NO. 03-CV-0801

RICK MONTALBANO, ET AL.	*	IN THE DISTRICT COURT OF
VS.	*	GALVESTON COUNTY, TEXAS
THE LINCOLN ELECTRIC COMPANY, ET AL.	*	56TH JUDICIAL DISTRICT

ORAL DEPOSITION OF

DAVID H. GARABRANT, MD

OCTOBER 19, 2006

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

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INDEX

EXHIBITS

NO.	DESCRIPTION	PAGE
1	Cross-notice to take oral deposition	5
2	Unified PD rating scale	21
3	Hoehn & Yahr staging of PD	21
4	Article entitled Basal Ganglia Neurotoxins	33
5	Drawing of respiratory region	41
6	1979 article on the effects of welding on health	76
7	List of studies	87
8	Binder of manganese critical studies	88
9	Written formula example	93
10	Characterization of arc welding fume	106
11	Billing invoices from Dr. Garabrant	111
12	Curriculum vitae of Dr. Garabrant	111

NO.	DESCRIPTION	PAGE
13	Declaration from MDL products liability litigation	111
14	2004 report by Dr. Garabrant	111
15	Reference list	111
16	Copy of deposition given by Dr. Garabrant dated 1/14/05	111
17	Report concerning Dr. Sethi	111
18	Current curriculum vitae of Dr. Garabrant	113
19	Handwritten and typed notes of Dr. Garabrant	117
20	Cross-notice to take oral deposition	118
21	ICD codes	135
22	Excerpt from the 2000 Standard of Occupational Classification Manual	168
23	Six three-ring binders of articles provided by Dr. Garabrant	168
24	Case disclosure	171

(Garabrant Exhibit No. 1 was marked)

DAVID H. GARABRANT, MD,

having been first duly sworn, testified as follows:

EXAMINATION

BY MR. LUBEL:

Q. Please state your full name.

A. David Hay Garabrant.

Q. What is your profession?

A. I'm a physician.

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

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

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

A. I am a physician and an epidemiologist.

Q. What state do you live in?

A. Michigan.

Q. What city?
A. Ann Arbor.
Q. How long have you lived there?
A. Almost 18 years.
Q. How long have you been a medical doctor?
A. I graduated medical school in 1976.
Q. Who hired you in this case?
A. I believe it was Ms. Haley McIntosh at O'Melveny & Myers.
Q. And where is she located?
A. Los Angeles.
Q. And how did you know her?
A. I had worked with her on other welding cases.
Q. And when were you first consulted with regarding any welding case?
A. I'm not sure I know exactly. I would estimate 2002.
Q. And have you pretty much on a regular basis worked on various welding related matters or cases since then?
A. I have at various times. I wouldn't say regularly, but it comes and goes.
Q. And here's the point that I'm driving at and the question: Would it be a fair statement that since you started working on the welding related litigation four or

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

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

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

A. Yes.

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

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

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

A. I was trying to finish my previous answer.

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

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

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

MR. LUBEL: I hear you.

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

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

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

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

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

Q. Health effects.

A. Oh. I have some familiarity with it.

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

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

Q. Why not?

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

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

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

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

A. A systematic review, yes.

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

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

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

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

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

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

A. That's correct.

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

A. That's correct.

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

A. Correct.

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

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

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

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

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

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

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

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

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

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

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

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

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

Q. What are the clinical manifestations of Parkinsonism?

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

Q. Are those the most common?

A. I believe so.

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

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

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

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

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

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

A. Yes.

Q. Is that a sign or a symptom?

A. That's a sign.

Q. It's something a doctor would observe?

A. Yes.

Q. What is it?

A. Slowness of movement.

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

A. Based upon experience.

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

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

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

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

Q. And what are the tests that are commonly

available to a neurologist to determine whether there is bradykinesia?

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

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

A. Yes.

Q. What is that?

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

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

A. Or an internist, for example.

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

Q. What was the second one?

A. A pull test.

Q. What is that?

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

Q. What's the Romberg test?

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

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

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

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

A. Yes.

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

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

Q. What does that mean?

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

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

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

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

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

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

Q. What do you call that?

MR. LUBEL: Recalcitrance.

(Laughter)

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

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

A. I believe so.

Q. There's others, correct?

A. I believe so.

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

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

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

Q. Is that also called levodopa?

A. Yes.

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

A. "L" stands for levo.

Q. It does?

A. Yes, sir.

Q. And what is levodopa?

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

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

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

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

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

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

A. I don't understand the question.
Q. What other features are you referring to other than you've told us about one of them being a response to levodopa?
A. I'm still not understanding. Features of what?
Q. The differences between PD and Parkinsonism.
A. Well, that's the clearest one. Other forms of Parkinsonism have different signs and symptoms that overlap with Parkinson's disease, but a good neurologist can differentiate them.
Q. How so?
A. By the history and physical exam as to what the findings -- what the pattern of findings and symptoms and signs are.
Q. And what would one expect to see in the history and physical exam that would be different between a PD and a Parkinsonism?
A. I can't answer that the way you've asked it. PD is a form of Parkinsonism.
Q. Well, I thought you told us that a good physician can pick up the difference based upon a physical exam and history. Did I hear you wrong?
A. Well, you've got -- no, you heard me right; but that's only half of my answer.
Q. What was the rest of it?

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

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

A. I've given you -- yeah.

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

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

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

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

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

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

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

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

A. I have heard of that.

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

A. Yes.

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

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

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

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

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

Q. Grab them if you don't mind.

A. Sure.

Q. Thanks.

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

A. I have them in front of me.

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

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

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

(Discussion was held off the record)

(Garabrant Exhibit Nos. 2 and 3 were marked)

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

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

A. Yes.

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

A. No.

Q. All right. Let me get those back real quick.

What is the unified Parkinson disease rating scale?

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

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

A. I don't recall any.

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

A. I'm not easily surprised.

Q. You see anything wrong with it?

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

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

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

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

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

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

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

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

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

quantify them.

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

A. Yes.

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

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

It includes signs and symptoms.

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

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

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

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

MR. LUBEL: That will be denied.

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

THE WITNESS: Could you repeat the question?

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

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

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

A. Yes.

Q. What do you call that form?

A. Manganism.

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

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

the question as asked.

Q. Let's do it this way.

A. Well, let me finish.

Q. Go ahead.

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

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

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

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

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

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

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

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

Q. Trauma?

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

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

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

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

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

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

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

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

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

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

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

A. Yes.
Q. Do you know what that list is, what that looks like?
A. I don't understand the question.
Q. What the other possible causes are.
A. Well, I've listed two of them. That's all I can think of from memory.
Q. And that was trauma and cerebrovascular disease?
A. Yes.
Q. Commonly known as stroke?
A. Could be a stroke, could be vascular insufficiency. I suppose it could also be vasculitis. And so, there -- I do not recall from memory what the differential diagnosis of causes of Parkinsonism is. So -- but there would be a list of things to rule out.
Q. From your experience do you know whether or not there's any type of scan that can be done that will objectively tell a neurologist whether or not the patient has PD? And by that I mean like MRIs, CTs, PET scans, SPECT scans, anything like that.
A. Well, there are scans that will allow a neurologist to get an image of the basal ganglia and to evaluate the size and radiodensity of the various ganglia which assists in defining whether, for example, the ganglia are the right size or they have adequate blood supply,

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

Q. In and of themself?

A. In and of themselves.

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

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

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

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

Q. The neurons die?

A. Yes, in the substantia nigra pars compacta.

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

A. Depends how many neurons die.

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

A. I don't know.

Q. Does anybody?

A. I don't know.

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

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

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

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

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

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

Q. Right.

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

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

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

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

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

A. Not to my knowledge.

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

A. The globus pallidus.

Q. Are there any others?

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

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

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

MR. LUBEL: We can go off the record.

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

(Garabrant Exhibit No. 4 was marked)

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

A. That's correct.

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

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

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

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

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

A. The question is too vague to answer.
Q. Let me ask it better.
A. You can find manganese in other basal ganglia besides the globus pallidus, and there's plenty of literature that will support that. But as to whether it's actually doing damage in those other structures or is simply present is a much more subtle question.
Q. All right.
A. And we'd have to look at that literature before I could answer that.
Q. Are you prepared to answer that today?
A. I did not prepare on that question today. I prepared on the epidemiology.
Q. All right. So, you're not prepared to discuss it today, correct?
A. That's correct.
MR. BISSELL: Other than what he's told you.
MR. LUBEL: Yeah, I can understand what you're saying.
Q. (BY MR. LUBEL) But you would agree that there is a substantial amount of evidence that manganese can accumulate in other areas of the brain than the globus pallidus, correct?
A. I would agree that manganese can be found. I'm not sure that it's fair to characterize it as accumulating.

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

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

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

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

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

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

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

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

A. Typically the globus pallidus.

Q. Any other areas?

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

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

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

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

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

Q. What do they assume?

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

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

MR. BISSELL: Object to form.

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

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

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

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

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

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

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

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

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

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

Q. How does it get into the blood?

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

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

Q. You said the alveolar space?

A. Yes.

Q. What is that?
A. The air in the alveoli.
Q. What is the alveoli?
A. It's the basic unit of the gas transfer region of the lung in which the transfer of oxygen from the air into the blood and carbon dioxide from the blood back into the air occurs.
Q. So, it's believed that it's that particular area in the lung to where this manganese transport takes place?
A. Well, it's in the respiratory region of the lung which includes all of the structures downstream from the terminal bronchioles.
Q. Okay.
A. Including the alveoli.
Q. All right. So, the actual transport of manganese from the lung into the bloodstream occurs at numerous areas including the alveoli?
MR. BISSELL: Objection; form.
Are you including diffusion? I mean, he said it could be diffusion or transport. You're not -- you don't mean transport necessarily by any kind of mediation?
MR. LUBEL: Correct. Just transport.
A. Well, let's try and break the question down. Is your question directed at understanding where in the lung anatomically this occurs; or is it directed at

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

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

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

Q. Okay.

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

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

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

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

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

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

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

(Garabrant Exhibit No. 5 was marked)

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

A. Yes.

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

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

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

MR. BISSELL: Objection; form.

