they work.

1 And as to whether confined-space welding is being done in a
2 safe manner is within the purview of an industrial
3 hygienist who is responsible for ensuring the work
4 practices are being properly done.

5 Q. (BY MR. LUBEL) I'm just asking if you have an
6 opinion as to whether it's possible for a welder using, for
7 instance, a 6010 or a 7018 welding rod to be overexposed to
8 welding fume in a confined space?

9 A. I don't believe that that occurs if there is
10 appropriate industrial hygiene oversight of the
11 confined-space welding.

12 Q. But if you don't have appropriate oversight, it's
13 possible, is it not?

14 A. Then you have a welder potentially doing the
15 tasks that are unsafe and against safety policies and
16 corporate policies, and you're not supposed to be working
17 in that manner.

18 Q. They're not; but under those circumstances they
19 could be overexposed, correct?

20 A. Again, without knowing the circumstances, I can't
21 say. What I can say is confined-space welding carries
22 substantial danger unless it is monitored by a professional
23 who's qualified to see that it's being done properly. And
24 the issue is not only welding fume. You can get into
25 oxygen deficient environments and electrical --

1 electrocution hazards when working with electrical
2 equipment in small spaces. There are a lot of issues in
3 confined space welding that require professional oversight.

4 Q. And can the industrial hygienist actually through
5 monitoring measure the actual fume to determine whether it
6 was overexposed or not?

7 A. That is an issue that should be addressed by an
8 industrial hygienist.

9 Q. Do you know the answer?

10 A. It is my belief that industrial hygienists who
11 are well qualified to assess exposures in confined spaces
12 have the tools to reasonably well measure the exposures of
13 people who are put in those environments.

14 Q. What tools are you referring to?

15 A. The various sampling devices that industrial
16 hygienists use for measuring oxygen content, gas content of
17 the air, welding fume, totally suspended particulates,
18 et cetera, et cetera. And that would be a whole range of
19 equipment they might choose to use.

20 Q. They can measure manganese, can they not?

21 A. Industrial hygienists can measure a total
22 suspended particulate typically on filter cassettes and
23 then they can -- depending on the analytical equipment
24 available in the laboratory, they may be able to measure
25 the metal's content of that particulate they collected on

1 the --

2 THE REPORTER: I didn't hear the end of
3 that.

4 THE WITNESS: That they have collected on a
5 filter cassette.

6 THE REPORTER: I need you to speak up a
7 little bit for me.

8 THE WITNESS: I'll do my best.

9 Q. (BY MR. LUBEL) Do you know if a person in a
10 welding environment if they're not adequately ventilated
11 whether or not they can be overexposed? Want me to re-ask
12 it?

13 A. Could you clarify that question?

14 Q. Sure. Do you know if a welder is welding in an
15 environment where they are not adequately ventilated
16 whether that means they can be overexposed to the welding
17 fume?

18 A. Well, exposure to welding fume is determined by
19 many factors, of which ventilation is one; and it's the
20 responsibility of the employer to ensure that the exposures
21 are properly controlled and the safety of the welder is
22 adequately ensured. That may include providing
23 ventilation. That may include restricting certain types of
24 welding in certain locations. I'm not familiar with all of
25 the approaches and tools available to a professional --

1 certified industrial hygienist to ensure that. But it is
2 the responsibility of the employer to provide oversight
3 over typically confined-space welding to ensure safety.

4 Q. What is your definition of adequate ventilation
5 in a welding environment?

6 A. I am not a certified industrial hygienist. To
7 the extent I'm aware of it, adequate ventilation is such
8 that the exposure levels to all of the materials that are
9 in the air are below the permissible exposure level.

10 Q. Which in welding would be what, five milligrams
11 per cubic meter as a ceiling guide?

12 A. That's where we have to get out one of the books
13 we just put away.

14 MR. BISSELL: Are you asking about welding
15 fume?

16 MR. LUBEL: Yes, sir.

17 MR. BISSELL: Do you know the answer?

18 MR. LUBEL: I think I do. I want to make
19 sure ours is the same, though.

20 MR. BISSELL: Do you want me to tell you?
21 You don't want to short-cut this? Okay. That's fine.

22 (Brief pause)

23 (Garabrant Exhibit Nos. 11 through 17 were marked)

24 Q. (BY MR. LUBEL) Sir?

25 A. I'll try to answer -- we sent them a letter that

1 there is not a permissible exposure limit for welding fume.
2 Welding fume is covered under nuisance dust and is also
3 covered under the PELs for each of the components of the
4 welding fume. So, for example, if the welding fume
5 contains manganese, one of the PELs that would cover it
6 would be the PEL for manganese.

7 Q. What is the PEL for manganese? Do you think it's
8 5 milligrams per cubic meter? I'm not going to hold you to
9 it. I just --

10 A. I believe it is, but the trouble is this is in
11 such small print, I can't read it. I've got to reprint it.
12 I think it is.

13 Q. You can look it up later?

14 MR. BISSELL: He's told you that's the
15 answer he wants is what you think it is.

16 Q. (BY MR. LUBEL) Okay. That is what you think it
17 is; and if you're wrong, I won't call you a criminal. Fair
18 enough?

19 A. Well, thank you.

20 Q. Okay. Exhibit No. 11, can you just identify that
21 for us, Doctor? Seems to be your billing invoices.

22 A. Appears to be and I think it is copies of all of
23 my billing invoices related to welding litigation.

24 Q. I take it it's not through today; is it?

25 A. No.

1 Q. It's through what?

2 A. Appears the most recent one was August 27. Most
3 recent invoice was August 27th, 2006, for a deposition
4 taken on May 17th, 2006.

5 Q. Have you done any work since August 27th?

6 A. Yes.

7 MR. BISSELL: Actually since May. That only
8 goes through May, I think; isn't that right?

9 THE WITNESS: Yes.

10 Q. (BY MR. LUBEL) Can you attach to that exhibit
11 any invoices you have between May and today?

12 A. I don't know that I have created any invoices.
13 Well, I mean, this invoice was created in August covering
14 services in May. I don't know whether I've created any
15 invoices since August.

16 Q. Okay. Exhibit 12 is a copy of your current
17 resumé?

18 A. That one is out of date. I brought a new one.

19 Q. All right. I'm going to mark your current one as
20 Exhibit 18, correct?

21 (Garabrant Exhibit No. 18 was marked)

22 Q. (BY MR. LUBEL) What has changed of any
23 significance between Exhibit 12 and Exhibit 18?

24 A. I added a bunch of papers and presentations.

25 Q. Anything specific to the topics related to the

1 lawsuit?

2 A. No.

3 Q. Can you identify Exhibit 13 for us?

4 A. I believe this is a declaration I wrote in the
5 MDL welding rod products liability litigation.

6 Q. Is it a sworn statement?

7 A. I think it's a declaration and --

8 Q. Did you swear to it?

9 A. I did, yes.

10 Q. Okay.

11 A. And I wrote it in 2004.

12 Q. Exhibit 14, can you identify that?

13 A. That's a report I wrote in the MDL welding
14 products liability litigation, and I wrote that in 2004.

15 Q. All right. Can you identify Exhibit 15?

16 A. This is a reference list, and I'm not sure when I
17 created this. It contains 263 references, and I believe
18 these are the references that I have brought with me today.
19 There may be others I have brought with me today that are
20 not on this list simply because those others are recently
21 published.

22 Q. I assume that that exhibit includes the
23 literature and articles that you're relying upon in the
24 case or the litigation?

25 A. Exhibit --

1 Q. 15.

2 A. -- 15 includes articles I'm relying on in this
3 case. There are additional articles that I'm relying upon
4 that are not listed there but which are in the book that
5 you have marked as Exhibit 8.

6 Q. I take it that between Exhibit 15 and Exhibit 8
7 and possibly other notebooks that we're going to talk about
8 later today, that would comprise the literature that you've
9 reviewed and that you're relying upon in the case?

10 A. Yes.

11 Q. Unless you find something new?

12 A. That's correct.

13 Q. Between now and trial, right?

14 A. Yes.

15 Q. Okay. Exhibit 16 appears to be a transcript of a
16 deposition you gave in the MDL, and it looks like it got an
17 attachment of something in the Elam case?

18 MR. BISSELL: That may be a mistake. I'm
19 not sure we intended to attach --

20 MR. LUBEL: I just didn't want to tell him
21 it was the MDL and then have it -- you know, because it's
22 got Elam attached it to.

23 MR. BISSELL: -- asserted on my behalf. I'm
24 sure we shouldn't have attached something that wasn't
25 responsive that we just put in there.

1 MR. LUBEL: Thanks.

2 MR. BISSELL: Sure.

3 A. Exhibit 16 appears to be my deposition taken
4 January 14, 2005, in the MDL welding rod products liability
5 litigation. It appears to have attached to it what I think
6 is trial testimony from the Elam case, October 22nd in
7 2003.

8 Q. (BY MR. LUBEL) Okay. Doctor?

9 A. Yes.

10 Q. Counsel for the defendants has represented to me
11 that he did not believe that you've been asked to offer any
12 specific causation opinions as to Mrs. Godwin. Is that a
13 true statement?

14 A. Well, I've been asked to answer or to give
15 opinions regarding general causation as to whether welding
16 causes Parkinsonism and as to whether welding causes
17 Parkinson's disease. In the instance of Catherine Godwin,
18 it would be my opinion that insofar as it's not known to
19 the world of science that welding causes either of those
20 diseases, there is no basis for attributing her disease to
21 her welding.

22 Q. But that's the only opinion you have that is
23 specific to her, correct?

24 A. Yes.

25 Q. And 17 is just a copy of the report that the

1 defense lawyers supplied you regarding Dr. Sethi, correct?

2 A. That's correct.

3 (Garabrant Exhibit No. 19 was marked)

4 Q. (BY MR. LUBEL) And Exhibit 19, just generally
5 can you describe what's in that stack of documents?

6 A. These are various handwritten and typed notes
7 that I created at various times over the past three or four
8 years. They're really separate documents. The first is
9 titled "Criticism of Racette, 2005." These are criticisms
10 that I wrote down I believe at the time of the Pressler
11 trial, which was --

12 Q. '05.

13 A. Yeah, I was going to say about a year ago, I
14 think. The second --

15 MR. BISSELL: January, '05.

16 A. The second is my notes on a number of case
17 reports of Parkinson's disease in welders. The third is my
18 notes titled "diagnosis of Parkinson's disease" and then at
19 the bottom of that page titled "review of literature" which
20 is actually identical to the previous document. It's a
21 case reports relied upon by plaintiffs. The next document
22 in that exhibit is titled "studies of incidence and
23 prevalence of PD and Parkinsonism in men." And this is
24 nothing more than a table giving background incidence and
25 prevalence of PD and Parkinsonism in various populations.

1 A fifth document is attached to Exhibit 19 and
2 probably doesn't belong there. It's a bunch of
3 advertisements by plaintiffs' law firms to attract welders.

4 Q. (BY MR. LUBEL) And you're not using that to form
5 a basis of any opinions in this case; are you?

6 A. No.

7 Q. You can take that out; and if you just put that
8 thing -- what do you call it -- back on the exhibit, I'll
9 stack it over here.

10 A. (Witness complies)

11 (Garabrant Exhibit No. 20 was marked)

12 Q. (BY MR. LUBEL) Thanks. And then Exhibit No. 20
13 you recognize as my notice of deposition of you along with
14 the request-for-documents subpoena?

15 A. Yes.

16 Q. You brought your file with you today, correct?

17 A. I did.

18 Q. Okay. Who is Nathan Schachtman?

19 A. He's an attorney at McCarter & English in
20 Philadelphia.

21 Q. Have you met with him on these cases?

22 A. I have met with him on a number of occasions. I
23 don't know what you mean by "these cases."

24 Q. The welding litigation.

25 A. Some of the welding litigation, yes.

1 Q. Have you met with him in the context of any other
2 litigation?

3 A. No.

4 Q. Just welding?

5 A. Yes.

6 Q. In other words, he hasn't asked you to consult in
7 the area of asbestos?

8 A. No.

9 Q. Silica?

10 A. No.

11 Q. Have you done any work on behalf of any asbestos
12 companies?

13 MR. HENDERSON: Objection; form.

14 A. It's not clear to me whether I have or have not.
15 I have testified on behalf of defendants in cases in which
16 it was alleged that gastrointestinal tract cancer was
17 caused by asbestos. I don't know whether any of those
18 would fit the rubric of asbestos companies.

19 Q. (BY MR. LUBEL) Okay.

20 A. They were typically end users of asbestos
21 products.

22 Q. What companies were they, like GE?

23 A. I don't recall offhand. Armstrong World
24 Industries. I think Exxon. I don't recall other names,
25 but that's the typical setting.

1 Q. Have you had any other role in asbestos other
2 than testifying as an expert in some of these cases? And
3 let me be more specific: Have you been a consultant to any
4 companies pertaining to asbestos issues outside of the
5 litigation?

6 A. No.

7 Q. Have you performed any epidemiological studies
8 pertaining to asbestos?

9 A. Yes. I published a couple of papers pertaining
10 to asbestos.

11 Q. On behalf of the car companies?

12 A. I published a meta analysis of mesothelioma and
13 lung cancer risks among auto repair workers and vehicle
14 mechanics. I did not do that on behalf of the car
15 companies. Some of my co-authors did.

16 Q. They just brought you in because of your
17 expertise?

18 A. I was a co-investigator and co-author of that
19 study. I don't know why they brought me in, but I was a
20 contributor to the work.

21 Q. But your co-authors were hired by Big Three?

22 A. I believe that their efforts on that research
23 were supported by payment from the Big Three auto makers.

24 Q. Who were they? Who were the co-authors?

25 A. From memory, Michael Goodman, Pat Hessel, Val

1 Craven, Jane Teta. I've probably forgotten a couple of
2 people.

3 Q. Have you done any work for the tobacco industry?

4 A. No.

5 Q. Have you participated in any epidemiological
6 studies that touched on relative risk or prevalence of
7 incidence of any cancers that may be associated with
8 smoking?

9 A. Yes.

10 Q. What studies were those?

11 A. I'd have to go through my CV. From memory we did
12 a study of lung cancer among welders in which we looked at
13 smoking and found that once we adjusted for smoking, there
14 was no association between welding and lung cancer risk.
15 They were stainless steel welders. So, it touched on
16 smoking related lung cancer.

17 I know some of the studies I've done of
18 gastrointestinal tract cancers, I believe we looked at the
19 role of smoking, I think, for stomach cancer; and I know we
20 looked at it for colon cancer. I know we've looked at it
21 in pancreas cancer. In some number of studies we've looked
22 at cancer risks associated with smoking.

23 Q. Who hired you to do the study regarding the
24 relationship, if any, between lung cancer and welding?

25 A. Nobody hired us. We did that on our own time.

1 Q. When did you do that study?

2 A. Back in the early 1980s.

3 Q. Is that published?

4 A. Yes. It's on my CV.

5 Q. Who are the co-authors?

6 A. First author is Corky Hull, Hull, H-U-L-L.

7 Q. Any others?

8 A. John Peters. I'm not sure I recall who else. We

9 could look quickly. It wouldn't be hard to find it. On

10 Page 12 of my CV, No. 28. The title of the study is

11 "Case-control study of lung cancer in Los Angeles County

12 welders." First author, Hull, C. You want me to read the

13 other authors?

14 Q. Let me just see them.

15 A. No. 28.

16 Q. Thanks. That was in 1989?

17 A. Yes.

18 Q. When did you start that study?

19 A. I don't recall that.

20 Q. Roughly? How many years before it was published?

21 A. Probably two years before that. Probably '87.

22 Q. You have the underlying data from it?

23 A. No.

24 Q. Who does?

25 A. I don't know at this point. It's been almost 20

1 years.

2 Q. Well, who was the last to have it?

3 A. Well, Corky Hull was a resident in occupational
4 medicine at USC; and he actually did the study and wrote it
5 up. He might have it. I don't.

6 Q. What function did you serve?

7 A. I was a faculty member. I think I was his
8 adviser.

9 Q. So, did you actually perform the study; or did
10 you just check it off?

11 A. No, I actually participated with him and
12 supervised his work.

13 Q. Was it his thesis?

14 A. We didn't require a thesis. It was a research
15 project he did.

16 Q. Did you look into any neurological effects at
17 that time?

18 A. No. It was a case-control study of lung cancer.
19 You cannot look at neurological effects in a case-control
20 study of --

21 THE REPORTER: I'm sorry. I didn't hear
22 what you said.

23 THE WITNESS: You cannot look at
24 neurological effects in a case-control study of lung
25 cancer.

1 Q. (BY MR. LUBEL) But you can do a case-control
2 study on neurologic effects from welding; can't you?

3 A. Well, you could do theoretically a case-control
4 study of neurologic disease in which you looked at past
5 exposures to welding if you had the data.

6 Q. Take a long time?

7 A. Well, it depends on what it took to get the data.
8 I mean, if the data were readily available, it wouldn't
9 take a long time. If you had to create the data set, it
10 could take a very long time.

11 Q. Have you performed any epidemiological studies on
12 silica?

13 A. A number of the studies I have performed have
14 included silica exposure as one of the factors we
15 addressed. And typically in a case-control study we might
16 have asked about silica exposure or might have looked at
17 occupational histories and inferred which ones involved
18 silica exposure. For example, we did a study titled
19 "Adenocarcinoma of the stomach and exposure to occupational
20 dust"; and we published that in 1988. To the best of my
21 recollection, one of the dust types that we addressed in
22 that was silica.