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

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

A. (Witness complies)

MR. BISSELL: He's got it labeled.

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

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

A. Yes.

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

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

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

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

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

A. Hold on. Let me finish explaining.

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

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

A. Yes.

Q. -- that's the respiratory region?

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

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

A. Right here (indicating).

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

A. That is the respiratory region.

Q. Where the red circle is?

A. Yes.

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

MR. BISSELL: Objection; form.

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

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

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

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

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

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

I'm asking.

A. Blood moves to every tissue in the body.

Q. Including the brain?

A. Yes.

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

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

Q. Has anybody studied that?

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

Q. What do you call that area?

A. An unfamiliar area.

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

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

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

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

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

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

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

Q. How did it get --

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

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

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

have any increase of manganese in their brain.

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

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

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

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

Q. Do you agree --

A. In the world's literature.

Q. Do you agree?

A. Do I agree with what?

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

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

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

A. I believe those two case reports suggest that

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

Q. Do you believe it?

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

Q. So, you agree it can happen?

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

Q. Under what circumstances?

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

Q. What were the levels?

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

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

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

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

working in?

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

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

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

Q. Are there any from other countries?

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

Q. How is that? Why not?

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

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

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

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

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

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

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

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

A. More than that.

Q. What else?

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

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

A. You want it for that plus more.

Q. What else do you want it for?

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

Q. Can't those be measured?

A. Yes, they can be measured.

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

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

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

solubilized.

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

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

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

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

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

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

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

A. That is correct.

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

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

Q. What are the features of that syndrome?

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

Q. Which number is that, Doctor?

A. No. 4.

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

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

A. Akinetic-rigid Parkinsonian syndrome.

Q. Okay. Go ahead.

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

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

I think that describes it.

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

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

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

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

A. I believe they can.

Q. Can a person have manganism without dystonia?

A. I don't think so.

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

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

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

A. I believe so.

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

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

Q. Is any tremor characteristic of manganism?

A. Postural tremor.

Q. What is that?

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

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

A. (Indicating)

Q. Your hands can shake?

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

Q. Oh.

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

Q. Okay. What is an intention tremor?

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

Q. Is an intention tremor consistent with manganism?

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

characterized by postural tremor.

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

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

Q. How does a doctor recognize rigidity?

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

Q. Are they tight?

A. They resist.

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

A. Yes, based on experience.

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

A. I believe so.

Q. But not necessarily required?

A. Well, not diagnostic of.

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

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

Q. What is bradykinesia?

A. Slow movement.

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

A. No.
Q. Are they consistent with a diagnosis of manganism?
A. I believe they've only been seen in one setting, and that is in manganese miners. So, they've been seen. It's been described. It's my belief that in other settings, they have not been seen.
Q. Do you know whether depression is characteristic of manganism?
A. I don't believe it's characteristic of manganism.
Q. Can it be caused by manganism?
A. I believe it is sometimes seen in the setting of manganism.
Q. Akinetic-rigid Parkinsonian syndrome, is that different than bradykinesia and rigidity?
A. It's quite similar.
Q. How would you characterize it or define it?
A. Akinesis is absence of movement. Bradykinesis is slowed movement.
Q. Can you have both at the same time?
A. Well, I think they reflect just different degrees of the same phenomenon.
Q. You were looking at Exhibit 4 to give us the signs and symptoms, correct?
A. Yes.

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

A. I don't see that it does.

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

A. I'm not aware that it is.

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

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

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

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

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

A. I have seen that.

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

A. I believe that would be synonymous with manganism.

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

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

thing.

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

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

Q. Right.

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

Q. Can Parkinsonism be synonymous with manganism?

A. I don't believe so.

Q. What's the difference between the two?

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

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

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

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

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

your --

Q. Correct.

A. -- question?

Q. Yes, sir.

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

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

Q. All right.

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

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

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

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

Q. You think that's what neurologists do?

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

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

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

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

A. What standard of care are you referring to?

Q. Standard of care applicable to physicians.

A. It would depend on the circumstances.

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

MR. BISSELL: Object to form.

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

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

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

Q. Let's talk about neurology.

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

Q. How about internal medicine?

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

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

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

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

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

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

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

A. To some extent.

MR. LUBEL: Take a short break?

MR. BISSELL: Yeah.

(Discussion was held off the record)

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

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

A. Yes.

Q. What do you call that system?

A. Circulation.

Q. The circulatory system?

A. Yeah.

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

or something to that effect?

A. Yes.

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

A. It dies.

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

A. I believe there is.

Q. Do we know where it is?

A. In the globus pallidus.

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

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

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

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

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

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

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

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

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

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

Q. Parkinsonian's syndrome. Sorry. Parkinsonian's syndrome.

A. Parkinsonism?

Q. For instance.

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

Q. Anything else?

A. I believe there's been characterization in the

manganese mining industry.

Q. Any other industries?

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

Q. How about the steel industry?

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

Q. How about foundries?

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

Q. Have you seen in it any foundries?

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

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

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

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

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

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

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

Q. Do you have those studies with you?

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

Q. Great. We need to find them.

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

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

A. What was the question?

(Requested question was read back)

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

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

A. (Nods head affirmatively)

Q. Is that a "yes"?

A. Yes.

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

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

body of literature.

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

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

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

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

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

A. That's correct.

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

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

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

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

Q. But you're not sure?

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

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

not the manganese is in a different form?

A. No.

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

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

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

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

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

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

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

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

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

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

Q. Of the total fume?

A. Of what?

Q. Of the total fume?

A. No, of the respirable fume.

Q. What do you mean by respirable?

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

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

A. No.

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

A. Yes.

Q. We're talking about something that's invisible to the naked eye, correct?
A. You can't see the individual particles. You could see evidence of fuming in the air. I don't understand the question the way you've asked it.
Q. The particle that you're saying is respirable, you can't see with the naked eye; can you?
A. You can't see an individual particle, no; but you can see evidence of fume in the air when the fume is made up of particles that are respirable.
Q. You can?
A. Yes, you can.
Q. Have you seen it before?
A. Yes.
Q. When?
A. I've seen welding done.
Q. Have you welded?
A. I have.
Q. Did you see the fume?
A. I did.
Q. Could you smell it when you welded?
A. I don't recall smelling it. I have been in welding environments where I could smell it.
Q. Were you exposed to it when you smelled it?
A. Why, yes.

Q. Have you ever tasted it before?
A. I can't say as I've ever tasted it, no.
Q. So, you don't know what it tastes like?
A. Well, I've been in many settings in which welding was being done right in front of me. I've never tasted it. So, I'd say yeah, it has no taste.
Q. So, when you're looking at this fume -- you say you can see if you're welding or if you've seen welding done -- how much of that visible fume is made up of these microscopic respirable particles that are invisible to the naked eye?
A. Let's clarify the question a little bit. I don't understand it the way you've asked the question. I don't understand what you mean by fume made up of microscopic particles that are invisible to the naked eye.
Q. You want me to ask it a different way?
A. Well, I'm not sure what you're actually asking. The fume is visible even though the individual particles in the fume are too small to be seen individually.
Q. And I'm asking you what percentage of the fume is made up of the invisible particles, the less than ten micron sized particles?
A. The majority of them, a high percentage.
Q. What does that mean, 50 percent, 70 percent?
A. Oh, for welding fume I don't know -- certainly

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

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

A. Yes.

Q. And where do they go?

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

Q. Settle on the ground?

A. Settle out on whatever horizontal surface there is.

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

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

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

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

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

Q. Now, is this --

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

Q. And you call it re-entrainment?

A. Yes.

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

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

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

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

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

A. To see the individual particles?

Q. Right.

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

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

A. Yes.

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

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

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

in the air.

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

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

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

(Garabrant Exhibit No. 6 was marked)

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

A. I have a tag on Page 29.

Q. Okay.

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

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

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

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

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

Q. So, is it authoritative or not?

A. Well --

MR. BISSELL: Object to form.

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

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

MR. BISSELL: Object to form.

A. It --

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

MR. BISSELL: Object to argument.

MR. LUBEL: Oh, okay.

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

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

Q. It doesn't?

A. It doesn't.

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

A. Yes, 27 years ago.

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

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

Q. By them?

A. Who is "them"?

Q. The American Welding Society.

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

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

A. I don't know.

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

A. I haven't seen what?

Q. Anything that the welding society did?

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

kept it catalogued in that manner.

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

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

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

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

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

MR. BISSELL: Yes, you are.

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

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

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

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

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

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

Q. ESAB?

A. I don't know.

MR. BISSELL: Yes.

A. Yes.

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

MR. BISSELL: Yes.

A. Yes.

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

A. I don't know.

Q. ITW?

A. I'm not aware of it.

Q. Illinois Tool Works?

A. I'm not aware of it.

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

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

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

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

Q. And there's been a bunch of companies, correct?
A. I believe I have worked on behalf of a number of companies that manufacture welding consumables.
Q. And when you got hired by these people, did you ever ask them or their lawyers, "Hey, have y'all got any studies on the potential neurologic effects from welding"?
A. I think I have discussed with the lawyers on numerous occasions all of the scientific literature regardless of who wrote it; and to the best of my knowledge, I have received whatever studies those attorneys were aware of.
Q. Okay. So, what I'm asking you is: For approximately 20 years after this 1979 version of effects on welding health, what did these welding companies do to try and come up with answers to questions posed by welding and potential neurological deficits?
A. Well, I think it would be fair to say that there was no evidence or reason for concern about Parkinsonism related to welding in the scientific literature adequate to have warranted that any studies be done. Every existing study had failed to show evidence of any problems, any neurologic problems whatsoever related to welding.
Q. So, the answer is they didn't do any studies in that 20 years, correct?
MR. BISSELL: Objection; form.

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

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

A. I've seen many studies.

Q. From the welding companies?

A. No, many studies in the scientific literature.

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

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

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

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

Q. No.

A. -- did they need to do it?

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

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

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

A. I mean --

Q. -- they --
A. -- as a scientist --
THE REPORTER: Wait, wait --
A. -- I do have opinions. That's what I spend my career doing.
Q. (BY MR. LUBEL) You've said it. My question is: Did they do any?
A. I'm not aware that they did any, which I regard as an appropriate response to the lack of evidence of a health concern.
Q. Did they ever do any?
A. Well, I know that they did studies to characterize the manganese content of welding fume and found that it was low, from which it was a reasonable conclusion that welders who worked with mild steel would not have been overexposed to manganese.
Q. Is that the only study they ever took part in?
A. It's the only one I recall as I sit here. To answer your question, there may have been others.
Q. How about Fryzek? How about Fryzek? Didn't they participate in some study, some guy you know from Michigan?
A. You asked me about studies prior to 2000 --
Q. And then I said open it up to ever.
A. Well, then I misunderstood your question. I'm sorry.

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

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

Q. Right.

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

Q. Welding defendants for Parkinson's like disease.

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

Q. Let me ask --

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

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

A. I do not understand the question.

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

welding and neurological issues ever?

A. Yes. Yes.

Q. What were the studies called?

A. What were they called?

Q. Right. How would you identify them?

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

Q. Any others?

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

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

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

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

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

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

give you an example.

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

MR. LUBEL: He --

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

MR. LUBEL: Okay. Anyway --

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

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

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

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

Q. How do you spell Clewell?

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

Q. Okay. Any others?

A. Well, I got to keep looking.

Q. Yes, sir.
A. It's going to take awhile.
(Recess from 3:08 p.m. to 3:44 p.m.)
(Garabrant Exhibit No. 7 was marked)
Q. (BY MR. LUBEL) Exhibit 7 is the list of studies that you could find with your proviso that you're not sure that this constitutes every study that fits the question I asked?
A. Let me try to answer. Exhibit 7 is a list of studies that address Parkinsonism and Parkinson's disease related to manganese exposure and/or welding that are funded by industries or industry groups.
Q. Okay. That you could locate?
A. That I could locate within about a 15-minute period.
Q. All right.
A. And there may be --
MR. BISSELL: Among the materials that you have with you.
A. Among the materials that I have with me today.
Q. (BY MR. LUBEL) You're not saying that the studies referenced on Exhibit 7 references every study funded by industry or a company related to manganese, true?
A. Oh, my goodness, no.
Q. All right. Fair enough. Okay. One of your

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

A. Yes.

Q. Is that something you put together?

A. Yes.

Q. You call it "manganese critical studies"?

A. Yes.

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

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

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

A. Sure.

(Garabrant Exhibit No. 8 was marked)

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

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

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

A. Epidemiological studies.

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

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

A. Correct.

Q. Exhibit 8 is epi studies, right?

A. Yes.

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

A. Yes.

Q. What is it?

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

Q. And what is it designed to tell somebody?

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

Q. If it existed, right?

A. If it, in fact, existed.

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

A. Yes.

Q. In your studies did you perform power calculations?

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

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

A. Yes.

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

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

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

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

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

A. No.

Q. How about after the data has been analyzed?

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

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

A. That is far more common than the converse.

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

A. Yes.

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

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

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

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

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

Q. Start with the planning phase.

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

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

A. Yes.

Q. What is your answer for that?

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

Q. Doesn't help you interpret the study?

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

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

A. No.

Q. Do they mean the same thing?

A. No.

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

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

Q. Why?

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

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

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

(Garabrant Exhibit No. 9 was marked)

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

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

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

A. And what is it that you measured?

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

A. Now I can interpret it.

Q. Talk to me.

MR. BISSELL: Can he mark on your exhibit?

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

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

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

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

Q. Okay. I'm sure that doesn't surprise you.

Do you understand what I'm trying to ask? Can you ask the question for me?

A. If you'll answer it.

Q. I can't. I'm not the scientist. You are.

You know what I'm trying to ask?

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

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

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

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

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

A. Yes.

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

Q. All right. Let me ask --

A. Because the answer it gave is relatively uninformative.

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

A. I would typically write that the relative risk

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

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

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

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

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

Q. Gotcha. All right.

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

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

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

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

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

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

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

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

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

A. (Witness complies)

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

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

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

A. I don't know.

Q. What is the log scale?

A. You take the --

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

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

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

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

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

A. Neither of those statements is correct.

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

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

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

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

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

Q. Okay. Let me ask you this --

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

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

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

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

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

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

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

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

Q. What should it say?

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

A. Can you ask the question again?

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

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

MR. BISSELL: Take a short break?

MR. LUBEL: Yeah.

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

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

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

Q. Thank you. That would be helpful.

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

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

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

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

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

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

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

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

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

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

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

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

A. No, I haven't done it.

Q. Okay.

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

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

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

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

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

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

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

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

use that for?

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

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

A. Yes.

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

(Garabrant Exhibit No. 10 was marked)

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

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

Q. Why?
A. Because it shows that the welding fume contains about 4 to 5 percent manganese when those rods are used.
Q. The fume or the electrode?
A. Huh?
Q. The fume?
A. It's the fume.
Q. Have you seen any characterization of the 7018 MIL-SPEC?
A. I do not recall it.
Q. Do you know what the portion of the manganese in the fume of that particular rod is?
A. I am not an expert on the differences among the rods. If the 7018 MIL-SPEC is the same as the 7018, I would assume they have similar manganese content; but I do not know that there's a difference between the MIL-SPEC and the non MIL-SPEC rods.
Q. Do you know whether a welder using either the 6010 or the 7018 can be overexposed in conditions such as a confined space?
MR. BISSELL: To what?
MR. LUBEL: To welding fumes.
A. Well, it would depend on many factors. Confined-space welding is an important issue for industrial hygienists to ensure that welders are safe when they work.

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

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

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

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

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

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

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

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

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

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

Q. Do you know the answer?

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

Q. What tools are you referring to?

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

Q. They can measure manganese, can they not?

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

the --

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

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

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

THE WITNESS: I'll do my best.

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

A. Could you clarify that question?

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

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

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

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

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

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

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

MR. BISSELL: Are you asking about welding fume?

MR. LUBEL: Yes, sir.

MR. BISSELL: Do you know the answer?

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

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

(Brief pause)

(Garabrant Exhibit Nos. 11 through 17 were marked)

Q. (BY MR. LUBEL) Sir?

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

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

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

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

Q. You can look it up later?

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

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

A. Well, thank you.

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

A. Appears to be and I think it is copies of all of my billing invoices related to welding litigation.

Q. I take it it's not through today; is it?

A. No.

Q. It's through what?
A. Appears the most recent one was August 27. Most recent invoice was August 27th, 2006, for a deposition taken on May 17th, 2006.
Q. Have you done any work since August 27th?
A. Yes.
MR. BISSELL: Actually since May. That only goes through May, I think; isn't that right?
THE WITNESS: Yes.
Q. (BY MR. LUBEL) Can you attach to that exhibit any invoices you have between May and today?
A. I don't know that I have created any invoices. Well, I mean, this invoice was created in August covering services in May. I don't know whether I've created any invoices since August.
Q. Okay. Exhibit 12 is a copy of your current resumé?
A. That one is out of date. I brought a new one.
Q. All right. I'm going to mark your current one as Exhibit 18, correct?
(Garabrant Exhibit No. 18 was marked)
Q. (BY MR. LUBEL) What has changed of any significance between Exhibit 12 and Exhibit 18?
A. I added a bunch of papers and presentations.
Q. Anything specific to the topics related to the

lawsuit?

A. No.

Q. Can you identify Exhibit 13 for us?

A. I believe this is a declaration I wrote in the MDL welding rod products liability litigation.

Q. Is it a sworn statement?

A. I think it's a declaration and --

Q. Did you swear to it?

A. I did, yes.

Q. Okay.

A. And I wrote it in 2004.

Q. Exhibit 14, can you identify that?

A. That's a report I wrote in the MDL welding products liability litigation, and I wrote that in 2004.

Q. All right. Can you identify Exhibit 15?

A. This is a reference list, and I'm not sure when I created this. It contains 263 references, and I believe these are the references that I have brought with me today. There may be others I have brought with me today that are not on this list simply because those others are recently published.

Q. I assume that that exhibit includes the literature and articles that you're relying upon in the case or the litigation?

A. Exhibit --

Q. 15.

A. -- 15 includes articles I'm relying on in this case. There are additional articles that I'm relying upon that are not listed there but which are in the book that you have marked as Exhibit 8.

Q. I take it that between Exhibit 15 and Exhibit 8 and possibly other notebooks that we're going to talk about later today, that would comprise the literature that you've reviewed and that you're relying upon in the case?

A. Yes.

Q. Unless you find something new?

A. That's correct.

Q. Between now and trial, right?

A. Yes.

Q. Okay. Exhibit 16 appears to be a transcript of a deposition you gave in the MDL, and it looks like it got an attachment of something in the Elam case?

MR. BISSELL: That may be a mistake. I'm not sure we intended to attach --

MR. LUBEL: I just didn't want to tell him it was the MDL and then have it -- you know, because it's got Elam attached it to.

MR. BISSELL: -- asserted on my behalf. I'm sure we shouldn't have attached something that wasn't responsive that we just put in there.

MR. LUBEL: Thanks.

MR. BISSELL: Sure.

A. Exhibit 16 appears to be my deposition taken January 14, 2005, in the MDL welding rod products liability litigation. It appears to have attached to it what I think is trial testimony from the Elam case, October 22nd in 2003.

Q. (BY MR. LUBEL) Okay. Doctor?

A. Yes.

Q. Counsel for the defendants has represented to me that he did not believe that you've been asked to offer any specific causation opinions as to Mrs. Godwin. Is that a true statement?

A. Well, I've been asked to answer or to give opinions regarding general causation as to whether welding causes Parkinsonism and as to whether welding causes Parkinson's disease. In the instance of Catherine Godwin, it would be my opinion that insofar as it's not known to the world of science that welding causes either of those diseases, there is no basis for attributing her disease to her welding.