23 Q. You're aware that silica is a carcinogen to
24 humans?

25 A. I'm aware that IARC has classified it as a

1 Category 1 agent.

2 Q. Do you agree with that?

3 A. I haven't reviewed that for today. I'd have to
4 say I'm not in a position to give an opinion off the cuff.

5 Q. Okay. Do you agree that asbestos is a
6 carcinogen?

7 A. Yes.

8 Q. How about benzene?

9 A. I believe benzene can cause acute myelogenous
10 leukemia in humans.

11 Q. Have you done any epi studies on benzene?

12 A. Benzene has been one of the exposures that we
13 have addressed in a number of studies I've done.

14 Q. Are they on your resumé?

15 A. Yes.

16 Q. Have you done any studies on benzene that have
17 not been published?

18 A. We just finished a very large cohort mortality
19 study for the United Auto Workers Ford Motor Company joint
20 national committee in which we reconstructed exposures to
21 about 90 different agents. Many of those agents were
22 various petroleum distillate products, some of which likely
23 contained low levels of benzene.

24 Q. Who funded the study?

25 A. The United Auto Workers Ford Motor Company

1 national joint committee.

2 Q. Who did you deal with from Ford Motor Company?

3 A. Gordon Reeve.

4 Q. Who did you deal with from United Auto Workers?

5 A. Frank Myra (phonetic).

6 Q. Has that study been published?

7 A. No.

8 Q. When will it be?

9 A. After we finish the dioxin study that we are deep
10 in the middle of right now. It's filling my time and
11 everyone else's time on my team.

12 Q. What are you doing the dioxin study for,
13 pesticides?

14 A. No. Did you say who or what?

15 Q. For what purpose are you doing a dioxin study?

16 A. We're doing a dioxin study to identify exposure
17 pathways in the environment to humans.

18 Q. Who's funding that one?

19 A. The Dow Chemical Company.

20 Q. Is dioxin a carcinogen?

21 A. One of the compounds, 2378 tetra --

22 THE REPORTER: Say that again. 23 what?

23 A. 2378-dibenzo tetrachlorodioxin is currently
24 categorized by IARC as a Category 1 agent.

25 Q. (BY MR. LUBEL) And are you dealing with the

1 director of epidemiology at Dow Chemical?

2 A. I know the director of epidemiology at Dow
3 Chemical, but he is not my contact person for the study.

4 Q. So, you know Collins?

5 A. Jim Collins?

6 Q. Yeah.

7 A. Yes.

8 Q. You know him from his Monsanto days or Ford days,
9 or did you just meet him in his Dow Chemical days?

10 A. I know him through the Dow Chemical Company.

11 Q. So, who's your contact at Dow?

12 A. Dr. Michael Carson.

13 Q. What's his title, if you know? If you don't
14 know, that's okay.

15 A. I don't know offhand.

16 Q. He's in the epi department?

17 A. No.

18 Q. What department is he in? Medical department?

19 A. Medical department.

20 Q. Are you aware that Dow Chemical bought Union
21 Carbide?

22 A. I am aware of that.

23 Q. Have you done any work for Union Carbide before?

24 A. Not to my knowledge.

25 Q. Have you ever been over to the Dow Chemical

1 Company's medical department?

2 A. I have.

3 Q. Where is it located?

4 A. Are you talking about in Midland?

5 Q. Yes, sir.

6 A. Yes.

7 Q. That's the headquarters, is it not?

8 A. Your questions are not answerable. Which of
9 "that" are we talking about and headquarters of what?

10 Q. Dow Chemical.

11 A. Which "that"?

12 Q. Dow Chemical has a headquarters where the medical
13 department is, correct?

14 A. I believe the world headquarters of Dow Chemical
15 Company is in Midland, Michigan. They have a medical
16 department for the Midland plant in Midland, but it is not
17 in the same location as the corporate headquarters.

18 Q. Do you know whether or not Dow Chemical has
19 epidemiologists in house that work for them?

20 A. I believe they do.

21 Q. How about toxicologists?

22 A. I believe they do.

23 Q. Do you know whether Union Carbide had those
24 specialties?

25 A. I do not know offhand.

1 Q. Have you done any other work for Dow Chemical
2 other than the dioxin study that you're doing now?

3 A. I did a research study for chlorpyrifos.

4 Q. When did you do that?

5 A. 1999 to 2001 or 2002.

6 Q. Was that report published?

7 A. We published a number of papers from that study.

8 Q. When do you expect the dioxin work to be done and
9 in a format that you can publish?

10 A. I've already presented 31 papers from that study.
11 We presented those in August of this year; and we have
12 another, oh, two years of work to finish analyzing the
13 data.

14 Q. Have you ever done any consulting work for the
15 American Petroleum Institute?

16 A. I don't believe so.

17 Q. Have you ever done any work for any oil company
18 or chemical company in the context of a chemical exposure
19 lawsuit?

20 A. I have been an expert witness on behalf of
21 chemical companies in chemical exposure lawsuits.

22 Q. Involving what chemicals?

23 A. It's really hard to recall. I don't know
24 offhand. I'd have to think for a while.

25 Q. How about benzene?

1 A. I have, yes.

2 Q. Toluene?

3 A. I've been an expert in lawsuits in which it was
4 alleged that exposures to mixtures of many solvents had
5 adverse health effects. I believe benzene and toluene have
6 been on the list and many other solvents as well.

7 Q. Are these in California?

8 A. Some have been in California.

9 Q. Was it the IBM litigation?

10 A. No.

11 Q. Which litigation was it?

12 A. I was involved with the Lockheed litigation.

13 Q. On behalf of defendants?

14 A. On behalf of defendants.

15 Q. Have you ever performed any epidemiological
16 studies or reviews that have not been published?

17 A. Yeah.

18 Q. A bunch of them?

19 A. Unfortunately, yes.

20 Q. A bunch of them?

21 Well, not everything gets published unfortunately.
22 Sometimes you have findings that are -- rather than
23 scientific they're called negative findings. And if you
24 don't find any associations, it's hard to get them
25 published. It's much easier to get positive findings --

1 THE REPORTER: Easier to get what?

2 THE WITNESS: Positive findings published.

3 Q. (BY MR. LUBEL) I take it that you're referring
4 to studies that you've submitted for publication that were
5 not accepted?

6 A. Well, I'm referring to studies that have been
7 submitted for publication and not accepted and studies we
8 never bothered to submit because we believed the journals
9 would not be interested in them.

10 Q. What studies have you done that have not been
11 submitted?

12 A. Oh, for example, we did a study of N-acetyl
13 glucose amidase and renal function among a cohort of lead
14 exposed workers hoping to find evidence that lead affected
15 their glucose amidase excretion; and we didn't find it.
16 So, it was a negative study, and we never got around to
17 publishing it.

18 Q. Any others?

19 A. There must be a few others, yeah. I don't know
20 from memory. These are the --

21 Q. Let me give you an example. Let me ask you some
22 specifics. Have you done any studies that could refer or
23 relate to welding that have not been published?

24 A. The only one I can think of is the current study,
25 the cohort mortality study we've done for UAW Ford in which

1 welding was a very important exposure. That has not yet
2 been published.

3 Q. Do you have a draft of it?

4 A. I have a technical report that is submitted to
5 the sponsor.

6 Q. Are you prevented by your rules of ethics to
7 tender that to us, or do you have to wait until the study
8 is published?

9 A. I believe I'm not allowed to distribute that.

10 Q. I take it, then, that you won't offer any
11 opinions of that at trial; is that correct?

12 A. That's correct.

13 Q. Any other studies that you've done that reflect
14 in whole or in part welding or manganese that have not been
15 published?

16 A. I don't think so.

17 Q. How about benzene?

18 A. Same answer.

19 Q. Asbestos?

20 A. Same answer.

21 Q. Silica?

22 A. Same.

23 Q. Vinyl chloride?

24 A. I don't think there's anything.

25 Q. Have you consulted with the American Welding

1 Society specifically?

2 A. No.

3 Q. Have you specifically consulted with any company
4 other than Lincoln Electric?

5 A. I don't believe I've ever consulted with Lincoln
6 Electric or any other welding company.

7 Q. Have you met any of their people?

8 A. I have met a couple of people from Lincoln
9 Electric.

10 Q. Who is that?

11 A. I don't recall their names. It was simply at
12 trial in a previous case.

13 Q. Mr. Brown?

14 A. I don't know.

15 Q. What do they have in the way of a medical
16 department? Do you know?

17 A. I do not know.

18 Q. Have you seen any of their mortality or morbidity
19 data?

20 A. No.

21 Q. Have you seen any mortality or morbidity data
22 from Union Carbide?

23 A. No.

24 Q. From any of the other companies in the
25 litigation, the welding litigation?

1 A. Not to my knowledge, no.

2 Q. For PD, as a person gets older, does their risk
3 of PD go up?

4 A. Yes.

5 Q. When do you start to see that increased risk?
6 What age?

7 A. Probably in their 30s.

8 Q. Most victims of PD over the age of 60?

9 A. Yes.

10 Q. Vast majority?

11 A. The majority.

12 Q. Do you see clear distinction or delineation once
13 you get over 60, 65 years old as to the prevalence of PD?
14 Is there a bright line of sorts?

15 A. No.

16 Q. How about Parkinsonism?

17 A. What's the question?

18 Q. Do you see any bright lines in the age of onset?

19 A. No.

20 Q. As a person gets older, does their risk go up for
21 Parkinsonism?

22 A. I believe so.

23 Q. Similar to PD or different?

24 A. I haven't compared them.

25 Q. Within your materials have you brought with you

1 any animal studies?

2 MR. BISSELL: If you're going someplace

3 else --

4 We've all taken a break, but you haven't had
5 one.

6 MR. LUBEL: Take a break. Take a couple of
7 minutes.

8 (Recess from 5:17 p.m. to 5:27 p.m.)

9 (Garabrant Exhibit No. 21 was marked)

10 Q. (BY MR. LUBEL) Exhibit 21, that's the ICD codes?

11 A. Yes.

12 Q. That's because a lot of the studies reference ICD
13 codes?

14 A. Some of the studies reference ICD codes.

15 Q. You know a Dr. Brain from Harvard?

16 THE REPORTER: Dr. who?

17 Q. (BY MR. LUBEL) Is it Brain or Bain?

18 A. Joe Brain? I don't know a Dr. Bain from Harvard.
19 I know a Dr. Joseph Brain from Harvard.

20 Q. He must be really smart for his parents to have
21 named him Dr. Brain.

22 A. I don't think they gave him that name.

23 Q. Have you seen any of his work in the welding
24 area?

25 A. No.

1 Q. Were you aware that he was doing some animal
2 studies for the welding companies?

3 A. I was not.

4 Q. Have you relied on any animal studies for your
5 opinions in these cases?

6 A. I don't believe so.

7 Q. Is there a place for animal studies in
8 epidemiology or for an epidemiologist to interpret?

9 A. They say there is animal epidemiology done. It's
10 not an area I work in.

11 Q. What's that area called?

12 A. Animal epidemiology.

13 Q. They have a speciality for that?

14 A. No. It's not a speciality, but people do study
15 the patterns of disease in animals. For example, right now
16 there's a tremendous amount of epidemiology being done in
17 bird flocks to track bird flu worldwide.

18 Q. Okay. Latency, do you have any opinions on the
19 appropriate latency from the studies for manganese induced
20 Parkinsonism or is it person by person?

21 A. We're not talking -- that question is not talking
22 about welding. I assume we're talking about in the
23 industries in which there is high level of manganese
24 exposure?

25 Q. Just for any manganese induced Parkinsonism.

1 A. Well, I have an opinion with respect to welding
2 and Parkinsonism. There's no latency because there's no
3 known association. You can't define latency until you know
4 that there's a cause of association. Since there isn't
5 one, latency is not an issue.

6 Q. So, in an individual that's been overexposed to
7 manganese and welding, you're saying --

8 MR. BISSELL: Object to form.

9 Q. (BY MR. LUBEL) -- that individual's Parkinsonism
10 could not have been caused by manganese from welding, true?

11 A. We've already discussed the two case reports in
12 which I believe two welders using high manganese content
13 rods in a confined space suffered from manganism.

14 Q. I'm just saying what is the latency that you
15 require to connect up a person that's been exposed to
16 manganese that has manganese induced Parkinsonism?

17 A. In those industries in which manganism has been
18 observed in some instances, the latency is a matter of a
19 few months.

20 Q. To what?

21 A. To years.

22 Q. How many years?

23 A. I don't know. I don't know what -- I don't know
24 how many years. In fact, I don't know if it's been defined
25 or not.

1 Q. Is it your opinion, Doctor, that there is no set
2 of exposure circumstances in which a welder can get
3 manganese induced Parkinsonism?

4 A. It's my opinion that there is a substantial body
5 of epidemiological literature that has looked at welding in
6 many, many settings and looked at the risks of Parkinsonism
7 and has failed to find any evidence of an association.

8 Q. I'm just asking you: Is it your opinion that
9 manganese exposure under no set of circumstances can lead
10 to manganese induced Parkinsonism in a welding environment?

11 MR. BISSELL: Objection; asked and answered.

12 A. We've already discussed the two case reports of
13 the welders in the rock crusher who I believe got manganese
14 induced Parkinsonism as a consequence of welding in that
15 setting.

16 Q. (BY MR. LUBEL) So, it can happen under the right
17 exposure circumstances, correct?

18 A. I know of two instances in which I believe it
19 happened.

20 Q. Can it happen under the right exposure
21 circumstances?

22 A. I think I just answered that.

23 Q. "Yes" or "no"? Can it happen under the right
24 exposure circumstances?

25 A. The question is so vague that I cannot answer it

1 because I don't know what you mean by "the right exposure
2 circumstances." I have already answered that I believe
3 there was a situation in which two welders working in a
4 rock crusher using high manganese welding rods were
5 overexposed to manganese and suffered from manganism.

6 Q. How much exposure would a welder have to be
7 exposed to of manganese to have manganese induced
8 Parkinsonism?

9 A. We do not know that for welders. We -- we don't.

10 Q. There's no threshold?

11 A. We do not know the answer for welding exposures.

12 Q. But is there a threshold for any manganese
13 exposures?

14 A. In other industries we believe there is a
15 threshold.

16 Q. What is it?

17 A. Well, it is somewhere above the OSHA PEL of
18 5 milligrams per cubic meter as an eight-hour time weighted
19 average that one could have five days a week throughout a
20 working career. It's believed that that PEL is adequate
21 and protective of the people who would not get manganism.
22 As to where the level is that actually induces manganism,
23 it's clear from the studies in the mining industry which
24 exposures were virtually uncontrolled that levels in the
25 range of 100 milligrams per cubic meter are adequate to

1 cause manganism; and that's to pyrolusite. I don't know
2 all of the data in between that setting and the PEL but
3 somewhere above 5; and certainly by the time you get to 100
4 for a product exposure, that's enough to do it.

5 Q. Is there no evidence to suggest that exposures
6 below 5 milligrams per cubic meter on a time weighted basis
7 can cause manganese induced Parkinsonism?

8 A. When you say "no evidence to suggest," I don't
9 know how to interpret that. I am not aware of any evidence
10 that provides a sufficient basis for a conclusion that
11 exposures less than 5 cause manganism.

12 Q. Why did the ACGIH lower their standard to
13 .2 milligrams per cubic meter?

14 A. I don't believe they did.

15 Q. They didn't?

16 MR. BISSELL: I think they did. I think
17 that's right.

18 A. I take it back. I stand corrected. They did
19 adopt a value of .2 milligrams per cubic meter.

20 Q. (BY MR. LUBEL) Why did they do that if 5
21 milligrams per cubic meter is safe?

22 A. The ACGIH adopts values that are often more
23 stringent than OSHA. After their expert panels reviewed
24 the data, they -- I do not know the background of that
25 decision. They may have chosen to lower it to achieve a

1 wider margin of exposure than OSHA felt was necessary.

2 Q. Do you routinely rely on the ACGIH in your
3 evaluation of safe levels?

4 A. I routinely look at the ACGIH TLVs and BEIs in my
5 work and --

6 Q. Are they typically thought of as being reliable
7 in their field?

8 A. Well, the issue, I believe, is how much of a
9 margin of protection one wants to build in between a
10 recommended exposure level and the level at which there is
11 increased risk of adverse effects; and one can somewhat
12 arbitrarily decide to use the ten-fold protection factor or
13 100 or 2 or 20. And I do not know the background of the
14 ACGIH's decision to recommend a time weighted average of
15 .2 milligrams per cubic meter.

16 Q. I mean, that's almost 25 times lower than the
17 OSHA level, correct?

18 A. I think it is 25 times lower than the OSHA PEL.

19 Q. Have you seen any references with regard to TLVs
20 or PELs that they're not to be viewed as fine lines between
21 safe and dangerous?

22 A. I have not seen references to that. I think that
23 setting exposure limits appropriately should be based on
24 identifying levels at which adverse effects occur and then
25 building in substantial margins of protection. And so, the

1 fact that the ACGIH has chosen a level substantially below
2 OSHA does not in any way imply that the level at which
3 adverse health effects of manganese occur is any different
4 for the ACGIH than it is for OSHA. It may simply say they
5 want to be more protective. In other words, they want a
6 wider margin below the level at which there are adverse
7 health effects.

8 Q. Do you know that's, in fact, what they mean?

9 A. No. I said that may well be what they have
10 decided.

11 Q. You don't know?

12 A. I do not know offhand without going back and
13 reading the history of their decision.

14 Q. Is there such thing as individual's
15 susceptibility or vulnerability with regard to any
16 neurological diseases that can be caused by manganese
17 exposures?