Q. But that's the only opinion you have that is specific to her, correct?

A. Yes.

Q. And 17 is just a copy of the report that the

defense lawyers supplied you regarding Dr. Sethi, correct?

A. That's correct.

(Garabrant Exhibit No. 19 was marked)

Q. (BY MR. LUBEL) And Exhibit 19, just generally can you describe what's in that stack of documents?

A. These are various handwritten and typed notes that I created at various times over the past three or four years. They're really separate documents. The first is titled "Criticism of Racette, 2005." These are criticisms that I wrote down I believe at the time of the Pressler trial, which was --

Q. '05.

A. Yeah, I was going to say about a year ago, I think. The second --

MR. BISSELL: January, '05.

A. The second is my notes on a number of case reports of Parkinson's disease in welders. The third is my notes titled "diagnosis of Parkinson's disease" and then at the bottom of that page titled "review of literature" which is actually identical to the previous document. It's a case reports relied upon by plaintiffs. The next document in that exhibit is titled "studies of incidence and prevalence of PD and Parkinsonism in men." And this is nothing more than a table giving background incidence and prevalence of PD and Parkinsonism in various populations.

A fifth document is attached to Exhibit 19 and probably doesn't belong there. It's a bunch of advertisements by plaintiffs' law firms to attract welders.

Q. (BY MR. LUBEL) And you're not using that to form a basis of any opinions in this case; are you?

A. No.

Q. You can take that out; and if you just put that thing -- what do you call it -- back on the exhibit, I'll stack it over here.

A. (Witness complies)

(Garabrant Exhibit No. 20 was marked)

Q. (BY MR. LUBEL) Thanks. And then Exhibit No. 20 you recognize as my notice of deposition of you along with the request-for-documents subpoena?

A. Yes.

Q. You brought your file with you today, correct?

A. I did.

Q. Okay. Who is Nathan Schachtman?

A. He's an attorney at McCarter & English in Philadelphia.

Q. Have you met with him on these cases?

A. I have met with him on a number of occasions. I don't know what you mean by "these cases."

Q. The welding litigation.

A. Some of the welding litigation, yes.

Q. Have you met with him in the context of any other litigation?
A. No.
Q. Just welding?
A. Yes.
Q. In other words, he hasn't asked you to consult in the area of asbestos?
A. No.
Q. Silica?
A. No.
Q. Have you done any work on behalf of any asbestos companies?
MR. HENDERSON: Objection; form.
A. It's not clear to me whether I have or have not. I have testified on behalf of defendants in cases in which it was alleged that gastrointestinal tract cancer was caused by asbestos. I don't know whether any of those would fit the rubric of asbestos companies.
Q. (BY MR. LUBEL) Okay.
A. They were typically end users of asbestos products.
Q. What companies were they, like GE?
A. I don't recall offhand. Armstrong World Industries. I think Exxon. I don't recall other names, but that's the typical setting.

Q. Have you had any other role in asbestos other than testifying as an expert in some of these cases? And let me be more specific: Have you been a consultant to any companies pertaining to asbestos issues outside of the litigation?

A. No.

Q. Have you performed any epidemiological studies pertaining to asbestos?

A. Yes. I published a couple of papers pertaining to asbestos.

Q. On behalf of the car companies?

A. I published a meta analysis of mesothelioma and lung cancer risks among auto repair workers and vehicle mechanics. I did not do that on behalf of the car companies. Some of my co-authors did.

Q. They just brought you in because of your expertise?

A. I was a co-investigator and co-author of that study. I don't know why they brought me in, but I was a contributor to the work.

Q. But your co-authors were hired by Big Three?

A. I believe that their efforts on that research were supported by payment from the Big Three auto makers.

Q. Who were they? Who were the co-authors?

A. From memory, Michael Goodman, Pat Hessel, Val

Craven, Jane Teta. I've probably forgotten a couple of people.

Q. Have you done any work for the tobacco industry?

A. No.

Q. Have you participated in any epidemiological studies that touched on relative risk or prevalence of incidence of any cancers that may be associated with smoking?

A. Yes.

Q. What studies were those?

A. I'd have to go through my CV. From memory we did a study of lung cancer among welders in which we looked at smoking and found that once we adjusted for smoking, there was no association between welding and lung cancer risk. They were stainless steel welders. So, it touched on smoking related lung cancer.

I know some of the studies I've done of gastrointestinal tract cancers, I believe we looked at the role of smoking, I think, for stomach cancer; and I know we looked at it for colon cancer. I know we've looked at it in pancreas cancer. In some number of studies we've looked at cancer risks associated with smoking.

Q. Who hired you to do the study regarding the relationship, if any, between lung cancer and welding?

A. Nobody hired us. We did that on our own time.

Q. When did you do that study?
A. Back in the early 1980s.
Q. Is that published?
A. Yes. It's on my CV.
Q. Who are the co-authors?
A. First author is Corky Hull, Hull, H-U-L-L.
Q. Any others?
A. John Peters. I'm not sure I recall who else. We could look quickly. It wouldn't be hard to find it. On Page 12 of my CV, No. 28. The title of the study is "Case-control study of lung cancer in Los Angeles County welders." First author, Hull, C. You want me to read the other authors?
Q. Let me just see them.
A. No. 28.
Q. Thanks. That was in 1989?
A. Yes.
Q. When did you start that study?
A. I don't recall that.
Q. Roughly? How many years before it was published?
A. Probably two years before that. Probably '87.
Q. You have the underlying data from it?
A. No.
Q. Who does?
A. I don't know at this point. It's been almost 20

years.

Q. Well, who was the last to have it?

A. Well, Corky Hull was a resident in occupational medicine at USC; and he actually did the study and wrote it up. He might have it. I don't.

Q. What function did you serve?

A. I was a faculty member. I think I was his adviser.

Q. So, did you actually perform the study; or did you just check it off?

A. No, I actually participated with him and supervised his work.

Q. Was it his thesis?

A. We didn't require a thesis. It was a research project he did.

Q. Did you look into any neurological effects at that time?

A. No. It was a case-control study of lung cancer. You cannot look at neurological effects in a case-control study of --

THE REPORTER: I'm sorry. I didn't hear what you said.

THE WITNESS: You cannot look at neurological effects in a case-control study of lung cancer.

Q. (BY MR. LUBEL) But you can do a case-control study on neurologic effects from welding; can't you?

A. Well, you could do theoretically a case-control study of neurologic disease in which you looked at past exposures to welding if you had the data.

Q. Take a long time?

A. Well, it depends on what it took to get the data. I mean, if the data were readily available, it wouldn't take a long time. If you had to create the data set, it could take a very long time.

Q. Have you performed any epidemiological studies on silica?

A. A number of the studies I have performed have included silica exposure as one of the factors we addressed. And typically in a case-control study we might have asked about silica exposure or might have looked at occupational histories and inferred which ones involved silica exposure. For example, we did a study titled "Adenocarcinoma of the stomach and exposure to occupational dust"; and we published that in 1988. To the best of my recollection, one of the dust types that we addressed in that was silica.

Q. You're aware that silica is a carcinogen to humans?

A. I'm aware that IARC has classified it as a

Category 1 agent.

Q. Do you agree with that?

A. I haven't reviewed that for today. I'd have to say I'm not in a position to give an opinion off the cuff.

Q. Okay. Do you agree that asbestos is a carcinogen?

A. Yes.

Q. How about benzene?

A. I believe benzene can cause acute myelogenous leukemia in humans.

Q. Have you done any epi studies on benzene?

A. Benzene has been one of the exposures that we have addressed in a number of studies I've done.

Q. Are they on your resumé?

A. Yes.

Q. Have you done any studies on benzene that have not been published?

A. We just finished a very large cohort mortality study for the United Auto Workers Ford Motor Company joint national committee in which we reconstructed exposures to about 90 different agents. Many of those agents were various petroleum distillate products, some of which likely contained low levels of benzene.

Q. Who funded the study?

A. The United Auto Workers Ford Motor Company

national joint committee.

Q. Who did you deal with from Ford Motor Company?

A. Gordon Reeve.

Q. Who did you deal with from United Auto Workers?

A. Frank Myra (phonetic).

Q. Has that study been published?

A. No.

Q. When will it be?

A. After we finish the dioxin study that we are deep in the middle of right now. It's filling my time and everyone else's time on my team.

Q. What are you doing the dioxin study for, pesticides?

A. No. Did you say who or what?

Q. For what purpose are you doing a dioxin study?

A. We're doing a dioxin study to identify exposure pathways in the environment to humans.

Q. Who's funding that one?

A. The Dow Chemical Company.

Q. Is dioxin a carcinogen?

A. One of the compounds, 2378 tetra --

THE REPORTER: Say that again. 23 what?

A. 2378-dibenzo tetrachlorodioxin is currently categorized by IARC as a Category 1 agent.

Q. (BY MR. LUBEL) And are you dealing with the

director of epidemiology at Dow Chemical?

A. I know the director of epidemiology at Dow Chemical, but he is not my contact person for the study.

Q. So, you know Collins?

A. Jim Collins?

Q. Yeah.

A. Yes.

Q. You know him from his Monsanto days or Ford days, or did you just meet him in his Dow Chemical days?

A. I know him through the Dow Chemical Company.

Q. So, who's your contact at Dow?

A. Dr. Michael Carson.

Q. What's his title, if you know? If you don't know, that's okay.

A. I don't know offhand.

Q. He's in the epi department?

A. No.

Q. What department is he in? Medical department?

A. Medical department.

Q. Are you aware that Dow Chemical bought Union Carbide?

A. I am aware of that.

Q. Have you done any work for Union Carbide before?

A. Not to my knowledge.

Q. Have you ever been over to the Dow Chemical

Company's medical department?