18 A. There is hypothetically such individual
19 variations, but I do not believe that any basis is known
20 for claiming an individual is more or less susceptible to
21 manganese. So, it's a hypothetical that is not known to be
22 true.

23 Q. But as you sit here today, it's your opinion that
24 there is no individual susceptibility to manganese at least
25 for neurological purposes?

1 A. No. It's my opinion that we have absolutely no
2 ability to identify who is more or less susceptible.

3 Q. I'm not disagreeing with you. I'm just trying to
4 find out what your opinions are.

5 Do you agree that there are people or people in
6 general have different individual susceptibilities to
7 manganese exposures as far as what type of neurological
8 outcome, if any, will happen to them?

9 A. No, I do not agree because the way you've asked
10 the question presumes that we know that it exists; and the
11 answer is we don't know that it exists. We have no ability
12 to identify who has or does not have that susceptibility.
13 It's a hypothetical that might exist. You might be very
14 susceptible to manganese but we have no way of knowing that
15 and I might be very susceptible and we have no way of
16 knowing that either. It's just a hypothetical.

17 Q. Same true with smoking?

18 A. I'd have to say yes. I wish it were different.
19 I wish we could identify who was going to get cancer and
20 stop them from smoking.

21 Q. Well, how do you explain that some people that
22 smoke a pack a day for five years get lung cancer caused by
23 smoking; and some people that smoke for 60 years, you know,
24 die at 100 from a car accident and never get cancer?

25 A. We have no idea how to explain that.

1 Q. How about individual susceptibility?

2 A. Again, it's a hypothetical. It could be -- it
3 could be that it's something about them. It could be
4 nothing other than the roll of the dice. You roll a bunch
5 of dice. Why does one of them come up six? Why does one
6 of them come up one? It might simply be random. There are
7 a lot of random processes. There could be a reason behind
8 it we haven't discovered, but right now it appears to be a
9 random process.

10 Q. I mean, not everybody that is exposed to asbestos
11 gets the deadly form of cancer called mesothelioma; do
12 they?

13 A. That's correct.

14 Q. Why is that?

15 A. Well, again, we don't know. To some extent risk
16 is clearly related to dose. People who had higher doses of
17 the amphibole fibers are at higher risk than people who
18 have had lower doses. But as to why among people who have
19 had heavy doses, some get mesothelioma and some don't, we
20 simply do not know. It's possible there is susceptibility,
21 but nobody can identify what it is. Nobody can say who's
22 susceptible. It's also possible it's just a random
23 process, luck of the draw.

24 Q. For manganese exposures does anybody know whether
25 it's the cumulative exposures that matter or the higher

1 short-term doses?

2 A. Well, what we do know is the people that have
3 high short-term exposures get their manganism quickly.
4 There's very little lag time. We know that in a number of
5 industries, high-level exposures achieve a sufficient dose
6 in the globus pallidus that the onset of manganism follows
7 very quickly. That's particularly important with respect
8 to Catherine Godwin because her last welding exposure was
9 four years before her symptoms were ever --

10 THE REPORTER: I didn't hear the end of
11 that.

12 THE WITNESS: It was four years before her
13 symptoms as I recall.

14 Q. (BY MR. LUBEL) So, we don't know whether it's
15 the cumulative exposures that make the difference or the
16 high short-term exposures?

17 A. Well, we know in some settings high short-term
18 exposure is sufficient to cause it. We know in other
19 settings high level long-term exposures are sufficient to
20 cause it.

21 Q. Don't we see some circumstances even in the
22 industries that you acknowledge a connection between
23 manganese exposures and Parkinsonism where the workers have
24 worked for 20 or 25 years doing essentially the same job,
25 had exposures to manganese and then nothing happens and

1 then all of a sudden, pow, they have got a disease?

2 A. You'd have to show me examples of that.

3 Q. You've never seen any of those?

4 A. Well, I don't know from memory any that fit your
5 description.

6 Q. But are you generally familiar with the
7 literature enough to know that when you look at it, you
8 don't see necessarily everybody within three months of
9 working around manganese at high exposures coming down with
10 neurological deficits that resemble Parkinsonism? You
11 agree with that, right?

12 A. We've never seen that exact setting. We do know
13 one from all the studies done in Taiwan that high level
14 exposure led to manganism; and I think it was -- it was in
15 the ferro manganese plant. I think earlier I said it was a
16 battery plant. It was a ferro manganese plant.

17 MR. BISSELL: You did say that earlier.

18 THE WITNESS: Yeah, I think I got that
19 wrong.

20 A. That people came down within months of their
21 first exposure, exposure was very high.

22 Q. (BY MR. LUBEL) That's the exception to the rule
23 in the literature; isn't it?

24 A. I don't think it's the exception to the rule. I
25 think it's a clear example of what happens with high level

1 exposure. People can achieve a sufficient dose in the
2 globus pallidus quickly within months, and they get sick
3 quickly.

4 Q. How long does the manganese stay deposited in the
5 lung?

6 A. In the lung?

7 Q. Right.

8 A. I don't know offhand.

9 Q. I mean, do you know if it's released slowly into
10 the bloodstream?

11 A. Your question presumes all sorts of factors that
12 I don't think we've established. The first is that it
13 stays in the lung at all, which I'm not sure we know about.

14 Q. I'm asking you if you do know it.

15 A. Well, typically inhaled dusts that reach the gas
16 exchange region are picked up by alveoli macrophages and
17 those macrophages migrate up and out of the respiratory
18 tract up to the terminal bronchioles and then they get
19 caught in the mucus and go up and out of this area like an
20 escalator. Sometimes those macrophages can migrate out of
21 the alveoli and back into the interstitial tissue and get
22 picked up by the lymphatics and then get collected in the
23 peribronchial lymphatic aggregations. And so, you
24 typically see collections of dust, inert dust, that can't
25 be degraded in those regions. I don't know and I'm not

1 aware that it is known whether that happens for welding
2 fume. In other words, what I don't know is the clearance
3 time of welding fume in the lung.

4 THE REPORTER: The clear what?

5 THE WITNESS: Clearance time.

6 THE REPORTER: Clearance time. I'm sorry.

7 A. In other words, how quickly the macrophages
8 remove it and how much is left after, say, 24 hours,
9 48 hours, 72 hours.

10 Q. (BY MR. LUBEL) How long --

11 A. I simply don't know how long it stays, and I
12 don't think anybody knows about whether manganese is
13 released from any material that does persist in the lungs.
14 It may simply be cleared out of the lungs rapidly and is
15 gone.

16 Q. Once it gets to the brain, how long does it stay
17 in the brain?

18 MR. BISSELL: Object to form.

19 A. Which "it" are we talking about now? Are we
20 talking about welding fume?

21 Q. (BY MR. LUBEL) Just manganese.

22 A. Well, we've got to talk about those situations,
23 if manganese gets to the brain, because it's not clear yet
24 that welding fume gets to the brain or manganese in welding
25 fume gets to the brain. There are many, many reasons why

1 that doesn't occur such as a lot of welding fume not being
2 absorbed --

3 Q. Okay. And so --

4 A. -- welding fume being bound to proteins and being
5 excreted in the kidneys efficiently, some obstacles to
6 welding fume or obstacles to manganese crossing blood
7 barriers. Lots of issues.

8 Q. So, welding fume -- the manganese portion of
9 welding fume cannot get to a human's brain, correct?

10 A. No. Like I said, there are lots of obstacles
11 that keep it from getting to the brain; and there are lots
12 of welders in which whatever manganese exposure they get is
13 handled physiologically and excreted without any
14 appreciable accumulation in the brain.

15 Q. Well, if it gets to the brain, how long does it
16 stay there?

17 A. I don't know exactly. I did not prepare on that
18 topic.

19 Q. Have you attended any seminars on welding?

20 A. I'm not sure what you mean by "seminars on
21 welding."

22 Q. Okay. Have you spoken to any groups of lawyers
23 regarding any opinions you have on the welding cases?

24 A. No.

25 Q. Have you spoken at any industry meetings

1 regarding welding?

2 A. No.

3 Q. Have you met with any other experts regarding
4 welding?

5 A. I have met other experts. I don't know what you
6 mean meet "with."

7 Q. Who are the ones you've met?

8 A. I've met -- I think I've met Dr. Keyberts
9 (phonetic).

10 Q. Anybody else?

11 A. I think I've met Dr. Olonew.

12 THE REPORTER: Say that name again.

13 THE WITNESS: Olonew, O-L-O-N-E-W.

14 Q. (BY MR. LUBEL) Have you spoken with him?

15 A. I think I was introduced. Basically said "Hello.
16 Nice to meet you."

17 Q. Where was that?

18 A. I'm not sure at this point.

19 Q. Who introduced you?

20 A. I don't recall. It was at one of the trials. I
21 don't remember which one. I don't remember where.

22 Q. Any other defense experts other than those two
23 that you've met or spoken to?

24 A. I do not recall any others.

25 Q. Are you a member of any organizations?

1 A. Yes.

2 Q. What are they?

3 A. American College of Occupational and
4 Environmental Medicine, Society for Epidemiologic Research
5 the National Epidemiological Association, Michigan
6 Occupational Environmental Medical Association. I think
7 I'm a member of the International Society for Environmental
8 Epidemiology. There are probably some others I don't
9 recall.

10 Q. Have any of the organizations that you're a
11 member of been affiliated with any of the epidemiological
12 studies that have been done on welding?

13 A. I don't understand the question.

14 Q. Are you a member of IEU or something like that?

15 A. I don't know what IEU is.

16 Q. Okay. So, none of the organizations that you're
17 a member of have had any part in any welding study?

18 A. I'm not aware that they have, nor am I aware if
19 any of those organizations have any part in any study.
20 It's not part of what they do to my knowledge.

21 Q. And you're not a member of any group that has
22 played a part in any of the welding studies, correct?

23 A. I don't even know what that means.

24 Q. Okay.

25 A. What is a "group"?

1 Q. You're not a member of nor have you ever been a
2 member of any organization that has played any role in any
3 of those welding studies, correct?

4 A. It's hard to answer that. It's so vague.

5 Q. Okay.

6 A. I've been on the faculty of the International
7 Epidemiology Institute, which is one of the entities that
8 performed one of the welding studies.

9 Q. What was so hard about that?

10 A. 'Cause I have no idea what you mean by a "group."

11 Q. What do you call them?

12 A. The IEI.

13 Q. Yeah. What is it --

14 A. It's a --

15 Q. -- if it's not a group?

16 A. -- consulting firm.

17 Q. It's a consulting firm?

18 A. Yes.

19 Q. Are you paid by them?

20 A. No.

21 Q. What do you do for them?

22 A. I'm on their faculty. I occasionally talk with
23 them.

24 Q. How does that group finance themselves?

25 A. They work.

1 Q. For whom?

2 A. Clients.

3 Q. Such as who?

4 A. Well, I think that they did a study on behalf of
5 the welding consumable -- or the welding consumables
6 manufacturing industries.

7 Q. Who else have they done work for?

8 A. I believe they do work for the National Cancer
9 Institute by the Federal Government. I believe they do
10 work for other agencies within the National Institutes of
11 Health.

12 Q. So, their only private client was the welding
13 group?

14 A. I don't know their client list. I believe they
15 have other private clients.

16 Q. Do they have anything to do with your studies
17 over at Ford or the Big Three?

18 A. No.

19 Q. As a faculty member, did you have access to the
20 raw data from their welding study?

21 A. No.

22 Q. Can you get it?

23 A. No.

24 Q. Why not?

25 A. I have no right of access to the data.

1 Q. Have you asked for it?

2 A. No.

3 Q. Have you talked to any of the authors about the
4 study?

5 A. Not until after it had been published.

6 Q. Did you know that they were working on it before
7 it was published?

8 A. I was aware of that.

9 Q. How did you learn that?

10 A. I think I was told by attorneys for the welding
11 industry that Dr. Fryzek was working on the study.

12 Q. Schachtman, Nathan Schachtman?

13 A. To the best of my recollection, I don't believe
14 it was Dr. -- Mr. Schachtman who told me that.

15 Q. I call him Dr. Schachtman, too. I am not kidding
16 you. I do. It's a joke, but I do. 'Cause he thinks he's
17 smarter than you.

18 A. He may well be.

19 Q. He probably is.

20 MR. BISSELL: I know he's --

21 MR. LUBEL: He's --

22 MR. BISSELL: He's a lot smarter than I am.

23 (Laughter)

24 Q. (BY MR. LUBEL) Have you prepared any
25 demonstrative aids for your trial testimony?

1 A. In this case, no.

2 Q. Do you have demonstrative aids from your prior
3 trial testimony?

4 A. Yes.

5 Q. Have you brought those with you?

6 A. No.

7 MR. LUBEL: Can I get those from you, John?

8 MR. BISSELL: Probably.

9 MR. LUBEL: Okay.

10 Q. (BY MR. LUBEL) Have you ever done any work with
11 the Navy?

12 A. No.

13 Q. Were you in the Navy?

14 A. No.

15 Q. You said you welded before?

16 A. I have.

17 Q. Where did you weld?

18 A. Washtenaw Community College.

19 Q. What were you doing there?

20 A. I took occupational medicine residents over to
21 see welding so they could understand what it was about and
22 we learned to weld.

23 Q. When was that?

24 A. Ten, twelve years ago.

25 Q. Was that a one-time deal?

1 A. Yeah.

2 Q. One day?

3 A. Yep.

4 Q. Were y'all trained before you welded?

5 A. Yes.

6 Q. Did they monitor your air?

7 A. No.

8 Q. Did they provide you with any respiratory
9 protection?

10 A. No.

11 Q. Did you weld with any rods that had any
12 potentially hazardous components, if you know?

13 A. Well, let's see. We did aluminum welding. It's
14 arc welding. I'm not aware of -- well, every chemical has
15 toxicity issues. Dose -- I'm not aware that we were
16 exposed to anything under circumstances that would have
17 created any hazard.

18 Q. Was your environment mechanically ventilated?

19 A. It had a general dilution ventilation.

20 Q. What is that?

21 A. It was a big room.

22 Q. How big?

23 A. 50 by 40 with a 20-foot ceiling, big room.

24 Q. Was it a garage door that was open? Was any of
25 it open?

1 A. I don't recall at this point to be honest.

2 Q. Was there any manganese in the air?

3 A. Was there any what?

4 Q. Manganese in the air?

5 A. I don't know but -- I don't know. We also did
6 some gas cutting on steel. So, if there was manganese in
7 the steel, we may have generated some manganese fume.

8 Q. But you don't know?

9 A. No, I don't know.

10 Q. Are you relying on the work of any other experts
11 in this case?

12 A. If you can answer the questions, it would save us
13 both time.

14 Q. I assume it's no, but I want to hear from you.

15 A. I don't know. I don't believe I am. There are
16 areas that are outside of my expertise that I may rely on
17 such as exposure assessment and such as neurologists who
18 have seen the plaintiff and reached a diagnosis.

19 Q. But as you sit here today, you've not relied upon
20 anybody else's work, correct?

21 A. Not in forming my opinions, no.

22 Q. Have you authored any case reports?

23 A. In my life, yes.

24 Q. Right. How many?

25 A. I don't know. Small number.

1 Q. Less than five?

2 A. Probably.

3 Q. Are they on your resumé?

4 A. I'd have to look.

5 (Recess from 6:04 p.m. to 6:08 p.m.)

6 A. I'd have to say I don't think I have authored any
7 case reports. I can't find any.

8 Q. (BY MR. LUBEL) What is a case report?

9 A. It's typically a written description of a single
10 patient or a series of patients who have some aspect of
11 their illness that is unusual or uncommon.

12 Q. Have you never seen anything that was unusual or
13 uncommon?

14 A. I have.

15 Q. Not enough to write about it?

16 A. Apparently have never bothered to write them up.

17 Q. All right. Let me ask you this: You gave us
18 earlier what you believe to be diagnostic of manganism, and
19 what I'm curious from you is what would you expect a
20 neurologist to have in his or her records to establish a
21 diagnosis of manganism?

22 A. I would expect that the patient would have a
23 clinical presentation characterized by the factors I
24 testified to earlier today. So, the constellation of
25 neurologic findings that were typical of manganism. I

1 would expect to have results from a diagnostic workup that
2 excluded other causes of movement disorders. I would
3 expect to have a history of working in an occupation that
4 is known to put people at increased risk of manganism to
5 expose them to manganese at levels adequate to cause
6 manganism. And I might expect to see results of imaging
7 studies that show hyperintense signal from the globus
8 pallidus indicating excessive manganese content in that
9 basal ganglia.

10 Q. Would you require the imaging study from the
11 neurologist?

12 A. It would provide strong confirmatory evidence.

13 Q. But is it a necessary prerequisite to a correct
14 diagnosis of manganism in your opinion?

15 A. It's a matter of probabilities. It should be
16 done if one's going to say that a person has manganism and
17 it should be -- I mean, it provides the strongest evidence
18 that the toxin is present in the tissue that is responsible
19 for the clinical syndrome. In the absence of that, I think
20 that the absence of that finding seriously detracts from
21 the confidence that one would have that that diagnosis is
22 correct.

23 Q. But would you require an imaging study in order
24 to assess or evaluate that, in fact, that person actually
25 has manganism? In other words, could you agree with the

1 diagnosis of manganism if everything else is present except
2 for the imaging study? That's a better way to put it, I
3 guess.

4 A. I would consider two conditions. In a person who
5 was currently exposed or currently employed in a job in
6 which it was believed that there was manganese exposure, I
7 would not accept the diagnosis of manganism in the absence
8 of evidence of excessive accumulation of manganese in the
9 globus pallidus. I think that would be inappropriate.