A. I have.

Q. Where is it located?

A. Are you talking about in Midland?

Q. Yes, sir.

A. Yes.

Q. That's the headquarters, is it not?

A. Your questions are not answerable. Which of "that" are we talking about and headquarters of what?

Q. Dow Chemical.

A. Which "that"?

Q. Dow Chemical has a headquarters where the medical department is, correct?

A. I believe the world headquarters of Dow Chemical Company is in Midland, Michigan. They have a medical department for the Midland plant in Midland, but it is not in the same location as the corporate headquarters.

Q. Do you know whether or not Dow Chemical has epidemiologists in house that work for them?

A. I believe they do.

Q. How about toxicologists?

A. I believe they do.

Q. Do you know whether Union Carbide had those specialties?

A. I do not know offhand.

Q. Have you done any other work for Dow Chemical other than the dioxin study that you're doing now?
A. I did a research study for chlorpyrifos.
Q. When did you do that?
A. 1999 to 2001 or 2002.
Q. Was that report published?
A. We published a number of papers from that study.
Q. When do you expect the dioxin work to be done and in a format that you can publish?
A. I've already presented 31 papers from that study. We presented those in August of this year; and we have another, oh, two years of work to finish analyzing the data.
Q. Have you ever done any consulting work for the American Petroleum Institute?
A. I don't believe so.
Q. Have you ever done any work for any oil company or chemical company in the context of a chemical exposure lawsuit?
A. I have been an expert witness on behalf of chemical companies in chemical exposure lawsuits.
Q. Involving what chemicals?
A. It's really hard to recall. I don't know offhand. I'd have to think for a while.
Q. How about benzene?

A. I have, yes.
Q. Toluene?
A. I've been an expert in lawsuits in which it was alleged that exposures to mixtures of many solvents had adverse health effects. I believe benzene and toluene have been on the list and many other solvents as well.
Q. Are these in California?
A. Some have been in California.
Q. Was it the IBM litigation?
A. No.
Q. Which litigation was it?
A. I was involved with the Lockheed litigation.
Q. On behalf of defendants?
A. On behalf of defendants.
Q. Have you ever performed any epidemiological studies or reviews that have not been published?
A. Yeah.
Q. A bunch of them?
A. Unfortunately, yes.
Q. A bunch of them?
Well, not everything gets published unfortunately. Sometimes you have findings that are -- rather than scientific they're called negative findings. And if you don't find any associations, it's hard to get them published. It's much easier to get positive findings --

THE REPORTER: Easier to get what?

THE WITNESS: Positive findings published.

Q. (BY MR. LUBEL) I take it that you're referring to studies that you've submitted for publication that were not accepted?

A. Well, I'm referring to studies that have been submitted for publication and not accepted and studies we never bothered to submit because we believed the journals would not be interested in them.

Q. What studies have you done that have not been submitted?

A. Oh, for example, we did a study of N-acetyl glucose amidase and renal function among a cohort of lead exposed workers hoping to find evidence that lead affected their glucose amidase excretion; and we didn't find it. So, it was a negative study, and we never got around to publishing it.

Q. Any others?

A. There must be a few others, yeah. I don't know from memory. These are the --

Q. Let me give you an example. Let me ask you some specifics. Have you done any studies that could refer or relate to welding that have not been published?

A. The only one I can think of is the current study, the cohort mortality study we've done for UAW Ford in which

welding was a very important exposure. That has not yet been published.

Q. Do you have a draft of it?

A. I have a technical report that is submitted to the sponsor.

Q. Are you prevented by your rules of ethics to tender that to us, or do you have to wait until the study is published?

A. I believe I'm not allowed to distribute that.

Q. I take it, then, that you won't offer any opinions of that at trial; is that correct?

A. That's correct.

Q. Any other studies that you've done that reflect in whole or in part welding or manganese that have not been published?

A. I don't think so.

Q. How about benzene?

A. Same answer.

Q. Asbestos?

A. Same answer.

Q. Silica?

A. Same.

Q. Vinyl chloride?

A. I don't think there's anything.

Q. Have you consulted with the American Welding

Society specifically?

A. No.

Q. Have you specifically consulted with any company other than Lincoln Electric?

A. I don't believe I've ever consulted with Lincoln Electric or any other welding company.

Q. Have you met any of their people?

A. I have met a couple of people from Lincoln Electric.

Q. Who is that?

A. I don't recall their names. It was simply at trial in a previous case.

Q. Mr. Brown?

A. I don't know.

Q. What do they have in the way of a medical department? Do you know?

A. I do not know.

Q. Have you seen any of their mortality or morbidity data?

A. No.

Q. Have you seen any mortality or morbidity data from Union Carbide?

A. No.

Q. From any of the other companies in the litigation, the welding litigation?

A. Not to my knowledge, no.
Q. For PD, as a person gets older, does their risk of PD go up?
A. Yes.
Q. When do you start to see that increased risk? What age?
A. Probably in their 30s.
Q. Most victims of PD over the age of 60?
A. Yes.
Q. Vast majority?
A. The majority.
Q. Do you see clear distinction or delineation once you get over 60, 65 years old as to the prevalence of PD? Is there a bright line of sorts?
A. No.
Q. How about Parkinsonism?
A. What's the question?
Q. Do you see any bright lines in the age of onset?
A. No.
Q. As a person gets older, does their risk go up for Parkinsonism?
A. I believe so.
Q. Similar to PD or different?
A. I haven't compared them.
Q. Within your materials have you brought with you

any animal studies?

MR. BISSELL: If you're going someplace else --

We've all taken a break, but you haven't had one.

MR. LUBEL: Take a break. Take a couple of minutes.

(Recess from 5:17 p.m. to 5:27 p.m.)

(Garabrant Exhibit No. 21 was marked)

Q. (BY MR. LUBEL) Exhibit 21, that's the ICD codes?

A. Yes.

Q. That's because a lot of the studies reference ICD codes?

A. Some of the studies reference ICD codes.

Q. You know a Dr. Brain from Harvard?

THE REPORTER: Dr. who?

Q. (BY MR. LUBEL) Is it Brain or Bain?

A. Joe Brain? I don't know a Dr. Bain from Harvard. I know a Dr. Joseph Brain from Harvard.

Q. He must be really smart for his parents to have named him Dr. Brain.

A. I don't think they gave him that name.

Q. Have you seen any of his work in the welding area?

A. No.

Q. Were you aware that he was doing some animal studies for the welding companies?
A. I was not.
Q. Have you relied on any animal studies for your opinions in these cases?
A. I don't believe so.
Q. Is there a place for animal studies in epidemiology or for an epidemiologist to interpret?
A. They say there is animal epidemiology done. It's not an area I work in.
Q. What's that area called?
A. Animal epidemiology.
Q. They have a speciality for that?
A. No. It's not a speciality, but people do study the patterns of disease in animals. For example, right now there's a tremendous amount of epidemiology being done in bird flocks to track bird flu worldwide.
Q. Okay. Latency, do you have any opinions on the appropriate latency from the studies for manganese induced Parkinsonism or is it person by person?
A. We're not talking -- that question is not talking about welding. I assume we're talking about in the industries in which there is high level of manganese exposure?
Q. Just for any manganese induced Parkinsonism.

A. Well, I have an opinion with respect to welding and Parkinsonism. There's no latency because there's no known association. You can't define latency until you know that there's a cause of association. Since there isn't one, latency is not an issue.

Q. So, in an individual that's been overexposed to manganese and welding, you're saying --

MR. BISSELL: Object to form.

Q. (BY MR. LUBEL) -- that individual's Parkinsonism could not have been caused by manganese from welding, true?

A. We've already discussed the two case reports in which I believe two welders using high manganese content rods in a confined space suffered from manganism.

Q. I'm just saying what is the latency that you require to connect up a person that's been exposed to manganese that has manganese induced Parkinsonism?

A. In those industries in which manganism has been observed in some instances, the latency is a matter of a few months.

Q. To what?

A. To years.

Q. How many years?

A. I don't know. I don't know what -- I don't know how many years. In fact, I don't know if it's been defined or not.

Q. Is it your opinion, Doctor, that there is no set of exposure circumstances in which a welder can get manganese induced Parkinsonism?

A. It's my opinion that there is a substantial body of epidemiological literature that has looked at welding in many, many settings and looked at the risks of Parkinsonism and has failed to find any evidence of an association.

Q. I'm just asking you: Is it your opinion that manganese exposure under no set of circumstances can lead to manganese induced Parkinsonism in a welding environment?

MR. BISSELL: Objection; asked and answered.

A. We've already discussed the two case reports of the welders in the rock crusher who I believe got manganese induced Parkinsonism as a consequence of welding in that setting.

Q. (BY MR. LUBEL) So, it can happen under the right exposure circumstances, correct?

A. I know of two instances in which I believe it happened.

Q. Can it happen under the right exposure circumstances?

A. I think I just answered that.

Q. "Yes" or "no"? Can it happen under the right exposure circumstances?

A. The question is so vague that I cannot answer it

because I don't know what you mean by "the right exposure circumstances." I have already answered that I believe there was a situation in which two welders working in a rock crusher using high manganese welding rods were overexposed to manganese and suffered from manganism.

Q. How much exposure would a welder have to be exposed to of manganese to have manganese induced Parkinsonism?

A. We do not know that for welders. We -- we don't.

Q. There's no threshold?

A. We do not know the answer for welding exposures.

Q. But is there a threshold for any manganese exposures?

A. In other industries we believe there is a threshold.

Q. What is it?

A. Well, it is somewhere above the OSHA PEL of 5 milligrams per cubic meter as an eight-hour time weighted average that one could have five days a week throughout a working career. It's believed that that PEL is adequate and protective of the people who would not get manganism. As to where the level is that actually induces manganism, it's clear from the studies in the mining industry which exposures were virtually uncontrolled that levels in the range of 100 milligrams per cubic meter are adequate to

cause manganism; and that's to pyrolusite. I don't know all of the data in between that setting and the PEL but somewhere above 5; and certainly by the time you get to 100 for a product exposure, that's enough to do it.

Q. Is there no evidence to suggest that exposures below 5 milligrams per cubic meter on a time weighted basis can cause manganese induced Parkinsonism?