10 In someone that was no longer employed in a setting in
11 which it was believed that there was excessive manganese
12 exposure, the issue is the timing between exposure and the
13 onset of disease. If the disease onset occurred in a
14 manner that was plausibly related to the manganese exposure
15 and the exposure had ceased in the distant past such that
16 the manganese would likely have been cleared from the
17 globus pallidus, I would not require that in the study.

18 Q. And generally how long does it take the manganese
19 to clear that part of the brain with the understanding that
20 it may differ for each individual?

21 A. It certainly takes months. I don't know
22 whether -- I don't know what the upper end on that time is.
23 Let's say one would expect it to have diminished
24 substantially over a matter of years. One would expect it
25 to remain elevated for certainly two, three, four months.

1 In between it's not clear, and it would depend on how much
2 manganese had been there.

3 Q. Now, with respect to what you expect a
4 neurologist to document in his or her records regarding the
5 exposure, what would you expect to see?

6 A. I'd expect to see a detailed occupational history
7 with the job description and the description of tasks
8 performed. I'd expect there to be dates on that, and I
9 would expect to see a history of holding jobs in which
10 there was a reasonable basis for thinking it was
11 overexposure to manganese. In other words, you'd have to
12 work in job in which manganism is known to have occurred.

13 Q. Would you expect the doctor to inquire into
14 whether there was confined-space exposure?

15 A. It would depend on the industry. If somebody
16 worked in dry cell battery manufacturing, I'm not aware
17 that confined spaces are an issue. Somebody working in
18 manganese mining, I'm not sure that confined spaces are an
19 issue.

20 Q. How about the use or nonuse of respiratory
21 protection or equipment?

22 A. Well, you know, if we're talking about welding,
23 that's an occupation that is not known to put people at
24 risk of manganism. So, your questions seemed to be
25 directed mostly toward welding environments such as

1 confined spaces and use of protective equipment. That's
2 not an occupation known to put people at increased risk.

3 Q. So, you wouldn't expect the neurologist to
4 perform a differential diagnosis to determine whether
5 welding was the cause at all, would you, if they were
6 welders?

7 A. You've mixed issues that I don't think have any
8 relationship to each other. Differential diagnosis --

9 Q. Let me do it this way --

10 A. -- doesn't have to do with causation. It has to
11 do with etiology.

12 Q. Let me do it this way --

13 A. It doesn't --

14 THE REPORTER: Wait --

15 A. Differential diagnosis doesn't have to do with
16 etiology. It has to do with reaching a diagnosis.

17 Q. (BY MR. LUBEL) Let's put it this way: For the
18 welder you don't expect a neurologist to perform any
19 analysis of whether manganese could have caused a manganism
20 induced Parkinsonism, correct?

21 A. The only circumstance in which it is reasonable
22 to even think of welding as putting people at risk of
23 Parkinsonism is in the setting that we described using high
24 manganese rods inside a rock crusher without ventilation.
25 And so, if you were the neurologist seeing those patients,

1 I would expect that there would be an exploration of the
2 work and what the current job was and what the recent jobs
3 were. And if there was a history of welding in confined
4 spaces, I would expect that to be asked.

5 Q. Would you expect that same doctor to inquire
6 under those circumstances into things as detailed as
7 respiratory protection? I mean, because if somebody's in a
8 space suit with fresh air even working in a confined space
9 around the highest, you know, manganese content rods, you
10 wouldn't expect that neurologist to go into those kinds of
11 details; would you?

12 A. It wouldn't be unreasonable to ask those
13 questions. But the hypothetical that you've posed is not
14 realistic. It's very hard to go into a confined space in a
15 self-contained breathing apparatus because it's confined
16 space, and you've got all this equipment on your back and
17 very hard to move around.

18 Q. Really? You couldn't get inside a closet with
19 one of those?

20 A. It would be very hard to move around and do a
21 welding job in a SCBA in a closet.

22 Q. What about --

23 A. It would be --

24 Q. -- if --

25 THE REPORTER: Wait --

1 A. If we're going to talk about confined-space
2 welding, we're back to a situation where an employer has a
3 duty to ensure that the job can be done safely, and I would
4 not think that wearing a SCBA when in a confined space
5 would necessarily meet --

6 Q. We're getting off track --

7 A. -- with those conditions.

8 Q. (BY MR. LUBEL) I'm just trying to find out if
9 you expect a neurologist to assess the conditions by which
10 the exposure took place and whether any respiratory
11 protection was used at all in evaluating whether it's a
12 manganese induced Parkinsonism or not. That's all I'm
13 trying to figure out.

14 A. I think the neurologist should get an
15 occupational history and should seek to identify
16 occupations in which it is known that people can be
17 overexposed to manganese. Now, welding -- as it is done in
18 virtually every setting that I've ever seen and as it is
19 done in all of the epidemiology studies that have been
20 conducted -- does not fulfill those criteria. It's not
21 known to put people at increased risk. I would not expect
22 a neurologist to devote a lot of effort to exploring that
23 occupation because that occupation is of no relevance to
24 the risk of manganese.

25 Q. Okay. So, all the neurologist needs to do is

1 say, "Although this constellation of symptoms could be
2 attributable to manganese, it doesn't apply in this
3 particular circumstance because the guy or the lady is a
4 welder." That's all they need to do, right?

5 A. I'm not sure we've established what constellation
6 of symptoms we're talking about. Are we just talking about
7 Parkinsonism in general, the big umbrella term?

8 Q. The symptoms you say are indicative of manganism.
9 You've told us what they are.

10 A. Okay. So, we're talking about that particular
11 constellation of symptoms and signs?

12 Q. Correct. Let's say those symptoms and signs
13 exist, not all of them because you said not all of them had
14 to exist but a number, enough of them to where if the
15 manganese exposure was high enough, you'd link it.

16 So, my question is: If the constellation of symptoms
17 could be indicative of a manganese induced disease and the
18 neurologist says, "What did you do for a living" and the
19 man or lady said, "I was a welder," you would not expect a
20 neurologist to go any further. That's what I'm trying to
21 get you to say, if that's what you believe.

22 A. If a patient presented to me and had that
23 constellation of symptoms and signs that fit very well with
24 manganism, I would explore in detail their current and past
25 occupations for a number of years, not only to know the job

1 but to know the materials they worked with, the tasks they
2 performed, and the circumstances under which they performed
3 them. And the reason is if you have a diagnosis that
4 strongly suggests an etiology due to a chemical agent, then
5 the level of inquiry about could you have gotten exposed to
6 that agent should be in greater depth. That's appropriate
7 to do.

8 But if you've got a constellation of symptoms and
9 signs that is not tightly focused on that disorder, say
10 manganism, then the inquiry into potential exposures or
11 past jobs would typically not be with as great precision
12 and with as great depth. I mean, I spend my career trying
13 to explore occupational histories with patients to uncover
14 what their exposures have been for that same purpose.

15 Q. And what I'm trying to find out, Dr. Garabrant,
16 is when they perform this in-depth analysis when there is
17 the constellation of symptoms that could be reflective or
18 indicative or consistent -- or whatever term you want to
19 use -- to manganese exposure, do you believe that
20 information is supposed to be recorded in the medical
21 records?

22 A. There's no one answer that fits all situations.
23 The answer has to do with how tightly that constellation of
24 symptoms and signs fits the syndrome that is known to
25 result from toxicity due to that agent. If this is a -- if

1 this is a constellation of symptoms and signs that -- let's
2 not do it as a hypothetical. Let's do it as manganism. If
3 you see a patient and they fit manganism perfectly and --

4 Q. Cock-walk and all?

5 A. Cock-walk and all.

6 Q. Okay.

7 A. You should spend considerable effort to explore
8 their exposure history not only in the occupational setting
9 but drinking water and pesticide spraying and you name it.
10 You really --

11 Q. I'm not disagreeing with you --

12 A. -- should go at it with a great deal of interest.

13 In my practice --

14 Q. Wait a second, Doctor.

15 A. -- commonly --

16 Q. We can end this quickly. I just want to find out
17 if that analysis that you're giving us is supposed to be
18 recorded in the medical records of the doctor. That's all
19 I'm asking.

20 A. If you ask the questions, you write down the
21 answers. The issue is -- perhaps I'm not understanding --

22 Q. I'm asking you a simple question. Okay?

23 MR. BISSELL: He just answered it.

24 A. Yeah. If you ask the questions, you write them
25 down. You write down the answers.

1 Q. (BY MR. LUBEL) That's what I'm trying to get.

2 A. I didn't understand that.

3 Q. Okay. I'm not trying to be horsey or
4 disrespectful. I am just trying to get us out of here
5 sooner, including you, since you're going back to another
6 state.

7 (Garabrant Exhibit Nos. 22 and 23 were marked)

8 Q. (BY MR. LUBEL) Exhibit 22 comes out of one of
9 your books. Mr. Bissell was kind enough to make a copy of
10 it, "Standard Occupational Classification Manual, 2000"; is
11 that correct?

12 A. Yes.

13 Q. All right. And I have taken Exhibit 23, which is
14 six three-ring binders of the remaining articles that you
15 brought with you today, correct?

16 A. Yes.

17 Q. And then I've left behind you -- because I'm not
18 going to mark them --

19 A. Wait a minute. You've also got a seventh binder
20 marked as a separate exhibit.

21 Q. But it's already marked. I'm saying I marked
22 Exhibit 23 as the remainder of the articles that you
23 brought, right?

24 A. Yes.

25 Q. All right. Now, then, behind you is two boxes of

1 medical records and information that's related specifically
2 to Mrs. Godwin, correct?

3 A. Yes.

4 Q. And you understand that in this case at least so
5 far you're not going to offer any specific opinions to
6 Mrs. Godwin short of you don't believe that her disease
7 could have been caused by welding exposure, correct? Isn't
8 that what you told me earlier?

9 A. Correct. Except that I don't know what you mean
10 "short of" other than --

11 Q. In other words, you've not been asked to assess
12 her diagnosis?

13 A. That's correct.

14 Q. And you hadn't done so, correct?

15 A. That's correct.

16 Q. You've not examined her?

17 A. That's correct.

18 Q. You've not reviewed her records in a light to
19 determine whether she's, in fact, correctly diagnosed as
20 PD, Parkinsonism, manganism, or anything else, correct?

21 A. I have reviewed her records to understand how her
22 diagnosis was reached.

23 MR. LUBEL: John, can you just stipulate
24 that that's not his role in the case?

25 MR. BISSELL: Yeah. I think so. I mean, if

1 I understand what "that" is. I mean, he is not offered as
2 an expert to make or refute a particular diagnosis in her
3 case.

4 MR. LUBEL: Let me tell you what I'm doing.
5 When I look at the disclosure responses on David Garabrant,
6 there is nothing in there that leads anybody to believe --
7 including me even with my pea brain -- that he's going to
8 make specific opinions on Ms. Godwin?

9 MR. BISSELL: I don't think he is. The
10 question is so broad that it might rule out something that
11 I'm just not clear -- I think we understand. He is not
12 here and will not be tendered to offer specific Godwin
13 opinions.

14 MR. HENDERSON: Other than what he already
15 said?

16 MR. BISSELL: Other than what you have
17 talked about today in that he knows she was a welder. He
18 knows she welded in the Navy; and, you know, he has
19 opinions based on what the literature shows and what he
20 believes about capacity of certain exposures to cause
21 disease. Now, she fits in some of that in there but he's
22 not working from her out. He's working from what he knows
23 and as it may apply to her. He's not --

24 MR. LUBEL: But he's really not working on
25 her. He's really --

1 MR. BISSELL: Right.

2 MR. LUBEL: -- going to express what the
3 study shows, and then y'all can argue about where she fits
4 is what I'm --

5 MR. BISSELL: I think that's -- doesn't that
6 sound fair to you?

7 THE WITNESS: Let me put it this way: It is
8 my opinion based on reading her records that she does not
9 have manganism.

10 MR. LUBEL: See, now, we're -- see, that's
11 the kind of stuff that I would expect to see in the
12 disclosure. Y'all go talk.

13 MR. BISSELL: Let's talk a short break.

14 (Recess from 6:30 p.m. to 6:34 p.m.)

15 (Garabrant Exhibit No. 24 was marked)

16 Q. (BY MR. LUBEL) Can we agree that the opinions
17 that you offer as you understand them today to be limited
18 to the disclosure that was made by your lawyers that I've
19 marked as Exhibit 24?

20 A. Yes.

21 Q. Despite the fact that it's getting kind of late
22 and it's hot in here, have I been courteous to you?

23 A. Yes.

24 MR. LUBEL: Thank you for your time.

25 MR. BISSELL: We are going to reserve our

1 questions until the time of trial. Specifically we will
2 offer at trial evidence relating to Dr. Racette's study
3 related to a comparison of welders in Alabama to a Copiah
4 County, Mississippi, control group.

5 (Deposition concluded at 6:36 p.m.)

CHANGES AND SIGNATURE

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1 I, DAVID H. GARABRANT, MD, have read the foregoing
2 deposition and hereby affix my signature that same is true
3 and correct, except as noted above.
4

5 _____
DAVID H. GARABRANT, MD

6
7 THE STATE OF _____)
8 COUNTY OF _____)
9

Before me, _____, on this day personally
10 appeared DAVID H. GARABRANT, MD, known to me to be the
person whose name is subscribed to the foregoing instrument
11 and acknowledged to me that they executed the same for the
purposes and consideration therein expressed.

12
Given under my hand and seal of office this ____ day
13 of _____, 2006.
14

15 _____
NOTARY PUBLIC IN AND FOR THE STATE OF _____
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1 NO. 03-CV-0801
2 RICK MONTALBANO, ET AL. * IN THE DISTRICT COURT OF
*
3 VS. * GALVESTON COUNTY, TEXAS
*
4 THE LINCOLN ELECTRIC COMPANY, *
ET AL. * 56TH JUDICIAL DISTRICT

5
6 REPORTER'S CERTIFICATION
ORAL DEPOSITION OF DAVID H. GARABRANT, MD
OCTOBER 19, 2006

7
8 I, Suzi Gladney, a Certified Shorthand Reporter in and
for the State of Texas, hereby certify to the following:

9 That the witness, DAVID H. GARABRANT, MD, was duly
sworn by the officer and that the transcript of the oral
10 deposition is a true record of the testimony given by the
witness;

11
12 That the deposition transcript was submitted on
_____ to the witness or to the attorney for the
witness for examination, signature, and return to me by
13 _____, 2006;

14 That the amount of time used by each party at the
deposition is as follows:

15 Mr. Lubel - 4 hours, 47 minutes

16
17 That pursuant to information given to the deposition
officer at the time said testimony was taken, the following
includes all parties of record:

18 FOR THE PLAINTIFFS:

19 Mr. Lance H. Lubel
Mr. J. Robert Black
20 HEARD, ROBINS, CLOUD & LUBEL, LLP
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24
25

1 FOR THE DEFENDANTS AIR LIQUIDE AMERICA, LP, AIRGAS GULF
2 STATES, INC., CBS CORPORATION, F/K/A VIACOM, INC.,
3 SUCCESSOR BY MERGER TO CBS CORPORATION F/K/A
4 WESTINGHOUSE ELECTRIC CORPORATION, EUTECTIC
5 CORPORATION, HOBART BROTHERS COMPANY, MATHESON
6 TRI-GAS, INC., PRAXAIR, INC., THE BOC GROUP, INC.,
7 F/K/A AIRCO, INC., THE ESAB GROUP, INC., TDY
8 INDUSTRIES, INC., THE LINCOLN ELECTRIC COMPANY,
9 SANDVIK, INC., UNION CARBIDE CORPORATION, AND UNION
10 CARBIDE CHEMICALS AND PLASTICS COMPANY, INC.:

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16 FOR THE DEFENDANTS ITW AND MILLER ELECTRIC COMPANY, A/K/A
17 MILLER ELECTRIC MANUFACTURING CO.:

18 Mr. John R. Henderson
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20 2001 Ross Avenue, Suite 2000
21 Dallas, TX 75201-6929
22 Tel: (214) 999-6109

23 I further certify that I am neither counsel for,
24 related to, nor employed by any of the parties or attorneys
25 in the action in which this proceeding was taken, and
further that I am not financially or otherwise interested
in the outcome of the action.

Further certification requirements pursuant to
Rule 203 of TRCP will be certified to after they have
occurred.

Certified to by me on this 21st day of October, 2006.

SUZI GLADNEY, CSR, RPR
Texas CSR No. 6857
Expiration: 12/31/2006
RPR Certification No. 853095
Expiration: 9/30/2009
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REPORTER'S FURTHER CERTIFICATION UNDER RULE 203 TRCP
TO THE ORAL DEPOSITION OF DAVID H. GARABRANT, MD
TAKEN ON OCTOBER 19, 2006

The original deposition was/was not returned to the
deposition officer on _____;

If returned, the attached Changes and Signature page
contains any changes and the reasons therefor;

If returned, the original deposition was delivered to
Mr. John G. Bissell, Custodial Attorney;

That \$_____ is the deposition officer's charges to
the DEFENDANTS for preparing the original deposition
transcript and any copies of exhibits;

That the deposition was delivered in accordance with
Rule 203.3, and that a copy of this certificate was served
on all parties shown herein on and filed with the Clerk.

Certified to by me this ____ day of _____, 2006.

SUZI GLADNEY, CSR, RPR
TEXAS CSR No. 6857
Expiration: 12/31/2006
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Expiration: 9/30/2009
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