A. When you say "no evidence to suggest," I don't know how to interpret that. I am not aware of any evidence that provides a sufficient basis for a conclusion that exposures less than 5 cause manganism.

Q. Why did the ACGIH lower their standard to .2 milligrams per cubic meter?

A. I don't believe they did.

Q. They didn't?

MR. BISSELL: I think they did. I think that's right.

A. I take it back. I stand corrected. They did adopt a value of .2 milligrams per cubic meter.

Q. (BY MR. LUBEL) Why did they do that if 5 milligrams per cubic meter is safe?

A. The ACGIH adopts values that are often more stringent than OSHA. After their expert panels reviewed the data, they -- I do not know the background of that decision. They may have chosen to lower it to achieve a

wider margin of exposure than OSHA felt was necessary.

Q. Do you routinely rely on the ACGIH in your evaluation of safe levels?

A. I routinely look at the ACGIH TLVs and BEIs in my work and --

Q. Are they typically thought of as being reliable in their field?

A. Well, the issue, I believe, is how much of a margin of protection one wants to build in between a recommended exposure level and the level at which there is increased risk of adverse effects; and one can somewhat arbitrarily decide to use the ten-fold protection factor or 100 or 2 or 20. And I do not know the background of the ACGIH's decision to recommend a time weighted average of .2 milligrams per cubic meter.

Q. I mean, that's almost 25 times lower than the OSHA level, correct?

A. I think it is 25 times lower than the OSHA PEL.

Q. Have you seen any references with regard to TLVs or PELs that they're not to be viewed as fine lines between safe and dangerous?

A. I have not seen references to that. I think that setting exposure limits appropriately should be based on identifying levels at which adverse effects occur and then building in substantial margins of protection. And so, the

fact that the ACGIH has chosen a level substantially below OSHA does not in any way imply that the level at which adverse health effects of manganese occur is any different for the ACGIH than it is for OSHA. It may simply say they want to be more protective. In other words, they want a wider margin below the level at which there are adverse health effects.

Q. Do you know that's, in fact, what they mean?

A. No. I said that may well be what they have decided.

Q. You don't know?

A. I do not know offhand without going back and reading the history of their decision.

Q. Is there such thing as individual's susceptibility or vulnerability with regard to any neurological diseases that can be caused by manganese exposures?

A. There is hypothetically such individual variations, but I do not believe that any basis is known for claiming an individual is more or less susceptible to manganese. So, it's a hypothetical that is not known to be true.

Q. But as you sit here today, it's your opinion that there is no individual susceptibility to manganese at least for neurological purposes?

A. No. It's my opinion that we have absolutely no ability to identify who is more or less susceptible.

Q. I'm not disagreeing with you. I'm just trying to find out what your opinions are.

Do you agree that there are people or people in general have different individual susceptibilities to manganese exposures as far as what type of neurological outcome, if any, will happen to them?

A. No, I do not agree because the way you've asked the question presumes that we know that it exists; and the answer is we don't know that it exists. We have no ability to identify who has or does not have that susceptibility. It's a hypothetical that might exist. You might be very susceptible to manganese but we have no way of knowing that and I might be very susceptible and we have no way of knowing that either. It's just a hypothetical.

Q. Same true with smoking?

A. I'd have to say yes. I wish it were different. I wish we could identify who was going to get cancer and stop them from smoking.

Q. Well, how do you explain that some people that smoke a pack a day for five years get lung cancer caused by smoking; and some people that smoke for 60 years, you know, die at 100 from a car accident and never get cancer?

A. We have no idea how to explain that.

Q. How about individual susceptiblity?

A. Again, it's a hypothetical. It could be -- it could be that it's something about them. It could be nothing other than the roll of the dice. You roll a bunch of dice. Why does one of them come up six? Why does one of them come up one? It might simply be random. There are a lot of random processes. There could be a reason behind it we haven't discovered, but right now it appears to be a random process.

Q. I mean, not everybody that is exposed to asbestos gets the deadly form of cancer called mesothelioma; do they?

A. That's correct.

Q. Why is that?

A. Well, again, we don't know. To some extent risk is clearly related to dose. People who had higher doses of the amphibole fibers are at higher risk than people who have had lower doses. But as to why among people who have had heavy doses, some get mesothelioma and some don't, we simply do not know. It's possible there is susceptibility, but nobody can identify what it is. Nobody can say who's susceptible. It's also possible it's just a random process, luck of the draw.

Q. For manganese exposures does anybody know whether it's the cumulative exposures that matter or the higher

short-term doses?

A. Well, what we do know is the people that have high short-term exposures get their manganism quickly. There's very little lag time. We know that in a number of industries, high-level exposures achieve a sufficient dose in the globus pallidus that the onset of manganism follows very quickly. That's particularly important with respect to Catherine Godwin because her last welding exposure was four years before her symptoms were ever --

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

THE WITNESS: It was four years before her symptoms as I recall.

Q. (BY MR. LUBEL) So, we don't know whether it's the cumulative exposures that make the difference or the high short-term exposures?

A. Well, we know in some settings high short-term exposure is sufficient to cause it. We know in other settings high level long-term exposures are sufficient to cause it.

Q. Don't we see some circumstances even in the industries that you acknowledge a connection between manganese exposures and Parkinsonism where the workers have worked for 20 or 25 years doing essentially the same job, had exposures to manganese and then nothing happens and

then all of a sudden, pow, they have got a disease?

A. You'd have to show me examples of that.

Q. You've never seen any of those?

A. Well, I don't know from memory any that fit your description.

Q. But are you generally familiar with the literature enough to know that when you look at it, you don't see necessarily everybody within three months of working around manganese at high exposures coming down with neurological deficits that resemble Parkinsonism? You agree with that, right?

A. We've never seen that exact setting. We do know one from all the studies done in Taiwan that high level exposure led to manganism; and I think it was -- it was in the ferro manganese plant. I think earlier I said it was a battery plant. It was a ferro manganese plant.

MR. BISSELL: You did say that earlier.

THE WITNESS: Yeah, I think I got that wrong.

A. That people came down within months of their first exposure, exposure was very high.

Q. (BY MR. LUBEL) That's the exception to the rule in the literature; isn't it?

A. I don't think it's the exception to the rule. I think it's a clear example of what happens with high level

exposure. People can achieve a sufficient dose in the globus pallidus quickly within months, and they get sick quickly.

Q. How long does the manganese stay deposited in the lung?

A. In the lung?

Q. Right.

A. I don't know offhand.

Q. I mean, do you know if it's released slowly into the bloodstream?

A. Your question presumes all sorts of factors that I don't think we've established. The first is that it stays in the lung at all, which I'm not sure we know about.

Q. I'm asking you if you do know it.

A. Well, typically inhaled dusts that reach the gas exchange region are picked up by alveoli macrophages and those macrophages migrate up and out of the respiratory tract up to the terminal bronchioles and then they get caught in the mucus and go up and out of this area like an escalator. Sometimes those macrophages can migrate out of the alveoli and back into the interstitial tissue and get picked up by the lypmphatics and then get collected in the peribronchial lymphatic aggregations. And so, you typically see collections of dust, inert dust, that can't be degraded in those regions. I don't know and I'm not

aware that it is known whether that happens for welding fume. In other words, what I don't know is the clearance time of welding fume in the lung.

THE REPORTER: The clear what?

THE WITNESS: Clearance time.

THE REPORTER: Clearance time. I'm sorry.

A. In other words, how quickly the macrophages remove it and how much is left after, say, 24 hours, 48 hours, 72 hours.

Q. (BY MR. LUBEL) How long --

A. I simply don't know how long it stays, and I don't think anybody knows about whether manganese is released from any material that does persist in the lungs. It may simply be cleared out of the lungs rapidly and is gone.

Q. Once it gets to the brain, how long does it stay in the brain?

MR. BISSELL: Object to form.

A. Which "it" are we talking about now? Are we talking about welding fume?

Q. (BY MR. LUBEL) Just manganese.

A. Well, we've got to talk about those situations, if manganese gets to the brain, because it's not clear yet that welding fume gets to the brain or manganese in welding fume gets to the brain. There are many, many reasons why

that doesn't occur such as a lot of welding fume not being absorbed --

Q. Okay. And so --

A. -- welding fume being bound to proteins and being excreted in the kidneys efficiently, some obstacles to welding fume or obstacles to manganese crossing blood barriers. Lots of issues.

Q. So, welding fume -- the manganese portion of welding fume cannot get to a human's brain, correct?

A. No. Like I said, there are lots of obstacles that keep it from getting to the brain; and there are lots of welders in which whatever manganese exposure they get is handled physiologically and excreted without any appreciable accumulation in the brain.

Q. Well, if it gets to the brain, how long does it stay there?

A. I don't know exactly. I did not prepare on that topic.

Q. Have you attended any seminars on welding?

A. I'm not sure what you mean by "seminars on welding."

Q. Okay. Have you spoken to any groups of lawyers regarding any opinions you have on the welding cases?

A. No.

Q. Have you spoken at any industry meetings

regarding welding?

A. No.

Q. Have you met with any other experts regarding welding?

A. I have met other experts. I don't know what you mean meet "with."

Q. Who are the ones you've met?

A. I've met -- I think I've met Dr. Keyberts (phonetic).

Q. Anybody else?

A. I think I've met Dr. Olonew.

THE REPORTER: Say that name again.

THE WITNESS: Olonew, O-L-O-N-E-W.

Q. (BY MR. LUBEL) Have you spoken with him?

A. I think I was introduced. Basically said "Hello. Nice to meet you."

Q. Where was that?

A. I'm not sure at this point.

Q. Who introduced you?

A. I don't recall. It was at one of the trials. I don't remember which one. I don't remember where.

Q. Any other defense experts other than those two that you've met or spoken to?

A. I do not recall any others.

Q. Are you a member of any organizations?

A. Yes.
Q. What are they?
A. American College of Occupational and Environmental Medicine, Society for Epidemiologic Research the National Epidemiological Association, Michigan Occupational Environmental Medical Association. I think I'm a member of the International Society for Environmental Epidemiology. There are probably some others I don't recall.
Q. Have any of the organizations that you're a member of been affiliated with any of the epidemiological studies that have been done on welding?
A. I don't understand the question.
Q. Are you a member of IEU or something like that?
A. I don't know what IEU is.
Q. Okay. So, none of the organizations that you're a member of have had any part in any welding study?
A. I'm not aware that they have, nor am I aware if any of those organizations have any part in any study. It's not part of what they do to my knowledge.
Q. And you're not a member of any group that has played a part in any of the welding studies, correct?
A. I don't even know what that means.
Q. Okay.
A. What is a "group"?

Q. You're not a member of nor have you ever been a member of any organization that has played any role in any of those welding studies, correct?

A. It's hard to answer that. It's so vague.

Q. Okay.

A. I've been on the faculty of the International Epidemiology Institute, which is one of the entities that performed one of the welding studies.

Q. What was so hard about that?

A. 'Cause I have no idea what you mean by a "group."

Q. What do you call them?

A. The IEI.

Q. Yeah. What is it --

A. It's a --

Q. -- if it's not a group?

A. -- consulting firm.

Q. It's a consulting firm?

A. Yes.

Q. Are you paid by them?

A. No.

Q. What do you do for them?

A. I'm on their faculty. I occasionally talk with them.

Q. How does that group finance themselves?

A. They work.

Q. For whom?
A. Clients.
Q. Such as who?
A. Well, I think that they did a study on behalf of the welding consumable -- or the welding consumables manufacturing industries.
Q. Who else have they done work for?
A. I believe they do work for the National Cancer Institute by the Federal Government. I believe they do work for other agencies within the National Institutes of Health.
Q. So, their only private client was the welding group?
A. I don't know their client list. I believe they have other private clients.
Q. Do they have anything to do with your studies over at Ford or the Big Three?
A. No.
Q. As a faculty member, did you have access to the raw data from their welding study?
A. No.
Q. Can you get it?
A. No.
Q. Why not?
A. I have no right of access to the data.

Q. Have you asked for it?
A. No.
Q. Have you talked to any of the authors about the study?
A. Not until after it had been published.
Q. Did you know that they were working on it before it was published?
A. I was aware of that.
Q. How did you learn that?
A. I think I was told by attorneys for the welding industry that Dr. Fryzek was working on the study.
Q. Schachtman, Nathan Schachtman?
A. To the best of my recollection, I don't believe it was Dr. -- Mr. Schachtman who told me that.
Q. I call him Dr. Schachtman, too. I am not kidding you. I do. It's a joke, but I do. 'Cause he thinks he's smarter than you.
A. He may well be.
Q. He probably is.
MR. BISSELL: I know he's --
MR. LUBEL: He's --
MR. BISSELL: He's a lot smarter than I am.
(Laughter)
Q. (BY MR. LUBEL) Have you prepared any demonstrative aids for your trial testimony?

A. In this case, no.
Q. Do you have demonstrative aids from your prior trial testimony?
A. Yes.
Q. Have you brought those with you?
A. No.
MR. LUBEL: Can I get those from you, John?
MR. BISSELL: Probably.
MR. LUBEL: Okay.
Q. (BY MR. LUBEL) Have you ever done any work with the Navy?
A. No.
Q. Were you in the Navy?
A. No.
Q. You said you welded before?
A. I have.
Q. Where did you weld?
A. Washtenaw Community College.
Q. What were you doing there?
A. I took occupational medicine residents over to see welding so they could understand what it was about and we learned to weld.
Q. When was that?
A. Ten, twelve years ago.
Q. Was that a one-time deal?

A. Yeah.
Q. One day?
A. Yep.
Q. Were y'all trained before you welded?
A. Yes.
Q. Did they monitor your air?
A. No.
Q. Did they provide you with any respiratory protection?
A. No.
Q. Did you weld with any rods that had any potentially hazardous components, if you know?
A. Well, let's see. We did aluminum welding. It's arc welding. I'm not aware of -- well, every chemical has toxicity issues. Dose -- I'm not aware that we were exposed to anything under circumstances that would have created any hazard.
Q. Was your environment mechanically ventilated?
A. It had a general dilution ventilation.
Q. What is that?
A. It was a big room.
Q. How big?
A. 50 by 40 with a 20-foot ceiling, big room.
Q. Was it a garage door that was open? Was any of it open?

A. I don't recall at this point to be honest.
Q. Was there any manganese in the air?
A. Was there any what?
Q. Manganese in the air?
A. I don't know but -- I don't know. We also did some gas cutting on steel. So, if there was manganese in the steel, we may have generated some manganese fume.
Q. But you don't know?
A. No, I don't know.
Q. Are you relying on the work of any other experts in this case?
A. If you can answer the questions, it would save us both time.
Q. I assume it's no, but I want to hear from you.
A. I don't know. I don't believe I am. There are areas that are outside of my expertise that I may rely on such as exposure assessment and such as neurologists who have seen the plaintiff and reached a diagnosis.
Q. But as you sit here today, you've not relied upon anybody else's work, correct?
A. Not in forming my opinions, no.
Q. Have you authored any case reports?
A. In my life, yes.
Q. Right. How many?
A. I don't know. Small number.

Q. Less than five?
A. Probably.
Q. Are they on your resumé?
A. I'd have to look.
(Recess from 6:04 p.m. to 6:08 p.m.)
A. I'd have to say I don't think I have authored any case reports. I can't find any.
Q. (BY MR. LUBEL) What is a case report?
A. It's typically a written description of a single patient or a series of patients who have some aspect of their illness that is unusual or uncommon.
Q. Have you never seen anything that was unusual or uncommon?
A. I have.
Q. Not enough to write about it?
A. Apparently have never bothered to write them up.
Q. All right. Let me ask you this: You gave us earlier what you believe to be diagnostic of manganism, and what I'm curious from you is what would you expect a neurologist to have in his or her records to establish a diagnosis of manganism?
A. I would expect that the patient would have a clinical presentation characterized by the factors I testified to earlier today. So, the constellation of neurologic findings that were typical of manganism. I

would expect to have results from a diagnostic workup that excluded other causes of movement disorders. I would expect to have a history of working in an occupation that is known to put people at increased risk of manganism to expose them to manganese at levels adequate to cause manganism. And I might expect to see results of imaging studies that show hyperintense signal from the globus pallidus indicating excessive manganese content in that basal ganglia.

Q. Would you require the imaging study from the neurologist?

A. It would provide strong confirmatory evidence.

Q. But is it a necessary prerequisite to a correct diagnosis of manganism in your opinion?

A. It's a matter of probabilities. It should be done if one's going to say that a person has manganism and it should be -- I mean, it provides the strongest evidence that the toxin is present in the tissue that is responsible for the clinical syndrome. In the absence of that, I think that the absence of that finding seriously detracts from the confidence that one would have that that diagnosis is correct.

Q. But would you require an imaging study in order to assess or evaluate that, in fact, that person actually has manganism? In other words, could you agree with the

diagnosis of manganism if everything else is present except for the imaging study? That's a better way to put it, I guess.

A. I would consider two conditions. In a person who was currently exposed or currently employed in a job in which it was believed that there was manganese exposure, I would not accept the diagnosis of manganism in the absence of evidence of excessive accumulation of manganese in the globus pallidus. I think that would be inappropriate.

In someone that was no longer employed in a setting in which it was believed that there was excessive manganese exposure, the issue is the timing between exposure and the onset of disease. If the disease onset occurred in a manner that was plausibly related to the manganese exposure and the exposure had ceased in the distant past such that the manganese would likely have been cleared from the globus pallidus, I would not require that in the study.

Q. And generally how long does it take the manganese to clear that part of the brain with the understanding that it may differ for each individual?

A. It certainly takes months. I don't know whether -- I don't know what the upper end on that time is. Let's say one would expect it to have diminished substantially over a matter of years. One would expect it to remain elevated for certainly two, three, four months.

In between it's not clear, and it would depend on how much manganese had been there.

Q. Now, with respect to what you expect a neurologist to document in his or her records regarding the exposure, what would you expect to see?

A. I'd expect to see a detailed occupational history with the job description and the description of tasks performed. I'd expect there to be dates on that, and I would expect to see a history of holding jobs in which there was a reasonable basis for thinking it was overexposure to manganese. In other words, you'd have to work in job in which manganism is known to have occurred.

Q. Would you expect the doctor to inquire into whether there was confined-space exposure?

A. It would depend on the industry. If somebody worked in dry cell battery manufacturing, I'm not aware that confined spaces are an issue. Somebody working in manganese mining, I'm not sure that confined spaces are an issue.

Q. How about the use or nonuse of respiratory protection or equipment?

A. Well, you know, if we're talking about welding, that's an occupation that is not known to put people at risk of manganism. So, your questions seemed to be directed mostly toward welding environments such as

confined spaces and use of protective equipment. That's not an occupation known to put people at increased risk.

Q. So, you wouldn't expect the neurologist to perform a differential diagnosis to determine whether welding was the cause at all, would you, if they were welders?

A. You've mixed issues that I don't think have any relationship to each other. Differential diagnosis --

Q. Let me do it this way --

A. -- doesn't have to do with causation. It has to do with etiology.

Q. Let me do it this way --

A. It doesn't --

THE REPORTER: Wait --

A. Differential diagnosis doesn't have to do with etiology. It has to do with reaching a diagnosis.

Q. (BY MR. LUBEL) Let's put it this way: For the welder you don't expect a neurologist to perform any analysis of whether manganese could have caused a manganism induced Parkinsonism, correct?

A. The only circumstance in which it is reasonable to even think of welding as putting people at risk of Parkinsonism is in the setting that we described using high manganese rods inside a rock crusher without ventilation. And so, if you were the neurologist seeing those patients,

I would expect that there would be an exploration of the work and what the current job was and what the recent jobs were. And if there was a history of welding in confined spaces, I would expect that to be asked.

Q. Would you expect that same doctor to inquire under those circumstances into things as detailed as respiratory protection? I mean, because if somebody's in a space suit with fresh air even working in a confined space around the highest, you know, manganese content rods, you wouldn't expect that neurologist to go into those kinds of details; would you?

A. It wouldn't be unreasonable to ask those questions. But the hypothetical that you've posed is not realistic. It's very hard to go into a confined space in a self-contained breathing apparatus because it's confined space, and you've got all this equipment on your back and very hard to move around.

Q. Really? You couldn't get inside a closet with one of those?

A. It would be very hard to move around and do a welding job in a SCBA in a closet.

Q. What about --

A. It would be --

Q. -- if --

THE REPORTER: Wait --

A. If we're going to talk about confined-space welding, we're back to a situation where an employer has a duty to ensure that the job can be done safely, and I would not think that wearing a SCBA when in a confined space would necessarily meet --

Q. We're getting off track --

A. -- with those conditions.

Q. (BY MR. LUBEL) I'm just trying to find out if you expect a neurologist to assess the conditions by which the exposure took place and whether any respiratory protection was used at all in evaluating whether it's a manganese induced Parkinsonism or not. That's all I'm trying to figure out.

A. I think the neurologist should get an occupational history and should seek to identify occupations in which it is known that people can be overexposed to manganese. Now, welding -- as it is done in virtually every setting that I've ever seen and as it is done in all of the epidemiology studies that have been conducted -- does not fulfill those criteria. It's not known to put people at increased risk. I would not expect a neurologist to devote a lot of effort to exploring that occupation because that occupation is of no relevance to the risk of manganese.

Q. Okay. So, all the neurologist needs to do is

say, "Although this constellation of symptoms could be attributable to manganese, it doesn't apply in this particular circumstance because the guy or the lady is a welder." That's all they need to do, right?

A. I'm not sure we've established what constellation of symptoms we're talking about. Are we just talking about Parkinsonism in general, the big umbrella term?

Q. The symptoms you say are indicative of manganism. You've told us what they are.

A. Okay. So, we're talking about that particular constellation of symptoms and signs?

Q. Correct. Let's say those symptoms and signs exist, not all of them because you said not all of them had to exist but a number, enough of them to where if the manganese exposure was high enough, you'd link it.

So, my question is: If the constellation of symptoms could be indicative of a manganese induced disease and the neurologist says, "What did you do for a living" and the man or lady said, "I was a welder," you would not expect a neurologist to go any further. That's what I'm trying to get you to say, if that's what you believe.

A. If a patient presented to me and had that constellation of symptoms and signs that fit very well with manganism, I would explore in detail their current and past occupations for a number of years, not only to know the job

but to know the materials they worked with, the tasks they performed, and the circumstances under which they performed them. And the reason is if you have a diagnosis that strongly suggests an etiology due to a chemical agent, then the level of inquiry about could you have gotten exposed to that agent should be in greater depth. That's appropriate to do.

But if you've got a constellation of symptoms and signs that is not tightly focused on that disorder, say manganism, then the inquiry into potential exposures or past jobs would typically not be with as great precision and with as great depth. I mean, I spend my career trying to explore occupational histories with patients to uncover what their exposures have been for that same purpose.

Q. And what I'm trying to find out, Dr. Garabrant, is when they perform this in-depth analysis when there is the constellation of symptoms that could be reflective or indicative or consistent -- or whatever term you want to use -- to manganese exposure, do you believe that information is supposed to be recorded in the medical records?

A. There's no one answer that fits all situations. The answer has to do with how tightly that constellation of symptoms and signs fits the syndrome that is known to result from toxicity due to that agent. If this is a -- if

this is a constellation of symptoms and signs that -- let's not do it as a hypothetical. Let's do it as manganism. If you see a patient and they fit manganism perfectly and --

Q. Cock-walk and all?

A. Cock-walk and all.

Q. Okay.

A. You should spend considerable effort to explore their exposure history not only in the occupational setting but drinking water and pesticide spraying and you name it. You really --

Q. I'm not disagreeing with you --

A. -- should go at it with a great deal of interest. In my practice --

Q. Wait a second, Doctor.

A. -- commonly --

Q. We can end this quickly. I just want to find out if that analysis that you're giving us is supposed to be recorded in the medical records of the doctor. That's all I'm asking.

A. If you ask the questions, you write down the answers. The issue is -- perhaps I'm not understanding --

Q. I'm asking you a simple question. Okay?

MR. BISSELL: He just answered it.

A. Yeah. If you ask the questions, you write them down. You write down the answers.

Q. (BY MR. LUBEL) That's what I'm trying to get.
A. I didn't understand that.
Q. Okay. I'm not trying to be horsey or disrespectful. I am just trying to get us out of here sooner, including you, since you're going back to another state.
(Garabrant Exhibit Nos. 22 and 23 were marked)
Q. (BY MR. LUBEL) Exhibit 22 comes out of one of your books. Mr. Bissell was kind enough to make a copy of it, "Standard Occupational Classification Manual, 2000"; is that correct?
A. Yes.
Q. All right. And I have taken Exhibit 23, which is six three-ring binders of the remaining articles that you brought with you today, correct?
A. Yes.
Q. And then I've left behind you -- because I'm not going to mark them --
A. Wait a minute. You've also got a seventh binder marked as a separate exhibit.
Q. But it's already marked. I'm saying I marked Exhibit 23 as the remainder of the articles that you brought, right?
A. Yes.
Q. All right. Now, then, behind you is two boxes of

medical records and information that's related specifically to Mrs. Godwin, correct?

A. Yes.

Q. And you understand that in this case at least so far you're not going to offer any specific opinions to Mrs. Godwin short of you don't believe that her disease could have been caused by welding exposure, correct? Isn't that what you told me earlier?

A. Correct. Except that I don't know what you mean "short of" other than --

Q. In other words, you've not been asked to assess her diagnosis?

A. That's correct.

Q. And you hadn't done so, correct?

A. That's correct.

Q. You've not examined her?

A. That's correct.

Q. You've not reviewed her records in a light to determine whether she's, in fact, correctly diagnosed as PD, Parkinsonism, manganism, or anything else, correct?

A. I have reviewed her records to understand how her diagnosis was reached.

MR. LUBEL: John, can you just stipulate that that's not his role in the case?

MR. BISSELL: Yeah. I think so. I mean, if

I understand what "that" is. I mean, he is not offered as an expert to make or refute a particular diagnosis in her case.

MR. LUBEL: Let me tell you what I'm doing. When I look at the disclosure responses on David Garabrant, there is nothing in there that leads anybody to believe -- including me even with my pea brain -- that he's going to make specific opinions on Ms. Godwin?

MR. BISSELL: I don't think he is. The question is so broad that it might rule out something that I'm just not clear -- I think we understand. He is not here and will not be tendered to offer specific Godwin opinions.

MR. HENDERSON: Other than what he already said?

MR. BISSELL: Other than what you have talked about today in that he knows she was a welder. He knows she welded in the Navy; and, you know, he has opinions based on what the literature shows and what he believes about capacity of certain exposures to cause disease. Now, she fits in some of that in there but he's not working from her out. He's working from what he knows and as it may apply to her. He's not --

MR. LUBEL: But he's really not working on her. He's really --

MR. BISSELL: Right.

MR. LUBEL: -- going to express what the study shows, and then y'all can argue about where she fits is what I'm --

MR. BISSELL: I think that's -- doesn't that sound fair to you?

THE WITNESS: Let me put it this way: It is my opinion based on reading her records that she does not have manganism.

MR. LUBEL: See, now, we're -- see, that's the kind of stuff that I would expect to see in the disclosure. Y'all go talk.

MR. BISSELL: Let's talk a short break.

(Recess from 6:30 p.m. to 6:34 p.m.)

(Garabrant Exhibit No. 24 was marked)

Q. (BY MR. LUBEL) Can we agree that the opinions that you offer as you understand them today to be limited to the disclosure that was made by your lawyers that I've marked as Exhibit 24?

A. Yes.

Q. Despite the fact that it's getting kind of late and it's hot in here, have I been courteous to you?

A. Yes.

MR. LUBEL: Thank you for your time.

MR. BISSELL: We are going to reserve our

questions until the time of trial. Specifically we will offer at trial evidence relating to Dr. Racette's study related to a comparison of welders in Alabama to a Copiah County, Mississippi, control group.

(Deposition concluded at 6:36 p.m.)

CHANGES AND SIGNATURE

PAGE/LINE	CHANGE FROM/CHANGE TO	REASON

I, DAVID H. GARABRANT, MD, have read the foregoing deposition and hereby affix my signature that same is true and correct, except as noted above.

_____

DAVID H. GARABRANT, MD

THE STATE OF _____)

COUNTY OF _____)

Before me, _____, on this day personally appeared DAVID H. GARABRANT, MD, known to me to be the person whose name is subscribed to the foregoing instrument and acknowledged to me that they executed the same for the purposes and consideration therein expressed.

Given under my hand and seal of office this ____ day of _____, 2006.

_____

NOTARY PUBLIC IN AND FOR THE STATE OF _____

NO. 03-CV-0801

RICK MONTALBANO, ET AL.	*	IN THE DISTRICT COURT OF
	*	
VS.	*	GALVESTON COUNTY, TEXAS
	*	
THE LINCOLN ELECTRIC COMPANY, ET AL.	*	56TH JUDICIAL DISTRICT

REPORTER'S CERTIFICATION
ORAL DEPOSITION OF DAVID H. GARABRANT, MD
OCTOBER 19, 2006

I, Suzi Gladney, a Certified Shorthand Reporter in and for the State of Texas, hereby certify to the following:

That the witness, DAVID H. GARABRANT, MD, was duly sworn by the officer and that the transcript of the oral deposition is a true record of the testimony given by the witness;

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 _____, 2006;

That the amount of time used by each party at the deposition is as follows:

Mr. Lubel - 4 hours, 47 minutes

That pursuant to information given to the deposition officer at the time said testimony was taken, the following includes all parties of record:

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I further certify that I am neither counsel for, related to, nor employed by any of the parties or attorneys 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
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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.

_____
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